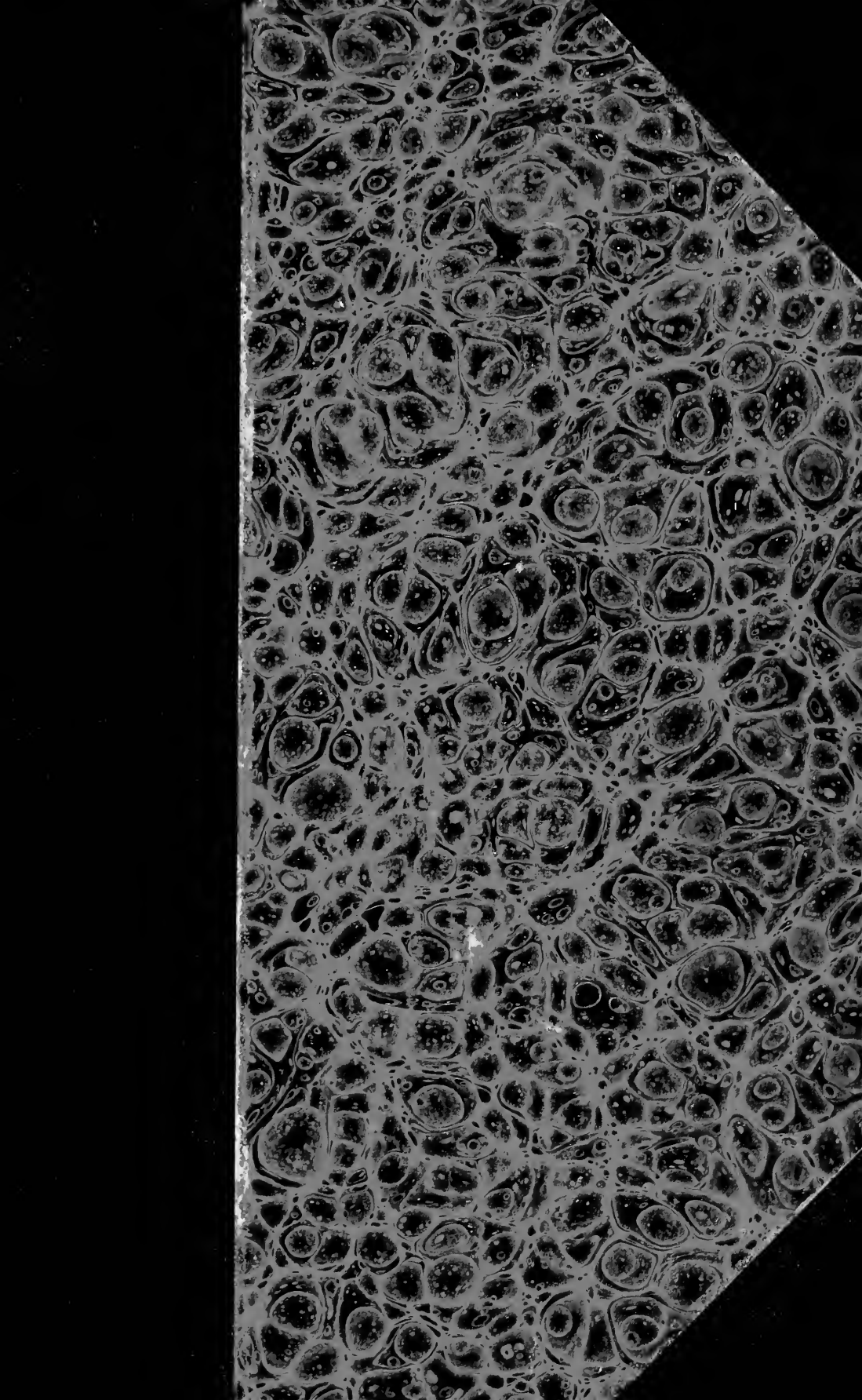

A

PRACTICAL TREATISE

ON

MEDICAL DIAGNOSIS

FOR STUDENTS AND PHYSICIANS.

BY

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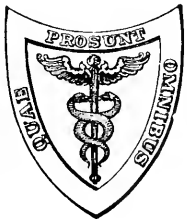

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TO THE

MEMORY OF MY FATHER

BENJAMIN MUSSER, M.D.,

AND

MY GRANDFATHER

MARTIN MUSSER, M.D.

PREFACE TO THE FIFTH EDITION.

THE most effective way in which an author can evince his gratitude for favor shown to a book is to keep it a fair exponent of its subject. In each successive edition of this work, accordingly, no effort has been spared to maintain it abreast of a department of basic importance and incessant advance. The present volume embodies a most thorough revision, embracing not only every detail of the text, but also a fundamental rearrangement, planned with the object of explaining the subject in the most logical and natural sequence, and facilitating thereby its comprehension. The department of illustrations has likewise been improved.

The original purpose of the work was to make it an exponent of objective medicine, and to point out the way to acquire *precision* in diagnosis by means of modern methods. It emphasized the paramount importance of the clinical laboratory, public and private, then relatively rare, and perhaps was instrumental in extending such facilities, so that nowadays any physician in town or country can equip his own laboratory, and at once simplify his practice and increase its success. No expensive outfit is necessary, as the entire plant can be placed in the corner of an office, and indeed much of the work can be done at the bedside. In the earlier editions greater stress was placed upon bacteriological diagnosis and the principles that underlie it than was customary in similar works. The object was to impress upon the profession the essential advantages to be derived from this method. That which was then a new story is now universally recognized, hence in the present edition the principles of diagnosis have yielded space for greater elaboration of the practical features. So many new and valuable methods have been developed, and so many new points of importance have arisen, that in spite of earnest efforts at condensation, the information presented has required increase of space. Laboratory methods, the application of the *x*-rays, and the later developments of physical diagnosis have been especially detailed.

Experience as a consultant and as a teacher has convinced the author that as practitioners we must hold ourselves to the highest pitch of energy and effort. Any lapse in methods or laxity of application means failure, partial or complete. To minimize this, and to provide for the exigencies and strain inseparable from the most responsible of the professions, a

tabulated scheme of diagnosis has been furnished, by means of which every method may be at the command of the practitioner, and none may be overlooked because of wearied brain or body.

The entire work is divided to accord with the natural employment of the methods. Hence we have in sequence Historical Diagnosis, Subjective Diagnosis, Objective Diagnosis, Physical Diagnosis, and Laboratory Diagnosis. By observing these methods exhaustively we find not alone the disease, but also the *health-value* of the patient, that upon which restorative efforts must be based. True diagnosis estimates the morphological and physiological characteristics of the patient and recognizes the cause as well as the effect of disease.

I am again indebted to many colleagues for valuable services: to Dr. Joseph Sailer for assistance in the chapter on Nervous Disease; to Dr. W. C. Posey for the chapter on the Eye; to Dr. A. O. J. Kelly for suggestions in the sections on Cardiac Disease; to Dr. J. Dutton Steele for aid in the chapter on Gastro-intestinal Disease. Professor Allen J. Smith very kindly made for me many illustrations and revised the chapter on Animal Parasites. To Dr. Henry Pancoast I am indebted for the section on *X*-ray Diagnosis. Dr. R. Max Goepp, Dr. Myer Solis-Cohen, and Dr. W. E. Rahte rendered valuable literary services, and the latter prepared the index.

Finally, I must express renewed acknowledgment to the publishers, Messrs. Lea Brothers & Co., for their uniform courtesy, their great resources, and most of all for such devotion to the interests of the book itself as can only be appreciated by those who have assumed the responsibilities of authorship.

J. H. M.

1927 Chestnut St., Philadelphia,
April, 1904.

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GENERAL DIAGNOSIS.

SECTION I.

GENERAL CONSIDERATIONS.

CHAPTER I.

THE DATA, METHODS, AND OBJECTS OF DIAGNOSIS.

THE sufferings of one who consults a physician are indicated by *symptoms*. The symptoms of which the patient complains are known as the *subjective symptoms*. The symptoms which the physician observes are known as the *objective symptoms*.

The symptoms have a history. It may be the brief one of sudden onset, or a long one of rise and fall, of ebb and flow, of the mingling of complex phenomena from time to time. The story of the evolution of the disease is written as the *history of the present disease*.

The present disease may be due to or modified by previous disease. We may be consulted for the effects of one link in a chain of morbid disorders which began in infancy or early adult life. We should learn, therefore, of the occurrence of *previous disease*. Certain types of constitution and some few diseases are transmitted by parents to offspring; we should, therefore, inquire into the *family history*. A farther insight into the nature of the suffering may be obtained by a knowledge of the age, sex, habits, occupation, environment, etc.—in short, by a knowledge of the *social history*—for, if the cause of the disease under consideration is determined, a distinction from other affections with allied phenomena can frequently be made.

The *subjective symptoms*, the *history of the present disease*, the *previous history*, the *family history*, and the *social history*, are learned by *inquiry* of the patient or the friends of the patient by methods and within limitations hereafter to be described. It is proper that they should be ascertained, if practicable, before the objective symptoms are studied.

After the history of the patient has been ascertained in full, the

objective symptoms are sought for by the use of the senses of sight, of touch, of hearing, with the instruments of precision to aid them—the *physical examination*, and by chemical and bacteriological methods.

The phenomena of disease are ascertained, therefore, by INQUIRY and by OBSERVATION. The facts or data thus collected and the discriminate interpretation of them constitute *diagnosis*.

Methods of Diagnosis. The facts of the *social history*, the *family history*, the *previous medical history*, and the *history of the present disease*, along with the subjective symptoms, are those upon which the old practitioners, prior to the nineteenth century, depended upon largely to establish a diagnosis. They are obtained by the employment of the mind alone, and their value is based upon experience and analogy and is subject to many fallacies. The diagnosis rested upon knowledge acquired from the recorded experiences of others, as well as that of the inquirer, and was in a sense *empirical*. Conclusions based upon such knowledge were arrived at by a process of deductive reasoning, a method at present of little, if any, consideration in scientific research. Such facts are gained almost exclusively by narration; their truth or falsity cannot be proved by observation or experiment; they are valuable only as a check upon the facts acquired by other methods; they are facts of history. The collection and interpretation of these facts represent the first or *historical* method of diagnosis.

The *subjective symptoms* likewise are gained by inquiry. Their value as facts upon which conclusions may be based is considered in Chapter VII., Section III. It is interesting to observe that they are symptoms that may be of psychic origin and hence can be simulated. No organic disease is necessary for their production. Inasmuch as they constitute a very important array of facts, and the methods of ascertaining and analyzing them differ from the methods to ascertain and analyze other facts known to be of more precise value, the collection and interpretation of the subjective symptoms constitute the second or *subjective* method of diagnosis.

The *objective symptoms* consist of signs evident of themselves, and of signs elicited by special methods of exploration. The collection and interpretation of the signs evident of themselves represent the third or *objective* method of diagnosis.

Signs not at once manifest, but elicited by special methods of examination, are known as *physical signs*. They are restricted to phenomena secured by physical exploration, chiefly in the examination of the chest and abdomen, hence we have physical signs of diseases of the chest. The collection and interpretation of facts obtained by this method constitute the fourth or *physical* method of diagnosis.

Modern methods of investigation have given to medical science instruments of research which, to secure accuracy, are employed in the laboratory. The obtaining and interpretation of facts by this method make the fifth division of our subject, and constitute *laboratory* diagnosis.

Object of Diagnosis. The object of diagnosis is to determine the condition of the living patient who may be suffering from disease.

Diagnosis implies that the phenomena of disease are detected—*clinical diagnosis;* that the effects of the disease on the organism and the nature of the morbid process are ascertained—*pathological diagnosis.* In addition to naming the disease and its cause, we should include in the diagnosis a determination of the stage of the disease and the recognition of its complications. Even this is too restricted an idea of diagnosis. It should include also the recognition of the cause of the morbid process; in other words, the *ætiological diagnosis.* Moreover, diagnosis implies such a knowledge of the patient's condition as will enable the physician to estimate the possible dangers and the outcome of the disease—the *prognosis.*

The diagnosis is made not only to give the disease a name, but also to treat it; and as it is not disease that we treat, but a patient with an ailment, a full knowledge of the patient and of his environment, mode of life, habits, occupation, etc., should be obtained by inquiry.

The practical result of diagnosis is the ability to remove or prevent the occurrence of morbid processes, or to mitigate their effects by rational therapeutics.

Methods of Reasoning. But we should not only secure facts, we should also be able to utilize them for analysis and induction—the result of which is the formation of the diagnosis. The diagnosis is obtained by three methods—the *direct*, the *indirect*, and the *differential.* In the direct method the data collected are sufficient to warrant a positive conclusion. An indirect diagnosis is made by *exclusion.* Thus, a symptom-group may represent several diseases, and each affection must be passed in review and excluded until one is found which closely corresponds to the data of the case under consideration. It is not one condition, because of the absence of certain symptoms; it is not another, because of the presence of certain essentially different symptoms: and so a negative proposition is proved. By the *differential* method the diagnosis of one of a few possible diseases is made after passing in review the positive and negative data. The direct method is scientific, rational, and the most practical. It is a process of purely *inductive reasoning.*

Clinical Sense. It is seen that observation and reasoning are essential in diagnosis. Something more is desirable, though not possessed by all observers. Long-continued precise observation develops in the student a *clinical sense.* He acquires the ability to place a precise value upon the symptom or sign, and to form with the various facts secured a clinical picture of the disease under consideration. The student will soon learn that under varying conditions the same symptom may have different values in different individuals. In consequence, the picture is true or false as the value is properly or improperly rated. So rapidly does the observer with this gift arrive at conclusions that it is often said he is a diagnostician by intuition. If such a faculty were possible, it would be a valuable possession; but the ability to make a "snap" diagnosis is acquired only by patient, repeated observation, by systematic arrangement of the knowledge acquired and by fixing it on the mind. Observe; place in order; fix in memory; reason.

Diagnosis sometimes Impossible. Notwithstanding our efforts to collect data by inquiry and observation, we are often unable to make a diagnosis. This arises when premises are wanting for the process of induction. The subjective symptoms may not tally with the known processes of disease; or the narrator of the history of the present disease may omit important evidence from lack of memory or knowledge, from design, or for other reasons. The objective phenomena may be developed in an ill-defined way; or they may be obscure, as the state of the abdominal contents in a person who is obese; or they may point to one or more processes the subjective symptoms of which are not present. At the time of observation the disease may not have developed fully, may not have "spelled itself out." Under these circumstances a provisional diagnosis must be made and the final conclusion held in abeyance. If there is a suspicion that the patient is suffering from a contagious disease, the latter should for sanitary reasons be given the benefit of the doubt; and if the patient's condition requires prompt remedial action, the symptoms must be viewed first of all as possible indications for therapy.

Avoid Haste. If prompt action is not required, haste should be avoided. It is not necessary to make a diagnosis at once, and it is not a confession of ignorance if time is asked before an opinion is given. Repeated observation and reflection should be employed before a conclusion is arrived at. This applies particularly to those conditions which result from improper environment, for the proper detection of which social data, knowledge of temperament, etc., must be acquired. Then, again, it may be necessary to observe the patient under changed circumstances, or to study the effects of diet on renal secretion or the function of other organs. Haste leads to faulty diagnosis, and therefore to misdirected therapeusis.

Diagnosis should not be Limited. It is not sufficient to give a name to a group of symptoms and be satisfied that the diagnosis is made. Every method should be used to collect data. The exact physical condition of the patient must be ascertained and the functional powers of all the organs correctly determined. We thus learn if the more evident disease is the single expression of a morbid process, or if it is only the surface storm, the currents of which are underneath. A pleurisy or pneumonia may be the outcome or a complication of a latent nephritis. A peritonitis may be the sequel of an appendicitis or pyosalpinx. Or diseases due to the same process may exist at the same time in two or more organs, as suppurative pleuritis and pericarditis. In such a case it would obviously not be sufficient to recognize one of the affections alone.

For purposes of treatment it is not sufficient to recognize a neuralgia or a spasm. The state of the patient on account of which the neuralgia developed must be ascertained. Attention should be called to the importance of not being lulled into a false security by the belief that the diagnosis of the first day is sufficient. Complications may arise or the morbid process invade new territory. Thus, in the course of pneumonia, in a few days a meningitis may arise or an ulcerative endocarditis ensue.

Modern Diagnosis. Anyone who takes the trouble to recall the methods of diagnosis in use twenty years ago will be struck by the wonderful expansion of the means now at hand to unravel the mysteries of disease. Then, a few instruments of precision and a few chemical reagents were required. The microscope was employed to examine only a few of the excretions and the blood. Now, the instruments of precision are multiplied and the scope of their explorations is increased. Chemistry among other things helps to fathom the mysteries of gastric disease. The microscope has extended its domain, and, with the new methods of staining fluids and tissues, has become the key that unlocks many of Nature's secrets. The new science of bacteriology has come to our aid, and now, instead of waiting to establish a diagnosis until an epidemic counts its victims by hundreds, it is obtained at once.

Certainty in diagnosis, for these reasons, has made a decided advance. The number of diseases that can be positively diagnosticated has increased. Methods of investigation and new instruments of precision are increasing daily. May we not hope that in the future the horizon of absolute knowledge will be extended far beyond its present limits? New instruments and new methods will surely avail.

The use of the large number of instruments that are essential, and the chemical and bacteriological examinations that are made, requires much time. Often the diagnosis is a question of hours, or even of days. The patient profits thereby. The tax on the physician is far greater than it was a few years ago. The bedside labor is great, and, in addition, he must have a laboratory at his command for microscopical, chemical, and bacteriological work. The outcome is that the scientific physician must have a *clientèle* limited in number, or else have one or more assistants to aid him in his investigations. Without doubt the latter will soon occur. Not as in days of old will we find in the practitioner's office the apprentice, compounding drugs and rolling bandages, assisting in the operations of bleeding and dressing ulcers, but the highly trained, scientific assistant, who by labors in the laboratory and at the bedside has become competent to collect data suitable for scientific methods of reasoning.

Case Records. Records of cases should be kept for obvious reasons. The habit compels a general survey of the case, and tends to prevent oversight in the examination. It naturally aids in the training of the powers of observation. It teaches precision in the narration of cases. The memory is aided by repetition and by lack of haste in ascertaining phenomena. The data are on record for more mature reflection, and to aid in the study of the literature of similar cases. The record is of value in case the patient returns for advice after a lapse of time. It may be of medico-legal value. The mental effect on the patient is good, for the taking of notes requires time and accurate, studied observation. In case it is desired to study a large number of cases, records are scientific data. The records may be kept on loose sheets and filed for future use. When a sufficient number are secured they may be classified and bound in volumes devoted to the various diseases, or they may be noted in a blank-book. At the end of the year the book is indexed according to the diseases and the names of

the patients. A better method is the card-system. The cardboard should be six by eight inches. One card is devoted to each case, although more can be used. They are arranged and catalogued according to the library system of card catalogues.

Method of Record. A systematic plan should be pursued in noting the cases. It need not correspond to the lines of inquiry in the examination of the patient, which are modified by the circumstances of the case.

The following outline explains itself. The various data should be recorded in sequence, and in such manner that the facts of each line of investigation can be readily culled for review and analysis. (See Chapters II. and III., Part I.)

RECORD OF CASE No. —.

Diagnosis. *Result.*

Name and residence, place of birth, and former residence.

I. SOCIAL HISTORY: Age, sex, race, married or single, children, the number and health; miscarriages.

Occupation: Present and previous home surroundings, sanitary conditions, etc.

Habits: Tobacco, alcohol, tea, narcotics; sexual habits; regularity of meals; character of food and method of eating; number of hours of sleep, degree of fatigue; brain-use, exercise.

II. FAMILY HISTORY: Hereditary tendency; health of parents, brothers, sisters, etc. Cause of death and age at which it occurred.

III. HISTORY OF PREVIOUS DISEASES: Character of convalescence from; syphilis and gonorrhœa; injuries.

IV. HISTORY OF THE PRESENT DISEASE: Data, mode of onset, and probable exciting cause of present trouble; evolution of the disease to date of examination.

V. THE PRESENT CONDITION:

A. *Inquiry:* The *subjective symptoms.*

B. *Observation:* The *objective symptoms.*

External appearance, development, color, figure, height and weight, attitude, expression of face.

Temperature, perspiration, eruption, swelling. Condition of limbs and joints.

Examination of the *digestive apparatus:* Mouth, tongue, gums, and pharynx; abdominal organs; contents of stomach; feces.

Examination of *respiratory apparatus:* Nose, mouth, and larynx. The lungs: inspection, palpation, percussion, auscultation, mensuration. Cough and expectoration.

Examination of *circulatory apparatus:* Inspection and palpation of cardiac area, percussion, auscultation of heart; similar examination of arteries and veins; the pulse; examination of the blood.

Examination of the *urinary apparatus:* Kidneys, ureters, and bladder; examination of urine.

Examination of the *nervous system:* Intelligence, subjective nervous phenomena, sleep, gait, station, reflexes, paralysis, tremor, pain, convulsions, headaches, disturbances of sensation, disturbances of speech. The organs of special sense.

Examination of fluids obtained by puncture.

Bacteriological examination of blood, sputum, secretions, exudations, etc.

Diagnosis.

Prognosis.

Treatment.

CHAPTER II.

THE MORBID PROCESSES AND THEIR SYMPTOMATOLOGY.

Although we may have secured all the data obtainable by inquiry and observation, and, if possible, made a diagnosis based upon them, it frequently happens that the conclusion arrived at is not final, and perhaps from the nature of the case can not be. We are prompted, therefore, to view the case from a different standpoint, to utilize our knowledge of the phenomena of morbid processes, and for the purpose of comparison to review the features that apparently resemble those of the process under consideration. Thus, for instance, in an obscure case of fever, the objective and subjective phenomena have been fully inquired into—we are unable to decide whether the disease under consideration is a septic process with an obscure lesion, a form of miliary tuberculosis, or malignant endocarditis. The known symptoms of each are considered—our knowledge of such symptoms depending upon our knowledge of the phenomena of the respective morbid process—and compared with the symptoms presented by the case in question. In this manner a *diagnosis by exclusion* is made. Moreover, after a diagnosis is made, a review of the symptomatology of morbid processes serves as a check upon the conclusions that have been reached. We should also, after making a diagnosis, compare the symptoms of the process as exhibited in the patient with the symptoms which we know to be common in the suspected disease.

It is necessary, therefore, that the student should fully know the symptoms of morbid processes. Each process is characterized by special phenomena by which it can be recognized. The symptoms are modified by the functional and anatomical structure of the organ in which the process takes place. Thus the pathological products of inflammation of the mucous membranes of the bronchial tubes and of the stomach are the same; but the symptoms differ because of the difference in their functions, and hence we have cough in the former case, and vomiting in the latter. Very frequently the symptoms differ because of the physical alterations. Thus inflammation of the pericardium is similar to inflammation of the pleura, but owing to the anatomical relations the pressure-symptoms of pericarditis are entirely different from the pressure-symptoms of pleuritis.

The morbid processes are not many. They include: I. Alterations in the blood and circulation. II. Disturbances of nutrition. III. Neoplasms (or abnormal growths).

I. Alterations in the Blood and Circulation.

The composition and distribution of the blood affect all the tissues for weal or woe. Changes in quality will be considered under Diseases of the Blood. Practically the symptoms, when the quality is affected,

are those of *anæmia* plus the physical and functional symptoms of the primarily diseased organ—as the spleen in leukæmia. The quantity of the blood may be increased or diminished.

1. Increased Quantity of Blood, or Plethora. Formerly this was considered an entity, and the symptoms of flushed face, hot and full head, throbbing pain, throbbing temporals, a full, strong pulse, sluggish intellect, were thought to indicate an excess of the general bulk of the blood. True plethora is rarely permanent. In transitory plethora the veins and not the arteries are over-filled. The symptoms are not due to general plethora, but to excess of blood-pressure or to special fluxions of blood to superficial vessels, determined by a nervous mechanism. Increase in one of the cellular elements of the blood, the leucocytes, is not a plethoric condition.

2. Diminished Quantity of Blood, or Anæmia. Anæmia embraces the diminution of the bulk of the blood as well as of the red blood-cells and their hæmoglobin.

The term might be used for loss of water from the blood, as in Asiatic cholera (see Infectious Diseases) or in serous purging. The symptoms are those of *collapse*.

Oligæmia and spanæmia are terms that may be used to define the general thinness or poverty—atrophy of the blood. Clinically, anæmia is divided into simple anæmia, general poverty of the blood; pernicious or idiopathic anæmia, reduction in the number of red cells; chlorosis, reduction in the quantity of hæmoglobin; leukæmia, relative loss of red and increase of white corpuscles. (See Diseases of the Blood.)

3. Local Disturbance of the Circulation. Hyperæmia or Congestion. The process may be acute or chronic. It is usually local, although it may be general; in the latter case many organs may simultaneously be involved from a common cause.

Acute Hyperæmia. The acute or active form of hyperæmia is always local and arterial. There is an excess of blood in the part. If the skin is the seat, there are redness and increased heat, and throbbing or pulsation may be seen. The parts are swollen. The excitability of the nerves is increased, with local symptoms of warmth, fulness, or itching.

The morbid blushing or flushing that occurs at the menopause or reflexly from internal disorder is a hyperæmia, and in erythema of the skin hyperæmia is also very marked.

Causes. Arterial hyperæmia is caused by (1) neuroparalysis of the inhibitory or vasoconstrictor fibres, of the cervical sympathetic, splanchnic, and other sympathetic and some mixed nerves, as the sciatic; (2) neurotonic stimulation of the actively dilating or vasomotor dilator nerves, as the chorda tympani. There is relaxation of the arterial walls. This may also occur directly through the vasomotor system, being induced by thermic, electrical, or chemical irritants, or from paralysis of muscular fibres after spasmodic contraction due to cold, as in frost-bite.

1. *Neuroparalytic Hyperæmia.* Destruction of the cervical sympathetic nerve by abscess, wounds, or a tumor pressing upon it, produces hyperæmia of one side of the face, with rise of temperature, and

contraction of the pupil. Later on the vascular conditions are reversed. Lesion of the fifth nerve or one of its branches causes hyperæmia of the iris, the conjunctiva, the cheek, the gums, and other structures supplied by it, with associate loss of sensation followed by atrophy. The sensory symptoms have nothing to do with the vascular paralysis.

2. *Neurotonic Hyperæmia.* After wounds of the brachial plexus hyperæmia of the fingers is seen. (See Fingers.) The local temperature rises and there is neuralgic pain. Local hyperæmia with hyperæsthesia, known as erythromelalgia, belongs to the same class of symptoms, being due to affections of the nerve-trunks or of the peripheral nerve-endings. It must be remembered that a reflex hyperæmia is possible.

Chronic or Venous Hyperæmia—Passive Congestion. The blood accumulates in the veins, and, by backward pressure, in the capillaries. The venous capillaries are over-distended, and, as compared with the arterial, much enlarged. They contain venous blood.

Any congested part, as the exterior, is bluish or purple in tint, often swollen (clubbed fingers), cooler than normal, with lessened sensation, and without pulsation. (See Cyanosis.) The dependent parts are first affected, as the legs, or the lungs. In fevers a weak heart and recumbent posture predispose to congestion of the lungs.

Causes. Obstructive heart and lung diseases cause *general* venous congestion. *Local* venous congestion is caused by tumors, the pregnant uterus, or collections of feces pressing upon the veins; it is also caused by inflammation of the veins, and thrombosis.

Local Anæmia. This may be due to arterial thrombosis or embolism, arterial obstruction through endarteritis, or to arterial spasm. Raynaud's disease is a form of arterial spasm. The grave effects of arterial obstruction are seen in cerebral anæmia from endarteritis, and in myocarditis from obstruction of the coronary arteries.

Œdema and Dropsy. The changes of the circulation which produce these conditions have been referred to in Chapter XXIV. (page 228). The symptoms and signs of the condition are also noted in the same place.

Thrombosis and Embolism. The student should be familiar with the symptoms of these conditions, and, what is fully as important, with the causes that give rise to them. Thrombi may form in the heart, the arteries, or the veins. Emboli may be formed either in the heart or vessels, but lodge in the vessels only.

Thrombosis. The *symptoms* of thrombosis are: 1. *Mechanical.* The channel is obstructed; hyperæmia, engorgement, œdema, and cyanosis arise. Its most typical form is seen in femoral thrombosis, with cyanosis and œdema of the leg. When an artery is obstructed, the symptoms are like those of occlusion under other circumstances (see Embolism); when a vein, the mechanical symptoms vary according to the particular vein affected. Thus in thrombosis of the coronary vein the heart's action is interfered with. In thrombosis of the portal vein, jaundice (not because of the obstruction), œdema (ascites), and congestion of mucous membranes (gastric and intestinal) occur, as from obstruction in any vein. In thrombosis of the cerebral veins

disturbance of the function of the brain is seen; in thrombosis of the pulmonary veins dyspnœa. 2. *Inflammatory or septic.* If the thrombosis develops secondarily to an inflammation of septic origin, as in the extension of an inflammation into the radicles of the portal vein from an abscess about the rectum or vermiform appendix, the liver would be infected with micro-organisms: an infectious inflammation, with chills, fever, sweats, and other phenomena of a septic character, would result (pyelophlebitis). 3. *Embolic.* From the thrombus emboli are sometimes swept off; hence, embolic symptoms arise in the course of thrombosis.

While thrombosis is easily recognized, as a rule, it is necessary to call attention to the very great importance of going a step farther and looking for the *cause.* A thorough knowledge of the causes of thrombosis often leads to the diagnosis of a thrombus when without such knowledge its presence would never have been suspected. The causes are not many: 1. Stagnation or stoppage of blood. It is seen chiefly in the veins and the heart. External pressure upon the veins, as upon the pelvic veins in pregnancy or abdominal tumor, upon the hemorrhoidal veins, upon the portal veins by tumor, upon the pulmonary veins by mediastinal tumor. It must be remembered that some change takes place in the internal coat of the vein also, but that the pressure is primary. Weakness of the heart is another cause of stagnation; feeble contractions lead to the formation of cardiac thrombi. 2. Changes in the vessel-walls. The change is usually inflammatory and often proceeds from wounds. If the wound was septic, the inflammation will be septic. In the heart, endocarditis; in the aorta, atheroma leads to the development of thrombi. 3. Entrance of a foreign substance into the vessels. A carcinoma or other new growth may extend into the veins. Micro-organisms penetrate the veins and cause inflammation and thrombosis, or infect a previously existing thrombus. The clot is then broken and distributed throughout the system, causing pyæmia. 4. Extension from an already existing thrombus. A clot enlarges by coagulating the blood next to it. A large venous distribution may become blocked, as, first, the uterine veins, then the internal iliac, then the external iliac, and after that the femoral—causing the affection which frequently occurs in the puerperal form, *phlegmasia alba dolens.*

Embolism. An embolus is a substance which is swept into and plugs a vessel. Emboli may be fragments of a blood-clot (thrombus), vegetations from valves of the heart, parasites, new growths which had entered the veins, fat, or air. If obstruction of the vessel alone is produced, the embolism is said to be *simple;* if a new process, as inflammation, accompanies the obstruction, it is *specific.* A fragment from a thrombus in the systemic veins may become an embolus and block the pulmonary artery; a clot or portion of valve-leaflet from the left heart may block a systemic artery, as a cerebral artery or the femoral artery or one of its branches; a clot in the portal vein may obstruct branches in the liver.

Symptoms. The symptoms occur suddenly and depend upon the particular artery obstructed. The cutting off of the blood-supply causes cessation of function beyond the point of obstruction. In pul-

monary *venous* embolism dyspnœa is pronounced, the heart's action is rapid and irregular, and many cases are said to be "heart-failure." In the middle cerebral artery the embolus causes aphasia and monoplegia or hemiplegia. In embolism of the pulmonary artery cough and hemorrhage with dyspnœa occur suddenly. The patient in whom this occurs usually has had antecedent mitral regurgitation and dilated right heart.

The blocking of an artery may lead to various symptoms. If, for instance, the main artery of the leg is blocked, anastomosis may be set up; if it does not, gangrene ensues. If an artery supplying any internal organ is blocked, anastomosis may occur if the artery is not terminal. If the artery is terminal, there results rapid necrosis or softening, as in the brain; gradual wasting, as of the kidney; or engorgement of the arterial area and diffuse hemorrhage. The latter is known as a *hemorrhagic infarct.* This may occur in the lungs (pulmonary artery), spleen, kidneys, retina, and, rarely, the intestinal canal. The symptoms of hemorrhagic infarct are swelling and hemorrhage. In the lungs there are physical signs of consolidation, with hæmoptysis, cough, and dyspnœa; in the kidneys, pain and hæmaturia; in the spleen, pain and at times enlargement; in the retina, blindness with ophthalmoscopic changes; in the intestine, pain and hemorrhage with sloughing of mucous membrane.

Infective emboli cause abscesses. *Capillary embolism* is seen in the skin and mucous membranes in many infective diseases, notably ulcerative endocarditis. *Fat-embolism* occurs in the pulmonary capillaries, and is due to fat-globules which sometimes enter the circulation in pregnant women or in patients with bone disease, as osteomyelitis, or fractures. The symptoms are those of intense dyspnœa. It may cause sudden death. *Air-embolism.* Air may enter wounds of the veins of the neck. It accumulates in the heart, and as the ventricle cannot contract on it, the blood is not propelled. Death takes place with the symptoms of heart-clot, the heart being in asystole.

Hemorrhage. Hemorrhage may be *arterial*, *venous*, or *capillary.* It may occur because the blood soaks through the walls, by diapedesis; or it may occur from rupture, or rhexis. Hemorrhage by diapedesis takes place in venous engorgement, stasis, and inflammation. It is the small, passive hemorrhage of congestion, as in pulmonary congestion from heart disease; it is venous or capillary; the blood is dark. Hemorrhage by rupture is arterial, venous, or capillary. If the artery ruptures, it has been torn by violence, destroyed by ulceration or suppuration, or it is the seat of endarterial change. Veins are also diseased, or their walls destroyed, before rupture takes place. Rupture of capillaries occurs from violence or great internal pressure. In death from suffocation the capillaries are the seat of hemorrhage because of the increased venous pressure. Such capillary hemorrhage occurs in typhus, hemorrhagic smallpox, and scarlatina. The state of the blood is sometimes the cause of hemorrhage, as in scurvy, purpura, and other conditions. *Hæmophilia* is a peculiar hereditary affection possibly due to the state of the blood; more likely, however, due to the condition of the bloodvessels.

The special forms of hemorrhage, and their symptoms, etiology, and

diagnosis, will be considered in the sections to which the names in the following list point :

Bleeding from the nose—*epistaxis.*
Vomiting of blood—*hæmatemesis.*
Bleeding from the lungs—*hæmoptysis.*
Blood passed with the urine—*hæmaturia.*
Blood passed from the uterus—*menorrhagia* or *metrorrhagia.*
There is also intestinal hemorrhage—*melæna.*

Hemorrhages underneath the skin are known as *petechiæ* if small, and *ecchymoses* or *suffusions* if large.

Hemorrhage into internal organs receives its name from the organ affected, and is known as a *parenchymatous hemorrhage. Apoplexy* is applied to hemorrhage into the substances of organs, particularly if it occurs suddenly and is localized—as pulmonary apoplexy, cerebral apoplexy, spinal apoplexy. Long usage has associated the term with hemorrhage into the brain, so that it is applied to that part alone by most writers. *Hæmatoma,* or blood-tumor, is a collection of blood that has coagulated in a cavity, organ, or tissue. (See Ear.)

Symptoms. The symptoms of hemorrhage vary in degree, depending upon the amount of blood that escapes from the vessel, and whether the hemorrhage is external or internal. By external hemorrhage we mean one which is accompanied by a discharge of blood visible to the bystander. An internal or concealed hemorrhage is not apparent by any outward sign of blood.

The symptoms by which *external hemorrhage* is recognized need not be detailed. The show of blood in situations or at times other than normal is sufficient. It must be remembered that arterial blood is bright red, venous blood dark. It must also be remembered that the character of the blood coming from internal organs is modified by the secretion of the affected organ. Thus the blood from the stomach is coagulated and black, like coffee-grounds; blood from the intestine, tarry. The general symptoms of the various degrees of external hemorrhage are similar to the symptoms of internal hemorrhage, which will be described later. Both vary with the rapidity of the flow of blood. If the bleeding is slow, large quantities may be lost and more or less profound anæmia result. It is often more difficult to determine the source of hemorrhage. The mode of recognition of the anatomical varieties of hemorrhage will be discussed under the respective systems which are the seat of the bleeding. Hemorrhage may take place into a cavity, as the stomach, bowels, or bladder, and after the blood has undergone changes it may cause symptoms of, and be discharged as, a foreign body.

Although *internal hemorrhage* presents vivid phenomena, they may not be characteristic, and its recognition is often impossible without some knowledge of the history of the case. The symptoms are complex. First, we have pain, a symptom due to rupture of a vessel or to the filling of a tissue with blood. In the beginning the pain is sharp, severe, and of itself may cause shock. In the second place, the symptoms due to loss of blood arise. After pain, sudden prostration ensues; pallor spreads rapidly; the extremities become pallid and cold; a cold sweat breaks out on the forehead; the features become pinched and shrunken; the pulse becomes weak and rapid, and later thready, or

disappears altogether at the wrist; the carotids pulsate; the heart throbs violently, and a diffuse impulse is seen, at first vigorous, soon like a slap against the chest-wall, and then it fades away completely. On examination of the heart and vessels so-called anæmic murmurs are heard. The patient is restless, and sighs and yawns frequently. The respiration becomes slow and shallow. Nausea and sometimes vomiting may occur. He may faint but once or repeatedly, to be restored again and again, or the syncope may terminate in death. In the intervals between the syncopal attacks the mind is clear. If, however, profound shock is associated with the hemorrhage, there is dulness or stupor; the intellect is dazed; otherwise delirium and agitation may be present. When the hemorrhage is profuse, convulsions may take place. The temperature of the body falls. If the patient has fever at the time, the temperature suddenly falls to or below normal. We have, therefore, the following conditions in hemorrhage: syncope, shock, and collapse. They may all be present in the same subject, or one or two may be absent. The same symptoms may, however, occur from other causes which must be excluded. Sometimes the shock may be due to the same cause as the hemorrhage. The causes of shock are so evident that they serve to distinguish it from the collapse of hemorrhage. They are injury, anæsthetics, railway accidents, surgical operations, perforative peritonitis, strangulated hernia, intestinal obstruction, profound mental impression, and pain.

Shock from hemorrhage must be distinguished from *concussion*. In the latter the intellectual disturbance occurs at once, and is more marked than the circulatory symptoms. The absence of the usual phenomena of hemorrhage serves to distinguish syncope due to concussion from that due to the many well-known causes of fainting.

There are many forms of internal hemorrhage sufficiently grave to have a probably fatal result, or at least to create alarming symptoms. In the chest, diseases of the lungs or of the aorta cause hemorrhage. In concealed pulmonary hemorrhage the blood accumulates in a large phthisical cavity. When the aorta or an aneurism ruptures, the blood may enter the mediastinum or the pleura. Under these circumstances a knowledge of the previous history is essential. Careful examination of the lungs and of the heart and bloodvessels must be made in a case which presents the above-mentioned symptoms of internal hemorrhage. Internal concealed hemorrhage into organs or cavities of the abdomen occurs in gastric, duodenal, and intestinal ulceration; in aneurism, and in ulceration of large vessels from septic inflammation around them. It must not be forgotten that alarming or fatal internal concealed hemorrhage may be due to hæmophilia or purpura.

II. Disturbances of Nutrition.

Hypertrophy and Atrophy. (See Size, Chapter XIX., Part I., and Muscles.)

Inflammation. Inflammation is a process largely attended with vascular alteration, but also with disturbance of nutrition. It may be acute or chronic. It is due to injury, mechanical, physical, chemical, or vital. The invasion of micro-organisms or the irritation of their

products is the most frequent cause in cases that come within the province of the physician. The symptoms are modified by the structure affected and by the cause of the inflammation. The intensity and the character also modify them. The classical symptoms—pain, heat, redness, and swelling—are indicative of the tissue-process. In addition we have exudation and alteration of function. *Pain* varies in degree with the sensibility of the part. It is increased by pressure or movement and by the functional activity of the affected organ. *Heat* is detected by the hand or surface-thermometer. In abscess within the peritoneum and in pyosalpinx it may be described by the patient as a ball of fire. The surface-temperature over an inflamed lung or pleura is higher than over the healthy side. *Redness* can only be observed in parts open to inspection, as the nasal, oral, faucial, and other cavities. *Swelling* is observed with the redness; it is shown by enlargement of the affected organ, if the latter can be measured by palpation or percussion. *Exudation* takes place from mucous surfaces, into serous cavities, into the connective or any affected tissue, or into tubes or channels (heart and bloodvessels, lymphatics). The symptoms are: characteristic discharges from mucous surfaces; pressure and physical signs from accumulation in cavities; symptoms of the obstruction of channels. Grave pressure-symptoms arise when the exudation presses upon the nerves, nerve-centres, or nerve-tracts (brain, cord, peripheral nerves). In simple and in tuberculous meningitis the pressure-symptoms are often more pronounced than the inflammatory. *Alteration of function:* the symptoms can not be detailed here; each organ and structure must be referred to. The function may be stimulated at first, but is soon perverted or suppressed.

General Symptoms. *Fever* is the general expression of the local process. It may be primary from reflex irritation of afferent nerves which influence the heat-centre and disturb the thermotaxic mechanism. It may be secondary, the product of inflammation (pus, toxins) irritating the centres. The degree depends upon the cause. Active inflammation may not be attended by fever.[1]

Suppuration. The character of the fever indicates the variety of the inflammatory process. In most inflammations the fever is continuous. When there is suppuration, however, it becomes intermittent or remittent. The presence of suppuration is also made known by the *hectic state*, in which the fever is attended by chills and sweats. The appetite is lost or impaired. There is also leukocytosis. The urine contains a large amount of indican. In obscure inflammations about the peritoneum the indicanuria points to a suppuration. While fever symptoms in inflammation are similar, save in degree and in the peculiar type of the temperature-range—intermittent, remittent, or continuous—*septic inflammations* are attended early by cerebral symptoms, prostration, and the typhoid state. (See Fever, page 350.)

As a corollary, when fever is present, local inflammation must be sought for. Chronic inflammations may only give rise to altered function and cause exudation (swelling, effusion).

Inflammation of Various Structures. The symptoms vary according to the anatomical and physiological peculiarities of the structure.

[1] Musser. "Abscess of Liver," Univ. Med. Magazine, 1892.

Mucous Membranes. Pain is not excessive; heat is complained of (rectum); redness is marked and varies with the intensity from bright to dark red; swelling is always present, and in narrow channels like the nose or the gall-ducts causes occlusion. The *exudation* is at first mucous, then mucopurulent, and finally purulent. Before exudation there is a stage of dryness. The microscopical appearance of the exudate varies with the anatomical character of the membrane affected. Its peculiar epithelium is always present, also micrococci, pus, red cells; exudate from the lungs or liver contains special crystals. The functions are impaired. Fever is usually not very high and is continuous. The causes are direct local irritants or congestions from external impressions (cold?).

Serous Membranes. Pain is extreme and may cause collapse. Heat, swelling, and redness cannot be estimated. The surface-temperature rises. Exudation occurs after a brief dry stage. The cavities—pleura, pericardium, peritoneum, joints, cerebrospinal canal—are filled, causing mechanical symptoms and physical signs. Fever is excessive in some forms. Function is impaired or abolished. General symptoms are more pronounced. Shock or collapse is common in peritonitis. The affections are always secondary to a general process (rheumatism), to infection, to disease of neighboring structures, or to Bright's disease, diabetes, cancer, scurvy, or other diathetic condition.

Inflammation of *muscles* (rare), of *connective tissue*, and of *glands* is characterized by symptoms common to the morbid process, with alteration of function.

Inflammation of *bone* and *periosteum* presents the same group of symptoms. The pain may be intense or of a dull, aching, or boring character.

Inflammation of the *heart* and *vessels* is also attended by the cardinal symptoms. When the central organ is the seat of the disease pain is not common, but in the arteries or veins it is of frequent occurrence. The most striking symptom, however, is the obstruction to the channels. It is characteristically seen in phlebitis, as of the femoral vein. Œdema of the leg and cyanosis reveal the obstruction. In the heart the acute process or the results of the process give rise to all the symptoms of obstructive heart disease.

Inflammations of the *nerves*, the *spinal cord*, and the *brain* are followed more strikingly by pressure-symptoms and by the symptoms of degenerations secondary to the inflammatory process. Hence, while pain and tenderness are present in the exposed nerves, increased irritability, then abeyance, perversion, or abolition of function are the principal signs of inflammation of these structures.

Inflammation of internal organs, *lung*, *liver*, *kidneys*, and *pancreas*, is made known by pain (minimum amount) and swelling (enlargement of the liver), and by change in the function, indicated by modifications of the respective secretions as well as by functional and physiological symptoms.

Local Death—Necrosis and Gangrene. If nutrition is not complete, the life of the cell is endangered, and the process known as necrosis or gangrene results. The nutrition is annulled: 1. By stoppage of the circulation. 2. By the direct action of an irritant which destroys the

cells. 3. By abnormal temperature. A combination of the three causes quickly produces gangrene. Stoppage of the circulation may be due to an embolus or thrombus, or to stagnation by pressure, or to capillary stasis alone. Sloughing and "bed-sores" ensue in the two latter instances; gangrenous eschars in the former. The cells may have been destroyed by corrosives or caustics, by heat or cold, or by bacteria. When decomposition takes place, as in retained and infiltrating urine, cell-destruction and sloughing ensue. All pathogenic bacteria cause necrosis to a greater or less degree. Frost-bite and burn illustrate the destructive power of abnormal temperature.

Nerve-lesions may give rise to trophic disorders and thus produce necrosis. We have, allied to bed-sores and known as decubitus, a form of necrosis in spinal cord diseases. The sloughing is extensive and rapid. Trophic disorders cause paralytic hyperæmia, and hence necrosis.

It must not be forgotten that debility, cachexia, and feeble circulation play a great part in assisting the local changes.

Gangrene of internal structures concerns us. This form is nearly always due to stoppage of the circulation. It is seen in constriction of the intestine from hernia or obstruction. It occurs in phthisis from thrombi. Clinically, we see it frequently in diabetes. The lung, the brain, and the intestines are most frequently affected.

The symptoms of necrosis or gangrene are modified by the tissue involved, the function interfered with. In external gangrene the decomposing structures emit a foul odor, there is rapid prostration, and the patient sinks into the typhoid state. Fever ensues from intoxication by decomposing substances—sapræmia. Often the symptoms are latent. A man aged sixty, in my ward, was about all the time. He died suddenly of pulmonary hemorrhage the result of gangrenous ulceration of a large vessel; at the autopsy gangrene of the lung was found. The only symptom was the characteristic odor. In the course of inflammatory processes the onset of gangrene is frequently attended by the cessation of pain, the peculiar odor when it communicates with the exterior, and the development of exhaustion and the typhoid state. The character of the discharge points to gangrene. When the lungs are affected, the expectoration is like prune-juice; when the bowels, the discharge is dark and putrid.

Fever is a morbid process with the cause and symptomatology of which the student should be familiar. It will be fully treated in another place. (See Fever.)

Degenerations. The symptomatology varies with the form of degeneration and the organs affected. The prostration of the general economy is due to the same cause as the degenerations themselves.

Albuminous degeneration occurs in fever, and causes the weak heart and defective gland action. The weak heart of the convalescent period in diphtheria and other infective diseases is well known.

Fatty Degeneration and Infiltration. In fatty degeneration there is cell-destruction. The brain, the heart, the kidneys in Bright's disease, the liver, all undergo degeneration. It may be due to phosphorus-poisoning, to snake-bite or other toxic agents. It is seen in acute yellow atrophy of the liver. Fatty infiltration, or lipomatosis, is seen in the "fat" heart of brewers, the enlarged liver, the

excess of fat in the abdomen, etc. The affected organs are enlarged, but they are functionally weak. Fatty infiltration of organs is recognized by its ætiological associations. In alcoholic subjects of sedentary habits, in those patients who eat an excess of fatty foods, in over-fed and pampered children, and in tuberculous patients it is commonly seen. In fatty infiltration the cells are not destroyed. If with the above conditions the liver is enlarged or the heart is weak, or both these conditions are present, we may expect to find fatty infiltration. There is enlargement of the affected organ, which is painless, smooth, not usually soft on palpation. The condition occurs at any age, but usually in later life. Emaciation may not be present. Lithæmia is common in fatty infiltration.

Amyloid Degeneration. This is rarely confined to one organ of the body. The causes are syphilis, malaria, tuberculosis, and prolonged suppuration. The liver and spleen are enlarged, hard, smooth, and painless. There are great pallor, and œdema of the feet and face. There is anæmia, but no fever. The kidneys are affected, hence polyuria and low specific gravity of the urine; a few casts are found. The bowels are likely to be loose because the process has involved the intestine. It occurs at any age. The diagnosis rests on the presence of a cause, the painless enlargement of organs, the pallor, and the polyuria.

Fibroid Degeneration. This is not so much a degeneration as an overgrowth of connective tissue with coincident primary or secondary atrophy of the parenchyma. The function of the organ is impaired or abolished. Increase of connective tissue in the nerve-structures is known as sclerosis, in the liver or kidney as cirrhosis. In the arteries it leads to the changes known as endarteritis. Whatever the pathology may be, whether the atrophy of cell-elements of the affected structure be primary or secondary, the condition is productive of serious, even grave consequences. It is part of the senile process. It leads to the manifold symptoms of endarteritis; it is the cause of many nervous affections which will be discussed in the proper chapters.

The varied phases of so-called interstitial nephritis are due to the fibroid change primarily in the kidneys, and secondarily in the arterial system. In the lungs it attends emphysema, or may even be productive of that condition. The fibroid heart is another manifestation of the same process. The tubes and channels are closed by the same process as in fibrous stricture of the duodenum. Wherever situated its development means gradual abolition of function.

Mucoid Degeneration. This form of degeneration is seen in myxœdema. The albuminous intercellular substance is replaced in the connective tissue by mucin.

Pigmentary, calcareous, and colloid degeneration are local morbid processes without other symptoms than those of the primary affection.

III. Neoplasms or Abnormal Growths.

Tumors, other than cancer or sarcoma, produce only mechanical symptoms, and will be considered in their appropriate places. The mechanical symptoms are due (1) to the tumor (foreign body) and (2) to obstruction of some channel in near relation.

Symptoms. Neoplasms cause local symptoms. This is most striking in structures which must necessarily be destroyed as the growth increases in size, as in the brain or spinal cord, or where tubes or channels are closed, as in cancer of the stomach or œsophagus. Local symptoms may precede the general symptoms; on the other hand, general symptoms may arise for which no local cause can be assigned. The local symptoms of cancer are variable and depend upon the anatomical nature and physiological offices of the organ affected, and upon its anatomical relation to surrounding organs. This class of symptoms will be referred to in the section on Special Diagnosis. Suffice it to say they cause gradual abolition of the function of the organ, or closure of the channels in connection with it, as the intestinal canal, the pharynx, or the hepatic ducts.

Cancer and *sarcoma* are accountable for a group of symptoms to which the term *cachexia* has been applied. In addition, a few symptoms belong to the cancerous process wherever situated. They may or may not all be present; in the large majority of cases one or more are wanting; they should always be sought for in order to confirm a diagnosis of cancer. These symptoms are:

1. **Pain**, recognized by peculiar characteristics in most cases: (*a*) it is sharp and lancinating; (*b*) it is paroxysmal; (*c*) it is increased by irritation, as food when the stomach is affected; (*d*) it is increased by functional activity, as speaking or swallowing in carcinoma of the larynx or pharynx; (*e*) at the outlet of canals, as the bladder or rectum, it gives rise to tenesmus.

2. **Hemorrhage.** If the malignant mass is in communication with the exterior, the blood may be discharged *per vias naturales.* In malignant disease of the upper air-passages or the lungs hemorrhage is likely to occur. It is common in gastric carcinoma as well as in uterine cancer. If the organs do not communicate with the exterior, and the lesion gives rise to exudations or transudations, the latter are frequently bloody, as in carcinoma of the pleura or peritoneum.

3. **Abnormal Discharge.** This occurs especially in cancer of the hollow viscera and of the canal-structures. The discharge is the result of inflammation, suppuration, and necrosis, and particularly microbic inflammation. It is recognized by its more or less *bloody character* and by its *odor,* which is peculiar. It is most offensive and penetrating, and, particularly in uterine cancer, is almost pathognomonic. Even the utmost cleanliness will not obviate it.

4. **Tumor.** It may be readily detected or elude all search. Some swelling is certainly present. It is discovered by external examination, by the objective physical signs of enlargement or change of contour of the affected organ.

5. **Foreign Body.** The growth gives rise to symptoms similar to those present when a foreign body is fixed in any portion of the hollow viscera, as the respiratory tract, the gastro-intestinal, including the hepatic and the genito-urinary tracts. *a.* Through reflex influence an attempt is made to remove it; hence cough, vomiting, diarrhœa with tenesmus, repeated and painful micturition with tenesmus, etc., the particular symptoms varying with the organ affected. *b.* Obstruction of

the channels, with all the accompanying symptoms, depending upon the location of the growth.

6. **Temperature.** A morbid process is often recognized by its negative symptoms, if the term may be used. Thus, fever is absent, or the temperature is even subnormal in carcinoma.

7. **The Cancerous Cachexia.** Wherever situated the disease is sooner or later attended by extreme general symptoms, which are, in a measure, striking. It is to be admitted that cases of carcinoma often occur without marked cachexia. *a.* One symptom may always be looked for; it is *emaciation*. It may be rapid or gradual and extend over one or two years; toward the end it is always rapid. Ultimately, if the patient does not succumb to other conditions, it presents an extreme picture. The eyes are sunken; all normal accumulations of fat disappear; the fat in the rectal fossæ disappears, causing deep depression of the rectum; the abdomen is retracted. The appearances are most striking in cancer of the œsophagus. *b. Pallor* may be present. (See Color.) *c. Anæmia*, with breathlessness, palpitation, vertigo. *d. Exhaustion.* This with accompanying emaciation is progressive and may be the first symptom. Progressive weakness is often seen without fever or local disorder to account for it. Toward the end it becomes so extreme as to forbid exertion. *e. Malnutrition.* Evidences of malnutrition appear; the skin is hard and dry; its elasticity is impaired and it becomes the field for parasitic invasion. Tinea and other parasites may flourish. Bacteria invade the susceptible areas, and boils make their appearance. The secretions are perverted. In the mouth ulcers develop; the fungi of this region (the throat, etc.) become more active; the gums are inflamed. In the later stages the "typhoid state" (see Fever) may ensue. If the gastro-intestinal tract is invaded, symptoms of acute intoxication may arise.

8. **Metastasis.** We are often aided by the occurrence of this event, particularly by involvement of the glands. In gastric carcinoma or secondary hepatic disease enlarged glands above the left clavicle are found; in rectal carcinoma, secondary hepatic cancer. In many instances the presence of cancer is revealed by the metastasis, even when the primary growth cannot be recognized.

Diagnosis. In obscure cases the age, the sex, the associate pathological conditions, the duration of the disease become important factors in the diagnosis. Cancer usually occurs after forty, or, some authorities say, after fifty years of age. The female sex is more frequently affected. It may be associated with a history of previous lesion or irritation, as ulcer in vaginal, gastric, or rectal cancer; the irritation of teeth or a pipe, in labial and lingual cancer; of gallstone, in cancer of the bile-ducts; of renal or visceral calculus, in disease in that situation. A disease of grave and malignant character, the duration of which is over eighteen months or two years, is not, in all probability, cancer.

MORBID PROCESSES IN TUBES OR CHANNELS.

When tubes or channels are the seat of disease, symptoms arise apart from the special morbid process, which are due to obstruction and are common to all tubes or channels. The symptoms of obstruction of the

bloodvessels and lymph-channels—cyanosis, œdema, gangrene (thrombosis and embolism)—have been described. But in addition we have hypertrophy, a secondary condition, not referred to above, which nevertheless follows obstruction of any channel. In the case of vascular obstruction the hypertrophy is seen in the heart and the arteries. (See Diseases of the Heart.)

In obstruction, therefore, of tubes or channels we have to a greater or less extent (1) hypertrophy behind obstruction; (2) diminution of the normal flow of fluid and consequent accumulation of material which normally passes through the channels; (3) atrophy and cessation of functional activity beyond the point of obstruction; (4) dilatation following the primary hypertrophy; (5) degeneration, ulceration, low-grade inflammation (bacterial), secondary rupture of the affected viscera. The morbid anatomist can readily point out the examples of the morbid changes sequential to obstruction. Thus in cancer of the œsophagus there are hypertrophy of the muscular coats, regurgitation of food, atrophy of the stomach, dilatation with accumulation of food, secretions from the glands of the œsophageal mucous membrane, secondary ulceration, rupture into the lungs, with gangrene or pneumonia. In obstruction at the pylorus there are (1) hypertrophy; (2) accumulation; (3) intestinal atrophy; (4) dilatation of the stomach, with its train of symptoms. In obstruction of the biliary channels, or the bladder, or ureters, the same secondary conditions arise *plus* obstruction to the flow of bile or urine. Secondary symptoms arise from accumulation of the non-escaping fluids. Subjective symptoms, it may be said, are not marked; there are pain and difficulty in the performance of the usual functions. It need scarcely be said that the obstruction sometimes gives rise to symptoms directly referable to the abnormal obstructing material which acts as a foreign body. The symptoms are reflex and depend entirely upon the seat of the foreign body.

The *causes of obstruction* in whatsoever channel situated are, first, pressure from disease outside (growths, hernia); second, disease of the walls, with contraction; third, occlusion by a foreign body, as gall-stone, renal calculus, worms or other material, depending on the channel obstructed. The symptoms are most marked when the obstruction is due to disease outside the walls or to obstruction by occlusion within the walls.

In all cases of obstruction—nasal, faucial, laryngeal, bronchial, œsophageal, gastro-intestinal, biliary, renal, or pancreatic—look for the symptoms of the secondary morbid change. Each form of obstruction will be specially considered elsewhere. (See Special Diagnosis.)

Obstruction of Bloodvessels. It must not be forgotten that the bloodvessels are in a measure distinct from other tubes, although subject to the same physiological and pathological laws. They contain fluids, and have a continuous function by which the fluids are propelled. They are subject to the laws that govern the flow of fluids under all circumstances in nature. Any derangement or disease will effect changes which are explainable by hydrostatic or hydrodynamic laws. Fluids within vessels exert pressure. Pressure produced by weight of the fluid is known as the hydrostatic pressure; that produced by the

flow is known as the hydrodynamic pressure. Pressure can be gauged by proper instruments.

Blood-pressure. In the case of bloodvessels the pressure of the contained fluid is called the blood-pressure. The blood-pressure is estimated at the pulse by the educated finger and by the sphygmomanometer. A graphic representation of the character of the pulse is obtained by means of the sphygmograph. A certain definite pressure is always present in health. It is subject to slight fluctuations, but sphygmographic tracings follow a definite course. In the description of the pulse, modifications of blood-pressure will be given in detail; it is sufficient here to say a few words regarding hydrostatic and hydrodynamic pressure.

Hydrostatic pressure is modified by the weight of the fluid. It is of pathological importance in the veins only, and especially in those of the lower limbs. When the pressure is raised, the increased weight of the blood-column causes increased bulk and over-distention, as in varicose veins, unless the support to the blood-column is increased. Inflammations of the lower limbs are attended by venous accumulation and followed by ulceration. For this reason dropsies arise more readily in these portions. The common occurrence of gout in the feet may be due to slow circulation.

Hydrodynamic pressure is variable. Its changes indicate increase or diminution of blood-pressure. The bloodvessels are resisting elastic tubes; the resistance is always equal to the pressure within, hence blood-pressure and *arterial tension* are equivalent terms. We speak of increased or diminished pressure, or correspondingly of high or low tension. Now, the hydrodynamic or blood-pressure depends upon: (1) variations in the volume of blood; (2) variations in the capacity of the vascular system; (3) velocity of the capillary circulation; (4) the force of the heart. The tension of the artery depends upon the same conditions.

1. Variations in the Volume of the Blood. *a*. Volume increased. Causes: absorption of fluid after meals or drinking to excess. Result: increased blood-pressure and increased tension. Controlled in health by action of the vasomotors relaxing the vessels, and by enlargement of the veins. *b*. Volume diminished. Cause: hemorrhage, serous purging. Result: diminished blood-pressure, lowered tension. Controlled in health by contraction of arteries through vasomotor nerves. In hemorrhage the loss of blood produces anæmia. The latter is a stimulant to the vasomotor centre in the medulla, and produces contraction of peripheral arteries and high tension.

2. Variations in the Capacity of the Vessels. *a*. Diminution of the capacity of the blood-channels (volume of blood not lessened). Cause: cutting off of a vascular area by ligation or obstruction, by narrowing the calibre of the wall, as in arterial spasm or endarteritis, by disease of the kidneys, contracting and lessening channels in the aortic circuit, or disease of the aorta, causing obstruction to the outflow of blood. Result: increased pressure, high tension. Controlled by normal regulating vasomotor apparatus, or by diminution of the volume of blood. *b*. Increase of capacity of blood-channels. Cause: relaxation of muscular coats of vessels. Result: diminished

blood-pressure, lowered arterial tension. Controlled by contraction of vessels or increase in amount of blood. In shock, the vasomotor sympathetic system of the splanchnic arteries is so disturbed that the arteries are dilated and all the blood is sent into the abdominal vessels (fall of pressure).

The mode of action of the vasomotor apparatus is as follows: centres in the medulla, in the spinal cord, and locally in the sympathetic ganglia of different parts, control the vasomotor nerves, which influence hydrodynamic pressure. When the centres are stimulated, tonic contraction of the vessels is produced which may be general or local. Increased pressure or heightened tension is the result. It may be reflex from the periphery, or due to some state of the blood. When the centres are paralyzed, or inhibited, or cut off from the arteries, the latter become relaxed (dilated). The pressure is lowered, the tension is less. *Shock*, pain, certain drugs, reflexes (probably) produce inhibition of the vasoconstrictors.

3. Velocity of Capillary Circulation. Obstruction to outflow of blood from capillaries into the veins increases blood-pressure. Cause: the same as when arteries contract. Result: increased blood-pressure, high tension. Regulated in the same manner as arteries. Relaxed capillaries produce opposite conditions.

4. The Force of the Heart. *a*. Heart's action (left ventricle) increased. Cause: hypertrophy, palpitation. Hence, the greater force of blood-impact, greater resistance by arteries. The tonic resistance narrows the calibre of the vessels. Result: increased pressure, higher tension. *b*. Heart's action weakened. Hence, less force of blood, less resistance. Result: lessened pressure, low tension.

High arterial tension is recognized by (*a*) incompressibility and tension of the arteries; (*b*) accentuation of the aortic second sound; (*c*) prolongation of the left ventricle first sound; (*d*) increased flow of urine, pale, and watery; (*e*) the characteristic pulse-tracing. Permanent high tension leads to hypertrophy of the heart and more or less atheroma.

Low arterial tension is recognized by (*a*) soft, compressible, often dicrotic pulse; (*b*) enfeebled sounds, aortic second and left ventricle; (*c*) scanty, high-colored urine; (*d*) the characteristic pulse-tracing. Permanent low tension leads to stases, congestions, and cyanosis, with general weakness and impaired nutrition.

SECTION II.

HISTORICAL DIAGNOSIS.—DATA OBTAINED BY INQUIRY.

CHAPTER III.

THE SOCIAL HISTORY.

Mode of Procedure.

THE subjective symptoms of the disease are elicited first, so that, if necessary, measures may be directed to the patient's relief at once. We have the advantage of observation of the patient's intelligence, expression, and general bearing, and at the same time ascertain the direction further inquiry should take. The patient's embarrassment may pass off and composure ensue before an objective examination is made. It seems preferable, however, to begin the record with the social history of the case, and then record the facts of the family history, the previous history, and the history of the present disease. It is immaterial how they are considered in the following discussion, and for convenience, therefore, the above order will be followed.

The aid to diagnosis obtained from inquiry into the social history cannot be considered exhaustively. Works on hygiene must be consulted. General ideas will be given; reference to the influence of various factors will be found under the individual diseases. That such data are of value is illustrated in various forms of colic. For instance, knowledge that the patient labored in lead will often simplify the diagnosis of the nature of this symptom.

Age.

The age is learned, for each period in the evolution and involution of life has its peculiar physiological processes susceptible to variations by external influences.

A large group of affections arise in the first period of *infancy*, from inheritance or congenital malformations, from accidents incident to parturition, and from improper management of the cord. At a later period, when the feebly resisting organism is adapting itself to its environment, disturbances of digestion from poorly prepared or improper food arise; pulmonary disorders from improper clothing, ventilation,

etc., occur. The developing nervous system has more acute susceptibilities, and hence a long array of reflex symptoms or diseases is observed at this period. Another group of diseases, the exanthemata, are more prevalent in *childhood*, because they arise from exposure to the specific causes, such exposure happening before the child attains many years. The anatomical arrangement of the larynx, disproportionately small, makes the diseases of that organ most frequent in childhood, and a serious factor in mortality. The gastro-intestinal tract, unaccustomed to irritating and infectious agents, is liable to various forms of inflammation at this age. At *puberty* the perversions (from earlier years) are liable to arise, increasing in frequency as adolescence advances. Anæmia and chlorosis are prone to develop at this period. Exophthalmic goitre, hysteria, and epilepsy develop, while the infections to which persons at this age are liable to be exposed, as tuberculosis, may occur. In the *middle period of life* the diseases that arise from occupation, from exposure to external agencies, from habits, are seen. Moreover, processes beginning in adolescence are reaching their acme and find expression in later life, as the cysts of hydatid disease, renal calculi, and manifestations of gout. To these may be added aneurism, angina pectoris, cancer, diabetes, myxœdema, and degenerative nervous diseases. In *later life* degenerations of the vascular and cerebrospinal systems occur; affections due to fibrosis, a resultant of wear and tear, as atheroma; cancer; calculous disease; and, as a final event upon most of these, the terminal infections.

Nasal and Laryngeal Affections. A consideration of the influence of age in diseases of the respective organs shows that among nasal and laryngeal affections the acute inflammations secondary to measles and other exanthemata occur at an early age, while the chronic attacks occur late in life, as do also tumors, except adenoid. Foreign bodies are more likely to be found in children and in the feeble-minded.

Diseases of the Lungs and Pleura. Infancy and old age appear to offer least resistance to bacterial invasion, hence streptococcus and pneumococcus infections are common at these extremes. Tuberculosis, however, is more common in early adult life, although it does not respect age. The degenerations and the morbid growths are more common in later life.

Diseases of the Heart and Bloodvessels. The age at which we are wont to find cardiovascular affections varies with the character of the lesion. Apart from congenital cardiac affections, acute inflammations are more common at the age when infections are more operative, as in the early decades. On the other hand, it goes without saying that degenerative lesions are found in later life. But as man is no older than his arteries, and as these degenerative lesions may occur at a comparatively early age, from a cardiovascular standpoint a man may be senile at thirty-five or earlier.

Gastro-intestinal Disorders. Early age predisposes notably to gastro-intestinal disorders. In later life the catarrhs due to improper exposure, indiscretions in eating, or to occupation are common. The menopause is often associated with gastric disorders of a neurasthenic type. Obstruction from intussusception occurs early in life; from

bands or through apertures, in adult life—usually prior to forty years of age; from volvulus, between forty and sixty years. Obstruction due to a gallstone occurs during the middle or later period of life—always after the fortieth year.

Diseases of the Liver. Diseases of the liver usually occur late in life, because the causes upon which they depend are operative only at that period. In a case, therefore, of ill health in a young subject, when the cause cannot well be determined, the liver is not so likely to be the seat of disease as in older subjects. Late in life we have gallstones with their multiple consequences, inflammation, cirrhosis, and cancer. We may, however, have the congestions and the degenerations in early life, although not so frequently.

Sex.

The prevalence of various diseases in the sexes in undue proportion arises because of difference in the anatomical structure and physiological offices of the two, and because of the difference in exposure to varying causal agencies. Diseases more common in the *male* sex occur on account of occupation, from exposure, from over-activity of mind and body, and, finally, from the formation of bad habits. The diseases that are more prevalent in the *female* sex, apart from affections arising out of menstruation and childbearing, find their origin in the more or less sedentary nature of women's lives, and hence, among other things, the opportunities for introspection. Hysteria, neurasthenia, and nervous disorders abound with them. Males are more subject to epilepsy, angina pectoris, hæmophilia, gout, diabetes, locomotor ataxia, and vesical disease. Females are more subject to exophthalmic goitre, gallstones, tuberculosis, rheumatoid arthritis, chorea, and the above-mentioned nervous disorders. Among *liver diseases* cirrhosis is more common in the male sex; while cancer, both of the liver and of the biliary passages, is more common in women because primary carcinoma elsewhere is more frequent. Among *heart diseases*, females are more prone to acute infectious processes and to the neuroses from immobile nervous systems; males, to degenerative lesions and the intoxication neuroses.

Occupation.

This should be ascertained in the inquiry, for each occupation demands effort in one particular direction or compels exposure to deleterious influences. Writers' cramp, emphysema, eye-strain, laryngeal affections, and a series of disorders thus arise. Knowledge of exposure to particular irritants, coal dust or fine particles of metal or stone, gases, chemicals, effluvia of all kinds, or of close association with animals capable of transmitting disease, is valuable in diagnosis. Those occupations which are pursued indoors in overheated apartments, and which compel the inhalation of noxious vapors, predispose to nasal and laryngeal inflammations. Miners, stonecutters, grinders, and those employed in similar occupations are subject to chronic bronchitis and emphysema, which in turn invite tuberculous infection.

It is noticeable that those occupations which prevent outdoor exercise and regular habits, and which are attended by undue strain of body and mind, as, for example, the work of a locomotive engineer; or overtax special organs, as the eye or ear, predispose to, if they do not actually cause those neurasthenic conditions which have local or general expression, especially in those who are subject to gastric disorders. Lead-poisoning, mercurial poisoning, phosphorus-poisoning, arsenical and brass-poisoning are incident to the occupations which compel contact with these poisons. Infections from animals are to be thought of in tanners, hostlers, and others.

The manner and degree of employment of the mind should be inquired into in functional and organic nervous disorders.

It is not to be forgotten that the occupation at different periods of life must be found out, the age at which life's battle began, and the circumstances that surrounded the early career. The deleterious influence of a former occupation may be observed after the patient is in an entirely different sphere of labor.

Habits.

Habits as to clothing (catarrhal affections and rheumatism), as to hours of rest and sleep (neurasthenia), as to character of food, time, regularity, and manner of eating (the indigestions, gout), as to exercise, and as to the use of alcoholic stimulants (cirrhosis of the liver, neuritis, brain affections), to tobacco (amblyopia, cardiac palpitation), of tea or coffee, of narcotics, must be inquired into. Methods of work, methods of recreation, domestic joys or sorrows, must be ascertained. A knowledge of the habits, of the life (of the inner life, indeed) of the individual, is essential to a rational diagnosis, and hence a true therapeusis.

In gastric affections it is necessary to inquire closely into the habits of eating, the amount and character of the food, and the regularity of the meals. In liver diseases, alcoholism suggests cirrhosis; large amounts of food, hyperæmia; sedentary habits and an excess of starches and fats, gallstones.

Place of Residence and Dwelling.

A knowledge of the place of residence is of service. Town residence and country residence, a residence in a damp locality, by the sea and in the mountains, in particular valleys, in different water-sheds, in tropical or frigid clime, each makes an impress on the constitution, even if actual disease is not created. Malarial regions, goitre districts, localities in which individuals have to an unusual degree vesical calculi, or in which special epidemic diseases abound, as yellow fever, cholera, or dysentery, are matters for inquiry. Knowledge of the places of residence at different periods of life and the duration of such is often important information.

The situation of the dwelling and its degree of comfort must be learned. The sanitary arrangements—drainage, ventilation, water-supply, heating—are to be scrutinized.

Family Relations.

Marriage and the number of children, with their degree of health, must be recorded; when the patient is a woman, the number of children born, the character of the labors, the number of miscarriages.

Is there trouble in the marital relation? Has there been sorrow, or sudden shock, or long nursing, or great care? Are the financial circumstances easy? Has there been recent malfeasance? How many invalid women arise out of such ashes!

Questions so personal can only be put after long acquaintance, or the information may be obtained through judicious inquiry of friends.

Frequently more delicate questions must be put, as to masturbation or excessive venery, but with great caution and only when conditions make such an inquiry imperative. In epileptiform convulsions, profound hysteria, neurasthenia, prompt, clear, manly questions as to these habits are to be put, not reference made to them in prudish or mawkish suggestion.

Exposure to Infection.

If the suspected ailment partakes of the nature of a contagious disease, the probability of exposure to the disease must be looked into and the presence of epidemics ascertained. The period of incubation must be known in such cases. The prodromal symptoms must also be known. We inquire for all those circumstances which contribute to the origin of the infection. Hence, we inquire into the nature of the food and water. We inquire whether an opportunity for inhalation of infectious material, as dried tuberculous sputum, or direct exposure to an affected patient could have occurred. We learn the hygienic conditions and place of residence (malarial districts, the tropics). The occupation—wool-sorter, hostler, farrier—may point to the nature of the infection. In short, we inquire whether the patient has been exposed to any infection.

CHAPTER IV.

THE FAMILY HISTORY.

This inquiry is instituted in order to determine the affections which may or may not be hereditary. We learn also the average duration of life in the family and the relation of the mortality to the physiological epochs in life. Data of the latter character are of value in estimating the possible duration of life for purposes of life insurance, and they also throw light on abnormal conditions; thus to learn that most of the members of a family have died of apoplexy, some form of aneurism, or arterial degeneration at a comparatively early age, is to learn that vascular changes developed earlier than usual. The family physician, who comes in contact with one or more generations, profits most by the knowledge of the family history. He learns the predisposition to various minor ailments—to headaches and attacks of indigestion, "bilious attacks," for instance; he learns of a family type which leads a parent to say that his child "has a tendency to croup," a popular expression which has in it an element of truth. That condition or state which predisposes to "colds" belongs also to family groups. He learns the power of resistance to disease in the family, or their capability to undertake large duties in life; he learns their susceptibility to drugs and their tendency to take stimulants. Nerve-force is the capital with which the battle of life is kept up; if it is at a minimum in groups of families, diseases or conditions of poor health due to its use—a use not excessive in others—arise.

To secure accurate data, the age and state of health of parents, brothers, and sisters, if living, are ascertained; or, if dead, the cause of death, and age at which it took place. Similar questions may be applied to several generations of the family and to collateral branches.

Difficulties. A correct family history may not be secured because of family pride, because the patient may not understand the medical terms, or because the inquirer forgets the interdependence of many diseases.

Caution must be employed in order not to arouse family pride if evidence of "scrofula" is sought for, or to provoke undue alarm when inquiry into the family history of cancer is made. Disarm suspicion by inquiring for the symptoms of the disease in various organs in which it may occur, as jaundice, uterine hemorrhage, etc., or ask about growths or tumors. Do not use the specific terms consumption and cancer.

Obscure Terms. Moreover, care must be exercised to secure definite data, and not to over-estimate statements. Thus, when the cause of death is stated to be "dropsy," or "jaundice," or "cold," or "teething," or "change of life," the term is meaningless. Control questions must be put by inquiry into the character of the symptoms that attended the fatal illness, and by giving the affections the various popular names that are given them in different countries.

Inherited Diseases.

Only a small number of diseases are strictly hereditary. Of these, nervous diseases are the most common, as progressive muscular atrophy, hereditary chorea, Thomsen's disease, Friedreich's ataxia, migraine, asthma, epilepsy, and forms of insanity. To these may be added family types occurring generation after generation or as racial characteristics, manifested in hysteria, hypochondriasis, and other psychoses. The physical stigmata of degenerates are associated with these nerve affections. The writer has seen chronic Bright's disease, or a state of the constitution that predisposes to it, occur in several generations without the usual exciting causes of that affection. Syphilis may be inherited. No investigation is complete in certain eye, skin, nasal, or throat affections, in diseases of the nervous system, and to a less degree in other ailments, without settling the question of ancestral syphilis, if it can be done in a judicious manner. Hæmophilia is the most striking affection that is transmitted by inheritance. Diabetes is an affection that may well be spoken of as inherited. Generally, it is not the diseases themselves that are hereditary, but types of tissue that predispose to disease, such as tuberculosis or cancer; or conditions of the organism that favor imperfect metabolism, as is seen in gout and rheumatism. In consequence of this tendency in successive generations, we inquire for a history of gout or rheumatism or those affections in which these processes are most likely to occur: hence in affections of the nose, pharynx, and larynx, in diseases of the heart and vessels, and in certain articular and skin affections.

A tendency to the diseases or perhaps the metabolic causes productive of them is inherited in emphysema and arteriosclerosis. The observing physician will often find not that the patient is a victim of an inherited disease, but that the generation to which he belongs is the last perhaps of a long line. The family is dying out. The patient presents the stigmata of degeneracy or of hysteria, or he is neurasthenic, susceptible to infections, easily affected by, and perhaps the victim of the alcohol, tobacco, or drug-habit. He does not bear up in adversity, and lacks the mental poise to withstand the temptations of prosperity.

Atavism. The fact that diseases skip a generation (atavism) must be remembered. A generation may be small or decimated by accidental disease, and hence the force of the family history be weakened. At times in a family sufficient time has not elapsed for predisposition to arise, as when we inquire into the illness of a child whose parents are in early adult life. Finally, all negative answers must be recorded. Such knowledge has a controlling value in estimating the significance of the family history.

Nervous Diseases. The family history is perhaps of more importance in connection with nervous diseases than in connection with those of any other system. By *neurotic heredity* we mean that in certain families a tendency to the development of various forms of nervous disease exists, which may be manifested, however, only in certain members of a generation. Certain forms of nervous disease, the causes of which are unknown, are spoken of as hereditary or familiar, because two or more examples have been observed in the same family. Various

terms are employed to indicate the nature of the inheritance. *Direct inheritance* means that the child acquires the disease at birth. If both parents have the disease, the child is likely to suffer more severely, and this is spoken of as *cumulative inheritance.* By *indirect inheritance* is meant the condition in which the collateral ancestry and not the parents have had the disease. Both parents may appear to be healthy, although the grandparents, or earlier ancestors in the direct line, have suffered from the same disease, and this is called *atavistic inheritance.* By *similar inheritance* is meant the occurrence in the offspring of a disease similar to but not identical with that from which the parents have suffered. Examples of such diseases are Huntington's chorea and Goldflam's periodic paralysis. By *dissimilar inheritance* is meant the development in the offspring of a form of nervous disease differing from that which existed in the parents, as an epileptic child born of parents suffering from neurasthenia, hysteria, or insanity. The indications of neurotic heredity are manifold. Inquiries must be made in regard to insanity and epilepsy, to instances of suicide, to peculiarities of character, to criminal tendencies, to addiction to the use of drugs, such as alcohol or opium; to congenital deformities; or to congenital diseases, such as deaf-mutism. Charcot has called attention to the fact that certain of the so-called rheumatic manifestations may occur in the antecedents of a patient suffering from nervous disease.

Contagious Diseases.

In the inquiry it may be well to ascertain the probability of disease being transmitted from husband to wife, or the opposite. Syphilis, gonorrhœa, and tuberculosis are examples. Not only may this probability apply to the transmission of disease from husband to wife, but to their offspring as well. Then, too, we must inquire of mothers for manifestations of syphilis in the children.

Caution must be exercised in the pursuit of knowledge of this kind, as strained, or even ruptured, marital relations may result from injudicious intimations.

Common Morbid Processes.

The data of the family history are of no avail unless it is remembered that many fundamentally identical affections have various modes of expression. Various diseases may be allied to the one suspected to exist in the patient, and be overlooked because of this difference of expression. One member of a family may die of heart disease, another of rheumatism, or some may have had chorea, or cutaneous affections, or renal calculi; such ailments are expressions of the same morbid process. Finlayson well puts them into groups and fittingly portrays them as follows:

"In regard to scrofulous [tuberculous] diseases, we ask for swollen glands or 'waxy kernels,' or running in the neck; diseases of the spine and other bones, bad joints, white swellings, or 'incomes,' as they are termed in Scotland; disease of the glands, of the bowels, water in the head, consumption of the lungs, or decline, or weakness of the chest, with spitting of blood, and so on.

"Heart disease, rheumatism, chorea, psoriasis, and some other cutaneous affections, and perhaps renal concretions and emphysematous bronchitis, appear to replace each other in different members of the same family.

"The neurotic group includes the various forms of neuralgia, epilepsy, hypochondriasis, hysteria, and insanity; apoplexy and hemiplegia may (perhaps doubtfully) be included in this group; their hereditary character seems rather to be associated with vascular disorders. Gout, disease of the liver, contracted kidney, renal calculus and gravel, and angina pectoris form another allied group; and these have also some affinity with the disorders connected with arterial degenerations. Syphilis, which, of course, has marked hereditary characters, assumes such a multitude of forms as to preclude enumeration; but the tendency is for such syphilitic diseases to fail in the course of time from early death or sterility. Abortions, stillbirths, early deaths in infancy, associated with cutaneous eruptions on the buttocks and with snuffles, are important in many family histories; nervous deafness, opacities of the cornea, notched teeth, epilepsy, and imbecility are occasional manifestations of the same disorder in those children who survive."

Conclusions.—It is thus seen that in securing the family history data are acquired which may be (1) complete and of value in estimating family tendencies, or (2) vague and of doubtful value. The latter may be due to lack of memory on the patient's part or to his ignorance of technical terms. The difficulties must be overcome by control questions prompted by our knowledge of the nature of the disease and its frequency at different ages, by an inquiry for symptoms, and by investigation into collateral and remote branches of the family.

CHAPTER V.

THE PREVIOUS MEDICAL HISTORY.

THE medical history is secured to determine (1) if the present disease has occurred previously, (2) if it is the sequel of a former disease, or (3) if a similar affection has occurred, and may therefore be excluded from the diagnosis.

1. The following diseases are likely to recur: asthma, delirium tremens, erysipelas, gout, gallstones, malarial fever, pneumonia, tonsillitis, rheumatism, and renal colic.

2. The diseases which have sequelæ are syphilis, gonorrhœa, scarlet fever, rheumatism, and other general infectious diseases. Gallstones, renal calculi, local infections, as of the appendix or gall-bladder, are often followed by secondary disorders.

3. The diseases which are likely to occur only once are most of the exanthemata. In the diagnosis of obscure cases if a history of their occurrence is ascertained, they can usually be excluded.

The date of occurrence and character of the disease, the duration, the degree of severity, and the completeness of convalescence must be determined.

Diseases of the Nervous System. In the investigation of diseases of the nervous system it is important to inquire in minute detail into the previous medical history.

The infectious diseases sometimes are followed by peripheral neuritis or lesions in the central nervous system, or they may produce an early tendency to arteriosclerosis. It is of importance to know whether the fœtal existence of the patient was normal, and, if possible, to obtain data concerning the condition of the mother during this period. Inquiry should be made regarding the nature of the birth; the existence of infantile spasms, at what age they occurred, when they ceased, if at all, and if there was any suspected reason for their development. It should be noted when the child first walked, when it was first able to talk, the rapidity of its intellectual development and progress at school, whether the character was normal, if there were night-terrors, nocturnal enuresis, symptoms of nasal obstruction, or indications of visual defects.

In the case of boys the physician should endeavor to discover if there is any history of severe injury, particularly to the head; whether the boy had the opportunity for free exercise or was restricted in this respect; if his habits were good; if he smoked early in life; if he was overworked at school or obliged to work hard during early adolescence; if he masturbated. In the case of females the physician should inquire at what period puberty occurred, and whether there has been any difficulty with menstruation; the occurrence of childbirth, or miscarriages, or of gynecological disorders or operations.

The existence of neurasthenia or gastro-intestinal disorder at some early period should be determined.

The history of luetic infection is often difficult to elicit. Occasionally it will be admitted, but more frequently it is necessary to discover the fact by indirect questioning.

Diseases of the Nose and Larynx. Diseases of the nose and larynx are more readily explained if we have knowledge of the previous occurrence of syphilis, the exanthemata of early life, also gout or rheumatism. It is also important to inquire for rhachitis or previous nervous diseases.

Diseases of the Lungs and Pleura. Pneumonia is likely to be followed by subsequent attacks. Pleurisy may be an expression of rheumatism, and it may be preceded by other rheumatic phenomena. On the other hand, it may be the earliest expression of tuberculosis, and may precede the latter by two or more years, being separated from it by an interval of health. In affections of the pleura, we must inquire for previous infections and note the occurrence or not of disease of contiguous structures, as the ribs, the muscles of the chest, and the viscera beneath the diaphragm. Then it must be borne in mind that pulmonary tuberculosis may succeed a long antecedent articular or glandular tuberculosis, the history of which should be inquired for. The previous state of the circulation should be studied, and the occurrence of heart disease sought for.

Diseases of the Heart. The occurrence of any one of the numerous infections may have been the initial step in the production of the affections we are considering. The determination of the nature of a cardiac lesion may hinge upon the correct decision of this question. The infection of acute rheumatism is sought for. A history of chorea, of various skin affections related to gout and rheumatism, of eye affections, of tonsillitis, of other related affections, must be sought for. If found, such history is more than suggestive.

Diseases of the Stomach. Infectious diseases predispose to gastric disorders, either because of the attendant gastritis or of the resulting defective innervation. The excessive feeding during convalescence from typhoid fever, it seems to the writer, is frequently the cause of gastric dilatation; and dilatation which may become permanent often occurs in the course of severe infectious diseases. In a number of recorded instances the dilatation was so acute and severe as to be rapidly fatal. Any prolonged illness that weakens the muscular system and lowers the nervous tone is likely to cause gastric disease.

It has already been stated that gastric affections may be secondary to many *local diseases*, as of the heart, the lungs, and the kidneys. Inquiry as well as objective investigation must be made to determine the presence of possible primary diseases; disorders that interfere with the mechanical support of the intra-abdominal organs must be inquired for; *pregnancy*, antecedent *ascites*, or a large *tumor* may so weaken the abdominal muscles as to lead to gastro-enteroptosis; finally, a history of the ingestion of *corrosive poisons* must be sought for in cases of gastritis.

It is very important to learn whether the patient has been subjected to the various causes of *neurasthenia*, which, with the history of the

occurrence of neuropathic symptoms, are valuable data pointing to the source of many gastric neuroses.

Diseases of the Intestine. In *obstruction* by bands of adhesion there is a history of *peritonitis*, or, as Treves points out, previous attacks of *obstruction* more or less marked. In *volvulus* the patient has been subject to constipation prior to the attack, but in *intussusception* there is no previous history of intestinal trouble, unless it be a polyp, causing dragging, colicky pains, and occasional discharge of blood.

Diseases of the Liver. It is absolutely essential to inquire into the patient's previous history to establish a diagnosis, as liver disease is usually secondary. The occurrence of heart disease or obstructive lung disease points to a congestion; infectious diseases to cirrhosis, if the latter is not otherwise accounted for; dysentery to abscess; ulceration or suppuration in the portal area to multiple abscess; syphilis to syphilitic disease of the liver; tuberculosis, suppurations, bone disease, and syphilis, to amyloid disease; pyæmia to multiple abscess; tuberculosis to fatty liver.

Diseases of the Kidney. A previous history of an infection, of some constitutional disorder, as gout or rheumatism, or of renal calculus. Antecedent heart or lung disease must be inquired for.

Infectious Diseases. As previously indicated, the object of determining whether an infection has existed previously is to exclude those which are not likely to occur a second time; to consider seriously those in which one attack predisposes to subsequent attacks; and, finally, to explain phenomena which we know to be sequelæ of infections. In this manner the Bright's disease or ear disease following scarlatina may be recognized; the various affections of the nose, throat, and larynx, of the lungs and gastro-intestinal tract, which have been spoken of above, are similarly more easily diagnosticated.

It is most important to appreciate the sequelæ of syphilis and of gonorrhœa as described in the chapters treating of these diseases in Special Diagnosis. Undoubtedly obscure toxic phenomena, especially forms of headache, of neuritis, and of rheumatism are due to the occurrence of these diseases at some remote period.

Previous Injury. In the medical history valuable information is secured if we know of previous injury, or of the performance of a surgical operation for a given disorder. This is particularly true in the case of abdominal diseases. Knowledge that a patient has been operated on for appendicitis might aid in the explanation of symptoms which point to intestinal obstruction. In a similar manner a clue to other abdominal affections may be found.

CHAPTER VI.

THE HISTORY OF THE PRESENT DISEASE.

Scope of Inquiry.

THE history of the present disease includes an account of the *subjective symptoms*, of the *duration* of the disease, of its mode of *onset*, and of the *evolution* of the symptoms to the date of examination. The patient also gives an account of such objective symptoms as could be noted by him, as swelling of the legs, the date of its commencement, mode of onset, and progress. Practically, it is better to learn the symptoms on account of which the patient applied for treatment, and, with them as a guide, to inquire into the date of origin and mode of development of the disease. We thus ascertain the *clinical course* of the disease. Many diseases are diagnosticated only by their clinical course. It is obvious, therefore, that the greatest care should be employed to secure accurate facts. Thus, an infectious disease which has a definite period of incubation, a definite symptom-group preceding the eruption, a fixed day for the appearance of the eruption and for each of its stages, and a symptom-group for this period, can often be recognized only in this way, as the specific eruption may simulate two or more infections. This statement may be applied to many other diseases, and, as a matter of fact, if the clinical course is not carefully mapped out, the diagnosis may be impossible, or at least the clinical picture may remain obscure.

Method of Inquiry.

The history and subjective symptoms are learned best in the language of the patient. If the memory fails or the symptoms are not clearly narrated, judicious questions may be put to complete the story; but leading questions must never be asked, or only after the patient's own account has been fully given.

Often the patient will be too voluble and introduce irrelevant matter, or too taciturn from modesty or a desire to conceal facts, as when illegitimately pregnant. While much time is lost in listening to a prolix account of sufferings, the student will do well at first to bear with the patient, for it gives him the opportunity to study character, observe the patient's mental and emotional characteristics, and expression of the countenance. To suppress the loquacious, free the tongue of the silent, gather scintillations of intelligence out of the dense clouds of ignorance, requires knowledge of human nature of a high degree, acquired only by long practice. Allied difficulties have been discussed in the paragraphs devoted to the family history. Indeed, the wonderful faculty of seeking information in this manner has been the capital of many physicians not otherwise well prepared. It is by this means

and by tricks that the charlatan plies his vocation. A favorite method of the quack, after a few words from the patient, is to tell him how he —the patient—feels. He has some knowledge of the march of the disease, and portrays its full development to the surprised and credulous victim. Elsewhere (see Subjective Diagnosis) the reliability of such data is discussed, and the student must not for one moment consider the data obtained by inquiry as of equal value with those obtained by observation.

It is particularly important to secure a *chronological order* of events in the disease. The diagnosis is much easier if such sequence is established. Of course, there are times when only the minimum amount of information of this character can be secured. The patient may be unconscious, or in a convulsion, or unable to speak from dyspnœa. It then becomes necessary to rely on the testimony of friends or to gather the information from the patient's surroundings.

Mode of Onset and Duration of the Disease.

It is well to learn if the onset of the disease was *sudden* or *gradual*. If the former, the most striking phenomena are to be ascertained; a chill, convulsion, sudden pain, sudden vomiting, a profuse diarrhœa—each points to lines of further inquiry. If the latter, did it follow upon an acute illness, or did each symptom gradually increase in intensity, and as each week or each month passed by, new phenomena creep into the symptom-complex? We thus learn if the affection under consideration is acute or chronic—its *duration*. It must not be forgotten that certain affections may be two or three days—or, on the other hand, as many weeks—in developing, which are nevertheless acute; typhoid fever is a good example. It is to be remembered, also, that diseases may have sudden acute expressions, and that a chronic disease may be in existence a long time without the patient's knowledge. An acute colliquative diarrhœa or a convulsion is often the first intimation of a chronic nephritis, and an attack of angina pectoris the first symptom of organic heart disease of long standing. To appreciate the relationship of acute to chronic disease, or of acute phenomena to chronic morbid processes, requires a full knowledge of the processes of disease.

Evolution of the Disease.

In making inquiry concerning the evolution of the subjective symptoms, the *frequency*, *duration*, *character*, and *degree of severity* of each symptom, and its relationship to the function of the organ apparently affected, must be inquired into. Thus in the case of pain in the abdomen, we must learn its character, frequency, duration, intensity, and location, and whether it is associated with functional disturbance of any of the viscera in which the pain presumably has its origin. Or, if there is frequency of micturition, the length of time the symptom has been present; the degree of frequency: the time in the twenty-four hours when the micturition is most frequent; its relation to food, exercise, or emotions; the character of the act of

micturition, and its association with other evidences of functional disorder or organic disease of the genito-urinary tract.

The steps thus far taken in the diagnosis are four in number. While a great deal that is not essential may be gathered, the very gleaning of the facts enables the student to acquire objective information of the highest value by observing the patient's speech, gesture, expression, and general bearing. Moreover, the facts ascertained are of value in determining a scientific and rational line of treatment, for in addition to the diagnosis the causal factors of the disease are often found.

SECTION III.

SUBJECTIVE DIAGNOSIS.—DATA OBTAINED BY INQUIRY.

CHAPTER VII.

THE PRESENT CONDITION.—GENERAL SUBJECTIVE SYMPTOMS.

We now come to the second step in the investigation—the determination of the present condition, the *status præsens*. To determine the present condition *inquiry* and *observation* are necessary. In this section will be discussed only the data obtained by inquiry. It includes the subjective symptoms that are common to special organs or systems or to morbid processes.

The *subjective symptoms* are expressive of the sensations of the patient, and vary in accordance with the sensibilities of the individual affected. Thus acute pain may apparently represent a severe process in one, while in another the same severity of process may be represented by the minimum amount of pain. (See Chapter VIII., Part I.) The subjective symptoms are "conditions of the patient's consciousness." (Pye-Smith.) They can be simulated, and are therefore sometimes fallacious.

Feigned Disease.

It is to be remembered that it is our province not only to ascertain the cause of suffering in the sick, but also to detect the flaws in the testimony of him who would feign sickness. The malingerer utilizes subjective symptoms to hide his deception because they can not be seen, felt, weighed, measured, or ascertained by hearing.

To detect feigned sickness demands much acumen on the part of the physician. He must not only be able to make an accurate and exhaustive objective examination of the patient, but also be alert to appreciate surroundings and conditions. Feigning may be suspected if there is a *motive*, as in the case of prisoners, pension applicants, students at school or college, hospital "beats," and persons who hold health policies.

If sickness recurs frequently without definite cause, while the subjective symptoms are mild and quickly relieved and the objective symptoms negative, the use of instruments of precision will detect the malingerer. With their aid we can usually find out if the subjective and objective phenomena tally. The thermometer frequently exposes the

deception, as fever can rarely be simulated, although tricks with the thermometer may be practised. A favorite method is to rub it, and thus cause the mercury to rise. Frequently the suspected person must be placed under close surveillance, unknown to him, and dodges of all sorts, suggested by the surroundings and circumstances, employed to make him unwittingly testify to his deception.

The student will learn later that there is a *mimicry* of disease, and that in certain nervous affections the simulation of subjective symptoms plays the chief *rôle*. In hysteria subjective and objective symptoms are masked. Long experience and acumen are required by the physician to see through the deceptions. The age of the patient, the sex, the state of the emotions, the varying expressions of the symptoms (under varying circumstances) —with attention fixed or removed—the mobility of the symptoms under excitement or emotional disturbance, the lack of harmony between functional disorder and organic change, are the elements to be considered in order to fathom the mysteries. Often anæsthesia must be induced in order to dissipate simulated tumors, or to relax rigid joints and contracted limbs. Magnetism, electricity, and other tests are likewise employed. In the chapter on Hysteria its manifold expressions will be adverted to, and it will be seen that functional disorder of almost every organ or special sense is simulated in this affection. Organic processes even are imitated, as joint-inflammations, peritonitis, and other grave conditions.

Value of Subjective Symptoms.

Notwithstanding the fallacy of subjective symptoms in that they may be feigned or mimicked, they are valuable evidences in the hands of the scientific inquirer. If the patient is a good witness, their value is much enhanced. He must be intelligent and truthful. His testimony is of value if he can array in logical order the sequence of symptomatic events which culminated in the condition for which he seeks relief. If he can clearly narrate the events in his past life, or in the lives of his ancestors, that appertain to physiological aberrations, his story is an aid to the searcher for truth.

Individuals vary not only as to pain sense, but also as to other subjective symptoms. The *morale* is shattered in some more readily than in others —thus, for instance, oppression of the præcordia may strike terror to some, while to others it would be simply a sense of discomfort. Moreover, subjective symptoms are constantly before the patient while in distress, if only in the mind's eye. Because of this perturbed state they grow in magnitude rather than diminish. We must study them from many points of view. The mode of onset, frequency, degree, and character of symptoms must be inquired into. The competency of the witness under the circumstances, from lack of accurate noting of symptoms, failure of memory, varying degree of susceptibility to impressions, etc., may well be doubted.

If the physician is possessed of a scientific turn of mind, considering evidence without allowing previous conceptions to influence him, capable of discerning the truth and discarding the false, of analyzing and weighing

statements, and of appreciating their relationship to what is known of morbid processes, the patient's statements of subjective symptoms are of value in the discernment of disease.

But not only does the varying "personal equation" of the patient render subjective symptoms fallacious, the same factor in the physician contributes also to the fallacy. The latter may have unfortunately formed, by hearsay regarding the patient, a preconceived notion of the nature of the disease; or from personal bias in favor of particular diseases, on account of narrow lines of study or lack of breadth of view of pathological processes, he may set out to prove a theory rather than to establish a fact. In either case, by leading questions, by placing emphasis on certain parts of the testimony, the subjective symptoms can be juggled with and made to tell any but the truthful story. Adroitness, combined with tact and good judgment, is more essential to secure a true account of the patient's sufferings even than to obtain a correct history of his disease.

General Subjective Symptoms.

The general subjective symptoms—that is, the abnormal and disagreeable sensations which extend more or less over the whole body, or are referable to more than one organ or apparatus—are few in number and are not diagnostic of any particular affection. They are at times the only symptoms complained of by the patient. They include abnormal sensations of strength or weakness, general numbness or tingling, and general paræsthesias of all kinds; general vasomotor disturbance, causing sensations of heat—flushing, or sensations of cold—from mild chilliness or "creeps" to the pronounced chill or rigor—sudden perspirations, general throbbings or pulsations, described by S. S. Cohen as phenomena of *vasomotor ataxia;* and general discomfort, to which the term *nervousness* is applied. Irritability, disorders of sleep, and the more distinct nervous manifestations above mentioned, will be referred to in the chapter on Nervous Diseases, and fully discussed under Hysteria and Neurasthenia.

A feeling of *strength*, or the idea of an ability to perform great feats of strength or endurance, or a great mental feat, is a subjective symptom that is dwelt upon by the patient who is developing or passing through certain stages of paretic dementia. It is accompanied by other evidences of exhilaration. *Exhilaration* attends chlorosis and forms of hysteria and neurasthenia, the physical or mental exhibition of strength taking place in the after part of the day and evening or upon undue excitement. Corresponding depression usually follows.

A sense of *weakness, exhaustion*, or *fatigue*, is often complained of. If an absolute demand is made upon the bodily strength, it can respond, but otherwise it is not exerted. The patient complains of being more tired in the morning than upon retiring, or of a sense of inability to perform accustomed or special duties. Mental depression usually attends the phenomenon. It is due to neurasthenia generally, but is a frequent accompaniment of the forms of toxæmia to which malaria, gout, and rheumatism belong; of the toxæmia of certain varieties of indigestion, of tobacco, alcohol, and other narcotic poisons (tea or coffee), and of

mineral poisons. The same sense of fatigue attends the prodromal stage of the specific fevers. It is a symptom that has been observed frequently of late in the sequential period of influenza.

The sensation of weakness must not be confounded with true weakness or *muscular prostration*, seen in diabetes, tuberculosis, exophthalmic goitre, and anæmia. While the patient is aware of its presence, it is well to consider it under the objective phenomena of disease, for it is a readily recognized sign of disease.

Numbness or *tingling*, and *burning* (paræsthesias), may be general or local. Numbness, when local, may precede the eruption of herpes zoster, may be the *aura* of an epileptic attack, the premonitory symptom of neuralgia, or a symptom of neuritis. It will be discussed in the chapter on Nervous Diseases. It must be remembered that general paræsthesia, while a disorder of sensation, is due to morbid conditions outside the pale of the nervous system. It may be of reflex origin, from irritation at a distant point; or it may be toxic, as in carbolic acid or aconite poisoning. The sensation is complained of in neurasthenia and hysteria, and is a common accompaniment of locomotor ataxia, multiple neuritis, and chronic spinal meningitis. Other subjective vasomotor disturbances that are of frequent occurrence are likewise manifestations of nerve disorder from reflex or toxic causes. Flushing, and a constant sensation of heat, with or without perspiration, which attend the perturbation of the menopause, are common in uterine disorders and in chronic gastritis.

Faintness, or a sensation of weakness with a syncopal tendency, occurs in anæmia, myocardial disease, pulmonary thromboses, and pneumothorax, from increased abdominal pressure. This symptom is present when the action of the diaphragm is interfered with. Emotion, fatigue, pain, and shock cause it. It occurs in neurasthenia, and frequently accompanies physiological acts like defecation.

Formication, or itching, is a variety of pruritus attending many skin affections, hysteria and neurasthenia, many organic spinal diseases, toxic conditions, as from lead, uræmia, gout, and jaundice. Some drugs give rise to it. It is a local symptom in diabetes and leucorrhœa affecting the genitals, and of seat-worms or hemorrhoids affecting the anus.

Throbbing may be felt over the entire body in aortic regurgitation, hysteria, neurasthenia, and anæmia. Pulsation of the aorta may be functional or due to aneurism. Throbbing of the head and neck is seen in exophthalmic goitre. It is a sensation experienced in aneurism, in palpitation, and in cardiac hypertrophy limited to the aorta or the heart.

Other abnormal local sensations are:

1. A sense of *fulness* located in the chest or epigastrium, due to nervous dyspepsia or dilated stomach; hypertrophy of the heart, emphysema, and asthma; or to distention of the abdomen, as from tympanites.

2. *Oppression* or *weight* in the chest in cardiac and pulmonary affections and in hysteria; in the epigastrium, in gastric neurasthenia; in the head, in neurasthenia and hypochondriasis, or as an *aura;* in the abdomen or pelvis, when a tumor is present.

3. *Girdle sensation* in spinal cord disease, or temporarily after violent cough or vomiting, and in diaphragmatic pleurisy.

4. *Præcordial distress* in myocarditis and angina, in heart weakness and dilatation, in acute forms of indigestion and of diarrhœa, and in hysteria and neurasthenia.

5. *Chill and fever.* Both are subjective as well as objective phenomena, but as one can be accurately estimated by an instrument of precision (thermometer), and as both are generally associated, the discussion of them will be postponed. (See Objective Signs.)

The abnormal sensation of cold or of heat will be discussed in the chapter on Nervous Diseases.

6. *Coldness* may be due to myxœdema, as well as to functional nervous disorders. When localized, with pain or pricking sensations, it may be due to one of the forms of mild neuritis, and may occur in locomotor ataxia or syringomyelia. (See Sensation in Nervous Diseases.)

The student will learn that the curious manifestations to which reference has been made are all evidences of ill health, of a depressed vitality, of a condition in which there are malnutrition, poverty of nerve-force, and lack of blood-richness (anæmia). There may be peripheral irritation or a toxæmia, but the under-current of ill health is the fundamental derangement.

CHAPTER VIII.

PAIN.[1]

Definition. Pain is a general term used in medicine to describe a number of subjective symptoms connected with morbid processes. It may be defined as a sensation which produces on the part of the organism, as a whole, the desire to abolish or escape from it. It is the expression in consciousness of injury to the peripheral or central nervous system by irritation or lesion; at times the central end of the peripheral nerves may be the seat of irritation, causing so-called referred pains. This definition, however, fails to include the hyperæsthesias, the hyperalgesias, and all simulated pains. But the latter are to be included in this chapter for the sake of clinical convenience, while the two former are of significance only as conducing to the production of pain.

Pathology. The pathology of pain is generally believed to be a state of impaired nutrition, and hence of injury, gross or microscopical, either at the periphery or in the afferent nerve-tract. The cause may be purely functional, as, for example, when pain is due to over-stimulation of the tract by its normal stimulus and its consequent exhaustion; or to strictly local conditions, as pressure, injury, or inflammation; or to systemic conditions acting locally, as the neuralgias of anæmia. There is also the so-called sympathetic or reflex pain, due to irritation in a part removed from the locality to which the sensation is referred.

Pains in reference to the general nervous system may be classified according to the localization of the lesion into (1) peripheral, (2) central, and (3) general. *Peripheral* pains are local and due to some alteration either in the structure or in the nutrition of the peripheral nerves. The disturbance may be situated at the sensory terminations, or anywhere in the course of the nerve or nerve-roots. Pains due to causes situated in the latter place are usually perceived at the peripheral distribution of the nerve, and are, therefore, spoken of as *referred pains*. The nature of *central* pain is not at present clearly understood. Certain cases have been reported in which pain had been felt in one part of the body, usually an extremity, and at post-mortem no lesion whatever could be found in any portion of the afferent nerves coming from the painful region. Lesions, however, have been found in these cases in the brain itself, and it is supposed that these are responsible for the painful impression. *General* pains are those due to some toxic condition of the blood or impair-

[1] Pain is treated in a suggestive manner, and so much space is given to it because it is too frequently improperly managed. Its cause is never thoroughly investigated. Anodynes are given for its relief, thus too frequently creating victims of the morphine-, chloral-, or other habit. The following articles are suggestive: Head: "On Disturbances of Sensation, with Especial Reference to the Pain of Visceral Disease," Brain, vol. xvi., Part I., 1893; Ross: Brain, 1888; Mackenzie: Medical Chronicle, 1888: Mackenzie: "Points Bearing on the Association of Sensory Disorders and Visceral Disease," Brain, vol. xvi., Part III., 1893. Also, papers by Starr. See chapter on Nervous Diseases.

ment of the nutrition of the nervous system as a whole, and manifested as pain in the regions of least resistance.

Causes. Conditions acting upon the *peripheral ends* of the sensory nerves are injuries or disease of the surfaces or of the viscera. Conditions acting upon the *nerve-trunk* may be either internal or external. Among the internal causes are the acute and chronic forms of neuritis. Among the external causes are tumors, perineural inflammatory processes, or anything causing mechanical injury to the nerve itself. Nerve-roots are usually involved in intraspinal growths, in spinal meningitis, and, occasionally, as a result of disease of the vertebral column. The lesions causing the *central* pain are cerebral embolism, hemorrhage, softening, inflammatory processes, tumors, and injuries. General causes are the anæmias, the intoxications, the infectious fevers, and perhaps certain drugs when taken habitually, as morphine, although it is usual to include the pains complained of by opium-eaters among those due to simulation.

Variations in Disease. Pain is, perhaps, the most variable symptom in disease. It ranges from a sensation of mere discomfort, as the dull ache of chronic lumbago, to the stabbing pain of pleurisy or the intolerable anguish of heart-pang. It is at times compatible with the highest mental endeavor or the severest physical exertion, at other times the whole energy of the organism is absorbed in resisting it. It may be definitely localized in any part of the body, in any of the tissues, or distributed over an ill-defined area.

The Recognition of Pain.

The Mode of Expression. As a rule, the physician learns of the existence of pain by the complaints of the patient. Thus he learns more or less accurately its location, character, degree, and duration, and usually something concerning its causation. But the value of this source of information is variable. The patient may be voluble and describe too much; or taciturn and shrink from admitting his suffering; or ignorant and unable to give a clear account. Fortunately, there are other ways by which suffering is expressed which may be grouped among the objective symptoms. They are:

Facial Expression. The expression of the face is the most common interpreter of the emotions, and is far more reliable than the patient's statements. The tense and drawn lineaments, the clenched jaws, the dilated pupils, the livid countenance, make a picture of agony which, with the labored respiration, the general shrinkage of the body, is unmistakable. (See Chapter XX., Part I., The Face.) Or, in a less intense form, the shrieks and struggles or the groans of more prolonged suffering are no less impressive in their suggestiveness.

Attitude. Not less characteristic are the various postures assumed: the sudden fixity of heart-pang; the retracted head of meningitis; the immobile side of pleurisy; the crouching attitude or restlessness of colic; the flexed thighs and immobile trunk of peritonitis; the shoulder drooping to the affected side in renal colic; the bent knee of arthritis.

Reflex Actions. Further, there are certain reflex actions that are associated with local irritations: thus the closing of the eyelid on irritation

of the conjunctiva; the sneeze or cough on irritation of the nasal or laryngeal mucous membrane; the erection following irritation of the urethra; or even the limp characteristic of pain on moving or resting the weight of the body on an affected limb. Then there is the sudden shrinking of the whole body, the attempt to defend, or the sudden movement of the hand to the affected part, or the sudden jerking away of the part itself if the act be possible; these are true reflexes, and sufficiently diagnostic of local suffering. It scarcely need be mentioned that in children, in the insane, in persons unable for any reason to communicate their thoughts, the bodily expression of pain is of the greatest diagnostic value in determining its seat.

Objective Signs of Disease. The phenomena of the associate morbid processes may serve to indicate the occurrence of pain and its seat. Thus pain is one of the cardinal symptoms of inflammation; it is commonly associated with nerve-injury; it is frequently accompanied by local flushing or herpetic eruptions in neuralgia.

Sources of Error. Unfortunately, pain is one of the most unreliable of symptoms. It is necessarily a subjective symptom, with, in all probability, qualitative as well as quantitative variations. The particular degree in either respect is of importance in diagnosis, and as only the roughest means, if any, are available to estimate it objectively, the physician is compelled to rely almost wholly upon the statements and appearance of the patient.

Error as to the presence or absence, or the degree of pain, arises because of: 1. The character of the patient; 2. The surroundings of the patient; 3. The psychic state of the patient; 4. The physician.

1. The patient may *exaggerate* his sufferings or *belittle* them. The tendency to exaggeration is most marked in those of nervous temperament; in those suffering from chronic disease of long standing; in those accustomed to indoor and mental labor; in women; and in the young. The tendency to depreciation is most marked in the phlegmatic temperament; in those accustomed to hardship, especially if of small intellectual development; in men, and in the aged. Both tendencies are to be neutralized by observing the associated symptoms and the character of the patient, and by skilful questioning. The appearance may deceive because of undue susceptibility to suffering on the part of the patient, or unusual inhibitory power. There can be no question that painful stimuli, usually easily borne, in some produce almost unbearable misery. Such exaggerated sensibility occurs in the emotional, in the weak and debilitated, and in the delicately nurtured.

2. *Mental association* is a powerful factor; it is well known that soldiers, who in the heat of battle disregard serious and necessarily painful wounds, will suffer intensely under the probably less painful offices of the surgeon; and it is unfortunately a common experience that the surroundings of the operating-room exaggerate the pain of trifling operations.

3. *Inhibition* is a much more serious source of error, for while undue attention to one part is only reprehensible when practised to the neglect of others, a patient who disregards pain may fail to direct attention to the real seat of disease. It is sometimes exercised to a most remarkable

degree. The stoicism of the American Indian under torture is attested by many observers; certain religious sects among the Hindus habitually inflict the most ingenious tortures on themselves; the early Christian martyrs rejoiced in misery. It is common to find this disregard of pain among those exposed by occupation to discomforts and injuries; and the Teutonic and Slavic races appear to possess it in a higher degree than the Celtic or Semitic. *Shock* either inhibits pain or diminishes the normal response to it. Habitual use of opium seems to increase the susceptibility in a remarkable manner. Patients will even submit to operation for the relief of a supposed ailment that is found to have no physical basis; and this occurs in cases in which there is no reason to believe that the pain is simulated as an excuse for the indulgence. Moreover, a pseudoneuralgia is wont to occur in victims of the morphine-habit. It may simulate a gastralgia or an intestinal colic. The writer has seen an innocent victim of morphine suffer from pseudohepatic colic, withdrawal of the drug causing subsidence of the periodic attacks of pain and vomiting.

4. Lastly, and by no means to be neglected, a most common source of error is *undue credulity* or *scepticism* on the part of the physician, for he may, on the one hand, be deceived by an eloquent and persuasive complainant, or, on the other, discredit true suffering. In fact, great insight and an acute clinical sense are required on the part of the physician to estimate the value of pain. Herein he displays his innate skill, although large experience and a wide knowledge of human nature equip him to a large degree.

Simulated Pain (see Feigned Disease) is to be recognized by the existence of a motive for deception.

Malingering. Simulation is common in those who seek damages for injuries, or in those who have a morbid craving for sympathy and attention. Its detection depends upon the skill of the physician, who, by distracting the attention from the part complained of, observes that the pain disappears, or, on the other hand, that pain is admitted in a part to which attention is directed; moreover, the physician observes an absence of adequate physical alteration, and usually inconsistency in the symptoms, for the malingerer is seldom able to simulate a correct clinical representation for any length of time. Especially in the latter case is the observation of the invalid's surroundings of considerable importance.

Hysteria. The so-called hysterical mask is of much value, for the bitter complaints and the placid or even smiling features cannot fail to strike the observer by their incongruity. True hysteria is apt to be deceptive, and more than one humiliating failure is recorded of even the most skilful of our craft. The difficulty is increased because actual physical changes occur, as amaurosis with dilatation of the pupil, contracture and induration about the joints, unquestionable anesthesias, and palsies. True hysteria is often to be detected only after prolonged and painstaking study of the case; the careful exclusion of organic visceral disease; by the absence of the characteristic symptoms of the nervous degenerations, such as ankle-clonus, or altered electrical reactions, or changes of the eye-ground; and often by the impossibility of associating the sensory lesions with the known anatomical distribution of the nerves.

Objective Investigation of Pain.

In order to estimate accurately the diagnostic value of pain, the statement of the patient must be corrected by his expression, posture, and manner, and the apparent nature of the disease. Pain is one of the cardinal symptoms of inflammation; vasomotor and muscular disturbances are often associated with neuralgia; any morbid condition exerting pressure on a nerve-trunk, as a neoplasm, callus, etc., commonly causes pain. Hence, if the objective phenomena of these disorders are present, they lend color to the complaint of pain; and if not present, they should be inquired for.

Attempts have been made to estimate the acuteness of the pain sense with scientific accuracy, or at least to secure a practical method for measuring its varying intensity in different localities in the same case. Björnström, of Upsala, has contrived a pair of forceps that compress a fold of skin; the amount of pressure required to produce pain, which can be read from a scale, indicates the degree of sensibility or rather resistance to painful impression. Another instrument, Buch's, accomplishes the same thing by direct pressure, and hence can be used over the superficial nerve-trunks. Another method more generally available is the application of an induced faradic current of variable strength—single naked-wire electrodes being best for this purpose. The common clinical method, by far the most inaccurate and only applicable in cases of marked analgesia, is a pin or needle forced through a fold of skin. No method has yet been suggested for even the approximate estimation of the acuteness of sensibility to internal pain, and it must still be left to the judgment of the patient.

Clinical Value of Pain.

We can judge of the character, temperament, and nervous susceptibility and perturbability of the patient by the symptom pain and the mode of its expression. It aids us in the recognition of hysteria and helps to detect the malingerer. We learn the patient's capability of resistance, and hence, in a measure, his strength. We learn the quickness of receptivity in consciousness of the peripheral irritation, or the degree of intelligence, or the amount of stupor; or, if conditions are present that usually cause pain, its absence may show disease of the conducting paths to the brain.

The absence of pain which otherwise is usual points to the occurrence in the local process of such change as has destroyed peripheral nerve-endings. Thus, when pain ceases in dysentery gangrene has ensued. In intestinal obstruction its cessation indicates the same process. In profound shock pain is not complained of; the amount of pain, therefore, indicates the degree of shock. Hence, in peritonitis, in which shock frequently occurs, pain may be wanting entirely. The abdominal surgeons welcome its occurrence after an operation, as it indicates the absence of shock.

Diagnostic Significance of Pain.

Pain enables one to determine the location of disease and the *nature* of the causal morbid process.

The Location. The location of the disease is determined (*a*) by the seat of the pain (see 5 below) and in part (*b*) by the mode of expression. The modes of expression also indicate its point of origin in a general way and its probable cause. They are (1) the facial expression, (2) the posture, (3) the reflex actions, (4) the associate phenomena. They need not be referred to again. (See page 61.)

The Morbid Process. The nature of the causal morbid process is judged by the study of pain from various standpoints. Thus we must learn (1) the mode of onset, (2) the duration, (3) the time of occurrence, (4) the character or variety, (5) the location, (6) the modifications produced by pressure, temperature, rest, motion, posture, electricity, drugs, and climate.

1. Mode of Onset. The mode of onset of pain in the majority of cases indicates whether the morbid process is acute or chronic.

The onset may be *sudden:* (1) in gout or acute inflammations of serous membranes, as pleurisy or peritonitis; (2) in certain headaches, particularly in those of congestive or emotional origin; (3) in acute obstruction of canals; (4) in contraction of muscular structures in their effort to expel a foreign body, as in the intestines, the gall-ducts, the vermiform appendix, the ureters and bladder, or uterus; (5) in rupture of the structure in which the pain is developed. In the latter instance we have the most typical sudden pain, providing no line of defence had been set up by previous inflammation. Thus in rupture of an aneurism or of the heart there is sudden, sharp pain. In rupture or perforation of the stomach or intestines, or any of the hollow viscera, this kind of pain arises. (6) Sudden pain also occurs in certain neuralgias or neurosal affections. It is seen in angina pectoris, locomotor ataxia, and in acute brow-ague, or trigeminal neuralgia.

The onset may be *gradual*, and followed by continuous increase in intensity or variation. Such onset indicates that the process is one of slow development and not attended by a "solution of continuity," as from rupture or tear. It is observed in various forms of rheumatism, in inflammations of muscles and of mucous membranes, in chronic inflammations of serous structures, in chronic bone disease, and in slowly developing mechanical pressure, as by tumors.

2. Duration. The duration of the pain also indicates the acuteness or chronicity of the causal morbid process. (*a*) Pain of *short* duration is seen in the affections in which it develops suddenly (see Mode of Onset), in acute serous inflammations, and in neuralgias. (*b*) Pain of *long* duration, if constant, is usually due to organic lesions; if intermittent, it may be due to neuralgia. Pain that is continued over a long period of time excludes the sudden accidents that have been mentioned, unless change in the character of the pain takes place.

Pain is also divided, as to duration, into temporary and constant pain. *Temporary* pain indicates total or partial abeyance of the morbid process,

while *constant* pain points to its continuance. Constant pains are seen in bone affections and in inflammation of muscles; the reflex pains due to chronic disease elsewhere, as the backache of uterine disease or the inframammary neuralgias from the same cause, are also constant.

Pain may also be *intermittent* or *remittent*, *paroxysmal*, or *periodic*. (*a*) Intermittent and remittent pains are characteristic of neuralgias, or point to a functional origin; they recur because the cause which superinduces them again becomes operative. Thus recurring headaches due to eye-strain may be intermittent or remittent in the sense that they occur only when the eye is used. Attacks of such pain recur over a long period. (*b*) Paroxysmal pain is the form which occurs when there is obstruction of channels, as the gall-ducts in biliary colic, the intestines, the uterus, and the ureters in the various forms of colic to which they are liable. The paroxysms of pain recur in the course of the attacks. (*c*) The term periodic is applied to pains that occur at distinct intervals. Pain that is periodic has frequently for its cause malaria in some form. The toxic headaches and nerve headaches, as migraine, are often periodic. (Consult Headaches.)

3. Time of Occurrence. *Diurnal* pains are usually reflex from functional disorders. Some pains, as headache due to cardiac weakness and to forms of anæmia, are present during the day, because the patient is in the upright position; they disappear in the recumbent position, and hence are absent at night. Neurasthenic pains are worse in the forepart of the day. *Nocturnal* pains are common in syphilis. They are usually due to periosteal inflammation.

The *time-relation* of pain to functional acts is of importance. Thus in gastric pain its relation to the taking of food is to be ascertained. Pain coming on before meals is gastralgic; occurring after meals, it is due to ulcer, cancer, or gastritis, sometimes to indigestion; occurring several hours after, it may be due to pyloric obstruction or duodenal ulcer. Chest pains, increased by the act of breathing, are muscular or pleuritic.

4. Character. The character suggests the location and the cause. *Sharp*, *lancinating*, *gnawing*, or *stabbing* pain is usually due to inflammation of serous membranes, to various forms of colic, and to forms of neuralgia. *Cutting* pain is a sharp form that occurs in flatulent colic. *Cramp* is pain due to sudden spasm of muscles or to spasm of the intestines, as in colic. *Gnawing* or *boring* pain attends the pressure-necrosis of aneurism, and is characteristic of gastralgia or hyperacidity. *Throbbing* pain is usually associated with acute inflammation, whether superficial or deep. It may be rhythmic with the pulsations of the heart. *Dull* pain is due to slow, chronic inflammation in the bones and in the viscera; it is the pain of myalgia and of fatigue in the muscles. It may be of an aching character. But *aching* pains may also be general; they are found among the prodromata of the acute diseases, attend and follow a chill, and occur in most characteristic form in influenza and dengue. *Pressing* pain is complained of when it attends an attempt to remove material from the viscera, as the passage of water when the bladder is inflamed; the passage of feces in dysentery. The term *tenesmus* is applied to it, so that

we may have vesical tenesmus and rectal tenesmus. The passage of clots or other material from the uterus is attended by pain with pressure or "bearing-down," as it is termed.

NATURE OF THE DISEASE. Finally, the character of pain is often an indication of the nature of the disease as well as of the tissue affected: 1. Thus boring and constant pain is seen in bone and periosteal disease. 2. Soreness or aching in muscular affections. 3. The pain is sharp and stabbing when serous membranes are affected. 4. Smarting and burning, or, perhaps, dull pain and soreness, when mucous membranes are inflamed. 5. Burning or itching in affections of the skin. 6. Dull and usually constant in visceral affections, although in malignant disease of various organs it may be sharp and paroxysmal. 7. Aching, burning, and throbbing in the nerve-trunk and its distribution, with tenderness, commonly indicate neuritis. 8. A sense of swelling, distention, or bursting attends the pain of obstruction of hollow viscera, as in renal or hepatic colic. 9. Rending or tearing pain may be complained of when a hollow viscus or sac is ruptured, as notably in rupture of the sac of extra-uterine pregnancy. (See "Pain Crises," page 69.)

5. Location and Distribution. It may be of questionable advantage in some cases that the localization of pain generally indicates the situation of the morbid process. Too often an apparently adequate explanation of the symptoms may thus be found, while other pathological changes may be overlooked. But, on the other hand, the conditions to which attention has been called by the pain might, on account of its obscurity or unusual location, otherwise escape observation altogether. For the diagnostic significance of local pains see the respective chapters devoted to local diseases.

Pains may be general or local.

a. GENERAL PAINS. They are due either to central or to peripheral disturbance of the nervous system by a poison circulating in the blood. This may be the poison of fevers, or it may be a rheumatic or gouty poison. It is seen in the common affection known as "cold," when the pains are probably myalgic. In syphilis, malaria, lead-poisoning, and toxæmias generally, there are general pain, soreness, and fatigue. General muscular soreness is a constant accompaniment of rachitis and scurvy. General pains are not confined to the muscles, but may be seated also in the fibrous structures and bones. In their more severe forms such pains occur in dengue, which is also known as "break-bone" fever.

b. LOCAL PAINS. The location is, in general, an indication of the seat of the disease. It may be accepted as an almost universal rule that pain due to a local process is limited to the immediate or associated nerve-supply of the diseased region.

Referred Pains. The above statement holds true even when the referred pains—that is, those felt in the associated nerve-supply—are as far distant from the site of the morbid process as the knee pain of coxitis, the shoulder pain of hepatic disease, pain in the neck from pericarditis or diaphragmatic pleurisy, the ear and temporal pain of lingual carcinoma, the pain in the legs from cancer or ulcer of the rectum, the testicular and

thigh pain of renal colic, or the umbilical pain of vertebral disease. (See Figs. 1 and 2.)

Sympathetic Pains. Hilton lays down the rule that pain in any part, in the absence of a local process, is due to exalted sensitiveness of the nerves of that part, and depends upon a cause remote from the painful area. Further, Hilton remarks that pain on the surface of the body must be expressed by the nerve which resides there, and, hence, the cause of the pain must be situated between the peripheral termination and its central origin. This applies particularly to the pains which arise from disease of the vertebræ and to the referred pains described above. It follows therefore that in the investigation of the cause of pain, the nerve, its anastomoses, and the organs supplied by it should be investigated.

Local pains may be (*a*) superficial or deep-seated; (*b*) circumscribed or diffused; (*c*) bilateral; (*d*) unilateral.

(*a*) *Superficial* pains are due to involvement of the superficial nerves distributed to the skin or to the muscles directly underneath, or to the structures in close relation to the skin, as the peritoneum, the pleura, or the pericardium. *Deep-seated* pains, when in the extremities, are due to bone disease; when in the abdomen, to disease of the viscera, particularly inflammatory affections, to aneurism, or to bone disease; when in the chest, to disease of the aorta or mediastinum.

(*b*) *Circumscribed* pain is always due to a limited area of disease, or is reflex. Thus, in ulcer of the stomach the pain is usually circumscribed in one spot of the epigastrium; in inflammation of the appendix, to the region of that structure. *Diffused* pain indicates involvement of a large area with less intensity of process than when the pain is circumscribed. When the pain is diffused, or, as it is sometimes called, radiating, over an area of nerve-distribution, its point of origin may be found somewhere in the course of the nerve, either in the trunk or in one of its branches. *Corollary:* Given pain in a locality, study the nerve-supply of that region and the nerve-anastomoses connected therewith. We learn much from the study of this distribution. The referred pains have been indicated. (Page 67.) Among others, the pain of angina radiates down the arms. The pain of diaphragmatic pleurisy is referred to the front of the abdomen above the umbilicus. Radiating pains, however, are chiefly due to disease in the course of the nerve, the pain being referred to its trunk and terminal distributions, as pain in the foot in sciatica. Pain from pressure upon the nerves at their exit from the spinal canal is felt at the periphery of the nerves, as in the centre of the abdomen, and not at the point of exit. Pain in this locality is frequently an indication of disease of the vertebræ, the pain being propagated by the sixth or seventh dorsal nerve. Pain between the shoulders is often due to an aneurism pressing upon the vertebræ. (See Pain in the Heart.)

(*c*) *Bilateral*, or symmetrical, and surface pains indicate a central or bilateral cause; while, on the other hand, (*d*) *unilateral* pain implies that the causal lesion is one-sided.

c. Peripheral Pain of Central Origin. We have referred to pains of the extremities or trunk due to central disease. In meningitis and other general organic affections of the brain and cord peripheral pains

are frequent, and may be the earliest and most striking symptoms. Indeed, it is very common to find patients with spinal cord disease who have been treated for a long time for what was supposed to be rheumatism. The pains in the joints of central origin may be constant, or paroxysmal and lancinating when the disease is chronic. (See Character.) The cardinal rule that all peripheral pains without obvious local cause should lead to an examination of the nervous system must never be forgotten. The paroxysms of pain may be most excruciating, and sometimes cause collapse. They are known as *painful crises.* Pain may be complained of in various viscera, as well as in the joints. Sudden, intense pain, with functional disturbances of the affected viscera, occurs independently of any lesion of the part or of any apparent exciting cause. One class of these attacks has received the name *gastric crises.* The pain is in the epigastrium, and is associated with vomiting. In another class *laryngeal crises* occur, with pain in the larynx and violent spasmodic cough, with dyspnœa ; the pain extends over the shoulders. Or we may have *rectal crises,* with sensation of burning in that situation ; *urinary crises,* simulating renal colic ; and *genital crises.* Pains in crises also occur in the muscles. Crises occur chiefly, if not entirely, in locomotor ataxia. They are distinguished from pain due to other causes by their sudden onset, their extreme severity, the absence of organic disease or local cause in the affected viscera, the sudden termination, the normal condition between the attacks.

Reference must be made to the curious change that takes place in persons with chronic morphine intoxication. Such persons are very prone to have functional pain. This form of pain is usually paroxysmal and severe, and may simulate organic pains. The most common clinical form seen is gastralgia. The subjects of locomotor ataxia suffer much from pain, and have to take enormous doses of morphine. This habit is soon acquired, but notwithstanding the large doses of the drug paroxysmal pain continues, and in its severity simulates the crises of the primary disease. It is very difficult and often quite impossible to decide whether the pain is due to the morphine-habit or to the primary affection. (See Source of Error, p. 62.)

6. Pain Modified by Pressure, Movement, Rest, or Mental Diversion. We also study pain under the influence of pressure, movement, temperature, rest, etc.

Pain that is modified by *pressure* is generally superficial. It is usually of an inflammatory origin. The variety of the pressure gives some clue to the nature of the pain. If the pain is increased by pressure of the finger-tips, it is due to ulcer or inflammation when internal, and to inflammation if external. Although of visceral origin, gastralgia and colicky pains in the intestine, which may be neurotic in origin, are relieved by pressure, particularly if the whole hand is applied. Pain from the dislocation of an organ, as a movable kidney or displaced uterus, or from dependent viscera, may be relieved by judicious pressure in the proper direction, so as to correct the displacement. Pain from affections of the nerve-trunks can be distinctly localized by pressure in the course of the nerve-trunk, and particularly at the points where the cutaneous filaments of the nerves come through the fascia. These points in the thorax are along the vertebral column, in the axillary

region, and anteriorly about the parasternal line—the points of Valleix. We distinguish neuralgias from myalgias by the presence of these tender points. Pain due to bone disease can frequently be distinguished in this way. By pressure or weight upon the head or shoulders we may ascertain if pain is due to vertebral disease. The presence of renal calculus or of gallstones may be determined by the excitation of pain by pressure.

Pain increased by *movement* points to an affection of the bone, muscle, joint, or nerve in the part moved; groups of muscle may be isolated for the tests. Some few pains are relieved by movement of the body only because the mind is diverted in this act. Pain, when superficial and increased by movement, is due to neuritis, myalgia, or rheumatism.

Almost all pains are modified by *rest*. Its influence has but little diagnostic significance. In some cases of doubt as to the nature of a visceral pain, functional rest of the organ, by which relief is obtained, may aid in determining its locality. Thus, resting the eyes may relieve a headache, the nature of which was obscure until this respite was secured.

Pain modified by *temperature* (cold or heat applied to the spine, ice or hot water in a sponge) and by *electricity* usually gives information as to the seat of the disease in the spinal column, of which the pain is the external expression.

Pain modified by *climate* is rheumatic or neuralgic; if modified by weather or season, it is due to neuralgia or neuritis, whether of gouty or traumatic origin.

The patient may describe an excruciating pain in an area, but not exhibit outward evidence or physiological change which should accompany such suffering. Thus the pain may simulate that of peritonitis. Such pain is often modified and mollified by fixing the *attention* of the patient on some other part or on some extraneous subject, when the previously alleged tender area may be pressed upon without causing any evidence of suffering. Similarly, attention may be called by a leading question to pain in some other region. The admission of the occurrence of such pain, with other evidences of hysteria, points to the underlying causal factor in the production of pain. A most important characteristic of pain, and one that serves to distinguish the pain of organic disease from that of hysterical origin, is its variability with excitement or on fixation of the attention of the sufferer on other parts. Moreover, the subject will fall into the trap of describing it as having characters contrary to the usual attributes of pain or as being associated with phenomena not compatible with the pain—if judicious leading questions are put.

Résumé. Notwithstanding clinical investigation we may not be able from the character and locality to determine the real cause of the pain. In general it may be borne in mind that pains are due:

1. To disease of the central nervous system or of the nerve-trunks;
2. To inflammations;
3. To intoxications (neuralgia), as from malaria, lead, and other forms of toxæmia.
4. To pressure on the nerve-trunks;
5. To reflex influences.

If in doubt, therefore, the general symptoms and condition of the patient must be ascertained in order to determine the causal origin, and hence the true nature of the pain. In all cases of pain the controlling motive in diagnosis should be to determine the *general condition* of the patient and to find the local *cause* of the pain.

Pain in the Head.

Pains in the head may be classified, according to location, into those due to affections of the scalp, those due to affections of the cranium, and those due to intracranial conditions.

1. Affections of the scalp are to be further classified as those of the skin, those of the occipitofrontalis muscle, and those of the nerves.

The Skin. The occurrence of itching and burning commonly indicates some local condition of the skin; if the itching is slight, seborrhœa should be looked for; if more severe, eczema; burning and itching of a severe type commonly indicate dermatitis venenata; *Pediculus capitis* should not be forgotten. A feeling of tension, with soreness, accompanies the eruption of erysipelas. Intense local irritations are caused by burns and scalds; the latter, however, are alone likely to give rise to error, because the hair is not immediately destroyed. A sore feeling, with tenderness limited to a sharply defined swelling, with a sensation of less resistance in the centre and some darkening of the skin, is diagnostic of a bruise. Hyperæsthesias of the scalp frequently accompany meningeal and cranial affections, and there are even local changes, such as the so-called puffy tumor of necrosis of the inner table of the skull.

The Muscles. Sharp pains in the occipital or frontal region, increased by wrinkling the scalp or brief pressure, but generally relieved by firm and constant pressure, occurring with irregular periodicity, and associated with meteorological changes, are suggestive of occipital *myalgia*. The diagnosis is confirmed by the presence of other symptoms of lithæmia.

The Nerves. Neuralgia occurs in paroxysms, accurately located in the course of one or more of the nerve-trunks, and presenting points of special sensitiveness where the nerve emerges from the skull and where it divides for its cutaneous distribution. The pain is usually relieved by firm pressure, but it is to be remembered that sharply localized pressure on the nerve-trunks against the hard skull will cause a traumatic tenderness. The character of the pain is variable; it may be of the most acute or rending form, or, but more rarely, a persistent dull ache; it may be throbbing, or occur in successive paroxysms at brief intervals, or it may be regularly periodic. There are often associated vasomotor, secretory, and motor disturbances; local blushing or sweating may be observed along the course of the nerve; and spasms may occur in the muscles of the eyelids for instance, or more general spasms, as in the terrible tic douloureux, distinguished by pain from tic convulsif. The commonest seats are the supraorbital, the dental, the auricular, and the occipital nerves. In the great majority of cases the pain is unilateral.

The sensory nerves of the scalp and face are the trigeminus and the branches of the cervical plexus. The distribution is as follows: the

ophthalmic division of the trigeminus is distributed to the eyeball, lachrymal gland, the mucous membrane of the nose and eyelids, the integument of the nose and upper eyelid, the forehead, and the anterior half of the hairy scalp. The *superior maxillary* division supplies the skin over the malar bone, and that of the lower eyelid, side of the nose, and upper lip; the upper teeth, the upper part of the pharynx, the antrum of Highmore, and the posterior ethmoidal cells; the soft palate, tonsil, and uvula, and the glandular structures of the roof of the mouth. The *inferior maxillary* division is distributed to the side of the head, the upper anterior portion of the external ear, the external auditory canal, the lower lip, and the lower part of the face; the tongue, the mouth, the lower teeth and gums, the salivary glands, and the articulation of the jaw. The *great occipital* is distributed to the back of the head, the *small occipital* to a narrow region just in front of it, and the greater *auricular* to the skin of the posterior portion of the pinna and the skin over the mastoid process and parotid gland.

Pain simulating neuralgia is frequently due to some *local irritation;* foreign bodies have been known to cause paroxysmal attacks for a number of years until removed; diseases of the bones are a prolific source, especially in the case of the jaws and the cervical vertebræ. Enlarged cervical glands occasionally irritate the great auricular and small occipital nerves. Bilateral occipital pain is very characteristic of cancer of the cervical vertebræ. In these cases there is usually pain on movement of the head or pressure upon it, and some stiffness of the neck. Intracranial growths occasionally cause pains, usually paroxysmal, limited to one of the branches of the trigeminus.

Reflex Neuralgia. Certain of the cephalic nerve pains are symptomatic of disturbance in the associated but distant nervous distribution. Pain in the region supplied by the ophthalmic division is very common in influenza. It is usually dull, aching, and continuous, increased by pressure and anything tending to increase congestion. A severe, acute attack of indigestion will produce ocular and supraorbital pain. Refractive lesions of the eye cause the same kind of pains, which are, however, increased by using the eye and relieved by rest and atropine. The use of the latter is an important diagnostic procedure. Pain in the temporal region and in the external auditory meatus is often due to intense irritation of some of the branches of the inferior dental nerve; the usual cause is cancer of the tongue, but irritable lingual ulcer may also produce it, and even severe inflammatory conditions of the lower jaw. The pain is described as sharp, lancinating, and paroxysmal, liable to exacerbations, especially when the primary lesion is irritated, and relieved when it is alleviated. Pain may be caused in the ear alone when there is irritation of the inferior dental nerve.

Systemic Neuralgia. Perhaps in the majority of cases of cephalic neuralgias the cause is to be found in some systemic disturbance. If the attack is preceded by a desire to sleep, occurs when the dew-point is high, and is associated with increase of urates in the urine, it is probably a so-called *lithæmic* neuralgia; the pure gouty forms are most likely to succeed indulgence in rich food or red meat, and there is ordinarily irritability of

temper. *Diabetic* neuralgias are invariably worse as the amount of sugar excreted is increased, and there are usually similar affections of the nerves in other parts of the body. Regularly periodic pains, worse in the spring and fall, occasionally preceded by a slight chill or malaise, suggest chronic *malaria*. The diagnosis can readily be confirmed by examination of the blood and by the detection of enlargement of the spleen. *Syphilitic* neuralgias are usually worse at night; the pain is described as boring, and may be periodic. There is likely to be some thickening of the bones, and perhaps a diminution of elasticity of the tissues, and almost always local tenderness. The pain is relieved almost immediately by potassium iodide. In *anæmic* neuralgias the pain is not characteristic, but it is improved temporarily by the recumbent posture and stimulants, and is worse during menstruation. The general appearance of the patient and an examination of the blood readily suggest the cause. In *locomotor ataxia* there are occasionally cephalic crises of a neuralgic nature; these come on suddenly and are exceedingly severe, but usually occur only at long intervals; the pain is shooting or stabbing, and does not remain located in one nerve-trunk. Chronic *lead-* and *alcohol-poisoning* also cause neuralgias, but they are not of themselves characteristic, and never occur as isolated symptoms, being frequently associated with peripheral neuritis.

Secondary Neuralgia. Dull, burning pains, commencing perhaps with a chill, and accompanied by febrile symptoms, indicate *inflammations* of the mucous membranes of the head. A dull persistent headache located just beneath the eyebrows often accompanies coryza, and indicates extension to the frontal sinuses; if the nose alone is involved, there are a feeling of fulness and occasional sharp pains or tickling sensations. A feeling of dryness and some discomfort on swallowing accompany the various forms of stomatitis and pharyngitis; in the latter there is also a sensation of tickling and fulness in the ear, due to extension along the Eustachian tube.

Pain at the angle of the jaw, with tenderness, and increased on swallowing, almost invariably unilateral and associated with swelling of the parotid, is unmistakably due to parotitis.

The neuralgias and inflammations of the middle ear are exceedingly painful; they may consist of a sharp continuous pain, or a series of regular exacerbations and remissions, or a throbbing sensation; pain often radiates to the jaws and side of the face. As suppuration progresses the feeling becomes one of extreme tension until the membrane is perforated, when there is immediate relief. Tinnitus is common throughout the whole course of the case.

Inflammations of the eye produce local pain, usually causing the sensation of a rough foreign body. There is usually a slight supraorbital tenderness, and in iritis sharp pains radiate over the whole area of distribution of the two upper branches of the fifth nerve.

Certain ulcers of the mouth are comparatively painless, noma often developing insidiously. Syphilitic ulcers are painless. Simple and tuberculous ulcers are very irritable, but, like carcinoma, are apt to cause paroxysms of pain even when not irritated.

It may not be out of place to mention the value of certain anæsthesias as diagnostic signs; thus in neuritis of branches of the fifth there may be cutaneous anæsthesia while there is tenderness over the nerve-trunk.

2. Affections of the Cranium. A *dull constant headache*, limited to a small area, later increasing in severity, and the pain assuming, perhaps, a boring character; tenderness, often very severe, over the affected area, and probably slight œdema of the scalp, with some rigidity of the muscles of the neck, and the ordinary signs of the inflammatory process, indicate inflammation of the cranial *bones*. In the simple cases there will usually be some history of injury, the pains will not be especially periodic, and the fever will be irregular. In the syphilitic cases there will be the history and symptoms of infection, the pain will become worse at night, and usually there will be a concomitant rise of temperature. The pains will also be controlled by potassium iodide, but as it often requires enormous doses to accomplish this result, the failure of a moderate dose should not be considered as excluding syphilis.

3. Intracranial Headaches. Intracranial headaches are functional or organic. Both forms may be acute or chronic. The typical acute functional headache is seen in the more or less common type known as *migraine* or *hemicrania*.

Migraine is a periodic neurosis characterized by pain in the distribution of the trigeminus and other cranial nerves. The headache is usually unilateral, and, as it is probably due to vasomotor disturbances, is always associated with vasomotor symptoms. It occurs more particularly in women, frequently begins in early childhood, and continues throughout adolescence. It is often hereditary. It occurs most frequently in women who suffer from anæmia or from menstrual difficulties. It sometimes occurs in the early stages of secondary syphilis. The habit which predisposes to the headache may develop after prolonged physical or mental over-exertion. The attacks, however, are excited by over-exertion, mental excitement, or disturbances of digestion. The pain of migraine is situated possibly in the pia and dura mater.

Symptoms. The attack develops with or without premonition. In each individual different prodromal symptoms are recognized as indicating the approach of an attack. Undue nervousness, a general sense of discomfort, pressure or heat in the head, vertigo, tinnitus, spots before the eyes, excessive yawning, and repeated chilliness are the most common.

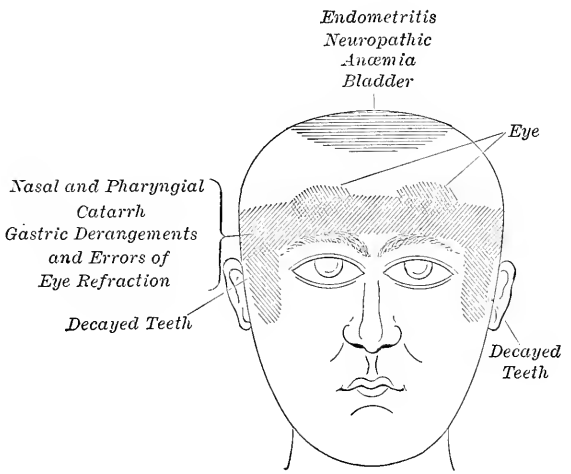
The pain is most frequently felt on the left side of the head first. It may be seated in the anterior frontal, temporal, or parietal region. The pain is continuous, and increases in intensity to the height of a paroxysm. Painful points are not usually detected, although the entire cutaneous area may be hyperæsthetic. The patient is sensitive to light and sound; intolerable nausea intervenes, and vomiting may occur at the height of an attack. The eye symptoms are very pronounced. Flashes before the eyes, scintillating scotomata, or hemianopia may occur.

The vasomotor symptoms that attend the attack are of two varieties, dividing the disease into the spastic and the angioparalytic forms. In spastic migraine the skin on the affected side is cool, the forehead and ear are pale, the temporal artery is contracted, the pupil is dilated, and the

flow of saliva is increased. In the paralytic form there is redness of the face on the affected side; the temporal arteries are dilated and pulsate strongly; the face is hot, the pupils are contracted, and there is often unilateral sweating.

Chronic Headaches. Chronic functional headaches are usually habitual in the sense that the attacks are constant, but there may be longer or shorter intervals of freedom from pain. The nerves affected are the trigeminus, the four upper cervical, and the sensory branches of the vagus to the posterior fossa of the skull. Three types of such head pains are seen: ordinary headache, migraine, and neuralgia. Headaches are caused, as a rule, by diffuse irritation of the peripheral ends of the nerve-tracts above referred to. Neuralgias, on the other hand, are caused by irritations of the trunks of these nerves.

FIG. 1.



Location of pain in various forms of headache.

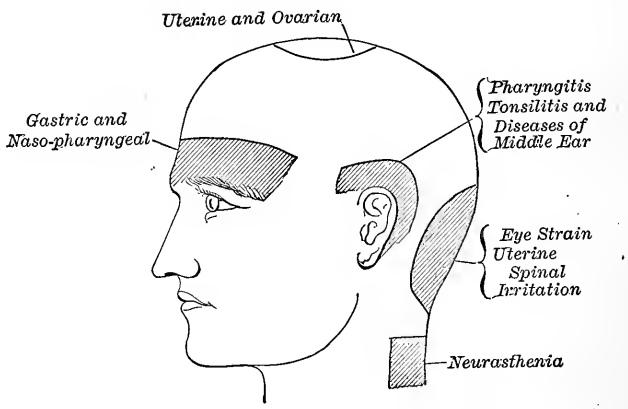
CAUSES. 1. Hæmic: (*a*) anæmia; (*b*) diathetic states (gout, rheumatism, diabetes); (*c*) infections (malaria, syphilis, specific fevers). 2. Toxic (lead and other mineral poisons, alcohol, the poison of uræmia, tobacco). 3. Neuropathic states (epilepsy, neurasthenia, chorea, hysteria, neuritis). 4. Reflex causes (ocular, nasopharyngeal, auditory, gastric, sexual, uterine). 5. Organic disease.

Headaches are divided according to their *situation* into frontal, occipital, parietal, vertical, diffuse, and combinations of any of these forms. The most common forms are the frontal, the fronto-occipital, and the diffuse. Ocular headaches are usually frontal when due to errors of refraction. When due to muscular insufficiencies they are occipital and cervical. Nasopharyngeal headaches are dull, and frontal or diffuse. When the pharyngeal tonsil is enlarged the headache may be dull, frequently recurring, and seated in the occipital region. In follicular tonsillitis and in obstruction of the Eustachian tubes the headache is diffuse. In disease of the middle ear it is temporal and occipital. Gastric or dyspeptic

headaches without constipation are often occipital, sometimes frontal. With constipation and intestinal irritation they are diffuse and frontal. Uterine and ovarian headaches are occipital and vertical. In neuropathic headaches the pain is referred to the top of the head, as in clavus; or it may be associated with spinal irritation. Neurasthenic headaches are usually associated with a sense of pressure or weight, and are seated in the frontal and vertical regions. In spinal irritation the pain is of a boring character and situated in the occipital region. The earliest symptom of the neurasthenic headache is neck-weariness and pain in the neck. Neurasthenic headaches occur in brain-workers when the brain and eyes are overtaxed. Headaches in epilepsy are severe, and are limited to the vertical or occipital region.

Organic headaches are usually violent, associated with fulness and throbbing of the head. They may be remittent, becoming more intense

FIG. 2.



Location of pain in various forms of headache

with each exacerbation. Organic headaches may be due to inflammation, to abscess and softening, to tumor, to congestion of the brain, or to inflammations in the meninges. Anything that increases the quantity of blood in the vessels of the head will increase the pain in organic headaches. In acute inflammation of the brain the pain is agonizing, continuous, associated with vomiting and fever and sometimes delirium. In abscess of the brain the pain is less violent. It is occasionally paroxysmal and attended by paralysis and disturbed intellection. In tumor of the brain the headache is severe and paroxysmal. In congestion the pain is dull, increased by stooping, by sleep, and by bodily or mental fatigue. Some congestive headaches are due to violent exercise, and are relieved by bleeding at the nose. In all congestive headaches the face is flushed, the bloodvessels are turgid, and the vessels in the eye-ground are overfilled. In meningitis the pain is constant, is more or less fixed, and sometimes very sharp. Syphilitic headaches are frontal or temporal, worse at night, and often periodic.

Headaches are divided according to the *character* of the pain: 1. Pul-

sating and throbbing. 2. Dull and heavy. 3. With constriction, squeezing, or pressing. 4. Hot and burning. 5. Sharp and boring. The headaches of the first class are usually associated with vasomotor disturbances, as in migraine. To the second class belong the toxic and dyspeptic headaches; to the third, the neurotic and neurasthenic; to the fourth, the rheumatic and anæmic; to the fifth, the hysterical, neurotic, and epileptic. Vertigo is a common accompaniment of the dyspeptic type of headache situated in the frontal regions. Somnolence is more marked in the syphilitic, anæmic, and malarial headaches. Nausea is more common in occipital forms of headache.

Duration. Eye-strain causes occipital pain, which is rarely persistent, but comes on after prolonged use of the eyes. It may be associated with headache in other parts of the head, due to other causes. In chronic meningitis the headache is persistent and located in the vertex or parietal regions. When thickening of the meninges, with adhesions, takes place from trauma, there are constant pain with frequent exacerbations, sensitiveness of the head, incapacity for study. Uræmic headache is not constant. Persistent headache may be present in the later stages of Bright's disease and in diabetes. In atheroma pain in a part or the whole of the head is common; it may be persistent, though subject to exacerbations in case of excitement or violent exercise. Headache following study, in children, is due to brain-strain, to eye-strain, or to indigestion. Persistent headache is sometimes due to asthma. In rare instances headache is said to be idiopathic. Neuralgic headaches are usually periodic, and may be associated with throbbing or pulsations. They are associated with vasomotor signs. Hysterical headaches are irregular and shifting; they persist after all causes are removed; they are replaced by pain in other parts of the body. They are usually associated with other manifestations of hysteria.

Neuralgia. Neuralgia is characterized by pain in the course of distribution of the affected nerve. The pain is of pronounced severity, and occurs in remissions and intermissions. The symptoms of a neuralgic paroxysm may be preceded by hyperæsthesia over the part subsequently affected. The pain is of a burning or shooting character. It is usually limited to the distribution of the affected nerve, but may extend into other regions. It may be excited by external irritants, by mental excitement, and often by movement of the part. On examination the area of distribution of the affected nerve may be found to be anæsthetic, but usually there is hyperæsthesia of the skin. Wherever the affected nerve is accessible to pressure pain can be elicited. The nerve-trunk may be tender during the attack as well as during the intervals. In neuralgia there is often some spasm of the muscles supplied by the affected nerve.

Vasomotor symptoms are common. The skin may be pale or reddened. When the trigeminal nerve is affected, both the skin and the conjunctivæ are reddened. The lachrymal secretion may be modified. Eruptions like urticaria or herpes may develop along the course of the nerves. Protracted neuralgia may cause marked nutritive disturbances.

General Conditions. A patient who is subject to neuralgia may be in apparent good health, and the neuralgia be due to trauma or to cold. As

a rule, however, the neuralgia is due to constitutional causes, as rheumatism or gout; to some form of toxæmia, as malaria; or to some condition of the blood, as anæmia.

The following individual forms of neuralgia are seen:

1. Neuralgia of the trigeminus, or *tic douloureux*. The entire fifth nerve or only some of its branches are affected. The pain is often severe, and may be associated with twitching, with vasomotor disturbances, with eruptions, and with changes in the secretions. Trophic changes, as the hair turning gray or ulceration of the cornea, may follow. Usually a single branch is affected, either the first (ophthalmic), the second (supramaxillary), or the third (inframaxillary). Points of pressure are, as a rule, readily detected at the foramina for the exits of the nerves. Trigeminal neuralgia must be distinguished from headache due to other causes, affections of the bones and periosteum, and affections of the teeth. The distribution and paroxysmal character of the pain and the points of tenderness assist in the diagnosis.
2. Occipital neuralgia.
3. Neuralgia of the brachial plexus.
4. Intercostal neuralgia.
5. Neuralgia of the lumbar plexus, divided into lumbo-abdominal, crural, and obturator. This form of neuralgia (lumbar plexus) must not be confounded with bone and joint disease, with lumbago, renal colic, appendicitis, and uterine affections.
6. Sciatica.
7. Genital and rectal neuralgia.

Pain in the Nose.

Pain, varying in degree, occurs in all acute affections of the nose. Its seat and character are of some diagnostic significance. Smarting or burning pain at the root of the nose accompanies acute *rhinitis* and attends *post-nasal catarrh*. The pain is diffuse and indefinite in *dry catarrh* and in *diphtheria*. The most severe pain occurs when *foreign bodies* are present in the nose, and in cases of *glanders* and *primary syphilis*. Foreign bodies of a vegetable nature by swelling and germinating induce pain, which increases gradually in intensity.

In tropical regions the larvæ of *Lucilia hominivora* may be found in the nostrils. The pain is so severe at the root of the nose, extending backward, as to cause maniacal delirium. There is a fetid, sanious discharge followed by necrosis.

Pain over the Frontal Sinus.

The pain of an inflamed frontal sinus is more severe than the pain of inflamed nostrils. It is sometimes intense and agonizing. Pain from inflammation or tumors of the antrum may be located in the cheek. In diseases of the nose, if the pain radiates to the *ear*, the Eustachian tubes are probably involved.

Pain in the Larynx.

Pain in the larynx may be sharp, stabbing in character, or simply a tickling or burning with a feeling of pressure. It is increased by pressure and by speaking or swallowing. The pain is sometimes so intense as to render speaking or swallowing impossible. In acute laryngitis the pain is cutting and burning. In the milder inflammations, in dry catarrh, and in lupus it amounts to soreness only. The pain is severe and sharp in cases of cancer and tuberculosis, rarely in syphilis, or when foreign bodies are present in the larynx. The pain may be very severe and intense when there is destructive ulceration. It is a diagnostic symptom of *perichondritis*.

Pain in the Arms.

Unilateral Pain. The pain may be due (1) to neuritis associated with tenderness of the nerve-trunk; (2) to neuroma, as, indeed, any peripheral nerve may be affected; (3) to simple neuralgia, or to neuralgia from the pressure of enlarged axillary lymphatic glands, of a morbid growth, or of an aneurism pressing on the nerves; (4) to rheumatism or myalgia; (5) to bone disease.

Bilateral Pain. Bilateral pain in the arms is of central origin, due to diseases of the vertebræ or of the spinal cord; or neuralgic, due to anæmia or toxæmia of some form.

Pain in the Thorax.

Pains of reflex origin are seated usually in the *shoulder* or the *back*, and are due to liver or gastric disease. The pain of liver disease is referred to the right shoulder; of ulcer of the stomach, to the interscapular and lumbar regions, or to the top of the shoulder, as in a case observed by Wood.

Pain is rarely a symptom of disease of the *lungs* unless the pleura is involved.

In *bronchitis* there may be some soreness and some oppression behind the sternum, but otherwise pain is absent.

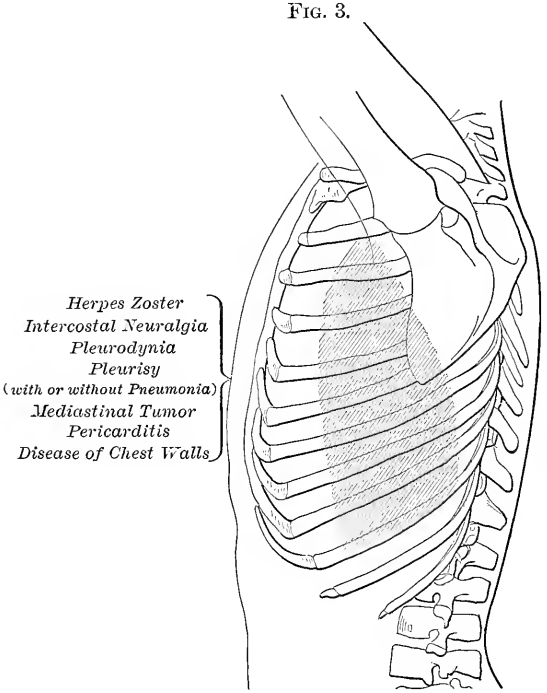
In *pleurisy* pain occurs before the exudation. It is sharp and lancinating, and so severe as to impede the respiration and to cause the cough to be short and catchy. It is seated usually at the base of the chest, in the lateral or anterior region. It occurs when the patient attempts to take a full breath, and causes him involuntarily to check the inspiratory excursion before it is half completed. The patient usually places his hand upon the affected part, and involuntarily leans to the affected side. The pain of pleurisy may be increased by local pressure, but is always relieved by general pressure, as by the whole hand, a broad bandage, or strap of adhesive plaster. In the pleurisy that attends phthisis pain is quite common, being of the same character as the pain of acute plastic pleurisy, but varying in situation and in degree. It occurs in paroxysms and follows a slight exposure to cold, undue exertion, or fatigue. It may persist for twenty-four hours, and then disappear, until brought on again

by a repetition of the cause. It must be distinguished from the myalgia of phthisis which is due to cough and exposure.

In *myalgia* the muscles and fasciæ at the bony attachments are very tender.

The pain of pleurisy must be distinguished from pleurodynia, from intercostal neuralgia, and from the pain due to disease of the ribs.

FIG. 3.



The site in general of thoracic pain in various conditions.

In *pleurodynia* the muscles are sensitive if palpated or compressed between the fingers. A large area is affected, but the physical signs of pleurisy or of pneumonia can not be elicited. Cough and fever are usually absent. The pain is associated with pain in other muscular or fibrous structures. There may be a previous history of exposure to cold and dampness. Usually there is a history of recurrent myalgia.

Intercostal neuralgia is sometimes difficult to distinguish. The pain is sharp, localized, and may modify the movements of the chest. General pressure relieves it; local pressure at the points where the terminal filaments of the nerve come to the surface may increase it. The so-called Valleix's tender points, however, are not always present in cases of intercostal neuralgia. The patient is usually anæmic, is often the subject of uterine or exhausting disease, and may suffer from neuralgia in other situations. Cough and physical signs are absent.

Fracture of the rib, or *caries of the rib*, may be recognized by the physical signs of these conditions and by the local tenderness. Both may,

however, be attended by localized pleurisy, indicated by more severe pain on coughing or on deep breathing. Caries or fracture is determined by pressure upon the diseased rib, which elicits the crepitus of fracture.

An *empyema* that is about to point will cause pain in some area of the chest. The pain is seated usually at the points of election for the discharge of the empyema, as the fifth interspace on the left side, and is soon followed by swelling, heat and redness of the skin, and œdema.

More or less constant pain at the apices, when undoubtedly independent of affections of the muscles, is a suspicious sign of *tubercular disease*. It may be aggravated by pressure.

Pain behind the *sternum* is often a reflex neurosis from gastric disorder. It may occur in bronchitis. It may also be due to cancer of the mediastinum, to aneurism, or angina. Pain in the sternum or ribs is syphilitic, or is due to periostitis or necrosis following typhoid fever, rarely to cancer.

Chronic *fibrous inflammation* of one or more of the attachments of the muscles is of common occurrence. The pain lasts for years. It is persistent, sometimes associated with stiffness; it is increased by movement, and there may be extreme aching pains in the parts.

The pain of *vertebral caries* transmitted along the course of the corresponding nerve has been referred to.

Pain referred to the Heart. **1. In Disease Outside of the Heart.** Pain in the region of the heart is usually due to other causes than disease of the heart or of the pericardium. Pain other than heart pain is frequent in the præcordia, or more precisely in the fifth or sixth interspace on the left side and in the epigastrium.

The causes are: (1) neuralgia; (2) pleurodynia; (3) myalgia; (4) local pleurisy; (5) periostitis; (6) aneurism; (7) abscess.

The *neuralgias* may be associated with points of tenderness, which are usually the seat of greatest intensity of the pain. These points of tenderness correspond with the positions at which the nerves have their exits through the fascia to the surface, and are found along the sternum, in the mid-axilla, and along the vertebræ. The pain is paroxysmal, occurring at variable periods of the day; it is observed in anæmic and neurasthenic subjects. It may precede the development of herpes zoster, in which case the exact nature of the pain is not discoverable until the eruption appears. In gout or diabetes we may have local neuritis, causing neuralgic pain.

In *pleurodynia*, which is thought to be an affection of the pleural nerves, the pain is more diffuse, and is increased by pressure with the finger-tips. It is relieved by pressure with the whole hand.

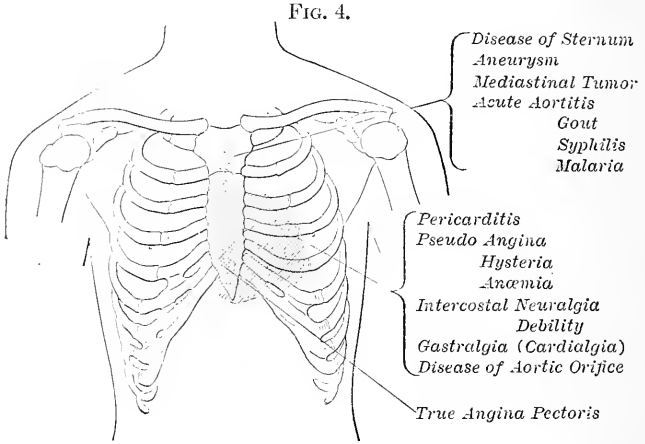
In *myalgia*, which is seen so frequently in phthisis on account of severe coughing, in rheumatism, and in debilitated subjects generally, the pain is more or less diffuse, interferes with the movements of the chest, is relieved by uniform general pressure, and is associated usually with myalgia in other parts.

The pain of *pleurisy* is recognized because it usually inhibits the act of breathing and is associated with cough; friction-sounds may be detected.

Periostitis. The pain is associated with tenderness and swelling. One or more of the costosternal articulations may be extremely tender from the periostitis of syphilis, or from that which follows typhoid fever. In one of my cases the rib had to be resected.

The pain may be due to internal pressure and erosion of the ribs from *aneurism.* This affection may cause neuralgic pains.

FIG. 4.



Pain referred to the heart.

Abscess. Pain in the præcordia may be due to a localized tuberculous abscess between the pericardium and the walls of the thorax, or to empyema.

The Epigastrium. Pain in the epigastrium is often held to be due to cardiac disease, but as a rule it is of gastric or gall-duct origin. The pain of angina pectoris may, however, be limited to this region. Acute, severe, and excruciating pain in the epigastrium may be due to *rupture* of the heart, and also to *pericarditis.*

2. **In Disease of the Pericardium.** *Pericarditis* is a common cause of pain in the region of the heart. The pain may radiate to the left shoulder and extend down the arm. It is paroxysmal and may have some of the characteristics of angina pectoris. It is increased by movement, by pressure, and by the action of the diaphragm. It may be referred to the epigastrium. The patient is often obliged to sit up in bed, and suffers from orthopnœa. A pericardial friction-sound is usually detected.

3. **In Disease of the Aorta.** In *acute inflammation of the aorta* the pain may extend along the course of the aorta, it may be referred to the sternum, or it may extend along the spine. The pain is severe, causing an expression of extreme suffering, and is continuous. In gouty subjects with *atheroma* paroxysmal pain may occur in this situation. There is usually valvular disease at the aortic orifice. Similar pain occurs in syphilitic and in alcoholic subjects, and may be due to malaria.

Aneurism. The pain usually results from pressure of the aneurism upon adjacent structures. The pain is of a boring character, localized at one point, if the aneurism presses on a bone and causes erosion. The

pain is of a dull, aching character, increased by movement, relieved by rest or by change of position; or it may be acute and of a neuralgic nature when nerves are pressed upon. It may follow the course of the nerves and be associated with numbness and tingling. The long duration of the pain, its localization, and its aching character are sufficient to exclude angina pectoris. When the pain is unilateral, it may be due to pressure of an aneurism upon the nerves at their exits from the vertebral canal; the pain extends along the course of the intercostal nerves. It is severe and burning, but there are no localized points of greater intensity. The pain may extend down the arms, and when the abdominal aorta is affected, it may extend down the legs. If *rupture* of the aneurism takes place, the pain is sudden and sharp. Death, however, ensues quickly, so that the pain is rarely complained of.

4. In Disease of the Heart. Three forms are seen: (1) pain due to disturbances of the rhythm; (2) pain due to valvular disease; (3) pain due to angina pectoris.

PAIN DUE TO DISTURBANCE OF THE RHYTHM. In the large majority of cases palpitation, intermission, and irregularity of the heart occur without pain. Paroxysms of palpitation are sometimes attended with severe præcordial pain and distress, as in reflex palpitation; in the palpitation of Graves' disease and of anæmia. The palpitation of organic disease is induced by exertion. The rapid action of the heart is painful and the throbbing causes distress.

While intermission and irregularity may continue without pain, the patient is often conscious of disturbance of rhythm, and complains of the stoppage, the distress of which is more alarming than severe pain, particularly when the heart-action is tumultuous, as in *pericarditis* and in *valvular disease*.

PAIN DUE TO VALVULAR DISEASE. Pain is of more frequent occurrence in disease of the *aortic* valves. It is situated in the region of the aorta at the base of the heart, and is aggravated by exertion. Constant dull pain and pseudo-anginoid attacks are of frequent occurrence in mitral stenosis. (See Atheroma.)

PAIN DUE TO ANGINA PECTORIS. Heberden first described the attacks of true angina pectoris, which are not of common occurrence. The pain of angina pectoris is severe and agonizing. It comes on suddenly, lasts but a brief interval, and recurs in paroxysms. The patient realizes that the pain is in the heart, and complains of feeling as if the organ were held in a vise. There is a sense of impending death with sinking and oppression. From the heart the pain radiates to the neck and down the arms. It extends particularly to the left arm, and may be severe in the wrist or at the tips of the fingers. During the few minutes of pain the patient's face becomes pale or of an ashy hue, perspiration breaks out on the forehead, the extremities become cold, the breathing is short. Prostration usually follows the attack, but the præcordial distress disappears entirely. The attack may occur in patients who are entirely free from valvular disease of the heart. Most commonly, however, it is associated with some lesion. The lesions frequently found are disease of the coronary arteries, atheroma of the aorta, aortic valvular disease, and myocarditis

with fatty degeneration. It occurs after middle life, and is more frequent in males. It may occur without exciting cause, or may follow undue exertion, exposure to cold, mental excitement, or profound emotion.

The points upon which the diagnosis is based are:

1. *The seat of the pain.* This is usually behind the middle or the lower part of the sternum, and more to the left than to the right, or in the epigastrium. Thence it extends to the posterior portion of the axilla or it may radiate to the neck. In some instances it extends to the occiput. Frequently the pain extends to the left arm as far as the elbow or even the fingers. It may extend to the abdomen or to the right arm. I have seen it affect both arms. It is not influenced by external pressure.

2. *Character.* The sense of constriction with the indescribable torture is most characteristic.

3. *The respiration.* The respirations are shallow, or may even cease, but there is no dyspnœa.

4. *The position of the patient.* The attitude is one of fixation.

5. *The expression.* The pale face, extremely anxious countenance, and the cold sweat on the forehead, make a striking picture, which when once seen can never be forgotten.

6. *Subjective sensation.* Such extreme depression and sensation of impending death occur in no other affection. Particularly characteristic is the immediate relief, without hysterical manifestations or dyspeptic symptoms, which follows an attack.

7. *The pulse.* During the attack the frequency of the pulse is not much influenced, and the action of the heart may be uniform and regular. Rarely its frequency may be lessened. The tension of the pulse is increased during the attack.

Some authors speak of various grades of angina pectoris, and call all forms of præcordial pain and oppression, with radiation of the pains to the arms and neck, mild forms of angina. Such attacks have often obvious causes in disturbance of digestion and in emotional excitement. When associated with increased arterial tension and signs of arteriosclerosis, they may be of an anginoid nature. The greatest difficulty exists in distinguishing them from true angina.

Hysterical or *pseudo-angina* can be distinguished only with difficulty. It occurs much more frequently than true angina. One attack predisposes to others. It occurs in females, usually before the age of forty, who present other symptoms of hysteria or are otherwise neurotic. The attacks most frequently come on at night, and may be periodic. They are associated particularly with menstrual disorders. The pain is less severe and the oppression is not so marked in pseudo-angina; coldness of the hands and feet with the occurrence of syncope, or a general feeling of sinking, are common symptoms. The pain is of long duration and is associated with great agitation. It is preceded by neuralgia, and neuralgic pains persist after the attack. Low tension, feeble second aortic sound, and soft arteries may be present, although the opposite is also seen. The disease is never fatal. In one of my patients attacks of hysterical hæmoptysis alternated with the anginal attacks.

Pain in the Mouth and Fauces.

Pain occurs in all inflammations and ulcerations of the mouth except those due to syphilis. It is aggravated by food, by movements of the lips, cheeks, or tongue, and by attempts to eject saliva. Pain is absent in gangrene.

In affections of the fauces and pharynx pain is one of the most common subjective symptoms. The functional acts of the pharynx require movement of all the structures. When they are the seat of inflammation or ulceration, movement excites pain; and the latter is therefore a symptom of great severity in inflammation of the tonsils and pharynx, of rheumatism of the muscular structure of the pharynx, and of tuberculous and cancerous ulceration. Pain in the pharynx is a frequent accompaniment of post-nasal inflammations, although the pharynx itself may not be affected.

Pain in the Œsophagus.

Pain is a common symptom of disease of the œsophagus. In acute inflammation it is extreme, and is complained of in the neck, between the shoulders, and for a short distance along the vertebræ. Its character depends upon the cause. Severe burning pain, often agonizing, is due to inflammation caused by hot or caustic fluids. Absence of pain after the ingestion of such substances, or its disappearance in a short time, points to extreme corrosive action and gangrene. Pain attends and is a part of the symptom known as *dysphagia* (*q. v.*).

Pain in the Abdomen.

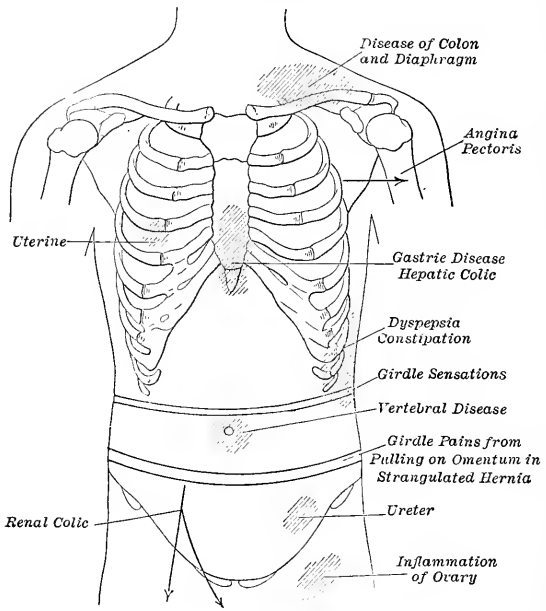
Pain Confined to the Abdominal Walls. The skin, the nerves, the muscles and fasciæ, the connective tissue, may be the seat of pain. When the skin is affected, the pain is usually localized and moderate in severity. There is superficial tenderness. Evidences of inflammation, such as erythema or ulcers, are present. Pain due to affections of the nerves is seen in simple neuralgia and herpes zoster. The latter is recognized by the neuralgic character of the pain, by the fact that its distribution corresponds to that of superficial nerves, and by the characteristic vesicular eruption which follows. Neuralgias are recognized by the well-known points of tenderness, the intermittent character of the pain, and the association with anæmia; neuritis, with the usual objective signs, may be present.

The muscles and fasciæ may be the seat of *rheumatism*, with severe pain and tenderness. Movement always increases the pain, and sighing, laughing, or coughing aggravates it. The pain may be diffuse, and so severe as to be confounded with that of peritonitis. The presence of rheumatism in other muscles and of moderate fever, without gastro-intestinal disturbance, points to the true condition.

Referred Pain. A common cause of abdominal pain is disease of the vertebræ, with pressure upon the peripheral nerves at their emergence

from the spinal column. The pain is situated in the median line, either below the ensiform cartilage or around the navel. It is an intermittent pain. Aneurism of the abdominal aorta, causing erosion of the vertebræ by pressure, gives rise to the same kind of pain.

FIG. 5.



Referred pain in various disorders.

Pain within the Abdomen.

I. Character. Pain in the abdomen may be sudden in onset, or it may represent the culmination of slight sensations of discomfort progressively increasing in severity. The pain may be of brief duration or continue over a long period of time. Sudden acute pain points to inflammation, perforation of one of the hollow viscera, gastralgia or enteralgia, flatulent distention of the stomach or intestines, or to obstruction of one of the numerous channels. Thus, in a case of gastric ulcer, sudden pain suggests that perforation may have taken place; chronic pain indicates the existence of some chronic process such as ulcer, or a gastric or intestinal neurosis. Sudden paroxysmal pain is spoken of as *colic:* the successive spasms being often attended by vomiting, rapid pulse, coldness of the extremities, cold sweat, and more or less collapse, except in lead colic. Such pain is seen in intestinal, hepatic, renal, and in uterine and vesical colic.

II. Location. The *seat* of the pain, whether general or local, will be considered in discussing the individual organs and their diseases.

Diffused Pain. When the pain is general it points to rheumatism, to intestinal colic, to peritonitis, or to tympanitic distention.

Local Pain. It may be said that pain is a fairly trustworthy sign of the existence of disease of some structure in the part to which the pain is referred. Pain over the liver or spleen is generally due to involvement of the peritoneal coverings of these organs, and partakes of the character of local peritonitis. It may, however, be due to extreme congestion, to malignant disease, or to displacements, and the diagnosis must be made by noting the character of the pain, its intensity, duration, seat, and the other general and local symptoms with which it is associated.

Pain in the Epigastrium. Pain referred to the stomach is situated in the upper zone of the abdomen, below the ensiform cartilage, between the ribs of the two sides, usually in the median line. It may extend along and under the ribs of either side. Pain in this situation may be due to a number of causes: 1. To myalgia, neuritis, or neuralgia of the intercostal nerves which terminate in this situation. 2. Localized peritonitis or perigastritis, which may be secondary to infection or injury of the peritoneum from disease of contiguous organs. 3. Affections of the pancreas: *a.* Pancreatic colic, a rare condition associated with diarrhœa, intestinal dyspepsia, and salivation. The pain is paroxysmal, the attacks lasting two or three hours. *b.* Pain due to carcinoma of the pancreas, darting or lancinating in character, associated usually with tumor, jaundice, and emaciation. *c.* Pain due to pancreatic hemorrhage; sudden and extremely severe, attended by collapse. 4. Aneurism of the aorta or of the cœliac axis. The pain is constant, of a boring character, and may be associated with shooting pains along the course of the lumbar nerves. The physical signs of aneurism are present. 5. Disease of the gall-bladder and gall-ducts. 6. Disease of the vertebræ. We should look for the sixth or seventh dorsal vertebra to be affected, hence higher up posteriorly than the area affected in front would indicate. 7. Affections of the stomach—gastric pain.

Pain of Gastric Origin. In diseases of the *stomach* pain is a very common symptom, and presents all degrees of severity, from a mere sense of discomfort or uneasiness to agony. In *atonic dyspepsia* there may be no local gastric symptoms except a feeling of weight and fulness, while in *nervous dyspepsia* there is usually uneasiness or discomfort after eating. *Cardialgia* is a form of discomfort in the epigastrium scarcely amounting to pain, but attended by heartburn or acidity.

In *gastralgia* the pain is characteristic: it usually comes on while the stomach is empty, and frequently recurs daily at the same hour. At first the pain is slight and easily borne; but it gradually increases in severity, each succeeding paroxysm being worse than the preceding one, until a climax of agony is reached. In character the pain is gnawing and cramp-like, doubling the patient up, and leaving him moist with cold sweat and in partial collapse. A not uncommon form is that seen in locomotor ataxia (gastric crises) and in opium-eaters.

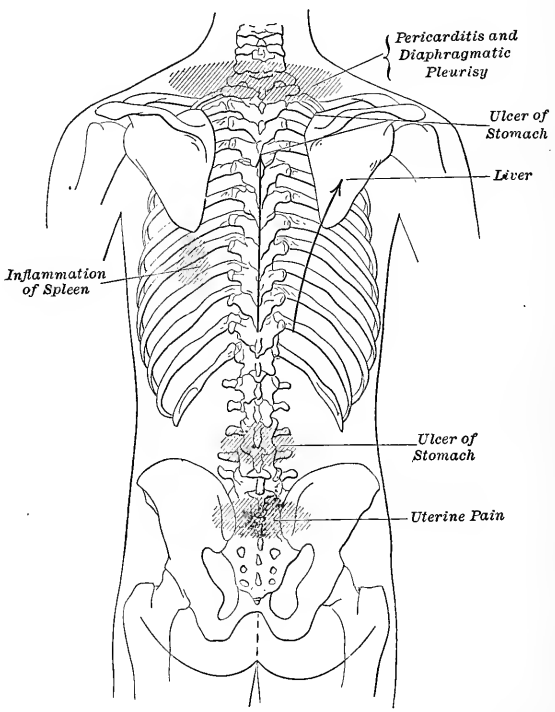
Catarrhal dyspepsia is marked by pain and uneasiness in the epigastrium after eating, with tenderness on pressure; if flatulence coexists, there will be temporary relief to the discomfort upon the eructation of gas.

In *ulcer* there is a more or less constant feeling of soreness in the epigastrium, while after taking food the dull pain is aggravated and becomes

sharply localized. Frequently there is pain in the back at the same level, a little to the left of the spine and between the midscapular region and the lumbar vertebræ. The pain usually occurs sooner after taking food than in the case of cancer, and is more frequently relieved by vomiting. Attacks of gastralgia are not rare, and the pain may shoot down the arm.

In *hyperchlorhydria* (excess of hydrochloric acid) the pain may be exactly like that of ulcer, and it may be impossible to differentiate the two conditions except by the occurrence of hemorrhage and the presence of severe local tenderness. Usually, however, the ingestion of albuminous foods relieves the pain of hyperchlorhydria while it aggravates that of cancer.

FIG. 6.



Direction of radiation of pain in various disorders.

In gastric *cancer* pain may be wholly absent throughout the entire course of the disease; but, as a rule, pain is more continuous than in ulcer, less severe, not so sharply localized, does not come on so soon after taking food, and is not relieved to the same degree by vomiting. Paroxysms of gastralgia are not so common.

In *acute gastritis* the pain and its character vary with the intensity of the inflammation. When due to the irritation of some toxic agent that has been swallowed, the pain is severe and burning; when the result of imprudence in eating and drinking, the pain is of a dull, sickening

character. In either case there is more or less tenderness on pressure. Sometimes, in mild cases of catarrhal gastritis, firm pressure from a broad surface affords at least temporary relief to the distress.

Time of Occurrence. The significance of pain in gastric disease depends on the time of its occurrence. Pain coming on before eating or when the stomach is empty is usually due to hyperchlorhydria, though it may be associated with no alteration in the gastric secretions, and is then termed simply gastralgia; it is relieved by food. When it comes on after eating, it is usually due to organic disease of the stomach, as ulcer or carcinoma; but it may be due to neurasthenia. It must not be confounded with the pain that occurs from two to four hours after meals, caused by intestinal indigestion or some pancreatic affection. When the pain is *diffuse*, it is due to hyperacidity and bacterial fermentation, as in dilatation, catarrhal gastritis, and simple indigestion. When *localized*, it is due to ulcer or cancer, and is associated with tenderness; in such cases it may extend to the back.

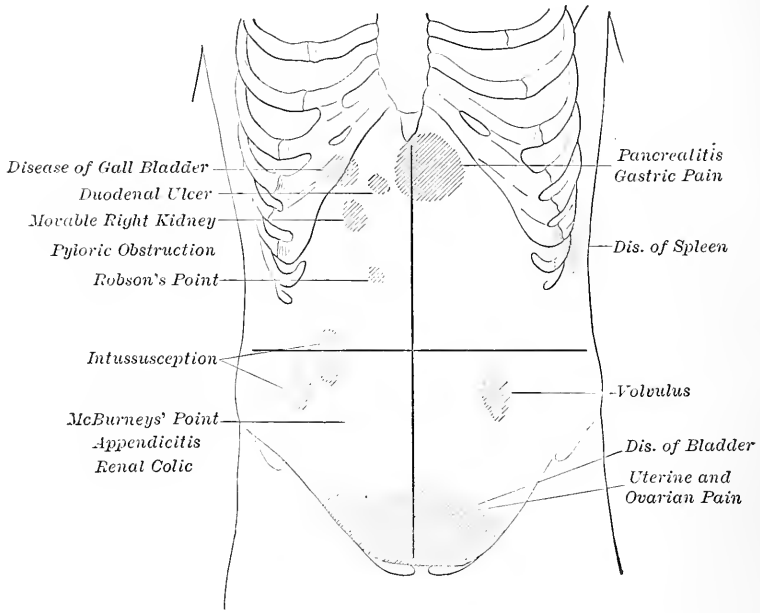
Pain of Intestinal Origin. Intestinal Colic. The colic of intestinal indigestion occurs suddenly, or it may be preceded by other signs of intestinal indigestion. The *pain* is chiefly in the umbilical region and radiates from that point. It is relieved by moderate pressure and by warmth. The patient is restless and irritable; the face is anxious; the skin breaks out in a cold sweat, and the pulse is small and hard. The pain at its height is described as agonizing, and of a boring or shooting character, abating for a time and then increasing again; it may shoot from the region of greatest intensity to the shoulders, back, chest, or iliac region. Prostration or *collapse* rapidly ensues. *Nausea* and vomiting follow the pain, and there are gaseous eructations. *Distention:* the abdomen is distended and tympanitic on percussion. The pain may be relieved by the passing of flatus. *Cramps:* painful spasm of the muscles of the calves is common; the muscles become knotted. The hands and feet are cramped also. The pain is said to be due to spasm of the intestine, and is known also as spasmodic colic. It is certainly due to distention or to irritation.

If the colic is due to *indigestible food*, it may have been preceded by an attack of acute indigestion, and the griping pains may have developed at long intervals, with gastric and intestinal flatulence. Vomiting may precede or attend the attack, and diarrhœa may follow. If the colic is due to *gas* alone, there is much tympanites; if it is due to *feces*, there is an antecedent history of constipation, and fecal masses may be detected in the rectum or in the course of the colon. The presence of *fever* is against intestinal colic, and points to inflammation in some portion of the abdomen; moreover, in inflammation the pain is constant, but localized and aggravated by pressure; the skin is hot and dry.

Diagnosis. The sudden severe pain, often relieved by the discharge of gas, with gastro-intestinal disorder, tympanites, the occurrence of cramps in the extremities, and the localization of pain to the umbilicus, all point to the intestinal origin of the affection. A history of exposure or of indiscretion in diet aids in the diagnosis. In colic the pain may come on suddenly or increase gradually from a mere sense of discomfort or soreness.

Intestinal colic must be distinguished from *enteralgia*. The latter comes on slowly and lasts for hours or days. The pain is situated around the umbilicus, and is relieved by deep pressure, although the skin may be hyperæsthetic. Sometimes the abdomen is retracted; there are no signs of indigestion, and flatulence and borborygmi are absent.

Fig. 7.



Situation of pain in abdominal and pelvic diseases.

Lead Colic. If the enteralgia is due to lead, there is a history of exposure to that metal. The blue line on the gums, with obstinate constipation but no vomiting, and the occurrence of neuritis due to saturnine poisoning, point to the nature of the case. Grawitz considers basophile granulations in the red blood-corpuscles valuable early evidence of lead-poisoning.

Hepatic Colic. In hepatic colic the pain is situated in the region of the gall-bladder, and may radiate to the right shoulder or to the back. It is sometimes fixed in the right parasternal line at about the level of the cartilages of the sixth and seventh ribs. The attack is attended by vomiting, usually of bilious fluid; and may be followed by jaundice. A history of cholelithiasis can usually be elicited. There is local tenderness, and there may be some swelling of the gall-bladder. Gallstones may be found in the stools.

Renal Colic. In renal colic pain begins in the kidney and thence extends along the ureter. It is always localized more to the right or left of the median line in the abdomen. It is more frequently in the lower portion of either of the upper quadrants, three inches to either

side of the median line, depending upon the kidney affected. From this region the point of maximum intensity and of local tenderness moves in the lower quadrant in an oblique direction toward the median line, rarely an inch below the transverse umbilical line. The pain then extends to the region above the pubis and down the thighs. From the first there is increased frequency of micturition. The urine is scanty, high-colored, and may contain blood. Free micturition is followed by relief.

Uterine Colic. In uterine colic the pain is situated in the pelvis. There is some abnormal discharge, and the patient gives a history of uterine disease. Care must be taken not to confound the sudden pain of extra-uterine pregnancy with intestinal colic or other forms of abdominal pain. In *extra-uterine pregnancy* the pain is in the lower quadrants of the abdomen to the right or left of the median line. It is sudden and intense, attended by more or less collapse, or even by all the symptoms of internal hemorrhage. It may cause vomiting. The history of cessation of menses, or other signs of pregnancy, of discharge of decidua, with the local signs on physical examination, indicate the true nature of the pain.

Pancreatic Pain. In *disease of the pancreas,* either from the passage of calculi (extremely rare) or because of pancreatic hemorrhage, there may be sudden severe pain. The pain is localized to the region below the sternum. It may be severe in the back and extend up the thorax. It occurs in paroxysms, and is attended by great anxiety, some dyspnœa, and collapse.

Gastric Pain. Intestinal colic must be differentiated from the pain of gastric ulcer, gastric cancer, and gastralgia. The characteristics of pain in these affections have been discussed. When *perforation* occurs in gastric ulcer, the pain is usually seated in the epigastrium, but may be complained of in the back as high as the midscapular region. It is sudden and severe, preceded by a history of ulcer and attended by collapse. There are no evidences of indigestion.

Intestinal colic must be distinguished from that due to *perforation of the biliary passages,* which is attended by pain in the hepatic region. The pain is sudden, and is usually preceded by symptoms due to obstruction of the biliary passages by gallstones.

Appendicitis. Intestinal colic must not be confounded, although it frequently has been, with the pains that attend appendicitis. This is particularly the case with relapsing appendicitis. In this form only mild fever attends the attack. The patient is seized with severe pain, which may be described as occurring in the lower right quadrant, but is sometimes complained of about the umbilicus. It frequently follows indiscretion in diet and may be attended by vomiting; it is usually relieved by eructation, but not by the passage of gas, a point of great importance in the diagnosis. The attack occurs mostly in young subjects, and lasts from twelve to twenty-four hours. It may be so severe as to cause collapse. If fever attends it, and there is a mass present, the diagnosis is much easier. In the relapsing as well as in the initial form there is tenderness at McBurney's point. (See Appendicitis.)

Peritonitis. Intestinal colic must not be confounded with peritonitis, which may follow in any of the above conditions or develop at other points in the abdomen. The purulent peritonitis that succeeds gonococcus

pyosalpinx may be attended by severe pain without much constitutional reaction. The pain, however, although complained of about the umbilicus, can be localized by pressure in the lower quadrant and in the pelvis. It may disappear after eight or ten hours, to be followed by a recurrence. The recurrence of pain is usually attended by *fever*. During the first twenty-four hours the bowels are loose, or at least readily moved. If the peritonitis continues beyond this period, it is often impossible to move the bowels.

Intestinal Obstruction. Intestinal colic must not be confounded with organic disease of the bowels with resulting obstruction. In these affections there are sudden constipation and rapid prostration. The vomiting, if present, persists and soon becomes stercoraceous. In *intussusception* the stools are characteristic. Strangulation, or ileus, is associated with a history of previous peritonitis or the presence of hernia; in the latter there may be characteristic signs at the hernial points. The pain may be transverse and with a sense of constriction if the omentum is incarcerated or adherent and drags on the mesentery. In obstruction from external pressure the presence of tumors has been known previously or can be recognized. In fecal obstruction or in obstruction by gallstones the local signs may be pronounced, and the pain is usually in the ileocæcal region. The affection is acute. Pain extending over a long period, and not due to an acute process or attended by severe acute symptoms, has been considered elsewhere. (See Abdomen.)

Rheumatism and Neuralgia. Intestinal colic may be mistaken for *rheumatism* of the abdominal walls. In the latter there may be a history of exposure. The muscles are extremely tender. There are no gastro-intestinal symptoms, the urine is loaded with uric acid and urates, and there may be muscular pain in other situations, or a pronounced history of previous attacks of rheumatism. In lumbo-abdominal neuralgia the pain may simulate intestinal colic. Pressure-points are detected where the respective nerves have their exits through the fascia.

Pain in Vertebral Disease. Just here may be considered the pain about the navel which occurs paroxysmally in disease of the vertebræ. There may be caries from tubercular disease or from pressure of an aneurism or a malignant growth. Examination of the vertebræ may determine its nature.

Girdle-pain. This is a peculiar pain or sensation in the trunk, due to disease of the spinal cord. It is described as the sensation of a band drawn tightly around the body. It varies from a simple drawing sensation to extreme pain encircling the trunk. It is situated above the level of the umbilicus. In mild forms it is due to chronic myelitis or spinal sclerosis; in severe forms to inflammation of the nerve-roots, or to cancerous, syphilitic, or tubercular disease of the meninges.

Pain in the Spine.

Pain in the spine is due less frequently to organic disease of the cord than to acute or chronic inflammation of the meninges, to disease of the bones of the vertebral column, or to curvature of various forms from

muscle weakness. Rhachialgia and tenderness in the course of the spine occur after concussion.

I. Disease of the Spinal Cord. In organic disease of the cord pain may be referred to the loins, the sacrum, or to the parts about the spine, but not to the spinal column itself. We may have also the eccentric or radiating pains, of which mention has previously been made, due to irritation of posterior nerve-roots. These pains may be dull, resembling those of rheumatism. In acute cases the pains are accompanied by febrile symptoms which may suggest rheumatism, especially when the other spinal symptoms are in abeyance. In chronic cases these peripheral spinal pains are influenced by the weather, and this likewise makes it difficult to distinguish them from the pains of rheumatism. Rheumatic pains in the limbs occurring after middle life, with or without joint-changes, should suggest locomotor ataxia. In this affection sharp and darting pains, "pain crises," and girdle-sensations occur.

II. Disease of Vertebræ. Fixed localized pain at some point in the vertebræ points to traumatic, syphilitic, or tubercular caries, to rheumatoid arthritis, or to pressure-necrosis as by an aneurism. Pain due to vertebral disease is both local and radiating. It is increased by pressure directly on the spinal column (on the head), by heat or by cold, or by electricity, applied over the part. It is relieved by removing the pressure of the weight above, as by raising the head or shoulders. It is relieved by the absolutely recumbent posture. With this pain the movements (flexibility) of the spine are interfered with because of spasm of the muscles or anchylosis; there may be deformity. When the patient is placed upon a flat surface, the normal lumbar arch is changed. (Plate I.)

III. Disease of Meninges. Pain due to meningeal disease is local and radiating. It is associated with muscular spasm and rigidity of the spinal column.

IV. Spinal Curvature. The pain of curvature from muscular weakness extends along the nerves. The patient is afebrile. The signs of organic disease above mentioned are absent, but muscle-weakness and general signs of debility are present. Pain in the spine frequently attends *scurvy* and *rhachitis*. It may be accompanied by paresis of the muscles and closely simulates an organic brain or cord disease.

Pain in the Side.

Pain in the left side—the so-called inframammary pain—is one of the most frequent complaints heard by the practitioner. By discussion of it we can show how the symptom pain, wherever situated, must be investigated in order to determine the tissue affected and the nature of the disease. The tests used in the study of nerve affections are given in the corresponding chapter. Pain in the side may be due to many causes, to exclude any one of which inquiry as to the mode of onset, duration, and character of the pain must be made. Then the structures underneath and about the seat of pain must be examined. 1. The *skin:* to exclude any swelling or tumor or herpes zoster, and to determine the tender nerve-points. 2. The *muscle:* to exclude myalgia or pleuro-

dynia. Examine for tenderness; note the effect of movement. Does full breathing increase the pain? Palpate with the fingers and with the whole hand. Negative results exclude any muscular affection. 3. The *nerves:* (*a*) tender points; (*b*) herpes; (*c*) the vasomotor appearance. The presence of anæmia, other neuroses and neurasthenic phenomena, or toxic conditions, as malaria, lead, or gout, lend color to the view that the pain is neuralgic. 4. The *pleura.* Auscultate for friction if there is pleuritis. Inquire for cough. Note the character and effect of breathing. 5. The *pericardium.* Note friction of pericarditis or thrill by palpation. Is the heart disturbed in function? 6. The *heart.* It is rare that disease of this organ causes pain, although it may be present in dilatation. Is it affected in a reflex manner, causing palpitation or irregularity? Look for distant disease. Angina or pseudo-angina pectoris may be present. 7. The *stomach* and *colon.* A dilated stomach or loaded colon may cause pain by pressure upward. Gastralgia may also be the cause.[1] 8. The *spine.* Determine if it is diseased, or if there is pressure by an aneurism or a mediastinal growth. If a local cause is not ascertained, look for a central or reflex origin. (Plate II.)

Although any one of the above conditions may cause pain in the side, it is usually (1) a reflex pain from gastric disorder; (2) pain from neuritis; (3) a true neuralgia from anæmia; (4) a neuralgia from heart-fatigue. (Hilton.)

It is to be observed that every local tissue must be examined, and questions asked as to the various attributes of the pain.

Pain in the Loins.

Acute Pain. When acute, *without fever*, pain in the loins may be due to lumbago, to a sudden uterine retroversion, to a suddenly moved kidney, or to calculus of the kidney; *with fever*, acute Bright's disease, smallpox, muscular rheumatism, tonsillitis, influenza, dengue, or spinal meningitis must be looked for.

Chronic Pain in the Back—Backache. Backache may be due to many causes. When the pain is in the region of the kidneys, the latter may be at fault.

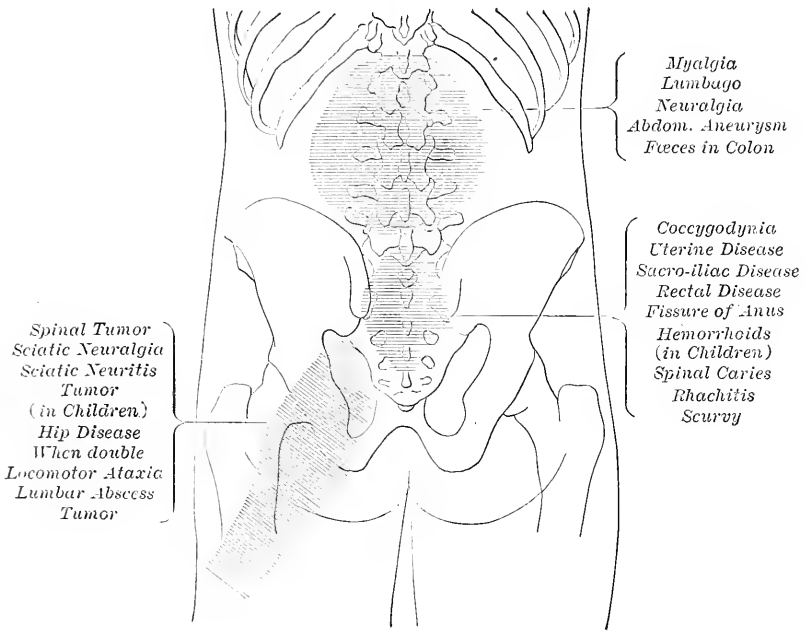
Organic disease may be associated with backache (see next paragraph); more frequently pain, if in one kidney, is due to a calculus or to accumulation of uric acid gravel. Pressure over the kidney or a sudden jar from a false step will usually excite the pain. It may be constant in

[1] Shoulder-tip pain, due to anastomosis of the phrenic nerve with the third and fourth cervical and to parts of the liver and round ligament (Hilton); or of the phrenic nerve and subclavius (Rolleston); or of the vagus with the spinal accessory, which communicates with the third and fourth cervical. The vagus and spinal accessory are sensitive to pressure. (Embleton.)

Inframammary pain (sixth, seventh, and eighth intercostal spaces). The aorta at the left side, third dorsal vertebra, is in relation with the fourth, fifth, and sixth intercostal nerves through the sympathetic ganglia, through which also the heart sympathetics are in anastomosis. The fourth, fifth, and sixth intercostal nerves supply cutaneous branches to the sixth, seventh, and eighth intercostal spaces. Inframammary pain is a reflex neuralgia expressive of some heart-distress. The latter is brought about by exhaustion of the medullary and vasomotor centres, from worry or over-work, or from long-continued irritation of the uterine nerves. This pain is most common in leucorrhœa. (Jacobson: Hilton on "Rest and Pain.") (See Fig. 5.)

movable kidney. When low down, just above or over the sacrum, it is due to disturbance of the pelvic viscera. The uterus, the colon, and rectum (impaction, cancer) must be examined. *Sacral* pain is also due to disease of the sacro-iliac joints, as from gonorrhœal rheumatism, when the characteristic attitude and gait are present.

Fig. 8.



Pain in the back.

Otherwise we may have—(*a*) Pain due to affections of the *muscles*. 1. *Myalgia of rheumatic origin*. Increased by movement, by dampness, by pressure; often relieved by warmth, by the recumbent posture, or by rest. It is associated with symptoms of lithæmia and with the passage of red sand in the urine. When the fasciæ or the ligaments of the vertebræ are affected, the upright position and pressure in small areas increase the pain; other muscles may be affected alternately. 2. *Myalgia from sprain*. A history of injury is obtained. Usually one side is larger than the other. Tenderness is present and movement increases the pain. There may be increased swelling, vasomotor disturbance, or ecchymoses. A neurosis of the so-called spinal or traumatic type (hysteria) attends the pain. 3. *Myalgia from fatigue*. Not only acute fatigue after exertion, but also chronic muscle-tire (and nerve-tire). The pain is increased on exertion, after mental, physical, or *emotional* effort. *Neurasthenia*, *anæmia*, or local exhaustive disease (uterine, gastro-intestinal, etc.) is present. The muscles are usually flabby, and the vertebral column is not supported. The patient lounges or supports the back. Spinal curvatures are observed.

(*b*) Pain due to affections of the *nerves*. Nerve-pain is recognized by the tender points and by vasomotor phenomena.

(*c*) Pain due to disease of the spine, the membranes, or the cord. (See above.)

Pain in the Region of the Liver.

Pain is a frequent symptom of liver disease. When sudden in onset, acute, and increased by pressure or movement, it is due to *perihepatitis*. Acute paroxysmal pain below the ribs in the seventh or eighth interspace or in the epigastrium points to *gallstones*. Pain with distention occurs in *congestion*. Stabbing or darting pains belong to *cancer*. Abscess may be attended by pain from associated perihepatitis.

Pain in the liver must not be confounded with that of pleurisy. In pneumonia there is often congestion of the liver and perhaps perihepatitis. The associated pain has been mistaken for the pain of hepatic colic.

Pain in Diseases of the Kidney.

Pain of the kidneys is referred to the loins. *Bilateral* pain is complained of as a dull aching pain, sometimes increased by movement, often attended by a sense of weight or pressure. Pain of this character extends over the entire lumbar region, and is due to disease of both kidneys, as renal hyperæmia, nephritis, pyelitis, tumors, and malignant disease. Indeed, we have seen renal pain and hæmaturia in a case of commencing appendicitis. It is not generally mistaken for pain due to other causes, as myalgia or disease of the vertebræ. If myalgic, it may follow exposure to cold and be associated with pain in other muscles.

We have also unilateral renal pain. The pain may be seated in the region of the kidney behind, opposite the two lower dorsal and two upper lumbar vertebral spines, or deep in the abdomen, to the right or left of the spinal column, below the level of the umbilicus.

Unilateral pain may be constant or paroxysmal. *Constant* pain is usually due to organic disease of the kidney, as *carcinoma* or *tuberculosis*. (See Palpation.) It may, however, be due to the impaction of a *calculus* in the pelvis of the kidney. It may also be due to a displaced or *movable kidney*. In *tumors* the pain may follow the course of the sciatic nerve and simulate sciatica. In *pyelitis* and *hydronephrosis* the pain is of a tearing character, whereas in movable kidney it is variable.

Paroxysmal and *lancinating* pain, the paroxysms occurring at intervals, is usually due to *renal calculus*, to obstruction or twist of the ureter, as in Dietl's crisis, or rarely to blood in the pelvis of the kidney. Neuralgia of the kidneys no doubt occurs. It may be due to malaria, lead-poisoning, gout, or anæmia. It partakes of the character of neuralgia elsewhere. It must not be forgotten that sometimes in case of disease of one kidney the pain is *referred* to the normal kidney.

Pains in the Legs and Feet.

Pains due to Affections of the Long Nerve-trunks. **Paroxysmal Pain.** Pain in one leg may be due (1) to sciatic neuralgia or (2) to neuritis. The former does not exhibit localized tenderness and is not aggravated by movement. The latter, also called *sciatica*, is recognized by tenderness in the course of the sciatic nerve or at its exit from the pelvis, and by increase in the pain when the limb is extended by forced movement. The pain is constant, worse at night, and characterized by agonizing paroxysms. It follows exposure to cold or may be caused by rheumatism. One of the many branches of the sciatic may be affected, exhibiting tenderness in its course. If the sciatica persists, wasting of the muscles, herpetic eruptions, and areas of anæsthesia over the affected leg may be found. Such neuritis is usually traumatic (cold), alcoholic, rheumatic, gouty, or syphilitic; the exact cause in each case must be ascertained by the associate phenomena and by excluding other causes. Pain in one leg may also be due to (3) pressure on the sciatic nerve by a pelvic growth, (4) neuroma, (5) rheumatism, (6) syphilis of bone or a syphilitic gumma of muscle or connective tissue, (7) to a spinal cord tumor, (8) to inflammation of the veins.

Cramps in the calves may be due to excessive exercise, to deep varicose veins, or to the toxæmia of chronic nephritis.

Fixed pain in the leg, in counterdistinction to the mobile pains of neuritis, is usually situated in the *fasciæ* or *muscles* or in the *bones*. It may be due to *rheumatism*, when the pain is diffused and the nerve-points of tenderness are wanting. It may be the result of *strain* or *injury*, a history of which must be carefully inquired for. In a person of rheumatic diathesis the latter may be the only exciting cause, the fixed pain at the situation of the injury being due to rheumatism. Fixed traumatic pains usually are accompanied by tenderness on pressure, and aggravated by movement, both active and passive, the tenderness on pressure not necessarily being in the nerve-trunk. In malignant disease of the long bones, mobile neuralgia-like pains may precede for some time the fixed pain of the permanent process.

Bilateral pains in the extremities, when not rheumatic or due to trichinosis, are often of central origin, and may be due to spinal sclerosis or spinal tumor; to malignant disease of the vertebræ pressing on the cord; or to pelvic growth or lumbar abscess, causing pressure on both nerve-trunks in the pelvis.

Pains in the Feet not due to Affections of the Large Nerve-trunks.

1. Pain in the Articulations due to Flat-foot. The pain may be in the tarsus or at the metatarsal articulations. Deformity is a common cause of pain in the extremities, and may be unilateral or bilateral. Flat-foot from breaking of the arch can readily be recognized; pressure on the sole of the foot may increase the pain.

2. Pain in the Heel. This is often of gouty origin. Rarely, it is due to ovarian disease.

3. Pain in the Interosseous Spaces between the Distal Ends of the Third and Fourth Metatarsal Bones (Morton's painful affection of the foot). It

occurs in people who are on their feet a great deal, is relieved by a night's rest, increases as the day goes on, and is increased by pressure or by wearing a tight shoe. It is worse in wet and cold weather. Localized pressure at the point on the sole indicated above causes extreme pain.

4. **Pain in the sole of the foot** is often due to disease of the prostate.

We can not leave the extremities without a word regarding pains in the extremities of distinctly central origin—the *forerunners of hemorrhage into the brain.* Mitchell has called attention to these pains. They occur suddenly without evidence of local disease; they are located in one of the extremities, usually the leg, are excruciating, and are not influenced by position, local applications, or pressure. In a patient with hard arteries and high pulse-tension they should be looked upon with suspicion.

CHAPTER IX.

DYSPNŒA.

Dyspnœa means difficult breathing, "shortness of breath." The respirations may be deeper than normal, more frequent than normal, or they may be both deeper and more frequent. The patient is usually conscious of suffering or of some distress in breathing.

While a common and almost constant symptom of lung disease, it may be absent in a patient with extensive disease of the lung. Many patients with chronic fibroid phthisis or emphysema, even when the disease is extensive, may not have dyspnœa unless an unusual demand is made upon the system. These subjects usually are under-weight, move slowly, and otherwise show that they are deprived of one of the essentials to active life.

Dyspnœa is recognized by the increase in the rapidity of the chest movements, with increased action of both the essential and the auxiliary muscles. The expression, as in orthopnœa, is characteristic. The alæ nasi move; the eyes and countenance are indicative of more or less distress; the pupils are dilated. As the dyspnœa continues cyanosis develops. Frequently the patient breaks out into a cold sweat, either general or limited to the forehead, the face, and the extremities. The hands and feet become cold. In complete obstruction stupor sets in; carpopedal spasm or general convulsions follow; the respirations become slower; and death takes place in coma or from heart-failure (asystole).

While dyspnœa is usually easy of recognition, it may be simulated by attacks of acute indigestion with thoracic symptoms of oppression. This latter phenomenon, however, is temporary and not associated with increased rapidity of respiration.

Ætiological Varieties of Dyspnœa.

I. Lessened Amount of Air-supply. A. Obstruction of the air-passages. B. Diminution of air-space from (1) consolidation; (2) compression; (3) impaired expansion.

A. Obstruction of the Air-passages.

1. From Occlusion of the nares, unless compensated by mouth-breathing.

2. From Faucial Obstruction. Dyspnœa is seen in tumors, in inflammation of the tonsils, in the rare form of erysipelas of the pharynx, and in retropharyngeal abscess. It occurs from occlusion of the passages, and is more marked in retropharyngeal abscess and erysipelas than in other conditions. In certain forms of abscess of the tonsils it may be extreme.

3. From Laryngeal Obstruction. Dyspnœa is a frequent, as well as the most serious symptom of laryngeal disease. It may be due (1) to obstruction by inflammatory or œdematous swelling; (2) to spasm; (3) to tumors or foreign bodies in the larynx; (4) to the cicatrization of ulcers after syphilis or lupus; (5) to paralysis of the abductors or adductors of the larynx. It may be, therefore, organic or spasmodic.

Dyspnœa may vary in degree from slight inconvenience in breathing, noticed by the patient, to the violent struggling for breath that is seen in cases of extreme stenosis of the larynx. If carefully observed in either case, the larynx is seen to rise and fall. In extreme forms of obstruction the head is bent back, the neck stretched, the muscles of the neck contracted. The spaces above the sternum and at the sides of the trachea are retracted during inspiration, and the alæ of the nose work vigorously. Further evidence that sufficient air does not enter the lungs is found in recession of the epigastrium and drawing in of the ribs at the base of the chest during the act of inspiration. The countenance is dusky or ashy-gray, the lips become cyanosed, and the nails bluish as the dyspnœa persists and increases; a cold perspiration breaks out on the forehead; and finally, from exhaustion, the respiration becomes slower and slower until it is reduced to mere gasps. The heart's action increases in frequency as the stenosis increases. Death usually takes place from asphyxia, the child first falling into a stupor on account of carbon dioxide poisoning.

Certain characteristic sounds attend the act of inspiration, depending on the nature of the obstruction. In obstruction from simple spasm, or from intense inflammation of the larynx, without secretion, the sound of the inspiration is *harsh* and *stridulous*. In obstruction from œdema or from exudation, as in laryngeal diphtheria, the sound of the inspiration is *loud* and *stridulous*, but not shrill. The expiration is usually noiseless and prolonged. The short, stridulous, or gasping inspiration is followed by prolonged gentle expiration. In spasmodic croup the expiration resembles snoring. The interval between expiration and inspiration is lessened; the respirations are hurried.

In some forms of laryngeal obstruction the *exit* of air is interfered with, as in a movable tumor below the vocal cords. We have expiratory dyspnœa. The act of inspiration is complete, the act of expiration is suddenly checked by the obstruction on account of which the lungs become overfilled with air and emphysema develops.

Duration. Dyspnœa from disease of the larynx may develop gradually and continue over a long period of time, or it may be acute in onset, depending upon the character of the morbid process that has brought about the obstruction. *Acute* paroxysms of dyspnœa, one of which may end in death, sometimes occur in the course of affections in which *chronic* dyspnœa is present; thus sudden œdema may occur in cases of syphilitic or tuberculous ulceration.

Diagnosis. Laryngeal dyspnœa must be distinguished from other forms of dyspnœa: 1. Dyspnœa from diseases of the heart and lungs. 2. Dyspnœa from pressure upon the trachea. The larynx is not markedly moved during the respiratory acts, and the patient bends the head

forward instead of backward. 3. Dyspnœa from pressure on the larynx. Cellulitis of the neck, tumors of the lymph-glands, goitre, and retro-pharyngeal abscess are provocative of this form of laryngeal dyspnœa. Examination of the respective localities by inspection and by touch reveals the cause. It may be worthy of remark that dyspnœa in diphtheria, frequently thought to be due to internal occlusion, may be due to pressure of enlarged glands on the bronchus and larynx.

4. From Tracheal Obstruction. In this form of dyspnœa there is no increased movement of the larynx and no change in the voice. The voice may be weakened, however, and the sonorous quality diminished. It will be modified, if disease of the larynx coexist, or paralysis of its muscles from pressure on the recurrent laryngeal nerves be the cause of the tracheal stenosis. In the latter instance laryngoscopic examination may show a tumor pressing upon the larynx, or an aneurism bulging into the trachea. The latter must not be confounded with pulsation of the lower end of the trachea, due to transmission of the impulse of the aorta, a phenomenon which occurs in healthy persons.

The dyspnœa is *expiratory* in type, and is never so extreme as that in laryngeal stenosis. The lower ribs therefore are not sucked in during inspiration until late in the disease. A stridor attends the dyspnœa, heard with the stethoscope over the trachea and over every part of the chest. Sometimes over the trachea a point can be isolated at which the sound is heard loudest, which point may indicate the seat of the stenosis. When the lower part of the trachea is obstructed, the sound is often more marked over the larynx than over the sternum. In cases of prolonged obstruction in the lower air-passages Demme has pointed out that the upper portion of the thorax may diminish in size. In the course of constant dyspnœa, paroxysms may occur during which the distress is unusually severe. These paroxysms of dyspnœa may be due to spasm of the vocal cords; but very likely, as Bristowe has shown, they are due to swelling of the mucous membrane, or to mucus that has accumulated at the point of obstruction and can not be dislodged, or to spasm of the muscular tissue of the trachea. The patient may complain of pain or oppression behind the sternum, or possibly only of slight soreness. Cough, with expectoration of mucus, usually attends the dyspnœa. Sometimes the mucus is blood-tinged; and in cases of leaking aneurism small quantities of blood may be expectorated when the condition has existed a considerable time.

If the obstruction is due to a foreign body, the dyspnœa is of the same type, but occurs suddenly.

The *causes* of tracheal obstruction, which are also some of those of bronchial obstruction, are: (*a*) *External pressure:* (1) *tumor of the thyroid gland;* (2) *thoracic aneurism;* (3) *mediastinal tumor* from other causes than aneurism, as cancerous or tubercular disease of the glands, or mediastinal abscess; (4) *cancer of the œsophagus;* and (5) in rare cases, a *dilated auricle.* (*b*) *Diseases of the walls of the trachea.* They cause obstruction by narrowing the calibre. *Syphilis* is the most frequent cause of such obstruction. (*c*) *Foreign body.* The presence of a foreign body within the lumen of the trachea causes obstruction. The foreign body

may remain free for a time, moving up and down as the patient coughs, and, indeed, may be felt against the side of the trachea by a finger placed against the outside of the neck. Later, the foreign body usually becomes fixed in the right bronchus or in one of its main divisions, the opening of the right bronchus being more direct than that of the left. In some instances the body may be dislodged and fall into the opposite bronchus. It rarely falls first into the left.

5. From Bronchial Obstruction. This is not accompanied with increase in laryngeal movement or with change in the voice. When a bronchus is obstructed suddenly, compensatory emphysema occurs rapidly in the opposite lung; when gradually, it develops slowly, the degree depending upon the amount of obstruction. The physical signs over the cut-off lung are absence of the vesicular murmur and vocal fremitus, and impaired movement of the affected side. The percussion-sound at first is normal, although it is influenced less by forced inspiration and expiration; later, it progresses from impaired resonance to dulness. As the case advances, the affected side may fall in and measure less than the opposite side. A snoring or whistling sound may be heard over the root of the lung, between the scapula and the vertebræ. Moist râles may be present.

B. Diminution of Air-space. 1. Consolidations, as from congestions, inflammations, or morbid growths. The degree of dyspnœa depends upon the extent and suddenness of onset of the consolidation.

2. Compression from Intrathoracic Causes.—Any variety of *pleural effusion* causes dyspnœa from compression. Obstruction of the tubes on account of spasm as in asthma, or on account of inflammation as in bronchitis, causes a characteristic form of dyspnœa. The dyspnœa is more marked in bilateral effusions than in unilateral, and its severity depends somewhat upon the rapidity with which the effusion takes place. In cases of sudden effusion of air, as in *pneumothorax*, the dyspnœa is very alarming at first, but is gradually relieved as accommodation takes place.

3. Impaired Expansion. *External Pressure.* Dyspnœa may be caused by anything that presses upon the thorax and interferes with expansion. It is therefore seen in affections below the diaphragm. In enlargements of the various abdominal organs, such as the liver, spleen, kidneys, pancreas (cystic disease), and uterus dyspnœa always occurs. In accumulations of gas (flatulence) or of fluid (ascites) the diaphragm is pressed upward and encroaches on the thoracic capacity. In abdominal tumor, tumor of the ovary, of the omentum, and of the above mentioned organs dyspnœa is a distressing feature.

4. Impaired Action of the Respiratory Muscles. 1. *Weakness or Paralysis of the Muscles. Phrenic dyspnœa* is a peculiar form due to paresis of the phrenic nerve and consequent interference with the action of the diaphragm. It may not be observed so long as the patient is at rest. Slight exertion, however, causes distress and an increase in the frequency of the respirations. After taking a few steps the patient experiences a sense of suffocation, and upon making an ascent he must stop frequently to take breath. Other physiological processes are affected. In

the act of sighing the patient feels as though the abdominal organs were drawn up into the chest. Any straining effort, such as defecation, is rendered difficult. The voice is weak, and there is difficulty in coughing and sneezing, because a full inspiration cannot be taken. This latter fact may render very serious a slight attack of bronchitis.

Physical Signs. Instead of the natural expansion of the ribs and chest, the thoracic movements are reversed. The epigastrium and the hypochondriac regions are drawn in during inspiration and pushed forward during expiration. The abnormality may be detected by palpation with both hands below the cartilages of the ribs. Unilateral paralysis of the diaphragm causes retraction of the corresponding hypochondriac region.

In progressive muscular atrophy from general *lead-poisoning*, and in *multiple neuritis* from other causes, paralysis of the diaphragm may take place. It is said to occur in *hysteria*, and Walshe states that he has seen it after *diphtheria*. In *fatty degeneration* of the diaphragm, and in *inflammation* extending from the peritoneum to the pleura, the same phenomenon has been seen. It may occur in *trichinosis*.

Paralysis of the diaphragm must be distinguished from inaction during the act of inspiration, which is the usual cause of retraction of one or both hypochondriac regions. Paralysis of other muscles always accompanies paralysis of the diaphragm, and the shadow of the diaphragm is not seen (see page 471).

Dyspnœa due to *paralysis of other respiratory muscles* can be recognized by the atrophy of the groups of muscles concerned. Electricity may aid in the diagnosis.

2. *Inhibition of Muscular Action by Pain.* The seat of the pain may be in the pleura, in the muscles, or in the intercostal nerves. Frequently, as in peritonitis, hepatitis, etc., it is below the diaphragm, interfering with the action of that muscle. The dyspnœa that occurs from pain resulting from pleuritis, or from inflammation of the chest-wall, is recognized by the posture taken by the patient in order to relieve the affected side, by local tenderness, and by the physical signs of pleurisy or of pleurodynia.

II. Lessened Amount of Arterial Blood in the Lungs. 1. Tumors pressing on the vessels (rare).

2. Stases in the vessels, as in obstructive heart disease.

Cardiac dyspnœa is clinically divided into :

a. Dyspnœa increased or caused by exertion.

b. Paroxysmal dyspnœa.

c. Orthopnœa.

d. Rhythmical dyspnœa, or Cheyne-Stokes respiration.

The dyspnœa of effort comes on after the slightest exertion or excitement.

In paroxysmal dyspnœa the attack comes on without apparent cause. It must be distinguished from the paroxysmal dyspnœa of uræmia, asthma, and emphysema. Pulmonary disease can usually be recognized by the physical signs. The paroxysmal dyspnœa of heart disease is attended by more violent respiratory efforts than the physical state of the lungs admits,

and the difficulty attends both inspiration and expiration. Wheezing is not so marked as in certain forms of asthma. There is some obstruction to the outgoing of air; but, on account of the air-hunger, all the efforts of the patient are exerted to fill the chest. In paroxysmal dyspnœa the breathing usually becomes quiet when the patient is placed in a comfortable position, provided there is no pulmonary or pleural complication. Posture does not modify the severe dyspnœa of asthma or emphysema.

Orthopnœa and Cheyne-Stokes breathing are described on page 105.

3. Thromboses. In cases of cardiac weakness the vessels may become occluded (hemorrhagic infarct). After labor a clot of blood may escape from a uterine sinus, be carried to the right heart, and thence to the pulmonary veins. A clot may arise from inflammation of veins situated in any part of the body.

4. Embolism. Foreign substances, such as fat, may be present in the blood. They may occur in parturient women three or four days after labor. Fat-embolism has developed after fracture and in cases of diabetes.

5. Impaired quality or deficient quantity of blood, as in forms of anæmia.

III. Interference with the Nervous Mechanism of Respiration. The dyspnœa may be of central or of peripheral origin, and may be due to various causes.

1. Tumor, Hemorrhage, or Degeneration about the Respiratory Centre in the Medulla.

2. Irritation of the Centre by Toxic Agents. This may occur in *uræmia*, in *diabetes*, and in *auto-intoxication* from gastro-intestinal disorder. The disease at present known as *asthma* may be of central origin, due to irritation of the pneumogastric centre. The dyspnœa of *hysteria* also belongs to this class. To this class belongs "heat dyspnœa," which occurs in all febrile conditions, the warm blood acting as a direct irritant to the respiratory centre in the medulla oblongata. (Landois.) This explains the curious fact, pointed out by Cohnheim, that the respirations in pneumonia lessen as soon as the fever disappears, notwithstanding the persistence of the physical condition.

The nature of each variety is recognized more particularly by the associate symptoms. The dyspnœa due to the poison of uræmia usually occurs in paroxysms, but may be constant. It sometimes is the first intimation of the presence of renal disease. The presence of albumin and casts in the urine, the odor of the breath and of the exhalations, and the presence of hypertrophy of the heart and of an accentuated second aortic sound point to a uræmic origin. The dyspnœa of diabetic coma, known as "air-hunger," is characterized by slow and deep respirations. The history and symptoms of diabetes, the odor of acetone on the breath, the presence of sugar in the urine, and the absence of organic pulmonary disease point to diabetes. The dyspnœa of uræmia often cannot be distinguished from dyspnœa due to other causes, unless cardiac and pulmonary disease can be excluded. This is often difficult, as uræmia frequently develops after an hypertrophied heart has failed, the physical signs of dilatation then being sufficient to explain the dyspnœa. Irritation in the medulla may also

give rise to Cheyne-Stokes respiration, which may be the type of dyspnœa occurring in uræmia.

3. **Reflex Irritation of the Terminal Endings of the Pneumogastric Nerve, or of Nerves Intimately Associated in the Medulla with the Pneumogastric.** This is the usual cause of the paroxysmal dyspnœa of asthma. It may be due to a number of conditions: (*a*) Disease in the upper air-passages, as polyps, hypertrophy of the turbinates, and adenoid growths which are the most frequent source of paroxysmal dyspnœa. Temporary irritants applied to the nares, such as various animal and vegetable odors, micro-organisms or pollen in the inspired air, and attacks of nasal congestion may also produce paroxysmal dyspnœa. The irritation is propagated through the ethmoidal and posterior nasal branches of the pneumogastric nerve, the Vidian and the nasopalatine nerves, to the septum, and through the anterior palatine nerve to the middle and lower turbinates. (*b*) Irritation in the fauces and larynx. This is not so likely to cause dyspnœa; yet there is no doubt that the presence of a constant irritant in these situations tends to keep the respiratory tract in a state of excitability, so that asthma is more likely to persist. (*c*) Irritation of the terminal branches in the stomach of the pneumogastric nerve. Peptic asthma, or the asthma of indigestion, may be due to this cause.

Clinical Varieties of Dyspnœa.

The clinical varieties of dyspnœa are determined (1) by the influence of exertion; (2) the respiratory rate; (3) the respiratory rhythm; and (4) the constant or paroxysmal nature of the symptom.

1. **Dyspnœa Influenced by Exertion.** *a.* **Shortness of Breath on Exertion Only.** This is seen in cases of simple debility, and of interference with respiratory action on account of obesity. It is the form met with in anæmia and in moderate cardiac debility. It may not be observed by the patient unless he walks hurriedly or ascends a flight of stairs.

b. **Shortness of Breath Independent of Exertion.** This is of more serious import, and may be due to a number of causes. It is that seen in severe cardiac and pulmonary disease. The latter includes asthma and emphysema, bronchial obstruction, and pulmonary consolidation and compression (by effusions).

2. **The Respiratory Rate.** Dyspnœa varies clinically according to the frequency of the respiration. In its most extreme form it is known as *orthopnœa*, on account of the upright posture assumed by the sufferer. (See Posture.)

a. **Respirations Slow or Normal.** In dyspnœa the inspirations may be deep, and the frequency of respiration less than normal. This is one of the forms of dyspnœa seen in diabetic coma, "breathlessness without dyspnœa." The breathing may be slow and stertorous. Such breathing is likewise associated with coma; but the coma is of central origin, being due chiefly to apoplexy or tumor. The causes of such respiratory rhythm are usually either central or toxic, or both.

Toward the end of life the respirations, even if hurried before, become slower through carbon dioxide intoxication.

b. **Respirations Increased.** The respirations may be hurried, and may create distress in *simple nervousness* and in *hysteria*. In the latter affection the rapid breathing is often attended by distress. The respirations are quickened, and may equal half the pulse-rate or may even be as frequent as the pulse. The term "panting" is applied to such respiration. It is seen in exophthalmic goitre. In all forms of dyspnœa on exertion (see above), and in all forms due to heart or lung disease, the respiratory rate is increased.

3. The Respiratory Rhythm. Slow and shallow alternating with quick and deep breathing is seen in the peculiar form of breathing known as Cheyne-Stokes respiration. A period of apnœa intervenes, accompanied by simultaneous alteration in the size of the pupils. (See Uræmia and Diseases of the Brain.)

4. Constant and Paroxysmal Dyspnœa. Constant Dyspnœa. This implies a persistence of the cause. It is frequently subject to paroxysmal aggravations.

Paroxysmal Dyspnœa. Paroxysmal dyspnœa is seen in its most typical form in reflex and toxic asthma. It is often of cardiac origin, but may be due to central or reflex causes. (See Asthma.)

5. Inspiratory and Expiratory Dyspnœa.—Dyspnœa is divided into inspiratory and expiratory dyspnœa, according as the ingoing or outgoing of air is interfered with.

Emphysema is possibly due to lessened power of all the respiratory muscles and to bronchial obstruction due to inability to empty the chest of air (expiratory dyspnœa). Inspiration is short and quick, the scaleni being strongly contracted and the serratus magnus, the latissimus dorsi, and the pectorales all aiding in elevating the ribs. Expiration is prolonged and requires the aid of all the auxiliary muscles for its completion. The powerful abdominal muscles are seen to contract vigorously, thus aiding in pressing up the diaphragm; the quadratus lumborum and the serrati postici superior et inferior draw down the ribs. Knowledge of the processes involved in forced expiration renders the diagnosis comparatively easy. The contraction of the broad abdominal muscles confirms the diagnosis.

Laryngismus Stridulus. In this form of dyspnœa the act of breathing ceases in the midst of inspiration, and is attended by a characteristic sound. It is seen usually in poorly nourished children. It is of frequent occurrence in *rhachitis*, its presence suggesting that disease when other manifestations of it are obscure.

The symptoms occur suddenly and are very alarming. The child awakes in the night, and suddenly stops breathing after a few short whistling inspirations; terror is depicted on its countenance; the eyes stare; the face is pallid at first, but rapidly becomes livid. The alæ nasi are distended, the head is thrown back, and the spine arched. A cold perspiration breaks out over the forehead. Carpopedal spasms may occur, and the urine and feces may be discharged involuntarily. In a few seconds, or at most two minutes, the child draws two or more deep, noisy inspirations, each one lessening in depth and volume of sound, when the cyanosis gradually disappears, the color returns

to the face, and the child becomes tranquil. In mild forms the child "catches its breath." It holds its breath, and then makes a noisy inspiration.

Attacks of laryngismus stridulus are more rare in adults. They may occur in *hysterical* subjects. During the attack there occurs a series of long, harsh, whistling or stridulous inspirations, followed by short, noisy expirations. Rarely is there complete closure of the glottis.

In both children and adults general convulsions may occur during the attack, or carpopedal spasms alone may be seen. Among adults the convulsions occur only in hysterical subjects.

The *diagnosis* of laryngismus stridulus is based upon the absence of laryngeal symptoms prior to the attack, the absence of cough or hoarseness, and the complete disappearance of all laryngeal symptoms when the attack subsides. The absence of pain and fever and of laryngoscopic signs is noteworthy. This applies, of course, to spasm occurring independently of laryngeal disease.

CHAPTER X.

COUGH.

COUGHING is a reflex act. A deep inspiration is taken and the glottis closed. Immediately a sudden expiratory effort is made, during which the glottis is opened. A loud sound accompanies the forcible passage of air outward, which carries along with it any substances that may have been in the air-vessels.

Causes of Cough.

Cough may be due to (1) irritation of the respiratory tract, to (2) central and to (3) reflex irritation.

1. Irritation of the Respiratory Tract. This usually begins in the respiratory mucous membrane, the purpose of the cough being to expel accumulations of mucus or pus, or of some foreign substance. It occurs in all forms of bronchitis and in lung affections associated with bronchitis. The cough of phthisis, when not laryngeal, is due to a localized bronchial catarrh. Nodules situated in the lung-substance outside of the bronchi, as the calcareous and fibrous nodules of healed tuberculosis, do not provoke the act of coughing. The irritation is not limited to the bronchial tubes, but may occur in any portion of the respiratory tract. Cough is excited by the presence of a foreign body of any kind in a bronchus. It is notably present in pharyngeal and laryngeal diseases. (See Diseases of Nose and Larynx.) The cough of the latter is of peculiar character, which renders it easily distinguished from cough due to other causes.

The presence of an irritant does not always excite cough. When the sensibilities are obtunded, as in typhoid fever, in disease of the brain, or in the last stages of any disease, the presence of mucus will not excite cough, although the presence of mucus in the trachea may be indicated by rattling in the throat. In phthisis sudden cessation of the cough and expectoration from weakness is a bad prognostic sign and denotes approaching death. It is also a bad sign in pneumonia.

2. Central Irritation. Cough may be of centric origin. Kohts has found by experiment that irritation of the floor of the fourth ventricle, above the centre for respiration, excites cough. This centric origin may possibly explain the cough of hysteria; and the short, barking cough which arises in hysterical or nervous states when the patient is afflicted with the idea that he is about to have hydrophobia. Irritation of nerves in anatomical relation with the pneumogastric also excites cough.

3. Reflex Irritation. There are several varieties of cough due to this cause.

a. **Nasal Cough.** (See Nose.)

b. **Pharyngeal Cough.** This may be due to hypertrophy of adenoid tissue, inflammation, paralysis, or new growths of the pharynx, to an elongated uvula, or to the presence of secretion from the nose.

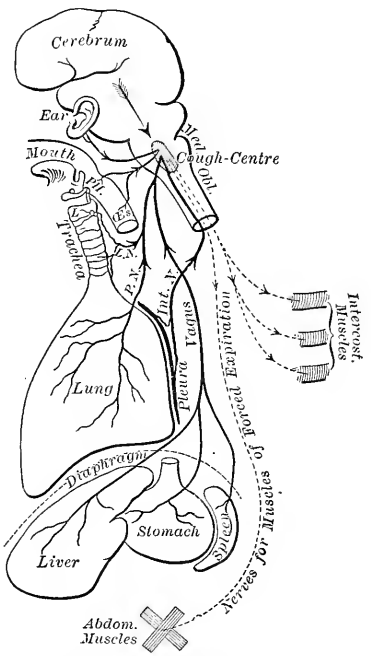
c. **Pleural Cough.** This is not of infrequent occurrence and is due to the inflammation of the pleura.

d. **Ear-cough.** The most characteristic cough of this form is that due to the presence of a foreign body in the meatus of the ear, or to disease of the ear. It sometimes is difficult to examine the external auditory meatus on account of the coughing excited. According to Fox, the afferent nerve that transmits the irritation is the auriculotemporal branch of the fifth nerve, and not the minute auricular twig of the vagus.

e. **Tooth-cough.** Fox called attention to the occurrence of cough from the irritation of the stump of a tooth, and referred to the cough of infants during the first dentition.

f. **Stomach-cough.** The popular opinion that cough is very frequently due to disease of the stomach is not substantiated by the experiments of Kohts. Nevertheless, cough is frequently observed in patients who are suffering from mild gastric catarrh, the treatment of which relieves the cough. This is in all probability due to the fact that with the gastritis there is a secondary pharyngitis which causes the cough, and which disappears entirely when the gastritis is relieved. Irritation of the gastric filaments of the pneumogastric may be a possible cause of this so-called stomach-cough.

FIG. 9.



Schema of the afferent nerves through which coughing may be excited reflexly. The efferent nerves through which efferent impulses are transmitted to the expiratory muscles are dotted. (LANDOIS.)

g. **Liver-cough.** It is said that cough may occur in cases of hepatic enlargement or abscess, hydatids, perihepatitis, cholelithiasis, and subphrenic abscess.

h. **Miscellaneous Reflex Causes.** Cough may accompany *cardiac disease* in consequence of secondary bronchial congestion from hemorrhagic infarct. It may exist with enlargement of the bronchial glands, with mediastinal tumor or abscess, with thoracic aneurism, and with caries of the dorsal vertebræ. It has even been attributed by some authors to irritation from disease of the sexual apparatus.

It will be seen, therefore, that when investigating the cause of a cough in diseases in which it is a prominent symptom, it is necessary not only to examine the respiratory tract throughout its whole extent, but also to examine the condition of the ears and of the teeth, and to bear in mind a possible centric origin.

Clinical Characteristics of Cough.

The cough may be dry or moist; constant or paroxysmal.

Dry Cough. This occurs when the source of irritation is not removable. (See Dry Cough of Laryngeal Disease.) It is seen in the first stage of *bronchitis* and in the earlier stages of *phthisis*. During the first stage of pleurisy the cough is short, hacking, and suppressed; in the second stage it is superficial, as if the sound-waves were checked. In cases of emphysema the cough for a long time may be dry and unproductive, only to be relieved after the discharge of a small pellet of tenacious mucus. Dry cough is characteristic and very familiar, although it is described with difficulty. It is the type met with when the cough is due to irritation outside of the respiratory tract, and is characteristic of the ear-cough, the tooth-cough, the nervous cough (which is nothing but a bad habit), the cough of hysteria, and the cough of a peculiar barking character occurring at puberty and described by Sir Andrew Clark.

Moist Cough. This is attended with the production of sputum of a mucopurulent, purulent, or bloody character, which is removed with comparative ease.

Constant Cough. This usually implies a persistence of the cause, which is invariably pulmonary or bronchial, as pleurisy, phthisis, bronchitis, and consolidations generally.

The cough is almost constant when the irritation is permanent, and when a large amount of secretion is rapidly being poured out. The latter is seen in *bronchorrhœa*, in *bronchial dilatation*, and in the later stages of *tuberculosis*. In these affections the paroxysm of moist cough may occur three or four times in twenty-four hours.

Paroxysmal Cough. The moist cough may occur in paroxysms only, each paroxysm being relieved by the removal of the irritant, and the subsequent paroxysm not taking place until the irritating secretion has reaccumulated. In the second stage of *bronchitis* paroxysms of cough may occur every few hours, or only once in twenty-four hours, usually in the morning on arising. The accumulated secretions of the night are thus disposed of, after which the patient remains free from annoyance. Paroxysmal coughs occur in cases of *bronchorrhœa*, of *bronchial dilatation*, and of *cavities*, either of the lung or of the pleura opening into the lung, being excited whenever the cavity fills with secretion. The paroxysm may occur daily or several times a day, and an enormous amount of sputum is thrown off, until the cavity has been emptied, as accumulation of matter excites coughing only after a certain level has been reached. In these affections the cough is further characterized by the fact that it is aggravated by change of position. The association of retching and vomiting with the paroxysm is of some diagnostic significance, and is seen not only in whooping-cough, but also in phthisis. In *pertussis* the character of the paroxysms is of special diagnostic significance. The expiratory efforts are frequent and rapid, followed by a noisy, prolonged inspiration, accompanied by the characteristic whoop. The appearance of the countenance at the same time is striking. The face is cyanosed, the eyes stare, and the patient's distress is most

evident. The labored efforts at coughing frequently terminate in an attack of retching or vomiting.

Diagnostic Value of Cough.

The diagnostic significance of cough is estimated by the character, whether dry or loose, whether constant or paroxysmal; by the sound, which, however, is usually modified by the condition of the larynx (consult the section on Laryngeal Diseases), and by means of which laryngeal is distinguished from bronchial disease; by its frequency; by its duration; and by its development at particular times or under particular circumstances, as on rising in the morning, or on changing to a cold atmosphere, or in speaking, or upon movement. The diagnostic value of cough further depends on a knowledge of the character of the expectoration. (See Sputum.)

Cough is a constant accompaniment of diseases of the larynx. Several forms are noted:

1. The *dry* cough, as seen in acute laryngitis. It is almost constant, and is aggravated when the patient speaks, takes fluid, or inspires deeply. In children it is abrupt, brassy or metallic, stridulous or whistling, so-called "croup-cough," as seen in cases of "false croup" and laryngitis with œdema.

2. A *dry, hoarse* cough occurs in the course of chronic laryngitis.

3. *Cough with whoop.* With the act of coughing a whooping sound may be heard during an inspiration, succeeding rapid violent expiratory acts. It is spasmodic and convulsive, and is followed by retching, and often by vomiting. (See Pertussis.)

4. The cough is of such a character as to give one the idea that it is *suppressed* in membranous and œdematous laryngitis.

5. A cough frequently occurs without any local anatomical changes in the larynx, which seems to be purely of *nervous* origin. Two forms are seen:

a. Paroxysmal. Severe coughing occurs suddenly, and can not be controlled by the patient. It ceases without cause, returning in a few hours. There is no expectoration.

b. Continued and rhythmical. The cough is not so severe as in the paroxysmal form, but also occurs at more or less regular intervals, and varies somewhat in intensity from time to time. It does not occur during the act of eating or speaking, and ceases entirely during sleep. It is usually worse when the patient is under observation. Examination with the laryngoscope reveals absence of disease. Nervous cough is seen after diphtheria, when sexual disturbances are present, at puberty, and in some cases of anæmia, chlorosis, of neurasthenia and hysteria. The pitch is usually high.

CHAPTER XI.

PALPITATION, ARRHYTHMIA, AND PULSATION.

Palpitation.

In palpitation the patient is conscious of the action of the heart. Although it occurs in organic disease of the heart, it is more frequently due to disease outside of the heart.

Symptoms. The symptoms vary in degree. In mild forms the patients may complain of a fluttering or a sensation of sinking in the præcordial region. In the more severe forms the heart seems to beat violently against the chest. The arteries throb, the action of the heart is increased, and the area of impulse against the chest-wall is visibly enlarged. The patient complains of distress in the præcordial region. The pulse may be increased to 150 per minute. In nervous palpitation the face becomes flushed, and after the attacks large quantities of urine are passed. Sometimes, in this form of palpitation, exertion relieves the attack. The heart-sounds are found to be clear and metallic in character, and no murmurs are present. The diastolic sounds are greatly accentuated. If anæmia is present, murmurs due to that condition are increased in intensity. The attack may last but a few minutes or continue for hours.

a. Palpitation is most common in patients whose nervous system is in a state of increased excitability; it is very common in hysteria and in neurasthenia; it follows emotional disturbance; it is more frequent in women than in men. The attacks occur most frequently about puberty and at the menopause.

b. Palpitation may be due to the action of toxic substances, as tobacco, tea, coffee, or alcohol.

c. From strain and over-exertion, particularly if associated with excitement, palpitation may occur and continue for a long period. This is one of the forms of irritable heart described by Da Costa as common in young soldiers.

d. When compensation fails in valvular disease of the heart and in myocarditis, attacks of palpitation occur which are distinctly due to over-exertion.

e. Palpitation is of frequent occurrence in *indigestion* from flatulence or dilated stomach.

f. Palpitation is an early symptom of anæmia, especially chloro-anæmia.

Arrhythmia.

The alteration in rhythm is usually due to nervous disturbance. In organic disease it is not, as a rule, appreciated by the patient.

Arrhythmia is the general term applied to irregularity of the action of the heart. The heart intermits—that is, one or two beats are dropped at intervals of a few seconds or half a minute, a minute, or longer. The systole may be omitted altogether, or so weak as not to produce a pulsation in the radial. The heart's action is said to be irregular when the beats are unequal in volume or force, or occur at unequal intervals, without periodic intermissions. The causes of disturbance of the rhythm have been classified by Baumgarten [1] as follows:

1. Central causes in the medulla either from organic disease, as hemorrhage or concussion, or from psychical influences.
2. Reflex influences, as in dyspepsia and diseases of the liver, lungs, and kidneys.
3. Toxic influences—tobacco, coffee, and tea are common causes; various drugs, such as digitalis, belladonna, and aconite.
4. Changes in the heart itself. Mural changes, as in dilatation, fatty degeneration, and myocarditis; changes in the cardiac ganglia; sclerosis of the coronary arteries.

It must not be forgotten that both irregularity and intermittency may occur in persons otherwise in good health, and continue for a long time without any evidence of arterial or cardiac disease.

The following varieties of arrhythmia have been described:

1. The Paradoxical Pulse. (Kussmaul.) The beats are more frequent, but less full during inspiration than during expiration. Hurried respirations may induce this in healthy individuals. It is found with weak heart and in chronic pericarditis.

2. The Bigeminal and Trigeminal Pulse. Two or three beats follow each other in rapid succession, a longer interval of normal pulsation separating the groups. In the bigeminal pulse the first beat of the pair is the stronger. These pulsations are seen in mitral disease, and are characteristic of the digitalis pulse.

3. Fœtal Heart Rhythm—Embryocardia. The long pause is shortened and the two sounds are of the same character. It occurs in extreme dilatation and myocarditis, in shock and hemorrhage, and in the late stages of fevers.

4. Delirium Cordis. The heart's action is very irregular.

5. Gallop Rhythm. (See Rhythm, under Physical Diagnosis.)

Pulsation.

Pulsation or *throbbing* is a striking feature of *hysteria* and *neurasthenia*. The abdominal aorta is frequently thus affected. The pulsation may be constant or intermittent. There may be dyspeptic symptoms. The pulsation of the carotids may cause disagreeable sensations in the head, and the beating transmitted to the ear may be the source of extreme annoyance. Pulsation in the neck may be due to exophthalmic goitre.

Pulsation, especially of the carotids, the abdominal aorta, and the brachial arteries occurs in *anæmia*. Pulsation of the abdominal aorta

[1] See Transactions of the Association of American Physicians, vol. iii.

may be reflex from organic disease in the vicinity. Similar localized pulsation in the innominate arteries may be mistaken for aneurism.

The pulsation that attends organic heart disease may be due to hypertrophy of the heart, but is particularly characteristic of *aortic regurgitation*.

The differential diagnosis of the various forms of pulsation and of the conditions which they may simulate are considered in the section on Physical Diagnosis.

CHAPTER XII.

DYSPHAGIA.

Difficult swallowing is due to a number of causes, the chief of which are diseases of the mouth and fauces, disease of the larynx, and disease of the œsophagus.

The affections of the mouth are glossitis, cancer of the tongue, and the various forms of stomatitis.

Inflammation of the tonsils and all forms of *pharyngitis* cause painful swallowing. The various infections, the eruptions of which are located in these regions, as well as diphtheria cause dysphagia. Rheumatism of the pharynx and retropharyngeal abscess are causes that create the greatest difficulty in diagnosis. In the former there is but little ocular evidence of disease; in the latter a tumor may be detected by inspection or palpation.

Difficulty in the act of swallowing in *laryngeal diseases* is most marked when tissue-destruction in the larynx has taken place, or when there is acute inflammation about the muscles or their attachments; hence, when tuberculous or malignant ulcers are present, or perichondritis arises, the difficulty is so great as to prevent the taking of food. There is great dysphagia when the epiglottis is the seat of acute inflammation. Laryngeal dysphagia is recognized by pain and by the falling of particles of food into the larynx, exciting cough. It may be distinguished from the dysphagia of pharyngeal affections by ocular examination, the location of the pain, and the non-association of rheumatism. Mis-swallowing, or "swallowing the wrong way," occurs in all conditions in which food is allowed to enter the larynx. Although conditions favorable for its occurrence are present, it may not take place unless the patient is off his guard during the act of swallowing, as when he is laughing. It may then occur even in normal individuals. It may be associated with anæsthesia of the larynx and occur in central nerve affections which cause that condition.

Dysphagia is a symptom common to all *diseases of the œsophagus.* It may vary from simple painful swallowing to complete obstruction of the tube.

It may be due to paralysis, to spasm, or to obstruction. Dysphagia from obstruction of the œsophagus or stricture is due (1) to disease outside of the canal (external pressure), (2) to disease of the canal itself, and (3) to the presence of a foreign body in the canal. In the consideration of this symptom, therefore, these three conditions must be studied.

1. External Pressure. The œsophagus at different parts of its course is in intimate relationship with the trachea, the thyroid gland, the carotid artery, the left bronchus, the bronchial glands, the arch and the descending

portion of the aorta. Disease of these structures may therefore cause difficulty in swallowing. It is not likely that difficulty of deglutition from disease of the trachea, thyroid gland, or carotid arteries will be overlooked. If the trachea is affected, dyspnœa will be a prominent symptom ; if the thyroid gland, dyspnœa will be associated with dysphagia, and the enlarged gland will be visible from the outside. Disease of the vertebræ is not likely to cause obstruction of the œsophagus, for it would not press that organ against any other solid structure. Disease of other structures may, however, cause difficulty of deglutition by pressing the œsophagus against the vertebræ. Within the thorax disease of the mediastinal glands, aneurism of the arch or descending portion of the aorta, an enlarged left auricle, a pericardial effusion, or disease of the left bronchus might cause constriction of the œsophagus. Enlargement of the *mediastinal glands* occurs in tuberculosis, carcinoma, sarcoma, and syphilitic disease. Physical signs of a mediastinal tumor, with a history of syphilis or the general symptoms of tuberculosis, sarcoma, or carcinoma, would point to the presence of these affections. In *aneurism* of the aorta, in its arch or transverse portion, the physical signs and subjective symptoms of aneurism—with accentuation of the aortic second sound and the presence of atheroma—would lend color to the view that the obstruction was of this nature. In both instances just mentioned the obstruction rarely goes to the extent of preventing the passage of liquids. In enlargement of the *left auricle* and in *pericardial effusion* the degree of difficulty may amount simply to a temporary sense of obstruction or pain about the point where food passes these structures. If the early physical signs are associated with an enlarged auricle, with mitral stenosis, or with pericardial effusion, the diagnosis of the causal condition is easy. It is particularly important, in considering difficulty of deglutition from external pressure, to remember that the œsophagus is in close relation with the bronchus on the left side, at about the fourth dorsal vertebra—ten inches from the teeth—in case it is desirable to investigate the obstruction with a probe. Obstruction from aneurism of the descending portion of the arch of the aorta is also located in the upper portion of the œsophagus, nine inches from the incisor teeth.

2. Organic Disease. Difficulty of deglutition due to disease of the œsophagus itself occurs in acute inflammation, in chronic inflammation, and in stricture, which is always the result of traumatic inflammation, syphilis, or cancer.

3. Foreign Body. Stricture from the presence of foreign bodies is usually recognized with ease. The difficulty of deglutition is due both to the foreign body and to the spasm it excites. In consequence of the latter regurgitation of food takes place. In the first place, there is a history of the swallowing of a foreign body. Sudden pain succeeds the act, while there are great anxiety and distress, particularly if the body is a large hard mass. Not only is there difficulty in deglutition, but also dyspnœa. The latter is due to pressure, but is aggravated by the nervous state. When the foreign body is small, the dysphagia is moderate in degree and the reflex irritation slight, although nausea and vomiting

may be present. If the obstruction cannot be removed, ulceration and abscess result, the further course of which depends upon the seat of the obstructing material. Pain, hemorrhage, subcutaneous emphysema, and the emission of air are the symptoms that follow. The exact location of the foreign body may be ascertained by the use of the Röntgen rays, as in the remarkable case of White's.

Harrison Allen has called attention to several features. Many of the symptoms are primary and some are secondary. The former are due to the trauma and to the presence of the foreign body; the latter to the secondary ulceration. This softening and ulceration of the walls may take place rapidly. Allen does not think that pain or the occurrence of convulsions is of much significance, but that emphysema, the excessive secretion of mucus, and the emission of air are important signs. Anxiety he considers a very common and very suggestive symptom.

CHAPTER XIII.

VOMITING.

During the act of vomiting the stomach is compressed by the abdominal muscles and diaphragm, while the so-called cardiac sphincter of the œsophagus is at the same time relaxed. Sometimes there are nausea and violent efforts at expulsion on the part of the stomach without the occurrence of vomiting, because the cardiac orifice of the stomach is not opened at the same time. Again, there may be profound relaxation of the œsophagus, but no compression of the stomach by the diaphragm and abdominal muscles. Both factors must operate at the same time to produce vomiting, which explains why it is that some persons suffer extreme nausea and even have violent retching, but are unable to vomit.

It is to modern physiologists—Schiff and Budge and Brunton—that we owe a correct explanation of the physiology of vomiting. From them we learn that there is a nervous centre for vomiting, which is seated in the medulla oblongata in close proximity to, and intimately connected with the respiratory centre. It is to this centre that impressions are sent from the brain itself or from various portions of the body through their nerve-supply, and from this centre motor impulses are transmitted to the muscles concerned in the act of vomiting, and to the stomach and œsophagus. In his usual graphic manner Brunton has described the entire mechanism.

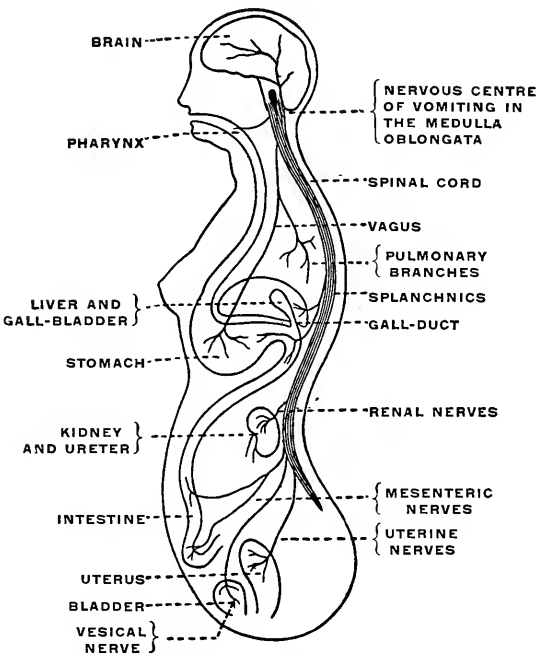
By a very good diagram (see Fig. 10) Brunton indicates the afferent nerves which transmit impulses to the vomiting-centre and excite its action. They are: pharyngeal branches of the glossopharyngeal; pulmonary branches of the vagus; gastric branches of the vagus; gastric branches of the splanchnic; renal, mesenteric, uterine, ovarian, and vesical nerves. Fibres pass downward from the brain, transmitting impressions to the vomiting-centre from the organs of special sense, from the brain-substance or its membranes when they are diseased, or from central ganglia excited by emotion or imagination.

From this it is seen that vomiting is a reflex act; that its mechanism is quite simple; and that a proper understanding of this mechanism is essential to a correct appreciation of its pathology and treatment. Reference has not been made to the vomiting that occurs in the initial stage of many fevers, and in septicæmia, uræmia and allied affections; nor to the vomiting of hysteria. The former is doubtless due to the direct action of the poisoned blood on the vomiting-centre; but it is also quite possibly due to the propagation of impulses to the vomiting-centre from the brain when the latter is irritated by toxins in the blood. If the phenomena of hysteria are due to an abeyance of the processes of inhibition, vomiting

in that disease can be said to arise from the non-control, by higher centres, of the vomiting-centre.[1]

The significance of vomiting in a given case can sometimes be determined very readily; at others it may remain in doubt even after very careful examination and questioning of the patient. In seeking for an explanation of vomiting it is of importance to find out the *previous state of health* of the patient—whether the vomiting occurred after the patient had been ill for a longer or shorter time, or suddenly when he was in apparent health; or whether it formed one of the initial symptoms of an acute disease.

FIG. 10.



The nervous mechanism of vomiting. (After BRUNTON.)

Inquiry should be made as to the supposed *cause* of the vomiting; whether it was excited by the taking of food, drink, or medicine, or by some disgusting sight or odor.

The *time* when the vomiting occurred should be ascertained, as well as its frequency, and whether it was preceded by nausea, pain (noting its locality), injury, cough, jaundice, or constipation.

The *position of the patient* at the time of vomiting sometimes furnishes a valuable clue to its cause.

The *effect* of the vomiting is sometimes of aid in diagnosis. In ulcer and in migraine, for example, it affords marked relief.

Finally, the appearance and quantity of the matter vomited are very important subjects for investigation. (See Objective Signs.)

[1] From "Vomiting, Physiological and Clinical." Trans. Penna. State Med. Soc., 1887. Musser.

Character. Vomiting may occur occasionally, persistently, or periodically; it may be projectile and painless, or difficult and painful. Projectile vomiting is characteristic of cerebral disease or points to a reflex origin; the latter type indicates local gastric disease. *Sudden* vomiting without antecedent illness usually indicates some local affection of the stomach, or is due to some nervous impression, or marks the onset of some acute general disease, especially *pneumonia*, the *eruptive fevers*, and *yellow fever*. Excessive vomiting generally indicates that the case will be severe.

Vomiting in Gastric Disease. The local affections of the stomach attended by vomiting are acute and chronic gastritis—especially the catarrhal form—dyspepsia, ulcer, cancer, and dilatation.

In *acute gastritis* there will be a history of an acute illness marked by severe local and general symptoms. The cause of the gastritis may be found to be overeating highly seasoned or indigestible food; abuse of alcohol, narcotics, or sedatives; drinking water to which the patient is unaccustomed; poisoning with such drugs as arsenic and mercury; or, in susceptible persons, sudden changes in atmospheric conditions. The vomiting is preceded by nausea and epigastric pain and tenderness, and is often followed by profound prostration. The vomited matter consists, first, of the gastric contents, which may throw light on the cause of the attack; then of mucus, saliva (which has been swallowed), bile, and in grave cases altered blood.

In *chronic gastritis* vomiting often occurs in from half an hour to an hour and a half after eating, the food being only partly digested and sometimes coated with mucus; it does not produce the prostration incident to vomiting in acute gastritis, and is followed by some relief of the gastric uneasiness and pain. The emaciation may suggest cancer of the stomach.

In *ulcer of the stomach* vomiting is rarely absent. Occurring usually soon after the taking of food, it relieves the gastric pain, but there is nothing characteristic in the vomit unless it contains blood. Welch believes that gastric hemorrhage in recognizable amount occurs in about one-third of the cases.

In *cancer of the stomach* vomiting is an almost constant symptom; but it may not occur until comparatively late in the disease, although in rare instances it is one of the earliest symptoms. Usually it does not appear until dyspeptic symptoms have persisted for some time. There is no uniformity in the frequency of its occurrence nor in the character of the vomit. As a rule, the interval that elapses after taking food is longer than in the case of ulcer, and the ejection of food does not give so much relief to the patient. The symptom may occur every day or several times a day in the early stages; but if *dilatation* of the stomach develops, as it usually does in cancer of the pylorus, vomiting may be deferred for several days and then be correspondingly more copious. Blood, frequently altered by gastric juice so as to resemble coffee-grounds, is a common constituent of the vomit. Some pus is often found by microscopical examination. (See under Inspection.)

Reflex Vomiting. Nausea and vomiting are excited in some persons

by the sight of blood, or by a horrible or loathsome spectacle; others are more susceptible to foul odors and disgusting tastes.

Vomiting is frequently reflex; that is to say, irritation at some point is transmitted by the proper afferent nerve to the vomiting-centre and then reflected to the stomach. Vomiting of this character occurs in *pregnancy*, and in diseases of the *appendix vermiformis, ovaries, uterus, bladder, prostate gland, lungs, nose, eyes, kidneys, intestine, peritoneum, liver, gall-bladder*, and *bile-ducts*.

Vomiting is of reflex origin when there is no local affection of the stomach nor general disease to account for it, and when a remote source of irritation can be discovered, the removal or mitigation of which checks the vomiting. The particular organ that is the source of the irritation must be determined by a careful physical examination guided by the indications furnished by age, sex, time of occurrence, habits, and the symptoms that accompany the vomiting.

The nausea and vomiting from which many women suffer during the early months of *pregnancy* are most marked on rising in the morning; they are aggravated if the patient has been on her feet much or has been subjected to any exhausting or worrying influence; on the other hand, they are relieved by quiet and the recumbent posture. In diseases of the ovary, uterus, bladder, and prostate there is local pain, catarrhal symptoms, inflammation, or noticeable enlargement.

The *lungs* are probably not often the cause of reflex vomiting. Rarely, however, *phthisis* is so masked by gastric symptoms and vomiting as to be overlooked. More frequently it is the act of coughing and the effort to expel the sputa from the throat that produce the vomiting; expectoration tickles the throat, and may have the same effect as the finger or feather in inducing vomiting. This seems to be the explanation of the vomiting which follows a hard spell of coughing in pertussis.

Peritonitis may be suspected to be the cause of vomiting if there has been injury to the peritoneum from a surgical operation, or if it has been exposed to infection through the uterus and tubes, or from disease of organs surrounded by it, as the vermiform appendix. Vomiting may be the only symptom present except collapse. The fluid is not only ejected, but also regurgitated, and may appear to flow from the stomach. Large amounts of fluid are discharged, clear and of a green color.

In the vomiting due to the passage of a *renal* calculus or a *gallstone* the colicky pains and their location definitely point to the source.

Vomiting in Toxæmias. Vomiting is also a marked symptom of toxæmias, being probably produced by direct irritation of the vomiting-centre. Among such diseases are the *specific fevers*, notably *scarlet fever* and *yellow fever; sewer-gas poisoning;* diseases of the liver and kidney, which produce *cholæmia* and *uræmia*, particularly cirrhosis of the liver and interstitial nephritis. The vomiting of uræmia usually occurs in the morning. It is accompanied by nausea and depression. Whenever morning nausea and vomiting occur in an adult without obvious local cause, the urine should be examined. Other confirmatory signs are high-tension pulse, accentuation of the aortic second sound, and hypertrophy of the heart.

Cyclic Vomiting. This condition was described by Leyden in 1882 as periodic vomiting. Cases in children have been recorded by Snow and others. Clinically, the vomiting is sudden in onset, severe, and consisting first of the contents of the stomach and later of acid mucus. There is usually a febrile reaction at the onset, but this may be absent in adults. The abdomen is almost invariably retracted. There is usually a degree of prostration which is out of proportion to the local manifestations, and may be dangerous. There may be narcosis, delirium, or great restlessness. These gastric crises recur at intervals of six weeks to six months, and recur periodically in spite of the utmost care as to diet. This disease is probably a gastric neurosis, and has analogies with migraine. There is no reason to believe that it is reflex in origin. It may be due to the accumulation of toxic substances. Many of the cases are instances of periodic gastrosuccorrhœa.

Cerebral Vomiting. Vomiting due to *cerebral disease* is a well-recognized symptom; in early life it is a characteristic feature of meningitis and tumor of the brain, and is likewise of moment in later life. I am of the conviction, however, that it is not sufficiently recognized as one of the first symptoms of apoplexy. True, we find that apoplexy occurs after a full meal, when the attack is associated with indigestion, with efforts at vomiting; and I do not here refer to such cases, but to cases of painless, often watery vomiting, occurring without nausea and without retching. A sudden, violent expulsion of the stomach-contents, ceaseless, unrelieved by remedial measures, has been seen by the writer to precede other signs of apoplexy by from thirty minutes to twenty-four hours. In all cases of apoplectic character the pulse is slow and full, while in nausea and vomiting from other causes, in the aged particularly, it is weak and feeble. Moreover, some alteration of breathing is noticed. It is either irregular, or slow, or unduly hurried, and proves the intimate relation of the vomiting to the respiratory centres. Further, strength is seen, not weakness; in the apoplectic the face is congested, not pallid as in simple sick stomach. The other peculiarities of cerebral vomiting have been indicated.

Crises. Sudden attacks of vomiting with hyperacidity, with or without pain, often occur in *locomotor ataxia* and in other affections, as hysteria. They also occur in movable kidney, and are then known as Dietl's crises.

Diagnosis. Vomiting is readily recognized. It is often productive of serious lesions. It may cause apoplexy or cerebral congestion; or acute over-distention of a dilated heart, as in aortic regurgitation. If it continues for any length of time, and much fluid is ejected, vomiting is attended by anuria and rapidly followed by collapse. It also induces thirst.

CHAPTER XIV.

CHANGES IN THE APPETITE—THIRST—FLATULENCE—HICCOUGH.

The following subjective symptoms may be complained of, and while they are common in gastric disorders, may be present in other general and local diseases: disorders of appetite, bad taste in the mouth, thirst, eructations, pyrosis, distress or weight after meals, burning after meals, flatulence, nausea, vomiting, constipation, diarrhœa, pain, vertigo, and cardiac palpitation. Nearly all the subjective symptoms represent gastric neuroses, and will be more fully described in the section devoted to that subject.

Bad Taste. This is usually due to acute catarrh, but may be present in chronic catarrh. It is said to be characteristic of the acute form of gastritis popularly known as "biliousness." It may be due to local causes, as decayed teeth, disease of the mouth, tonsils, or nose. It may be due to medicines—*e. g.*, potassium iodide—or to metallic poisoning.

Thirst. Thirst is not a symptom of gastric disorder alone; it is a symptom of diabetes and all conditions in which the body has lost fluids, by sweating, vomiting, or purging; by evaporation and combustion (fever); or by hemorrhage. It is common in acute and chronic gastritis, particularly in the alcoholic form.

Distress, Weight, and Burning. These complaints are very common and may come on immediately after meals; they may be due to dyspepsia, hyperacidity, dilatation, bacterial fermentation, or flatulence, and exist in varying degree, either singly or combined. (See Gastric Hyperæsthesia.)

Nausea. This symptom is usually associated with vomiting. In some persons it is impossible to excite vomiting, although they may suffer intolerably from nausea. Nausea is akin to vomiting in its mechanism and clinical associations (*q. v.*). It is a common incident in chronic interstitial nephritis, and in old people with arterial sclerosis and defective renal elimination. It may be due to irritating ingesta, to hyperacidity, to gastrectasia, or to toxins formed in the stomach.

For a consideration of vomiting (Chapter XIII.), constipation and diarrhœa (Chapter XV.), pain (Chapter VIII.), vertigo (Chapter XVIII.), see the chapters indicated.

Alterations of Appetite. Loss of appetite, or *anorexia*, may be due to a number of causes. It is present in all forms of organic disease of the stomach except occasionally in ulcer. The appetite may or may not be impaired in gastric neuroses. Everyone is familiar with the loss of appetite due to nervous impressions, as emotions, anxiety, or mental care. Anorexia is of frequent occurrence in disorders remote from the stomach that modify the condition of the organ reflexly. In the section on Vomiting will be found statements showing the influence of central dis-

ease and disease of distant organs upon the stomach; through the same channels and through the same mechanism, and hence by the same group of causes, loss of appetite may be produced. Loss of appetite is a constant accompaniment of the moderate gastritis which attends all fevers. It is seen in dyspeptics who have carried out a restricted dietary—anorexia from disuse. In hysteria it is fairly common, and when extreme is known as *anorexia nervosa.* In all forms of anæmia, in all chronic wasting diseases, and in functional and organic disease of the nervous system the appetite is lost. The writer has been particularly impressed with the importance of determining the presence or absence of suppuration in some portion of the body in all cases in which there is loss of appetite or distaste for food, the cause of which is not of gastric origin.

Boulimia, or excessive appetite, sometimes occurs in children, and is popularly thought to be due to worms. Excessive desire for food is a common symptom in the earlier periods of diabetes, and is said to be present in disease of the mesenteric glands. It occurs also in gastric neuroses. *Perversion* of the appetite, in which all sorts of substances are greedily swallowed, occurs in hysteria, dementia, and pregnancy. It is known as *pica.*

Flatulence. Flatulence is an accumulation of gas in the stomach or intestines, and is a very common source of complaint. Gastric flatulence is marked by distention of the stomach, with the discomfort which it occasions, and by the eructation of gas at variable intervals after the taking of food. When the gas is the result of the fermentation which accompanies the production of the fatty acids, flatulence is frequently accompanied by pain which is relieved by eructations. When the distention is great or long-continued, disturbances in the action of the heart, particularly palpitation and intermittency, are likely to occur. Occasionally flatulence interferes with the breathing, and, from the apprehension which this symptom and palpitation excite, faintness and inaptitude for mental and physical work may arise.

Flatulence may be due to *carbon dioxide* being generated and retained on account of motor deficiency. It is seen in the middle-aged and in the old. *Air* swallowed with the food or the saliva is an occasional cause. Flatulence may also be due to the regurgitation of pancreatic juice, as in fixation of the stomach-wall and open pylorus. It comes on four or five hours after eating, and is caused by the decomposition of the carbonates of the pancreatic juice which liberates carbonic acid. Flatulence from bacterial fermentation is seen in dilatation of the stomach, and is usually continuous. It also occurs in chronic indigestion. Flatulence in rare instances is due to disturbance of the interchange of gas between the blood and the contents of the stomach. Normally it is known as *gastro-intestinal respiration.*

Excessive flatulence is a common manifestation of hysteria. Such patients may complain of something rising in the throat from the stomach and smothering them (*globus hystericus*). There may also be tympanites and even phantom tumor. It may be necessary to anæsthetize the patient completely in order to diagnosticate the latter from genuine tumor.

Regurgitation of gases or ingested matter, belching, or eructation is a frequent symptom of gastric disorder. It may be limited to the discharge of gas, although sometimes imperfectly digested food also regurgitates. (See Rumination.) Regurgitation of the gastric juice alone causes an unpleasant taste, and the fluid is hot and acrid. The juice is usually brought up with the belching of gas.

Pyrosis, or waterbrash, is a common symptom in some forms of dyspepsia, and usually occurs in the morning when the stomach is empty, at which time large amounts of fluid are ejected. The fluid is thin and watery, sometimes acid, sometimes tasteless; in other cases the fluid is slightly alkaline. The fluid is ejected without vomiting. Sometimes the discharge begins immediately after eating. The late Dr. Chambers thought that the fluid was saliva, swallowed and retained in the lower part of the œsophagus by a spasm of the cardiac orifice, which, when a sufficient amount had been collected, gushed back into the mouth. Pavy and Handfield Jones believe that the fluid is secreted by the stomach; while, on the other hand, Roberts, who found the liquid to possess diastatic power, believes it to be saliva. *Acid eructations* from hyperacidity or fermentation begin one or two hours after meals. They rarely occur in dilatation, but are common in over-feeding.

Hiccough, or singultus, is a spasm of the diaphragm. The contractions take place at more or less regular intervals, and are attended by a peculiar clicking sound, due to the sudden passage of air through the glottis. Hiccough may be a serious symptom; it may last but a few minutes or continue for several days. In the latter case it causes extreme exhaustion. In chronic disease its occurrence is of bad prognostic omen. It may be due to exhaustion alone, as in the typhoid state or in shock; to central nervous disease, as brain tumor or meningitis; to hysteria and mental emotion; it may be the result of toxæmia, as in uræmia, diabetes, or gout; or it may be of purely local origin, as in gastric carcinoma, intestinal obstruction, peritonitis, hepatic and pancreatic disease.

CHAPTER XV.

DIARRHŒA AND CONSTIPATION.

DIARRHŒA.

DIARRHŒA may be due to nervous causes, the direct action of irritants in the intestine, to catarrhal inflammation, or to infectious inflammation of the tract. It is a symptom which is common in the course of gastric neuroses, particularly those attended by hyperacidity. It may be conservative, the result of an attempt to eliminate poisons from the system. It may be a symptom of constipation. (See Spurious Diarrhœa, page 129.)

Diarrhœa is a symptom of disorder of the intestine, which in turn is itself the cause of symptoms; just as jaundice, a symptom of hepatic disorder, is the cause of various symptoms. In diarrhœa there is increased frequency of the movements of the bowels. This is due to increased peristalsis of the intestine, which in turn is due to a number of causes. Not all increased peristalsis results in diarrhœa.

Nervous Diarrhœa. Increased peristalsis may be due to some impression upon the nervous mechanism of the intestines. This may explain the diarrhœa of emotion, or that which occurs from other psychical influences.

Catarrhal Diarrhœa. In the larger number of cases the diarrhœa is due to catarrhal inflammation of the intestinal tract. The causes of the catarrhal inflammation are many, and have been divided into primary and secondary causes.

Primary catarrh is due to the direct influence of causal factors upon the mucous membrane. (1) It occurs after cold or exposure, (2) from the direct irritation of undigested food or hyperacidity, and (3) from the action of irritants, as of bacteria or their products. *Infectious Diarrhœa.* Catarrhal inflammation due to micro-organisms is the most frequent form in children. *Secondary* catarrhs follow other lesions, as ulcers. The catarrh, and hence the diarrhœa, that attends the ulceration of typhoid fever and of dysentery; that which occurs in the course of Bright's disease; and the diarrhœa that attends carcinoma or other organic disease of the bowel, are of this nature. In addition, a catarrh of the bowels may arise from venous stasis in the mucous membrane, with chronic congestion, as in organic heart disease with congestion of the liver.

Toxic Diarrhœa. Diarrhœa is a symptom of the action of certain poisons, such as mercury, arsenic, and other corrosive agents. The diarrhœa which occurs from the irritant action of food-products and in cholera infantum is due to a toxic ptomaïn or to actual infection. In food-poisoning *Bacillus enteritidis* of Gärtner is often the irritant.

Vicarious Diarrhœa. Diarrhœa sometimes fulfils a vicarious office. This is the case with the diarrhœa which comes on in cases of chronic

Bright's disease, and in acute Bright's disease before the supervention of uræmia. When diarrhœa occurs in a person with pallor, dimness of vision, and œdema, the urine should always be examined.

Symptoms. Increased Movements of the Bowels. The frequency of the movements varies with the cause. In diarrhœa of nervous origin, after five or six movements have occurred, the patient usually is relieved, because by that time the cause for the nervousness has disappeared. In catarrhal diarrhœa the number of stools varies from half a dozen in twenty-four hours to the same number in an hour; indeed, in some severe cases the evacuations may be almost constant.

Character of the Stools. 1. The stools are *fecal*, with a small amount of *water*. They are light in color, softer than natural, but yet retain their form—the kind of movements seen in simple catarrh.

2. The fecal matter is mixed with *undigested food*. The feces are in scybalous masses, and the watery element is increased. They are the stools of the so-called dyspeptic diarrhœa.

3. Along with the feces more or less *mucus* is seen. The amount of mucus depends upon the seat as well as the intensity of the inflammation. Inflammations of the large intestine are attended with mucous discharge, which may be mixed with and stained by feces so that it can be recognized only by close inspection. In milder degrees of catarrh mucus is seen on the surface of the fecal masses.

4. Formed feces disappear almost entirely, and instead the evacuations are *watery*. The watery stools may be discolored, as in the pea-soup evacuations of typhoid fever; or they may be almost clear water, as in the rice-water discharges of cholera.

5. The evacuations may contain *blood*. Bloody discharge usually accompanies the discharge of mucus; when the catarrh is in the lower bowel, blood may occur independently of mucus and small amounts of free blood are seen. When both are present, the mucus is tinged with reddish specks. The blood may be bright in color, and then usually comes from the rectum. It must be remembered that the blood may emanate from bleeding hemorrhoids, or fissure that has been unduly irritated by the diarrhœa. It is then bright red and is not mixed with the stools, and from its position it is readily seen that the blood was discharged after the fecal matter. On the other hand, bloody stools may be due to cirrhosis of the liver with venous congestion, to the ulceration of typhoid fever, to the intense inflammation of enteritis, to carcinoma of the bowel, or to intussusception, in which they almost have the value of a pathognomonic symptom. It must be remembered that blood of this character is discharged from the bowel independently of diseases of that tube, as in purpura, scurvy, and other blood diseases. (See Embolism, page 26.) If mixed with the stool, the blood may be black, as in all forms of *melœna;* or it may be dark red in color. Black blood usually comes from the stomach or the first part of the duodenum, and may be the result of ulceration, or even the swallowing of blood.

Microscopical and Bacteriological Examination. (See Feces.) In simple catarrhal inflammation of the tubules but little is found on microscopical examination except an excess of epithelium from the mucous lining. In

more intense inflammations, in addition to epithelium pus and blood and mucus are found. Micro-organisms are found, the kind depending upon the cause of the diarrhœa. In health Booker has found at least forty varieties of micro-organisms in the intestine, many of which, in all probability, are not pathogenic. Even *Bacillus coli communis* and *Bacterium lactis aërogenes* are found in health. In the diarrhœa of children both forms are present in excessive numbers because the conditions are favorable to their growth, and in all probability are the cause of the irritation of the bowel. In that form of inflammation of the bowel known as dysentery, in addition to the bacteria that attend inflammation, *Amœba coli* is often present. It has been found that dysentery may be due to a number of causes, but that the so-called tropical dysentery is due to the bacillus of Shiga or to the protozoa first described by Kartulis, in Egypt, and in this country by Osler. (See Feces.)

Pain. The symptoms that attend diarrhœa depend upon the cause and the frequency of the evacuations. The most frequent symptoms are *pain* and *flatulent distention*, with *borborygmi* and *tenesmus*. The pain depends largely upon the cause. When the irritant is a product of indigestion or a bulky mass, pain is more or less severe and situated in the centre of the abdomen, or diffuse. The pain occurs before defecation; it is sharp, lancinating, and is usually relieved by the act. When the inflammation is in the large intestine, the pain may be complained of in the course of the large bowel or be more intense over the cæcum and the sigmoid flexure. The rectum may be the seat of pain or of painful sensations, as if there were a hot ball in the lower pelvis.

Flatulent Distention. The flatulent distention is usually not very great. The abdomen is distended, tympanitic on percussion, and tender on palpation; the signs are more marked in the middle of the abdomen when enteritis alone is present, or they may be observed over the entire colon, as in the so-called enterocolitis of children. With the distention there are borborygmi, which usually subside after the evacuation.

Tenesmus occurs in all forms of diarrhœa when the evacuations are frequent. In severe cases it may be almost continual, and may lead to prolapse of the bowel. It is of common occurrence in prostatic enlargement, pelvic growths, rectal diseases, vesical calculus, impacted feces, worms or foreign bodies in the rectum,—conditions which must be excluded.

General Symptoms. The general symptoms that attend diarrhœa depend upon the cause. In simple diarrhœa there may be slight feverishness only, with a little weakness. In diarrhœa with copious stools containing mucus, with or without blood, the fever is marked and may rise as high as 103° F. The fever that attends dysentery is high, and usually rises rapidly at the beginning.

Prostration. More or less prostration attends all cases. It is, however, more marked when there are frequent watery evacuations. In its most pronounced degree it is seen in cholera and cholera infantum. *Collapse* rapidly ensues under these circumstances on account of the depleting effects of the excessive watery discharge. In catarrh of the intestines secondary to typhoid fever and other conditions the general symptoms depend upon the primary disease.

Chronic Diarrhœa.

Chronic diarrhœa may be due to chronic inflammation of the bowels, as in chronic intestinal catarrh. It may be secondary to the ulceration of dysentery, tuberculosis, syphilis, or cancer. It may be secondary to chronic gastritis with anacidity, or attend the neuroses of gastro-enteroptosis. It is the common diarrhœa of amyloid disease. In chronic diarrhœa the number of the stools varies, but seldom amounts to more than ten to fifteen in a day. In chronic intestinal catarrh three or four movements occur in the twenty-four hours, the first evacuation taking place immediately on rising, and the remainder during the morning hours. Women suffer more than men in this respect, the movements being readily excited by exhaustion or nervous influence, as grief, emotion, or excitement of any kind. The stools are watery, and contain fecal matter which is usually coated with mucus. The color of the feces is not changed. The patients usually suffer from intestinal dyspepsia, or they are subject to some gastric neurosis. They are not under-weight, and except for the inconvenience of the morning hours quite equal to the ordinary demands of life. They are more nervous than most people and are liable to attacks of hemicrania.

Membranous or Paroxysmal Diarrhœa.

In a number of cases the discharge from the bowels resembles membrane. The disease is also called membranous enteritis. The discharges contain much mucus and may be quite watery. After the feces have been passed, membrane is discharged. This may be in shreds or large masses, or may represent a cast of the bowel. The patients are usually women who are hysterical and have some menstrual disorder. Pain may precede the discharge, and continue until there is complete relief.

Spurious Diarrhœa.

This is the diarrhœa of constipation. Fecal impaction may be associated with diarrhœa. The mass of feces may be channelled or a colitis may occur secondarily. The evacuations are scybalous, coated with mucus, and often bloody. Tenesmus is present.

CONSTIPATION.

Constipation may be due to a number of causes. It may be due to alteration or diminution in the secretions of the intestinal tract, as is seen in all fevers, except when they are attended by specific intestinal catarrh as in typhoid fever. Such diminution of secretion occurs in the summer, when there is more free perspiration than in other seasons, and is present in affections attended by excess of perspiration or exhaustive diuresis. Constipation, therefore, is a common symptom of diabetes.

In addition to alteration of the secretion, diminution in the sensibility of the nerves may exist. This is the one chief cause of habitual consti-

pation which is so prevalent. Through carelessness the patient loses the habit of having a regular movement of the bowel each day, and in consequence the usual stimulus is removed. Constipation also occurs from weakness of the muscular coat of the intestinal wall.

The three conditions—diminution or alterations in the secretions, debility of the muscles, and impairment of the sensibility of the nervous mechanism—combined account for the prevalence of constipation among persons of sedentary habits and those living upon improper diet. General diseases and local disorders that influence either of the above elements cause constipation. Thus in anæmia and chlorosis, in neurasthenia and hysteria, constipation is a common condition. Its occurrence in fevers has been mentioned. In the convalescence from exhausting disease and prolonged confinement to bed constipation is apt to ensue.

Local Causes. Atony of the abdominal muscles or of the bowel is a cause. Atony is most strikingly seen in peritonitis and typhlitis, in both of which a paretic state of the bowels develops. It is seen in the aged and in cachexia along with atony of other muscles. Obstruction of the bowels, acute or chronic, usually causes constipation (*q. v.*); if the obstruction is not complete, there may be diarrhœa on account of catarrhal inflammation. Constipation often is due to pain, particularly pain seated in the rectum, as from fissures, hemorrhoids, or fistula. The pain is such that the patient shrinks from an evacuation, and frequent postponement soon causes constipation. Constipation occurs also from local diseases in other portions of the body, in all probability influencing the nervous mechanism by which peristaltic action is excited. In acute and chronic disease of the brain and cord, as meningitis and myelitis, constipation is a usual attendant. It also occurs in tetanus. If the bowel is deprived of fecal matter, evacuation ceases; constipation is, therefore, a common sign of stricture of the pylorus and of stricture or cancer of the œsophagus.

Symptoms. Constipation is characterized by diminution in the frequency of the bowel movements. The frequency of the movements varies in health. Some persons are comfortable with an evacuation once a week, or at most every third or fourth day. There are cases on record in which the evacuations took place but once a month. Cases of this class are usually due to muscular paralysis of the bowels, with secondary dilatation. The accumulation of feces is removed by a sharp attack of diarrhœa, attended by much pain. The diarrhœa sometimes continues for twenty-four hours. When it sets in, fever may be present until there is thorough evacuation.

Local Symptoms. The symptoms other than cessation of bowel movements that attend constipation are local, due to the discomfort of the accumulation of feces. The local symptoms may be limited to the rectum or extend throughout the abdomen. If to the former, there is a sensation of fulness from a mass. It may cause some pain, or may set up tormina and tenesmus, and portions of it may be discharged from time to time. In other words, a diarrhœa may occur—the diarrhœa of constipation, or spurious diarrhœa. The evacuation does not give relief, and the desire for a movement may be more or less constant. In constipation with

fecal accumulations the outline of the colon may be marked out by palpation and percussion of the distended abdomen. In its course masses are felt varying in size from that of a marble to that of a base-ball, and in consistence they may be soft to the palpating finger; they are never indurated like a calcareous mass, as gallstones, or a mass due to malignant disease. The abdomen is distended; there is considerable rumbling, and sometimes peristaltic waves are seen. (See Fecal Tumor.)

General Symptoms. While in many instances the general symptoms are of no consequence, in others the patients are nervous and may be in a more or less impaired state of health from the secondary effects upon the stomach. Digestion is impaired, and the form of indigestion is that which attends neurasthenia. The patients are of spare habit, usually of dark or muddy complexion. They may be depressed and more or less hypochondriacal, and there is inaptitude for mental exertion. The tongue is constantly furred, the appetite variable; a feeling of weight and fulness after eating and some degree of flatulence are usually present.

In this connection must be mentioned the constipation that occurs on account of lead-poisoning, and the exhibition of drugs, as opium or other astringents. The constipation of lead-poisoning is usually attended by colic, the blue line on the gums is seen, and wrist-drop or other manifestations of lead-poisoning may be present.

The Secondary Effects of Constipation. The effects of constipation upon the intestines are various and sometimes disastrous. They are *dilatation* and *ulceration*. The dilatation may be so great as to distend the entire abdomen, as in cases reported by Formad and Osler. Ulceration may be localized to the rectum or cæcum, or extend throughout the entire large intestine. On palpation the course of the colon is tender, and fecal masses may be outlined which are painful because of their pressure upon the adjacent ulcer. Rectal ulcers are often deep, and may be followed by perirectal abscess. In the cæcum the accumulation may cause a large boggy swelling—stercoral typhlitis—which is tender on pressure and dull on percussion.

Fecal impaction, with secondary ulceration, is of frequent occurrence in typhoid fever. This must be borne in mind, for serious general and local symptoms often arise because it is overlooked. Recently I saw a case with diarrhœa of constipation and some fever, which persisted for weeks after the usual course of typhoid fever. It was thought the patient had tuberculosis, or that the typhoid process was abnormally prolonged. Examination disclosed ulceration into the vagina, and the feces were constantly discharged from this orifice. It had been thought that the discharges of feces were due to diarrhœa. Of course, fever attended the process and rendered the case all the more obscure.

Incontinence of feces arises because the centres controlling the act of defecation are impaired. Afferent fibres from the rectum pass to centres in the lumbar cord, from which efferent fibres return to the sphincter ani. Through higher centres, as in the brain, the lumbar centre may be inhibited. When the rectum is full, impulses are sent to the lumbar centre and the brain, and unless they are resisted by the will, the act of defecation takes place reflexly.

Incontinence of feces occurs in the insane, in delirious persons, in coma, and in profound prostration. It may be due to paralysis of the sphincter or to injury of the pelvic floor.

Painful defecation is usually attended by bloody stools when an anal fissure is present. Hemorrhoids and enlarged prostate and prolapse of the rectum give rise to pain. Disease of the pelvic viscera may also cause painful defecation.

CHAPTER XVI.

THE VOICE AND SPEECH.

Aphonia.

THE most common symptom of affections of the larynx is disturbance of phonation. The voice is changed in character, or may be lost in any affection which causes swelling of the mucous membrane, or occlusion of the orifice, or which interferes with the action of the vocal cords. The voice may be *hoarse* in acute and chronic inflammations, in tumors, in specific ulcerations about the larynx, and in paralysis of the cords. From simple hoarseness it may vary in intensity to complete aphonia. Laryngoscopic examination is necessary in order to detect the presence or absence of paralysis of the vocal cords. (See Paralyses.)

Chronic hoarseness may be due to chronic laryngitis. Slight hoarseness, deepening to aphonia, attended by soreness, and later some dysphagia, is seen in *lupus*. Infiltration and scar-contractions cause dyspnœa later in some instances. Aphonia from inflammation or œdema is also a symptom of *leprosy*, which, however, is present in other situations as well. The *duration* may be significant. Hoarseness of long duration (years) is said to be prodromal of *cancer*. (Ziemssen.)

Functional aphonia may occur after excessive use of the voice and in hysteria. Hysterical aphonia occurs in women and young girls; the laryngoscope reveals nothing; the acts of coughing, laughing, and sneezing are normal, and a sound may be created in either act; it appears and disappears suddenly.

Tone of the Voice. The character of the voice may change. When one-sided paralysis of a cord is present, the voice is flat and toneless. In cases of paresis of the tensors of the cords a falsetto voice results. Diplophonia occurs in one-sided paralysis, and in some cases in which small tumors lying between the cords come up during the act of phonation and form nodes. Two tones are formed at the same time. Frequently only certain tones are doubled.

Disturbances of Speech.

Disturbances of speech may be divided into two groups: *aphasia*, the disturbance of the central nervous mechanism controlling speech, writing, and mimicry; and *anarthria*, the disturbance of the peripheral motor mechanism of speech.

Aphasia. By aphasia is meant the loss or impairment of the ability to understand spoken, written, or mimic language, and to express thoughts by the same means. It is divided ordinarily into two forms: *motor aphasia*, or the inability to innervate the motor apparatus for speech,

while the sensory or perceptive functions are intact; and *sensory aphasia*, or the inability to recall or understand words, although the ability to produce sound is preserved. A variety of other forms, however, have in the course of time come to be recognized. Oppenheim recognizes the following five varieties:

1. **Motor Aphasia.** This consists of the loss of power to speak, with persistence of the understanding of spoken, written, and mimic speech. This is the first form of aphasia in which it was possible to locate with accuracy the portion of the brain involved. The lesion is cortical or subcortical, and involves the foot of the third frontal convolution on the left side. The symptoms vary according to the extent and destructiveness of the lesion.

2. **Sensory Aphasia.** The perception of sound as such is preserved, but there is inability to recognize the significance of words, although spontaneous and occasionally voluntary speech is preserved. The lesion is found usually in the auditory centre—that is, the first temporal convolution on the left side. The symptoms are variable, alexia being often present.

3. **Pure Alexia, or Word-blindness.** Although sight is preserved and objects may be recognized, the ability to understand written or printed language is lost. Spoken language is still understood, voluntary speech and writing possible, and occasionally written words may be read if the patient is permitted to trace the letters with a pencil or the finger, recognizing each one as it is formed. The lesion is found usually in the left occipital lobe on the external surface, but sometimes involves the gyrus angularis.

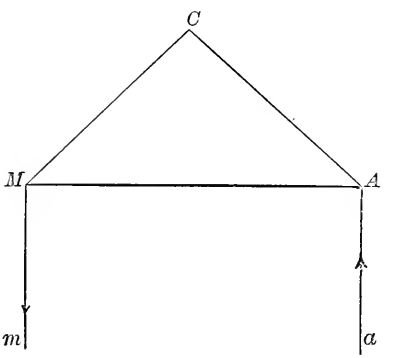
4. **Pure agraphia,** or loss of the power of writing, all the other qualities remaining normal. Lesions have been found in the left upper parietal lobe.

5. **Optic Aphasia.** Objects may be seen and recognized, but it is impossible for the patient to find the proper name for them. If the objects are recognized by some other sense, as, for example, hearing or touch, the name may be recollected instantly. The lesion is found usually at the junction of the first temporosphenoidal and the occipital lobes. This form is frequently a symptom in otitic abscess. Loss of the stereognostic sense may also be regarded in some respects as an aphasic manifestation.

In order to explain aphasia, it has been customary, since the time of Wernicke, to employ the diagram shown in Fig. 11. The triangle *ACM* represents the intracerebral paths and centres for the mechanism of speech, and the lines *Aa* and *Mm* the peripheral apparatus. *A* represents the centre for auditory perception; *M*, the centre for the emission of motor impulses; and *C*, the concept-centre, in which the intellect analyzes the impressions received and from which the directing influence for the choice of language is transmitted to the motor centre. *Aa* represents the auditory nerve; *Mm*, the motor nerves to the pharynx, tongue, and lips. Auditory impressions may, therefore, be transmitted along *Aa* to *A*, thence directly to *M*, and thence to the larynx. This is the mechanism supposed to be involved in ordinary mechanical speech—that is to say, the mechanical repetition of spoken words. The auditory impressions may, however, pass from *A* to *C*, there

to be analyzed or understood, and then transmitted to *M*, either in the same or in an altered form. This constitutes the intelligent repetition of spoken language. If the alteration of form is considerable, or if, without immediate auditory impressions, impulses are transmitted from *C* to *M*, voluntary or intelligent speech is said to occur. Although this diagram probably does not accurately represent the conditions existing in the brain, it has been found that the varieties of aphasia that can theoretically be deduced from it correspond more or less closely to those that may be recognized in actual practice. These varieties are as follows: Destruction of the motor centre *M* gives rise to the so-called cortical motor aphasia with the following symptoms: loss of (1) voluntary speech; (2) repetition; (3) reading aloud; (4) voluntary writing; (5) writing from dictation; while (1) the understanding of speech, (2) the understanding of writing, and (3) the ability to copy writing are preserved. Destruction of the auditory centre *A* gives rise to cortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing; (3)

FIG. 11.



the ability to repeat speech; (4) the ability to write from dictation; (5) the ability to read aloud; while (1) voluntary speech, (2) voluntary writing, and (3) the ability to copy writing are preserved. A lesion at *C* would give rise to cortical apperceptive aphasia. The symptoms of this form would differ very slightly from those due to interruption of the tracts supplying it. The centre is probably complex and its parts are widely distributed. The speech-disturbances of general paresis are due possibly to partial destruction of the concept-centre. Lesions of the various tracts of fibres connecting the different centres with each other or with the periphery also produce symptoms. Lesions between *A* and *M* produce the symptom known as *paraphasia*. (1) Voluntary speech; (2) repetition of speech; (3) the understanding of spoken and written language; and (4) the ability to copy writing, all are preserved. The only symptom, therefore, of this condition is the misuse or false pronunciation of words. Thus objects may be misnamed, one word used in place of another, different syllables of the words misplaced (literal paraphasia), or the words jumbled in a sentence (verbal paraphasia). There is usually also *paragraphia*—that is, a similar disturbance of written language; *paralexia*,

manifest when the patient attempts to read aloud; and sometimes the symptom known as *agrammatism*—that is, the misuse of cases, moods, or tenses. Paraphasia, however, occurs also in certain general diseases of the brain, and is practically always present if the intrinsic tracts concerned in speech are disturbed. Interruption of the tract uniting *C* and *M* causes transcortical motor aphasia. There are lost (1) voluntary speech and (2) voluntary writing. There are preserved (1) the understanding of speech; (2) the understanding of writing; (3) the ability to copy; (4) the ability to repeat words; (5) the ability to write from dictation; (6) the ability to read aloud. The most characteristic symptom is the inability of the patient to remember words, although he is able to repeat them fluently. The interruption between *A* and *C* gives rise to transcortical sensory aphasia. There are lost (1) the understanding of speech; (2) the understanding of writing. There are preserved (1) voluntary speech; (2) voluntary writing; (3) the repetition of speech; (4) reading aloud; (5) writing from dictation. Usually both voluntary speech and writing are affected by the paraphasia resulting from the interruption of the intrinsic tracts. It differs from the preceding form particularly in the fact that the meaning of words spoken upon repetition or written from dictation is not grasped by the patient. In this form, communication with the patient, even by gestures, is often impossible. Finally, lesions may occur in the tracts uniting the centres concerned in speech with the periphery. Lesions in the tract *Mm* give rise to subcortical motor aphasia. There are lost (1) voluntary speech; (2) the repetition of speech; (3) the ability to read aloud. There are preserved (1) the understanding of speech; (2) the understanding of writing; (3) the ability to copy; (4) voluntary writing, and (5) writing from dictation. This is, of course, the purest form of motor aphasia. Interruption of the tract *Aa* gives rise to subcortical sensory aphasia. There are lost (1) the understanding of speech; (2) the repetition of speech; (3) the ability to write from dictation. There are preserved (1) voluntary speech; (2) voluntary writing; (3) understanding of writing; (4) reading aloud; (5) copying.

In actual pathology this theoretical classification with groupings of symptoms must sometimes be modified on account of a variety of conditions. The most important modification is that produced by the existence of possible lesions of other centres concerned in speech. Thus the share taken by the visual receptive and apperceptive centres is of great importance in all persons who have been taught to read. They necessarily are concerned also in the production of writing. It is not, however, possible to represent the mechanism of these functions by a diagram as we have represented auditory and motor speech; for it appears that impulses from the visual centres must pass through the receptive centre for speech, or *A*, before being transferred to the arm-centre or the speech-centres. The same is true for tactile impressions. These are of importance chiefly in blind persons who have been taught to read with their fingers, in whom, indeed, they may be equal in importance to the visual centres in normal persons. Various complicated diagrams have been devised for the purpose of exhibiting the influence of all these centres upon speech, and Mills has introduced

an additional naming-centre, situated in the third temporal convolution, in which perceptions are given the names that properly belong to them. A source of error is the fact that lesions may be only partially destructive, or may be so extensive as to involve two or more tracts or centres at the same time. Under these circumstances the symptoms become very complex, and it is often impossible to determine the extent of the physiological disturbance that has been produced. Usually, however, the localization of these lesions is not difficult, on account of the predominance of certain characteristic localizing symptoms.

It will be obvious from this description that it is necessary in each case of aphasia to test a variety of functions. These can best be examined as follows:

1. *Voluntary Speech.* If the patient is able to answer questions intelligently or makes spontaneous intelligent remarks to the physician, voluntary speech is preserved. Voluntary speech may, however, exist and the remarks of the patient be nevertheless unintelligible when there is an extreme degree of paraphasia.

2. *The Ability to Repeat Words.* This may be tested by merely saying a word or several words and getting the patient to repeat them. Mechanical speech, the centre for which is supposed to be located in the speech-area of the right hemisphere, may also be tested by requesting the patient to repeat some well-known series of words, such, for example, as the names of the months or of the days of the week, the alphabet, or a series of numbers. Sometimes familiar songs may be remembered and spoken when it is absolutely impossible for the patient to give an intelligent answer to a question. Under the influence of great emotion epithets or oaths may also be uttered. The ability to repeat words may sometimes be present when it is impossible, on account of transcortical sensory aphasia, to determine its integrity, because it is impossible to make the patient understand what he is expected to do.

3. *Reading Aloud.* It must not be forgotten that in some cases this symptom is masked by defects of vision. If possible, the eyes should always be examined and the patient be given his glasses if he has been in the habit of using them. It is advantageous to select large type, such as that used in the headlines of newspapers.

4. *Voluntary Writing.* This symptom may be masked by the existence of right hemiplegia and inability to write with the left hand.

5. *Writing from Dictation.* As in the repetition of speech, this symptom may be masked by inability of the patient to understand what he is expected to do.

6. *Copying.* Errors of vision, as well as paralysis and other motor disturbances of the arm, must be excluded.

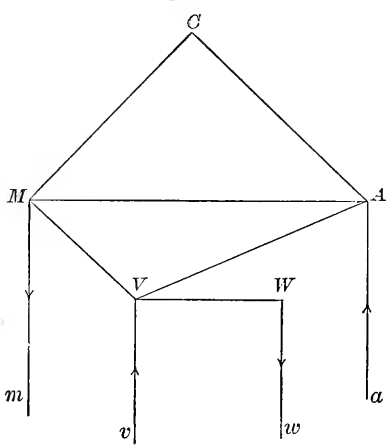
7. *Speech-perception.* This is perhaps one of the most difficult of all aphasic symptoms to determine. Usually the patient is requested to perform some simple action, such as putting out the tongue, touching the ear with the hand, etc. This may be performed perfectly, but more complex commands or long statements may not be understood. It is supposed that this is due perhaps to incompleteness of the lesion, or to a general disturbance of intellect, such as must occur, in a more

or less pronounced degree, in any case of aphasia. It is, therefore, important to attempt to converse with the patient, getting him to reply by gestures, or in writing, according to his ability, and gradually to employ more and more complex statements. In cases of marked paraphasia the improper use of words in the replies may lead to the belief that the patient does not understand what is said to him, when, as a matter of fact, word-perception is perfect.

8. *Understanding of Writing.* The diagnosis of this condition is subject to the same errors as the understanding of speech, and in addition the possibility of visual defect is to be remembered.

9. *The Existence of Paraphasia.* This, of course, can be detected only when either voluntary speech or the ability to repeat words is present. Under these circumstances it may be recognized when it is only slight in degree by getting the patient to repeat words of many syllables, such as "incomprehensibility," or sentences of several words.

FIG. 12.



Disturbances of writing, apart from disturbances of speech, may also occur. These may be better understood by a consideration of Fig. 12, in which the writing-centres as well as the speech-centres are shown. It will be seen that there may be destruction of *W*, or *agraphia*. Voluntary writing and the ability to copy are lost, while the ability to read is preserved. Destruction of *V*, or *cortical alexia*, is characterized by the loss of (1) the recognition of written words and (2) voluntary writing, while speech may be intact. In destruction of *VW*, or *conduction-agraphia*, voluntary writing and voluntary copying are lost; while the ability to read is preserved—that is to say, it corresponds exactly to the preceding form. Under such circumstances paragraphia may exist in this type. *Transcortical agraphia:* loss of voluntary writing, and ability to do mechanical copying and reading. *Transcortical alexia:* loss of the ability to read, with preservation of the power of voluntary writing and of copying. There may be interruption of the tracts to the periphery, giving rise to *subcortical agraphia*, in which voluntary writing and copying are impossible,

but the power of reading is preserved. Paragraphia never occurs in this form. *Subcortical alexia:* inability to read and copy; voluntary writing is preserved. All of these forms may coexist with the various types of aphasia. In testing the patient for alexia the following symptoms should be examined: (1) voluntary writing (see above); (2) writing from dictation (see above); (3) copying; and (4) the recognition of letters either spoken or written. In testing patients for voluntary writing with the left hand, it must be remembered that many aphasics give *mirror-writing*. The following terms also are used in connection with aphasia: *aphrasia*, the inability to form sentences with words; *dysphrasia*, the imperfect formation of sentences; *apraxia*, the total loss of speech.

Anarthria. By anarthria is meant a disturbance in the peripheral motor mechanism of speech resulting from disease of the nuclei in the medulla or of the peripheral nerves arising from them. This may vary in degree from complete aphonia, or loss of power to make sounds and words, which occurs in bulbar paralysis, or the aphonia of laryngeal paralysis, in which whispering speech is still preserved, to mere inability to pronounce correctly certain consonants as a result of local paralysis or paresis of the lips or tongue. Anarthria may be permanent or temporary; or, in cases of slight paresis, recurrent, giving rise to intermittent claudication of speech. It is tested by directing the patient to repeat letters of the alphabet, to count, or to repeat words with long syllables and difficult consonants, as "artillery," "extraordinarily," etc. Allied to anarthria, but perhaps the result of certain functional disturbances, are *stuttering* and *stammering*. In the former, if the patient attempts to speak, there is inhibition of motion for a longer or shorter interval, and then the word may be pronounced with explosive violence, and the following words of the sentence spoken normally. In *stammering* there is frequent repetition of the first two or three consonants of the word, particularly if these happen to be labials. Stuttering and stammering are associated sometimes with defective intelligence. Finally, there are a series of disturbances of speech in which intellectual derangement is apparently the chief factor. These may perhaps be forms of aphasia due to partial destruction of the concept-centre or centres. Among them may be mentioned the inability or unwillingness to speak that occurs in the *mutism* of the insane, in hysteria, and after a severe illness, as scarlet fever, causing cerebral fatigue; tendency to excessive speech, *logorrhœa;* the omission of syllables, with interruption of the speech and tremulousness of the tongue and lips, which is particularly characteristic of general paresis, difficult words, such as those mentioned above, being pronounced imperfectly, as "arlry" for "artillery," or even less accurately. *Scanning* speech, in which the words are separated by considerable intervals, and are spoken with a peculiar drawl and a descending cadence; it is particularly characteristic of multiple sclerosis, but may occasionally occur in general paresis and in hereditary ataxia. Other forms are: *explosive*, or *staccato* speech; and a peculiar, slow, drawling utterance, occasionally termed *bradylalia*, which occurs in certain states of mental depression. *Echolalia* occurs almost exclusively in imbeciles, and is characterized by the repetition of all sounds heard.

SECTION IV.

OBJECTIVE DIAGNOSIS—DATA OBTAINED BY OBSERVATION.

CHAPTER XVII.

CORRELATION BETWEEN THE OBJECTIVE SYMPTOMS AND OTHER PHENOMENA OF NATURE.

THE objective symptoms of disease are the most important to ascertain, because they are the data upon which a complete diagnosis is based. Without such data the diagnosis is mere guesswork—one of probability. With such data alone, if accurately and precisely collected, a positive diagnosis can very frequently be made. A correct diagnosis depends upon the skill and thoroughness of the physician and his ability to interpret the data secured, always provided that clear, succinct data can be obtained.

A general and physical examination of the patient is made, followed by an immediate study, or, if time permits, a study at leisure of the fluids of the body—microscopically, chemically, and bacteriologically, in order to secure objective symptoms. In the general examination we make a survey of the individual and form an estimate of his height and weight. In the physical examination the various organs and tissues are interrogated by the senses applicable to the investigation of each, aided by special instruments. The secretions and discharges, both normal and abnormal, exudates, transudates, and fluid obtained from cysts, are examined—laboratory investigation.

The student will soon learn that the process of ascertaining the objective signs of disease is in no respect different from that which obtains in the study of any object in nature or any like phenomenon. The chemist notices the form, the color, the density, etc., of the object under examination; the effects of heat and cold, of various reagents upon its structure; he determines its component parts and ascertains its relation to other objects in nature. From data thus obtained by the use of all his senses he classifies the object. The biologist notes not only the physical appearance of a given form of life, but also the phenomena of the living, sentient matter under all conditions in a varied environment. By comparison and analysis the living being is classified. Indeed, it should be emphasized

that diagnosis is a study in biology pursued by the same methods of observation and reasoning employed in other fields of biology.

By the same powers of observation and the same analytical process, the departures from health are recognized and classified. Is it not, therefore, a wonderful aid to the diagnostician to possess faculties which have been trained to minute observation by previous studies in sciences allied to medicine?

What has been thus imperfectly said is intended to emphasize the fact that no mystery attends the recognition of the objective signs of disease. Abundant opportunities of observing disease at the bedside, patient training, skill in technique, and a systematic procedure are essential.

Methods of Observation. The method by which the objective symptoms are ascertained is modified by the circumstances under which the patient is seen. It is obvious that the patient who comes to the office, or is not sufficiently ill to be in bed, has sufficient strength to stand, should be given an exhaustive examination. Moreover, we can observe certain abnormalities, as of the gait, not apparent in bed. On the other hand, in the case of a bedfast patient we observe the position he assumes, and have better opportunities for thorough examination of the various organs. Often the objective examination must be very brief on account of the patient's extreme illness. It may be advisable, although unfortunate, to exclude one or more methods, as percussion if there is pain, or auscultation if there is great restlessness or orthopnœa.

If a complete examination is to be made, it is well to begin with the exterior. After the *external examination* has been completed, the *internal examination* is made, by grouping together and examining organs functionally related, as the heart and bloodvessels, in diseases of the heart; the nose, larynx, and lungs, in pulmonary affections. The student will do well to begin at the head and take up the organs in their continuity.

The results obtained by observation are based upon comparison; the student must bear this constantly in mind. We *compare* the body as a whole with our conception of the normal individual, formed by a study of a large number of persons. We compare symmetrical parts —the right side of the chest with the left, the arm suspected to be the seat of disease with the healthy arm, etc. The cardinal rule in an examination is to base the significance of ascertained facts upon comparison with known normal conditions.

Physical Diagnosis. The methods of examination mentioned above are conducted by the sense of sight, by the sense of touch, by the sense of hearing, and by the combined senses of touch and hearing. The facts thus acquired give one full knowledge of the physical condition of the parts examined. The collection and discriminate interpretation of these facts constitute *physical diagnosis.* (See Section V.)

Bacteriological Diagnosis. Data obtainable by observation are further secured by methods employed to determine the presence of micro-organisms that are presumably the cause of the disease. This method of diagnosis is modern; it is as precise a means of reaching a conclusion as any in our possession. The methods of research required constitute the bacteriological diagnosis, and are detailed in full in Chapter

XXXVIII., Section VI. An examination of the blood, of the secretions and excretions, of pathological fluids, and of morbid products, is made in carrying out this method of diagnosis.

Laboratory Diagnosis. Chemical examination of normal and pathological fluids secures data essential in diagnosis. In this manner the blood, the gastric contents, the urine, the feces, and all pathological fluids are examined. The methods of examination are detailed in Section VI.

Data Obtained by Observation with the Aid of Special Instruments of Research. These examinations can be conducted at the bedside, although the results are more accurate if they are carried out in the clinical laboratory. The instruments of precision required are: (1) the microscope and its accessories; (2) instruments for examining the blood; (3) instruments for determining blood-pressure; (4) the spectroscope and polariscope. In Section V. the use of these instruments will be detailed.

CHAPTER XVIII.

FIRST-SIGHT IMPRESSIONS.

THE general appearance of the patient affords an idea of his ability to cope with the antagonistic forces of his environment or to overcome the deleterious effects of his occupation. It frequently indicates the effect of present or past disease or of inherited disease.

Location of Disease. The general appearance of the exterior will often indicate which system is the probable seat of the disease. For instance, violent respiratory action points to the lungs; paralysis, to the nervous system; an enlarged abdomen, to disease of the viscera in that region.

General Abnormal Vital Conditions.

The *first sight*, striking impression, is always to be noted. "Very sick," "comatose," "collapsed," etc., or "robust," "cyanosed," etc., are speaking memoranda. To the experienced practitioner the opinion formed at first glance is often of great diagnostic significance. It may happen that the patient is suffering from some unusually abnormal vital condition, a study of which must be made before the exhaustive survey of the case is conducted.

Impairment of consciousness and convulsions are readily recognized. The two often go hand-in-hand, but in some instances, as in fainting-fits, consciousness is not lost. The following list includes the various forms of convulsions with their associate phenomena. Only those are mentioned which occur suddenly. For their symptomatology and diagnosis the appropriate sections on special diagnosis must be consulted.

1. **Unconsciousness.** *a.* **Syncope.** The attack, which may be preceded for a few moments by yawning, sighing, or nausea, is sudden and short. The face is pale but calm, the pulse feeble or imperceptible, the extremities cool. The breathing is quiet during the attack. The pupils are dilated and respond to light. There is no pain.

b. **Cerebral Disease.** Spasm sometimes is associated. Head pain, congested face, hemiplegia, facial palsies, irregular and irresponsive pupils, insensitive cornea, incontinence of urine.

c. **Intoxications.** Alcohol, opium, and other narcotics; uræmia, diabetes, toxæmia from infections, sun-stroke.

d. **Coma.** Coma is a condition of complete unconsciousness, with suspension of perception and volition. Sensation and motion, however, are not always abolished. Coma always is of grave significance, indicating a serious disturbance of the cerebral functions; it occurs in a number of conditions, and presents different symptoms according to the cause; it

may arise from (1) injury to the head, (2) disease of the brain or spinal cord, (3) the action of poisons, or (4) auto-intoxication.

Injuries to the Head. These injuries may produce *laceration of the brain-substance*, with *cerebral or meningeal hemorrhage*. Coma resulting from such an injury is usually recognized by the history and the visible signs of a wound. A head injury in a comatose person, however, may be the result of a fall due to a sudden onset of unconsciousness.

Diseases of the Brain and Spinal Cord. Coma may be due to hemorrhage into the brain (apoplexy); to epilepsy; to cerebral embolism or thrombosis; to thrombosis of the cerebral sinuses; to cerebral abscess; to pachymeningitis, leptomeningitis, or cerebrospinal meningitis; to cerebral syphilis; to general paralysis; or to multiple sclerosis.

In a given case of coma the ætiological diagnosis is based on the history, if it is obtainable, and on the symptoms, which are fully described in the special part, in the sections devoted to the different diseases.

The Action of Poisons. Coma may be produced by the influence of certain poisons: alcohol, opium, chloral, cannabis indica, the bromides, and other narcotics, when taken in toxic doses.

Alcohol. The *coma* of acute alcohol-poisoning is *profound*, and is accompanied by *great muscular relaxation*. In the early stages the vessels of the face and neck are engorged, and the skin is moist and warm; later, the face is pale and ghastly, and the skin becomes cold. The *pupils* usually are moderately dilated and do not react. The *pulse* is rapid, strong at first, then more and more feeble. The *respiration* is stertorous and heavy. The *sphincters* usually are not relaxed. The *body temperature* is subnormal.

Opium. The patient falls into a deep sleep, from which he usually can be aroused momentarily by loud shouting or violent shaking, and which is succeeded by the profound coma of opium-poisoning. The *face* is red and often cyanotic at first, later becoming pale and livid. The *pupils* are contracted to the size of a pin-point. The *skin* is dry until the approach of death. The *breathing* is slow, deep at first, then very shallow. The respirations are 8 or 10, and finally only 4 or 5 in the minute.

Chloral. The coma of chloral-poisoning follows the deep sleep caused by the drug. It is deep, and the patient cannot be aroused. Absolute muscular relaxation exists. The *face* is white and livid; the *skin* is covered with a cold sweat. The *pupils* at first are contracted, but soon become widely dilated. The *pulse*, while perhaps a little slowed at first, soon becomes rapid and thready, finally being imperceptible. The *respirations* are slow and labored at first, and then are shallow and feeble.

Cannabis Indica. The coma following toxic doses of this drug resembles that occurring in acute alcohol-poisoning. The *pulse*, however, generally is fairly strong. Before the onset of the coma there is a peculiar sense of prolongation of time, of which the patient may have complained.

Auto-intoxication. Coma due to auto-intoxication may occur in the course of Bright's disease from uræmia, diabetes, the infectious fevers, pernicious malaria, acute yellow atrophy of the liver, Addison's disease, or heat-stroke.

The symptomatology of these conditions is discussed under the respective heads. The coma of the infectious fevers is produced gradually, and ordinarily is preceded by insomnia, by delirium, or by other signs of cerebral disturbance. The coma sometimes is due to a complication, such as the occurrence of an abscess or effusion at the base of the brain in the course of typhoid fever.

2. **Convulsions.** *a.* **Epilepsy.** (1) "Haut mal": aura, convulsions—(*a*) tonic, respiratory muscles affected, face livid, stupor follows; (*b*) clonic, tongue bitten, stupor follows. (2) "Petit mal": sudden pallor, no convulsions.

b. **Infantile Convulsions.** Usually reflex from indigestion; may be the onset of a specific fever or due to high temperature.

c. **Puerperal Convulsions.** Headache, amaurosis, œdema, suppression of urine and albuminuria; clonic convulsions, tongue bitten, complete coma; history. (See Uræmia.)

d. **Uræmia.** Unilateral or bilateral clonic convulsions. (See Renal Disease.)

e. **Alcoholism and Sun-stroke.**

f. **Organic Brain Diseases** (syphilis, tumor, softening, etc.).

g. **Convulsions without or with only Partial Loss of Consciousness.** Hystero-epilepsy, focal or Jacksonian epilepsy, hysteria, cerebral embolism, thrombosis, hemorrhage, spasms of various kinds.

h. **Convulsions with Vertiginous Movements.** The forms of vertigo are gastric, aural and labyrinthine (Ménière's, also paroxysmal), ocular, cerebellar, from congestion of the brain (reflex), epileptic.

3. **Collapse.** Collapse may occur in a person in apparent health and be the first indication of disease, as in rupture of a large bloodvessel causing internal hemorrhage; or it may occur in the course of disease, as typhoid fever, when intestinal hemorrhage takes place.

The symptoms are those of prostration, with partial loss of consciousness, or the mind may be perfectly clear. The face is pale, pinched, and bathed with perspiration. (See Hippocratic Facies.) The skin is wrinkled and cool and clammy. The hands are cold. The eyes are sunken and encircled by dark rings. The voice is weak or suppressed. The pulse is rapid and thready, or may be absent at the wrists. The heart-sounds are indistinct. The temperature falls below the normal. The respiration may be hurried or shallow, sighing and gasping. The urine is scanty or may be absent.

Collapse may be due to severe pain from any cause; to hemorrhage, external or internal; to perforation of abdominal viscera; to peritonitis; to excessive watery discharge, as in cholera or serous purging. It may be due to pernicious malarial fever, and is then attended by coma. It may mark the termination of an infection, notably pneumonia. Sudden pneumothorax, embolism of the pulmonary artery, or rupture of a pulmonary abscess is attended by collapse. It may be present in the terminal stage of ulcerative endocarditis, and is not unusual in acute pancreatitis.

4. **Shock** is a condition in which the vital powers are blunted or stunned, with or without mental terror or anxiety. It is likely to be seen after injury, surgical operation, hemorrhage, angina pectoris, severe pain

from any cause, any sudden cerebral or spinal lesion, undue mental and emotional strain. Its presence points to a grave antecedent condition, near or remote. The symptoms are those of collapse.

5. Vertigo. In vertigo the patient feels as if his head were swimming; or as if he were whirling around, or were falling forward or backward—*subjective vertigo;* or as if all surrounding objects were whirling around him or rising up toward him or pulling away from him while he himself remained stationary—*objective vertigo.* Vertigo is a symptom of many disorders, and usually accompanies any disturbance of the circulation within the cranium. It may occur in disease of the heart, liver, kidneys, stomach, bloodvessels—arteriosclerosis, cerebral hemorrhage or apoplexy, cerebral embolism and thrombosis—blood, nerves, brain, spinal cord, ear, and eye; in disturbance of equilibrium; after the taking of certain drugs; and after applications of electricity. The presence of a foreign body in the ear, the insufflation of air into the Eustachian tube, and disease of the labyrinth and of the auditory nerve may give rise to vertigo. Ménière described a disease associated with paroxysmal vertigo. Paralysis of the ocular muscles, eye-strain, and astigmatism may be accompanied by vertigo. Laryngeal vertigo is often closely associated with epilepsy. The larynx is usually normal in this affection, but in one case it contained a polyp. Tickling or burning in the larynx usually precedes the vertigo. The vertigo which develops in the sequence of infectious diseases probably results from the action of the toxins. This is the case in the vertigo of *malaria* and in the *paralyzing vertigo* of Gerlier. (See page 191.)

Reflex Vertigo. Vertigo is probably of reflex origin when it occurs in disease of the heart, stomach, intestines, nose, teeth, larynx, as well as of the sexual organs.

Toxic Vertigo. This occurs after the ingestion of large doses of quinine or salicylates, and after excessive indulgence in alcohol, tobacco, coffee, tea, or opium.

Electric Vertigo. This is caused by galvanization of the skull and adjacent parts.

Functional Vertigo. This includes the vertigo that occurs in sea-sickness and mountain-sickness, the dizziness felt when looking down from a height or at running water, and on jolting, swinging, or revolving.

This apparently hasty view has already given the practitioner much information. He then notes with more deliberation (1) the personal appearance; (2) the apparent age; (3) the temperament and constitution of the patient or the evidence of any diathesis or cachexia; (4) the position assumed in standing, walking, or in bed; (5) the general form and nutrition.

CHAPTER XIX.

GENERAL EXAMINATION OF THE EXTERIOR.

1. The Personal Appearance.

From the general appearance the patient's habits as to industry, neatness, or care of dress may be observed; these habits are of diagnostic importance, particularly in brain affections. The appearance also shows frequently whether the patient is addicted to alcohol or to the use of narcotics. Moreover, the slit boot to relieve the swelling of gout, the loosely fastened boots from swollen ankles, the unduly worn sole as in spastic paralysis, the unbuttoned waist-band because of dropsy or increased weight, the stained trousers from drops of urine, are seeming trifles, but of diagnostic value.

The occupation of the patient is often important in throwing light upon his disease: the brown, weather-beaten face of the farm laborer, sailor, or driver contrasts strongly with that of the merchant, clergyman, or clerk. A machinist can often be recognized by his grimy, oily hands. All this information can be obtained at a glance, and many details can be added before the patient has taken his seat in the consulting-room.

2. The Apparent Age.

The apparent age of the patient should be estimated from his appearance, and compared with the actual age when this is learned later. In this way the physician will be able to judge whether the patient is aging too rapidly or bearing his age well. An obvious advantage of noting the patient's age is that it enables us at once to exclude a large number of diseases which do not occur in the period of life to which the patient belongs. For example, if the patient is a child, we need not consider the chronic degenerations nor the visceral cirrhoses, which appear in middle and later life. Conversely, in an old person we do not expect to meet with the exanthemata which affect children almost exclusively. So, too, typhoid fever and pulmonary tuberculosis are more common in adolescence and early manhood than in childhood and old age. Again, in very young girls, the question of menstruation and its difficulties never has to be considered. *Gray hair* in a person under thirty-five generally indicates a feeble constitution and premature age. *Loss of hair* is not significant, for, apart from a tendency to baldness which is very marked in some families, professional men who do much brain-work, especially in hot, close rooms, are apt to become bald much sooner than other men. The presence of *wrinkles* at the corners of the eyes and of "crow's feet," and of dull, dry, lustreless eyebrows, should be noted as indicating aging, whether the person has lived long or not. In women approaching forty,

who do not gain in flesh, there is often a suggestive prominence of the angles of the jaw and sternomastoid muscles, with a certain loss of roundness and elasticity of the cheeks. The latter appearance, however, may be due to the loss of the molar teeth.

3. The Temperament and Constitution of the Patient.

Emphasis was formerly laid upon appearances pointing to a particular diathesis or type of inherited constitution. Five varieties of diathesis were described: the gouty or sanguine-arthritic, the strumous, the nervous, the bilious, and the lymphatic. While groups of individuals may be classified under one of these diatheses, it is well not to lay too much stress upon them for diagnostic purposes. As pointed out by Gairdner, it is not proper to designate the diathesis off-hand. Individual appearances should be carefully noted, so that after the examination a final conclusion as to the diathesis may be drawn.

In the *gouty* or *sanguine* diathesis the osseous system and muscles are well developed, the nutrition active, and the patient usually robust in appearance. The digestion is good, the respirations are deep, the circulation is well carried on (as shown by the florid skin and the large heart), the pulse is firm and steady, and the pressure in the arteries is high. The head is large and the jaw prominent; the teeth are good. The hair is of strong growth. The individual with this diathesis is predisposed to the arterial changes of advancing age. Apoplexy, aneurism, and angina pectoris, or complications resulting from the senile changes in the heart and arteries develop.

In the *strumous* diathesis the bones and the glandular system are changed and the appearance of the face is rather coarse; the bones of the chest are small; the long bones are slender, while their epiphyses are large; the forehead is broad and prominent, the lips full, the alæ nasi thick, the teeth carious, the lower jaw light and thin, the hair fine and often of a light hue, the eyelashes long, the eyebrows arched and often heavy. In this diathesis the nutritive changes are poor, inflammations are usually sluggish; disease of the bones, of the glands, and forms of tuberculosis are apt to be more severe.

In the *nervous* diathesis we see small, active, restless beings, with small bones and large muscles. They are full of energy, and carry on large business operations or other ambitious undertakings. The features are well formed, the eyes active. Such types readily become the victims of over-work, dyspepsia, and early breaking-down of the nervous system. They possess idiosyncrasies toward drugs, particularly opiates.

In persons of the *bilious* diathesis we find a dark skin, dark hair, muddy conjunctivæ. They are usually not well nourished. Their digestion is poor, and they are subject to attacks of so-called biliousness. Sick headaches are common. Fatigue is not borne well.

In the *lymphatic* diathesis there are lack of energy and sluggishness of nutritive processes; such persons are unable to keep up in the wear and tear of life. They are usually pallid and have soft muscles. (See Lymphatism.)

In addition to diathesis *cachexia* is also noted. Cachexiæ arise from the ravages of disease, especially when the number of the red cells of the blood is reduced and the hæmoglobin diminished. Cachexiæ are caused especially by syphilis, gout, and chronic malarial poisoning. In cancer of some parts of the digestive apparatus—and, indeed, in all forms of chronic disease of the digestive tract—a cachexia develops. The anæmia from poisoning with lead, arsenic, and other metallic poisons produces an appearance to which the term cachexia has also been applied, although in truth it only resembles that state. Each form of cachexia takes its name from its cause, as the syphilitic or the cancerous cachexia.

4. Stigmata of Degeneracy.

As a result of the careful studies of Lombroso and others we have learned that certain alterations in the structure of the body are frequently associated with disturbances in the mentality of the individual, or the capacity of resistance and assimilation of the individual tissues. Naturally the significance of these stigmata varies considerably in different individuals, and they should be regarded as suggestive rather than decisive.

Anomalies in the general growth include dwarfism and giantism. *Dwarfism* may be of various types, the most important being the true dwarf, in whom all portions of the body are too small; the rhachitic dwarf, in whom distinct anomalies in the skeleton occur; and the *cretin* or dwarf of congenital myxœdema. The two last-named varieties are really types of disease. *Giants* also may be of two types: the true giant and the acromegalic giant. The modification of growth may affect only certain parts of the body. The head may be abnormally small or large without any distinct evidence of disease. A more characteristic stigma is excessive elongation of the arms, approximating the simian type, or imperfect development of the lower portion of the body: small, thin legs associated with a well-developed torso. Not infrequently we notice in otherwise well-developed persons that the chest is abnormally narrow—that is, the thorax as a whole is poorly developed. This may be associated with the congenital hypoplasia of the cardiovascular system described by Virchow.

The more pronounced forms of degeneracy can not but affect the health of the individual. They consist in alterations of the ears: absence of the lobe; absence of the antihelix; position of the external ear with relation to the head, so that it stands out almost perpendicularly; presence of a small point upon the helix, a vestige of the animal type; adherence of the lobe to the side of the head. Abnormalities of the eyes: they may be unusually close together or far apart; placed at different levels; they may present various changes in the ocular muscles. Even the growing together of the eyebrows is looked upon as a slight stigma of degeneracy. Excessive breadth of the nose, imperfect closure of the nostrils, marked deviation of the septum—all these anomalies are included in this category. In the mouth: various irregularities, such as wide separation of the teeth; abnormal development of the canines; the prognathic or agnathic jaw; high arching of the palate; cleft palate—are all found more frequently among persons otherwise degenerate than in normal individuals. In the

hands: supernumerary digits; exaggerated webbing of the fingers, or the so-called cleft hand—that is, separation of two of the metacarpal bones—have been noted. Similar anomalies have been described in the feet. Hypospadias and epispadias, abnormally small size of the external genitalia, are supposed to be quite characteristic features. Anomalies of the hair consist in abnormal growth upon the forehead, upon the back, following the course of the spine, or excessive irregularities of the hairy growth on other parts of the body. Delayed puberty, or the persistence of the infantile, or development of the contrasexual type—that is, the feminine type in men, and the masculine type in women—are also examples of degenerate tendencies.

5. The Attitude and Gait of the Patient.

The *attitude* of the patient gives information as to his physical vigor, and, to a certain extent, of his alertness of mind. A man vigorous of mind and body will stand firmly upon both feet, with back straight, shoulders square, and head erect. When one is depressed by care or disease, the shoulders have a tendency to droop and the head to fall forward. Indecision and a vacillating disposition are sometimes indicated by the patient standing first on one foot and then upon the other while talking, or by an unsteady look from the eye.

When one shoulder is lower than the other and the patient is of phthisical build, pale, and emaciated, the attitude is strongly suggestive of phthisis or chronic pleurisy on the side on which the shoulder is depressed. Sometimes, in acute pleurisy, the patient will walk with the shoulder depressed and the arm firmly pressed against the affected side so as to restrict its movements as much as possible.

Decubitus. The attitude of the patient in bed is often significant. He may assume the *active dorsal*, or the *side position*, with the body arranged so that it is comfortable and unconstrained. Then slight indisposition only is present. On the other hand, the *side* position, the *dorsal* position, or the *upright* or *semi-upright* position may be assumed.

To the close observer the attitude of a patient in bed is sometimes reassuring. He lies easily upon his back, or turned slightly to one side with the arms uncovered, and may even turn or sit up to greet the physician as he enters the room—all these signs point to moderate illness or to the approach of convalescence.

Side Position. A patient with acute pleurisy or pneumonia will lie on the affected side, so as to limit its motion as much as possible and at the same time to allow the healthy side full liberty to expand. The breathing will be shallow and frequent, the expression of the face anxious, and occasionally a spasm of pain contracts it as the patient coughs or is obliged to take a full breath. If effusions are present, by lying on the side of the effusion pressure is removed from the heart and the unaffected lung—an obvious advantage.

At times, in case of thoracic aneurism, if situated on one side, or of movable thoracic tumors, the patient will lie on the side which is the seat of the disease.

Dorsal Position. The dorsal position, as assumed in health or slight disease, has been referred to. When the position is assumed in grave disease it is called *passive* dorsal, because it is often assumed without volition of the patient.

In grave cases of typhoid or other low fevers the patient lies upon the back and shows a marked tendency to slip down in the bed. The expression of the face is heavy or vacant. The lips and teeth require constant cleansing to keep them free from sordes; the tongue is dry and glazed or covered with sordes; the tendons of the wrist twitch convulsively, and the patient lies with open or half-open eyes (coma vigil), picking at the bedclothes or at imaginary objects which float before his eyes.

A healthy baby a few months old finds motion an almost ceaseless delight. It will lie on its back, kick up its feet, play with its toes or some object that attracts it, crowing, wriggling, squirming. In *rhachitis*, on the contrary, the little patient lies as quiet as possible, even refraining from crying, because all motion is painful. In *cerebrospinal meningitis* the head is drawn backward and downward, and the muscles at the back of the neck are rigidly contracted.

In *acute disease* involving the peritoneum or neighboring organs, such as acute peritonitis, appendicitis, or endometritis, the patient lies on the back with the legs flexed upon the thighs and the thighs upon the abdomen. Motion is avoided as much as possible, and so is any pressure upon the abdomen.

Lateral Position. The lateral or dorsal position, with legs drawn up and trunk and head bent over to meet them, is assumed, with groans of pain and possibly involuntary bearing-down, in hepatic and intestinal colic and during the throes of labor.

The Semi-upright or Upright Sitting Position. In an acute attack of *asthma* the patient is found sitting up in bed, or in a chair, possibly by an open window. The expression of the face is anxious, the skin dusky or pale, and moist. The breathing is loud, noisy, and scraping. The demand for oxygen is imperative, difficulty is experienced in inspiration and expiration, not sufficient air for physiological purposes being able to enter the alveoli; expiration is prolonged and labored (expiratory dyspnœa). The patient sits with the chin raised and the head erect, the hands grasping the arms of a chair or the bedclothing, so that, by fixing the chest, the accessory muscles of respiration can be of the greatest assistance in supplementing the diaphragm. In emphysema, in its late stages, or when complicated with bronchitis and asthma, the same position is assumed almost constantly.

In *pericarditis* with effusion, in *large pleural effusions*, and in *advanced heart disease* with anasarca the patient is unable to lie down on account of the smothering feeling which the recumbent position induces. In pericarditis the expression of the face is extremely anxious, the patient having a dread of impending death.

When there is a large pleural effusion, the expression is not usually so anxious, but the dyspnœa may be intense. The patient is propped up in bed, leaning slightly to the affected side, and devotes all his energies to breathing, avoiding every exertion, such as moving, answering questions,

or coughing, which taxes his breathing-muscles still more. One side of the chest may be observed to move violently while the other is motionless.

In heart disease and anasarca dyspnœa frequently amounts to *orthopnœa*. The patient may be found propped up in bed or seated in a large rocking-chair, some patients finding greater comfort in the latter. The face is pale, livid, or jaundiced, and may be swollen, while the cellular tissue throughout the body is œdematous, and the cavities, especially the peritoneum, are more or less filled with fluid. In diaphragmatic pleurisy the position assumed is very characteristic—the erect sitting posture, with the body leaning forward and laterally, to relieve the pain.

The Prone Position. Rarely the patient is found lying upon the abdomen. He assumes this position because it gives relief to abdominal pain or to colic of any form. Owing to the change in the relative positions of the organs brought about by this posture, the pain of an ulcer of the stomach, of aneurism, or of caries of the vertebræ may be mitigated.

In tetanus *opisthotonos* occurs. The body is supported on the head and heels and the trunk is arched upward, because of tonic contraction of the spinal muscles. In strychnine-poisoning with tonic convulsions the same position may be assumed.

Emprosthotonos, side-vault position, is occasionally assumed in tetanus and also in strychnine-poisoning.

Unclassified Positions. Irregular or bizarre positions are usually assumed in affections of the nervous system, particularly in hysteria.

Restlessness. Often the patient is unable to assume a position, or at least to remain fixed in any position. This may occur on account of pain, or because of irritation or anæmia of the nerve-centres. In cases of moderate *cerebral hemorrhage* and of *shock* there is great restlessness. The patient is restless without the appearance of agitation. In profuse *hemorrhage*, whether uterine, intestinal, or pulmonary, the resulting cerebral anæmia manifests itself in restlessness, with sighing and gasping. The pallor, the quickened pulse, the great thirst, with the history of bleeding, are sufficient to explain the restless state. In *chorea* there is more than restlessness—there is constant twitching of muscles with jerking from one side of the body to the other. The patient does not keep the covers on when in bed, and by her jerky movements often does herself considerable injury.

In *cerebral meningitis* the patient tosses from side to side or lies with the head retracted and pressed deeply into the pillow. The eyes are injected, the pupils contracted, and frequent sharp cries are uttered especially if the patient be a child.

In *hysterical convulsions* the patient, usually a young woman, tosses wildly to and fro, screaming, laughing, or crying; or coma may be mimicked. The moods often change with great suddenness. The appearance is very alarming at first sight; but the pulse and breathing are not much accelerated, there is no fever, and the patient is sufficiently conscious not to injure herself even to the extent of biting the tongue.

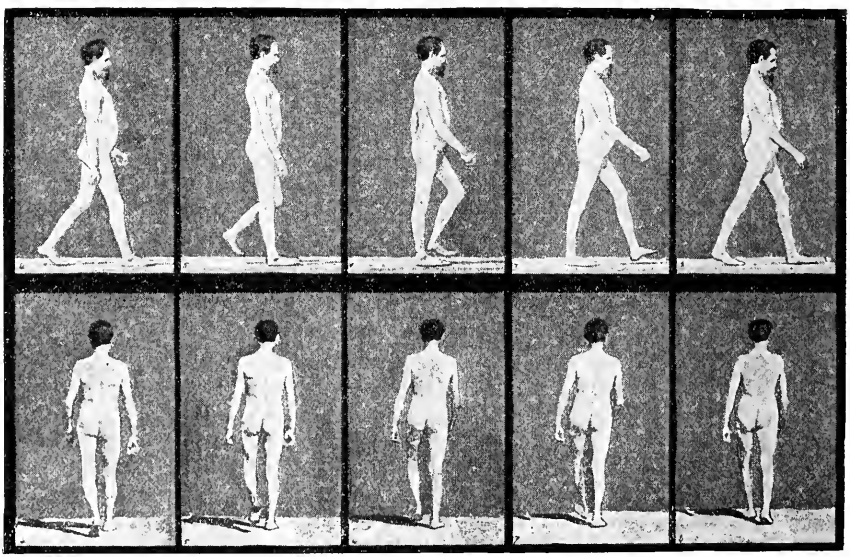
Gait. The gait is sometimes characteristic. (See Nervous Diseases.) The *hemiplegic* patient advances the sound limb, and then brings the other up to it by lifting the pelvis and swinging the paralyzed limb around by

a movement of circumduction. The shoe is worn down at the toe in an irregular way. Sometimes the shoulder on the sound side is thrown outward and forward, so as to facilitate the raising of the pelvis on the paralyzed side in order that the limb may be circumducted. The arm may be rigid, or bent at the elbow, the fingers being flexed upon the palm and the thumb turned in.

In *locomotor ataxia* there is uncertainty in the gait, which may only be felt by the patient or be apparent to the observer also. There is irregularity in the line of progression, or the movements become very jerky and erratic. As there is very little motion at the knee, because it is spasmodically braced, the pelvis is slightly tilted until the foot is released; the foot is then raised unnecessarily high, jerked rapidly forward and outward and brought down with a sudden stamp, or flail-like action, on the heel. The patient's centre of gravity undergoes several changes at each step, so that he swings from side to side. He cannot walk in the dark, and at a later stage requires the aid of canes to prevent him from falling forward.

In *paralysis agitans* the attitude and gait of the patient are peculiar. The head and body are thrown forward and fixed in that position; the

FIG. 13.



Gait in a case of locomotor ataxia: instantaneous serial photographs. (MUYBRIDGE and DERCUM.)

arms are slightly abducted and partly flexed, the hands being in the position in which a pen is held or a pill rolled. The legs are also bent at the knees. Rhythmical tremors affect the hands first and then the rest of the body, the head and neck usually escaping. The gait is *festinating* —that is to say, each step becomes more rapid than the preceding, until the patient is prevented from falling only by catching hold of something.

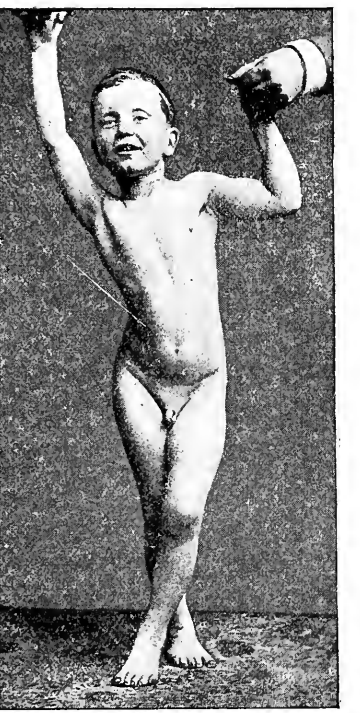
The tremors cease during sleep, and are independent of voluntary motion. (See Fig. 14.)

In *spastic paraplegia* the patient walks with two sticks. He leans on the left one, arches the back, and then lifts the pelvis and the right limb as far from the ground as possible, but cannot quite clear it. The leg is rigid and the foot dragged around in a semicircle. The toe has a marked tendency to stick to the ground, and is brought forward with a scraping sound. The knees have a tendency to interlock, and the foot that is brought forward is apt to cross in front of the other.

FIG. 14.

Side view of a case of paralysis agitans, showing forward inclination of trunk. Tendency to propulsion. (DERCUM.)

FIG. 15.



Spastic paraplegia, cross-legged progression. (DERCUM.)

In *disseminated insular sclerosis* the gait is somewhat jerky and resembles the gait of ataxia or of tumor of the cerebellum. Of course, the disease that causes such peculiarity in gait cannot be established without first observing the mental and nervous phenomena that attend such affections.

In *hysterical paraplegia* there is sometimes complete loss of power of standing or of walking. The patient falls if an attempt is made to compel her to stand; or she walks with the knees and the hips semiflexed or in awkward attitudes, implying greater muscular exertion than necessary for the normal gait. The condition is recognized by the fact of

its occurrence in young subjects in whom other striking phenomena of hysteria are observed.

Cross-legged Progression. This form of gait is seen in children with

FIG. 16.

Typical pseudohypertrophic muscular paralysis. (DERCUM.)

FIG. 17.

Hysterical astasia-abasia. (LLOYD.)

spastic paraplegia, and occurs because of contracture in the calf muscles. When the child begins to walk, one foot gets over in front of the other. Sometimes a swinging oscillation of the body occurs, which may persist throughout adult life. (See Fig. 15.)

The gait of *pseudohypertrophic muscular paralysis* is known as the waddling gait. This oscillating character is assumed in order that the body be so inclined "as to bring the centre of gravity over each foot on which the patient successively throws his weight, because the weak gluteus medius can not counteract the inclination toward the leg that is off the ground unless the balance is exact." (Gowers.) The *position* assumed in getting up from the floor, as described by Gowers, is pathognomonic. The patient turns over in the all-fours position, raises the trunk with his arms, rests the trunk upon the extended hands, then extends the knees, pushes back with the hands

until he can grasp one knee with the corresponding hand, then grasps the other knee, and pushes up the trunk by gradually raising the point of support for the hand upon the thigh. (Fig. 16.)

The *swaying* gait, like that of a drunken man (cerebellar titubation), is significant of cerebellar disease. (See Station.)

Feebleness of the gait attends general paresis and the early stage of chronic myelitis, but, of course, is of no significance unless it is attended by other symptoms of these affections.

The gait of *paramyoclonus multiplex* and that of *Thomsen's disease* are also peculiar. (See Muscles.)

Station. **Astasia** and **abasia** are terms employed to define the loss of power of standing and of walking, respectively, without paralysis. (See Fig. 17.) Hysteria is the most common cause.

Ataxic astasia in tabes dorsalis. The inability to stand is observed either with (1) the eyes closed, or (2) with the eyes open and the toes and heels in contact, or (3) with the eyes open and the feet apart. The latter occurs in the highest degree of ataxia, and may be followed later by complete loss of power of standing.

Swaying. If a healthy person stands with the eyes shut and the feet close together, the body will sway slightly. In a patient with locomotor ataxia this swaying is greatly exaggerated.

In *pseudohypertrophic paralysis,* when the patient stands, there is that extreme curvature of the spine known as lordosis. It disappears entirely when the pelvis is supported, as in the sitting posture. In the later stages of this affection there is posterior or lateral convexity of the spine with astasia.

In the paroxysms of *Ménière's disease* loss of the power of standing may be absolute. The patient may be hurled to the ground and be quite unable to rise or sit up. The nature of the paroxysm may be suspected from the sudden onset and the complaint of vertigo, together with the ear symptoms which attend this affection.

In disease of the *middle lobe of the cerebellum,* swaying from side to side in wide oscillations is observed. The appearance is like that of a drunken person. While the walk is peculiar, the patient can usually sit up.

6. General Form and Nutrition.

The general form and nutrition of the body are estimated by the color of the skin, the amount of subcutaneous fat, the degree of muscularity, and the size and shape of the osseous system. Hence, we estimate the degree of physical development of the individual by the *size* and *weight,* by the nutrition of the muscles, and by the state of other tissues. To recognize lack of development is often to be able to explain phenomena of a functional nature which otherwise could not be accounted for. The color will be considered in the chapter on the Skin.

It is extremely important that these observations should be made, particularly in childhood and adolescence. Not only are marked departures from the normal significant, but slight deviations point to the occurrence of processes which modify nutrition. Unless lack of development is detected, it is frequently impossible to explain the occurrence of some

functional disorder, as neuralgia, or of derangement of the viscera, or of indefinable ill health, as the result of which the patient shows inaptitude for exertion or inability to conduct the usual affairs of life. The recognition of malnutrition, as shown in lack of muscle-tone or diminution of weight, is often sufficient to point the way to successful treatment by hygienic methods.

Size. Size affords some information as to the degree of development of our patients and as to the class of diseases to which they are most liable. While there is no absolute standard by which to compare the relative proportion of height to girth in individual cases, there is a type generally recognized as being usual, and variations from it give rise to such expressions as *stout*, *spare*, *slender*, *thin*, *tall*, and *short*.

Stout usually expresses an increase in girth and a moderate excess of flesh over the normal. When used in this sense it becomes synonymous with lusty, and indicates an increase of flesh which is well distributed and due to healthy, active nutrition without impairment of physical activity. In some cases, especially in women, stoutness is used as a euphemism for corpulency, but not often for that excess of fat properly called obesity. Stoutness, in the sense of lustiness, up to middle life is an indication of physical and often of mental vigor. It is often found in gouty and rheumatic subjects.

A tendency to take on flesh after the age of forty-five, especially if the person's occupation is sedentary and his habit of body inactive, is not to be regarded as favorable. It may be compared to a warrior persisting in wearing an increasingly heavy weight of armor after the campaign is over. Increased weight under such circumstances is not increased strength but increased burden, and the burden becomes greater with advancing years. Those who are under forty and stout, in the sense of having too much fat in proportion to bone and muscle, bear fevers and exhausting diseases badly. Fat subjects after middle life, and to an increasing degree after that period, are liable to fatty degeneration of the heart, bloodvessels, and other important viscera. Women at the menopause are very prone to take on flesh rapidly.

Persons who are tall and thin, with a long, narrow thorax, especially if they have become tall rapidly after puberty, are commonly looked upon as delicate, and as especially liable to consumption. There is reason for this view; but if they live to be twenty-five without contracting any disease of the lungs or pleura, they may then live to a great age.

Some patients have an appearance which is well described and understood by the word *spare*. The figure is compactly put together, with small bones and a scanty allowance of fat, and there is a tendency to leanness rather than to roundness of form.

In still others muscle and bone predominate, and the figure is apt to be angular, as in those described as *wiry*. They are often possessed of great muscular power and resistance to strain. Those of spare and wiry habit bear disease very well. Inspection alone may leave one in doubt whether to regard an individual as thin and delicate, or spare. Light will be obtained from the patient's occupation and the amount of physical exertion of which he is capable, and also from the tonicity and hardness of

his muscles. If one stops to think a moment, he will see that, for the same amount of heart and lung power, a man will be better off if spare than if corpulent; because in the latter case he has an additional load to carry, and has to nourish and maintain a thick blanket of fat from which he derives no adequate advantage. Hence, a person of spare build who survives childhood and adolescence without disease probably has, on the whole, a better prospect for long life than a stout person.

Normal Habit. In estimating the patient's size or weight it is important to ascertain if he has a regular habit of taking on flesh at certain periods of the year, for instance, or if the obesity has developed suddenly or followed acute disease.

Change in Size. Change in size may be *general* or *local.*

General Change in Size. When the entire body is altered in size, the change may be due to increase or diminution of the muscles and fat, singly or combined. When large accumulations of fat take place, the word *obesity* is applied to the condition. The estimation of the patient's size as compared with his weight is usually based upon the amount of subcutaneous fat. General accumulation can readily be recognized by rotundity of the exterior. Over-stretching of the skin may cause lines upon the buttocks, abdomen, and thighs, like the lineæ albicantes of pregnancy.

The word *emaciation* is applied to excessive atrophy of fat and muscles. The skin becomes loose, wrinkled, and is readily raised in folds. If it is accompanied by great exhaustion and apparent loss of fluid, the word *marasmus* is employed.

Local Change in Size. Increase or diminution in size may be local. It is not to be forgotten that accumulations of fat may take place in special portions of the body; the *abdomen* is the favorite seat for excessive accumulation, particularly in women and in men of sedentary life, with habits of excessive indulgence in food and drink. When one part is increased in size and another is growing progressively smaller, the disparity indicates disease (see below).

The face may be thin and even much emaciated, while the abdomen is greatly distended from dropsy or from tumors of the various abdominal viscera or glands. The head is much increased in size in chronic *hydrocephalus*, while the face remains small.

Local changes in size of the head, face, thorax, abdomen, extremities, and other parts are discussed in the sections devoted to these regions.

Weight. Nothing has yet been said of the weight, but as it affords a precise estimation of the size, particularly if considered in relation to the height and age, the following discussion will include both size and weight.

While the eye can estimate approximately the weight of the body and the degree of emaciation, the physician should make it a rule to ascertain the weight accurately by means of scales. Machines are now made which can be used for weighing the patient and at the same time noting the exact height.

The relation of body-weight to height is of importance. It is also important to know the average weight of the individual at different periods of life. The progressive increase in weight which should take

place after birth should be remembered, as the opposite is positive evidence of malnutrition.

Hutchinson's table enables us to judge the average weight of a healthy man of a given height:

A man	of	4	ft.	6	in.	to	5	ft.	0	in.	ought to weigh about	92.26	pounds.
"	"	5	"	0	"	5	"	1	"	"	"	115.52	"
"	"	5	"	2	"	5	"	3	"	"	"	127.86	"
"	"	5	"	4	"	5	"	5	"	"	"	139.17	"
"	"	5	"	6	"	5	"	7	"	"	"	144.29	"
"	"	5	"	8	"	5	"	9	"	"	"	157.76	"
"	"	5	"	10	"	5	"	11	"	"	"	170.86	"
"	"	5	"	11	"	6	"	0	"	"	"	177.25	"

In some life insurance tables in this country the average weight for the height is lower, especially in persons over five feet ten inches.

Loss of Weight. The question of *weight* is an important one in disease. The whole body may exhibit considerable loss of flesh, the cheek bones and temporal fossæ being distinctly visible, the muscles soft, the limbs wasted, and the subcutaneous fat diminished. As has been stated, persons with an excess of fat do not bear fevers and exhausting processes so well as those who have a relatively larger proportion of firm muscle tissue. It is particularly important to note the weight from time to time. In the course of *wasting disease* we learn the effects of treatment, or, on the other hand, the march of disease in spite of treatment. In obscure cases, as in suspected *tuberculosis*, persistent loss of flesh is a grave diagnostic and prognostic sign. After acute disease, if the patient is weighed every week, the onset of insidious sequelæ; as tuberculosis, may be detected. It is important to notice not only whether flesh has been lost or not, but also how much and for how long a time the loss has been going on. Such facts furnish a clue not only to diagnosis, but to treatment as well. Flesh is lost in almost all diseases, acute or chronic; but it becomes of special diagnostic moment in the latter. It is most noticeable in *tuberculosis, cancer, marasmus, cirrhosis of liver* and *kidneys, diabetes,* in *anæmias,* and in cachectic conditions due to *prolonged suppuration* or *chronic diarrhœa,* in *gastric neurasthenia* and *anorexia nervosa.*

Remember, if emaciation is present, to ascertain its degree, its rate of development, and its possible relation to unusual mental care or to acute disease. Slow progressive emaciation is of serious moment. At any stage of phthisis a maintenance of the body-weight is one of the most favorable elements in prognosis.

Again, while loss of weight attends all the diseases of the digestive tract which interfere seriously with nutrition, it progresses more rapidly and steadily and attains a greater degree, in malignant disease than in the structural or functional diseases. Hence, the question of loss of weight is important in deciding between chronic catarrhal gastritis and gastric carcinoma. But still more important is the question of the time during which loss of flesh has been taking place, and whether it has been progressive or interrupted by periods of gain in weight. If during two or three years the patient has been vomiting occasionally and losing flesh, but gaining again from time to time, it is much more significant of gastric catarrh than of gastric cancer.

False Increase of Weight. In certain cases of great anasarca, in malignant disease of the abdomen, especially huge cysts of the ovary in women and sarcoma of the kidney in children, there may be actual increase of weight due to the accumulation of water or to the new growth, though the rest of the body is manifestly emaciated.

Weight in Children. In babies and children fat is more likely to be a sign of good health than in adults. Nevertheless the quality of the flesh is to be taken into consideration. There are fat and flabby babies and children, and there are others who are fat but whose flesh has a firm, solid feel. The former often gain and lose flesh rapidly, and when ill do not appear to have much resisting power. The size of a child gives a good idea of its nutrition. In fact, by the weight alone can we judge of the normal growth of the child. Griffith's weight chart enables one to note weekly changes. A child may have its growth stunted by improper food and unfavorable hygienic conditions, or the stunting may be the result of exhausting disease, such as whooping-cough.

Increase in Weight and Size. Increase in weight and size then may be due to changes in (1) the skeleton (see Chapter XXVII., Part I.); (2) the muscles; (3) the adipose tissue; (4) the subcutaneous connective tissue, giving rise to accumulations of serum or mucin, or to connective-tissue dystrophies (see Chapter XXIII., Part I.). *Diminution* in size is due to changes in (1) the skeleton, (2) the muscles, and (3) the adipose tissue.

7. Muscular Development.

Palpation shows whether the muscles are flabby or relaxed and large, or small, firm and elastic. It must be remembered that a person may be obese and yet have poor muscular development, or have little fat and yet a fair amount of muscle. General lack of muscular development or muscular weakness is an important sign of malnutrition and may explain the nature of many symptoms. The muscular weakness can be approximately estimated by the degree of firmness of the muscle. Weakness of the muscles of the spine, with resulting curvature or inability to keep the erect posture, is sufficient cause for the occurrence of neuralgic pains in the course of related nerve-trunks and for the displacement of organs within the thorax or abdomen, often causing functional disturbance. Various uterine displacements and functional disorders may be mitigated by toning up the nutrition of the muscles of the trunk. Forms of indigestion, sluggishness of secretions, particularly of the bowels, follow in the wake of muscular debility and pass away as the muscles regain their tone. The indigestion may not actually have taken place because the muscles are weak, although in a measure there is relation between the two conditions; but weak, flabby muscles indicate a state of the system which may develop indigestion. Moreover, weakness of the abdominal walls and separation of the recti muscles or *diastasis* favor dropping of the liver, stomach, and other organs, causing gastro-enteroptosis with its train of symptoms. The detection of muscular deficiency leads to correct lines of treatment. Atrophy of muscles occurs because of disuse, because of sedentary occupation or of a life of ease and luxury, with excessive feeding. It is sure to follow improper assimilation, as seen in extreme degree in *anorexia nervosa*.

CHAPTER XX.

EXAMINATION OF THE FACE, HEAD, AND NECK.

THE FACE AND ITS EXPRESSION.

The expression of the face, the shape of the head, its movements, the color of the hair, the state of the skin, enable one to recognize senility on the one hand, or infantilism on the other. By the expression and contour of the face we recognize the presence of adenoid disease of the nasopharynx.

The face is a mirror in which are reflected all degrees of ill health, from that which amounts only to temporary indisposition and depression up to the gravest cachexia. The face reflects also the degree of intelligence of the patient and his mental condition at the time, as well as his emotions and in a large measure his character. The face is usually a fairly good index of the temper of the individual; benevolence, amiability, and purity are written as plainly on some faces as anger, lust, dishonesty on others. (See Nose and Mouth in respective chapters in Special Diagnosis.)

The face frequently affords us valuable information concerning the health, habits, and temperament of the individual. Everyone is familiar with the bright eye and animated countenance of a friend which lead us to say, "You are looking very well to-day," and with that slight pallor, diminished clearness of the conjunctiva, with perhaps a dark circle under each eye, which lead us to infer that he is depressed or has passed a sleepless night. The face also gives unmistakable evidence of alcoholism by its bloated appearance, injected or glassy eye, dull expression, and nervousness when the patient is addressed suddenly.

Full-blooded persons, disposed to endarterial changes, frequently as the result of gout, often have at a little distance the *ruddy* appearance of blooming health. Closer inspection, however, shows that the ruddy color is due to a dilated or congested condition of the minute blood-vessels. This condition, when associated with high tension in the arteries and accentuation of the aortic second sound, is highly suggestive of chronic nephritis. (For color and complexion, see the Skin, Chapter XXIII., Part I.)

Moreover, the face tells of the presence or absence of pain, and to a certain extent of its character. Everyone has witnessed the sudden contraction of the brow and eyelids and the involuntary sucking in of the breath when some one has bitten upon a tender tooth. Other faces bear the imprint of long-continued, more or less constant suffering. According to Eustace Smith, pain in the head in children is indicated by contraction

of the brows; pain in the chest, by sharpness of the nostrils; and pain in the belly, by contraction of the upper lip. (See the Face in Children and Pain, Chapter VIII., Part I.)

The master mind in clinical medicine, the late Austin Flint, Sr., tersely described the various appearances of the face in disease, with their clinical significance, as follows:

The Facies of Renal Disease. In some cases of acute albuminuria and of chronic parenchymatous nephritis—the large white kidney of Bright—puffiness of the face from œdema, with notable pallor, renders the aspect highly diagnostic.

The Malarial Facies. Pallor of the face, sallowness, and slight puffiness, if renal disease be excluded, point to malarial disease.

The Facies of Carcinoma. Notable anæmia, a waxy or straw-colored complexion, and more or less emaciation, in combination, render the aspect marked in some cases of malignant disease. In a patient over forty years of age this aspect has considerable diagnostic import, although it is by no means always present when malignant disease exists.

The Typhoid Facies. In the middle and later periods of typhoid fever the countenance is often dull, besotted, expressionless. This facies may be present in the typhoid state, which is incident to diseases other than typhoid fever—*e. g.*, pneumonia. Coexisting with a dusky hue of the skin and congestive redness of the conjunctiva, it distinguishes typhus as contrasted with typhoid fever.

The Facies of Acute Peritonitis. The upper lip raised so as to expose the front teeth gives an aspect which characterizes, in a certain proportion of cases, acute peritonitis. It is often wanting, but when present it is strongly diagnostic.

The Facies of Acute Pneumonia and Hectic Fever. Circumscribed redness of one or both of the cheeks, with abruptly defined borders, is diagnostic of acute pneumonia. If it be observed in a case of chronic pulmonary disease, it denotes the so-called hectic fever and is a sign of phthisis. The wan, emaciated appearance with the bright eye and hurriedly expanding nostrils excites our fears that the progress of the latter affection is most rapid.

The Facies of Exophthalmic Goitre. Projection of the eyeballs, giving to the face a remarkably staring and sometimes ferocious expression, conjoined with enlargement of the thyroid body and frequency of the pulse, is distinctive of the affection known as exophthalmic goitre—Graves' or Basedow's disease.

The Choleraic Facies. In the collapse stage of cholera the face is contracted, sometimes wrinkled; the cheeks are hollow, the eyes sunken, the skin livid, and the expression denotes indifference. This combination of traits is quite distinctive. They are, however, to a certain extent combined in the state of collapse which occurs in some cases of pernicious intermittent fever and in other pathological connections.

The Hippocratic Facies. This facies denotes the moribund state. The skin is pale, with a leaden or livid hue; the eyes are sunken, the eyelids separated, and the cornea loses its transparency; the nose is pinched and the eyes are retracted; the temples are hollow and the lower jaw drops.

Hippocrates described this facies in graphic terms, and the name Hippocratic has ever since been used to designate it.

The Facies in Children. Inspection is even more important in the case of children than in adults. The pale, pinched, weazened face of some babies who have snuffles, ulcers, or striated lines at the corners of the mouth, and look prematurely aged, with prominent forehead and a depressed nasal bridge and retroussé tip, characterizes *inherited syphilis.* In older subjects the undeveloped face and skull are striking.

The head is unusually large with flattened vertex, projecting forehead, and open fontanelle in *rhachitis.*

The head becomes very much enlarged, the eyes prominent, the bones of the *face* remaining small, the expression vacant in *hydrocephalus.*

The dull, apathetic expression, with the thickened lips, the small nasal orifices, and the gaping mouth, are characteristic of *adenoid disease* of the pharynx with tonsillar hypertrophy.

The thickened lips, the protruded tongue, with saliva dribbling from the open mouth, the flattened nose, with the idiotic expression and pallid, waxy skin, are easily recognized in *cretins.* To a lessened degree such appearances are seen in "backward" children who may be said to be undeveloped cretins.

The red, swollen face, the reddened, weeping eyes, and running nose make a very striking picture in *measles.*

An irritating, excoriating discharge from the nose in a child may indicate the existence of nasal *diphtheria.*

The Facies in Nervous Disease. All varieties of mental aberration are reflected in the face: the suspicious, at times revengeful, look of the delusional monomaniac; the wild look and excited manner of the maniac; the plaintive, depressed, injured look of melancholia; the vacant, listless, peaceable, animal-like look of dementia—a look which changes to animation only at sight of food or some coveted luxury. All these expressions come to be recognized very readily by those who see much of the insane. In hysteria expressions of varied emotions are seen; in neurasthenia a worn and wearied aspect of countenance is noticeable.

The face often tells of the existence of some organic nervous disorder. The peculiar heavy expression, drooping eyelids, though they close imperfectly, and sluggishly moving lips betoken the early stage of the facio-humero-scapular type of *muscular atrophy,* and are sometimes seen in *Friedreich's ataxia.*

Spasm and Tremor. Change in the expression and appearance of the face more frequently occurs because of change in the function and nutrition of the muscles, on account of central or peripheral disease of the nervous system. On this account we have facial spasm or tremor, and unilateral, bilateral, or local facial paralysis. Further consideration will be given to these conditions in the local examination of the muscles (Chapter XXVI., Part I.) and under Diseases of the Nervous System.

In *peripheral facial palsy* the paralyzed side of the face has a staring, vacant expression, owing to the fact that the eyelid is motionless. The angle of the mouth on the affected side is depressed. The whole para-

lyzed side is devoid of wrinkles, and has a smoothed-out, glazed appearance; tears flow over the cheeks and saliva dribbles from the corners of the mouth. The contrast with the normal side is most marked when the patient smiles or frowns. (See Fig. 18.)

In *glossolabial palsy* there is progressive paralysis with tremulousness of tongue and lips; progressive failure of articulation, and dribbling of saliva. Sometimes the patient is able to open the lips, but unable to close them without the aid of the fingers. The mask-like expression of *immobility* in *paralysis agitans* has been described as Parkinson's mask.

A slow, hesitating, thick manner of speaking, with a tendency to slur the labial and lingual consonants, when associated with irregularity of the pupils, slight tremulousness of the lips and loss of the fine adjustment of other muscular movements, such as writing, is very suggestive of *general paralysis of the insane*, especially when the condition develops in a middle-aged man.

Facial hemiatrophy is a peculiar affection, characterized by progressive wasting of the bones and soft tissue of one side of the face. The disease is rare; it begins, as a rule, in childhood, but may develop in later life. The change is diffuse; in some instances, however, it slowly spreads from a spot in the skin, involving in succession the tissues underneath.

FIG. 18.

Complete facial palsy. Patient unable to close eye of the affected side. (DERCUM.)

FIG. 19.

Facial hemiatrophy. (LYMAN.)

The skin changes in color and the hair falls out. The eye is sunken on the affected side on account of wasting of the tissues of the orbit. The bone of the upper jaw atrophies to a more marked degree than the other bones that undergo wasting, and the teeth become loose and fall out because of the wasting of the alveolar processes. The wasting is sharply limited by the middle line. (See Fig. 19.) The disorder is easily recognized. The patient looks as if the face were made up of two halves from different persons. It must not be mistaken for the *facial asymmetry* which is associated with congenital wry-neck, and is distinguished by contraction of the sternomastoid muscle from birth.

Color of the Face. A full account of the color is given in Chapter XXIII., devoted to the Appearance of the Skin.

Contour of the Face and Head. The head and face are enlarged in *megalocephaly*. The changes in contour in *acromegaly*, *rhachitis*, and *osteitis deformans* are described in Chapter XXVII., Part I., on Bones and Joints. In *leprosy* the characteristic leonine countenance—*facies leontina*—is the result of the tuberous outgrowths about the eyes and forehead. The outline in *myxœdema*, and *scleroderma*, are described in the chapter devoted to the skin (Chapter XXIII.).

Swellings of the Face.—The face is swollen and deformed in *mumps*, *erysipelas*, and *smallpox*, and to a moderate degree in *measles*.

The puffiness of the eyelids and general swelling of the face in *anasarca* due to *Bright's disease* and in *trichinosis* will be referred to in the chapter on Œdema.

Of characteristic appearance is the swelling of the face in obstructive heart or lung diseases and in the spasmodic stage of whooping-cough.

Temporary swellings may be due to *urticaria* or *angioneurotic œdema*.

Local swellings occur as follows:

1. Of the *forehead*, in *glanders* and *trichinosis;*
2. Of the *upper jaw*, in *antrum disease* and *alveolar abscess;*
3. Of the *lower jaw*, in *alveolar abscess* and *actinomycosis;*
4. In *front* of the *ear*, in *mumps;*
5. Over the *mastoid process*, in *mastoiditis* from ear disease and *thrombosis* of the lateral sinus;
6. In the *cheeks*, in *gangrenous stomatitis*, and in obstruction of Steno's duct.

Anthrax and *boils* may cause swelling of any part of the face.

The Lips.

Color. The lips are pale in *anæmia*, although pallid lips do not necessarily imply the presence of this condition. They are livid in *cyanosis* from chronic lung or heart disease with feeble circulation. *Vesicles* (herpes) are apt to appear upon the lips in common colds, in certain febrile diseases, particularly pneumonia, and with many women during or immediately following menstruation. An *ulcer* due to a chancre or epithelioma, and mucous patches may be situated on the lips.

A child with hereditary syphilis may show ugly *fissures*, or the *scars* which result from them, at the angles of the mouth.

In facial palsy the angle of the mouth on the paralyzed side is depressed and the skin is free from wrinkles. In glosso-labio-laryngeal palsy the lips *tremble* and *twitch*, and may have to be closed with the fingers after they have been opened. In general paralysis of the insane the lips tremble, and speech is "thick," hesitating, and uncertain, with a tendency to elide syllables and slur the labial consonants.

The Bloodvessels.

The veins are enlarged and tortuous in tumors of the neck and thrombosis of the lateral sinus and in varicose aneurisms. The temporal arteries are prominent, rigid, and tortuous in atheroma.

THE HEAD.

Abnormal Movement. Irregular bizarre movements occur in *chorea.* A rhythmical *nodding spasm* of the head occurs in hysteria, in the epilepsy of childhood, or may be due to habit. Spasmodic jerking of the head with rotation to one shoulder, and elevation of the chin and rotation of the face to the opposite side, occurs in spasmodic torticollis.

Abnormal Fixation. Spasm of muscles, myalgia, torticollis, rheumatism and rheumatoid arthritis, scars, scleroderma, and retropharyngeal abscess cause more or less fixation of the head. In tubercular disease of the vertebræ the head is fixed and the pain is relieved by support. Fixation and retraction occur in meningitis, in tetanus, and in strychnine-poisoning.

Enlargement. Change in the size and shape of the head is seen in *leontiasis ossea, rhachitis, acromegaly, osteitis deformans,* which are discussed in the chapter on the Bones and Joints; in *myxœdema* and *leprosy;* in *sporadic cretinism* and in *hydrocephalus.*

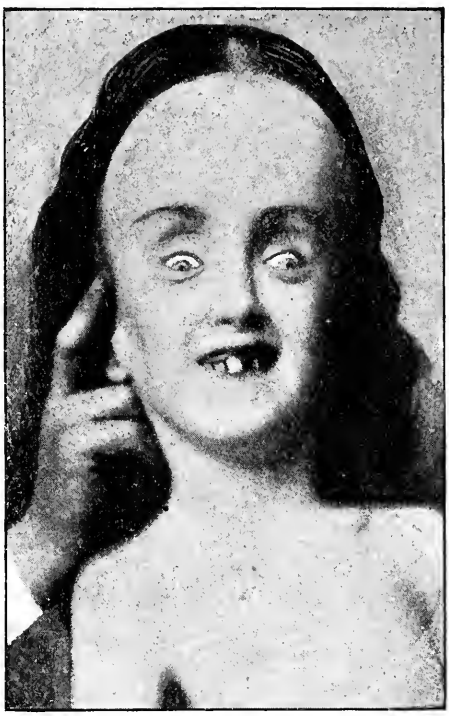
In *osseous hypertrophy* the bones are thickened. Gowers states that such thickenings may simulate hydrocephalus at any age. He thinks it doubtful whether the nature of osseous hypertrophy can be ascertained during life.

Hydrocephalus. The enlargement of the skull is very conspicuous, and the disproportion of the cranium to the face is striking. The cranium is rounded or globular in shape, and the fontanelles are seen to be very large, tense, and bulging; the sutures are widely separated. The disproportion in size between the face and head is apparently increased by the projection of the anterior portion of the skull. The axis of the eyes is directed downward, and they are partly covered by the eyelids because of the oblique direction of the orbital plates. The head is supported with difficulty. The eyeballs roll from side to side. Frequently there is strabismus. The skin is stretched tightly over the cranium and the hair is scanty. The veins are prominent. (See Fig. 20.)

Diminution in the size of the head is seen in microcephaly (circumference less than seventeen inches). The head is usually abnormal in shape.

Sweats. Sweating of the head is of common occurrence in rhachitis. It is attended by loss of hair on the back of the head induced by boring the head into the pillow.

FIG. 20.



Congenital hydrocephalus. Female, aged seventeen. The thinness of the hair could not be represented. (Original.)

The Hair.

The hair often indicates the state of the individual's nutrition. Changes in it may be significant of syphilis or other internal morbid processes. The abnormal growths and changes in the texture due to local parasitic disease will not be referred to. Undue and rapid *loss of hair* in patches, known as *alopecia*, is indicative of syphilis. The hair can be easily pulled out in large masses without causing pain. This falling of the hair must not be confounded with the excessive falling out which takes place during convalescence from acute disease, particularly typhoid fever, nor with that following an attack of gout or erysipelas. Loss of hair may follow severe neuralgias of the fifth nerve.

Color. Obscure paralysis or anæmia may be explained by noting if the hair is artificially colored. Lead and other forms of poisoning have repeatedly arisen from the use of hair-dyes. Other changes in the color are often significant. Early gray hair may go hand-in-hand with premature endarteritis. The term "canities" is applied to the diminished development of pigment. Premature gray color in well-defined patches occurs in nerve-lesions, as paralysis of one of the branches of the fifth pair, and is a trophic change. Sudden change in the color of

the hair, usually to gray, takes place at times under the influence of fright, mental anxiety, or deep emotion.

"Green" hair is seen in brass-founders and workers in copper mines; "blue" hair in laborers in cobalt mines and persons employed in the manufacture of indigo. Chemicals applied to the hair change its color —hydrogen peroxide bleaches the hair, pyrogallic acid turns it black. Drugs administered internally, as jaborandi and its alkaloid, change the color to a darker hue.

The Fontanelles.

We now turn our attention to an examination of the fontanelles and the bones of the head. The fontanelles in a healthy child, with the exception of the anterior, close during the early weeks of life. The anterior fontanelle closes some time between the sixteenth and the twentieth month. We note whether they are open or closed, prominent or depressed. New openings or fontanelles and loose bone plates, the normal fontanelles remaining open, are seen in so-called *craniotabes*—a condition found in congenital syphilis and rarely in rhachitis.

Prominence or *fulness* of the fontanelles may be temporary or permanent. When the former, a passing fever with cerebral congestion may be the cause; when the latter, hydrocephalus or some other brain affection in which there is increase of internal pressure.

Depression of the fontanelles occurs in general atrophy, marasmus, and wasting diseases generally. In collapse it is of grave prognostic omen. In pneumonia and other respiratory affections with dyspnœa retraction of the fontanelles is observed. The former affection, with cerebral symptoms, is thus distinguished from cerebral meningitis, in which the fontanelles bulge.

Delayed Closure. The fontanelles are neither prominent nor depressed in *rhachitis*, a point of distinction between this affection and hydrocephalus or enlargement from other internal causes. They may remain open in rhachitis long after the usual period of closure, even to the third or fourth year.

The Bones.

The *bones* of the cranium may be thickened; they may be the seat of periostitis, necrosis, or caries. Nodes and painful doughy swellings, becoming indurated and due to periostitis, occur in syphilis. Necrosis and caries of the *frontal* bone are almost pathognomonic of syphilis. Necrosis of the *jaw* bone belongs to phosphorus-poisoning. The *mastoid* process of the temporal bone should be examined in many affections; the symptoms that should call our attention to this region are pain and tenderness over the mastoid, rigor, and fever, with the symptoms of thrombosis of the cerebral sinuses, pain in the head, convulsions, and strabismus.

Examination of the mastoid region should include the *occipito-atlantal articulation.* Disease of this articulation, and particularly tubercular disease, causes stiffness of the neck or falling forward of the head. On

account of the stiffness, associated with difficulty of deglutition and pain, the writer has seen it mistaken for retropharyngeal abscess.

Auscultation and percussion. We have thus far limited our examination of the head to inspection and palpation. Auscultation has been practised, and at one time it was believed that the *continuous murmur* heard over the vertex in children was due to intracranial disease. Osler, however, pointed out its occurrence in healthy children; hence, unless heard in adults, its presence is not of diagnostic significance.

McEwen, of Glasgow, has found that in cerebral abscess, tumor, and also in meningitis secondary to ear disease a difference in the percussion-note is heard over the affected area, and at the same time the percussion-resistance is increased. The site of disease was indicated by a note higher in pitch than the usual osteal note.

THE NECK.

The position and movements of the larynx and trachea, the thyroid gland, the lymphatic glands, and the vessels of the neck should be observed.

The Larynx and Trachea.

Position. These structures occupy the median line in health. They may be deflected to the right or left. The deflection is more readily noticed at the lower part of the neck, and can be ascertained by comparing the position of the structures with the normal relation to the adjacent muscles. Pseudodeflection may be due to atrophy of the muscles of one side. True deflection is due to an external growth or aneurism, or to disease within the thorax, as a thoracic aneurism or a mediastinal tumor. In chronic fibroid phthisis the trachea is often drawn over to the side of the affected lung.

Movements. When the respiratory movement of the larynx and trachea is excessive and associated with dyspnœa, the source of the dyspnœa is of laryngeal origin. When, on the other hand, the movements are lessened, or the organs remain fixed in spite of violent efforts at respiration, the dyspnœa is due to disease in the mediastinum, as enlargement of the mediastinal glands or aneurism pressing upon a bronchus. *Tracheal tugging* is usually determined by palpation. It is particularly characteristic of aneurism of the descending portion of the aorta. (See Diseases of the Bloodvessels.)

Thyroid Gland.

Atrophy is shown by absence of fulness, which would otherwise be present. (See Myxœdema and Acromegaly.)

Enlargement of the thyroid can be detected without much difficulty. It may be limited to one lobe or affect both lobes.

The enlargement varies in size from a small localized swelling to large masses filling the median and lateral portions of the neck, pressing upon the trachea, and extending into the thorax. The swelling may be soft or hard to the touch. In the fibrous forms the swelling is not very large

and is very much indurated. In the cystic forms of thyroid enlargement fluctuation may often be detected; it may be localized to a small area of the lobe, or may be present over the entire affected lobe. In some cases a purring or thrill is transmitted to the fingers, synchronous with the heart's action and due to increased vascularity of the gland. Auscultation under these circumstances reveals a systolic murmur.

Causes. Enlargement of the thyroid gland may be due to simple hypertrophy, to fibrocystic enlargement, or to enlargement in which the vascular element is more prominent, as in exophthalmic goitre. 1. In simple *hypertrophy* the enlargement is often intermittent, increasing in size at each menstrual period, or coming on during pregnancy, to disappear after labor. It may then disappear entirely or return at the menopause. 2. The *fibrocystic* enlargement which is endemic in certain countries is persistent. 3. The enlargement of *exophthalmic goitre* generally continues throughout the course of the disease.

Enlargement of the thyroid gland from the above-mentioned causes must be distinguished from enlargement due to *abscess*, *cancer*, *sarcoma*, or *adenoma*. Abscess usually follows infectious diseases; in the writer's case it followed typhoid fever. With carcinoma and sarcoma there are anæmia, gradual loss of flesh, and the usual clinical phenomena of these processes. It must also be distinguished from other tumors in this region. It particularly must not be confounded with enlargement on the right side due to an innominate aneurism. (See Aneurism.)

The Vessels of the Neck.

(See Arteries and Veins, Physical Diagnosis, Chapter XXXIV.).

The Lymphatic Glands.

(See Chapter XXV.).

CHAPTER XXI.

EXAMINATION OF THE EYE AND EAR.

THE EYE.

Indirectly the eye and the skin are the external structures that most frequently present evidence of disease in other organs. This is particularly true of the eye because it can be examined with comparative ease, and because it is a highly specialized organ, bearing close relationship to the vascular and nervous systems. Its functions are subservient to the highest physiological cerebral action; hence any perturbation or organic change in the cerebrum is expressed in altered eye function, either of movement or of vision. Its nervous and vascular connections with the brain render it sensitive to internal changes, and in diseases of the nervous system the eye is therefore the one organ the examination of which is essential to make a diagnosis. In the chapter on Nervous Diseases constant reference will be made to this point; and the converse holds true, that in the study of the changes in the eye reference must be made to the nervous system. In addition, diseases of the heart and kidneys, and certain systemic conditions, such as gout, rheumatism, diabetes, etc., often find expression in some eye change.

Much may be ascertained regarding the state of the general system from an inspection of the eye and its adnexa. This is at once evident when we reflect that of the twelve pairs of cranial nerves four are devoted solely to this important organ, while in the eye itself we have unfolded to our gaze a living nerve-head, the optic papilla, and the retinal vessels, which offer to our view the perfect cycle of the supply of an organ with arterial and the escape of its venous blood. Moreover, the eye presents in compact form a representation of nearly all the tissues of the body.

In order to insure that nothing shall escape scrutiny in the inspection of the eye, it is necessary to follow some settled plan of investigation, and for this purpose it is well to pursue a definite anatomical order, proceeding from the superficial to the deeper structures.

The Lids.

Œdema is a not infrequent symptom of renal disease (see Œdema of the Face), and may occur in cases of profound anæmia and chlorosis; it may indicate the prolonged use of arsenic, or it may be originated by disease in the orbit or some of the periorbital sinuses of the same side. The accumulation may take place during the night and become evident in the morning on rising. Morning puffiness is natural to some individuals, and, like the swelling of the face following a debauch, is not to be confounded with œdema.

Ptosis, or drooping of the eyelid, may be congenital; more usually it is a symptom of disease within the brain. (See Paralysis of Third Nerve.)

Lagophthalmos, or imperfect closure of the lids, follows paralysis of the orbicularis muscle, due to lesions of the portio dura of the facial nerve. According to Bull and Hansen, paralysis of the orbicularis muscle is of common occurrence in leprosy.

Blepharospasm, or active closure of the lids from spasm, is of a reflex nature, originated by excitation of a filament of the fifth nerve. It is always present to a greater or less degree in *photophobia;* this latter symptom is a frequent associate of ocular disorders, and is found also in certain stages of meningitis, cerebral tumors, typhus, measles, etc. It accompanies many forms of headache, especially migraine, and may be the expression of a hyperæsthesia of the retina in nervous subjects, apart from any actual inflammation of the membrane. Spasm of the orbicularis muscle has been noted quite often as a symptom of hysteria. *Nictitation,* or abnormally frequent winking, occurs not infrequently in children as part of a choreic habit.

Styes, or small boils on the palpebral margin, and **blepharitis,** or inflammation of the margin of the lids, while often due to an error of refraction, may denote some defect in the general health, such as anæmia or scrofula.

Vaccinal eruption may appear on the eyelids, occurring at the commissures as a small ulcer with an indurated border and yellow floor; it is usually attended by some swelling of the lids and face and by enlargement of the preauricular glands.

Chancre may appear as a primary sore, and is generally situated on the palpebral conjunctiva.

Malignant pustule, or specific anthrax, is seen at times, though rarely, on the lids of those who are exposed to infection from diseased animals or decayed animal matter.

Xanthelasma consists in the formation of small, irregular, opaque, yellowish patches, slightly elevated above the surrounding skin. These areas may either remain localized, or the disease may involve the palms of the hands, the flexures of the fingers, and the inside of the mouth. (See Tongue.)

The Orbits.

Exophthalmos, or proptosis, abnormal prominence or protrusion of the eyeball, is usually occasioned by some disease of the orbit or of the neighboring sinuses which encroaches upon the cavity of the orbit. It is one of the diagnostic features of exophthalmic goitre (see Exophthalmic Goitre), and may also be caused by paralysis of the ocular muscles. It has been seen, though rarely, after spontaneous hemorrhages into the orbit in cases of hæmophilia and scurvy.

Enophthalmos, or recession of the eyeball, may be the result of exhausting diseases, such as peritonitis, or secondary to some orbital lesion. It is very pronounced in the sudden atrophy of the orbital tissues that occurs in cholera from loss of water.

Extraocular Muscles.

In order that the subject of paralysis of the extraocular muscles may be grasped more readily, a few words of explanation will be given regarding the anatomy and physiology of the muscles engaged in the ocular movements before detailing briefly the measures employed for its detection.

The eyeball is suspended in the orbital cavity by means of six muscles—the four recti, superior, inferior, internal, and external, and the superior and inferior obliques. Of these, the four recti and the superior oblique have their origin at the apex of the orbit, while the inferior oblique arises from its lower inner wall. These muscles exercise their action upon the movements of the globes in three pairs, each pair being composed of two antagonistic muscles: the rectus internus and externus, the rectus superior and inferior, and the superior and inferior obliques. The sixth nerve supplies the external rectus, the fourth the superior oblique, the remaining four muscles receiving their impulses from the third nerve.

When all of the muscles are in a state of equal tension, and the visual axes are directed straight forward in the horizontal plane, the eyes are then said to be in *the primary position*. Any deviation from this is known as *a secondary position*, the simplest of these being direct lateral or vertical movements.

Duane[1] has simplified the action of the extraocular muscles by dividing the twelve muscles which move the two eyes into three *groups* of four each: four muscles moving the eyes laterally, four moving them up (elevators), and four moving them down (depressors).

Each group is again divided into two *pairs*, one muscle of each pair being in the right eye and the other in the left. The muscles of any one pair form what are known as associated antagonists, each acting to move the eye to which it is attached in precisely the same direction and to the same distance that the other muscle of the pair moves the companion eye.

Thus, the four *laterally acting muscles* are divided into (*a*) a pair of *right-turners* (right externus and left internus), and (*b*) a pair of *left-turners* (right internus and left externus).

The four *elevators* comprise (*a*) the right superior rectus and the left inferior oblique, which act particularly to elevate the eyes when the latter are directed to the right, and act scarcely at all as elevators when the eyes are directed to the left. This pair then comprises what may be called the *right-hand* elevators. The other pair (*b*) of elevators, namely, the right inferior oblique and the left superior rectus, on the other hand, are most efficient when the eyes are turned to the left, and act scarcely at all when the eyes are turned to the right. They may be called the *left-hand* elevators.

Similarly the four *depressors* comprise (*a*) a pair of *right-hand* depressors (right inferior rectus and left superior oblique), and (*b*) a pair of *left-hand* depressors (right superior oblique and left inferior rectus).

[1] Ophthalmic Record, Dec., 1901.

Manner of Detecting Palsies of the Extraocular Muscles. Normally, the eyeballs move in perfect unison and harmony, so that the images of objects fall upon corresponding points of the retinæ, and single vision obtains. If this harmonious action be interrupted by paralysis of one or more of the extraocular muscles, however, limitation in the movement and deviation of the affected eye result, coupled with double vision, or diplopia.

Limitation in the Movements and Deviation of the Affected Eye. In studying limitations of motion in the eyes, the examiner seats himself before the patient and requests the latter to follow with his eyes the movements of a candle which is carried through all the different meridians of the visual field, any muscular deviation being made evident by a failure in correspondence of the images from the candle reflected from the cornea, as well as by the lagging in the movements of the eye owing to the deviation of the action of the affected muscle. Three general laws have been formulated which should be borne in mind in this connection: 1. The limitation in motion as well as the diplopia increases toward the side of the affected muscle. 2. The secondary deviation (the deviation which the sound eye makes while the affected eye is fixing the candle) is greater than the primary deviation (the deviation of the affected eye while the sound eye fixes). 3. The image formed on the retina of the affected eye is projected in the direction of the paralyzed muscle.

Diplopia. The character of the diplopia varies according to the muscle or muscles whose function has been disturbed. Generally speaking, diplopia is either simple or homonymous, or crossed or heteronymous. In the former the image seen by the affected eye lies on the corresponding side and betokens convergence of the visual axes, while in the latter the image seen by the affected eye is projected to the opposite side and indicates divergence of the visual axes. In order to ascertain the relation of the two images to the respective eyes it is essential that the diplopia should be carefully tested.

Test for Diplopia. For this purpose the patient is seated in a darkened room with a red glass placed before one of the eyes in order to facilitate the identification of each image by its color, and a lighted candle is held on a level with the head about five metres off. Having noted any deviation which the eyes make in the primary position upon a chart specially constructed for the purpose, the examiner moves the candle through the different meridians of the visual field, the patient being requested to regard the flame with both eyes while the head remains stationary, each deviation being carefully noted on the chart.

After the deviations have been recorded, the diagnosis of the affected muscle or groups of muscles may readily be obtained by a comparison of the diplopia manifested by the candle-test with the following table of diplopia in paralysis devised by Duane:

A. There is a *lateral* (*i. e.*, an homonymous or crossed) diplopia, which increases markedly as the eyes are carried *laterally* (to the right or left). A laterally acting muscle is paralyzed.

a. Diplopia increases in looking to the *right* (= paralysis of a right-turner).

Diplopia homonymous: paralysis of *right externus.*

Diplopia crossed: paralysis of *left internus.*

b. Diplopia increases in looking to the left (= paralysis of a left-turner).

Diplopia crossed: paralysis of *right internus.*

Diplopia homonymous: paralysis of *left externus.*

B. There is a *vertical* diplopia which increases in looking *up.* An elevator is paralyzed.

a. Vertical diplopia increases in looking *up and to the right* (= paralysis of a right-hand elevator).

Diplopia left (*i. e.,* image of right eye above): paralysis of *right superior rectus.*

Diplopia right (*i. e.,* image of left eye above): paralysis of *left inferior oblique.*

b. Vertical diplopia increases in looking *up and to the left* (= paralysis of a left-hand elevator).

Diplopia left (*i. e.,* image of right eye above): paralysis of *right inferior oblique.*

Diplopia right (*i. e.,* image of left eye above): paralysis of *left superior rectus.*

C. There is a *vertical* diplopia which increases in looking *down.* A depressor is paralyzed.

a. Vertical diplopia increases in looking *down and to the right* (= paralysis of a right-hand depressor).

Diplopia right (*i. e.,* image of right eye below): paralysis of *right inferior rectus.*

Diplopia left (*i. e.,* image of left eye below): paralysis of *left superior oblique.*

b. Vertical diplopia increases in looking *down and to the left* (= paralysis of a left-hand depressor).

Diplopia right (*i. e.,* image of right eye below): paralysis of *right superior oblique.*

Diplopia left (*i. e.,* image of left eye below): paralysis of *left inferior rectus.*

Additional Symptoms. In addition to the study of the anomalies in motion and of the diplopia, considerable information may often be gained by noting the position of the head in ocular paralysis. Thus, in paralysis of the sixth nerve the face is turned toward the paralyzed side; in paralysis of the fourth nerve it is turned downward and toward the shoulder of the paralyzed side; and in paralysis of the third nerve the face looks toward the shoulder of the same side. Not rarely dizziness is complained of, and there is false projection of the field of vision, causing faulty estimation of distance.

The Clinical Significance of Disturbances in the Motility of the Extra-ocular Muscles. In addition to the relation which paralysis of the muscles bears to lesions of the brain and of the cranial nerves, and which will be dwelt upon at length later, diplopia may proceed from some much less serious disturbance, as, for example, derangements of the digestive organs or abuse of alcoholic intoxicants. Transient attacks of diplopia may be

among the earliest symptoms of tabes dorsalis, and may occur at the very beginning of cerebral meningitis.

Monocular diplopia is a rare symptom, and when it can be dissociated from some local disturbance in the media of the eye, may be due to hysteria.

Ocular deviation or *paralytic squint*, which has just been described, must be differentiated from *concomitant squint* or *strabismus*. In this latter variety there is no great restriction in movement of the eyes in any one direction, the faulty position of the visual axes remaining constant while the eyes are moved from side to side, and the secondary deviation being equal to the primary. This is the condition which is commonly known as cast or cross-eye, and usually makes its appearance in children with high degrees of hypermetropia.

Nystagmus is a spasmodic condition of the muscles of the eye, producing rapid oscillations of the ball, usually horizontal, sometimes rotary, and rarely vertical. It is of great value as a symptom, being present in many brain lesions, usually those of the restiform bodies, the vermiform process, and of the cerebellum. It is also seen in about one-half of the cases of disseminated sclerosis, in Friedreich's ataxia, in miners, and often as the result of visual defects.

Muscular Insufficiencies. Of late years much attention has been given by ophthalmologists and neurologists to the study of *errors in the extraocular muscle balance* in different reflex psychoses. While the assertion which has been made by some, that chorea and even epilepsy may be originated by such deviations, is extreme, it is nevertheless quite true that many forms of headache, of vertigo, of nausea, and of vague neuralgic pain of a cephalalgic type can be traced to this source. It is important, therefore, that the clinician should be acquainted with such errors and with the methods employed for their detection.

Test. The device of Maddox is usually employed for this purpose. It consists of a glass cylinder which is fitted into a linear opening in a metallic disk. The patient is seated before a candle flame, five metres away from the examiner, and requested to fix the flame with both eyes. The rod is then placed before one of the eyes perpendicularly and an image of a perpendicular streak of light obtained from that eye. If the streak of light be deviated toward the same side as the eye before which it is held, a condition of excessive convergence or *esophoria* is present; but if the streak deviates toward the opposite side, then a divergence of the visual axes or *exophoria* exists. If the streak be on a higher or lower level than the flame, vertical imbalance or *hyperphoria* is present. Balance of the muscles is known as *orthophoria*.

The Conjunctivæ.

The conjunctiva being a transparent, vascular membrane, any changes in the quantity or constitution of the blood will at once manifest themselves in its folds. Thus in anæmia there is always pallor of the conjunctival vessels, while in plethora there is usually a passive dilatation

of the vessels which gives the eye an injected appearance and occasions the "bloated eye" of the drunkard. In jaundice the conjunctiva is yellow. Spontaneous hemorrhages into the membrane are seen in whooping-cough, asthma, epilepsy, and in calcareous degeneration of the blood-vessels, and it may be the seat of hemorrhagic infarcts in ulcerative endocarditis.

Inflammation of the conjunctiva is an early symptom in measles, and in typhus fever it is a constant sign and serves to distinguish this affection from typhoid. It is also present in yellow fever, and may likewise constitute one of the earliest signs of meningeal and cerebral diseases. A passive hyperæmia follows disease of the cervical sympathetic.

The Cornea.

The cornea being an avascular membrane which derives its nourishment from the surrounding structures, it is very prone to undergo inflammation whenever the vitality of the system becomes much lowered, and as a result of this inflammation *opacities* remain which have a very deleterious effect upon vision. These opacities may be either superficial or interstitial. When superficial they are not infrequently the result of burns, traumatisms, and extension of inflammation from the surrounding conjunctiva; in many cases they denote, however, that the eye has been the seat of a phlyctenular conjunctivitis, a form of ocular disease which is quite common in scrofulous children and in individuals below par.

Superficial *ulceration* of the cornea is observed also in all fevers of a typhoid type, when the patient lies in a semiconscious state with the lids partly closed. Dust and bacteria collect between the lids; and as the patient winks but seldom, a crust forms on the cornea, which is followed by extensive ulceration. *Abscesses* of the cornea form in the stage of desquamation of variola, and must be differentiated from those which arise in the pustular variety of the disease at an earlier period. Ulcers also form in the seventh week of typhoid, being usually coincident with abscesses in the scalp and skin of the back.

The type of *interstitial opacities* of the cornea is seen in inherited syphilis. Indeed, to the trained eye, the appearance of the haze in this class of cases is so characteristic that the diagnosis of the systemic affection can be made from the eye alone. Malaria and scrofula may also produce a similar form of corneal inflammation. The small areas of opacity which form in the upper and lower parts of the cornea near the limbus, and which at times encircle the cornea, are known as *arcus senilis*. This is commonly supposed to be indicative of arterial sclerosis, although the author has never found any ground to warrant this assertion. It may always be diagnosticated from a somewhat similar opacity of inflammatory origin by the fact that in the latter variety, the opacity being due to an inflammation usually beginning at the corneoscleral margin, the haze is continuous with the conjunction of the two membranes; while in arcus senilis there is a zone of clear corneal tissue between the margin of the cornea and the rim of the opacity.

After lesions of the fifth nerve the cornea may ulcerate from traumatic

and trophic causes; and after paralysis of the seventh nerve it may suffer from exposure due to inability to close the lids.

Iris.

Inflammation of the iris is a common symptom of secondary syphilis; it occurs in the form of a gummatous infiltration of the membrane in the tertiary variety, and is seen, though rarely, in inherited syphilis. It is not an infrequent symptom of chronic rheumatism and gout, and may be caused by tuberculosis and rheumatoid arthritis.

The Pupil.

The pupil may react either directly or indirectly to *light-stimulus*. In order to observe this, the patient is seated before a window and requested to gaze at the sky. The examiner, stationed in front of the patient with his back to the window, excludes one eye by placing his hand over it, and notes the size of the pupil under diffuse daylight. The eye is then covered with the other hand, and the dilatation which should follow is also estimated. The hand is then withdrawn, and, if nothing prevents, the pupil resumes its original size. The fellow eye is then tested in the same manner. This is known as the *direct reflex action* of the pupil; *indirect or consensual reflex action* being the contraction or dilatation which occurs in the shaded eye when the exposed eye is being examined, and should correspond precisely with the movements of the pupil of that eye.

After the reaction of the irides to light-stimulus has been noted, the patient is directed to transfer his gaze to the examiner's finger, which should be made to approach the eye slowly, while its fellow is screened off as in the preceding test. The degree of contraction induced by this *accommodative effort* is carefully noted, and the same procedure repeated with the fellow eye. The obstructing hand is finally removed, and the patient being requested to look fixedly at the tip of the examiner's finger with both eyes, observation is made of the contraction of the pupils, which should be induced by the *effort at convergence* occasioned by approximating the finger to the eyes in the median line.

Hippus is a spasmodic alternating contraction and dilatation of the pupil, which is seen at times in mania, hysteria, and allied disorders. Rhythmical alterations in the size of the pupils occur frequently during so-called Cheyne-Stokes respiration; the pupil contracting during the period of apnœa and dilating with the first few respirations.

Modification in the Size and Behavior of the Pupils as the Result of Disease. Pupillary reaction to light is a reflex phenomenon, the optic nerve being the afferent nerve, and the third nerve the efferent nerve supplying the sphincter of the iris; communicating fibres between the corpora quadrigemina and the centre for the third nerve making such a reflex possible. The mechanism of pupillary reaction being of an extremely complicated nature, and necessitating the activity of a number of nerves and nuclei, it is not strange that anomalies in its behavior should frequently be met with in disorders of the central nervous system.

Dilatation of the pupil (*mydriasis*), apart from local diseases, of which glaucoma is the type, may occur in certain psychical states, such as fright and emotion; or it may be caused by disease processes giving rise to irritation of the pupil—dilating centre or fibres (irritative or spasmodic mydriasis), or by paralysis of the pupil—contracting centre or fibres (paralytic mydriasis or iridoplegia).

Irritation mydriasis occurs (*a*) in hyperæmia of the cervical portion of the spinal cord and in spinal meningitis; (*b*) in the early stages of new growths in the cervical portion of the cord; (*c*) in cases of intracranial tumor and other diseases causing high intracranial pressure, according to Raehlmann, although Leeser points out that these may also give rise to paralytic mydriasis; (*d*) in the spinal irritation of chlorotic or anæmic

FIG. 21.

Ophthalmoscopic examination.

persons, after severe illness, etc.; (*e*) as a premonitory sign of tabes dorsalis; (*f*) in cases of intestinal worms, from irritation of the sensory nerves of the bowel, and sometimes in other forms of intestinal irritation; (*g*) in psychical disturbances—*e. g.*, acute mania, melancholia, progressive paralysis of the insane (in the last-mentioned disease often unilateral, with myosis of the other eye). (After Swanzy.)

Paralytic mydriasis (*iridoplegia*) may be due either to a paralysis of the pupil-contracting centre or to failure of the stimulus being conducted from the retina to that centre. It may be found: (*a*) sometimes in progressive paralysis in which at first there was myosis; (*b*) in various disease processes at the base of the brain affecting the centre for the third nerve; (*c*) in a late stage of thrombosis of the cavernous sinus; (*d*) in orbital disease associated with pressure on the ciliary nerves. (After Swanzy.) It is said to be present in acute dementia, when there is œdema of the cortex, and may be found in cerebral softening. It results from irrita-

tion of the cervical sympathetic and occasionally occurs in aortic insufficiency.

Contraction of the Pupil (*Myosis*). After myosis from local causes, especially from the sequels of iritis, has been excluded, it will be found that contraction of the pupil may be caused by a disease process irritating the pupil-contracting centre or nerve-fibres (the irritative myosis of Leeser), or by one causing paralysis of the pupil-dilating centre or nerve-fibres (the paralytic myosis of Leeser), or by a combination of both.

Irritation myosis is found in (*a*) the early stages at least of all inflammatory affections of the brain and its meninges, hence in simple, tubercular, and cerebrospinal meningitis (when, in these diseases, the medium myosis gives place to mydriasis, the change is a serious prognostic sign, indicating the stage of depression with paralysis of the third nerve); (*b*) in cerebral apoplexy the pupil is at first contracted, according to Berthold, who points out that this contraction is a diagnostic sign between apoplexy and embolism, as in the latter the pupil remains unaltered; (*c*) in the early stages of intracranial tumors situated at the origin of the third nerve or in its course; (*d*) at the beginning of a hysterical or epileptic attack; (*e*) in tobacco amblyopia, probably from stimulation of the pupil-contracting centre by nicotine; (*f*) in persons following certain trades, as the result of long maintained effort of accommodation (watchmakers, jewelers, etc.), the pupil-contracting centre being subject to an almost constant stimulus; (*g*) as a reflex action in ciliary neurosis: consequently, in many diseased conditions of those parts of the eye which are supplied by the fifth nerve. (After Swanzy.)

Paralytic myosis occurs in spinal lesions above the dorsal vertebræ, such as injuries and inflammations, especially of the chronic form. The contracted pupil occurring in gray degeneration of the posterior columns of the spinal cord has long been known as spinal myosis. In the simple form of this myosis the pupil is only moderately contracted, and reacts both to light and to accommodation. This condition is found exclusively in the early stages only, when the disease has attacked merely the ciliospinal centre, or is situated higher up, as far as the medulla oblongata; later on, when Meynert's fibres become involved, we have the Argyll-Robertson pupil. The very minute pupil often seen in tabes dorsalis is probably due to secondary contraction of the sphincter pupillæ.

Paralytic myosis is also found in general paralysis of the insane. In acute mania the pupil is usually much dilated; and when this mydriasis changes to myosis, approaching general paralysis may be predicted. Myosis, following on irritation mydriasis, is also found in myelitis of the cervical portion of the cord. If paralytic myosis occurs in bulbar paralysis, the disease is probably complicated with progressive muscular atrophy or with sclerosis of the brain and spinal cord. Myosis may also be due to paralysis of the cervical sympathetic, resulting from injury, from pressure of an aneurism of the carotid, innominate, or aorta, or from pressure of enlarged lymphatic glands. In apoplexy of the pons Varolii myosis is present, but it is not yet certain whether it is an irritation or a paralytic myosis.

Inequality of the pupils may denote a lesion of the third nerve, an affec-

tion of the cervical sympathetic in the cervical region of the spinal cord, general paralysis of the insane, or some unilateral lesion of the brain.

The Lens.

Cataract. An opacity in the crystalline lens should always awaken the suspicion of diabetes, as cataract is of not infrequent occurrence in this disease. Although renal disease also has been held accountable by some for the occurrence of cataract, no satisfactory evidence has been given to prove this assertion.

The Eye-ground.

In order to study the remaining structures of the eye it is necessary to have recourse to the *ophthalmoscope*. The essential part of this instrument consists in a concave mirror, whereby the light from a lamp which is placed behind and slightly to the side of the patient's head may be projected into the interior of the eye about to be examined. This mirror is provided with a small central aperture, through which the examiner studies the details of the back of the eye or fundus oculi, as it is technically called. When the instrument is held close to the eye, and the eye-ground studied without the mediation of other means, the procedure is known as the direct method of ophthalmoscopical examination. In the indirect method, on the other hand, the ophthalmoscope is held about sixteen inches from the eye, and an inverted image of the fundus is obtained by means of a convex lens interposed between the ophthalmoscope and the eye, which serves to collect the rays of light into a focus between the lens and the eye of the examiner. The former method possesses the advantage of magnifying the interior of the eye about fourteen times, while the indirect method permits of the examination of the greater part of the fundus at a glance, albeit under smaller magnification.

The ophthalmoscope, in addition to giving information in regard to the condition of the media of the eyes, as, for example, of the existence of commencing cataract, or of opacities within the vitreous humor, unfolds to our gaze the head of the optic nerve as well as the retina and the choroid, and renders patent to our view the different diseases to which they are liable.[1]

Retinitis. The systemic affection which is accompanied by a lesion of the retina more often than any other is disease of the kidneys, especially chronic interstitial nephritis. Indeed, about 30 per cent. of all cases of this variety of renal lesion have an ocular manifestation. Retinitis may also be seen as an early symptom in the nephritis of scarlet fever and pregnancy. Its occurrence in subjects with a cirrhotic kidney is of gloomy import, for patients with a retinal complication in this disease usually die within two years of its first appearance. Retinitis may also be occasioned by pernicious anæmia, leukæmia, diabetes, syphilis, and heart disease.

[1] It has not been thought proper in a work of this kind to give further details regarding ophthalmoscopy, the student being referred to text-books upon ophthalmology for that important subject.

Choroiditis is usually the result of syphilis; but in rare instances the choroid may be the seat of tubercles. Gout may also originate a subacute inflammation of the membrane.

Optic Neuritis. The optic nerve being really a prolongation of the brain, and being, of a consequence, liable to be affected in cerebral disorders, it is of the utmost importance that the clinician should be able to recognize changes in its appearance. Indeed, it is safe to say that the study of a "nervous case," so called, is never complete without the report of the ophthalmoscopic findings.

Papillitis, or choked disk, an inflammation of the head of the optic nerve, is rarely idiopathic, but is occasioned by cerebral growths and by meningitis, especially of the base of the brain, and by the same constitutional diseases which originate retinitis. It also occurs in acute fevers, and it may be the result of suppression of the menses. Usually, however, choked disk is the result of an intracranial tumor, occurring in 90 per cent. of all such cases; and as it is an early sign, its detection has frequently been the means of discovering an intracranial neoplasm. As a rule, tumors of the cerebellum and those of the cerebrum which interfere with the circulation of the lymph in the brain produce choked disk, the size and the character of the tumor not seemingly influencing its production.

The variety of optic neuritis just described is an ascending neuritis, the inflammation beginning at the intraocular termination of the nerve and spreading upward to the brain. There is also an interstitial or descending neuritis which is commonly caused by meningitis. Retrobulbar or toxic neuritis is a variety of inflammation of the optic nerve in which the disease is confined to the bundle of nerve-fibres which supply the macular regions. This disease is commonly caused by alcohol or tobacco, although it may be originated by quinine, the salicylates, lead, or iodoform. It may also be caused by rheumatism and exposure to cold, and there is a rare form in which the disease is transmitted through certain families from generation to generation.

Optic Atrophy. This may be secondary to some inflammation of the optic nerve or retina, or it may be a primary disease.

Secondary or *consecutive atrophy* is usually the result of optic neuritis; it may, however, be originated by local causes either within the eye or the orbit. *Primary atrophy*, on the other hand, though occasionally idiopathic, is generally found associated with some disease of the spinal cord, especially with tabes dorsalis. In this affection it is frequently an early sign, and it has been noted by Benedikt, of Vienna, that when it occurs the patient rarely becomes ataxic. It has also been remarked that cases, in which blindness is well advanced, suffer but little from the characteristic pains of tabes dorsalis. Simple atrophy occurs also in lateral and insular sclerosis, and is frequently seen in general paralysis of the insane.

Before proceeding further with the consideration of the cerebral expansion of the optic nerve, it becomes necessary to study the methods used in the determination of the visual acuity, both central and peripheral,

as these are valuable and often necessary adjuncts in establishing the diagnosis of many obscure cases of cerebral disease.

Central vision is tested by means of black letters printed on a white test-card, those devised by Snellen being usually employed on account of the admirable system upon which they are founded. The patient is seated five metres away from the card and, one eye being blindfolded, he is requested to read the lowest line of letters that he can distinguish. If the vision fails to correspond to the standard, it is necessary to exclude hypermetropia, myopia, and astigmatism by means of convex, concave, and cylindrical lenses before it can be definitely asserted that the vision is lowered as the result of disease.

Peripheral vision, or the extent of space of which the eye is conscious when it is fixed on any given point, may be estimated in several ways; it is accomplished, however, most accurately by means of the perimeter.

FIG. 22.

The McHardy perimeter.

This instrument consists of an upright rest for the chin and a semicircular arc or bar, graded in degrees, which revolves on a central pivot, and is capable of describing a hemisphere in space. The eye under examination being directed straight ahead at the fixation-point, the fellow eye being blindfolded, the test-object (consisting of a small square of white paper) is brought from the periphery toward fixation. The patient is then asked to indicate the instant the object is perceived, and the examiner marks the degree upon a chart provided for the purpose. If a

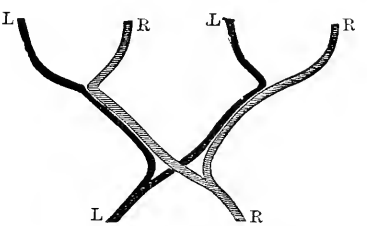
perimeter be not at hand, the field may be obtained fairly accurately as follows: The patient is seated opposite the surgeon with one eye bandaged. He is then directed to look at the corresponding eye of the examiner while the observer's finger is slowly brought in from the periphery toward the eye through the different meridians. In this way the examiner can ascertain whether the patient's eye wanders from the fixation-point, and at the same time he can compare the extent of the patient's field with that of his own. The field for form or white extends over 150 degrees horizontally and 110 degrees vertically, that for the different colors falling within this in the following order—yellow, blue, red, and green.

Scotoma. As the patient's macula corresponds to the fixation-point in the visual field, the physiological blind spot which is occasioned by the entrance of the optic nerve into the eye will be found in the temporal portion of the field. Pathological blind spots are known as scotomata, and may be either central, paracentral, or disseminated. When central they indicate disease either of the macula or of the fibres of the optic nerves supplying the macula, so that a central scotoma is one of the diagnostic features of retrobulbar neuritis.

Hemianopsia. This term is used to designate a defect in one-half the field of vision, the defect being named according to the blind area. Thus temporal hemianopsia means that the eye cannot perceive objects situated in the outer half of the field. The most common form of hemianopsia is loss of the temporal field in one eye and of the nasal field in the other, this condition being known as lateral homonymous hemianopsia. If the temporal portions of both fields are lost, the defect is known as bitemporal hemianopsia; binasal hemianopsia indicating loss of the nasal fields of both eyes. Superior and inferior hemianopsia are very rare.

It is often possible by studying the changes in the visual fields to locate quite definitely the seat of the cerebral lesion. By a reference to the diagram (Fig. 23) it will be at once evident that *a lesion of the chiasm* would

FIG. 23.



Showing the course of the optic fibres in the chiasm. (HIRT.)

necessarily comprise the crossed fibres of the optic nerve, and would occasion bitemporal hemianopsia. Such a lesion may be due to basilar meningitis, periostitis, hyperostitis, fracture of the body of the sphenoid, distention of the infundibulum and of the third ventricle, tumors, especially those of the pituitary body, and finally gumma. If the lesion is a gumma, there may be transient recurrent attacks of hemianopsia. Bitemporal hemianopsia is also an early symptom of acromegaly. The lesion

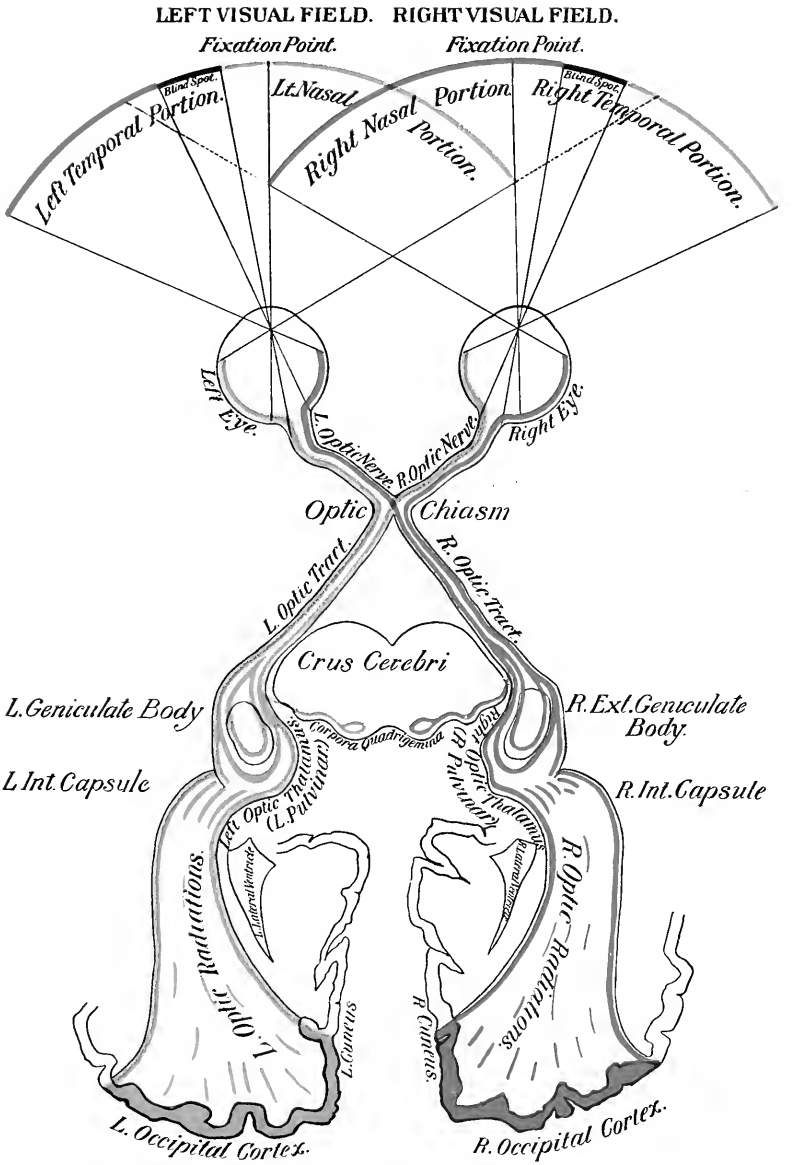
causing superior and inferior hemianopsia is usually in the chiasm also, affecting its superior or inferior portion; these defects in the fields may, however, be caused by symmetrical cortical lesions and by optic neuritis. (See Plate I.)

If the lesion affects the outer angle of the chiasm, then monocular nasal hemianopsia is the result.

Lesions of the Optic Tract and Centres. As shown in Plate I., the optic tract after crossing the crus to the hinder part of the optic thalamus divides into two branches. One branch goes to the thalamus and the external geniculate bodies, and to the anterior quadrigeminal bodies, from which fibres pass into the hinder part of the internal capsule, and, entering the occipital lobe, form the fibres of the optic radiations terminating in the cuneus, the perceptive visual centre; while the fibres of the other branch pass to the internal geniculate bodies and the posterior quadrigeminal bodies.

A lesion affecting the optic fibres anywhere posterior to the optic chiasm will produce lateral hemianopsia, so that this symptom of itself is of little value in localizations. There are, however, certain accessory symptoms which, when taken in conjunction with it, will serve to establish the seat of the lesion in most instances. Thus, in hemianopsia from lesions of the optic tract there is an absence of the symptoms which occur when the cortex is affected—as mind-blindness, word-blindness, etc.; while other symptoms indicating a basal lesion are apt to be present, as, for example, implication of the cranial nerves, especially those supplying the ocular muscles. Lesions of the optic tract are also frequently associated with disease of the crus cerebri, so that hemianæsthesia or hemiplegia of the opposite side of the body would be associated with the hemianopsia. There is, however, a sign which enables us at once to say definitely whether the lesion be in the optic tract or not, and this is known as the *Wernicke* or *pupillary inaction sign*. This is elicited as follows: The patient is seated in a darkened room with one eye blindfolded, and is directed to look straight ahead into the darkness. The eye being slightly illuminated by an assistant by means of diffuse light from a plane mirror, which is reflected into the eye from a light placed behind the patient's head, the examiner slowly throws a small beam of concentrated light from a concave mirror upon the blind half of the retina. If the pupil fails to react, the lesion is in the geniculate bodies or in the tract, inasmuch as the failure in the pupillary activity indicates that the lesion must have involved the sensory motor arc of the pupil as well as the visual fibres. Although when present the Wernicke sign is of great value, recent observations have shown that its absence is not conclusive. Lesions of the optic tract may be due to neoplasms or to tubercular or gummatous meningitis, or more rarely they may be the result of cerebral softening and hemorrhage. As yet clinical evidence is too meagre to make a diagnosis of lesions of the primary optic ganglia —pulvinar, anterior corpora quadrigemina, and external geniculate bodies —possible, although in lesions of the pulvinar two typical symptoms occur, viz., hemianopsia and athetosis, and hemianæsthesia may sometimes be present. In like manner, while it is generally believed that

PLATE I.


LEFT VISUAL FIELD.
RIGHT VISUAL FIELD.
Fixation Point.
Fixation Point.
Blind Spot.
Blind Spot.
Left Temporal Portion.
Lt. Nasal Portion.
Right Nasal Portion.
Right Temporal Portion.
Left Eye.
L. Optic Nerve.
R. Optic Nerve.
Right Eye.
Optic Chiasm
L. Optic Tract.
R. Optic Tract.
Crus Cerebri
L. Geniculate Body
R. Ext. Geniculate Body.
Corpora Quadrigemina
L. Int. Capsule
R. Int. Capsule
Left Optic Thalamus (L. Pulvinar)
Right Optic Thalamus (R. Pulvinar)
L. Lateral Ventricle
R. Lateral Ventricle
L. Optic Radiations.
R. Optic Radiations.
L. Sulcus
R. Sulcus
L. Occipital Cortex.
R. Occipital Cortex.

lesions of the optic radiations cause homonymous hemianopsia, it has not been definitely proved that these fibres have solely to do with vision.

The hemianopsia is usually assumed to depend upon cortical lesions in the occipital lobe when it is unaccompanied by any of the accessory symptoms which have just been detailed. The chief diagnostic symptom of a central lesion, however, is what is designated as negative vision, "vision nulle," for in these cases the patient has no subjective sensations of the defect in his visual field. Cortical hemianopsia may also be incomplete, one quadrant only of the field being lost.

Transitory hemianopsia, or *scintillating scotoma*, is the occurrence of symmetrical defects in the field of vision which usually conform to the hemianopic type, and in which a play of lights frequently appears as a precursor of an attack of migraine. (See Migraine.)

Visual hallucinations may also be hemianopic in character, and are due to irritation of the visual memory-centre.

Hysterical amblyopia may manifest itself either in complete blindness or in central scotoma, but more commonly as defective central vision with concentric contraction and reversal of the visual fields.

Paralysis of the Motor Nerves of the Eyeball. Although the various forms of ocular deviation and the different varieties of diplopia resulting therefrom have been mentioned at length in connection with the diseases of the ocular muscles (see page 175), it is necessary to refer still further to their causes, and to point out their connection with cerebral diseases.

Paralysis of the orbital muscles may be due to an orbital lesion or to one at the base of the brain; it may indicate a pontine lesion, or it may be originated by causes operating higher up in the cerebrum above the nuclei. In making the differential diagnosis between central and peripheral palsies, it must be remembered that those of central origin are frequently associated with other symptoms denoting intracranial involvement, while peripheral palsies are generally isolated and often complete.

Peripheral paralyses of the orbital muscles are generally the result of either rheumatism or syphilis. When due to the latter disease, they are usually tertiary manifestations, and especially is this likely to be the case if the third nerve is involved, which seems to be singularly prone to be attacked by gumma at the base of the brain. Paralysis of the sixth nerve is frequently of rheumatic origin.

Syphilis is responsible for fully one-half the cases of central paralysis, the gumma affecting either the nuclei of the nerves or the neighboring brain structure, the third and fourth ventricles, or the aqueduct of Sylvius.

Diphtheria usually causes paralysis of the ciliary muscle; it may, however, affect the nerves supplying one or more of the external muscles. Diabetes is complicated at times by paralysis of the external rectus. Influenza, herpes zoster, and whooping-cough are also rare causes of ocular palsies. Paralysis of the eye muscles is seen in paretic dementia, bulbar paralysis, and in multiple and posterior sclerosis. In locomotor ataxia the paralysis may be transient and appear at an early stage of the disease. Ocular palsies have also been observed after poisoning by lead, nicotine, sulphuric acid, carbon dioxide, and tainted meat.

Paralysis of the Third Nerve. In complete paralysis of the third nerve the upper lip droops, the pupil is partially dilated and immovable, and the power of accommodation is lost. The globe is slightly protruded and strongly diverged externally by the two unaffected muscles (the external rectus and the superior oblique). In *incomplete paralysis of the third nerve, as well as in paralysis of the fourth and sixth nerves,* the diagnosis is made by a study of the deviations and by the character of the diplopia, which has already been referred to.

There is a peculiar form of intermitting *paralysis of the third nerve,* known as ophthalmoplegic migraine, which occurs in the young and is associated with headache and at times with vomiting.

Paralysis of the ciliary muscle, or *cycloplegia,* follows a lesion of the trunk of the oculomotor nerve or of the anterior part of its nucleus. It is quite common as a sequel of diphtheria, and occurs, though rarely, in connection with spinal disease.

Ophthalmoplegia externa and interna refer to paralyses of all or nearly all of the external and internal muscles. As the lesion in this affection is central, it is frequently known also as nuclear paralysis. The *acute form* is due either to an acute inflammatory process in the nuclei or to hemorrhage; while the *chronic* variety depends upon a degeneration atrophy of the nerve-nuclei similar to that seen in progressive muscular atrophy and in chronic bulbar paralysis, with which these conditions may be associated.

In *conjugate lateral deviations* of the eyes, although the visual axes are deviated from the middle line, yet they remain parallel. The cause is generally a cortical lesion the result of apoplexy. A spasm deviation of the eyes in the same direction occurs as the result of irritative lesions involving the association centres or tracts, and also in hysteria.

The Localizing Value of Paralysis of the Orbital Muscles.[1]

Paralysis of the Third Nerve. Ptosis, the most frequent symptom of diseases of this nerve, may be present as a focal symptom in cortical lesions without paralysis of any other branch of the third nerve. This would seem to indicate a special centre for the elevator of the lids; and though not definitely ascertained, such a centre is believed to exist in front of the upper extremity of the ascending frontal convolution close to the centre. Ptosis on the side of the lesion, without paralysis of the other branches of the third nerve, has been seen in disease of the pons, and again, by forming a factor in crossed paralysis, may seem to localize a lesion in the crus cerebri, although when the third nerve is paralyzed by a lesion in this situation it is usually involved as a whole.

Crossed hemiplegia is a term used to express disease of the crus cerebri when there is paralysis of the third nerve on the side of the lesion, with hemiplegia, hemianæsthesia, and often facial and sometimes hypoglossal paralysis of the opposite side of the body.

Complete paralysis of every branch of the third nerve without any

[1] This section is an epitome of the excellent article on the subject in Swanzy's Handbook of Diseases of the Eye.

other paralysis is almost always basal; so, also, are those cases in which hemiplegia, when present, is slight as compared with the degree of the third-nerve paralysis. Lesion of the interpeduncular space and thrombosis of the cavernous sinus also produce third-nerve palsies; but in the latter the other orbital nerves, as well as the fifth and the optic nerve, may be involved as well. Third-nerve paralysis may also be a distant symptom of tumors of the cerebral hemispheres, more particularly if the palsy is accompanied by violent general head symptoms.

Paralysis of the Fourth Nerve. As a symptom of cerebral lesion, *solitary paralysis of the fourth nerve* is rare. It is generally due to a basal lesion. In combination with paralysis of the third nerve it speaks for a lesion in the cerebral peduncle extending back to the valve of Vieussens.

Paralysis of the Sixth Nerve. When *paralysis of the sixth nerve* occurs as the only focal sign, it is probably due to disease of the base. On account of the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure, they more frequently present a distant symptom than any other cranial nerve. Thus paralysis of this nerve is not infrequently a distant symptom of tumor of the cerebellum, whereas paralysis of the third nerve is more likely to be a distant symptom of a cerebral lesion.

Paralysis of the sixth nerve, when simultaneous in its onset with hemiplegia of the opposite side of the body, indicates a lesion in the pons, usually a hemorrhage, on the side corresponding to the paralyzed nerve. Basal paralysis of the nerve is frequently double, especially in syphilis. In combination with paralysis of the facial, paralysis of the sixth nerve is referable to a pontine lesion.

THE EAR.

Subjective Symptoms. Buzzing, roaring, hissing, singing, and other sounds in the ear—*tinnitus aurium*—are symptoms that may or may not be due to disease of the ear. When they are associated with vertigo, *Ménière's disease* should be thought of. They may constitute the *aura* preceding an epileptic attack or the subjective phenomena attending syncope. Many drugs when pushed to the physiological limit cause tinnitus.

The External Ear.

The external ear should always be examined. The thin ear may show the anæmic or chlorotic hue more strikingly than other portions of the body, or the opposite condition may be more vividly shown. *Hæmatoma auris* is seen in general paralysis of the insane and in other forms of insanity. It is a trophoneurosis. The ear is thickened and deformed from the effusion of blood between the cartilages and the perichondrium; discolored; and simulates a subcutaneous effusion due to injury. *Tophi* are observed in the external ears of patients with gouty diathesis. They are small, hard, gritty accretions, seen in the external ear along the margin or in the depressions. They consist of sodium urate.

Discharge. The presence or absence of discharge from the ears must be ascertained when cerebral symptoms or symptoms of infection (pyæmia) are present. Middle ear disease very frequently results in inflammation of the mastoid, which may extend to the sinuses and adjacent membranes of the brain; or the ear suppuration may be the primary focus from which general infection has taken place. It may not be possible in all cases to observe the discharge, which may have diminished or disappeared. Tenderness and œdema over the mastoid, perforation or bulging of the ear-drum, as well as other inflammatory signs, point to the occurrence of suppuration of the middle ear and mastoid cells. It must not be forgotten that a discharge of blood or cerebral fluid from the ear may take place in cases of fractured skull. The ears must also be examined in cases of coma from injury, or if the origin of coma is obscure.

The Auditory Nerve.

The Hearing. The hearing must be tested with the voice, a watch, or a tuning-fork. Normally, the instrument should be heard at an equal distance from each ear. If both sides are equally affected, the hearing of a patient must be compared with that of a healthy person. The ticking of a watch should be heard at a distance of about three feet. In some cases the voice may be easily heard, while the ticking of a watch can be distinguished only with great difficulty. The tuning-fork is used to determine by bone conduction whether deafness is due to obstruction or to disease of the auditory nerve. If it is due to obstruction, the vibrating tuning-fork is heard better on the vertex when in contact with the mastoid process than when held immediately in front of the external meatus (Rinne's test). Obstructive deafness is always due to disease of (1) the external meatus, (2) the tympanic membrane and middle ear, or (3) the Eustachian tube.

Deafness from internal ear disease may be due to affections of the labyrinth—as inflammation, caries, and necrosis—or of the auditory nerve. The tuning-fork is not heard on contact with the skull. The auditory nerve may be diseased in its course, or the auditory centre may be affected. (See Nervous Diseases, Chapter XXXI., Part II.)

It must not be forgotten that certain drugs, as quinine and the salicylates, may cause deafness. Deafness may be an early and premonitory symptom of typhoid fever or cerebrospinal meningitis, and may occur early or late in the course of mumps. Deafness due to *occupation* is worthy of mention. It is not uncommon in blacksmiths, boilermakers, locomotive engineers and firemen. In some instances the patients can hear better in the noise incident to their work than when the surroundings are quiet.

Hyperæsthesia of the Auditory Nerve. Abnormal acuteness of hearing (oxyacoia) occurs very rarely in certain cases of facial paralysis, and not seldom in hysteria. In some individuals suffering from hemicrania or tic douloureux, and in meningitis, the hearing of certain sounds —for example, high musical notes and whistling—is accompanied by pain. Nervous patients often complain of subjective noises, buzzing, roaring, hissing, and singing—the so-called tinnitus aurium.

Paralysis of the Auditory Nerve. No case of absolute unilateral deafness, due to a focal lesion in a hemisphere, has as yet been observed. Deafness from disease of the auditory nucleus is very rare. That due to disease of the peripheral nerve is much more common. We may have a rheumatic auditory paralysis similar to that of the facial nerve, or the deafness may be due to pressure from a tumor or inflammatory exudate at the base of the brain, or disease of the mastoid process of the temporal bone. The localization of the lesion is often extremely difficult. The only positive point is, that labyrinthine disease is apt to be accompanied by vertigo, while in disease of the nerve-trunk vertigo is absent.

Ménière's Disease. Aural Vertigo. We may define aural vertigo as a subjective feeling of motion referred by the patient either to his own body or to surrounding objects, with loss of equilibrium and without unconsciousness.

In this disease, first described by P. Ménière in 1861, the symptoms are paroxysmal vertigo (sometimes so sudden and intense as to throw the patient to the ground), tinnitus aurium, nausea, pallor, clammy sweat, and vomiting. The severity of the attacks varies greatly. There may be momentary unconsciousness; sometimes jerking of the eyeballs, nystagmus, or diplopia is present. The disease is paroxysmal in character, but a slight degree of vertigo and tinnitus often persist between the attacks. Some deafness is present. The attacks may vary in frequency from several in a day to only one in several months.

Paralyzing Vertigo. Gerlier describes a remarkable form of paroxysmal vertigo accompanied by weakness, paresis in the extremities, drooping of the eyelids, marked lassitude, and depression without unconsciousness. It occurs only in men, and is epidemic in the Canton of Geneva, Switzerland.

Hysterical or **functional deafness** is recognized by (1) its association with undoubted symptoms of hysteria; (2) its sudden occurrence after shock, emotional disturbance, or trauma; (3) the absence of a cause in the auditory apparatus for the deafness; (4) impairment of bone conduction and aërial conduction to the same degree; (5) the frequent coexistence of anæsthesia of the pinna and external meatus; (6) the tendency to sudden recovery.

Hysterical deaf-mutism is a rare condition, characterized by (1) sudden origin; (2) absolute aphasia and aphonia; (3) absence of signs of paralysis of the lips and tongue and of any paralytic phenomena except hysterical hemiplegia; (4) preservation of intellectual faculties and power of writing; (5) frequent coexistence of hysterical stigmata; (6) usually rapid recovery.

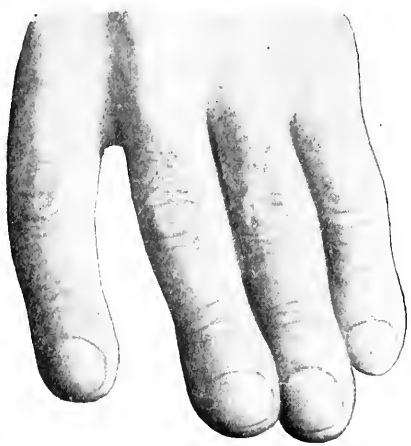
CHAPTER XXII.

EXAMINATION OF THE EXTREMITIES.

THE NAILS.

Shape. The appearance of the nails enables us to estimate the duration of certain diseases, or the time when convalescence began; it also indicates local interference with the nutrition of the parts. Thus

FIG. 24.



Clubbed fingers with curved nails, middle finger slightly flexed. (Original.)

curving of the nails, with clubbed fingers, occurs only in chronic diseases, as phthisis or emphysema, or in chronic cardiac disease and aneurism. In the latter it is sometimes found on one hand only. It is sometimes seen in other chronic wasting diseases. The nails may curve transversely or longitudinally. In transverse curvature the appearance is like that of a filbert, and in longitudinal curvature the nails are said to be incurvated. This change in shape may occur without clubbing of the fingers. The shape is altered in acromegaly and pulmonary osteo-arthropathy. (See Chapter XXVII., Part I.)

Color. *White marks* or *transverse grooves* on the surface are usually seen after an illness, and may indicate the date of recovery. The marks develop at the root of the nail, and as the nail grows the marks approach the tips of the fingers, and thus their position denotes the time that has elapsed since convalescence set in. If they are seen half-way up the nails, convalescence is probably of three months' standing.

Pallor. We get a good idea of the condition of the blood in the capillaries from the appearance of the tissue under the nails. If there is anæmia, pressure on the finger-tips will drive the blood from the capillaries. Stephen Mackenzie's rule, that if such pressure completely empties the vessels so that the nail becomes pale, it indicates that the globular richness of the blood is reduced one-half, is a fair and rapid test of the degree of the anæmia.

The *purplish* and *bluish-black* discoloration of *cyanosis* previously referred to is first seen under the nails.

Capillary Pulsation. Sometimes the *capillaries pulsate*, and this pulsation is more visible under the nails than in any other part of the body except the retina. It may occur in aortic regurgitation, after hemorrhages, and in anæmia.

Nutritive Changes. In various skin affections the nails undergo chronic inflammation with destruction, and the matrix is the seat of acute inflammation in onychia, which may be simple or syphilitic. It may be only a simple inflammation with ulceration, or it may result in loss of the nail and necrosis of the bone.

Deformity of the nails (toe) occurs in acute and chronic myelitis. In *locomotor ataxia* the nails sometimes fall out.

Trophic Changes. In *neuritis* the nail becomes dark and brittle, curved in its long axis, and the growth arrested, while lateral arching takes place. The cutis underneath thickens and the skin at the base retracts. The fingers may be clubbed. When growth is resumed, a distinct roughened line of demarcation is seen. In leprous neuritis there is destruction of the nails and of the distal phalanges.

Atrophy and *ulceration* at the base of the nails, followed by necrosis of the phalanges, is seen in so-called Morvan's disease, which is not really a disease, but a symptom of neuritis or syringomyelia.

Enlargement with thickening and sometimes twisting occurs after fevers, as typhoid; in the course of syphilis; and in sclerodactyly.

The nails in some cases of Raynaud's disease become dry, scaly, and cracked, or hypertrophied.

The *growth* is arrested on the paralyzed side in the hemiplegia from cerebral apoplexy. This is tested by staining the nails of the two hands at the same level with nitric acid; the relative position of the stain upon corresponding nails of the two hands will show whether there has been growth or not. Return of functional power is indicated by renewed growth.

THE FINGERS.

In gout and rheumatism the joints of the fingers are *enlarged* and *painful.* The swellings of the joints of each condition cannot well be distinguished. In gout, *tophi*, hard, white, sometimes glistening masses are likely to be present in the joints or along the tendons, on account of great accumulation of sodium urate. They are more prominent on the dorsal surface of the joints, and sometimes break through the skin, so that the "chalk-like" concretion exudes. It was said by Sir Thomas Watson

FIG. 25.

Subacute or chronic articular rheumatism.

FIG. 26.

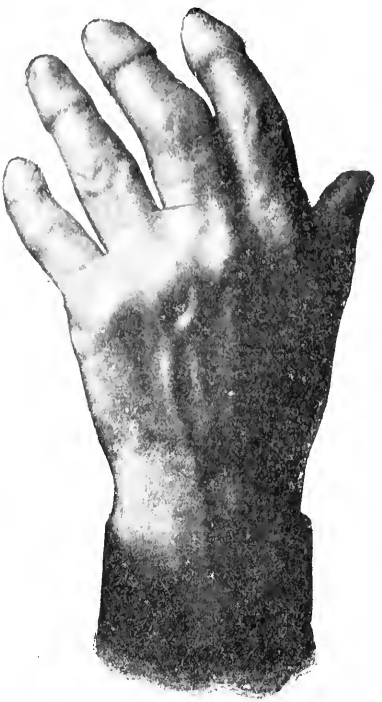
Chronic articular rheumatism, with nodular enlargement of interphalangeal joints. (STENGEL.)

that a gouty subject under his care used his joints to keep tally while playing cards.

Heberden's Nodes. (Haygarth's Nodosities.) The term "end-joint

arthritis" is also applied to this condition. This node belongs to the first of the three divisions of rheumatoid arthritis proposed by Charcot. The nodules develop gradually at the sides of the distal phalanges. The subject may be in good health, or may have had attacks of gout, or have suffered from acid dyspepsia. At first the joints may be a little swollen and tender. The swelling and tenderness may be periodic, and the size may be increased with each fresh paroxysm. The tubercles are seen at the side of the dorsal surface of the second phalanx, the corresponding cartilage becomes soft, the ends of the bone may be eburnated. A moderate anchylosis takes place. The nodules are often considered of good prognostic omen; it is even said that they are a sign of longevity. It is certain that the large joints are rarely involved when these nodules are present. (See Fig. 27.)

Fig. 27.



Heberden's nodes. (Original.)

Clubbed Fingers. The tips of the fingers may be *bulbous*, or *club-shaped*, in some cases of phthisis and other forms of chronic lung disease, and also in chronic heart disease. The condition is most common, however, in bronchitis and phthisis. The clubbing is associated with changes in the nails. (See page 192, and illustration of pulmonary osteo-arthropathy.)

Deviations in Position and Shape. *Eversion* is somewhat characteristic of rheumatoid arthritis; but deviations due to abnormal flexion or extension are the most characteristic digital features of the disease. (Fig. 28.)

Flexion of the first phalanx of the little finger is due to contraction of the palmar fascia or to paralysis of the common extensor from disease of the musculospiral nerve. *Contraction* of the fascia of the hand, causing more or less flexion of the little and ring fingers, is frequently seen, and may be an indication of the gouty diathesis. It is certain that this deformity may be seen in several members or in successive generations of a family in which gout is prevalent. It is called *Dupuytren's contraction.*

Abnormal *extension* is often very marked. *Hyperextension* of the middle phalanx is due to paralysis of the flexor sublimis from disease of the median nerve; hyperextension of the distal phalanges to paralysis of the flexor profundus muscle from disease of the median and ulnar nerves. Extension of the proximal phalanx, with extreme flexion of the two distal phalanges, contributes to form the "claw-hand." (See Muscles.) *Con-*

tractions due to chorea or to central lesions, as posthemiplegic contractions, will be considered under Special Diagnosis.

It is thus seen that the peculiar combined extension and flexion, causing abnormal shape of hands and fingers, may be due to (1) local joint-

FIG. 28.

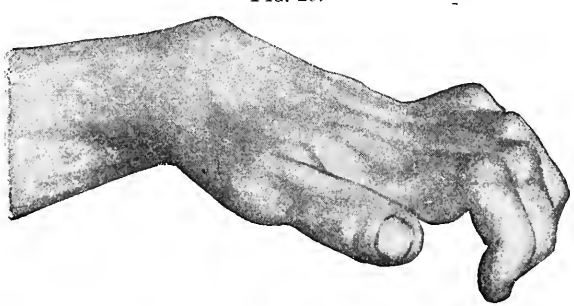
Rheumatoid arthritis. (Original.)

inflammation (subluxations); (2) local neuritis and paralysis; (3) progressive (spinal) muscular atrophy; (4) or rarely idiopathic muscular atrophy.

THE HANDS.

Shape. We bear in mind the variation in the *shape* of the hand in different types of individuals—the broad and heavy hand of the sanguine, the slender, dexterous hand of an individual of the nervous temperament (see Chapter XIX., Part I.), the large joints of the hand of

FIG. 29.

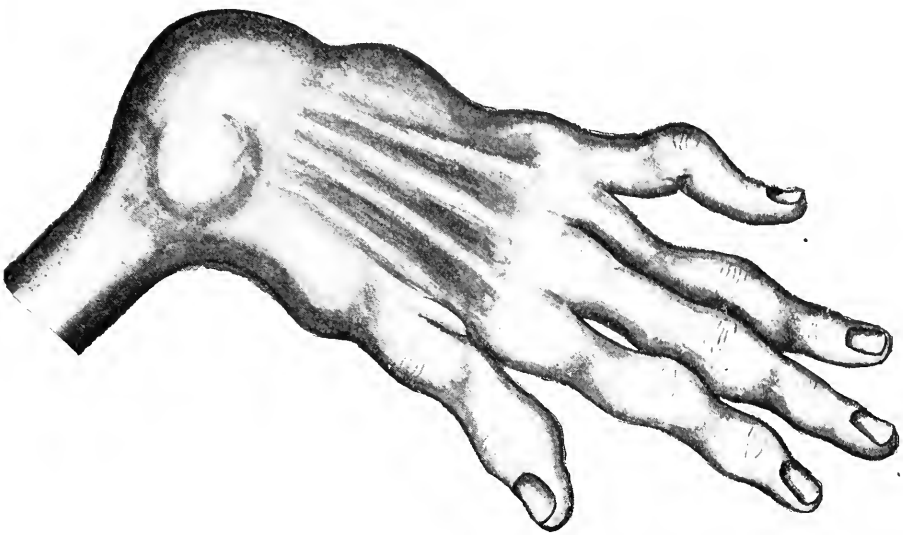


Pseudomuscular atrophy. Claw-hand. (GRAY.)

so-called strumous persons, and the effeminate hand of one who is inclined to tuberculosis, present sharp contrasts. Then, too, the "occupation" hand indicates in a general way the disease the patient is liable to—none more striking than the hand of the miner, the blue-black dottings of

which clearly indicate the possibility of anthracosis. Finally, we note the broad hand and clubbed fingers that are seen in congenital heart disease. The withered hand of age and wasting of the hands, as in

FIG. 30.



Rheumatoid arthritis. The tapering fingers are seen. The phalangeal joints are swollen; many are anchylosed. The wrist is stiff. The muscles are atrophied; the forearm muscles much wasted. (Original.)

FIG. 31.

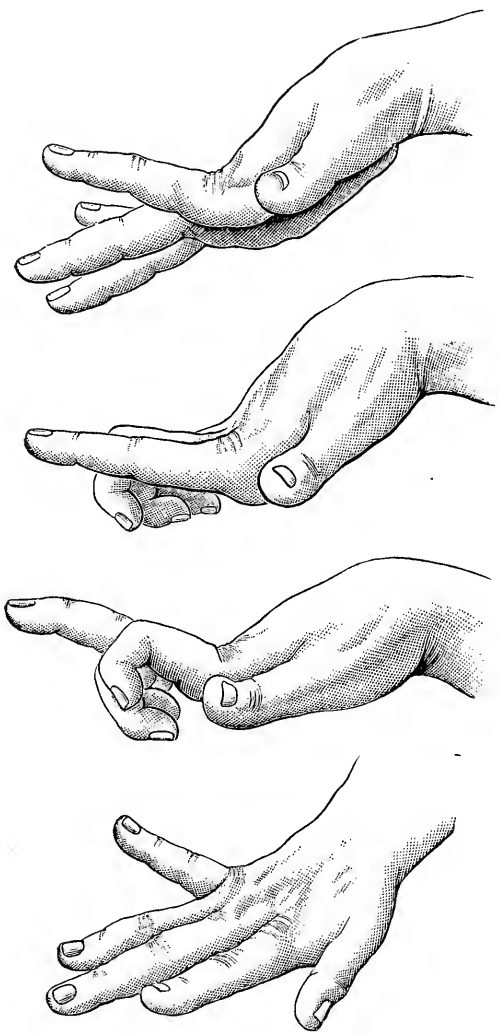
Lead-paralysis affecting the extensor muscles. (GRAY.)

phthisis or malignant disease, need not be referred to, as they are part of the general process.

Presenting more striking changes in shape are the peculiarly deformed

hands seen in affections of the muscles and joints. These deformities will be described in the respective sections. 1. We have the "*claw-hand*" (see Fingers) of progressive muscular atrophy, of inflammation of the ulnar and median nerve, and of chronic poliomyelitis, due to paralysis and atrophy of the interossei and lumbricales muscles, causing dorsal

FIG. 32.



Examples of the positions of the fingers in the movements of athetosis. (STRÜMPELL.)

extension of the proximal phalanges with flexion of the others. 2. The "*seal-fin*" hand of chronic gout and rheumatoid arthritis—spasm of the extensor muscles causing deflection to the ulnar side. 3. The "*ape-hand*," due to wasting of the thenar and hypothenar muscles, when the

thumb assumes a position parallel to the fingers. 4. *Deformity.* The deformed hand of rheumatoid arthritis and that of gout are characteristic. In the former the tapering, shining fingers, the bulbous phalangeal joints, the pallid, clammy surface dotted with freckles, the locked joints, the atrophied muscles, combined with exquisite tenderness of the involved parts, make a picture never to be forgotten. The peculiar deformity occurring in scleroderma is described in the chapter devoted to the Skin. Then we have the deformity resulting from flexion of the hand on the forearm, the forearm on the arm, as seen in *cerebral palsies* of children and in the *hemiplegias.*

Deformities. Deformities of the hand from other causes than the ones just mentioned are often observed. Temporary *contractures* occur in tetany, in temporary hemiplegia or monoplegia, and in paralysis of the extensors. *Dropping* of the hand from the radius toward the ulna occurs in acute poliomyelitis from paralysis of the extensors. Then we have paralysis of the median, ulnar, and other nerves, with their characteristic deformity. (See Nervous Diseases.) So-called *wrist-drop* is seen in *peripheral neuritis* (musculospiral nerve), and may be unilateral or bilateral. The hand hangs from the wrist on account of paralysis of the extensor muscles. Both hands may drop, although it sometimes happens that one is affected from a few days to a few weeks earlier than the other.

The "*spade-like*" hands of *myxœdema*, and the enlarged bones of the hands in *acromegaly* and *pulmonary osteo-arthropathy* are described in other sections.

Movements. One can infer the limitation of movements of the hands in the affections described above. The stiffened and immobile hand of chronic rheumatism, in which enlarged joints are prominent, contrast with the painfully locked hand of rheumatoid arthritis. Involuntary movements, as tremors and spasms, are also observed. The *tremor* of age, of hysteria, of paralysis agitans, of exophthalmic goitre, of mercurial and other intoxications, and of disseminated sclerosis, is most marked in the hands. It is in the hands and arms we see that most significant tremor or twitching with aimless picking at the bed-clothes, described in an account of the typhoid state (Chapter XXXII., Part I.), known as *subsultus tendinum. Twitching* and *spasm* of the hand or arm are seen in convulsive disorders, and may be unilateral or bilateral, as in hysteria, chorea, epilepsy (true and Jacksonian), tetanus, and tetany. When permanent, the convulsions are an expression of a chronic cerebral process, as hydrocephalus. Alternating spasm and relaxation of the fingers, hand, and arm are seen in athetosis.

Local Enlargement. The *swellings* of the hand, inflammatory or œdematous, do not differ in cause or appearance from swellings of the joints or of the subcutaneous connective tissues in other portions of the body. Several exceptions are to be noted: (1) the swelling that attends articular *rheumatism* with involvement of the wrist-joint extends over the dorsum of the hand frequently, while the fingers escape; (2) a localized swelling on the dorsum of the hand is often due to a *ganglion* or enlarged bursa of a tendon; (3) Gubler's *tumor* is a swelling that is seen in wrist-drop from displacement backward of the carpal bones;

(4) long-continued inflammatory swelling, with subsequent rupture of the skin, is seen in *mycetoma*. Finally, traumatic injuries produce tenosynovitis, bone affections, and palmar abscess. It is important to remember that syphilis and gonorrhœa may be causal factors in the production of such processes. (See Chapters XXIII., and XXVII., Part I.)

The Skin of the Hand.

The skin of the hand need not be considered apart from the skin of the rest of the body. It is smooth or rough, dry and harsh, moist and warm, under the same circumstances that affect the skin generally. In rheumatoid arthritis it has been described as peculiar. Both the dorsal surface and the palm are *moist* and very *soft*, and the former is dotted with freckles. In progressive muscular atrophy and exophthalmic goitre the skin is *moist*. The cold, *clammy* skin of one laboring under excitement, as may be caused by the first visit of the physician, is well known.

Color. The color of the hands is significant of the state of the circulation and the condition of the blood. The *blue* finger-tips and the *pallid* hand accompany similar color-changes in the lips, and are early signs of *cyanosis* and of *anæmia*.

The Circulation. Raynaud's Disease. Local Asphyxia. The hands or fingers become pale and intensely cold; they are the seat of numbness, and are without sensation. The term "*dead fingers*" graphically describes the appearance. The pallor usually comes on suddenly, and continues for a variable period. As the pallor disappears, there is a gradual return of warmth and the color changes to a livid-red, dark-blue, or even blackish hue. The paroxysms of alternating pallid and livid hue may occur several times in twenty-four hours. In some cases the lividity becomes so intense that gangrene ensues in small superficial spots, or even involves the whole finger. Pain may or may not be present, and does not increase when the hand hangs down. In my experience it is more frequently present and excruciating at the time the fingers are "dead." The tip of the nose and the lobes of the ears may be affected, and occasionally other parts of the surface. The sensitiveness to touch is markedly lessened. *Raynaud's disease* occurs usually in ill-nourished subjects, or after an acute disease, as typhoid fever. It may be associated with vascular spasm in internal organs, giving rise to epilepsy, hæmoglobinuria, temporary aphasia, or hemiplegia. It is usually worse in cold weather.

Erythromelalgia. Local changes in *color* are often due to neuritis either of the trunk or of the terminal endings of the nerves. When such changes are associated with pain we use the term erythromelalgia. It is characterized by redness of the surface with increased temperature, and is usually seen in the extremities, being limited to the distribution of the affected nerve. It is worse in summer, increased by artificial heat, and aggravated when the extremity is dependent or pressed upon. The redness is attended by burning and extreme local discomfort, in which all sorts of sensation are described—tearing of the finger-nails, pulling or pricking of the skin, twistings of thousands of needles, and other forms of torture. I know of no peripheral pain that is the source of greater agony.

Glossy skin is seen after nerve injuries and neuritis, and in central affections in which the trophic nerves are involved. The skin is shiny, smooth, drawn very tightly over the surface, and sometimes atrophied. Red and pale mottling may be seen. The surface is free from hair. Burning pain precedes and accompanies the change. The finger-tips become pointed. (See Nails.)

THE ARM.

The Vessels of the Arm.

Atheroma of the brachial artery and its branches in the forearm is indicated by enlargement, visible pulsation, and tortuosity of the vessels.

Œdema of one arm may be due to thrombosis of the axillary vein, as in heart disease or the phlebitis of gout; or to occlusion of this vein or of the subclavian from external pressure by a mediastinal tumor, aneurism of the aorta, the innominate or the axillary artery; or more commonly enlargement of the axillary glands.

The Bones of the Arm.

Enlargement of the ends of the bones is due to rhachitis; of the end of the humerus, radius, or ulna, with nocturnal pain, is usually syphilitic. Enlargements or nodes on the shafts of the bone are also syphilitic. Erythema nodosum is common on the extremities. (See the Skin.)

THE FOOT AND LEG.

Enlargement or deformities of the feet and legs may be due to changes in the joint, the bones, or the subcutaneous connective tissue. Hence, we would have *swelling* due to œdema and myxœdema, and enlargements due to acromegaly and pulmonary osteo-arthropathy. The chapters so frequently referred to will contain a discussion of these subjects, and the chapter on Joints will give a description of all articular changes. It must be recalled that *pain* may be due to flat-foot or to neuralgia of the third interosseous nerve. (See Pain.)

Flat-foot must always be looked for when inability to walk is complained of. Changes in the shape of the foot from muscular affections, bearing in mind that "claw-foot" is allied to "claw-hand," are found in progressive muscular atrophy and in Friedreich's ataxia.

Three *nutritional changes* take place in the feet that are of diagnostic significance: *perforating ulcer* of the foot, a trophic change occurring in locomotor ataxia; *gangrene* the result of endarteritis (usually senile) or occurring in the course of diabetes mellitus; *mycetoma*, or "Madura foot." Perforating ulcer usually begins as a blister, and goes through the successive stages of abscess and finally ulcer.

The *nails* of the feet are subject to the same changes that take place in the nails of the fingers.

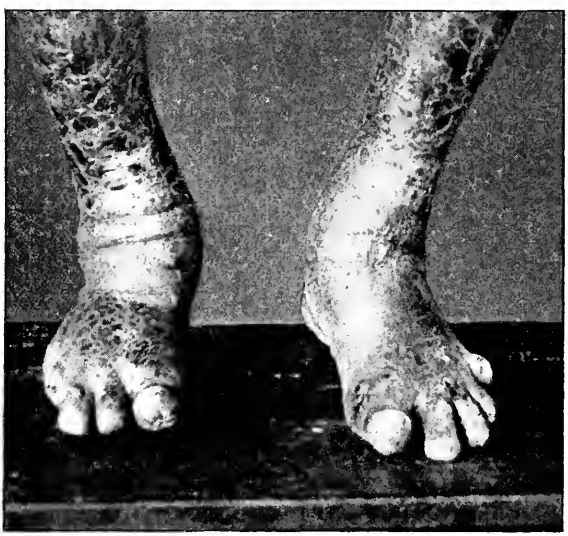
The Vessels of the Foot and Leg.

Varicose veins follow phlebitis or intra-abdominal pressure from tumors, fecal accumulation, or pregnancy. Phlebitis of the femoral vein is characterized by pain and tenderness of that vessel and by œdema of the foot. Deep-seated phlebitis may cause œdema and congestion of the foot and knotted veins. The femoral artery often pulsates and is the seat of a thrill simulating aneurism; the vessel is thickened and rigid. Absence of pulsation with localized pain is due to closure from thrombus or embolus, and is followed by gangrene.

The Bones of the Foot and Leg.

Bony growths or nodes are due to syphilis. Local or more general enlargement of the tibia, with pain, occurs in syphilitic periostitis during the tertiary stage and in the hereditary form. Swelling of the periosteum

FIG. 33.



Appearance of the legs in [a case of] leprosy showing pigmentations, dermatitis, ulcers, and amputations.

with redness, heat, pain, and high fever, is due to an infection, and is often followed by suppuration and necrosis. Red, swollen, and indurated areas over the extremities may be due to erythema nodosum, scurvy, leprosy, or some form of purpura. (For œdema, muscle-changes, and bone- and joint-lesions, see the respective sections.)

Cold Hands and Feet.

Coldness of the extremities is a common and often a serious complaint. It is natural to expect a peripheral coldness when the central organ of circulation is weakened, also in collapse, in hemorrhage, in shock, and during

the final hours preceding death. But we also see it in organic disease of the heart with impairment of the circulation. It is a common vasomotor condition in nervousness independent of hysteria. It is a marked feature in Nothnagel's *angina pectoris vasomotoria*, as well as in true and false angina pectoris. A visit to a physician or excitement from any cause is likely to be attended by coldness of the hands and feet. Under these circumstances the extremities are often bathed in a cold and clammy perspiration. In senile endarteritis coldness of the hands and feet is common.

The poisons of gout, rheumatism, and other diseases may cause cold hands and feet by irritating the peripheral and vasomotor nerves.

In gastric and intestinal dyspepsia the absorption of toxic principles, as leucomains, may produce this symptom.

Sensation.

Changes of *sensation* in the skin of the extremities will not be considered in this section. They will be taken up in the chapter devoted to the diseases of the nerves. It is sufficient to state that circumscribed *anæsthesia* due to causes limited to the skin is seen in morphœa, in the anæsthetic form of leprosy, and in certain ischæmic states (urticaria). It is accompanied by loss of tactile sensibility. *Hyperæsthesia* and *paræsthesia* attend various local affections, but save in nervous diseases are without diagnostic significance.

CHAPTER XXIII.

EXAMINATION OF THE SKIN.

INSPECTION.

Color. By inspection we determine local and diffused *color*-changes, *hemorrhages*, and the *lesions* indicated on page 215. A hand magnifying-glass assists the eye, and the microscope aids in recognizing parasitic affections. By palpation we determine the *nutrition*, *temperature*, and degree of *moisture* of the skin, and the physical characteristics of lesions. The portions exposed to the air exhibit more varied and pronounced changes of color than parts that are covered. The changes in color herein described refer more particularly to the face and hands. The color of other parts partakes of the same tint as that of the face, other things being equal, except that the intensity is less. Comparison of the two should always be made, and the mucous membranes should be examined as a control observation. The conjunctivæ, lips, and the mucous membrane of the mouth are sufficient, always remembering the possibility of hyperæmia of the conjunctiva from other causes. The *exposed surface*, the *age*, and the *habits* influence the normal color.

Exposed Surface. Local color-changes of the *face* will be particularized in this section. It is not to be forgotten that the color varies with the type—whether blonde or brunette—and that variations at times escape recognition in brunettes.

Age. The skin of a healthy *child* is of a faint pink color; as age advances it loses its fresh appearance and becomes paler, except in those whose occupation exposes them to atmospheric influences. In the latter, the skin becomes weather-stained, and may assume a mahogany or reddish-brown hue. In *old age* the color is apt to deepen and become duller, while the loss of subcutaneous fat allows the skin to lie in folds, especially about the jaws and neck, and wrinkles are marked, especially between the eyebrows, over the nose, and at the angles of the eyes and mouth.

Habits. Apart from these changes, which are physiological or the result of occupation, the skin exhibits changes which are the result of the *habits* or health of the individual. Some persons, especially if blondes, retain to old age the fresh, pink skin of childhood. In others is seen early a dull, *muddy complexion.* This is common in those who use coffee to excess and are habitually constipated. In others digestive derangements, particularly constipation, uterine disorders, and gout tend to produce, in addition to a muddy complexion, crops of acne pustules and comedones, or black-heads. It must be admitted, however, that some persons preserve a fresh complexion in spite of marked digestive disturbance. Con-

siderable congestion of the superficial bloodvessels, giving a person a *florid* appearance, may be due, especially in the young, to alcoholic excesses; and popular belief connects such an appearance, when coupled with a tuberous nose and a crop of angry-looking pustules, with prolonged use of spirits.

Secretion. The *sebaceous glands* of the skin of the face merit but a passing notice. Deficiency, excess, or alteration of secretion is usually due to some local cause. Excessive secretion of sebaceous matter, known as *seborrhœa*, or steatorrhœa, is seen in two forms: first, with oily exudation; second, with drying of the secretion and the formation of crusts. It may be more pronounced in strumous subjects. The opposite condition, or *asteatosis*, is seen in wasting diseases, particularly diabetes, and in xeroderma and ichthyosis.

Redness. Physiological hyperæmia has been spoken of. The color is intensified when the capillaries are over-filled or the blood-current is unusually rapid. The hyperæmia may be general or local, and is due to dilatation of the capillaries, possibly from nerve-influences. *General* hyperæmia is seen in fever, atropine-poisoning, and intoxication by organic poisons derived from food or the result of intestinal putrefaction. *Local* hyperæmia attends the phenomenon of blushing, and comes and goes in nervous persons with every psychical impression. Rarely in neurasthenics the hyperæmia may be extreme, amounting almost to an erythromelalgia. Abnormal redness may be diffused over the whole face or may present the circumscribed flush of phthisis; the local deep-red area, on one cheek, of pneumonia; the evanescent flush of anæmia, with cardiac palpitation; or the creeping flush, with raised border, of erysipelas, appearing on the bridge of the nose or at the nostril. In phthisis, moderate excitement or exertion, the taking of food, or the onset of fever, tinges the cheek with a hectic flush. In migraine, the burning may be limited to one side of the face. Capillary congestion on the cheeks or on the tip of the nose occurs with the endarteritis of the aged, but is seen also in early life in cases of hepatic cirrhosis or obstruction of the hepatic circulation from other causes.

Pallor. It is caused by diminution of the amount of blood in the capillaries or because its richness in hæmoglobin has been reduced. Diminished amount of blood in the capillaries occurs from active contraction or spasm of the arterioles, from hemorrhage, or from weak heart.

Temporary Pallor. The pallor, therefore, is usually *acute* or *temporary*, and may be recurrent. It attends fright, syncope, or nausea and vomiting. It occurs also in acute poisoning, in acute disease, such as diphtheria, and in hemorrhage. The pallor due to loss of blood may be instantaneous if the hemorrhage is sudden and large, or develop gradually if it is small and continued over a long period. The onset of sudden pallor is of diagnostic significance in diseases in which hemorrhage may occur, as aneurism, gastric or intestinal ulcer, and typhoid fever. Symptoms of collapse are seen with this form of pallor.

Permanent Pallor. Pallor of long duration, or *chronic pallor*, if we may so term it, is seen in a number of diseases. It is characterized by gradual onset. The number of red corpuscles is diminished, with destruction of

the hæmoglobin. *Pallor* is the normal condition in a large number of individuals, particularly in those who lead a sedentary life and are confined within doors. Generally speaking, pallor is characteristic of blood affections ; but it is not necessarily present in *leukæmia*—indeed, the cheeks and lips may be red. Moreover, *anæmic people are not necessarily pale*, and *pale people are not always anæmic*. Striking examples of pallor are seen in chronic *Bright's disease*, in *cancer*, in *chronic poisoning*, as from lead or arsenic, in *chronic catarrh* of the stomach or of the bowels, and in *chronic infectious processes*, as tuberculosis and syphilis. The pallor that attends *Bright's disease* is usually associated with slight puffiness under the eyelids, or local dropsical accumulations elsewhere. In chronic poisoning with *lead*, pallor is associated with a blue line upon the gums and wrist-drop ; while in arsenical poisoning there are frequently associated puffiness of the eyelids and looseness of the bowels.

Degrees of Pallor. *Greenish hue :* while *paleness* is recognized as the fundamental or prevailing color of the skin in many of the above-noted affections, an additional tinge gives a characteristic hue to the skin ; thus in *chlorosis* there is a *greenish* appearance of the face, which is in striking contrast to the pearly colored conjunctivæ. *Yellowish hue :* in *carcinoma* the *yellowish* tinge often causes the pallor to be mistaken for jaundice. In *pernicious anæmia* a *straw-colored* appearance of the skin has been frequently described, which may cause the disease to be mistaken for carcinoma. It is worthy of remark that the cachectic pallor in carcinoma is not likely to occur unless there are primary or secondary deposits in the gastro-intestinal tract or the liver, and it is well known that pernicious anæmia is usually secondary to gastric or hepatic disorder. The peculiar hue of the pallor, therefore, may be due to a common cause in these affections.

It is not well to lay much stress upon the variations in hue of the pallor. They are not of diagnostic importance in themselves, but only when associated with the characteristic symptoms and signs of the respective affections in which the respective hue occurs.

Abnormal Color. I. The Yellow Skin—Jaundice. The yellow coloration is seen not only in the skin, but also in the scleræ (see the Eye) and the mucous membranes. The discoloration of the skin is not difficult of recognition. It varies in shade from a slight yellow hue to yellow-green or olive-green, and in many forms of jaundice, to brownish-yellow. The yellow hue of the skin in jaundice may be preceded and is always accompanied by tingeing of the conjunctivæ ; its presence in this situation confirms the observation. The mucous membrane under the tongue early gives evidence of jaundice ; or, if the lips are everted and a glass slide pressed evenly on the surface, the yellow discoloration of the mucous membrane will shine through.

The yellow tint of the conjunctivæ must not be confounded with the some color due to subconjunctival fat. The latter is not uniform in the conjunctivæ, but may occupy cone-shaped areas.

The *physiological yellow* color of the skin that is seen in infants shortly after birth is not a true jaundice, but in all probability arises from excessive destruction of red corpuscles in the over-congested skin. On light

pressure with the finger the color changes. It fades from shades of yellow into the genuine flesh-color. The conjunctivæ are natural, and the urine is free from bile-pigment and the feces are normal. These symptoms suffice to differentiate the condition from true jaundice.

Jaundice is a symptom due to a number of diseases. In the first place, it is most frequently due to disease of the liver; this form is known as *hepatogenous* jaundice. It may possibly be due to destruction of the corpuscles of the blood and liberation of the hæmoglobin, the so-called *hæmatogenous* jaundice. The various causes of the former will be considered under diseases of the liver. The latter is said, not without objection, to be due to destructive agencies in the blood, such as ptomaïns, which are absorbed in gastro-intestinal disease, or to poisons that develop in the course of pyæmia, yellow fever, malaria, and relapsing fever; it may also be due to snake-bite or to poisons that are imported, as snake-venom, mineral poisons, chloroform, ether, or chloral. In both forms the yellow color of the skin is due to the deposition of bile-pigment (bilirubin) or blood-pigment in the cells of the rete mucosum.

Other symptoms due to the same cause are associated with *hepatogenous* jaundice. In doubtful cases their presence may be of diagnostic value in determining the nature of the yellow color of the skin. These symptoms are: (1) *Itching.* This symptom is intolerable; the surface of the body is often covered with scratch-marks on account of the irritation of the peripheral ends of the nerves by bile-pigment. (2) *Slow pulse.* (3) *Secretions* and *excretions.* The *saliva*, or expectoration, if present, is bile-tinged; and the color of the *urine* is dark from the presence of the pigment. (See Urine.) While the excretions are all tinged with bile in the hepatogenous form, the feces are free from bile; hence they are pale or of an ashy color. On account of the absence of bile in the intestines its physiological influence is absent, and therefore *flatulency* from fermentation becomes an important symptom.

II. The Blue Skin—Cyanosis. This peculiar hue is recognized without difficulty. The bluish or bluish-red appearance of the skin is first seen at points farthest from the central organ of circulation, as in the extremities. The mucous membranes, in which the capillary circulation is readily seen, also exhibit the change early. It is early seen also in the finger-tips, particularly underneath the nails, about the phalangeal joints, and in the lips. Subsequently the entire surface of the skin may become dusky or cyanosed as its cause increases in degree. Its onset, it is said, can be anticipated by the state of the veins on the under part of the tongue; overfilling or extreme distention of these vessels always occurs in cyanosis. At first the color, wherever situated, usually disappears on pressure, but as the hue deepens it remains in spite of pressure.

Causes. Cyanosis is (1) respiratory, due to over-filling of the veins and capillaries with blood not sufficiently oxygenated, or (2) vascular, to an excess of venous blood, oxygenation not being interfered with.

1. *Respiratory.* All conditions which interfere with the aëration of the blood cause more or less cyanosis. Practically, sufficient air cannot get to the blood, or sufficient blood to the air. Obstruction of the air-passages, diminution of the respiratory area, and diminished or inefficient

respiratory movements prevent oxygen from getting into the blood; interference with the circulation in the lungs prevents the blood from getting air. The two processes are often combined.

a. Obstruction of the Air-passages. This may occur in the upper respiratory tract, or in the capillary bronchi. (1) *Faucial* obstruction, by pharyngeal abscess or tonsillitis, or, in rare cases, by diphtheria, causes moderate cyanosis. (2) Obstructive *laryngeal* diseases produce cyanosis varying in intensity with the degree of obstruction and its persistence. The cyanosis is of *short* duration in spasmodic croup and in laryngismus stridulus; it is prolonged in the more persistent inflammatory affections. Its gradual onset, in moderate degree, as seen by the purple lips or dusky finger-tips, is of serious prognostic import in the course of tubercular laryngitis, even if symptoms of grave obstruction have not arisen. (3) *Tumors*, pressing on the trachea or bronchi, narrowing the air-channel, cause cyanosis. The tumors may be situated in the neck, as the thyroid gland, or within the mediastinum. (4) *Spasm* of the *bronchi*, as in asthma, *occlusion* of the bronchioles, as in bronchitis, both acute and chronic, and particularly the grave forms of capillary bronchitis in childhood, cause cyanosis. Chronic cyanosis is most diagnostic of *emphysema*. (5) *Foreign bodies* anywhere in the upper regions of the respiratory tract are fruitful sources of cyanosis.

b. Diminution of the Respiratory Area. Cyanosis from this cause occurs in *pneumonia*, in *œdema* of the lungs, in *tuberculosis*, and in all forms of *pleural effusion* and of *intrathoracic tumors* compressing the lung. It is an important diagnostic feature of acute tuberculosis.

c. Diminished or Insufficient Respiratory Movements. Deficient chest-expansion, because the action of the respiratory muscles is interfered with, lessens the respiratory area. This interference may be caused by (1) muscular or pleuritic pain; (2) impaired action of the diaphragm, on account of upward pressure by accumulations in the abdominal cavity, as large peritoneal effusions, an enlarged liver or spleen, or an abdominal tumor; (3) by paralyses: in *bulbar paralysis* and *peripheral neuritis*, in *paralysis* of the *diaphragm*, and in *spasm* of the muscles of respiration (as in tetanus) there is diminished respiratory movement. In forms of progressive muscular atrophy and in other rare affections of the muscles, as trichinosis, cyanosis is also observed.

d. Obstruction of the Pulmonary Vessels. Interference with the circulation within the lungs, from pressure on the pulmonary artery or vein by aneurism or mediastinal tumor, or from disease of the heart itself, is a most frequent cause of cyanosis. In affections of the heart it is not seen until—in the case of valvular disease, for instance—compensation is lost and the right heart is dilated, causing an accumulation of blood in the lungs. In the latter condition the bronchitis of passive congestion of the mucous membrane is an additional cause for the cyanosis.

2. *Cardiovascular.* Obstruction to the flow of venous blood anywhere in the circulation will lead to the development of cyanosis. This is the *cyanosis* of *passive congestion*. Cyanosis due to causes mentioned above is always general. Cyanosis arising from the causes presently to be detailed may be *general* or *local*, depending upon the seat of obstruction.

PLATE II.

Addison's Disease.

General cyanosis may occur in (1) *congenital heart disease;* (2) in *valvulitis,* when compensation is lost and dilatation has taken place; (3) in *incompetency* of the valves from dilatation; (4) in *weak heart* or enfeebled action from pericardial effusion. In congenital heart disease the cyanosis is so great and so persistent that the affection has been termed "blue disease" or *morbus cæruleus.*

Local cyanosis is seen when there is obstruction of the venous trunks from external pressure or from disease of the venous wall, causing thrombosis. It may be limited to the head and upper extremities in obstruction of the descending cava by tumor or aneurism; or to the lower portion of the trunk and the lower extremities in obstruction of the ascending cava by pressure from tumors within the abdomen or thorax. One extremity may be the seat of local venous stasis from pressure upon the veins, or their occlusion by thrombosis: the arm in cases of cancer of the breast and axillary glands, the leg in cases of femoral phlebitis, represent typical forms of venous stasis. A striking form is due to causes affecting the vasomotor nerves, giving rise to peripheral capillary spasm. (See under Fingers, Raynaud's Disease.)

III. The Bronzed Skin—Pigmentation. A bronzed hue of the skin is seen in sunburn; in toxic conditions, as Addison's disease, diabetes, and certain forms of liver disease; in uterine disease and in pregnancy; in argyria or the deposition of silver in the skin; in the unwashed (vagabonds' disease); and in parasitic affections of the skin.

General Bronzing. When the entire surface of the body is bronzed, it is usually due to *Addison's disease* (see page 855), although it must be remembered that extension over the surface may be very gradual, and some regions may be more deeply bronzed than others.

Local Bronzing. *Sunburn.* The discoloration of the skin in Addison's disease must not be confounded with that of *sunburn.* The latter discoloration is limited to parts that are exposed to the sun, is more uniform, and the mucous membranes are free. Moreover, the anæmia and debility of Addison's disease do not attend it. The pigmented areas in the mucous membrane of the mouth, seen in a certain class of negroes, must not be mistaken for the pigmentation of Addison's disease. (See Plate II.)

Filth. In persons living in filth general discoloration of the skin takes place, known as "*vagabonds' disease*"; but because it is so general and the skin is rough and thickened, and other evidences of filth are seen, it can easily be recognized.

Tuberculous Peritonitis. In certain cases of *tuberculous peritonitis,* even if the adrenals are not involved, a peculiar brown discoloration simulating Addison's disease is present.

Scleroderma. In *scleroderma* pigmentation occurs, although rarely.

Chloasma. The pigmentation that occurs in *uterine disease* and in *pregnancy* (uterine chloasma) resembles the bronzing of Addison's disease. It is usually confined to the forehead and cheeks and the normally pigmented areas of the skin. The mucous membranes are not affected, although in pregnancy there may be the characteristic blue discoloration of the vaginal mucous membrane. The vomiting and weakness that

attend pregnancy may sometimes lead to confusion—vomiting occurs early, pigmentation late in pregnancy.

Parasitic Affections. The affections just described must not be confounded with the discoloration—yellowish brown in hue—of *tinea versicolor*, a parasitic skin disease. The latter is recognized by its color and irregular dissemination. It especially occupies the chest and spreads to the abdomen. It rarely ascends above the neck. It does not usually, therefore, occur in parts exposed to the air or in parts that are the seat of normal pigmentation. Then, again, the surface desquamates in brownish scales. Examination of the scales in a drop of dilute liquor potassæ under the microscope shows both spores and mycelium of the fungus *Microsporon furfur*. Another skin affection is attended by bronzing—*leucoderma*.

Diabetes. In *diabetes* bronzing is often seen independently of any parasitic invasion of the skin, and apparently the result of the cachexia. It is possible that it is due to the cirrhosis of the liver which causes the glycosuria. But if the *pancreas* is primarily at fault, the skin change is more likely to occur.

Liver Disease. In certain forms of hepatic cirrhosis, as so-called Hanot's, or the hypertrophic form, bronzing undoubtedly the result of blood-destruction, *hæmochromatosis*, is seen in some instances. In the later stages of *jaundice* the dark-green, olive, or black hue of the skin might be mistaken for the general bronzing of Addison's disease, but the appearance of the conjunctiva is sufficient to indicate the cause of the bronzing.

Argyria. If silver nitrate is administered over a long period of time, fine black particles of the metal or its albuminate are deposited in the kidneys, the intestines, and the skin. The corium is the principal seat of the deposition. The discoloration of the skin is gray or grayish black. It is not changed by pressure, and is usually limited to the face and hands. Small specks may also be noted in the mucous membrane of the mouth. The cornea and nails are not affected. The subjects are usually in good health, although the presence of the discoloration, if seen in a patient with coma, would point to the possibility of epilepsy, on account of which the drug had been taken.

Freckles. Freckles are not usually of special diagnostic significance. Their occurrence in an unusual degree on the back of the hand and forearm has been observed, however, in case of rheumatoid arthritis.

Hemorrhages of the Skin.

Hemorrhages produce discolorations of the skin which vary in hue from deep red to yellow brown, according to their depth beneath the surface, and to the degree of absorption that has taken place since the hemorrhage occurred. They may be raised above the surface of the skin. They do not disappear upon pressure.

Hemorrhages in the skin are called, according to their size, *petechiæ*, *ecchymoses*, *vibices*, and *hæmatomata*. The petechiæ and ecchymoses are apt to appear in the hair-follicles, and vary in size from that of a pinpoint to that of a split pea.

Mode of Recognition. They are distinguished from erythematous and other eruptions because they do not disappear upon pressure, and are often slightly elevated.

Vierordt advises the following test to distinguish hemorrhages from erythemas: press a piece of glass (a microscope slide) upon the suspected spot; a hemorrhage is rendered more distinct, while the surrounding part becomes more anæmic; an inflammatory hyperæmia, on the other hand, disappears.

Significance. While subcutaneous hemorrhages are easily recognized, the determination of their cause is more difficult, and must depend upon the phenomena with which they are associated. The cause of the hemorrhage is often suggested by its situation; thus hemorrhages about joints are usually purpuric or hæmophilic.

1. **Hemorrhage with Fever.** Subcutaneous hemorrhages in the infections are due to changes in the quality of the blood, and indicate the severity of the infection, or to obstruction of the bloodvessels with emboli. To the former class belong *cerebrospinal fever* and *measles*, *variola*, *scarlatina*, and *rheumatic fever*. In the exanthemata they precede, accompany, or even replace the characteristic eruption, the latter being darker in color than normal. Hemorrhages usually take place at the same time from the mucous membranes; perhaps the nares are occluded, and the mouth and fauces filled with clotted blood. In milder infections sordes collect in the mouth only. They indicate the degree of malignancy of these affections. To the same class of affections belong *epidemic hæmoglobinuria* and *morbus maculosus neonatorum*, diseases of newborn infants but little understood, although no doubt of an infectious nature. To these may be added the severe forms of *purpura hæmorrhagica*, attended by fever, marked visceral disturbances, skin eruptions, and great œdema, the symptom-complex being the result of an obscure toxæmia.

2. **Hemorrhage with Anæmia.** Hemorrhages occur in all forms of *anæmia* attended by debility. In idiopathic or pernicious anæmia they are usually small, but may become extensive. They occur on the extremities, and, usually, on the dorsum of the feet or hands. There may also be retinal hemorrhages. They are also seen in the secondary anæmia that arises in the later stages of tuberculosis and of carcinoma, particularly of the stomach; in the later stages of Bright's disease, and of cirrhosis of the liver.

Scurvy is an affection characterized by anæmia, debility, and wasting, in which there are hemorrhages under the skin as well as from the mucous surfaces. The gums are particularly affected. They bleed easily. Hemorrhages also occur in the deep lymphatic spaces, in the muscles, underneath the periosteum, and in the joints. In *scurvy-rhachitis* similar hemorrhages are seen. (See Chapter XXVII., Part I.) When the subcutaneous hemorrhage is accompanied by profuse hemorrhages from other portions of the body and there is a history of such tendency in the family, the affection known as *hæmophilia* is likely to exist. Anæmia is a characteristic accompaniment of this condition.

3. **Cardiovascular Hemorrhages.** Hemorrhages due to obstruction of the vessels are known as hemorrhagic infarcts, and are seen in *pyæmia*

and *ulcerative endocarditis*. The hemorrhages are small, sometimes elevated, more abundant on the extremities, but also distributed over the trunk; they are seen in the mucous membranes of the lip and cheek, and on the tongue, in the conjunctivæ, and, on ophthalmoscopic examination, in the retina.

CAUSES.—The association of chill, fever, and sweat, the presence of pus in some region of the body, and the characteristic joint-affections point to pyæmia. On the other hand, if the hemorrhage is due to ulcerative endocarditis, the physical signs of that affection render the recognition of the cause comparatively easy. Finally, in *rheumatic fever* with involvement of the joints we have the occurrence of *purpura*. (See Erythema.)

4. Hemorrhage in Central Nervous Disease. NEURITIS. Purpura in some instances is believed by Mitchell to be due to primary disease of the nervous system; certainly we do see it in neuritis, in Raynaud's disease, in myelitis, and in locomotor ataxia. It may occur in hysteria, when drops of blood ooze through the skin at the time of the attack (hæmatidrosis).

5. Hemorrhage of Toxic Origin. The *venom* of snakes causes hemorrhages under the skin. In *jaundice* the blood is disintegrated and hemorrhages take place. In malignant types the mucous membrane bleeds and sordes collect on the tongue, lips, and gums. To the same class belong the subcutaneous hemorrhages that follow the administration of certain *drugs*, as copaiba, potassium iodide, quinine, and belladonna. (See Medicinal Rashes.)

6. Purpura. Subcutaneous hemorrhages occur in many instances in which thus far it has been impossible to classify them correctly. The term purpura is employed to designate them. They are known as primary purpura and secondary purpura. To the latter belong many of the varieties described above. For their consideration see Chapter III., Part II., Special Diagnosis.

7. Hæmophilia. (See Special Diagnosis, Chapter III.)

Eruptions of the Skin.

Eruptions may be *primary* and *local* (from causes operating directly on the skin) in the sense that they occur independently of any internal affection; or *secondary*, the resultant of an internal morbid process. The anatomical changes are the same, and morbid processes in the skin do not differ from such processes in other epithelial structures. The anatomical and physiological peculiarity of the part causes the difference in the phenomena. Hence, anæmias and hyperæmias, inflammations, acute or chronic with or without exudation; hemorrhages, atrophies and hypertrophies, new growths, and parasitic affections are found in both. But instead of a painless inflammation with transudation of mucus, as in mucous membrane inflammation, we have a more or less painful inflammation, with itching (nerve-supply), and with sebaceous and sudoriferous gland exudation. Otherwise the same symptoms attend each; but ocular examination of the inner mucous membranes is not possible.

While the reader is referred to special works on skin diseases for a description of the primary or local skin affections, the secondary affections will be briefly noted. It must not be forgotten that the local affections—eczemas, parasitic disease, etc.—are modified by the general condition of the patient.

Clinical Significance. To determine the clinical significance of the secondary or symptomatic skin lesions we must note carefully, first, the special character of the eruption, the *nature of the lesion;* second, its *distribution*—(*a*) in the layers of the skin, (*b*) over the surface of the body; third, its *association* with other symptoms.

I. The Nature of the Lesion. Observation concerning the nature of the lesion includes (1) its anatomical character, (2) the order of appearance, (3) its uniformity, and (4) the mode of invasion.

A knowledge of anatomical lesions is essential to enable one to define exactly the morbid process and determine the primary cause of the lesion. For a long time the lesions were divided into primary and secondary. The lesions known as scabs, scales, abrasions (raw), scratch-marks, and ulcers are always secondary. Scars and maculæ appear latest. The other lesions herein described are primary. The writer follows Pye-Smith in the description of them, as well as in most of the matter appertaining to cutaneous affections.

1. *Hyperæmia, or congestion.*

a. Mere over-fulness of the vessels from paralysis of the vasomotor nerves, with redness and heat, but without the exudation and tissue-changes which accompany inflammation. This hyperæmic blush, readily produced in the physiological laboratory, is rarely seen as an uncomplicated morbid condition (*e. g.*, Trousseau's *tache cérébrale*).

b. *Active, arterial, or inflammatory hyperæmia* varying in color from brilliant scarlet to rose-pink, and combined with heat, tingling, or other sensations.

c. *Passive, venous, or congestive hyperæmia*, dependent upon retarded circulation and distended venules. The color is purple, bluish, or livid, the surface is cold, and there are no painful sensations.

2. *Pimple, or papule.* A small solid elevation of the skin.

a. The acute inflammatory papule.

b. The chronic large inflammatory papule, discrete or confluent.

c. A solid non-inflammatory papule.

d. Solid elevations of the skin, which may be called false papules.

3. *Vesicle.* A visible cavity in the skin filled with transparent liquid.

4. *Pustule.* A cutaneous abscess.

5. *Bulla, or bleb.* A very large vesicle.

6. *Scab, or crust.* A dried-up concretion of the contents of a vesicle, pustule, or bleb.

7. *Scale* (*squama*). A dry flake of epidermic cells.

8. *Wheal* (*pomphos*). A flat solid elevation of the skin, much larger than a papule, and of ephemeral duration.

9. *Scratch-mark.* An injury to the skin, of linear form and curved outline.

10. *Raw.* A surface which has lost its horny layer of epidermis.

11. *Chap* (*rima*). A crack or fissure which goes through the epidermis.

12. *Sore* (*ulcus*). The result of destruction by inflammation, which has reached below the Malpighian layer and has destroyed the papillæ.

13. *Scar* (*cicatrix*). The result of the healing process after an injury or disease sufficiently deep to destroy the papillæ of the part.

14. *Nodule.* A solid elevation of the skin larger than a papule and seated in its deep layer.

15. *Stain* (*macula*). A patch of increased pigmentation of the skin.

16. *Hemorrhage* (*ecchymosis*). When a bloodvessel of the cutis vera gives way, a dark-red or purple mark is produced, which (like macula) does not disappear on pressure.

The recognition of the exact anatomical lesion is not sufficient for diagnosis unless the *mode of invasion* is observed at the same time. The rash often spreads from a single focus, or numerous foci appear and coalesce. The lesion is studied best in the most recent part. Not only is the mode of local invasion to be noted, but also the *uniformity* of the anatomical lesion. Often, instead of a simple lesion, various kinds are present at the same time, or they develop in successive order; thus in smallpox we have first the papule, then the vesicle, and finally the pustule.

II. Distribution. The location of the lesion in the various layers of the skin, and the distribution over the surface of the body, must be observed.

The layers of skin: (1) The horny layer of the epidermis manifests the pathological changes of hypertrophy, atrophy, dryness, or desquamation of the cuticle. Dead scales result, in addition to the hypertrophies and atrophies indicated in the outline. (2) The eruption in a large number of cases is limited to the living Malpighian layer of the epidermis and to the papillary layer of the cutis. The hyperæmias (erythemata), and inflammations of all kinds, are confined to these layers. In this situation they never leave scars. (3) The deep layer of the cutis is so intimately connected with the subcutaneous tissue that morbid changes in it involve the latter, and even extend more deeply. The affections are more severe, but less numerous than affections of the superficial layers, and are always followed by cicatrices. The changes in the sweat-glands, sebaceous glands, hair, and nails, so far as they refer to internal medicine, have been treated in another section.

Area of distribution: The distribution of the eruption over different areas of the body is of great importance in the diagnosis of the various erythemata due to exanthems and to morbid conditions of the gastro-intestinal tract. It will be noted more in detail when the specific eruptions are considered. The student should also bear in mind the relationship of eruptions or cutaneous changes of nutrition (trophic disorders) to the affected nerve-supplies.

III. Associate Morbid Phenomena. The student should particularly observe the associated symptoms in order to determine whether the skin affection is an expression of internal disorder. It will be learned later

that nearly all the secondary lesions belong to the *hemorrhages* or the *erythemata*. The true relationship of the cutaneous phenomena can be fully ascertained only by inquiry into the history and course of the eruption, and the concomitant symptoms. Thus, if the eruption is thought to be due to the infections to which the so-called exanthemata belong, the period of incubation, mode of infection, symptoms of the invasion, and the progress of the attack must be inquired into.

It must not be forgotten in the study of skin eruptions, to determine their relationship to chronic internal affections; other circumstances also should be inquired into, such as the occupation, the character of the clothing, degree of cleanliness of the patient, the effects of climate, the season, and other influences of environment.

General Symptoms.

The *subjective symptoms* are of great importance in the diagnosis. Pain, itching, burning, smarting, and tenderness are significant of the inflammations. *Pain* due to inflammation is constant and smarting, burning or throbbing in character. Sometimes, however, pains of a neuralgic character, intermittent and distributed in the course of nerve-trunks, precede the development of eruption. This is seen in herpes zoster. *Itching* is an important symptom in diseases of the skin. It is not as a rule present in the eruption due to the exanthemata, except in smallpox, chickenpox, and rubella. Its absence is a striking peculiarity of the eruptions of syphilis; but in erythema, especially if associated with œdema, it is a most annoying symptom. In other skin diseases, as eczema, psoriasis, and the parasitic affections, it is much more common and extremely annoying.

Itching may be present without any anatomical evidence of skin disease. It is seen in the troublesome *pruritus* that occurs in the aged, particularly about the intestinal and genito-urinary orifices, symptomatic of affections of the organs related thereto. It is a symptom which should lead to an examination of the urine, as diabetes is sometimes found to be the fundamental source of the complaint. It has been previously noted that itching occurs to a high degree in jaundice. It may also be produced by drugs, as opium and morphine, and sometimes quinine.

Classification. The following very concise outline, taken from the work of Pye-Smith, to whom the writer is indebted for much of the data here utilized, is here given to enable the student to appreciate more thoroughly the pathological relations of the various skin diseases. The schema also shows at once the relation of the eruptions to the internal disorders, which concern us more particularly in this work:

Diseases of the Skin Regarded as Physiological Processes.

(*Pathological Arrangement.*)

Acute Inflammation.—Diffuse, *e. g.*, scarlatina, morbilli, syphilis, roseola (eruptive fevers, erythema).

With venous congestion—Erythema nodosum (rheumatism).

With œdema—Urticaria, erythema nodosum (gastro-intestinal disorder and rheumatism).

With necrosis—Furunculus, anthrax (diabetes).

Localized in papules—Enterica (erythemata), syphilis, eczema, prurigo.

Localized in vesicles—Eczema, zona, variola, scabies, herpes, varicella (eruptive fevers, infectious diseases).

Localized in pustules—Impetigo, variola, scabies, syphilis, sycosis, acne.

Localized in blebs—Pemphigus, scabies, rupia.

Desquamating during involution—Scarlatina, etc.

Chronic Inflammations.—With venous congestion—Acne rosacea, pernio.

With over-production of epidermis—Psoriasis, pityriasis rubra.

With œdema—Elephantiasis.

With fatty degeneration—Xanthelasma.

With hypertrophy—Elephantiasis.

With cicatrization—Cheloid.

With ulceration—Lupus, syphilis, lepra.

New growths—Xanthelasma, lupus, lepra, syphilis, cancer.

Atrophy—The senile skin, lineæ gravidarum.

Hypertrophy—Ichthyosis, cornu cutaneum, clavus, verruca.

Hemorrhage—Traumatic (*e. g.*, flea-bites), typhus, scurvy.

Pigmentation—Syphilitic maculæ, melasma, chloasma, icterus, ephelis.

Congenital malformations—Ichthyosis, cutaneous nævus.

Neurosis—Pruritus (diabetes, jaundice).

Anomalies of Secretion.—Increased, diminished, or perverted—Seborrhœa, xeroderma, hyperidrosis, anidrosis, chromidrosis, etc. Obstructed—Comedo, milium, acne, sudamina.

A glance at the above outline will show that the eruptions which particularly concern us belong to the class of diseases to which the term *erythema* is applied.

Erythema. **Classification.** Erythemata may be divided, in accordance with the classification of Kaposi, into acute, contagious, exudative dermatoses, represented by measles, scarlatina, rubella, and smallpox, and the eruptions of typhoid fever and typhus; and the acute, non-contagious, inflammatory dermatoses. The *non-contagious* forms include the class which may be confounded with the eruptive fevers. These skin inflammations closely simulate in their symptoms the eruptive fevers, even to the affections of the mucous membranes. Besnier has named them the *pseudo-exanthems*, and divides them into rubeloids and scarlatinoids. Both simulate eruptive fevers throughout their course, and hence both are acute and febrile. The scarlatiniform erythemata are febrile at the beginning, subacute in course, but of longer duration than the fever they simulate. They are the most common forms, and arise from infectious diseases, such as puerperal fever, septicæmia, and gonorrhœa; or from toxæmia due to drugs or articles of food.

Diagnosis. CHARACTERISTICS OF THE ERUPTION. The *non-contagious* forms of erythema are recognized by the following characteristics: (*a*) Rose rash with injection of the surface; (*b*) either with or without general œdema, or circumscribed local œdema, forming wheals or papules, or in rare cases bullæ. (*c*) The rash is followed by a branny desquamation. (*d*) The exudation that attends the lesion is always watery, in counter-distinction to the seropurulent or purulent exudation of eczema and scabies. Sometimes slight *hemorrhages* attend the lesion, as in cases of purpura or of urticaria. (*e*) The course is of diagnostic significance. The onset is

sudden and usually attended by febrile symptoms, sometimes mild, again very intense. (*f*) The duration is short; at least it is not indefinite. Recurrent forms must not be regarded as one process of long duration. (*g*) The locality is not of precise diagnostic significance. The eruption is usually symmetrical, and the favorite localities may be defined as the extensor surfaces of the forearms and legs, the face, cheeks, neck, and the chest and abdomen. True erythema does not attack the scalp, the flexures of the joints, the palms (except erythema multiforme), nor the soles. (*h*) The local symptoms are mild. Local tenderness is more marked than in eczema. Smarting and tingling are complained of, but severe pain and excessive itching are rare. Only when wheals are present do we find pruritus. The rash of erythema does not spread. Patches occasionally coalesce, but an affected area never enlarges its borders. In addition, certain ætiological relations, some of which are obscure, are of diagnostic significance. The erythemata occur most commonly in children and young people. They are very frequent in men. In regard to age, the incidence coincides with that of rheumatism. As noted elsewhere (page 213), the nature of the *associate morbid phenomena*, if there are any, is first to be determined. Then the possibility that the rash may be medicinal (see page 220) or dietetic must be considered. Finally, is it a pre-eruptive rash of one of the exanthems? The presence of an epidemic, exposure to infection, and allied facts help to determine the answer to this difficult question. (See Roseola.)

Varieties. The non-contagious varieties of erythema are *erythema multiforme*, *urticaria*, *herpes*, and *roseola*.

Erythema Multiforme. In simple form it is seen with papules or with exudation; it may disappear in a few hours, or persist for a day or two and form rings (*erythema fugax* or *erythema annulatum*). With the fading of the redness faint desquamation ensues, and there may be a few pigment-marks. The annular form is observed in rheumatic fever. Both varieties may also be found associated with the following infections: typhoid fever, puerperal fever, gonorrhœa, cholera, infectious endocarditis and osteomyelitis, syphilis, leprosy, vaccination, and surgical septicæmia. Osler has called attention to the visceral complications of *erythema exudativum multiforme* associated with the skin-lesions—viz., gastro-intestinal crises, endocarditis, pericarditis, acute nephritis, and hemorrhage from the mucous surfaces. Arthritis is also seen in some instances. The skin-lesions range from simple purpura to local œdema, and from urticaria to large infiltrating hemorrhages of the skin and subcutaneous tissues. The gastro-intestinal crises are attended by colic, with vomiting and diarrhœa. (See pages 847 and 848.)

Erythema Nodosum. With the erythema there is great œdema. The spots are somewhat painful and tender, but do not itch. The redness of the erythema is modified by the hue of venous congestion. Small hemorrhages may be seen. The patches develop on the legs, their long diameter being parallel to the tibia. They rise slowly into hard masses. They may be seen on the ankles or the calf, and sometimes on the ulna. They occur frequently in those who have suffered from *rheumatic fever*.

Urticaria. A form of erythema in which wheals, sometimes sur-

rounded by an erythematous blush, are seen. It is an acute inflammatory œdema of the cutis. The serous exudation fills the lymph-spaces and expels blood from the venules. It takes place suddenly, and may be excited by chemical irritation or a mechanical irritant, as the finger drawn across the skin. Small patches or large white areas are seen, due to the coalescence of smaller ones (giant urticaria). All parts of the body may be affected, except the scalp, face, and soles of the feet. The eruption is not symmetrical. Its course may be acute, or it may be chronic and characterized by successive transitory attacks. It is the form of erythema in which intense itching is the most pronounced symptom. There are no other subjective symptoms. The itching causes restlessness and loss of sleep. Urticaria is symptomatic of gastric or intestinal disturbance, the ingestion of drugs or poisons, or the injection of diphtheria antitoxin. Another form follows the tapping of a hydatid cyst. Urticaria occurs sometimes in women at each menstrual period, and may be traced to ovarian disorder. It may occur, with high fever, after severe shock to the nervous system. It is not an infrequent complication of rheumatic fever. It occurs in men and women equally, but is most frequent in children and adolescents.

Erythema lævе often appears upon the tense skin of dropsical parts. It may be the result of acupuncture.

Herpes Zoster. This eruption is seen in the cutaneous distribution of one or more nerves. It consists of vesicles of flattened form, ranged in clusters of twenty or thirty, lying on a reddened, slightly swollen bed of skin. The number of clusters varies from one to ten. The vesicles develop in quick succession, beginning usually near the roots of the nerve whose branches they follow. A short papular stage precedes the vesicles, and some of the vesicles abort. The eruption tends to dry up in five or six days. The crusts form in yellowish or brownish clusters, which fall off in the third week, leaving purple stains.

When the disease attacks the face, it follows the course of the fifth nerve. The several twigs of the trifacial are traced out from their points of emergence from the bony canals. On account of the presence of loose areolar tissue great swelling of the eyelids sometimes takes place, so that the lesion may be mistaken for erysipelas. Ulceration of the cornea and iris sometimes occurs, and, when lower divisions of the trifacial are affected, vesicles may appear in the mucous membrane of the mouth and palate. The cervical nerves and those of the upper extremity are also affected in their distribution. The eruption on the arm rarely goes below the elbow. When the second and third intercostal nerves are affected, the intercostohumeral branch produces an eruption down the inner side of the arm. The eruption occurs frequently on the trunk; following the course of the dorsal nerves it slants downward as it approaches the pubes.

On the lower limbs the eruption rarely extends below the knee or buttocks. It follows the course of the external cutaneous or anterior crural nerve, or that of the small sciatic. Some of the branches of the sacral nerves may also be affected. The disease is unilateral, and its precise limitation to one-half of the body is of the greatest diagnostic significance

While fever or general symptoms do not usually attend its course in any marked degree, insomnia and depression are likely to occur, probably on account of the severe neuralgic pain which is the most important subjective symptom. It is localized in the nerves in the distribution of which the eruption takes place. It is not so likely to be present in the young. The pain may precede the eruption by several days, and persists long after the eruption has subsided. This is particularly the case in old people.

HERPES LABIALIS, or FACIALIS, is an eruption of vesicles arranged in groups or clusters upon an inflamed surface. They appear very suddenly upon the upper lip or the alæ of the nose, sometimes on the cheek or chin, and may occupy the inside of the mouth. They undergo some changes, as in herpes zoster, but are not attended by severe neuralgic pain. They are also symptomatic of an internal disorder, an acute catarrh (cold), or follow a rigor, as in intermittent fever or pneumonia. They may be present in epidemic cerebrospinal meningitis, but never in tubercular meningitis. Diagnosis of the former disease is confirmed by their presence. (Klemperer.) Herpes iris and herpes præputialis have no diagnostic significance of internal disease.

ROSEOLA. Roseola is an eruption of a deep-rose color, not arranged in crescentic patches as in measles, nor scarlet and capable of being resolved into innumerable red points as in scarlatina. It is not so diffuse as the latter. It precedes smallpox, scarlatina, measles, cholera, typhoid fever, syphilis, diphtheria, and malaria. In smallpox, in cases of cholera, and after parturition and surgical operations the rash is copious, but is characterized by being seated over the lower half of the abdomen and the anterior and inner aspects of the thighs. It may appear elsewhere, but is usually confined to that portion of the body.

The erythema of roseola may be mistaken for rubella, measles, or scarlatina. The following are points of distinction: (1) it is neither contagious nor epidemic; (2) there are no prodromal symptoms; (3) the rash does not come out after a definite period of fever; (4) it is not confined to any special locality; (5) the fever is of short duration and moderate degree, rarely above 101°; (6) there is no catarrhal discharge from the eyes or nose or in the pharynx; the fauces and palate are reddened but not swollen; (7) it is not seen in the mouth, like the eruptions of measles and scarlatina; (8) if present, the fever which precedes the eruption is of only a few hours' duration (in scarlatina it lasts twenty-four hours, in measles seventy-two hours); (9) the rash is not crescentic as in measles, nor punctiform as in scarlatina, though it must be admitted that severe cases of the affection cannot easily be diagnosticated, the development of the sequelæ alone establishing the diagnosis.

To add to the confusion, an erythema called roseola often precedes the eruption of a particular fever, as previously indicated.

ERYTHEMA OF SPECIAL DISEASES. Sufficient reference has been made to the erythemata that attend *rheumatism*. A few other internal (infectious) disorders are associated with the development of an eruption. In *cholera*, during the period of reaction, a rose rash which may resemble erythema, urticaria, or scarlatina appears coincidently with a rise of tem-

perature. It is most frequently seen on the forearms and backs of the hands, but it may cover the back and limbs. It may be slightly hemorrhagic and last two or three days. A slight desquamation usually follows. In *influenza* a roseolous eruption, covering the trunks and limbs and later becoming papular, is seen in rare cases.

Erythematous eruptions are sometimes seen in the course of *Bright's disease.* Two forms, quite distinct from the previously mentioned *erythema lœve,* are observed: the roseola on the feet, legs, and hands—rarely on the chest and abdomen; and the papular form on the thighs, arms, and shoulders. Itching and other subjective symptoms do not attend the eruption. A form with desquamation may begin on the limbs. These erythemata are common in the later stages of Bright's disease, but are not of ill-omen. In acute Bright's disease a transient roseola is observed very rarely; so also is purpura. If there is much anasarca in tubal nephritis, erythema is more common. The eruptions usually appear independently of uræmic symptoms, and disappear during their continuance. They are in all probability allied to the inflammation which attacks the lungs and serous membranes in Bright's disease.

Medicinal Rashes. Most of the so-called medicinal rashes belong to the erythemata.

The following drugs are known to cause erythema: potassium bromide and iodide, copaiba, cubebs, the essential oils, capsicum, santonin, chloral, opium, morphine, antipyrin, salicylic acid and its compounds, iodoform, belladonna and atropine, tar, carbolic acid, arsenic, cannabis indica, digitalis, mercury, silver, copper, and diphtheria antitoxin.

Belladonna produces in susceptible persons, or when administered in poisonous doses, a diffuse, bright-red erythema, closely resembling that of scarlet fever, but without the interspersed dark-red points which characterize the latter. *Atropine* also produces in some persons, especially on the shoulders, arms, chest, and face, an eruption of disseminated, small, hard vesicopapules showing no tendency to pustulation. They are seated on an inflammatory base, but are more superficial than acne lesions.

The *bromides* produce a characteristic pustular eruption which is most intense upon the shoulders, face, chest, and arms. Large doses, or long-continued administration, are generally required to bring it out. It is conspicuous upon the face of some epileptics.

The *iodides* produce an eruption which is not often pustular, but an erythematous or papular rash is not uncommon. It appears chiefly about the forearms, face, and neck. Vesicles, bullæ, and purpuric spots are also occasionally seen.

The eruption produced by *quinine* is generally erythematous and is attended with itching and burning; the face and neck are attacked first.

Opium and its alkaloids also produce in susceptible persons an erythematous scarlatinoid eruption which is accompanied by intense itching. Itching, especially about the nose, is much more common without eruption.

Copaiba produces a vesicopapular or papular eruption resembling urticaria and erythema multiforme, which is usually most abundant on the extremities and attended by itching. The lesions may be purpuric.

The eruption of *cubebs* is a diffuse erythema, with millet-sized papules, coalescent here and there. Unlike the eruption of copaiba, it is more copious over the face and trunk than over the extremities.

Antipyrin causes a measles-like or urticaria-like eruption.

General Diagnosis of Skin Affections.

(*Condensed from* PYE-SMITH.)

I. Factitious Eruptions. We must never forget the possibility of the affection before us being artificial. All kinds of dermatites, eczema, erysipelas, pemphigus, and impetigo may be simulated by the application of various irritants. Pigmentation also has often been imitated with success. Such artificial lesions will generally be found upon the arms, rarely on the face, and scarcely ever beyond easy reach of the patient's hands. Mustard, cantharides, and some other irritants can be distinguished with the aid of the microscope.

II. Traumatic Eruptions. In all cases of dermatitis we should seek for the irritant, and sometimes it is so directly the cause of the affection that the eczema or impetigo in question may be considered purely traumatic, and efficient treatment immediately follows accurate diagnosis: *sublata causa tollitur effectus.*

Pediculi in the hair should be carefully looked for in all cases of impetigo in children, and *Pediculi vestimentorum* in the prurigo of old people. The acarus of scabies, fleas, bugs, and gnats may be found. In adults *Pediculi pubis* may sometimes be found in the axillæ as well as in their proper region, and when they have been destroyed by mercurial ointment the patient is at once relieved from pruritus.

Frequently the irritant must be sought for in the objects which the patient habitually handles. The coarser kinds of brown sugar are a frequent cause of eczema of the hands (grocers' itch). So with many of the "chemicals" used in a variety of modern handicrafts. Constant washing of the hands in washerwomen, scrubbers, bartenders, and many others produces eczema rimosum. The heat of the sun is the cause of eczema solare and ephelides; the pigment-spots on the shins of elderly people are due to the heat of a fire. Sweat, again, is a very common irritant, producing the erythema which usually accompanies sudamina and also intertrigo of opposed surfaces. Scratching as a cause of traumatic dermatitis has been repeatedly referred to.

III. Febrile Rashes. We must never forget that a cutaneous eruption may possibly be part of an acute exanthem. The use of a clinical thermometer is a great help in this respect. Variola is frequently mistaken for syphilis and other affections.

IV. Medicinal Rashes. Some cases are due to certain kinds of food or to drugs. They have been described above.

V. Syphilodermata. When we have satisfied ourselves that the eruption before us is not factitious, nor directly traumatic, nor a symptomatic eruption, we may next consider whether or not it is due to syphilis. In this inquiry it is undesirable to ask questions the answers to which are as apt to mislead as to guide aright. A pustular eruption in

an adult should always suggest the question of syphilis when that of scabies has been answered in the negative.

1. We should first consider the *color* of the affected skin, remembering, however, that the pigmentation which gives the so-called coppery or rawham tint to a syphilitic eruption is the same as that which is sooner or later produced in all forms of dermatitis. Psoriasis, chronic eczema, lichen planus, and prurigo may all produce shades which bear the closest resemblance to the color of a syphiloderm.

2. The lesions of syphilis are *multiform*. It is rare in any but syphilitic affections to find mere hyperæmia in one part and associated pustules, papules, scales, or ulcers in others; and it is not often that a syphilitic eruption exhibits only a single elementary lesion.

3. Syphilitic eruptions for some unknown reason *do not itch*—the exceptions to this rule being remarkably few. If itching does occur, it is usually during the stage of scabbing of pustular rashes or during the healing of tertiary ulcers. An ordinary secondary syphilide may, however, as a rare exception be so irritating that wheals and scratch-marks are present. On the other hand, psoriasis is often free from irritation; while the degree of itching of eczema, and even of scabies and prurigo, varies greatly.

4. The local *distribution* of syphilitic disease is a great aid in diagnosis. Specific eruptions are certainly not, as a rule, symmetrical; the early roseolous rash is only so because it is general, and therefore upon a surface like the human body more or less symmetrical. Moreover, as it chiefly affects the face, chest, and trunk generally, it is near the middle line. But we do not see symmetrical patches of syphilides on corresponding parts of both sides of the face, both sides of the trunk, or the right and left limbs. In all but the earliest syphilides the affected patches are very decidedly and constantly unsymmetrical, irregularly scattered over head, trunk, and limbs, and chiefly remarkable for having no well-marked seats of predilection.

The forehead, especially about the roots of the hair, is however very frequently the seat both of the early and secondary erythematous, scaly, and pustular syphilides; and the palms of the hands and soles of the feet are frequently symmetrically affected with the later scaly eruption.

Practically, when we find a disease of the skin occupying some unusual position, we should at least consider the question of syphilitic origin.

5. These signs, alone or in combination, serve to distinguish early specific roseola from erythema, eczema, scarlatina, and measles, and the later eruptions from eczema, lichen, impetigo, and psoriasis.

The eruptions of *congenital syphilis* that are most liable to be mistaken are: the so-called pemphigus of infants, which is known by its affecting the palms and soles; rupia, which may always be distinguished from impetigo by the form of the crusts and the ulcerated surface beneath; an erythematous rash on the nates and genitals of infants, which is distinguished from eczema of the same parts, also common at that age, by its coppery color, its blotchy distribution, and more clearly defined margin.

The *tertiary ulcers* of syphilis are distinguished by their presence in

unusual places, by their punched-out edges, circular or so-called horseshoe shape, and by the fact that they usually give little pain or discomfort.

Tertiary ulcers have no predilection for the outer side of the leg; but inasmuch as the part above the inner malleolus is, from anatomical causes, the chosen seat of varicose ulcers, most ulcers in the former position will be syphilitic and in the latter not. Moreover, the age helps in the diagnosis, as varicose ulcers rarely occur before the fortieth year. Most ulcers on the arms are found to be tertiary syphilitic ulcers.

VI. Tineæ. The next group of skin diseases includes those which are due to vegetable parasites—tinea versicolor of the trunk, eczema marginatum of the perineum and thighs, tinea circinata of the neck and other parts, tinea sycosis of the chin, and tinea tonsurans of the scalp. In all doubtful cases the microscope should be employed.

Tinea of the scalp is rare in adults, and tinea circinata still more so; tinea marginata occurs only in adult males.

VII. Primary Superficial Inflammations. To distinguish the superficial from the deeper kinds of dermatitis, we should notice whether the cutis alone is infiltrated and thickened, or whether the skin as a whole is bound down by adhesions to the subcutaneous tissues. The presence of scars, however slight, is a proof that the process has gone deeper than the papillæ, and has more or less extensively destroyed the papillary layer. Superficial inflammations, excluding those due to acarus, to pediculi, and to other direct irritants, and excluding also those which are the result of vegetable parasites and of syphilis, fall, with respect to their treatment, into three large groups:

The first group, represented by impetigo and most forms of eczema, consists of subacute inflammations accompanied by burning, itching, and pain, and sometimes a slight degree of fever.

The second group of superficial inflammations of the skin is typically represented by psoriasis, but also includes lichen planus, the more chronic, dry, and obstinate forms of eczema, and true prurigo. These affections are chronic, with little irritation, exudation, pain, or active signs.

The third group is that of the erythemata. (See page 216.)

VIII. The Acne Group. Acne, both in its pathology and etiology, differs from other forms of dermatitis. The age of the patient and the distribution of the lesions are sufficient for diagnosis. It is at once a superficial and a deep dermatitis, and often leaves scars. Its treatment consists entirely, or almost entirely, in local applications directed to the correction of the sebaceous affection. With acne may be classed sycosis and furunculus.

IX. Deep Affections. When we have ascertained that the affection of the skin is deep—that is to say, that it involves the true skin—the field of diagnosis is limited.

Excluding erysipelas, which is distinguished by its acute character and febrile symptoms; excluding the pustular affections, which affect the skin deeply and produce scars only at isolated points (such as acne, variola, and herpes zoster); and excluding also leprosy and other exotic diseases, we have to distinguish in the great majority of cases which come before us in this country: (1) traumatic and varicose ulcers; (2) gummata and

syphilitic ulcers; (3) lupus; (4) rodent ulcer; and (5) carcinoma of the skin. With regard to the first of these, we must not assume that because a sore upon the skin is said to be the result of a blow or a kick it is purely traumatic, for syphilitic ulcers often arise in this way. Malignant ulcers are rare, and their nature is usually obvious from the age of the patient, the pain they occasion, their tumid margins, and their blood-stained secretions. Moreover, they are, with few exceptions, confined to the neighborhood of the orifices of the body, especially the lower lip, the urethra, the vulva, and the anus. Rodent ulcer, however, is very difficult to diagnosticate with certainty. Its locality, its slow and painless progress, and its occurrence during the latter half of life, usually serve to distinguish it from lupus; and its being single, excessively chronic, and unaccompanied by nodes or other syphilitic lesions are the best characteristics for diagnosis from a tertiary ulcer.

Scars. Scars are important proofs of the occurrence of previous disease, especially smallpox, chickenpox, and syphilis. Scars of the first two occur in the form of circular pits and almost always on the face. Scars of syphilis are larger, circular or oval in shape, and seen usually to the best advantage on the extremities, but the single scar on the forehead is strikingly suggestive. Scars upon the legs in persons under thirty years of age, when not traumatic, are almost always syphilitic. Scars as the result of suppurating glands are seen most frequently in the neck, but may be found wherever there are glands, especially under the jaw and in the axilla and groin. They are most liable to occur in tuberculous persons, either spontaneously or as the result of the exanthemata, erysipelas, or other infectious disease. When such scars are met with in a person with incipient tuberculosis, the prognosis is grave.

The appearance of the scar indicates its age in a general way, and hence throws light upon the patient's previous history, and also serves as a check upon the accuracy of his statements.

Scars the result of wounds, injuries, or operations may be seen anywhere; they are of importance only so far as they may furnish a clue to the cause of existing disease. Of such nature are the scars upon the head in cases of brain disease, particularly epilepsy.

The scars of pregnancy, the striæ seen upon the lower part of the abdomen and the upper part of the thigh, must not be confounded with similar scars that may remain after a severe attack of œdema, and which are sometimes found in fat persons. They are also seen after typhoid fever.

The Nutrition of the Skin.

The color, as determined by inspection, is a fair index of the nutrition of the skin, but further information is obtained by *palpation*. In *health* the skin is smooth, firm, and elastic. When pinched up between the thumb and fingers and then allowed to escape, it slips quickly back into its former position. When pressed or squeezed, it becomes pale from expression of blood, but resumes its natural hue immediately.

The readiness with which the blood returns after pressure shows the character of the capillary circulation of the skin. This is active in health

and sluggish in serious disease of the lungs, heart, and bloodvessels. In the eruptive fevers, especially in measles, scarlet fever, and smallpox, sluggish capillary circulation with dusky eruption is a grave sign. In measles it is usually due to pulmonary complication, and in other infectious diseases to the overwhelming effects of the poison.

As age advances the skin becomes less elastic, and in old persons may lie in wrinkles. When pinched up between the fingers the skin is more inclined to remain wrinkled. Fat persons whose skin is firm and hard are in much better condition than those whose skin is loose and flabby. The latter condition is frequently met with in babies, particularly those fed on artificial foods. When the skin is thin and dry and has lost its tone, so that when pinched into folds it regains its smoothness slowly and sluggishly, it is usually evidence, in a person under fifty, of some grave cachexia, as carcinoma.

Moisture of the Skin.

Moisture and *dryness* are in a sense correlated with the nutrition of the skin. It is quite certain that when the skin is abnormally dry its nutrition is impaired.

In health the skin is not perceptibly moist, except as the result of physical exertion or exposure to heat, or as the immediate result of imbibing a hot fluid or a sudorific drug. There is considerable individual difference, however, within physiological limits. Rheumatic and strumous persons may have a perceptibly moist and oily skin at all times, while others have a skin which perspires very little, even under influences that usually bring about perspiration.

Perspiration Increased—Hyperidrosis. It may be general or local.

A. General increased perspiration is seen—1. With fever; 2. With normal or subnormal temperature.

1. Generalized sweating occurs in the course of *rheumatism*, when the sweats are strong in odor and acid in reaction. It is seen in *tuberculosis*, especially in the miliary variety. It is sometimes marked throughout the course of *typhoid fever*. General perspiration also attends the violent muscular action of *tetanus*, but is not seen in *epilepsy*. An example of general sweating is seen in that curious affection to which the term "*sweating sickness*" has been applied. It is a fever the nature of which is not well understood, but in which this symptom is most pronounced. Sweating is extreme in *trichinosis*.

2. In the second instance, when the temperature is normal or subnormal, we may have sudden, temporary perspiration. Sweats occur from excitement or slight exertion during *convalescence*. A general profuse perspiration may be of short duration and occur suddenly after fright or shock in health. It is the characteristic perspiration of *collapse*. The forehead is covered with sweat, large drops stand out on the face, the hands and feet are moist or wet with perspiration, and the whole surface of the body "leaks." The skin at the same time is cold and clammy. In the collapse of all forms of shock, or after hemorrhage or profuse purging, as in cholera, this form of perspiration is seen.

More striking still is the perspiration that suddenly breaks out in the course of acute disease coincidently with a fall of temperature. We have the *critical sweats* of pneumonia and relapsing fever; sweats which terminate a paroxysm of intermittent fever, whether of malarial or infectious origin (see Fever); the profuse perspiration that attends pyæmia, breaking out with each fall of temperature to disappear as it arises; the night-sweats that attend tuberculosis and other exhausting diseases. In tuberculosis and in pus-formation or accumulation the oscillation of temperature, with or without chills, followed by sweating, is known as *hectic*. Sudden breaking out of general perspiration, but more notably seen on the face, attends dyspnœa of pulmonary origin and the attacks of dyspnœa in the course of organic heart disease. These sweats are at times the result of an effort at elimination on the part of the skin to relieve the kidneys or bowels, such as the perspiration of *uræmia*, which is attended by a urinous odor. At times sweats also occur in jaundice. In the conditions just mentioned the skin is cool and the extremities are cold.

Sudamina. Here may be mentioned another eruption, a localized moisture common in the course of internal diseases. Sudamina, or miliaria, are small clear vesicles seen in large numbers, usually on the abdomen, but also on any other part that reflects the light strongly. They are seen during and after the subsidence of profuse sweats. While actual perspiration is seen on the forehead, the trunk may appear free from moisture. When the hand is placed over it, as on the abdomen, the dryness is noted, but at the same time a roughened, nutmeg-grater-like sensation is felt. On close inspection this is observed to be due to the eruption just mentioned. The vesicles are usually of good prognostic omen in the course of febrile diseases, particularly typhoid fever. They are due to the accumulation of perspiration under the epidermis.

Prolonged Perspiration. In exhausting diseases, general and persistent perspiration may occur, particularly in the later stages, as in tuberculosis, and in any disease attended by persistent dyspnœa.

B. Local increased perspiration (*hyperidrosis localis*) occurs when there is local vasomotor paresis. Thus, in organic diseases of the brain and in affections of the peripheral nerves, in some forms of neuralgia, in migraine, and in hysteria, the symptom has been observed. Sometimes one side of the body alone is affected, even in a malarial paroxysm (hemidrosis).

Local sweats are sometimes significant. This is the case particularly with a sweat confined to the head, which occurs usually in children and is one of the striking characteristics of rhachitis. The patient rolls his head at night, so that the hair on the back of the head is rubbed off.

Unilateral sweating of the head may arise from destructive pressure on the sympathetic nerves, causing paralysis of the dilator fibres of the ciliospinal branches, in thoracic aneurism, and in caries of the lower cervical vertebræ. Contraction of the pupil and congestion of the face on the same side are usually noted.

Perspiration Diminished—Anidrosis. The skin is abnormally dry in the early stages of acute disease attended by fever, particularly if the febrile rise takes place suddenly, as in the acute digestive disorders of chil-

dren. In adults, when the disease is accompanied by high fever, as in thermic fever, the skin is dry. On the first day of the eruption of the exanthemata dryness is marked. Dryness of the skin is of frequent occurrence when there are copious discharges of water from the bowels or kidneys. In choleraic diarrhœa the dryness develops suddenly. In some affections, as diabetes and Bright's disease, the dryness lasts a long time and is frequently attended by eruptions or desquamation and by the formation of boils. When there are accumulations of serum in the lymph-spaces of the subcutaneous connective tissue, or changes in the connective tissue, as in dystrophies, myxœdema, or scleroderma, the skin is dry because of the stretching and compression of the bloodvessels.

CHAPTER XXIV.

EXAMINATION OF THE SUBCUTANEOUS CONNECTIVE TISSUE.

ENLARGEMENT or swelling of the subcutaneous connective tissue, other than skin tumors and papular eruptions on any portion of the surface of the body, is due to some change in the tissue or structure of organs directly underneath the swollen part. Œdema, myxœdema, subcutaneous emphysema, dystrophies, scleroderma, brawny induration, and local subcutaneous swellings are the principal conditions to be considered.

Œdema; Dropsy.

When the lymph-spaces of the subcutaneous connective tissue become over-distended with an accumulation of serum, the general term *dropsy* is applied to the condition. When the accumulation is local and confined to small areas, it is known as *œdema*. When it is general, and the large lymph-cavities, the pleura, peritoneum, and pericardium, also contain fluid, the condition is known as *anasarca*. Accumulation occurs because more fluid is poured out by the capillary vessels than can be removed by the lymphatics and veins. This may depend upon either obstruction of the veins and lymphatics or excessive *transudation* from the bloodvessels, or both. The former condition, however, is rare, and usually local, because unless the obstruction is very great the veins and lymphatics are able to carry away more fluid than is effused from the capillaries.

1. *Excess of fluid* transudes when there is *local capillary change* from inflammation or the effects of poisons. The change must be in the capillaries. It was thought that this general process was of an inflammatory nature, but at present it is believed to be due to the influence of poisons, probably absorbed from the intestinal canal, which alter the nutrition of the capillary vessels. Thus the œdema and general dropsy of albuminuria, particularly in the early stage of that affection, are thought to be due to a poison circulating in the blood which also causes the nephritis. Mahomed alleges that a pre-albuminuric stage of scarlet fever exists, during which he noted a peculiar reaction of the urine, which gave a blue color with guaiac. A brisk purgative administered when this reaction was noticed prevented the occurrence of albuminuria; whereas when the drug was withheld, albuminuria always followed. The purgative removed the poison which caused the nephritis and œdema.

In urticaria there is marked local œdema. Brunton thinks that some poisons circulating in the blood cause paralysis of the secreting power of the sweat-glands, resulting not only in effusion from the bloodvessels, but also in certain changes in the secreting cells, causing them to produce an acid, the local irritative action of which upon the capillaries causes a further

transudation of fluid. That acids circulating in the blood have the power of creating œdema the experiments of Cash and Brunton fully demonstrate. While, therefore, in the œdema of Bright's disease in its earliest stage and in urticaria we have this explanation of the phenomena, other factors are causal in other forms of œdema.

2. *Increased transudation* and *obstruction* to the flow of lymph are the causes of some forms of œdema. It may be of local origin, as in the œdema over the site of an inflammation or the œdema of an arm or leg from venous occlusion; or it may be of general origin, as in cardiac disease. The obstruction may be in the lymphatics or in the veins. In the former it may occur from (*a*) lack of muscular action; (*b*) deficient inspiratory action of the thorax; (*c*) diminution of the diastolic suction of the heart; (*d*) positive pressure on the veins. In the latter, obstruction is caused by conditions similar to those affecting the lymphatics, and arises from (*a*) lack of muscular action; (*b*) deficient movement of the thorax; and (*c*) feeble action of the heart; and, in addition, it is likely to be caused by (*d*) complete arrest of blood-flow from external pressure upon the vein or from plugging of the vein. It can readily be seen, with a little knowledge of physiology, how the above factors favor the development of œdema due to disease of the heart and to venous obstruction. The baneful factors are those which retard the flow of blood, preventing its return to the right heart. Hence it is called the œdema of passive congestion.

3. *Anæmia.* A third form of œdema, usually slight, is that which is seen in anæmia. Several factors combine to produce it: (*a*) the watery condition of the blood; (*b*) the condition of the capillaries; and (*c*) vasomotor paresis on account of imperfect nutrition of the vasomotor centres. It may be diffused, as in the anasarca that attends the anæmia of malaria.

4. *Neuritis, angioneurosis.* Œdema may be of nervous origin. Such is the œdema that occurs in diseases or injuries of nerves. To it possibly belongs the œdema of beri-beri. It may be a trophoneurosis with secondary alterations in the permeability of the vessel-walls, or it may be due to vasomotor paralysis, as in angioneurotic œdema.

Mode of Recognition. Whether the accumulation be in local areas or distend the entire subcutaneous tissue, the œdema is not difficult of recognition. The part is swollen and puffy, the surface is pale, smooth, and shiny, the temperature is usually low, and the affected area pits on pressure. Pitting is more pronounced if the finger is pressed over a part which is seated upon a firm foundation, as bone. Œdema of the ankle or over the tibia is more readily recognized than œdema in the calves.

The œdema obliterates normal depressions and increases the rotundity of the affected part. It causes deformity, as of the face and neck or of the penis, when the accumulation of serum is considerable. The swelling appears in the most dependent parts if the œdema is diffuse or the cause is general, as in cardiac disease; or in parts made up of loose connective tissue, as the eyelids or scrotum. The temporary *disappearance* of the œdema, either entirely or from one part to appear in another, is a prominent feature. It may disappear between morning and evening, or its position may alter with a change in the position of the body. The previous existence of œdema can often be discovered by the scars or striæ

that have resulted from over-stretching of the skin, as of the abdomen and thighs.

Diagnosis. Œdema is to be distinguished from: (1) *Inflammatory swellings*, by the absence of the other classical signs of inflammation: pain, heat, and redness. (2) The enlargement of *myxœdema* differs from œdema by the absence of pitting on pressure, by the permanency of the enlargement, the occurrence of induration, which resists the pressure of the finger, and by the occurrence of anæsthesia or analgesia. (3) The swellings of *connective-tissue dystrophies* are hard localized areas that do not pit on pressure and are not seated in dependent parts of the body. They are found on the arm, for instance, or on the thigh, or about the flanks and in the axillæ. (4) The swelling of *subcutaneous emphysema* differs from œdema in that it arises in the course of some disease of the air-passages, and on palpation the crackling sensation of air under the finger is distinctly felt, while there is no pitting on pressure. In the cases the writer has seen, the parts were particularly tender, although pain in subcutaneous emphysema is said to be usually absent.

The value of œdema as a diagnostic sign depends upon its location, its mode of development, and its association with disease of other organs or structures of the body.

Location. The œdema may be limited to small areas, as the eyelids, the face, or the feet, or to an arm or a leg; it may involve an arm and leg of the same side; or it may involve the extremities and trunk, and even include the face. We therefore have *local* and *general œdema*.

Local Œdema. Local œdema occurs over the *seat of an inflammatory process*, and is a valuable diagnostic sign. It is an indication of suppuration, and is known as "inflammatory" or "collateral œdema." The cause is *obstruction of the lymph circulation*. Collateral œdema is seen over the mastoid process when the cells are the seat of inflammation; in the parotid region when the gland is inflamed; over parts of the thorax in empyema; over the præcordia in purulent pericarditis; over the surface of the liver in some cases of hepatic abscess; in the abdominal parietes in purulent peritonitis, but more particularly over the primary focus of inflammation, as the gall-bladder region or the region of the appendix. Local œdema occurs when there is *pressure on a vein or occlusion of it by a thrombus*.

The Arm. Œdema of the arm from pressure on the veins by enlarged lymphatic glands in the axilla, and œdema of the leg from thrombosis of the femoral vein are examples of this form of local œdema. Dropsy of an arm often occurs when the patient has lain upon it. It is seen over muscles affected by trichinæ.

The Arms and Thorax. Another form of local œdema occurs when there is pressure upon the superior vena cava from aneurism or disease of the mediastinal glands. The œdema is then limited to the arms, head, neck, and thorax. Such œdema is usually associated with cyanosis of the hands and arms. There is also marked distention of the veins of the upper parts of the body. In a few instances the œdema has been found to be more marked on one side than on the other. This has occurred in cases of aneurism which communicated with the vena cava;

either the collateral circulation on one side had been established or pressure was greater on the left innominate vein. The œdema is sometimes limited to the *head* and *arms*. When the obstruction of the superior cava is situated below the entrance of the azygos vein, the chest shares in the venous congestion and resulting œdema. When, on the other hand, the obstruction is above the azygos vein, there is no œdema of the chest-wall. This form of œdema is, as a rule, easily recognized by the presence of the above-mentioned symptoms with other pressure symptoms due to disease of the mediastinum, and by the results of physical examination which reveals the presence of a tumor in the thorax. The œdema usually develops slowly, along with the other symptoms, but at times the onset is sudden. *Sudden œdema* in this situation is always due to an aneurism which has ruptured into the vena cava (see above). The sudden onset is attended by physical signs of aneurism, or, if they are not present, by a murmur characteristic of communication between an artery and a vein. It must be confessed that often the physical signs are not precise and the murmur is absent. The *suddenness* of the peculiar localized œdema is the chief diagnostic point in favor of this rare form of aneurism.

The *cause* of the above forms of œdema is local and in close proximity to or intimate anatomical relation with the dropsical swelling. But the cause of local œdema may be central, or in a sense general. It then develops gradually and begins in special localities, as in the *feet* or *face*.

The Feet. Œdema of the feet or ankles is usually due to disturbance of the circulation. It arises in heart disease, or in the course of any exhausting and debilitating disease in which the heart has become weakened. The organic change which takes place in the heart muscle (dilatation) in the course of *obstructive valvular disease* and in *lung disease* is often attended by œdema of the feet. Later a general dropsy may ensue. But œdema of the feet may occur from another cause—*i. e.*, *anœmia*. In all forms of this affection puffiness of the ankles may be seen. An explanation of the cause has been given. Similar localized œdema in individuals of relaxed fibre occurs in the evening after a day of considerable physical exertion. Œdema of the feet, subsequently becoming diffuse, occurs in beri-beri.

The Face. Œdema may begin or remain localized in the face. (See Face and Eyelids.) It may be limited to the eyelids as a simple puffiness, or may spread over the entire face, causing complete obscuration of the normal outlines. It is the œdema of *renal* disease, and differs from œdema of the feet due to weak heart in that it is more marked in the morning on rising, and disappears toward night. Of all forms of local œdema it is the most grave, and should at once call attention to the condition of the urine, particularly if the patient has just had an attack of scarlatina, or if it occurs in a woman who is pregnant.

Œdema of the face also occurs in *trichinosis*. It begins early in the disease, disappears after a few days, to return again later. There is pain, or rather a sense of tension and restriction of muscular movements, as of the eye muscles. The œdema is seen on the forehead and especially between the eyes. It is also localized over the affected *muscles*, and is associated with the growth of trichinæ in them. It is distinguished from

cardiac and renal dropsy by its course and situation, as well as by the fact that the scrotum and labia majora are never œdematous.

The *diagnostic significance* of primary local œdema may be summarized as follows: (1) *eyelids* or eyes ("Bright" eye, "tear that does not fall") in nephritis; (2) *face*, nephritis; (3) *forehead*, trichinosis; (4) *head*, pressure upon superior vena cava above the azygos vein; (5) one side of *head*, pressure upon innominate vein; (6) *head* and *arms*, or *head*, *arms*, and *thorax*, pressure upon superior vena cava; (7) one *arm*, pressure upon axillary veins; (8) one *leg*, pressure upon femoral vein; (9) both *feet* or

FIG. 34.

Face of a patient with general anasarca due to chronic parenchymatous nephritis. (HARE.)

legs, pressure upon inferior vena cava by abdominal tumor, loss of vasomotor tone, heart disease, anæmia, late nephritis; (10) the *loins* ("lumbar cushion)," nephritis, cardiac disease if patient is in recumbent posture; (11) the *scrotum*, nephritis and cardiac disease; (12) local, "collateral," œdemas over inflammations of structures underneath, as bones, the gall-bladder, the appendix, the pleura, peritoneum, or pericardium.

GENERAL ŒDEMA—ANASARCA. General anasarca in most cases is due to heart or to kidney disease. Œdema of the face and feet may become general. In cases in which the face is the first to become œdematous, extension of the process may be very rapid, so that twenty-four to forty-eight hours after the swelling is noticed, the whole body is in a state of anasarca. *Renal disease.* The extension throughout the rest of the body of œdema primarily seated in the feet and legs (*cardiac* dropsy) is more gradual, and is accompanied by other signs and symptoms of weakness of the heart. Hence, cyanosis gradually appears, first in the extremities and ultimately in the face and lips. On the other hand, in the general anasarca

that follows the local œdema of the face in Bright's disease pallor occurs, and as the œdema increases the color becomes more and more waxy, while the skin of the extremities becomes glistening in appearance. In the so-called "wet form" of *beri-beri* general œdema comes on rapidly.

ANGIONEUROTIC ŒDEMA. This curious affection is not of frequent occurrence. It may be present in the individuals of several generations of a family. The attack comes on suddenly. The swelling is circumscribed. It may appear on the face, on the brow, the lips, or cheek. The eyelid is a common situation. It may also occur on the backs of the hands, on the legs, or in the throat. It remains but a short time and disappears as quickly as it came on. The outbreaks sometimes exhibit distinct periodicity. Local symptoms of itching, heat, redness, or general urticaria may precede the swelling. The sudden swelling causes great deformity. If the upper lip is affected, the mouth can not be opened; if the hands, the fingers can not be bent. In the hereditary cases the attack recurs every three or four weeks. The danger to life is from œdema of the larynx, which caused death in two of Osler's cases. The general symptoms that attend the attack are gastro-intestinal. Nausea and vomiting occur, followed by severe colic.

It must not be confounded with simple urticaria, or the giant form of that affection, with which it may however have close affinities. It is regarded by Quincke as a vasomotor neurosis leading to increase of the permeability of the vessels.

Recapitulation. From what has been said the student will observe that œdema may be local or general; that local œdema may be unilateral or bilateral; that œdema may be further subdivided, in accordance with the cause, into inflammatory dropsy, œdema or dropsy of passive congestion, hydræmic dropsy, and vasomotor dropsy. The dropsy of passive congestion may be subdivided into cardiac dropsy, hepatic dropsy, and renal dropsy, according to anatomical causes.

While the account of œdema just given refers more particularly to the subcutaneous accumulation of serum, the same pathology and etiology apply to accumulations in the large lymph-cavities, and hence, in addition to general œdema, we may have *ascites*, *hydropericardium*, *hydrothorax*, *hydrocele*, and *effusion* in the joints. The methods of recognition of dropsy of the larger cavities will be deferred until diseases associated with these particular regions are discussed. It must be remembered that œdema or accumulations of serum in cavities may be of local or general origin.

It must not be forgotten that two or more causes may combine to produce a dropsy, or that a dropsy due to one cause may for a time be dependent upon a second and even a more pronounced factor later on in the development of the disease. Thus (*a*) the dropsy of hydræmia may be aggravated by (*b*) that of weak heart which arises from anæmia, to which may be added later the dropsy of vasomotor paresis. The dropsy in Bright's disease is due to (*a*) capillary changes produced by a poison circulating in the blood, and later to (*b*) the condition of the heart if, as is frequently the case, it undergoes dilatation.

Myxœdema.

Enlargement of the surface of the body, local or general, is also seen in myxœdema, a condition which simulates dropsy, as described in Chapter IV., Part II.

Subcutaneous Emphysema.

Enlargement or swelling of the surface, either local or general, may occur on account of *air* underneath the skin. The skin is pale and quite distended, and hence depressions are filled up, as the axillary, clavicular, and intercostal spaces. The primary seat of the swelling is in close proximity to the air-passages, and occurs because of communication between them and the subcutaneous tissue. It may occur in ulcerations of the upper passages, as the larynx or trachea; in ulcerations of the œsophagus into the mediastinum; in the ulceration and rupture of phthisical cavities into the chest-wall; and in rupture of the lungs from hard coughing, sharp crying, severe exertions, such as blowing of wind instruments. The air may escape under the pleura to the mediastinum and thence to the neck, or, when the pleura is adherent, air will pass from the lung into the connective tissue. The swelling gradually spreads over the entire body from the seat of rupture. In a case of laryngeal phthisis under the writer's care the œdema encircled the neck and spread uniformly over the anterior and posterior portions of the thorax; thence it extended downward until it met a corresponding infiltration of the lymph-spaces with serum in the thighs. The distinction between œdematous swelling and subcutaneous emphysema could thus be made: the latter offered no resistance, did not pit on pressure, crackled under the finger, and was quite tender on pressure. Spontaneous pain was not present; but any position was painful in which the weight of the body pressed upon the part affected.

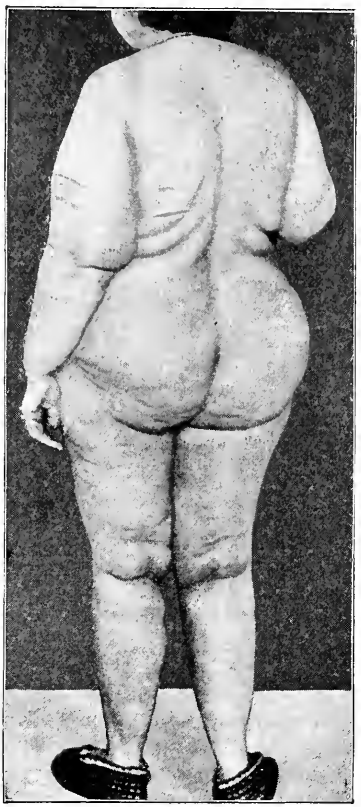
Connective-tissue Dystrophies.

Enlargements of the surface are seen in the so-called dystrophies. The dystrophy is usually due to a localized anomalous overgrowth of connective tissue, probably of trophic origin. It can easily be distinguished from œdema by the absence of the signs of œdema, and from local inflammatory swelling by the absence of pain, heat, and redness. The swelling occurs on the arms and legs, usually on the outer aspects, and may occur in various portions of the trunk. In one of the writer's cases the swellings were periodic; or, rather, the persistent swellings increased in size at irregular intervals.

Dercum has described a case of dystrophy in which the enlargements had been attributed to accumulations of fat. The patient presented marked subjective nervous phenomena, paræsthesias of all kinds, with flushings and sensations of sinking and depression. There were areas of anæsthesia, pain, and tenderness in the nerve-trunks. Pain preceded the advent of the swellings. Herpes zoster occurred in Dercum's case, and other

symptoms of neuritis were marked. The irregularity in the distribution of the swellings, their character and mode of development, the occurrence of neuritis, and the absence of perspiration distinguished dystrophy from lipomatosis or excess of fat. The patients were of a neurotic type, and mental impairment usually resulted in the course of the disease. The general nutrition failed, particularly as gastro-intestinal disorders ensued.

Fig. 35.



Adiposa dolorosa.—Note accumulations on back and on extremities. See knees and elbows; wrists and ankles unusually small. Patient aged fifty-six. Second attack of insanity. (Original.)

Scleroderma.

Scleroderma is a brawny hyperplasia of the subcutaneous connective tissue with swelling and induration. As the tissues are almost immovable, the term "hide-bound" is applied to this condition. Marked stiffness and pain are present.

In *localized* scleroderma, or *morphœa*, the skin has a waxy or dead-white appearance and is brawny and inelastic. There may be preliminary hyperæmia of the skin. Subsequently pigmentation of the hyperæmic area causes changes in color, or atrophy of the pigment may cause *leucoderma*. The secretion of sweat is diminished or entirely abolished. In the *diffuse* form the affection begins in the extremities or face and is accompanied by a sense of stiffness or tension; the skin is usually hard and firm, and gradually a diffuse brawny induration develops. The skin cannot be picked up in folds. It may appear normal, but is generally very smooth, glossy, and dryer than usual, rarely pigmented. Scleroderma may be confined to a limb or may become universal. The appearance of the face is characteristic. It is expressionless, and the lips can not be moved, while mastication is impossible; the eyes and the nose are deformed. The hands become fixed, and the fingers immobile and contracted from induration about the joints, the deformity being called *sclerodactyle*. The affection is thought to be due to a trophoneurosis or to fibrosis of the arteries of the skin, with connective-tissue over-growth in the adjacent areas.

Brawny Induration.

Œdema must not be confounded with the brawny induration of the calves of the legs in scurvy, probably from deep-seated hemorrhage. It must be remembered, however, that œdema of the ankles is very common in this affection. Brawny induration may also be found in syphilis. In a patient under the writer's care in the Presbyterian Hospital a brawny induration of the thigh, with painless swelling and stiffness of the leg, was apparently due to syphilis, as it disappeared rapidly under treatment with potassium iodide.

Localized Subcutaneous Nodules.

Sarcomata. The subcutaneous nodules are rarely if ever confounded with œdema or other swellings. In sarcoma the subcutaneous tumor becomes attached to the skin and may change its color. It is usually secondary to sarcoma in some other organ of the body. Primary sarcomata and such as are metastatic from organs in which there is normal pigmentation, as the eye, become blue or bluish-black. When the tumors are numerous, on palpation the surface is found to be rough and uneven.

Primary *melanotic sarcomata* of the skin can always be distinguished by their color. In both the primary and the secondary form the general symptoms of the disease daily become more and more pronounced, and subcutaneous hemorrhages are commonly associated with the local phenomena.

The first external evidence of *lymphosarcoma* may be subcutaneous nodules in unusual situations. Thus in a case under my observation a lymphoid nodule was first observed in the third costal interspace on the right side. Subsequently the glandular involvement followed.

Carcinomata. Subcutaneous lymphatic glands may be the seat of secondary carcinoma, and from their location may indicate the primary source of the disease. The glands above the left clavicle are sometimes secondarily affected in cancer of the stomach. In malignant disease of abdominal organs, glands in the abdominal wall are enlarged. The subcutaneous nodules should be removed and examined microscopically. The structures of the umbilicus (skin and subcutaneous tissues) enlarge, become nodulated, and sometimes the seat of fungoid ulceration in abdominal carcinoma, particularly of the stomach. It must not be forgotten that primary sarcoma or carcinoma of the skin, limited to one area, and simulating an intra-abdominal growth, may occur, as in a case under my care in the Philadelphia Hospital, and operated on by Horwitz.

Cysticercus Cellulosæ. The nature of the subcutaneous nodules of cysticercus is recognized by microscopical examination. They are usually associated with the presence of larvæ in other tissues, hence the patient complains of great soreness and stiffness and may become helpless.

Rheumatic Nodules. Subcutaneous nodules are seen in rheumatic patients in the course of rheumatism, or after the attacks, and in cases of rheumatic endocarditis. They are common in the young. They may

occur independently of the articular symptoms. They may be present in large numbers, and vary in size from that of a small shot to that of a large pea. They are of fibrous structure, and are attached to the tendons and fasciæ, particularly on the fingers, hands, and wrists, but may be found over the elbows, knees, scapulæ, and spines of the vertebræ.

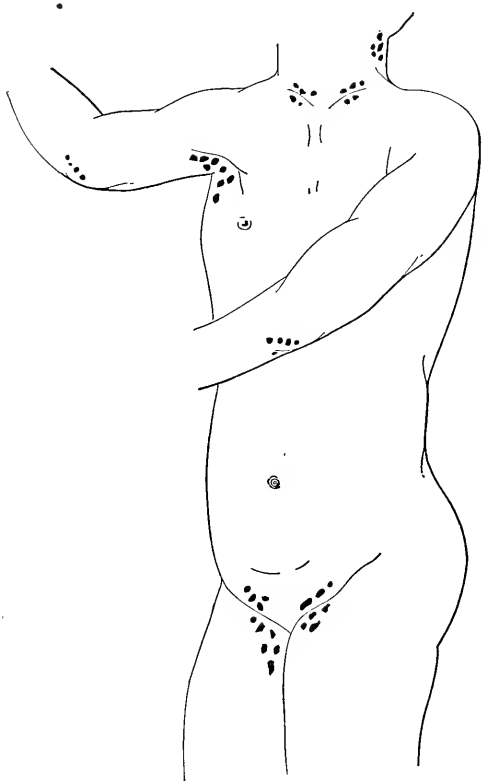
Syphilitic Nodes. Gummata are observed during the tertiary period of syphilis. They must not be confounded with the enlarged glands. They are attached to the skin, and may from time to time ulcerate. They may be seen on the back or buttocks; less frequently on other parts of the body.

CHAPTER XXV.

EXAMINATION OF THE LYMPHATIC GLANDS.

ENLARGEMENT of the lymphatic glands may be general or local. The former points to a systemic infection, as syphilis or glandular fever, to a blood disease, as leukæmia, or to malignant disease. Enlargement of a

FIG. 36.



Showing location of lymphatic glands.

single gland or group of glands indicates an infection or carcinoma in the area drained by the affected glands.

Lymphatism. Poor physical development has recently been observed with lymphatic over-growth, or the *constitutio lymphatica*. In this state sudden death is liable to occur. It is believed that one of the causes of

death from anæsthesia and from the antitoxin of diphtheria is a condition known as *status lymphaticus*. Hyperplasia of the lymphatic glands, the spleen, the thymus, and the bone-marrow constitutes the condition, and rarely is found in patients with rhachitis, and in hypoplasia of the heart and aorta. The internal lymphatic glands and the lymphatic structures of the alimentary tract are more frequently involved than the more superficial glands. With this over-growth of lymph tissue the spleen and the thymus gland are enlarged, and red marrow replaces the yellow marrow in young adults. Hypoplasia of the vascular system is not easily recognized. The left ventricle may be dilated and the peripheral arteries diminished in size.

General Enlargement of the Lymphatic Glands.

Certain types of individuals have a lymphatic system very sensitive to irritation. Such irritations may be caused merely by a mild infection, or by irritants arising from auto-intoxication, the enlargement depending not upon the virulence of the cause, but upon the individual trait of the patients.

The glands are enlarged in simple *adenitis*, *tuberculosis*, *Hodgkin's disease*, *leucocythæmia*, *sarcoma*, and *cancer*. The moderate enlargement of syphilis and the local enlargement from irritation in the area of lymph-drainage have been mentioned. In Hodgkin's disease many groups of glands are usually involved, and the spleen enlarged. The course of the disease is chronic. *Adenitis* is usually a local condition; the gland is tender and the connective tissue around it is affected. There are local heat and pain. At first the gland is hard, later it softens in the centre, and finally it exhibits fluctuation. In *tuberculosis* more than one gland is often affected. Usually the glandular involvement is bilateral, as in the neck. At first the glands are isolated; later they become matted. The local symptoms are not marked and the process is very indolent. Thick, cheesy pus which may contain tubercle bacilli is discharged. It causes tuberculosis when inoculated in lower animals—a method of diagnosis necessary in some doubtful cases. The tuberculin test must also be used. Fever and "decline" occur later, but often not until other structures, as the lungs, have become infected. (See Leucocythæmia.)

Enlargement of the cervical glands, and of the axillary and inguinal glands attended by *fever* occurs in that obscure infection described by Dawson Williams and others, and called glandular fever. Similar glandular enlargement is quite characteristic of German measles or rötheln. (See the Infections.)

Enlargement of the *post-cervical* glands, the *epitrochlear* glands, and lymphatic glands in other portions of the body points to syphilis. In the two first-mentioned localities the enlargement is of great diagnostic importance, as it is less likely to be due to any other causes.

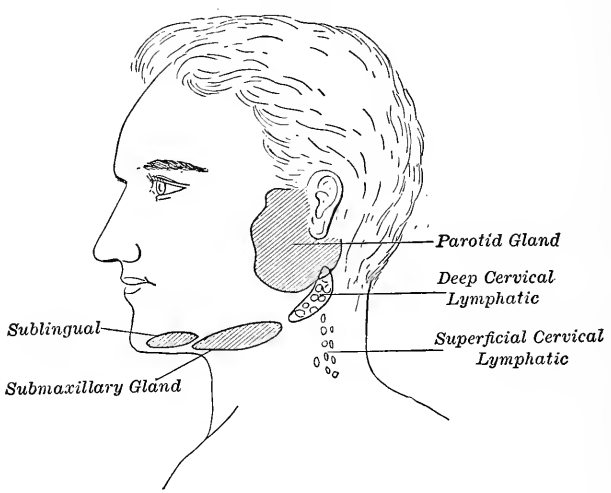
The Inguinal and Axillary Glands. The axillary glands are early affected and enlarged in mammary cancer as well as in affections causing inguinal enlargement. Secondary œdema may follow. The breast should always be examined in œdema of the arm.

Enlargement of the glands in the groin, if acute with inflammation, may result from the virus of chancroid or gonorrhœa. When enlarged from the virus of syphilis, they are hard and painless. It must not be forgotten that skin affections and infections of the extremities, as that of an ingrown toenail, cause enlargement of these glands. The enlargement may, from pressure on the vein, cause œdema of the leg.

Scars at the site of former glands point to tuberculous destruction or former bubo.

The Supraclavicular Glands. The enlarged and indurated glands may cause pressure-symptoms. The only local enlargement that is of diagnostic significance is that which is seen above the clavicle on the left side; it often points to carcinoma of the stomach, as Troisier announced.[1] Indeed, there are cases of this disease in which only the general symptoms of carcinoma are present. Local symptoms are wanting

FIG. 37.



Showing lymphatic glands of head and neck.

by which to make out the locality of the cancer. Enlarged glands above the clavicle are a fair indication that the stomach is the seat of the disease. The enlargement is probably due to transmission of the infectious material along the thoracic duct and its lodgement in the associated glands.

The Cervical and Submaxillary Glands. Enlargement of the submaxillary and cervical glands points to some infection of the mouth and throat or of the jaw and teeth. They are often the seat of nodular enlargement in *actinomycosis*. (See "collar" in adenitis of leukæmia.)

Acute enlargement of the upper deep cervical glands occurs in diphtheria, follicular and suppurative tonsillitis, and in many of the eruptive fevers. Chronic enlargement may be tuberculous if the glands are matted together and tend to suppurate; syphilitic if small and hard; in Hodg-

[1] Bulletin et Mémoires de la Société Médicale des Hôpitaux, January 13, 1888.

kin's disease the glands are large, separate, and non-suppurating; the enlargement may be secondary to carcinoma, as of the stomach. Infections of the ear and teeth may cause enlargement.

Lymphosarcoma is an infection of the glandular structures of obscure origin. A local group of glands may be involved or the glands throughout the body may be the seat of the over-growth. When the infection is general, the deep-seated glands, as the mediastinal and retroperitoneal, may be the first involved. Anæmia, fever, and signs of intrathoracic and abdominal pressure may be present without decisive indications of the nature of the disease. In a short time, however, a superficial gland becomes enlarged, and other glands follow in rapid succession. The presence of an enlarged gland in any part of the body may be suggestive of the nature of a deep-seated process. A positive diagnosis can be established by removing the gland and examining it microscopically. A case of this character which I saw with Hare showed the first evidence of glandular infection in the enlargement of a small gland over the third interspace on the right side of the chest, in front.

CHAPTER XXVI.

EXAMINATION OF THE MUSCLES.

The Data obtained by Inquiry.

THE gait of the patient, the position he assumes or in which he places the limbs would suggest inquiry as to the presence or absence of pain. Pain of a muscle or muscles may be due to *myalgia* or *myositis*, to *spasm*, or to *neuritis*. (See chapter on Pain.)

Myalgia is an inflammation of the muscles produced by cold or trauma causing pain on movement and spontaneous pain in the muscle, which is tender on pressure and may be the seat of spasm.

Palpation of the Muscles.

Nutrition. The nutrition of the *muscles* is observed by the hand of the examiner while the muscles are made to relax and contract alternately. We compare corresponding muscles of the two sides. Measurement of the limbs at corresponding situations makes the observation more accurate. The muscles may *atrophy* or *hypertrophy*. Either condition may be local, unilateral, bilateral, or general.

Spasm of the Muscles.

Myoidema is a local contraction of the muscle which occurs upon striking it with a plexímeter or the finger, as in percussion. It is more particularly seen in thin subjects, usually tuberculous, and elicited by tapping the pectoral muscles. The fasciculi rise in little humps, which persist for a short time and gradually subside. At one time myoidema was thought to be diagnostic of tuberculosis; but the symptom has no special significance.

No clinical sign is of greater importance than the spasm or rigidity of muscles in the vicinity of parts the seat of inflammation. Such spasm is a reflex contraction, and is known as *apprehensive spasm*, especially as referred to joints. It is nature's effort to protect the parts by fixation. The most notable groups, spasm of which suggests the presence of inflammation in the contiguous territory, are the abdominal, thoracic, lumbar, and cervical muscles, and the muscles attached to joints.

1. The abdominal muscle spasm, or rigidity of all the muscles of the abdomen, occurs in general peritonitis. It may be so extreme as to give to the abdomen the so-called "board-like" rigidity. Local spasm occurs under the following circumstances: Rigidity of the right rectus muscle occurs with inflammation of the gall-bladder, with localized peritonitis due to duodenal perforation, as well as other forms of inflamma-

tion in this vicinity. Spasm of the left rectus in its upper half suggests gastric perforation or pancreatic disease. Rigidity of the muscles over the right lower quadrant occurs in appendicitis, and in perforation from typhoid fever or other ulcerative lesions of the intestine.

2. Spasm of the thoracic muscles occurs over the affected side in cases of pleuritis.

3. Spasm of the lumbar muscles, of the right or left side, occurs when there is pyelonephritis or perineal abscess on the side of the spasm.

4. Contraction or rigidity of the muscles of the neck is an early sign of spinal meningitis.

5. Spasm or rigidity of groups of muscles along the course of the spinal column occurs in the various forms of osteitis.

The limitation of movement induced by the spasm points to the location of the inflammatory process. The student will do well, therefore, to keep in constant mind the significance of muscle spasm when it is found, and to consider as a valuable sign the presence of spasm or rigidity in the diagnosis of deep-seated inflammations.

Atrophy of the Muscles.

There are several varieties of atrophy: 1. The atrophy of disuse. 2. Myopathic atrophy. 3. Myelopathic atrophy, or the atrophy of degeneration, which follows lesions of the motor path, the cortex, the medulla, or the spinal cord, and develops as a result of neuritis. (See Nervous Diseases.)

The Atrophy of Disuse. It is also known as the atrophy of inactivity. The muscles are slightly lessened in volume. The atrophy takes place very slowly; it supervenes in cases of paralysis and in the joint-diseases which cause immobility. It occurs also in joint-disease from reflex influences. The electrical reactions of the muscles are qualitatively unchanged. By this reaction atrophy from disuse and atrophy from disease of the muscles can be distinguished from myelopathic atrophy, due to disease of the nerves (neuritis), or to degeneration of motor nerves and ganglia.

Myopathic Atrophy—Muscular Dystrophy. In this form of atrophy the muscle is diseased. It diminishes in volume and finally becomes completely shrunken.

Idiopathic Muscular Atrophy. *Dystrophia muscularis progressiva* (Erb). In this variety of dystrophy muscular wasting takes place with or without initial hypertrophy. Three forms are seen:

1. ATROPHY WITH PSEUDOHYPERTROPHY. This usually begins in childhood, and is often of congenital origin, being transmitted through the mother. It is first noticed just as the child is learning to walk. The extensors of the leg, the glutei, the lumbar muscles, the deltoids, and the triceps and infraspinati muscles are involved, but the first change takes place in the muscles of the calves. The muscles of the face, neck, and forearm are not usually affected in this form of the disease; the muscles of the hand are not involved. While hypertrophy progresses in certain muscles, others waste. The calves may hypertrophy, for instance,

while the extensors of the leg waste away and become weak. Attitude and gait are characteristic. (See page 156.) The patient stands erect, with the legs apart, the shoulders thrown back, the spine curved, and the abdomen prominent. The waddling gait prevails, and the method of getting up from the floor is pathognomonic. The course of the disease is slow; wasting follows the hypertrophy, but the weakness is greatest in the muscles first atrophied. Contractures and distortions of the spine and of the bones of the leg take place.

2. Primary Atrophy. This is likewise congenital or manifests itself in early life. It is divided into different types, according to the groups of muscles that are affected. The same process occurs as in the first, except that pseudohypertrophy is not primary. There may be several forms in different members of the same family. Of these we have *the juvenile form of Erb*. The upper arm and shoulder and the thigh muscles are first involved. Later the muscles of the gluteal region and calf may become enlarged and hard. The back muscles are gradually affected, inducing the attitude previously mentioned. The reaction of degeneration is not present. There is also an *infantile type*, first described by Duchenne, or the *facio-scapulo-humeral type*. Erb's form begins about puberty. The other forms usually begin in childhood, but may be delayed. The face is involved; it is expressionless, and in laughing the muscles move slowly; the child can not whistle, as the lips are thick and everted. The eyes remain partly open. The muscles of the group waste; later the thighs become involved. Erb has given a useful test to determine the strength of the shoulder and girdle muscles. When the child is lifted by the armpits, if the scapulohumeral groups are weak, the shoulders are forced up to the child's ears without resistance.

3. Peroneal Atrophy. A *peroneal type* of muscular atrophy has been described by Charcot. The extensors of the great toe and afterward the common extensors and peronei muscles are affected, resulting in club-foot. The muscles of the thigh may become involved later. When the disease occurs in childhood it gradually spreads to the upper extremities and affects the muscles of the hand, differing in this respect from other forms of muscular atrophy. The thenar, hypothenar, and interossei muscles are symmetrically involved, producing the claw-hand. Unlike the other forms of atrophy embraced under this heading, the peroneal type is attended by disturbances of sensation, pain, fibrillary contractions, and vasomotor changes. The reactions of degeneration may be present. It is thought by competent observers to be simply a form of neuritis, and is also called *progressive neural muscular atrophy*.

Diagnostic Features of Myopathic Atrophies. The disease is characterized by gradual progression of wasting and weakness in various groups of muscles not specially related. We never see wasting of the intrinsic muscles of the hands, as in the spinal forms of muscular atrophy, or of the tongue, pharynx, larynx, and eye. Complete paralysis rarely ensues. Electrical irritability is lessened. The reaction of degeneration is not present. Fibrillary twitching is not seen. Sensation is not affected. The reflexes are diminished and later may be lost. The sphincters are not involved; deformities about the joints or in the

spinal column may occur. The myopathies occur early in life, and are hereditary.

The *diagnosis* of idiopathic muscular atrophy is not difficult if the above-mentioned facts are borne in mind. The fact that it occurs in family groups is an important point in the diagnosis. In *cerebral atrophy* there is primary loss of power. In *chronic anterior poliomyelitis* (*spinal atrophy*) wasting begins in the muscles of the hands; in both the simple and the spastic form there are reactions of degeneration, fibrillary twitching, and increase in the reflexes, and in the latter, spastic contraction of the legs.

In *neuritis* the paralysis is proportionately greater than the atrophy. Sensory symptoms are often present. The cause is distinct. There is no family history.

General Atrophy. In cachexias the muscles as well as the tissues undergo atrophy. Even in nervous disease the atrophy of the muscles markedly increases when general wasting takes place.

RAYMOND'S TABLE OF ATROPHIES.

- Circumscribed atrophies
  - Atrophy from compression.
  - Atrophy in inflammatory conditions (pleurisy, joint-disease, etc.).
  - Atrophy from injury or inflammation of individual nerves.
- Progressive atrophies
  - Progressive spinal muscular atrophy: type Aran-Duchenne.
  - Progressive myopathic atrophy
    - Pseudohypertrophic muscular paralysis.
    - Type Leyden-Möbius.
    - Type Zimmerlin.
    - Type Erb.
    - Type Landouzy-Déjérine.
    - Type Charcot-Marie.
- Diffuse atrophies
  - Anterior poliomyelitis
    - Infantile form.
    - Acute of adults; spinal paralysis, with rapid course and curable (Landouzy-Déjérine); subacute and chronic form; chronic mixed form (Erb); diffuse subacute general spinal paralysis (Duchenne).
  - Syringomyelia.
- Facial hemiatrophy
  - Multiple neuritis (amyotrophic form)
    - Lead paralysis.
    - Leprous neuritis.
    - Alcoholic neuritis.
- Muscular atrophies of cerebral origin
  - With secondary degeneration involving the anterior cornua.
  - Without secondary degeneration involving the anterior cornua.
- Muscular atrophy in hysteria
- Muscular atrophy from systemic disease of the cord
  - Amyotrophic sclerosis.
  - Glosso-labio-laryngeal paralysis.
- Atrophy complicating other disease of the cord
  - Atrophy in myelitis.
  - Atrophy in compression of the cord.
  - Atrophy in multiple sclerosis.
  - Atrophy in tabes dorsalis.

Hypertrophy of the Muscles.

Hypertrophy of individual muscles occurs from over-use, as when an extremity or a portion of the trunk is used in excess. General hypertrophy of muscles occurs in Thomsen's disease. True hypertrophy is recognized by increased volume, great hardness, and increased vigor of the muscle.

Pseudohypertrophy (see under Muscular Atrophy) is associated with increased volume of muscle but diminished power.

Fibrous Tissues.

Intimately associated with rheumatic affections of the muscles is that of the fibrous tissues or fasciæ. Pain, fixation, and tenderness are noted, and if with them other rheumatic manifestations are found, the diagnosis is established; especially is the above true of trauma.

CHAPTER XXVII.

EXAMINATION OF THE BONES AND JOINTS.

Method of Examination. When the bones and joints, especially the spinal column, are to be examined, the patient should be stripped and after the movements and position in the upright or semi-upright posture have been noted, he should be made to lie down on a hard, smooth surface, and the trunk and joints examined in that position. Anterior, posterior, and lateral movements of the spinal column must be made to determine its flexibility. In this manner deformities, changes in the length of the bones, and abnormal postures can be carefully observed. In addition we must note muscular wasting, the presence of *local tenderness* and *swelling*, the *movements* of the joints, and loss of other functional activity causing lameness or joint-disability.

To distinguish joint-lesions from abnormal flexions or extensions due to *spasm* of muscles, *anæsthesia* must be employed.

THE BONES.

The Skeleton.

The bones may be the seat of nutritive changes involving the entire skeleton, on account of which the body is enlarged or diminished in size, in accordance with the change in size of the skeleton. Individual bones may be changed in position or may be the seat of inflammation or morbid growths. Many such changes will be considered in the section devoted to the extremities.

Enlargements. Nutritive changes giving rise to enlargements of the bones occur in acromegaly, osteitis deformans, and pulmonary osteo-arthropathy.

Acromegaly. Marie first described *acromegaly*, a skeletal change characterized by hypertrophy of the bones of the hands, feet, and face. The fibrocartilages of the ear and larynx are also enlarged. The enlargement of the inferior maxillary and frontal bones causes the face to assume a peculiar, elongated, elliptical outline. The nasal bones are enlarged, and the nose thickened; the temporal fossæ are deepened on account of enlargement of the malar bones. The forehead retreats because of the enlargement of the frontal sinuses and projection of the superciliary ridges; the chin is prominent and the lower teeth project beyond the plane of the upper; the lips and eyelids may be thickened; the tongue is enlarged and thickened. The hair is coarse and dry; the skin of the face dry and pigmented.

The hands are peculiar; they are much broader; the fingers are sausage-shaped, and the hand spade-like in form; the nails are flat, striated, and small. There is usually spinal curvature; the abdomen is prominent, and the height is increased. The muscles become weak and may atrophy; the skin is often pigmented; varicose veins have been observed, and the patient complains of hemorrhoids. The thyroid gland may be atrophied or hypertrophied. It may be well to state, in passing, that with these appearances nervous phenomena are observed and disorder of special senses complained of. Hemianopsia, limitation of the visual field, and blindness or deafness arise.

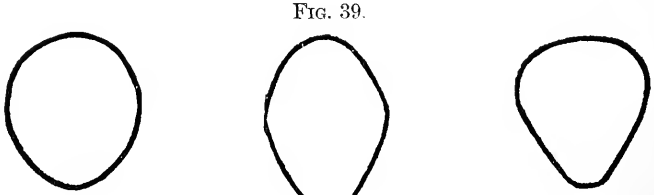
FIG. 38.

Case of acromegaly. (OSBORNE.)

Osteitis Deformans. Another remarkable change is seen in the skeleton, and has been described by Sir James Paget under the name of osteitis deformans. There is a marked change in the contour of the patient, who exhibits a peculiar mode of locomotion. The head is advanced and lowered so that the neck is very short, and the chin, when the head is at ease, is more than an inch below the top of the sternum. The chest becomes contracted, narrow, flattened laterally, deep from before backward, and the movements of the ribs and spine are lessened; the arms appear unnaturally long; the shafts of each tibia and femur are bent so that the patient becomes bow-legged. There is some stiffness, but no loss of power and not a great deal of pain. The skull is increased considerably in thickness.

These changes in the bones cause a dwarfed appearance of the trunk in comparison with the legs and arms, and the posterior lateral curvature

FIG. 39.



1
Outline of face in myxœdema.

2
Outline in acromegaly.

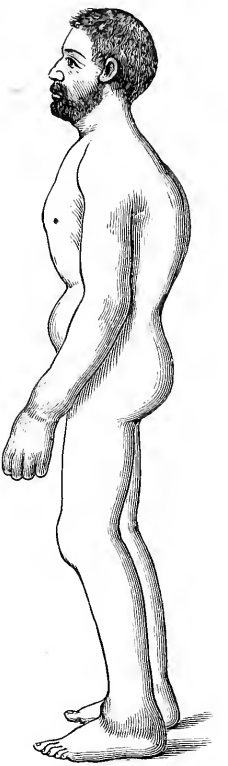
3
Outline in osteitis deformans.

necessitates a characteristic attitude. The skeletal changes are noted particularly in the long bones. As a result of the enlargement of the cranial

bones the face presents a triangular outline, with the base above and the apex below (see Fig. 39, outline 3), thus differing in appearance from the outline in acromegaly (Fig. 39, outline 2).

Pulmonary Osteo-arthropathy. Marie distinguishes acromegaly from another skeletal change in which there is hypertrophy of the bones of the extremities, including enlargement of the shafts. In this form of arthropathy the bones of the head and face are not affected. The hands and feet are enlarged, and the patellæ and other bones of the knee-joints increased in size. Curvature of the spine is present. The appearance of the fingers is different from that seen in acromegaly. The ends are enlarged and bulbous, and the nails are large and curved both in the transverse and in the longitudinal direction, like the clubbed fingers of phthisis; although the chief enlargement of the fingers is not terminal, and there is no cyanosis as in phthisical clubbing. The change seemed to be associated with pulmonary affections, and Marie called it *osteo-arthropathie pneumonique*.

FIG. 40.



Pulmonary osteo-arthropathy.

Diminution. Imperfect development of the bones is seen in idiots and cretins; general diminution in size of the skeleton with deformities may occur from rhachitis or from osteomalacia.

Osteomalacia. Among the general affections of the skeleton that may cause lessened size, osteomalacia must not be forgotten. As the lime salts are dissolved, the bones become preternaturally soft, break on the slightest provocation, or bend in various directions, depending upon the external pressure and the direction of the muscular force. The ribs are drawn in by the inspiratory force until the cavity of the thorax is lessened to a degree incompatible with life. The pelvis is deformed so that labor is impossible. (The disease frequently develops during pregnancy.) All sorts of fixed contortions are assumed. If the patient is able to be up, the body shortens, the back becomes rounded, and the neck flexed so that the chin is brought close to the sternum. On palpation the bones can be indented with the finger, and crackle like egg-shells.

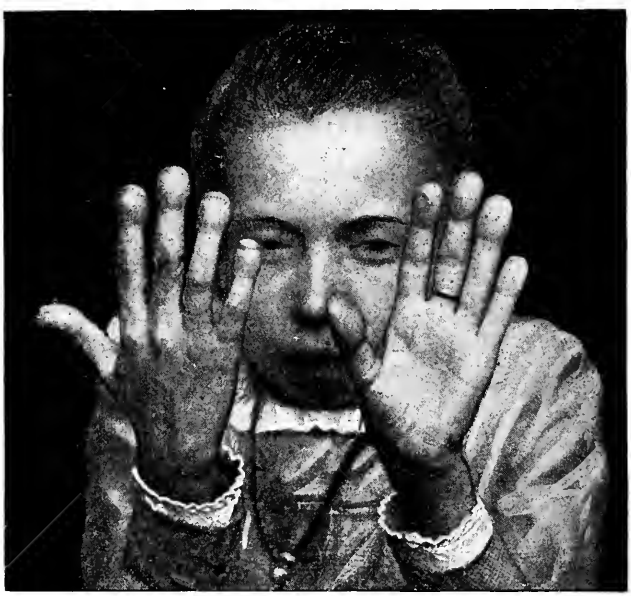
Osteomalacia is easily distinguished from *carcinoma* or *sarcoma* of the bones. In the latter spontaneous fracture occurs in various parts of the skeleton, but is generally preceded by pain and swelling at the seat of fracture. Then, in sarcoma, subcutaneous hemorrhages are present. When a single joint is affected in osteosarcoma, the same egg-shell crackling is observed.

Individual Bones.

Position and Shape. The peculiar position (falling downward) of the *scapula* in paralysis of the serratus magnus is diagnostic of that

affection, and indicates disease of the posterior thoracic nerve. In examination of the *clavicles* fractures must not be mistaken for disease of the bones, such as rhachitis. The examination of the *spinal column* is of the greatest importance. (See Spinal Joints.) A study of the diseases of the spinal column due to caries from tuberculosis is not within the province of this work; no physical examination, however, is complete without an investigation of the movability of the spine and the presence or absence of curvature. I refer to the curvature due to weakness of groups of spinal muscles. Functional disorders of the gastro-intestinal tract and the uterus are undoubtedly intensified by the presence of curvature, which

FIG. 41.



Pulmonary osteo-arthropathy. Female, aged eleven years. Tuberculous vertebral caries and pulmonary tuberculosis. Enlarged clubbed fingers and thickened ulna and radius. Private patient, 1885. (Original.)

leads to deformity of the body, and hence to the assuming of abnormal positions when sitting or walking. The recognition of lateral or anterior curvature leads to the adoption of lines of treatment which otherwise would not be followed, but without which weak muscles, improper aëration of the blood, and sluggish circulation would persist. Pain in the distribution of nerves or at their termination is often due to spinal caries pressing on them as they pass through the foramina. The most notable is the pain about the umbilicus due to Pott's disease in children.

The bones and cartilages connected with the thorax will be considered under Diseases of the Lungs.

Nodules or Nodes. Bone swellings are usually due to *periostitis*, the causes of which are trauma, infection, and syphilis. In syphilitic periostitis the swellings form on various portions of the skeleton, but are seen

most frequently on the skull, especially on the forehead; they are also found on the shafts of the long bones, preferably the tibia, ulna, and clavicles. They are usually multiple or bilateral. They are painful and tender on pressure, and may be the seat of heat and redness. *Exostoses* are hard and dense. They are situated on the outer aspects of the bone and in relation with the strongest tendons or muscles.

As an illustration of the importance of recognizing nodes, the writer recalls a case of persistent headache, the true nature of which was only ascertained by finding a small node on the skull. The headache had been of long duration (five years), and treatment for it had been sought in many countries.

Myelomata. It is interesting to note that multiple tumors of the skeleton or myelomata, according to Ellinger, sometimes present the clinical picture of pernicious anæmia. Of great diagnostic value, as pointed out by Fitz and insisted upon by Ellinger, is the presence of *albumosuria*, which has been found in pernicious anæmia. (See the Urine.)

Tenderness of the sternum upon pressure is often of diagnostic significance, and is usually indicative of syphilis. The pain and tenderness just noted, however, must not be confounded with local tenderness due to *necrosis*, which often arises in convalescence from fevers, notably those of an infectious nature.

Inflammation. The discovery of a slight swelling of the surface may lead to the recognition of a grave general process. Simple local inflammation or *periostitis* is recognized by localized pain, swelling, and slight œdema. It may be diffuse. It is seen most frequently on the tibia, sternum, and clavicle. It not infrequently follows typhoid fever. It may be due to syphilis.

Osteomyelitis. The occurrence of high fever, with or without chills but usually with pyæmic symptoms, without recognized cause, should lead to an examination of the bones. A spot of tenderness followed by local redness and swelling—on the tibia, for instance—would indicate the seat of suppuration in *osteomyelitis*.

THE JOINTS.

The Data Obtained by Inquiry.

Careful study of the bones enables us now and then to discover the nature of a general morbid process, as has just been indicated. It is true that *osteomyelitis* is less likely to be recognized than any other process; but when the patient has been exposed to an infection and fever is present, this condition must always be sought for, especially if no other infected area can be found.

This is not true, however, of joint-disease. We can determine which joint is affected as well as the nature of the morbid process. Other data are also needed, and we collect the usual data obtained by *inquiry*. The *social history* is not productive of valuable data. Acute rheumatism is more common in early life, rheumatoid arthritis in the middle periods,

and chronic rheumatism in late life. Females are more commonly attacked than males by rheumatoid arthritis, and the affection is more common in the poorer classes. Males and the well-to-do are the victims of gout.

From the *family history* one learns of the transmission of gout from generation to generation and of the occurrence of rheumatism or of the various allied processes in members of the same or previous generations. *Previous diseases* elicited are those of an infectious nature or an intoxication, as of lead. Such diseases must be sought for if the true nature of an arthritis is to be discovered. The history of the *present disease* is often that of recent infection or intoxication.

The *subjective symptoms* of joint-affections are worthy of note. *Pain* is the most prominent. This may be spontaneous, or may arise upon pressure, or follow attempts at movement. Spontaneous pain with tenderness is more pronounced in rheumatic and gouty inflammations of the joints. The pain in joint-disease is usually worse at night. This is particularly the case in tuberculous joints, and is due to removal of the apprehensive spasm of the muscles whereby the joints had been protected. It must be remembered that it is not unusual to see an hysterical joint. (See page 254.)

Pain in the joints must not be confounded with that of local or multiple neuritis. I have seen the pains of neuritis attributed to rheumatism of the phalanges, tarsus, and ankle until paralysis of the extensors took place. I have seen the pain of neuritis of the circumflex mistaken for shoulder-joint disease. Multiple neuritis is attended by pains that may be referred to the joints ; but neither in local nor in general neuritis are the joints ever swollen, tender, or painful on passive movement.

The Data Obtained by Observation.

Inspection. The *size, shape,* and *color,* the degree of *mobility* and the *position* of the joints are observed. The *number* of joints involved and the duration of the process in each joint must be carefully noted.

The Size and Shape. The joints may be *enlarged.* The enlargement may be due to infiltration of the tissues about the joints, to effusion within the joints, serous or purulent, or to inflammation of the ends of the bones.

1. When the enlargement is due to infiltration about the joint, the tissues are previously thickened, as shown by palpation, and the outline of the joint is changed. The normal contour is lost entirely, and instead there is a globular swelling beginning above and extending below the joint. 2. When the enlargement is due to effusion, it may be detected by palpation, as this elicits fluctuation. This is particularly so in the large joints. If the joint involved is the knee, the patella will float. The effusion changes the normal contour, but may in the earlier stages cause local swellings where the synovial sacs are near the surface ; hence, at the articulation of the tibia and fibula with the tarsus, on the inner and outer side, a boggy swelling is observed. At the knee the swelling is on each side above and below the patella. When the effusion is great,

the joint becomes immobile and may be flexed from distention of the sac. 3. When enlargement of the joints is due to hypertrophy of the bones, as in Charcot's disease, the latter are thickened and very hard. There may or may not be, and usually is not, fixation, and movement is but moderately interfered with.

Changes in the *outline* of the joint are also seen in rheumatoid arthritis. The loss of the cartilaginous substance of the joint, with the secondary osteophytic changes, causes deformity so that in the case of the small joints of the finger subluxation is seen; similar subluxations are seen in larger joints. The ends of the phalangeal bones are thickened.

The Color. Change in the *color* is usually noticed in inflammations. The surface is either bright red or dusky.

The Position. The *position* assumed is of diagnostic importance. Flexion of the limb of the affected joint occurs in over-distention. It must be remembered that the hip-joint is flexed in *appendicitis* and in *psoas abscess* or other affections in proximity to the psoas muscles. In rheumatoid arthritis there is subluxation. *Immobility* is observed. (See Palpation.)

Palpation. By palpation we determine the degree of *mobility*, employing passive motion of the joints; the presence of *fluctuation* and of *crepitation;* the condition of the *bones*, if *thickened* or *irregular;* the presence of *bulging* or *bogginess* along the margin of the joint from thickened synovial membrane.

1. The *mobility* of the joint is modified because of *pain*, muscular *spasm*, *distention* of the capsule by effusion, the presence of *osteophytes*, and *anchylosis*. Movement is inhibited in inflammation on account of the *pain*. Spasm prevents movement. A reflex muscular spasm takes place if osteitis and cartilage-destruction are present. In effusion there is less mobility, or even none at all. Movement is prevented in rheumatoid arthritis by the osteophytic growths which surround the joint. Fibrous anchylosis prevents movement.

2. *Fluctuation* is revealed by palpation, and points to liquid effusion within the joint. Œdema of the surrounding tissues occurs with purulent effusions.

3. A *crepitus* or grating sensation is observed in rheumatoid arthritis and other destructive diseases.

Joint-lesions (Secondary).

It must always be remembered that joint-lesions or processes may be expressions of *general infections*, as septicæmia, influenza, pneumonia, cerebrospinal meningitis, scarlet fever, and dysentery; of *blood diseases*, like purpura, hæmophilia, or scurvy; or of *nervous diseases*, like tabes dorsalis.

We have to consider synovitis or arthritis single and multiple, traumatic, toxic, or infectious; gonorrhœal and tuberculous infections being the most common monarticular causes. We shall then consider rheumatism and gout, rheumatoid arthritis, and finally the neuropathic joints.

Synovitis—Arthritis. The inflammation is recognized by pain, heat,

redness, and swelling. Effusion is present, and its physical signs are readily elicited. It is both periarticular and intra-articular. It may be due to traumatism; but we are now chiefly concerned with inflammations due to internal morbid processes. When single joints are affected, the most common causes are *tuberculosis*, *pyæmia*, and *gonorrhœal infection*. A mild degree of inflammation may be limited to one joint in subacute rheumatism. When many joints are affected, the cause is an infectious one, as *rheumatism*, *septicæmia*, *pyæmia*, *epidemic cerebrospinal meningitis*, *scarlet fever*, and *dysentery*, rarely *gonorrhœa*.

The Tuberculous Joint. In *tuberculosis* the joint is swollen and the neighboring tissue œdematous. Effusion may be detected. There is fever. The hip, the knee, the elbow, the wrist, and the ankle are most frequently affected. Inoculation of guinea-pigs with fluid from the joint may cause tuberculosis. Destruction ultimately takes place, with subluxations and subsequent fixation of the joint. With fever, wasting, and local signs of tuberculosis in other portions of the body the tubercular nature of the affection is indicated. The tuberculous process may be limited to the affected joint, extend to the tendinous sheaths, or secondary tuberculosis of internal organs may supervene.

The Joint of Gonorrhœal Rheumatism. The knee-joint is usually affected. Signs of acute or subacute inflammation are present, with œdema and effusion. The patient is a male in whom an acute or chronic urethral discharge is found. The pain is worse at night. The process is of long duration. Metastasis does not take place. Destruction rarely occurs, but anchylosis may. General pyæmic symptoms may ensue, and endocarditis, with or without emboli and other symptoms of the infections, so-called malignant form, may supervene. The micro-organisms (gonococci) can be found in the blood and in the pus of the affected joint. The general and local signs of rheumatism or of a rheumatic diathesis, and changes in the urine, skin eruptions, cardiac lesions, etc., are wanting. In certain cases several joints are affected, but the temperature is not so high nor the sweats so profuse as in acute rheumatism. Tenosynovitis is not infrequent.

The Tabetic Joint. In forms of nervous disease, particularly in sclerosis of the posterior columns, secondary joint-involvement sometimes occurs. The change in the large joints is preceded by pain, stiffness, and disability. Gradually nutritive changes take place. At first there is boggy swelling. The cartilages become eroded, the heads of the bone waste, the ligaments ossify, and irregular bony growths project. Wasting of the head of the femur is followed by dislocation. Sometimes an effusion takes place into the joint, and there may be periarticular œdema. The large joints—knee, hip, ankle, and elbow—are most commonly affected. Injury excites the abnormal atrophic process. When the tarsal bones and the articulations are affected, the foot becomes flat, and the tarsal and metatarsal articulations and the tarsal bones project forward or backward. This is called the tabetic foot.

The Joint of Hysteria. Symptoms of joint-disease are seen in hysteria. Pain and fixation of the joint are sometimes present. The joint rarely undergoes organic changes, but sometimes a plastic infil-

tration of the connective tissue outside of the capsule does occur. The hysterical nature of the pain and immobility are recognized by the absence of a cause for joint-lesion, the absence of fluctuation or of signs due to erosion, by the association of the local symptoms with the phenomena of hysteria, but more particularly by the fact that contraction and even wasting precede the joint-symptoms. In true affections of the joint both occur *after* the joint has become diseased; in hysteria muscular contraction takes place *first*.

The knee is the joint usually affected. Care must be taken not to be deceived by local vasomotor changes of hysterical origin which may be observed under the surface of the joint. This local increased temperature is not associated with general fever, however, while the vasomotor changes indicated by the swelling of the skin, increased tension, and shining appearance, with increased sensibility, are not persistent, but occur once or twice in the twenty-four hours. In a remarkable case of Mitchell's the local vasomotor change took place at night. The temperature of the affected knee increased 3 or 4 degrees, while the pulse remained at 80. The local symptoms of heat, redness, swelling, tension, and increased pain passed away by 3 o'clock in the morning. The fact that the same symptoms could be brought on by handling the knee or by pressure upon the patella pointed to its vasomotor origin.

In joint-cases of hysterical origin the reflexes must be studied. They do not change, and the electrical reactions are normal, although there may be atrophy from disuse, but not to the degree that occurs in organic disease. The muscles may be contracted, but, as previously noted, the contracture is primarily a relaxation, which takes place if the tension is removed. Concerning these vasomotor changes, Sir James Paget's expression, "A joint which is cold by day and hot by night is not an inflamed joint," is a safe guide to the recognition of an hysterical joint. When the joint becomes hysterical after injury, it is most difficult to ascertain its true nature.

Special Joints. The three joints that should concern the student more particularly are the shoulder, hip, and knee. When symptoms are referred to any one of these joints, they should not be passed over lightly. Grave consequences have followed the attributing of *hip-joint* inflammation to rheumatism when it was of tuberculous origin. Not only has hip-joint disease been mistaken for rheumatism, but the mistake has even been made of considering the process to be going on in the knee instead of in the hip. This is because there is often flexion of the leg, and because pain is so often referred to the knee-joint.

On the other hand, cases of *hip-joint* disease have been mistaken for suppuration in the pelvis or in the iliac fossa. Typhlitis and appendicitis have frequently been mistaken for hip-joint disease.

In the case of the *shoulder-joint* there is danger of confounding neuritis of the circumflex nerve, and consequent paralysis of the deltoid, with affections of the joint. Although the patient is unable to move the joint himself, it is quite readily moved by the physician, and the physical signs of joint-inflammation are wanting.

CHAPTER XXVIII.

EXAMINATION OF THE NOSE AND THE LARYNX.

THE NOSE

The Exterior. The external appearance of the nose is of diagnostic significance when marked deformity is present. Its shape is changed in myxœdema (*q. v.*) and in disease of the bone due to syphilis. The latter causes sinking or depression of the bridge of the nose, which must not be confounded with the depression that occurs in fracture. The nose may be broadened in cases of tumors of an expanding nature in the nasal cavities. The local change soon extends to the cheek. The nose is also the seat of eruptions, as acne and hyperæmia, but they are usually of local origin. They may be suggestive of a gouty diathesis.

The Interior. Examination of the cavities of the nose consists of two procedures, both of which are necessary to determine with accuracy the condition of the organ. These are:

1. **Anterior Rhinoscopy.** For this are needed a good light, a nose speculum of some form, probes, a 5 per cent. solution of cocaine, and a head-mirror with central opening.

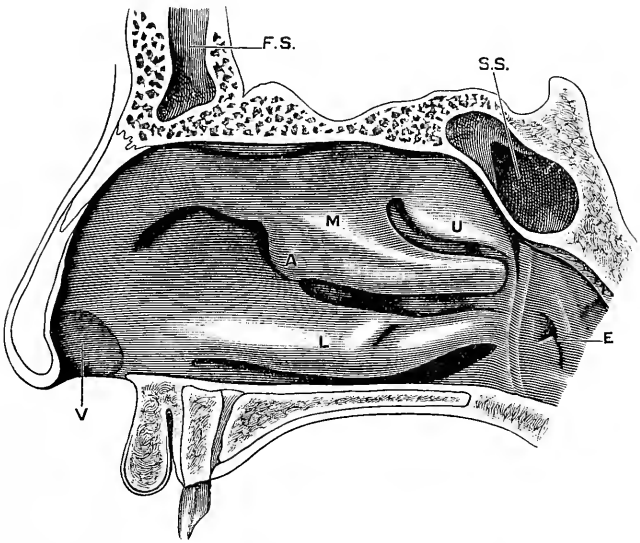
The examiner proceeds as follows: The patient is seated facing the surgeon, with the light behind and at the side of the head, as nearly as possible on a level with the examiner's eye. He must sit with shoulders and head a little forward. The operator adjusts the head-mirror so that the central aperture is in front of his own eye, and the reflected light falls on the patient's nose. It is very important in nasal examination that the operator look through the aperture and not *under* the mirror. The speculum is then taken in one hand and the nostril dilated, so that the view of the interior is unobstructed. Do not try to dilate the bony part of the nose, but only the vestibule Proceed from before backward with the examination, carefully focusing the light on each part in succession, and gradually tilting the head of the patient backward. Thus the floor of the nose, the septum, inferior turbinated bone, middle turbinated bone, and sometimes the superior turbinated bone, are brought into view successively. In a broad nose one may at times see the posterior wall of the pharynx, which is distinguished by its peculiar wave-like movement when the patient swallows.

The use of the probe is important, and without it no positive diagnosis can be made. With the probe the operator tries the condition of the mucous membrane, tests the consistency of tumors or hypertrophies, and so judges the character of the condition. After this the enlarged parts should be touched with cocaine and the result observed. Contraction of a swelling under its influence proves its vascular origin.

2. Posterior Rhinoscopy. This is the most difficult part of the examination and requires much practice on the part of the examiner. The instruments needed are a tongue-depressor, head-reflector, two sizes of throat-mirrors, a palate-hook or flat strings for holding forward the soft palate, and a curved applicator for cocaine, or a spray bottle with tip turned upward.

The patient is seated as before, the tongue held down with the tongue-depressor, and the patient is told to breathe freely through both mouth and nose. The light is directed into the pharynx and a mirror of the largest possible size inserted carefully behind the soft palate. The proper angle and the movement necessary to bring all parts into view can only be learned by practice. As a rule, it is best to hold the handle well up

FIG. 42.



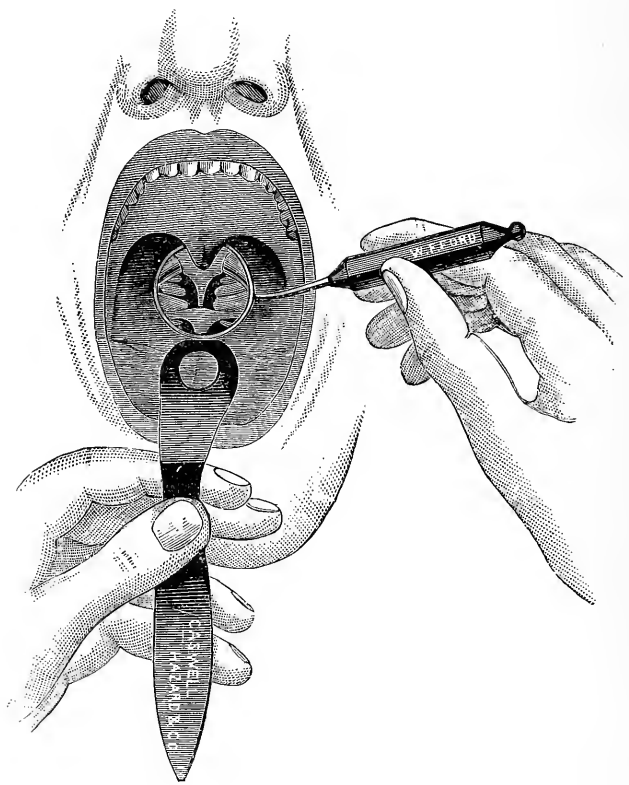
Anteroposterior section of the nose, showing the outer wall of the right nasal cavity. (Zuckerkandl.) *L*, inferior turbinate; *M*, middle turbinate; *A*, anterior end of middle turbinate; *U*, superior turbinate; *F. S.*, frontal sinus; *S. S.*, sphenoidal sinus; *E*, Eustachian tube; *V*, vestibule.

at first, and note the condition of the vault of the pharynx, then gradually depress it, examining the choanæ from above downward. Do not keep the mirror too long in the throat. It is better to insert it several times than to weary the patient by attempting to see everything the first time. After the choanæ have been examined, a turn of the mirror to either side will bring into view the orifices of the Eustachian tubes, and the examination is complete. If, after repeated attempts, it is found to be impossible to see the posterior nares, one must first seek to accustom the patient to the presence of the instruments; if this fails, it may be necessary to resort to the palate-hook or the cords to hold the uvula forward. The best hook is White's. It is necessary to apply cocaine to the soft palate before inserting the hook. Another plan, which is preferred by some, is to take the flat cords used for corset-laces, soak them in

mucilage and dry them. These are then stiff enough to pass through the nostril, yet flexible enough to pull down and out through the mouth with forceps. Then by drawing forward both ends the soft palate is pulled out of the way.

Sometimes a view of the posterior nares may be obtained by making the patient breathe in short, quick gasps, by which the uvula is released. In ordinary breathing it is often tightly pressed against the posterior wall of the pharynx.

FIG. 43.



Rhinoscopic mirror in position, (BOSWORTH.)

By the above methods the appearance and nutrition of the mucous membrane, relative size of the cavities, the nature of the discharge, and the presence of ulceration or perforation of the septum are determined. Deviations of septum, enlargement or contraction of turbinated bones, the size of the cavities, and the presence of foreign bodies or abnormal growths are also detected.

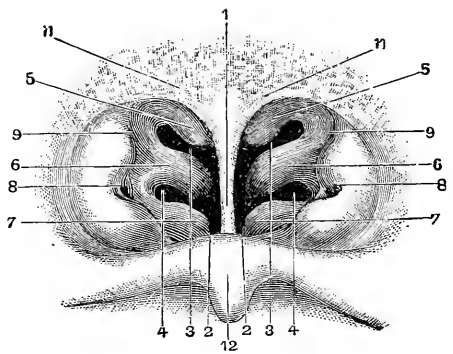
Appearance of the Mucous Membrane. The observer may find it unusually pale. This is seen in tuberculosis and in atrophic rhinitis. If a protuberant mass is observed to be transparent and shining, as well as pale, it is a *polypus*. If the mucous membrane is bright red, it may be due to *acute inflammation*, to *glanders*, or to *syphilis*. It is dull

red in *chronic catarrhs* and *caseous rhinitis*. The coatings of the mucous membrane are of significance. If a dry mucus covers the part, there is *dry catarrh;* on the other hand, a dirty-gray membrane is indicative of *diphtheritic rhinitis*.

The mucous membrane is swollen and bathed with a serous, seropurulent, or purulent discharge, the character depending on the stage of inflammation. The contractile tissue over the turbinated bones is congested and swollen. It feels elastic when touched with the probe, and shrinks when cocaine is applied.

In *chronic hypertrophic rhinitis* the uvula is thickened and elongated, on account of the hawking. The outer surface or the edges of the turbinated bones are enlarged throughout or in localities. The mucous membrane covering these spots is thickened, hard, and rough. If cocaine is applied, the mucous membrane does not contract, as in the swelling due

FIG. 44.



Rhinoscopie image. (SEILER.)

1. Vomer or nasal septum. 2. Floor of nose. 3. Superior meatus. 4. Middle meatus. 5. Superior turbinated bone. 6. Middle turbinated bone. 7. Inferior turbinated bone. 8. Pharyngeal orifice of Eustachian tube. 9. Upper portion of Rosenmüller's groove. 11. Granular tissue at anterior portion of vault of pharynx. 12. Posterior surface of velum.

to hyperæmia. The posterior ends of the inferior or middle turbinated bones are enormously enlarged, forming round tumors which obstruct more or less the posterior nares and project into the pharynx: polyps and deviation of the septum may complicate these cases.

The same appearances are seen in chronic post-nasal catarrh, and in addition, a mammillated and thickened appearance of the pharyngeal mucous membrane and that of the posterior third of the septum. In dry catarrh the mucous membrane is coated with mucus or covered with crusts. In atrophic rhinitis the membrane is thin, pale, hard to the touch, and covered with a layer of dried secretions and crusts. The nasal passages are abnormally wide, and one or all three turbinated bones are atrophied; or one side may be occluded by an enlarged turbinated bone or by deviation of the septum.

Nasal polyps are grayish-yellow or *greenish, shiny* masses, on a broad base, soft and yielding to the probe; they are usually not circumscribed.

Ulceration. Ulceration of the mucous membrane is usually a manifes-

tation of lupus, tuberculosis, or tertiary syphilis. In *lupus* the ulceration has extended from the exterior. If ozæna is present in a patient with lupus, it is probable that there is also lupus of the nasal passages. The ulcers may be followed by necrosis and caries of the bones. If the ozæna is not removable by antiseptic sprays, the bones are probably affected. A discharge of sequestra makes the diagnosis positive. Rhinoscopy and careful palpation may reveal the ulcer and a carious bone. *Tuberculous* ulcers are usually found in the septum. They are rarely primary. They present a whitish-gray surface, with elevations of infiltrated tissue. They bleed on the slightest provocation. The mucous membrane surrounding them is torn. Tubercle bacilli can be found in the scrapings from the ulcer. In *syphilis* the ulcers are situated anywhere in the nares. A history of infection or of secondary and tertiary manifestations can be obtained. The stench of the breath is sickening, and the patient complains of stenosis and loss of smell. There is some localized tenderness, and sleeplessness, debility, and emaciation may ensue. There may be mere superficial excoriations, or deep serpiginous ulcers surrounded by an inflammatory zone. Caries can be detected with a probe. The ulcerated surfaces are covered with a dry, greenish crust. *Foreign bodies* usually cause ulceration if impacted.

Neuroparalytic ulcers are painless and spread rapidly over considerable surface; they follow paralysis of the fifth nerve. They are dry and sluggish and do not extend to the skin. *Post-febrile ulcers* follow measles, scarlatina, typhoid, and variola, and are due to rupture of small abscesses, with the subsequent formation of ulcers. They are usually situated anteriorly on the septum or on the inner side of the alæ, covered with scabs, and very irritable.

Perforation of the septum frequently occurs in the anterior and lower part of the cartilaginous portion, and results not uncommonly from traumatism. A simple abrasion on the mucous surface is irritated by the patient's finger-nail until a depressed ulcer forms, covered by a succession of dry crusts. These crusts are picked away by the patient, and the ulcer, instead of healing, gradually extends through the cartilage and perforates into the other nostril. Such perforations are usually round in shape, with smooth margins, as though cut by a punch. They are frequently mistaken for the ulcerations due to syphilis.

Nasal Secretion. The *odor* of the nasal discharge may be suggestive of *diphtheria* or of the presence of *foreign bodies*. The discharge in the latter instance is sanious or purulent. Animal parasites, as well as peas and beans, cause pain, symptoms of obstruction, and ulceration. In syphilis with caries the odor is usually gangrenous. The physical, chemical, and microscopical characters of the nasal secretion are described in Chapter XXXVIII., Laboratory Diagnosis.

THE LARYNX.

The objective symptoms are determined by *inspection* and *palpation*. Inspection of the *exterior* of the larynx reveals the presence of swelling, and the movements of the organ as a whole. Local swelling of

the tissues over the larynx may occur in inflammations of the cartilages, which are usually of syphilitic origin, but may attend carcinoma or other tumor. More or less marked swelling attends the inflammation of the cartilages; fluctuation after a time is detected; and when the abscess is opened, pus and necrosed cartilage are evacuated. The objective signs of inflammation are noted.

The movement of the larynx is increased in cases of dyspnœa. It is accompanied by recession of the spaces above the sternum and the clavicles, with clonic contraction of the sternocleidomastoid muscle.

The *interior* of the larynx is studied by inspection (laryngoscopy) and by palpation (probe or fingers).

Laryngoscopy. The first requisite is a good light—sunlight, a good student's-lamp, or an Argand or Welsbach gas-burner; the electric light is not satisfactory. Second, a good reflector is required. It may be attached to a head-band or a spectacle-frame. It should be concave for artificial light, plane for sunlight, and pierced in the centre. Third, laryngeal mirrors of different sizes and a curved probe complete the instruments necessary for examination of the larynx.

Examination. The patient is seated with the source of light at one side and behind him; the head and shoulders are brought well forward and the head slightly raised. The operator takes a seat in front at a proper distance for the focal length of the reflector, and focuses the light on the patient's mouth, warms the laryngeal mirror over the flame and tests its temperature on the back of the hand. It should be moderately heated, so that when it is placed in the mouth the vapor of the breath will not be condensed on its surface. The patient must open the mouth and protrude the tongue, which is grasped gently but firmly between the folds of a napkin by the thumb and fingers of the examiner. The mirror is then inserted carefully and quickly, face downward, into the pharynx. Care must be taken not to touch the tongue or palate, otherwise the patient may retch and become alarmed. The mirror is passed to the posterior wall of the pharynx, and so directed that the image of the larynx is reflected to the eye of the operator. The patient is made to phonate "ā" or "ee," not "ah," and then to respire. The various structures and the action of the cords are observed. The appearances of the mucous membrane are studied during quiet respiration.

The epiglottis is very dependent, so that often the larynx can only be seen by having the patient stand while the operator remains seated. The patient's head is bowed on his chest and the examination proceeds.

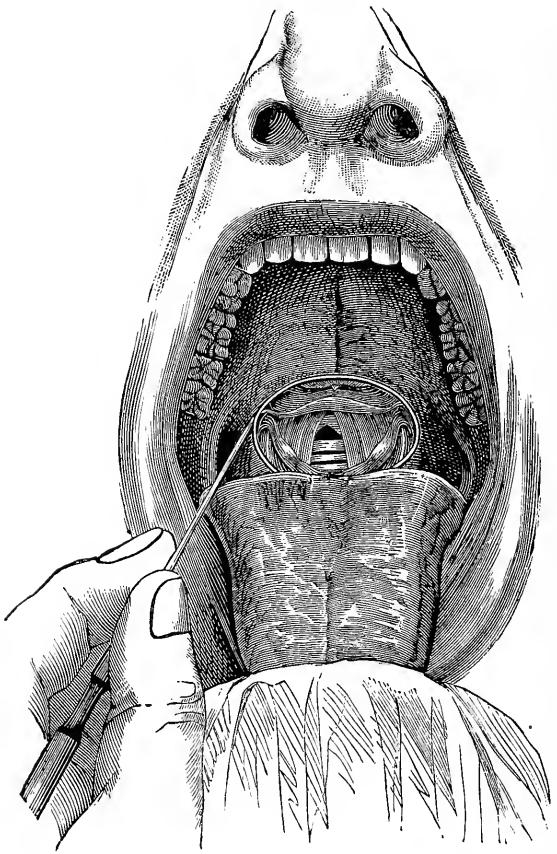
The first examination may not result satisfactorily, but little being observed on account of spasm of the pharyngeal muscles. Repeated sittings may allay apprehension and accustom the mucous membrane to the presence of the mirror. This object may be attained by administering bromides or by applying cocaine to the pharynx.

The probe is needed only to ascertain the consistency of tumors and growths. Cocaine must be applied before it is used.

Appearance of the Larynx in Health.

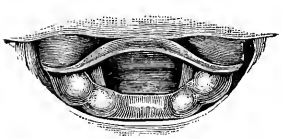
Fig. 45 shows the larynx as it is seen in the laryngoscopic mirror. Above (upper part) is the arched epiglottis, below it the cavity of the

FIG. 45.



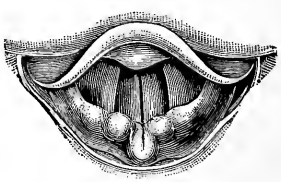
Laryngeal mirror in position, displaying the laryngeal image. (COHEN.)

FIG. 46.



Laryngeal image during respiration.

FIG. 47.



Laryngeal image during phonation.

larynx. In the centre are the vocal cords, white and glistening; on each side of these the pink folds of the false cords. At the bottom of the mirror are the arytenoid bodies, and between them the folds of the

PLATE II–a.

MODIFIED FROM SCHNITZLER'S ATLAS.

Fig. 1. The normal larynx as it appears during inspiration.

Fig. 2. The normal larynx as it appears during phonation.

a. Epiglottis.

b. Left ventricular band.

c. Left vocal cord.

d. Eminence marking the site of the left cartilage of Wrisberg in the ary-epiglottic fold.

e. Eminence marking the site of the left cartilage of Santorini and practically also that of the arytenoid cartilage in the ary-epiglottic fold.

f. Interarytenoid space with a slight amount of the upper portion of the posterior wall of the larynx visible.

g. Trachea with its rings.

h. Right ventricle of the larynx.

Fig. 3. The larynx in a moderate attack of acute laryngitis.

Fig. 4. Early stage of tubercular laryngitis. Localized hyperæmia and thickening on the posterior portions of the vocal cords. Infiltration of the posterior wall of the larynx with tubercle formation. Pale, œdematous swelling of both ary-epiglottic folds obliterating the eminences of Wrisberg. Anæmia of the laryngeal mucous membrane.

Fig. 5. Large, lobulated, tubercular infiltration on the posterior wall of the larynx. Commencing ulceration at the most prominent part of the growth.

Fig. 6. Tertiary syphilitic ulcer of the left ary-epiglottic fold and margins of the vocal cords. The right ary-epiglottic fold is the seat of a gumma that has begun to soften. The under portion of the epiglottis on the left side is superficially ulcerated from contact with the secretions of the ulcer on the ary-epiglottic folds.

Fig. 7. A papilloma attached by a broad base to the left vocal cord. It extends across the glottis and rests upon the right vocal cord. Owing to the weight of the tumor the action of the left crico-arytenoideus posticus is impaired and the left vocal cord is straighter and abducted less than the right one.

Fig. 8. An epithelioma involving both vocal cords. The left is merely infiltrated, while the right is ulcerated slightly. The deep red or purplish color of the passive hyperæmia should be contrasted with the appearance of the mucous membrane in other inflammatory conditions.

PLATE II-a.

Fig. 1.

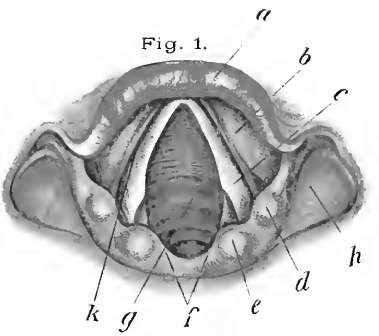


Fig. 2.

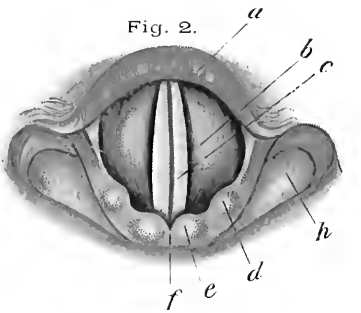


Fig. 3.

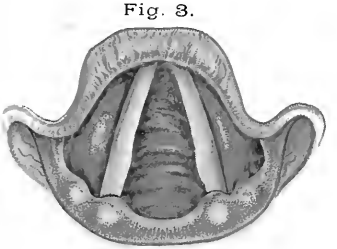


Fig. 4.

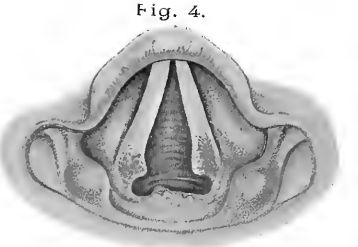


Fig. 5.

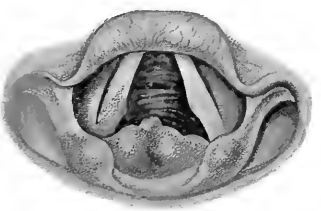


Fig. 6.

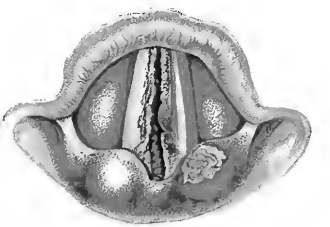


Fig. 7.

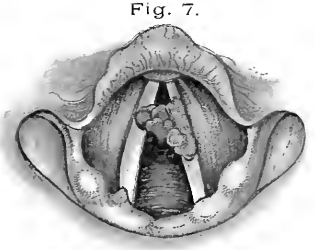


Fig. 8.

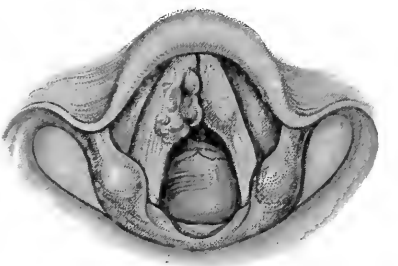


PLATE II-b.

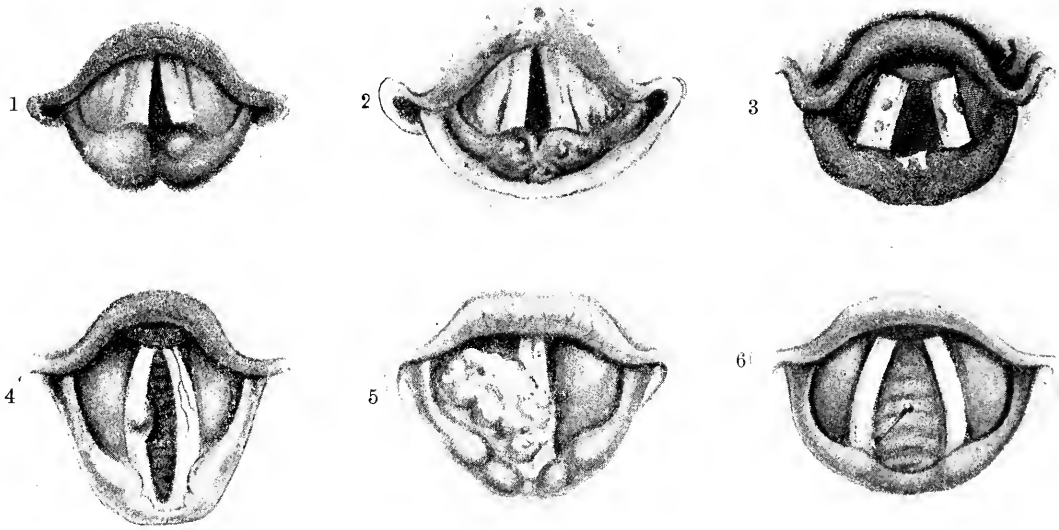


Fig. 1.—Laryngeal Image from a Case of Phthisis, showing the pyriform swelling of the arytenoid cartilages.

Fig. 2.—Tubercular Ulceration of the Epiglottis and Tubercular Nodules on the Ary-epiglottic Folds.

Fig. 3.—Syphilitic Ulceration of the Vocal Cords and of the Interarytenoid Space.

Fig. 4.—Fibro-cellular Tumor on the Right Vocal Cord.

Fig. 5.—A large Papillomatous Tumor springing from the Right Ventricular Band.

Fig. 6.—A pin embedded in the Posterior Portion of the Right Vocal Cord

interarytenoid space. Below and outside the arytenoid bodies are the fossæ. The mucous membrane is pink throughout except on the cords. In respiration the arytenoids separate, carrying the ends of the cords which are attached to them with them, and leaving a triangular opening —the glottis—through which the rings of the trachea can be seen. (See Fig. 46, and Plate II., A, Fig. 1.) In phonation the arytenoids approach each other, obliterating the interarytenoid space; the inner edges of the cords come in contact and close the glottis. (See Fig. 47, and Plate II., A, Fig. 2.)

Appearance in Disease.

A note must be made of the color of the various parts, of the presence or absence of swelling, of ulceration, of new growths, and of alterations of the movements of the parts concerned in phonation, particularly of the cartilages and the cords.

Color. The color is an indication of the degree of congestion. *Anæmia* of the larynx may be merely part of a general anæmia from any cause. In chlorosis it is seen before the external appearance is marked. An intense anæmia of the larynx is an early and valuable symptom of pulmonary tuberculosis.

Hyperæmia may be active or passive. It is readily recognized by the intense redness. Active hyperæmia occurs in acute laryngitis, either of the primary or secondary forms. Passive hyperæmia occurs in general obstruction to the circulation, as emphysema or valvular lesions; pressure on veins by tumors; forced expiration and holding the breath; in paroxysmal cough, especially whooping-cough. Active hyperæmia leads to catarrh, passive to œdema.

Swelling and Infiltration. Swelling of the epiglottis and of the aryteno-epiglottidean folds is seen in œdematous laryngitis, in acute, subacute, and chronic laryngitis. In *œdema* of the glottis the swelling is below the vocal cords. The swelling may be circumscribed and undergo suppuration. Swelling and œdema are also seen in *perichondritis*.

Tuberculosis. Swelling and infiltration succeed the primary anæmia or catarrh of the first stage of laryngeal tuberculosis. At first there are slight intumescences of tubercular infiltration, not well outlined, and gray in color. They are found most frequently in the interarytenoid space, less often on the false cords and arytenoid cartilages, rarely on the epiglottis.

1. A hill-like prominence between the arytenoid cartilages either in the middle or on one side. During phonation it comes up between the cords.

2. When a false cord is affected, the whole of it is usually infiltrated, forming a tumor-like swelling which often hides the vocal cords.

3. Vocal Cords. At first usually only one cord is affected. It is thickened and the free border is red. Sometimes the free edge seems split. The infiltration may extend to the subcordal region and cause a hypoglottic laryngitis.

4. Epiglottis. Infiltration of the epiglottis is rarer than œdema after ulceration, and care must be taken not to confound these conditions. The

whole epiglottis, or only portions of it, may be affected. The structure thickened and curled upon itself, and not freely movable.

5. Arytenoid Cartilages. They appear enlarged and puffy, and often fixed from perichondritis. Thickening of either one or both of the arytenoid cartilages is most characteristic, the general shape being pyriform, with greatest enlargement near the median line, but extending often into the aryteno-epiglottic folds.

Syphilis. In syphilis we have three forms of swelling:

1. Mucous Patches. These are flat elevations of 3 to 7 mm. diameter, oval or circular, and of a whitish-gray color. When the epithelium is lost they appear yellow and purulent. There is no tendency to ulceration, and the patches soon disappear even without treatment. They occur usually from three to nine months after infection.

2. Infiltrations. Usually these are overlooked, as they produce no symptoms. They are diffuse thickenings in various parts of the larynx, most often on the epiglottis, which may be thickened uniformly or only in part around the edge. The vocal cords may be so swollen as to cause dyspnœa. Usually an ulcerated spot is seen in the centre of the infiltration. The mucous membrane is either normal or reddened. Infiltration appears three to four or more years after infection.

3. Gummata. Gummata appear as round prominences of the same color as the surrounding tissue. They occur on either side of the epiglottis, on the aryteno-epiglottic folds, often in the interarytenoid space, on the false cords, and on the under surface of the vocal cords. If they break down, deep ulcers form, leading to extensive destruction of the parts.

Lupus. In lupus isolated or grouped nodes are seen coalescing into patches on the epiglottis. The disease is usually present on the face or in the pharynx and mouth.

Leprosy. In leprosy the epiglottis is swollen, and nodes from the size of a pin-head to that of a pea are seen on the epiglottis, arytenoid bodies, and false cords.

Fissures. Fissures and erosions are present in chronic laryngitis.

Ulcers. Ulceration is seen in tuberculosis, syphilis, carcinoma, leprosy, and lupus.

Tuberculosis. Ulceration occurs in tuberculosis in the following situations:

1. Interarytenoid Space. The mucous membranes are notched with irregular projections. When the ulcer is visible, it is irregular and of a dirty-gray color.

2. False Cords. The ulcers are flat and aphthous, with a pale-white base and a membranous deposit. The mucous membrane sometimes appears sieve-like.

3. Aryteno-epiglottic Ligaments. The ulcers are superficial and run lengthwise of the ligament.

4. Vocal Cords. The ulcers are either on the upper surface or on the edge of the cords. The former are superficial and seldom destructive. Those on the edge are either small and discrete, or linear ulcers affecting the whole border. The circumscribed ulcers occur usually at the poste-

rior portion of the cord and on the processus vocalis. Ulcers of the whole border are often very destructive.

5. Epiglottis. Tuberculous ulcers of the epiglottis occur only on its laryngeal surface, and are either aphthous and superficial, or deep; they arise from the breaking down of previous infiltration. Sometimes tubercles can be seen at the edge of the ulcers, but they are of no diagnostic value, as similar nodes are seen with non-tuberculous ulcers. The epiglottis is usually thickened and œdematous.

Syphilis. Syphilitic ulcers are circular, deep, with a sharp border and inflammatory areola, and overlaid with a whitish-yellow deposit. They develop from an infiltration or a gumma, and not on an unchanged surface. Ulcers on the upper surface of the epiglottis are always syphilitic.

Tumors. Benign Tumors. Papilloma. The most common form of the benign growths is the papilloma. The growth may spring from the true or false cords, the aryteno-epiglottic ligaments, rarely the posterior surface of the epiglottis. The tumor has a broad base. There may be one or several, and the size varies from that of a split pea to that of a walnut. Three varieties are met with: 1. Small warty growths, usually on the cords, dark red in color and seldom larger than a bean. 2. Groups of raised white papillæ on a broad base, also growing on the cords. 3. Large, red, mulberry-shaped or cauliflower-shaped growths, partly villous, partly warty, which fill up the whole larynx.

Fibroma. Fibroma appears as a hemispherical, pedunculated tumor of dirty-white, reddish, or dark-red color, more or less dense in consistency. It is usually single, and grows most frequently from the cords. The smallest examples are known as "singer's nodes"; they may be as large as a hazelnut.

Malignant Tumors. In addition to the symptoms of benign tumor, pain and hemorrhage occur. Both carcinoma and sarcoma are found, but the latter is very rare.

Carcinoma. The most common form is the epithelioma, although the medullary and scirrhous forms have been described. *Epithelioma* is seen as a circumscribed, hemispherical, warty or cauliflower-like formation, varying in size, or as a knotty infiltration projecting into the larynx. The *medullary* form is larger, soft and bloody, and rapidly ulcerates. *Scirrhus* is firm and hard. The structure of the larynx is gradually invaded, and necrosis of the tissues develops. Perichondritis and abscess frequently ensue.

In carcinoma of the *cords* two kinds of growth are seen. In the *polypoid* form the tumor develops on the cord like a warty growth, sometimes papillary and of a reddish-gray color. In *diffused* cancer of the cord the structures are red and knotty, and invade the surrounding tissue without distinct demarcation.

Sarcoma. The tumor has a broad base, is shining in appearance, and sometimes lobulated. Sometimes the structure is dark red or yellow.

Fever. Fever is present in acute laryngitis and tuberculous ulceration. It is high in acute laryngitis with stenosis; in tuberculosis it is of a hectic type.

Sputum. The sputum from the larynx is generally scanty; it is not

frothy, and is colorless and transparent; it is often discharged in small globules; it may be streaked with blood. Sometimes pseudomembranes are coughed up. It is doubtful if purulent sputum ever comes from the larynx, excepting in cases of perichondritis in which the abscess bursts into the larynx. Laryngeal sputum is found in catarrh and malignant tumors. It is blood-streaked when the catarrh is very intense, or after injuries.

The Epiglottis.

The epiglottis when inflamed is swollen and red, and may be palpated with the finger.

CHAPTER XXIX.

EXAMINATION OF THE MOUTH.

Inspection. We observe the color of the mucous membrane of the mouth, as well as the contour. The teeth, gums, and tongue are examined.

Color. The normal redness of the mucous membrane may be increased or diminished in intensity. Pallor is associated with anæmia. Increased redness attends inflammation. The mucous membrane is yellow in jaundice, bluish in cyanosis. Both of the latter changes are observed to greater advantage under the tongue. The mucous membrane is the seat of pigmentation in Addison's disease and in argyria. In the former, small oval purplish spots are seen, and must not be confounded with the pigmented spots common after stomatitis in negroes. Eruptions occur in the mouth, and may precede the cutaneous eruption. This is notably so in measles, in which the eruption is seen on the hard and soft palate twenty-four hours before the development of the skin rash. Vesicles are seen in smallpox and chickenpox.

Shape. Swellings are the result of disease of structures about the mouth. The floor of the mouth is encroached upon by glands underneath or by swelling of the cellular tissue. Bone diseases and some teeth affections cause swellings. The dental arch must be observed. (See Bones and Joints, General Morphology, Chapter XXVII.) Narrowing of the arch is due to adenoid disease or to the habit of thumb-sucking in childhood, much more likely to the former.

Fetor. The odor imparted to the exhaled air is peculiar in mouth-affections. It may be a simple fetor or of a metallic or gangrenous character. Fetor attends all inflammations; it is more pronounced in ulcerative and mercurial stomatitis; in the latter it may be metallic.

Hemorrhage. Petechiæ in purpura hæmorrhagica; submucous hemorrhages in scorbutus and severe forms of purpura—*morbus maculosus Werlhofii*—are common on the cheeks and on the gums. In ulcerative endocarditis hemorrhagic infarcts are seen. In grave anæmias petechiæ are also seen.

Capillary oozing of blood takes place from the mucous membranes in low typhoid states. The accumulated blood collects about the teeth and on the tongue, and in febrile states becomes dry. Dry incrustations are known as *sordes*.

Salivation. Increased flow of saliva occurs in all inflammations unless attended by high fever. It may be voluntarily discharged by the patient or dribble in a continuous stream. (See Saliva.)

The Gums.

The gums and the mucous membrane of the mouth are involved in inflammations and ulcerations, and in certain metallic poisonings. The gums swell and grow spongy in inflammations.

The Gingival Line. A red line at the junction of the gums and the teeth is frequently seen in cases of tuberculosis. It is sometimes present in other cachectic conditions, as carcinoma, and in diabetes.

The Gums in Scurvy. The gums are swollen and spongy in scurvy. They bleed easily, and are usually streaked with blood. Ulcers form along the alveolar margin. There is not much fetor of the breath. In mild cases the inflammation may be limited to the gums of four or five teeth. The gums of decayed teeth are usually the seat of the most marked inflammation. Infants may have scurvy as well as adults—especially if fed exclusively on sterilized milk or malt preparations. (See Scurvy-rhachitis.)

The Gums in Lead-poisoning. *The Blue Line.* In lead-poisoning a blue line is seen at the margin of the gums. Before the line makes its appearance, a row of discrete black dots occupying the seat of the papillæ of the mucous membrane is observed. If examined with a magnifying-glass, the line is readily seen to be an interrupted one. It does not always extend along the entire margin, but may be limited to a few front teeth in either the upper or the lower jaw. In the more advanced cases there is some salivation, and the patient complains of a sweetish metallic taste in the mouth and metallic fetor of the breath.

The Teeth.

Cases of indigestion, chronic gastritis, constipation, and diarrhœa are often due to defective mastication. Persistent aural, nasal, and ophthalmic affections may have their primary origin in disease of the teeth. Caries of the teeth may cause headaches or neuralgias, near or remote (see Headache), and may explain many cases of foul breath.

Pitting of the surface of the teeth and thinning of the enamel in transverse grooves are held by some to be due to mercury. There is no doubt that infantile stomatitis, independent of mercury, is the cause of these changes. Teeth so marked must be distinguished from the so-called Hutchinson teeth. In stomatitis the molars are often honeycombed to an extreme degree, the incisors becoming affected later. The color of such teeth may be darker than normal. A transverse furrow crosses all the teeth at the same level.

The Teeth in Gout. *Erosion* of the teeth takes place in gouty subjects. There are wasting and loss of polish of the labial surface, followed by deep grooves which extend into the body of the teeth. *Pyorrhœa alveolaris* is another expression of gout. There are (1) usually a marginal inflammation of the gums; (2) inflammation and necrosis of the pericementum; (3) loosening of the teeth; and (4) formation of so-called calculi.

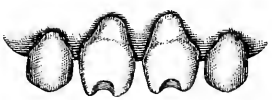
The Teeth of Congenital Syphilis. The upper central incisors of

the permanent set are affected. They are dwarfed, narrowed, and short.

The middle lobe of the tooth is so atrophied as to leave a single broad vertical notch in the edge of the tooth. A narrow furrow sometimes passes upward from the notch on both the anterior and the posterior surface, nearly to the gum.

Teething. During the period of infancy it is well to remember the influence of the eruption of the teeth upon the general constitution. While many prominent authorities believe that the eruption takes place without the occurrence of general or reflex symptoms, equally careful observers, on the other hand, believe that nervous phenomena often attend the process. The latter class of observers attribute the feverishness, insomnia,

FIG. 48.



Notched teeth. Malformation of permanent teeth found in hereditary syphilis. (JONATHAN HUTCHINSON.)

restlessness, loss of appetite, and gastro-intestinal disturbances of infancy to this cause. Convulsions at this period are believed to be due to the pressure of the tooth, which cannot break through the mucous membrane, upon highly sensitive nerves at the root. Even in later life reflex convulsions are held by some to be due to disease of the teeth.

Slowness in the development of the teeth may be due to rhachitis. The student should be familiar with the periods of development, and the number of teeth that appear at each period.

DATES OF ERUPTION OF THE TEETH.

Milk Teeth.

$$\frac{2\,M \quad 1\,C \quad 4\,I \quad 1\,C \quad 2\,M}{2\,M \quad 1\,C \quad 4\,I \quad 1\,C \quad 2\,M} = 20$$

Eruption of	central incisors	about	7th month.[1]
"	lateral incisors	"	9th "
"	first molars	"	15th "
"	canines	"	18th "
"	second molars	"	24th "

Permanent Teeth.

$$\frac{3\,M \quad 2\,B \quad 1\,C \quad 4\,I \quad 1\,C \quad 2\,B \quad 3\,M}{3\,M \quad 2\,B \quad 1\,C \quad 4\,I \quad 1\,C \quad 2\,B \quad 3\,M} = 32$$

Eruption of	anterior molars	about	7th year.
"	central incisors	"	8th "
"	lateral incisors	"	9th "
"	anterior bicuspids	"	10th "
"	posterior "	"	11th "
"	canines	"	11th "
"	second molars	"	12th to 14th year.
"	third molars (wisdom teeth)	about	18th to 25th "

[1] Lower incisors first.

The Tongue.

The surface of the tongue is the only mucous membrane of the body, except the oral and faucial, that is open to inspection. Its examination is supposed to enable us to judge of the effects of general diseases upon the system. Its appearance is thought to be indicative also of disorders of the gastro-intestinal tract because of its relations with it. Recent studies by Hutchinson, Butlin, and other observers have resulted in the promulgation of different views. They concluded that the changes in the coating were not due to changes in the epithelium, but depended largely upon parasitic invasion. The growth of micro-organisms is favored by local conditions induced by systemic causes. Since the writings of Hutchinson and Butlin, Dickinson returned to the investigation on the lines laid down by older observers, and has, in a measure, restored the tongue to its original position as a diagnostic factor in an estimation of the state of the general system and in diseases of the gastro-intestinal tract.

We study the tongue to ascertain its *color;* the character of *eruptions* if any are present; the occurrence of *indentations, excoriations, furrows,* or *fissures;* of *ulcers* and of *patches. Plaques, nodes,* and *nodules* are also seen on the tongue.

Morbid processes and *inflammation* of the tongue occur, and it is the seat of *atrophy* and *hypertrophy,* and of the various *tumors* in the parasitic diseases. Surgical affections of the tongue will not be considered; local affections will be referred to only in connection with general diseases.

Movements of the Tongue. When the patient is asked to put out his tongue, it is done in health without other movement than that required for its protrusion. Interference with its mobility occurs in disease, when protrusion is attended by abnormal movement. It may be tremulous, as in alcoholism or in simple weakness alone. It may be slow or impeded in the various stages of paralyses. It is tremulous and the seat of fibrillar contractions in general paralysis. It cannot be protruded at all in glossolabial paralysis; it is protruded with difficulty and perhaps only with the aid of the finger in general paralysis and diphtheritic paralysis, progressive muscular atrophy, and hemiplegia, because the paralysis is only partial. The tongue deviates to the paralyzed side of the body in hemiplegia with facial involvement.

Color. Yellowish-white oblong patches, soft, but slightly raised, are sometimes seen along the sides of the tongue, upon the eyelids, and upon the palms of the hands, rarely in other portions of the body. The patches, to which the term xanthelasma is applied, are sharply defined, and vary in size from that of a split pea to that of a three-cent piece. They occur in jaundice and in persons who are said to be subject to bilious attacks.

Pigmentations. Dark-purple, bluish-black or black marks are seen on the tongue as well as on the surface of the lips; the latter are sometimes the seat of brown pigmentation. The macules are sharply defined, neither raised nor depressed, and vary in size; they are seen after glossitis.

Other pigmented areas are also found in Addison's disease. *Blood-stains* are observed in purpura.

Bright-red spots the size of a split pea or larger, known as *ecchymoses*, are of frequent occurrence; they do not disappear on pressure. Hemorrhagic *infarcts* are sometimes seen on the tip of the tongue.

Black Tongue. This rare condition is of parasitic origin. It has recently been described anew by Cohen. It is also known as *nigrities*.

The affected portion is of a brownish-black or black color, varying in size and usually situated in the middle of the dorsum of the tongue. It looks like an iron-stain, and in some instances the surface is roughened. Beginning usually as a small spot it extends slowly, so that at the end of a month the dorsum is covered. The centre is blacker than the circumference. After the entire dorsum is covered, the spot begins to disappear from the circumference toward the centre, and desquamation ensues. Desquamation may last from a few days to two months. This series of phenomena is repeated and the entire affection subsides slowly. The papillæ of the affected surface are enlarged and look like "a field of corn laid by the wind and rain." The sensations of taste and touch are not altered, but a feeling of dryness is marked. It must be remembered that a black tongue is sometimes the result of deliberate deception.

Eruptions. Eruptions of variola, measles, erysipelas, herpes, and aphthous ulcers are seen on the surface of the tongue.

Excoriations. Excoriations on the surface of the tongue, or rawness, arise from injury and may also be seen in dyspepsia.

Patches and Plaques. First, there is the *smoker's patch*, on the middle of the dorsum about the point where the pipe-stem rests, or where the stream of smoke from the pipe or cigar strikes the tongue. This is a slightly raised area of oval shape; it is not ulcerated, but is smooth and red, or livid. Sometimes it is bluish-white or pearly in appearance. The smoothness is characteristic. White and bluish-white patches or plaques, also known as opaline plaques, are seen in *leucoma*, *leucoplakia*, *ichthyosis*, and *keratosis*. These patches are unknown under twenty years of age, do not commence after sixty, and are very rare in women. They are not attended by subjective symptoms usually. There may be a sense of induration and dryness. The course is always chronic.

Wandering Rash. *Ringworm*, or circular exfoliations—the geographical tongue—occurs most frequently in children. One or more smooth red patches are observed on the dorsum of the tongue, neither depressed nor elevated. The filiform papillæ are shed. The patch spreads and assumes a circular or oval outline; the border is faintly or decidedly yellow, usually slightly raised, and sharply defined. The circles may widen and contract from time to time. No subjective symptoms are noted except itching in a few cases. The cause is not known. The diagnosis is easy. The disease may continue for months or years.

Mucous patches are multiple lesions of syphilis in the mucous membrane. They have been referred to in the section on Diseases of the Mouth.

Ulcers of the Tongue. These may be simple, aphthous, traumatic, or infectious.

Simple ulcers follow long-standing superficial glossitis. They form in

the centre of the tongue or of the inflammatory area and are due to sloughing or simple melting away of epithelium. The ulcer is smooth, red, and glazed on the surface; the edges are callous and inactive, and the shape irregular. The ulcer is sensitive and may be painful, while the signs of chronic glossitis continue.

Dyspeptic or **catarrhal ulcers** occur on the tip or on the dorsum near the tip. The dorsum of the tongue, from the tip backward, is very red, and the filiform papillæ are destroyed. The ulcers are small and superficial without definite shape or character, except that they are red and irritable.

Aphthous ulcers are seen in children and adults, and when multiple are attended with fever and other symptoms observed with aphthous ulcers of the mouth. The odor of the breath is characteristic. Single ulcers are usually due to indigestion, or, in women, to the menstrual flow. The tendency to their formation is inherited.

Traumatic ulcers from the irritation of sharp teeth may persist a long time if the general health is bad. When indolent they may be mistaken for syphilitic, tuberculous, or cancerous ulcers. The rapidity of formation, the location opposite a rough tooth, and the absence of other signs of syphilis point to the correct diagnosis. *Chancre* is recognized by the induration, the circumscribed character of the lesion, its seat near the tip, and its association with enlargement of the lymphatic glands, which do not become enlarged from traumatic ulcer unless it is acute and angry. The latter is distinguished from *tuberculous* ulcers by the absence of tubercular signs in other organs and by the absence of tubercle bacilli in scrapings from the ulcer. Cancer is distinguished by the age, the characteristic appearance, and the secondary involvement of the glands.

Tuberculous Ulcer. The tuberculous ulcer presents an uneven, pale, flabby surface, covered with yellowish-gray, viscid or coagulated mucus. The edges are sometimes sharp-cut, sometimes bevelled, seldom elevated. They are not usually very red. There is little surrounding inflammation, and the adjacent portions of the tongue are but slightly swollen. The borders of the ulcer may be sinuous, and the shape oval or ovoid, or linear. In the neighborhood of an ulcer a number of tiny yellowish-gray points may be observed. The ulcer is painful, and attended by salivation. I saw in the Philadelphia Hospital a case of tuberculous ulcer of the tongue in a man twenty-five years of age with pulmonary and intestinal tuberculosis. The dorsum of the tongue was covered with a dozen ulcers, with sharp-cut edges and pale, flabby granulations, without induration or inflammation around them. They were yellowish-gray, and tubercle bacilli were found in the scrapings.

Tuberculous ulceration must always be carefully distinguished from syphilitic and cancerous ulceration. The associate symptoms offer the most trustworthy diagnostic points. Ulcers due to *lupus* are also seen upon the tongue.

Nodes. Nodules in the tongue are always tuberculous or syphilitic.

Cysts. Various cysts occur in the tongue. Mucous cysts and blood-cysts are the most common. *Cysticercus cellulosæ* and the *echinococcus* occur rarely. *Ranula* is a cyst underneath the tongue that causes suffering from mechanical obstruction. It is easy of recognition.

Parasitic Disease. Thrush is the most common. Other infections of the mouth usually extend to the tongue.

The Tongue in General and Remote Disease.

Coating. The following characteristics are observed: (1) the color; (2) the fur; (3) the degree of moisture; (4) the movements.

The student should bear in mind that changes in the condition of the tongue are frequently of local origin; that dryness, for instance, may be due to the habit of keeping the mouth open; and that a coating may be unusually marked because the tongue is not used during mastication. Often coating is seen on one side of the tongue. This has been referred to as due to disease of the nerves of one side. It is just as likely to be due to an absence of mastication on that side of the mouth, the bolus of food being kept on the other side because of pain, diseased teeth, or some other local cause.

Clinical experience has shown that certain conditions in the tongue are associated with certain general conditions which render the appearance somewhat diagnostic. The term diagnostic must be qualified, because the changes are so often local or are modified by conditions independent of the general system. For convenience, the classification of Dickinson as to the appearance of the tongue in disease may be utilized. In the Lumleian Lectures this eminent authority gives a description of the average healthy tongue which is based on extensive observations. Departures from the normal were noted and afterward arranged in the following eleven classes:

1. **The Stippled or Dotted Tongue.** The tongue is moist and dotted with little white points, due to an excess of white epithelium on the papillæ. It is usually seen in persons in poor health without fever. It is not, therefore, a febrile tongue, nor one indicative of grave constitutional disease. It is seen in cases of chronic disease, usually one in which there are no grave symptoms.

2. **The Dry Stippled Tongue.** This is found in mildly acute diseases, or in cases in which the constitutional disturbance is more marked.

3. **The Stippled and Coated Tongue.** Patients in whom this is found are very frequently the subjects of acute and constitutional affections. Fever is frequently present with this variety of fur.

4. **The Coated Tongue.** There is excess of white epithelium on the papillæ, and the coating is continuous. The intervals between the papillæ are more commonly filled with epithelium and accidental matters than in the preceding types. It is seen in acute and febrile diseases, and whether moist or dry in pneumonia, pleurisy, and typhoid fever. It is associated with a far greater degree of prostration and pyrexia, while the saliva is absent in the larger proportion of cases.

5. **The Strawberry Tongue.** The tongue is coated and injected; the fungiform papillæ shine through the coat, particularly at the tip and edges. It is the tongue of scarlet fever, but may be seen in any acute febrile disorder. In scarlet fever, however, it appears by the second or third day—most marked after the second. Pyrexia is more common in this class than in the preceding.

6. **The Plaster Tongue.** A thick, uniform coat, edges abrupt and striking, covers the tongue. The papillæ are elongated, and the intervals crowded with accumulations, among which are bacteria; it is the tongue of acute febrile disease. Fever was marked in a number of cases studied by Dickinson, and prostration was a common attendant. Saliva was deficient.

It is thus seen that, beginning with the healthy tongue, Dickinson described a series of groups, in each succeeding one of which the coating becomes more marked, with or without moisture. The clinical association that he found is a common experience. Each successive group was attended by more fever, greater exhaustion, and less saliva than the preceding group, and in each the tongue became more and more furred.

7. **The Furred or Shaggy Tongue.** When the tongue is moist, the papillæ are greatly elongated, and composed mostly of horny epithelium. The appearance is the same as if the tongue were dry. The moist, furred tongue is not so common as the other. It is seen most commonly in old age and in constipation. The dry, furred, or shaggy tongue may succeed the dotted tongue or the coated tongue in the course of advancing disease. It is the result of disease and lack of moisture, the saliva being deficient; it indicates that there has been fever, and that possibly but little food was taken.

8. **The Incrusted, Dry Brown Tongue.** Over the surface of the tongue there is a dry, thick, felted coat, which is continuous and dips down between the papillæ. The coat is largely made up of parasitic material. In the course of fevers it is the outcome of a preceding condition, the coated tongue, and is indicative of the typhoid state. It occurs in the infectious fevers with high temperature, but may be seen in conditions of low temperature, as cancer, phthisis, albuminuria, and chronic nervous diseases. There is much depression or prostration associated with it, and there is absence of saliva. If the patients with a dry brown tongue recover, the latter retrogresses to the furred or incrusted tongue, which in turn becomes bare gradually, at first in small layers; the bare tongue is thin, usually dry, but is more moist than the dry brown tongue.

9. **The red dry tongue** indicates a more serious condition usually than the dry and brown. It is the tongue of chronic wasting diseases. It occurs in the later stages of phthisis, and, like the raw-beef tongue, is associated with dysentery and liver abscess. There may be fever. It is in a measure the tongue of chronic diarrhœa. The tongue is shrunken, red, polished, and smooth. The papillæ have disappeared and the epithelium is stripped off in patches. It may be associated with aphthæ. If the patient is to improve, the redness fades, the papillæ become softer, and the moisture returns.

10. **Red and membranous**; otherwise like (9)—the red, denuded tongue.

11. **Cyanosis, or Venous Congestion of the Tongue.** The tongue is of a bluish or purplish color, the surface is smooth and wet, and the papillæ are almost indistinguishable. It is not confined to organic heart disease or cyanosis. It is of quite frequent occurrence in albuminuria. With the venous congestion in the albuminuric cases there is always a superabundance of deep epithelium. When the surface is examined, it looks as if the papillæ were fused together and overlaid by a moderate coat.

CLASSIFICATION OF TONGUES.

To the naked eye.	*Microscopically.*
1. Healthy, moist.	White epithelium in small amount on papillæ, not continuous or superabundant.
2. Stippled, moist, dotted with white.	Excess of white epithelium on papillæ, not extending between them.
2 (D).[1] Stippled, dry.	Ditto.
3. Stippled + coated; moist. Coat continuous in parts.	White epithelium on papillæ in excess, with partial filling of intervals.
4. Coated white; moist. Coat continuous.	Excess of white epithelium in papillæ. Intervals more or less filled with epithelium and accidental matter.
4 (D). Coated white, dry. Coat continuous.	Ditto.
5. Strawberry, coated+injected, especially showing in fungiform papillæ.	Like the coated or plastered, but with more injection.
6. White, plastered, thick, uniform coat; edges abrupt and striking.	More elongation of papillæ than with coated tongue, more filling of intervals with superficial accumulation.
7. Furred or shaggy, moist. Greatly elongated papillæ.	Extravagantly long papillæ, mostly of horny epithelium.
7 (D). Furred or shaggy, dry.	Ditto.
8. Incrusted, dry, brown; thick, felted, dry coat over papillæ.	Continuous crust on and between papillæ, largely of parasitic matters.
9. Furred or incrusted, becoming bare. Generally dry.	Crust breaking away, together with more or less of normal surface.
10. Red, denuded. Absence of normal covering.	General absence of all epithelium excepting the Malpighian layer; sometimes of that also.
11. Red, smooth, dry, membranous covering.	Level membrane replacing epithelial processes.
12. Cyanosed.	Injected; hypernucleated; excess of deep epithelium.

Moisture of the Tongue. The moisture is due to the saliva, any deficiency of which causes dryness of the tongue. It is natural, therefore, to conclude that any changes in the moisture of the tongue are due to altered secretion of the salivary glands. This is almost always deficient when fever is present, and hence the tongue is dry. At the same time, it must be remembered that this failure of secretion of the salivary glands does not depend upon gastro-intestinal disturbance.

Dryness of the tongue, it must not be forgotten, may be due to increase of evaporation from keeping the mouth open, as well as to diminu-

[1] The letter D is used to imply dryness. Thus to Class 2 a certain description is attached. Class 2 D presents the same characteristics with the addition of dryness.

tion of the salivary secretion. Again, in chronic fever, dryness of the tongue is a constant characteristic. Dryness is due to the effects of the temperature upon the secretions in general, but it is not the effect of high temperature, curiously, but rather a temperature which has persisted for a considerable length of time. Thus, in pneumonia, with a temperature of 105°, the tongue may be moist; whereas, in typhoid fever, with a temperature of 103°, the tongue is dry. General dehydration of the body causes dryness of the tongue, even without local diminution of secretion. This dehydration is seen in diarrhœa, in which disease dryness of the tongue is the common symptom. It is curious to observe that in cholera the tongue remains moist even until death; whereas, if the patient is about to improve and the discharge ceases, reaction and fever setting in, the tongue begins to dry and becomes quite brown. Local causes may explain this. The watery vomit may keep the tongue moist, and the low temperature of the body may contribute to the change. Excessive discharge of urine is a frequent cause; hence, in diabetes extreme dryness of the tongue is seen. The osmotic action of the sugar in the blood is the cause in diabetes mellitus, just as it is in cases of dehydration of the lens in cataract. Finally, prostration is a cause of dryness of the tongue. Asthenia in all forms continuing over a moderate period of time, as a week or ten days, causes lingual dryness.

The Effects of Food. These must be studied before deciding upon the clinical significance of changes in the tongue. The taking of food at once influences the coating and the degree of moisture. The act of eating cleanses the tongue. In disease, therefore, in which this act is not performed, it is natural that we observe more fur on the surface, and in conditions in which diet is limited to fluids the effect is marked. When the patient is restricted to a liquid diet, the tongue is likely to remain furred.

The Tongue in Relation to Diseases of the Alimentary Canal.

Dickinson does not believe any relationship exists between states of the tongue and dyspepsia, or ulcer of the stomach, apart from that which might occur from loss of appetite or restriction in the amount of food. Nevertheless, we associate the clean, red tongue, with or without excoriations and irritated papillæ, to gastric hyperacidity; a furred tongue, to anacidity with myasthenia. With regard to the bowels, some forms of constipation are often connected with changes in the tongue, but such connection is not constant. Dickinson thinks it is a coincidence, and can not even point to the diagnostic significance of the tongue in obstruction. The state of the tongue in the latter condition is dependent not upon the intestinal lesion, but upon the constitutional disturbance. A dry tongue is well known to occur in acute obstruction, and is due to deficiency of salivary secretion. In chronic obstruction, unless there is constitutional disturbance, the tongue will not change. In diarrhœa all conditions of dryness, furring, and incrustation are observed. The absence of saliva, dehydration, and pyrexia help the desiccation. In diarrhœa and dysentery, therefore, the change in the appearance of the tongue is more marked than in any other disease.

The Tongue in Relation to Individual Diseases.

As regards the relation of the tongue to other individual diseases, but little can be said. Of more direct association, we have the cyanotic tongue in heart disease; the dry tongue in chronic albuminuria and diabetes mellitus; the strawberry tongue of scarlet fever; and the dry tongue of typhoid fever. Of course, the so-called typhoid tongue represents but one stage of typhoid fever. Throughout the disease the tongue may present all varieties in direct succession, the stippled, the coated, the plastered, the furred, and the incrusted. In lobar pneumonia the same changes occur as the disease advances. In bronchitis the lower degrees of coating are presented, while in rheumatism the variety is considerable. In conclusion, it may be stated that the tongue seldom points to solitary organs or isolated disorders, but is a gauge of the effects of disease upon the system.

The Tongue in Prognosis and Treatment.

Clinical observers agree with Dickinson that the condition of the tongue is due very largely to four states—dehydration, exhaustion, pyrexia, and local conditions about the mouth. The degree of fever, the state of the nervous system, the maintenance or abeyance of secretions, and the failure of vitality are indicated by the condition of the tongue. The return of moisture, the disappearance of fur, and the subsidence of tremor indicate that these conditions are subsiding. The persistence and increase of these signs show that the patient is worse.

As to indications for treatment, the dryness, the furring, and incrustation are connected with lack of saliva. The processes by which this lack is brought about differ. They have previously been referred to. One can infer the saliva is lacking, as gastric secretions are also in abeyance. It is probable that this is the case except in the rare xerostomia, and possibly in diabetes. Such lessened secretion is followed by loss of appetite and impairment of digestion. The indication is to administer at once material that is digested with the least difficulty. Hence, liquid food and stimulants are to be used. The dry and brown tongue is of serious prognostic omen in all conditions. While it may be due to want of saliva alone, it also occurs as a part of the failure of nutrition in hectic fever, suppuration, and other conditions. It is an indication for the use of tonics, stimulants, and liquid and highly nutritious food. The weak pulse does not more surely tell of an asthenic tendency than the red, dry, and polished tongue.

CHAPTER XXX.

EXAMINATION OF THE FAUCES AND PHARYNX, AND THE ŒSOPHAGUS.

THE FAUCES AND PHARYNX.

Method of Examination.

THE unaided eye is sufficient, the throat being illuminated as in the examination of the larynx. The difficulties arise from the tongue and the uvula. The mouth should be opened as wide as is consistent with comfort. The tongue is pressed out of the way by the use of a tongue-depressor. In many cases, however, even with the tongue-depressor, the tongue muscles will contract and the organ bunch up in the mouth. Moderate, quiet, full breathing, the mouth being gently opened as the deeper inspirations are made, causes the tongue to relax and lie on the floor of the mouth and at the same time elevates the uvula. At the time of a full breath the part may be inspected throughout. Sometimes the fauces can be examined if the tongue is protruded and held with a soft napkin between the finger and thumb by the patient. In the fauces the tonsils and uvula are to be observed, following out the routine method of ascertaining all the facts. Attention is then paid to the posterior wall of the pharynx, with the same object in view.

Inspection. In examining the fauces and pharynx observation is made of the *color* of the parts, the appearance of the mucous membrane and its glands, the *appearance* and *position* of the uvula, the size of the tonsils, the character of the secretions on the pharynx, and the presence or absence of swellings and abnormal exudations.

Odor of the Breath. In follicular tonsillitis the breath has a peculiar intense and fetid odor. This is more marked in the milder forms of inflammation, with retention of the secretion of the glands. There is also fetor of the breath in cancer and syphilis. This symptom may be of diagnostic significance in distinguishing cancer from tuberculosis.

Color of the Mucous Membrane. The color of the mucous membrane is generally dark red. In the acute forms of pharyngitis it is bright red. In cases of heart disease, when there is cyanosis, the veins are congested and the surface dusky. In obstruction of the superior vena cava by tumor there is a cyanotic hue of the surface of the pharynx.

Appearance of the Surface. The capillary vessels may pulsate in aortic regurgitation. Pulsation of the internal carotid may be seen if an aneurism or aortic regurgitation is present. Bleeding points may be seen over the surface of the pharynx, the discharges of blood from which may simulate pulmonary hemorrhage. The blood may be swallowed

and then vomited, and hence gastric hemorrhage is simulated. When the hemorrhage occurs at night, it is seen on the pillow as yellowish stains. It is often due to adenoid vegetations in the nasopharynx. In chronic pharyngitis the membrane is *dry*, the glands are prominent, and the secretion viscid.

On examination of the posterior wall of the healthy pharynx little elevations due to glands are seen upon its surface, and moderate-sized vessels are seen coursing through the mucous membrane.

Projection forward of the posterior wall may be due to retropharyngeal abscess or caries of the vertebræ.

Eruptions. Eruptions may be observed in the pharynx in some of the specific fevers. Thus, in measles, the appearance of the rash on the pharynx and on the soft palate may be observed before the development of the rash on the skin. The eruption of scarlatina is also seen in the pharynx, and the papules and pustules of variola are frequently observed in that situation.

Ulceration. Perforation of the soft palate is usually the result of syphilis.

Follicular Ulceration. Small superficial ulcers corresponding to the follicles may be seen over the posterior wall of the pharynx. They occur in chronic catarrh, and are due to inflammation of the follicles. In addition, ulcers secondary to infectious processes are sometimes seen, as in *typhoid fever*. In the secondary stage of *syphilis*, small, shallow, and painless ulcers are seen on the posterior wall of the pharynx and on the palate. Mucous patches are observed at the same time, not only on the pharynx, but also in the mouth. In the tertiary stage deep ulcers, followed by scars, are seen on the posterior wall of the pharynx. Although the absence of pain renders it probable that these ulcerations are of syphilitic origin, nevertheless the history of infection and of the primary lesion, and the evidence of the disease in other structures, ought to be secured before a diagnosis is fully established. In the tertiary forms it may be necessary to resort to the therapeutic test. (See the Infections—Syphilis.)

Tuberculous ulcers are irregular in shape and have a grayish floor. They are seen in tuberculosis in its later stages and are the source of extreme pain. They are usually associated with ulceration in the larynx, and, in extremely rare cases, with ulceration of the tonsils. The surest method of diagnosticating tuberculous ulceration consists, after the application of cocaine, in scraping off a portion of the ulcerated tissue and examining it microscopically for tubercle bacilli, but it must be remembered that isolated tubercle bacilli may be derived from exudate coming from the lungs.

Cancer of the pharynx is rare, and is usually secondary, the disease having spread from other situations.

Exudations. On the pharynx the exudation may be due to diphtheria, to pseudodiphtheria, or to thrush. The method of distinguishing the various forms will be considered in the articles on the respective affections. In diphtheria the membrane is made up of fibrin arranged in a network, in the meshes of which epithelium, blood- and pus-corpuscles, and micro-organisms are found. When removed, hemorrhagic abrasions

and raw purulent inflammatory areas remain. Two forms of bacilli are found in the membrane—the pseudodiphtheritic bacillus and the true, or Klebs-Löffler bacillus. (See Bacteriology.) The Löffler bacillus is best detected by cultivations. After the membrane has been removed and washed in a 2 per cent. solution of boric acid, a blood-serum culture is made. The pseudodiphtheritic bacillus grows likewise, but its appearances are different.

Anæsthesia. Some of the results of inspection may be confirmed by means of the probe, and alterations in the sensibility of the pharynx may thus be detected. Sensations may be absent in the whole posterior wall of the pharynx. Loss of sensation may occur in hysteria, in bulbar and diphtheritic paralysis. On the other hand, there may be an apparent *hyperæsthesia,* as is sometimes observed in hysteria. In some individuals the pharynx is particularly sensitive to the presence of foreign bodies, such as inflammatory exudates, and may resent their presence by sudden coughing and retching. Inflammations increase the hyperæsthesia of the pharynx.

Spasm of the pharynx is a subjective symptom complained of in some cases of pharyngitis. The degree of spasm or the amount of choking sensation is largely dependent upon the neurotic constitution of the individual. It may be extreme when only a moderate amount of inflammation is present. It is seen in the most aggravated form in hydrophobia.

The Uvula.

In health the uvula hangs midway from the palate. It varies in shape from congenital causes, and may be elongated on account of disease. This takes place particularly in chronic nasal catarrh if there has been hawking or coughing. When elongated, it is pointed and may extend almost to the base of the tongue. The uvula may be swollen and œdematous, the latter condition being usually associated with subcutaneous œdema in acute Bright's disease, but may also occur in debility. In both conditions it may become so enlarged as to interfere with swallowing and breathing. In some cases of pharyngitis this organ is the seat of intense inflammation and great œdema. In addition to the constant cough which it causes there may be dyspnœa and repeated attacks of choking.

Hemorrhagic infarcts may take place in the uvula. In two instances under the writer's care the intense infarction led to sloughing, and in one, the uvula was swallowed. A superficial necrosis resembling diphtheria may take place if it is pressed upon by an enlarged tonsil.

The Cervical Glands.

The lymph circulation passes from the pharynx to the large lymphatic glands in the neck. The glands at the angle of the jaw are increased in size, while those extending along the vessels of the neck may also be enlarged.

The Tonsils.

The tonsils are situated at the sides of the pharynx, between the anterior and posterior folds of the palate. They are small bodies, not larger than a filbert in the adult. Their entire surface can be seen by ordinary inspection. If enlarged, the posterior surface can not be seen, although a better view may be obtained by causing the patient to gag or retch, during which they are brought forward to the light. They are pathologically of much importance. They are made up of glandular structure arranged in follicles and held together by connective tissue. The crypts of the follicles open on the surface, and in disease are visible. As far as known, the diseases of the tonsils have nothing to do with their function. The tissue and gland-follicles are liable to inflammations, which may be bacterial or the result of rheumatism.

Acute Enlargement. The tonsils become enlarged; the swelling takes place rapidly in the acute forms. They may be simply enlarged and the covering membrane intensely red. In other forms of inflammation the surface may be dotted over with white points, due to exudation from the follicles; these may be covered with a white or grayish membrane, which is removed with difficulty, leaving an abraded surface beneath.

Chronic Enlargement. Repeated attacks of inflammation cause chronic enlargement of the tonsils. They are enlarged sometimes to a great degree, filling almost entirely the lumen of the fauces. The surface is irregular and may be scarred. The mouths of the follicles may be dilated. Mouth-breathing is present. The tonsils may be the seat of *sarcoma* and *tuberculosis.*

Ulcers. Tuberculous ulceration is rare. In a patient, a lad of sixteen years, under the writer's care, the large tonsils were of a honeycombed appearance, on account of the grayish, irregular ulceration. Deglutition was absolutely impossible because of pain, and the young man died of starvation.

But while active disease is not common, there is increasing evidence, as previously indicated, that the tonsils more commonly give entrance to the tubercle bacillus, though the frequency of this occurrence is still in doubt. The histological appearance of tuberculosis without active clinical signs is not an infrequent observation. The disease in such cases is usually secondary to tuberculosis of the lungs.

Deep ulcers on both surfaces are usually syphilitic. An irregular spreading ulcer with fetor and sanious discharge is usually cancerous. The lymph-nodes are enlarged.

Exudations on the tonsils are due to inflammation of the follicles, to diphtheria, to the pseudodiphtheritic inflammation which attends scarlatina or which arises secondarily to other infectious debilitating diseases, and to thrush.

Leptothrix of the Tonsils. In healthy persons the plugs which block the tonsillar crypts are found to be made up of cells and segmented fungi. The latter stain bluish-red with iodopotassic iodide solution. Sometimes the micro-organisms extend beyond the follicles, covering the

surface of the tonsils with patches of various sizes. They are thus seen in follicular tonsillitis.

THE ŒSOPHAGUS.

Stiffness of the neck is seen in acute inflammation of the œsophagus, in peri-œsophageal abscess, and in traumatism.

The *expectoration* in diseases of the œsophagus is characteristic. It is usually a glairy mucus, often frothy or viscid. It is not coughed up, but after welling into the pharynx is hawked up. It is abundant in acute and chronic inflammation and in cancer.

Hemorrhage from the Œsophagus. Hemorrhage from the œsophagus occurs from varicosity of the veins at the lower portion of the gullet. It may occur in old people, from senile disease of the liver, kidney, and spleen, or at any age in cirrhosis of the liver. In hemorrhage from the œsophagus the blood is usually bright in color, has not been acted on by an acid, as in hæmatemesis, and is, therefore, alkaline in reaction, and is not discharged by vomiting, although vomiting may occur after the blood is poured out. In a grave case of purpura under the care of the writer, hemorrhage took place from the lower end of the œsophagus. Small bleedings from the œsophagus are usually indicative of cancer, especially if, in addition to the hemorrhage, there are present the symptoms of occlusion. Hemorrhage may also be due to foreign bodies, trauma, and ulceration.

Emaciation is the most characteristic general symptom of œsophageal disease. It is, of course, more striking in cancer, but occurs to a moderate degree in all forms of stricture.

Fetor of the breath attends dilatation of the œsophagus.

Emphysema of the subcutaneous connective tissue should always lead to investigation of the œsophagus. Usually it is found to have been preceded by pronounced symptoms of disease of the œsophagus. In rare cases ulceration of the œsophagus may progress without symptoms, and extend into the air-passages. The passage of air through the fistulous communication causes subcutaneous emphysema. It is of frequent occurrence when foreign bodies lodge in the gullet.

Physical Examination.

Examination of the œsophagus is made by inspection and auscultation, and by means of palpation with or without a bougie.

Inspection can be made with the œsophagoscope, but this instrument is difficult to use, and has not met with general favor.

Auscultation of the œsophagus, while the patient is swallowing fluids, normally reveals two sounds. One occurs directly after the patient swallows, and has no clinical significance. The second is heard normally after an interval of about six seconds, and is due to the contraction of the œsophageal muscle forcing the fluid onward through the cardia. This sound is delayed or entirely absent in stenosis of the cardia or when obstruction along the course of the œsophagus has caused great relaxa-

tion of the walls above. The proper points for auscultation are to the left of the ninth or tenth dorsal vertebra, or to the left of the tip of the ensiform cartilage. These sounds, however, have little diagnostic value.

Palpation. The œsophagus behind the trachea in the neck may be palpated when it is enlarged, as in abscess and in cases of diverticulum of the upper portion of the tube. In the latter case palpation may be quite distinct, the sac being found empty at times and at others full of fluid or semisolid material; and it may be possible, by using pressure, to empty the contents into the œsophagus, and thus to cause the disappearance of the palpable mass.

Percussion. Percussion of the neck in cases of sacculated diverticulum shows a localized dulness, which is often absent when the sac is empty. Percussion along the spine in cases of tumor or of dilatation above a stricture may show a dull area, usually on the left side.

The Röntgen rays have repeatedly been used with success to demonstrate the presence of dilatation above the stricture, and, more especially, sacculated diverticula in the lower portion. When empty, the sac gives a bright area; when filled with bismuth or other metallic solutions, it gives a deep shadow of regular outline.

Sounding. It must not be forgotten that the normal constriction of the œsophagus is situated nearly opposite the fourth dorsal vertebra, 10 inches from the teeth. The bougie is used to determine the cause of difficulty in swallowing. If the cause is due to paralysis or to spasm of the œsophagus, the bougie can usually be passed with ease. If, on the other hand, it is due to organic disease, an obstruction will be found, which is generally in the upper half of the œsophagus. If near the pharynx, the obstruction is due to cicatricial stricture. If the obstruction is encountered 9 inches from the teeth or about the position of the bronchus, it is usually due to cancer. The bougie should not under any circumstances be passed if there are grounds for believing there is an aneurism. Fatal rupture has followed its passage under such circumstances.

Method of Examination.

The patient should be seated with the head thrown back sufficiently far to make the passage from the pharynx to the œsophagus almost continuous. The operator may stand behind or in front of the patient. The bougie, held like a pen, should be passed through the pharynx, guided by the fingers, close to its posterior wall. But little force should be used. It should be passed slowly, for then the gagging will soon be overcome. The bougie should be warmed and oiled before it is introduced. The handle should be flexible and the bulb olive-shaped.

CHAPTER XXXI.

EXAMINATION OF THE NERVOUS SYSTEM.

Disturbances of sensation, of motion, of reflex action, of appearance and of contour, disturbances of the special senses, of the functional activity of the various organs of the body, and alteration of the condition of nutrition are considered in this examination.

Sensation.[1]

New varieties of sensation appear to be discovered every year, and it is often tedious and sometimes impossible to analyze even those which have been already described. Sensations may be described as those which are relatively simple—that is, involving but a single variety of perception—and those that are complex.

Simple Sensations. *Tactile sensation*, or the sense of touch, is usually spoken of as *œsthesia*. It is the ability to know when some external object has come in contact with the skin. Fibres of tactile sensation, in all probability, pass along the posterior columns of the cord through the fillet, and then into the posterior portion of the posterior limb of the internal capsule. Their cortical distribution is at present unknown, although there is some reason to believe that it is either the parietal lobe or the motor area.

No satisfactory instrument for the exact measurement of the touch sense has as yet been devised. It may be tested in a variety of ways. For ordinary clinical work it is sufficient to touch the skin lightly with the tip of the finger, and after some practice, this method is of exceptional value because the investigator becomes familiar with the degree of contact required to excite sensation in the normal skin in the various areas to be tested. A blunt instrument may also be employed, or, if sensation is still acute, a camel's-hair brush or cotton point. If a hard instrument is employed, the investigator should be careful that it is not sharp nor rough, so that the pain sense may be excluded, and also that no force is used in applying it to the skin in order that the pressure sense may not be involved. Von Frey has devised an instrument which consists essentially of a small hair fastened at right angles to the end of a handle. According as the stiffness of the hair varies, the instrument may be employed to detect slight or coarser changes in sensation. He has discovered by means of this instrument that tactile sensation is not diffusely spread over the skin, but is collected in small points which are ordinarily very close together, as many as one to two hundred occurring in a square millimetre

[1] Although, strictly speaking, all sensation is subjective, it is customary to include those forms that may be tested and to a certain extent measured, among the objective symptoms of disease.

in the sensitive parts of the skin. Other instruments are based upon the principle of projecting a rod a certain distance beyond a plane surface. The patient should close his eyes, or, what is better, permit them to be bandaged, and should then be instructed to indicate by some word or gesture the moment contact takes place. From time to time the patient should be asked whether he were touched when contact has not been made, although some movement indicating the approach of the instrument to the skin has been performed. Frequently in prolonged examinations the attention becomes fatigued, and the patient no longer recognizes whether he is touched or not, and answers at random.

Hyperæsthesia is an increased sensitiveness to contact. It may occur in a variety of conditions. Its most common cause is functional exaltation or irritability of the nerves, which may occur in neuralgia or neuritis. It also occurs in organic disease of the cord, and is then limited to the area of distribution of the spinal segment just above the destructive lesion. This is spoken of as the zone of hyperæsthesia. It is also occasionally present in functional conditions, such as neurasthenia and hysteria, and may be merely the result of some local irritation of the skin. The degree of tactile perception varies considerably in different persons.

Hypæsthesia, or decreased sensitiveness, may occur in neuralgia, in partial lesions of the spinal cord, particularly disease of the posterior columns, and rarely in cerebral lesions of various kinds, particularly those occurring in the parietal lobe, in the end of the posterior limb of the internal capsule, and in the pons. It also occurs in functional nervous conditions, and is quite common among the insane, and in cases of arrested or defective development.

Anæsthesia, total loss of the ability to perceive objects touching the skin. It results from solutions of continuity of the sensory nerves, from destructive lesions of the cord, or from brain-lesions. It is also the commonest form of hysterical stigma. Organic anæsthesia may be distinguished from functional anæsthesia by its distribution. If caused by nerve injury, it will exist in the region supplied by that particular nerve. If caused by disease of the spinal cord, the area of anæsthesia will be segmental in type—that is, bounded by two nearly horizontal lines passing around the body. In unilateral lesions of the spinal cord the anæsthesia is limited to the opposite side of the body. In cerebral disease the anæsthesia is commonly unilateral, and corresponds to the paralyzed side, if paralysis be present. If due to a lesion of the cortex, however, it may be limited to one extremity, but even in this case it is usually associated with paralysis. Functional anæsthesia may involve one-half of the body, or symmetrical areas, or areas with irregular or geometrical outlines.

Algesia. Pain-sense, or algesia, is the ability to perceive pain of any kind. It may be produced by various forms of irritants, such as cutting, bruising, caustics, electricity, etc., to each of which the response varies. Various instruments have been devised for testing its intensity. Among the best is that suggested by Kulbin, consisting of a needle which is thrust into the skin for varying distances, the amount of pressure required and the degree of penetration being indicated on a scale. Even this, however, is far from accurate, and for clinical purposes it is sufficient to

use a needle or pinch a small fold of skin between the finger-nails. In case of very pronounced disturbance of the pain sense it is sometimes possible to use the actual cautery or to thrust a needle entirely through a thick fold of the skin. For the purpose of testing the pain sense von Frey has also devised an instrument consisting essentially of a sharpened hair which may be pushed in or out of a hollow handle. The longer the exposed portion of hair the less the resistance, and therefore the greater the delicacy of the test. The advantage of this instrument is that it does not produce any wound or laceration of the tissues, and, therefore, only the nerve-terminals for pain are stimulated. As in tactile sensation, pain sensation is localized to minute points that are closely grouped in the skin. A faradic current is also frequently employed, and to a certain extent is accurate, if data can be obtained by comparing the healthy with the diseased side of the body. As, however, it appears that there is a special form of sensation for the induced current, its results can not be relied upon implicitly. Unlike touch, pain can also be elicited by irritation of the nerve-fibres that convey it to the cord, or perhaps of other nerve-fibres that exist in the tissues and in the nerves themselves. The sensation produced by this form of stimulation is somewhat different from that perceived when the pain-terminals alone are irritated, and is either of a rending or boring character. It can be elicited most readily by pressure upon a nerve-trunk, particularly when it crosses a bone, as, for example, the supra-orbital or the ulnar. The paræsthesias may, in cases of special intensity, be extremely painful. The painful sensations that are experienced in cases of irritation of the serous membranes or in pathological conditions of the muscles or bones are not susceptible to clinical investigation.

Hyperalgesia is increased susceptibility to painful impressions, so that the lightest contact may cause exquisite agony. It occurs in inflammation and in those conditions associated with hyperæsthesia. A variety of hyperalgesia is *tenderness*—that is, pain elicited by simple pressure. It is most frequently associated with local inflammation, and occurs along the course of the nerves in neuritis and neuralgia.

Hypalgesia, or decreased susceptibility to pain, occurs as a result of partial lesion of the nerves or of the central portion of the spinal cord, and occasionally as a result of focal lesions in the brain. It is also very common among idiots, immediately after epileptic attacks, and in cases of hysteria. Hypalgesia may also be acquired as a result of constant exposure to a mild form of irritation, as, for example, in those accustomed to going bare-foot.

Analgesia is an exceedingly important symptom. It results from local destruction of the nerve; from disease of the central gray matter of the spinal cord, such as occurs in transverse myelitis, in syringomyelia, and in tumors of the cord; and from focal disease of the brain, particularly if situated in the parietal lobe, or in the posterior limb of the internal capsule. It also occurs in a great variety of functional conditions, and may be general in certain forms of insanity. It is a very common lesion in hysteria, in which disease the area of distribution may assume the most curious forms, being limited to one-half of the body, or tracing geometrical figures on various parts of the skin. It may also be produced by

hypnotic suggestion. Organic analgesia is frequently associated with trophic changes, either as a result of the inability of the part to defend itself against irritation, or as a result of the intimate association of the sensory and trophic nerve-fibres.

Visceral pain may be elicited by strong pressure upon the testicles, ovaries, or breasts, or by a violent blow upon the abdomen. It is usually characterized by intense prostration and nausea. Visceral analgesia occurs in some cases of taber dorsalis and occasionally in hysteria.

Thermo-æsthesia. The heat sense, or thermo-æsthesia, enables us to recognize the difference in temperature between various bodies. It is usually tested by filling two test-tubes, one with hot and one with cold water, and applying them in irregular alternation to the region under investigation. The difference in temperature between the two tubes is a rough test of the delicacy of the sense. In health a difference of one degree centigrade can be recognized upon the more sensitive portions of the body (the anterior surface of the forearms, the skin of the face, and the chest). A rougher test is the use of metal and wooden objects. The former conduct heat more rapidly from the surface, and therefore give rise to a sensation of cold. In some cases simply blowing upon the skin either with the mouth open or with the lips pursed is sufficient. In the former the patient experiences a warm sensation, in the latter a cold sensation. The heat sense is rather complicated, and is not yet thoroughly understood. There seem to be special points upon the skin where the nerves for heat and cold terminate. (Goldscheider.) There may be loss of perception for cold objects, while perception for hot objects remains unimpaired, or the reverse may be present. Sometimes the patient calls all objects warm, and at other times he calls them cold.

Hyperthermo-æsthesia is practically of no value as a clinical sign, for our methods of testing the delicacy of the sense are at present imperfect, and hypothermo-æsthesia is also difficult to detect, and probably belongs to the category of conditions in which one of the sensations is more or less impaired.

Thermo-anæsthesia, or complete loss of the heat sense, is very important clinically. It occurs in neuritis or destructive lesions of the nerves, and in central disease of the spinal cord, such as transverse or pressure myelitis, tumor, and especially in syringomyelia. As a result of being most frequently associated with spinal cord disease, the thermo-anæsthetic area is usually segmental. The heat sense may, in connection with other forms of sensation, be diminished in functional nervous disease.

The above three forms of simple sensation are those usually regarded as of the greatest clinical importance. They may be equally affected, or one or two may be preserved and the others diminished or lost. The latter condition is known as *dissociation of sensation.* It occurs in neuritis, but is exceedingly rare. It also occurs in various forms of myelitis, particularly pressure myelitis, and is the most characteristic symptom, and for a long time was considered pathognomonic of syringomyelia. In this form of dissociation tactile sense is preserved, and the temperature and pain senses are lost. But it is now known that this type occurs whenever the gray matter of the cord is exclusively involved. In cases

of partial but extensive lesions of the cord it may coexist with complete anæsthesia in neighboring areas, and this constitutes one of the most valuable syndromes for the accurate localization of spinal lesions. When the tactile sense is lost, and the pain sense still present, the condition is termed *anæsthesia dolorosa*. It usually occurs as a result of partial injury to a peripheral nerve.

Tricho-æsthesia. Simple sensations of perhaps less clinical importance than the foregoing are tricho-æsthesia, or the consciousness that a cutaneous hair has been touched. This is really the sensation perceived when tactile sensation is tested with the cotton point; the latter is felt very well upon the forearm, on the back of the hand, and not on the palm, where sensation is distinctly more acute. Von Bechterew calls attention particularly to the fact that tricho-æsthesia and tactile sense are not equally delicate in various parts of the body. The former is most readily tested by touching the individual hairs with a small needle or cotton point. More elaborate apparatus of no particular value has, however, been devised.

The **tickling sense** is not clearly understood. It may be described as an involuntary effort to escape rapidly repeated slight stimuli. It differs greatly in different people and upon different parts of the body in the same person. It is diminished in conditions that diminish tactile sensation, but otherwise has no clinical significance.

The **sense of roughness** is allied to the pain sense; it serves to estimate the degree of friction between the skin and an external body. It plays a part in the stereognostic sense, and appears to be diminished in tabes and peripheral neuritis, but otherwise it is of no clinical value.

Internal Sensation. In addition to the pain of inflammatory processes and spasms, persons have claimed to be able to feel certain other conditions in the interior of the body, particularly moving foreign bodies. This sensation is usually indefinite, and often false.

The Sensation of Locality. When any part of the surface of the body is touched, we can, under normal conditions, tell the location of the point of contact. This varies considerably, however, in various parts of the body, being more accurate on the lips and less on the skin of the back between the shoulder-blades, where an error of from 6 to 7 centimetres is within the normal limits. It may be very much disturbed without loss of the delicacy of the touch sense. It may be tested by making contact with the finger or any blunt object, and directing the patient to close the eyes and to indicate by the finger or by description the point touched. Another method formerly much used by clinicians, and still employed by psychologists, is the use of the *æsthesiometer*, an instrument consisting essentially of two points that can be placed at a measured distance from each other. It has been found that in normal persons these can be detected as two points at the tip of the tongue when separated only 1 mm.; but may still be felt as one on the back when separated as much as 65 mm. This method is extremely inaccurate for the reason that it is difficult to apply the points with the same degree of force. Moreover, experiments have shown that the skin readily becomes educated and able to discriminate points much closer together than is normal for the part that is being tested.

The sensation of locality may be diminished in all forms of hypæsthesia, especially that associated with central disease, and without hypæsthesia in disease of the cord, such as tabes, and in injuries to the parietal lobe. In organic disease of cord the error in localization may be very great, and in some cases amount to a false localization, thus irritation of the hand will be felt in the shoulder, or of the foot in the thigh. When a single contact is perceived in several places, the term *polyæsthesia* is employed.

Allochiria (Obersteiner) is a term employed to describe the reference of a sensory stimulus to the corresponding location on the opposite side of the body. It is a very rare symptom, and its significance is not thoroughly understood. It may be general or local. When the former, it should be regarded as a stigma of hysteria. In the latter case, however, it is usually associated with some organic lesion of the cord, such as tabes, pressure myelitis, etc. A personal observation has led us to believe that it may indicate the restoration to functional activity of certain injured sensory tracts.

The Electrocutaneous Sense. This is really the degree of resistance to the irritation of the induced current. It varies considerably in different individuals, and in the same individual under different conditions and in different parts of the body. It is perhaps most delicate on the skin of the face, and least delicate on the back and the outer surface of the thighs. It is curiously affected in certain nervous diseases; thus in the periodic paralysis of Goldflam it is almost completely abolished during the attack. In *meralgia paræsthetica* it is also, as a rule, greatly diminished in the affected area. In all cases of muscular degeneration the electric current is better borne than when the muscles react. It is also greatly diminished when there is œdema of the skin or much subcutaneous fat. It sometimes persists, however, when tactile anæsthesia is present. In tetany it is greatly exaggerated (Erb's sign), and this constitutes one of the cardinal symptoms of that disease. It is also increased in some of the functional nervous conditions. The electrocutaneous sense is tested best by using a simple faradic battery, employing as the electrode for contact either the wire brush or the naked wire. No satisfactory system of measurement has as yet been devised, but it is of advantage to use invariably the same battery, and to note the position of the inner coil with reference to the outer one.

Pressure Sense. The clinical significance of this has not yet been determined. It is certain, however, that it undergoes considerable variation as the result of various pathological changes. It may be tested roughly by making various degrees of pressure with the finger or a blunt object upon the surface of the skin, the limb being so placed that it is impossible for the patient to make muscular resistance. It may be tested more accurately by using a series of little blocks that can be piled one on top of the other, or by filling a vessel more or less completely with shot or mercury.

Eulenburg has devised an instrument, called the *baræsthesiometer*, that consists essentially of a rod terminating in a flattened extremity, the other end of which is attached to a spring. The amount of pressure exerted is read off on a scale or dial. By means of this instrument very accurate

determinations can be made. In health a difference between two weights equivalent to one-thirtieth of the lesser should be correctly recognized. There are, however, many modifying conditions. The accuracy is greatly increased by placing the weights successively upon the skin with very brief intervals of time between them. If long intervals are allowed to elapse, the patient is less likely to give correct answers. The limb or surface that is being tested should always be supported, otherwise in addition to the pressure there will be added a muscle increment that ordinarily is much more accurate than the former; thus if the skin of the arm is being tested, the arm should be laid flat upon the table, and the patient should be particularly cautioned against raising it or employing the muscles in any way. The *sense of muscular resistance* may also be tested by means of weights or springs. Under these circumstances the limb should not be supported, but the patient should be instructed to hold the different weights in the hand and to estimate as nearly as possible their relative value. The delicacy of this sense is greatly increased by practice, and is practically of no value at all clinically. It seems, however, to be wholly or partly lost in cases of loss of muscle sense, or in cases in which the muscular sense is extremely impaired, as in ataxia, monoplegia, etc. The *sense of space*—that is, the recognition of the size of objects—depends partly upon the sense of localization, but chiefly upon the recognition of the positions of the limbs or fingers. The amount of movement necessary to separate the arms so that the two sides of the object may be touched at the same time is the distance required to move one limb over the surface of the object. As in the preceding, the delicacy of this sense depends largely upon practice, and in some cases is surprisingly accurate. It is also lost in the above-mentioned conditions. The *sexual sense* and the *sense of pressure upon the sphincters*—that is, the desire to micturate or defecate—is diminished or lost in various disorders of the spinal cord.

Functional Modifications of the Various Forms of Sensation.

Delayed Sensation. The perception of the various forms of stimulation that are appreciated in consciousness as sensations may be delayed for some time after the stimulus has been applied. This is spoken of as delayed sensation, and the interval may, in extreme cases, be several seconds. It is not known where this delay takes place, whether in the sensory bodies of the skin, or in the nerves, or in the central nervous system. This symptom is manifested particularly in tabes dorsalis, but may occur in functional nervous disease and in various forms of organic central disease. It has also been noted in peripheral neuritis. The delay can occur for one sensation alone, as the pain sense, even when tactile sense is normal.

Complex Sensations. These are probably very numerous, but only two have been so carefully studied that they are available for clinical purposes. These are the so-called position or muscular sense and the stereognostic sense. By the *position* or *muscular sense* we mean the ability to perceive and recognize the position of the limbs or of the body—that is, whether, for example, the joints are in a state of flexion or extension,

supination, pronation, or rotation; whether the spine is bent or erect, and the position of the head with reference to the trunk. It probably depends upon the co-ordination of the complex perceptions received from the muscles, joints, periosteum, tendons, and skin. It may be tested in a variety of ways. The patient should be instructed to close his eyes or have them bandaged; the finger is carefully grasped on either side and flexed or extended. After each movement the patient indicates its direction. After the fingers have been tested, the same process is employed for the wrist, elbow, and shoulder. Similar methods may be used for the feet, and the head may be rotated to the right or the left, bent forward, laterally, or backward. Another method is to take one arm, bend it into some particular position, and to instruct the patient to imitate the position with the other arm; the same thing being done with the legs; or the patient may be instructed to describe the position in which his arm has been placed, without attempting an imitation. This sense is lost when for any reason there is total anæsthesia of the part, and may disappear as an isolated symptom in case of disease of the posterior columns or in the ataxia due to central lesions. By the *stereognostic sense* we mean the ability to recognize the shape, consistency, surface, and nature of any object placed in the hand or brought in contact with the skin of any part of the body. This sensation is most readily tested by directing the patient to keep the eyes firmly closed; then to select a number of small objects, such as a pencil, match-safe, coin, key, etc., and place them in his hand and direct him to name or describe them. This sense depends upon a variety of perceptions. The size of the object is recognized by a combination of the locality and muscle senses; the nature of its surface by the tactile sense; its consistency chiefly by the pressure sense, perhaps aided by the pain sense; its nature—that is, whether of metal, wood, or any other substance—largely by the temperature sense. The stereognostic sense is always abolished when the tactile sense is absent. Occasionally in hysteria the patient may declare himself unable to perceive touch when the stereognostic sense is intact, but this is an exception. It may, however, be lost when tactile sense is still preserved, especially if the locality sense and the muscle sense have been greatly impaired. When due to organic causes, its absence usually indicates a lesion in the parietal lobe or in the projection-fibres coming from that region. It occurs frequently in hemiplegia, in cerebral monoplegia, and occasionally in peripheral palsy, involving two forms of sensation. It has also been observed as a transient symptom after brain-shock without disturbance of any other sense.

In testing any of the forms of sensation certain general methods should be employed. It is usually best to approach a suspected anæsthetic area from an area where sensation is normal. The boundary marks should be made with a dermograph, a brush dipped in eosin, a pen, or any other suitable utensil, and then this point tested again by passing from the anæsthetic to the normal area. As a rule, the transition is so distinct subjectively that there will be no difficulty in outlining the area by approaching it from various directions and by passing from its centre in the reverse manner. Not infrequently, however, the patients will state

that between the area of anæsthesia or extreme hypæsthesia and the region of normal sensation there is an indefinite zone in which sensation gradually changes from one extreme to the other, and this often leads to great difficulty in accurate delineation. As soon as the examination is complete, the results should be recorded upon a diagram. This is really the only way in which they can subsequently be accurately studied, and often a rough outline sketch is more satisfactory than any amount of verbal description. Sometimes it may be convenient to test all forms of sensation before transferring them, and the outlines of the different types may then be distinguished either by the use of different forms of interrupted or continuous lines, or, what is better, by using different colored pencils. It must be remembered, also, that for some reason not very clear the areas of anæsthesia, even in organic cases, vary from day to day, and the amount of variation is sometimes very considerable. When the results have been obtained, it must first be determined whether their distribution corresponds to the distribution of the cutaneous nerves or to the sensory areas supplied by the segments of the spinal cord. This is usually easy, and is greatly facilitated by reference to Plate III. It must then be determined what nerves or segments are involved, and a careful comparison made between the sensory and motor changes in order to determine either agreement or divergence. If they agree, the diagnosis is, of course, readily made; otherwise it is sometimes difficult to determine exactly what nerves or segments have been affected. (See section upon Localization of Lesions of the Cord.)

Disturbances of Motility.

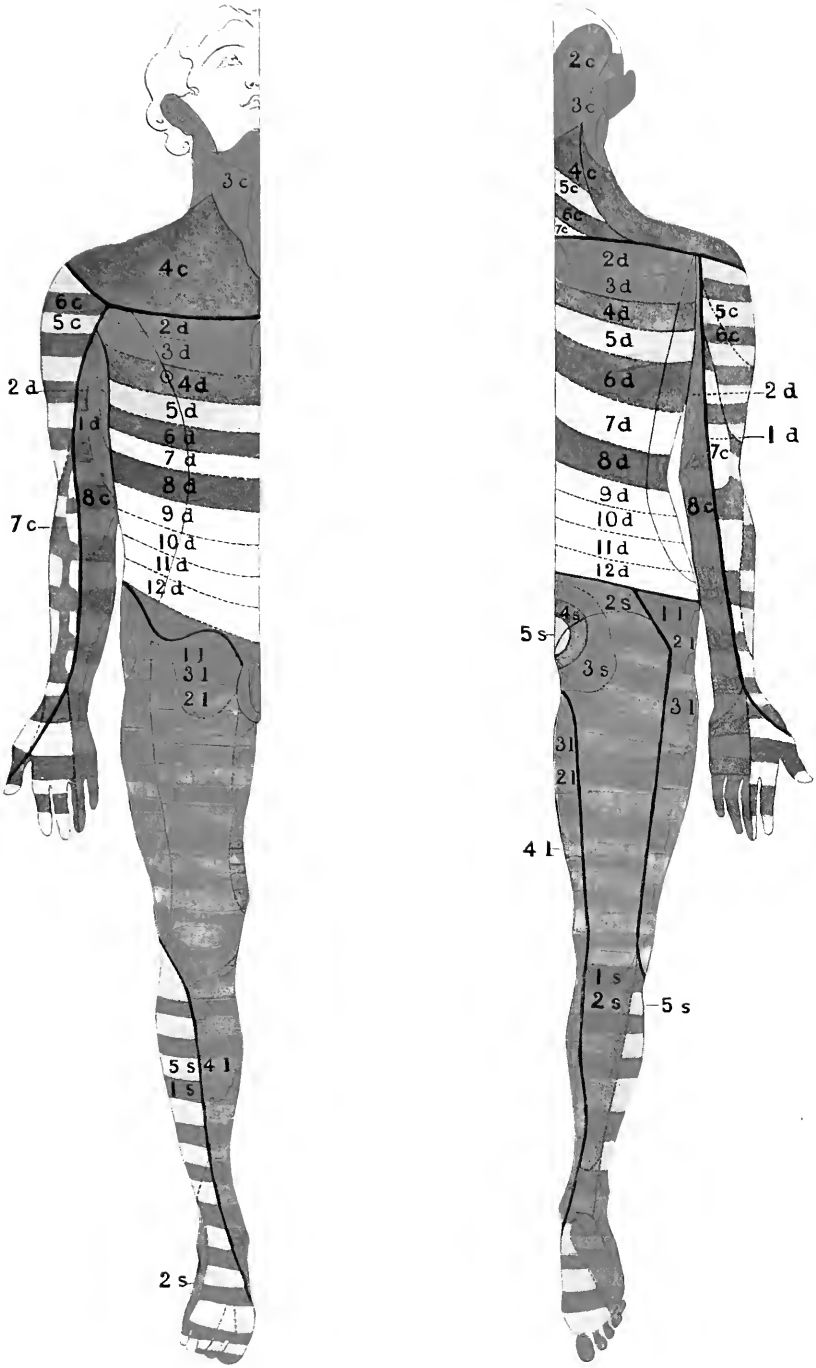
These may be grouped under a number of heads: First, loss of power, which may be either partial, *paresis*, or complete, *paralysis*. Second, impairment of movement, *inco-ordination*, or *ataxia*. Third, closely allied to this, *tremor*. Fourth, excessive muscular movement, *spasm*, or *convulsions*.

Loss of Power.

Paralysis. This is a loss of power in the muscles. It may be true, if the loss is due to disease of the muscle itself or the nervous influence controlling it; or false, if it is due merely to an inhibition of the muscular function produced by a disease of the muscle or joint that causes pain upon movement.

Paralysis is classified, according to the part affected, into *monoplegia*, when one extremity is involved; *hemiplegia*, when half of the body is involved; *paraplegia*, when two symmetrical extremities are involved (this term is usually restricted clinically to paralysis of both legs); *paraplegia cruralis*, if the legs are affected; *paraplegia brachialis*, if the arms are affected; *diplegia*, when two extremities are affected without involvement of the trunk. [Clinically, this is sometimes applied, although incorrectly, to paralysis of both arms (*diplegia brachialis*) or of both sides of the face (*diplegia facialis*).] *Crossed paralysis* is a term applied to paralysis of one side of the face and the opposite side of the body. *Local paralyses*, or *palsies*, is the term used when only small groups of

PLATE III.



These figures have been devised by Wichmann in order to show the distribution of the sensory areas corresponding to the segments of the spinal cord The colors used correspond to those of the normal spectrum, red, orange, yellow, green, blue, indigo and violet, with brown; respectively—the first, second, third, fourth, etc., segments in each portion of the spinal cord—cervical, dorsal, lumbar and sacral. The last four segments in the dorsal region are left white. The advantage of the plate is that it shows very clearly the overlapping of the segments that has been demonstrated by Sherrington. The dark heavy black lines on the arms indicate the division between the two sides of innervation—that from the upper portion of the cervical enlargement and that from the lower portion. The heavy black lines of the legs indicate the divisions between the sacral and lumbar areas of innervation. The figures and letters indicate the segments in which the supply has been drawn and have been introduced for the sake of making the diagram more available for ready reference. C—cervical, D—dorsal, L—lumbar, and S—sacral. The horizontal bands of color without dividing lines between them indicate that both segments innervate the areas involved.

muscles are affected. *Multiple paralyses*, or *palsies*, is employed when several parts of the body are involved at the same time. It may be limited to a muscle or group of muscles.

Another classification is based on the seat of the lesion: *cerebral*, *pontine*, *bulbar*, *spinal*, *neural*, *muscular* or *myopathic paralysis*. According to their nature, paralyses are divided into *organic*, *functional*, and *pseudo-paralysis*.

We also have *spastic paralysis*, in which the muscle-tone is increased and the reflexes are exaggerated, and contractures are either present or likely to ensue; and *flaccid paralysis*, in which the muscle-tone is diminished, the resistance to passive movement lost, and the reflexes abolished.

Paralysis is also classified, according to the situation of the lesion, into *central* and *peripheral* forms. The *central* or spastic type is characterized by absence of the reactions of degeneration, very gradual wasting, and increase in the tendon-reflexes; if anæsthesia is associated, it may affect a different part of the body, and often is partial—that is, it does not involve all forms of sensation. Sometimes there are other symptoms, indicating cerebral involvement, such as aphasia, hemianopsia, convulsions, and vertigo; or spinal involvement, particularly disturbance of the function of the bladder and rectum. The *peripheral* or flaccid type is characterized by rapid wasting of the muscles, the appearance of the reactions of degeneration, and loss of the tendon-reflexes; if there are sensory disturbances, they consist of total anæsthesia, and are found in the part affected by the paralysis.

Spastic paralysis is usually due to some lesion in the central motor neuron—that is, between the motor cortex and the terminations of the fibres of the pyramidal tracts in the anterior cornua of the spinal cord. The lesion, therefore, may be situated in the cortex, the corona radiata, the internal capsule, the pons, the pyramids of the medulla, or the lateral columns of the cord. Spastic paralysis must not be confused with the contractures that ensue after degeneration of the muscles, as in infantile palsy, neuritis, etc. In these cases the limbs are in a state of permanent flexion, and the resistance to extension and to passive movement is not due to increased muscular tone, but to an actual shortening of the muscle and its tendons, which can only be overcome by rupture of one or the other. Flaccid paralysis may be produced by cerebral lesions, but is more commonly due to lesions of the peripheral motor neurons—that is, from the anterior cornua of the cord to the muscle itself. It may, therefore, be produced by destruction of the ganglion-cells, by injury to the anterior roots or to the peripheral nerves, or by disease of the muscle. Flaccid paralysis frequently occurs as the result of functional conditions—for example, it is the type of paralysis that is usually observed in hysteria. As the trophic centres influencing the muscle are either cut off or destroyed, atrophy of the latter usually takes place (*atrophic paralysis*), which is characterized by decrease in bulk, alteration of the electrical reactions, and fibrillary twitchings. Paralysis of a single muscle or of a small group of muscles may be produced by lesions of the peripheral nerves or disease of the muscle itself.

Monoplegia, or paralysis of one limb, may be caused by small lesions in the cerebral cortex or the corona radiata. It is rarely produced by

lesions of the internal capsule, where the fibres are placed closely together, or of the spinal cord, unless the gray matter of the latter is involved. It occurs in circumscribed forms of infantile paralysis, in lesions of the peripheral nerves, particularly the roots or the plexuses, but rarely in disease of the muscles alone, in which case the lesions are more widely distributed. Monoplegia also occurs in hysteria and in the pseudoparalysis due to localized disease of the muscles or joints.

Hemiplegia is commonly due to a lesion of the opposite side of the central convolutions. This lesion may either be extensive and destroy the motor portion of the cortex or corona radiata, or more circumscribed, involving the internal capsule, the crura, the pons, or the medulla. Spinal lesions, also, if unilateral, which is rare, and situated above the fourth cervical segment, may produce paralysis of the same side of the body. (*Vide* Brown-Séquard syndrome.) In hemiplegia due to lesion of the cerebrum, the muscles of the trunk and those supplied by the upper branch of the facial nerve commonly escape. The lower half of the face and the arm and leg of the side opposite the affected hemisphere are paralyzed. If due to lesion of the pons below the decussation of the facial fibres—that is, in the posterior half—the arm and leg of the opposite side and the lower half of the face on the same side are paralyzed (*crossed paralysis, pontine palsy*). Lesions of the medulla, in addition to the motor tracts, involve other important nuclei and tracts.

Spinal hemiplegia is characterized by the absence of facial involvement.

Hysterical hemiplegia can only be recognized in some cases by the discovery of the other stigmata of that disease. It is almost invariably flaccid, and contractures rarely appear, while the form of paralysis in organic hemiplegia is ordinarily spastic, and usually in the course of time pronounced contractures occur.

Paraplegia cruralis is usually produced by a lesion of the spinal cord. If this lesion is situated above the lumbar portion of the cord, the type of paralysis is spastic; if in the lumbar or sacral region, or involving the cauda equina, there are often abolition of the reflexes and flaccidity of some of the muscles. Paraplegia, therefore, occurs in transverse or pressure myelitis, in tumor of the spinal cord, in hemorrhage into the spinal cord, and as a result of traumatism. It is occasionally produced by multiple neuritis of the legs, particularly that form known as Landry's paralysis, or in alcoholic neuritis, by bilateral cerebral lesions, and occasionally as a functional condition.

Paraplegia brachialis is a rare condition, occurring chiefly as the result of a localized meningitis in the cervical enlargement, particularly pachymeningitis hypertrophica cervicalis. As the result of the destruction of the anterior roots there occur atrophy and degeneration of the muscles, and the paralysis is flaccid. It may also occur in syringomyelia, and more rarely as a result of traumatic injury to both sides of the brachial plexus.

Diplegia facialis is almost invariably the result of bilateral facial palsy —which may occur as a result either of neuritis or of an injury to both facial nerves after they leave the medulla. The paralysis is, therefore, flaccid in type, characterized by loss of the normal wrinkles, inability to close the eyes, and drooping of the corner of the mouth.

Local palsies are usually due also to lesions of the peripheral neurons. They are commonly the result of some trauma injuring a single nerve-trunk. The paralysis is, of course, flaccid, and the reactions of degeneration are present. Occasionally, however, a very small lesion in the cortex will produce this condition.

Multiple palsies are usually due to some general condition affecting the peripheral neurons—thus in multiple infantile palsy the anterior cornua of the gray matter of the spinal cord are involved in various situations. The paralysis is usually flaccid and incomplete—that is, certain groups of muscles escape. In polyneuritis due to intoxication or infection there may be paralysis either of certain groups of muscles, particularly the extensors, or of the entire limb. This occurs most frequently in poisoning by lead, arsenic, and alcohol, or in infectious diseases, as beri-beri and diphtheria. The paralysis is nearly always flaccid; there is muscular atrophy, and the reactions of degeneration ultimately appear.

Myopathic Paralysis. Congenital absence or complete atrophy of the muscle gives rise to myopathic paralysis. In either case the diagnosis must usually be made by careful anatomical examination, because in the course of a very short time the patient learns to compensate the defect of the individual muscle by the excessive action of others in its neighborhood. The muscles most frequently congenitally absent are the pectorals, although many others also may be lacking. Total atrophy occurs in various myopathies, but with extreme slowness. In a special type of muscular atrophy (type of Duchenne-Aran), atrophy occurs in individual muscles or in small groups, and compensation is usually acquired for a considerable time until the progress of the disease renders it no longer possible.

Paresis is a term used to indicate partial loss of power in the voluntary muscles. In addition to the causes given for paralysis, it may be produced by exhaustion or by profound emotion; but in these conditions it is transient. There are two forms of paresis—one in which the muscle is unable to exert its normal force at any time, and the other in which the muscle may exert its normal force for a brief period, and then rapidly becomes exhausted and insufficient. In the former there is some deformity, such as foot-drop or wrist-drop. In the latter the symptoms do not appear until some effort has been made. Paresis may also be temporary, as after fatigue; stationary, as in cases of injury to the central nervous system; or progressive, as in the myopathies. In the latter condition the muscles waste and lose their power, but reactions of degeneration do not occur, and there are no fibrillary twitchings. Ultimately, the condition may go on to absolute paralysis. The power of the muscles may be tested very accurately by means of the *dynamometer*. This consists of a steel spring with a staff on one side and a sliding index on the other. The patient compresses the spring in the palm of the hand, and the amount of pressure is indicated in pounds or kilogrammes upon the index. By various mechanical devices the dynamometer may also be employed for the other muscles of the body. Care should be taken when it is used that the patient is not permitted to throw his weight against it. In using the instrument it is chiefly important to regard not so much the absolute power, as the difference between the two sides, the degree of muscular

force normally present varying very greatly in different individuals. Clinically, it is often sufficient to have the patient squeeze the physician's hand, first with one hand and then with the other; even moderate differences being readily detected by this means. The patient may also be instructed to resist passive movements, such as extension of the flexed arm; flexion of the extended arm; lateral movement of the head; opening of the eyelids, or the various movements of the lower extremities.

Intermittent claudication is a term applied to indicate the occurrence of transient, partial or complete paresis or lameness. Sometimes the patient suddenly becomes unable to continue locomotion, and falls to the ground; or one limb becomes weak, causing a pronounced limp and necessitating the aid of a crutch; while in other instances there is simply discomfort upon continued locomotion. This symptom occurs in various forms of functional nervous diseases, such as the periodic paralysis of Goldflam, *meralgia paræsthetica*, and as an idiopathic condition in diabetes and arteriosclerosis.

By **ataxia** is meant the inability to co-ordinate perfectly—that is, to give each muscle its due share in the performance of any action. As a result the movements are irregular and imperfect. Various types of ataxia have been distinguished: *spinal ataxia*, in which the disturbances of motion are more pronounced when the eyes are closed, and which is due to disease of the posterior columns; *cerebellar ataxia*, in which the disturbances are equally severe when the eyes are opened or closed, but which disappear when the patient lies down; *cerebral ataxia*, which occurs as a result of injury to the parietal lobe, and is accompanied by loss of muscular sense and marked persistent inco-ordination of movement; *pseudo-ataxia*, due to weakness of certain groups of muscles, so that they do not properly oppose the action of other groups. Finally, there is a form of ataxia apparently due to anæsthesia of the skin and loss of the muscular sense, in which the patient is able to perform movements perfectly as long as he can watch the part with the eye, but as soon as the eyes are closed the ataxia appears. Ataxia may be simulated by the groping exhibited by a person whose vision has recently become greatly impaired.

This condition may be tested in a variety of ways. Ataxia of the upper extremities may be recognized by directing the patient to touch the tip of the nose with the tip of the forefinger, or to extend the arms and bring the tips of the forefingers together with a rapid motion. In health, after one or two trials, either of these movements can be done perfectly. When ataxia is present, they are carried out awkwardly, and the forefingers are only brought in contact with each other or with the tip of the nose after several irregular coarse oscillations. The ataxia of the legs may be tested by requesting the patient while lying upon his back to touch with one of the toes some object held above his feet, or to bring the heel of one foot against the knee of the other. When the patient is erect, the ataxia may be tested by getting him to place the feet together, when there may be some swaying, which usually becomes much more marked when the eyes are closed.

Romberg's Symptom. If the ataxia is very slight, it may be necessary to have the patient stand on one foot with the eyes closed, or to attempt

to step backward under the same conditions. Under these circumstances the swaying is more pronounced than that noticed when a normal person attempts to perform the same movements. If the ataxia is at all severe, it produces a characteristic disturbance in the gait. (See Ataxic Gait.) Ataxia of the head is difficult to detect. Some observers contend that a peculiar form of grimacing whenever the patient attempts to move the lips or the eyes, or whenever the muscles of the face express some emotion, is an ataxic condition due to over-action.

Tremor. This is a disturbance of motion characterized by an oscillation of the part or parts involved. Tremor may be of various kinds. It may be fine or coarse, constant or irregular. It may disappear upon voluntary effort, or only be apparent when motion is attempted (*intention tremor*). It may be the result of paralysis, *paralytic tremor;* of poisoning, *toxic tremor;* of some functional nervous disease, as the *hysterical tremor;* or spasm of the muscle, *spasmodic tremor;* or it may occur as a family peculiarity without any discoverable cause, *hereditary* or *idiopathic tremor*.

Tremors are also classified as *rapid*, in which the movements occur more than five times per second; and *slow*, in which the oscillations may occur at intervals of several seconds. Nearly all forms of tremor are increased by placing the muscles upon a stretch. Tremor can usually be recognized by simple inspection. In some cases it is necessary to use peculiar methods for detecting it. Ordinarily it is sufficient, in order to detect tremor of the fingers, to have the patient extend them forcibly and keep them in that position. If the tremor, however, is exceedingly fine, its effect may be exaggerated by attaching long, light rods to the fingers, such as straws. This procedure is often useful in cases of tremor of the head or the feet. Tremors are nearly always exaggerated by fatigue or excitement. They develop in most normal persons in extreme forms of either of these states. These tremors are usually coarse, irregular, and either increased by or only apparent upon voluntary movements. Tremors may be recorded by attaching to the part affected rods whose ends are furnished with a pencil or stylet which writes upon a moving roll of paper. If a chronograph marks off seconds or fractions of a second at the same time, it is possible to measure very accurately the rate of oscillation. A more convenient method consists in requesting the patient to put the trembling part—for example, the hand—upon a small drum, which conveys each movement to an oscillatory stylet that marks upon a piece of smoked glass or paper. Seconds should be marked at the same time. Persistent fine tremor occurs particularly in paralysis agitans. In this the movements in the fingers are those of flexion and extension, and in the thumb of opposition, and it is therefore spoken of as the *pill-roller's tremor*. Voluntary effort causes it to cease for a brief interval.

Tremor also occurs not infrequently in exophthalmic goitre, and is increased by excitement or effort. The hereditary or idiopathic tremor becomes more apparent with advancing age, and is always increased by emotional disturbance. Irregular tremors occur as a manifestation of ataxia; often with cerebral lesions (the paralytic tremor); and after intoxications, as alcohol and tobacco. The hysterical tremor may be either irregular or regular. Its character is largely influenced by sur-

rounding circumstances; thus if the hysterical patient be in the hospital ward and have the opportunity of seeing a case of paralysis agitans, the tremor peculiar to that condition may be closely reproduced. Ordinarily, however, the hysterical tremor, being the result of voluntary and variable effort, is irregular. Intention tremor occurs particularly in multiple sclerosis. In this condition no tremor is observed while the parts are at rest, but as soon as voluntary motion is attempted a violent oscillation ensues, and continues until the effort ceases. Such a tremor can be particularly well elicited by asking the patient to convey a glass of water to his mouth. The movements become more and more violent as the lips are approached, and frequently more or less of the water is spilled. It may also be tested by asking the patient to touch some object with the forefinger. It will be observed, as the finger approaches, that the oscillations become more vigorous and wider. Intention tremor may, of course, be present in other parts of the body. In some respects it resembles the ataxic tremor. Generalized tremors are spoken of as convulsions or convulsive movements (*q. v.*).

Although the tremors of the extremities have been most carefully studied, other parts of the body are often affected. The best known are the tremors of the head. In this part the movement may be either forward and backward, lateral or rotary, rapid or slow. A peculiar tremor-like movement is the salaam tremor, a slow backward and forward movement that occurs in some forms of idiocy.

Muscular Spasm. By this is meant a condition in which the muscle is involuntarily contracted, either persistently (tonic or tetanic spasm) or rhythmically (clonic spasm); or there may be intermittent momentary spasm (tic).

Tonic spasms are characterized by vigorous contraction of the muscle, which becomes hard and painful. If only one group of muscles is affected, as, for example, the calf, the joint controlled by it is placed in the position normally assumed when the group is active. If all the muscles or even antagonistic groups of the upper extremity are affected, the flexors usually overcome the extensors. In the lower extremity the reverse occurs, but this is not invariable. When all the muscles of the body are involved, the powerful muscles of the back usually arch the spinal column, and there is more or less opisthotonos. Tonic spasms can usually be diagnosticated by simple inspection. They occur particularly in tetanus, strychnine-poisoning, and hysteria, and in these conditions may often be produced by peripheral irritation. These spasms are all spoken of as *active* in contradistinction to the so-called *passive* spasms resulting from actual shortening of the muscles or fixation of the joints. They may be distinguished from the latter by the fact that they almost invariably disappear during chloroform or ether narcosis, and sometimes also during sleep, and can be overcome by force without the tearing of tissue, although this is a less generally applicable test. Localized spasms in the upper extremities may occur as a result of disease of the cord above the cervical enlargement or of the brain, producing a spastic condition of the muscles, which, however, is rare. A more common type is the peculiar form of spasm seen in tetany, consisting in the closure of the fingers and

the opposition of the thumb, giving rise to the so-called *obstetrical hand*. Spasms in certain individual muscles of the hand or arm occur in the occupation neuroses. Spasms of the lower extremities are also occasioned by the various conditions giving rise to spasticity of the muscles. An idiopathic form of spasm not infrequently occurs in the calf muscles, particularly on awakening. It appears to be of no clinical significance.

Hysterical spasms are of various types. The tonic forms may affect a single limb or even a single group of muscles, and may persist for long periods of time, giving rise either to persistent extension or flexion of the limb. In the latter case shortening may ultimately ensue and cause persistence of the deformity. General hysterical spasms usually can be recognized by the fact that the patient assumes some extraordinary posture, as opisthotonos, pleurosthotonos, or emprosthotonos. These spasms are often precipitated by pressure upon some sensitive point (hysterogenic zone, ovaries), and may sometimes be abolished by pressing upon the same or a corresponding portion of the body. A peculiar form of localized tonic spasm is that occurring in the masseters, known as *trismus* or lock-jaw, which is a common symptom of tetanus. The *myotonic reaction* is sometimes spoken of as a form of tetanic spasm, and consists of a sudden, persistent contraction of the muscle or groups of muscles with which some voluntary movement has been attempted. It occurs, so far as known, only in Thomsen's disease.

Clonic spasms are of various types. They may affect a single extremity, half the body, or, in rare cases, the whole body. The movements are usually rhythmical and vary greatly in different cases. The most frequent causes of clonic spasms are injuries to the brain. Focal irritation in the motor region will produce at first a spasm in the part innervated by that area. If the irritation is sufficiently strong, or acts for a sufficiently long time, its influence will extend to the adjacent areas in the cortex, and a general unilateral or bilateral convulsion will ensue. This is the so-called epileptiform attack. If the local spasm is distinct and precedes by some time the development of the general twitching, it is spoken of as *focal*, or *Jacksonian epilepsy*. As a result of the violent irritation in the brain, unconsciousness usually ensues, but not invariably. Clonic convulsions may possibly be of local origin, although this is exceedingly doubtful. A localized form of clonic spasm, due to peripheral irritation in all likelihood, is *facial tic*, characterized by occasional or successive lightning-like contractions of the muscles of the face. Functional convulsions, particularly those occurring in hysterical patients, are very frequently clonic in character. Often there is a preliminary tetanic spasm, followed in a short time by the development of clonic movements. These assume various forms, the commonest being perhaps beating with the limbs, throwing of the head from side to side, and lateral or anteroposterior movements of the body. The attitudes and movements express fear, menace, ecstasy, eroticism, or other emotional states.

In certain conditions in which *ankle-clonus* is extremely exaggerated, it may occur as the result of slight pressure upon the sole of the foot and constitute a local clonic spasm of the calf muscles.

Irregular Movements in Chorea and Athetosis. Allied to the clonic spasms, but bearing also close affinity to tremors, are the irregular move-

ments that occur in chorea and athetosis. The typical movement of *chorea* is an irregular innervation of groups of muscles that appears to be voluntary in character, yet is not under the control of the patient, is much more rapid, as a rule, than a voluntary movement, and recurs at very frequent intervals. Choreic movements may be mild, or so severe that they produce irregular contortions of the body, causing the patient to throw himself or herself from side to side, and often producing severe bodily injuries and even death by exhaustion. *Athetosis* is a name given to a peculiar, slow, irregularly rhythmical movement of the extremities, generally spoken of as *worm-like* in character. It is ordinarily most marked in the fingers, which are gradually extended until they form almost a right angle with the back of the hand, and then slowly flexed and extended again, each finger moving more or less independently of the others. At the same time there is movement at the wrist-joint, the elbow, and sometimes of the trunk. The limbs may be affected, giving rise to a curious, staggering gait in which the patient seems ever to be about to lose his equilibrium, but maintains it almost like a miracle. Frequently the muscles of the face are involved, giving rise to curious, irregular grimaces and more or less disturbance of speech or *dysarthria*. The movements are usually continuous. Athetosis is a very common sequel to cerebral lesions occurring in early childhood.

More remotely akin to clonic spasms are the movements of *tic convulsif* or general tic; these are usually sudden, gesture-like movements, often repeated several times, and associated with violent grimaces. Sometimes the patient merely repeats certain purposive, co-ordinated movements a number of times, such as touching the palings in a fence. These are spoken of as *imperative movements*.

The term *convulsion* is used to designate general spasm with loss of consciousness. It is often employed, however, to indicate general clonic spasm of the whole body, even when consciousness is still present. This use is undesirable and should be avoided. General convulsions invariably indicate some disturbance in the brain; whether it be some chronic disease with occasional exacerbation of cortical irritation, some acute injury, or some systemic disease such as meningitis, uræmia, or a severe infection. A functional disturbance may be hysteria or epilepsy, although the latter is, of course, usually due to an organic lesion.

Muscular Tone. The term muscular tone means that condition of the voluntary muscles of the body by which they are maintained in a state of tension sufficient to enable them to respond promptly to nervous innervation. Muscular tone varies slightly under normal conditions. It is less in profound fatigue, and when the attention is distracted by external objects; it is more marked when the patient concentrates his attention upon the part being tested. It is invariably diminished after lesions of the peripheral motor neuron, in cases of profound cachexia, in coma, and during anæsthesia. It is also generally decreased in lesions of the posterior columns of the spinal cord. It is increased in lesions of the central motor neuron without involvement of the peripheral neuron, in neurasthenia, hysteria, and in conditions affecting the brain as a whole, as

meningitis, brain tumor, etc. It must be remembered that the reflexes may be increased in certain conditions, although the muscle-tone is apparently diminished. The usual test is the resistance to passive movement. The limb to be tested is grasped firmly, and if flexed, is suddenly but not too forcibly extended, or if extended, is flexed. If the muscle-tone is normal, there may be a transient involuntary resistance at first, but this disappears very soon, and then the limb may be moved in any position with comparatively slight effort. Any of the joints may be tested independently in this manner. It is important to inform the patient what is to be done. In children, in the ignorant, and in the insane it is often almost impossible to overcome the tendency to voluntary resistance, which is usually increased by the anxiety produced by the examination. Occasionally it is necessary to take measures to distract the attention, such as giving the patient a sum in arithmetic to perform, requesting him to look at the ceiling or some particular object, or engaging him in conversation. The increased resistance to passive movements may be so great that it is almost impossible to bend the limb at any of the joints, or so slight that it is difficult to discriminate it from the normal condition. The exaggerated forms are usually spoken of as *spasticity* of the muscles, and when associated with paretic or paralytic conditions the term *spastic paralysis* is employed. Diminution of the muscle-tone, or *hypotonia*, is usually difficult to detect by passive movements alone.[1] When it is entirely lost, the limb is spoken of as *flail-like*. The joints seem to have no tendency to remain in one position. If the limb is shaken, with every movement they pass from extension to flexion, or *vice versa*. Under these circumstances the passive movements are entirely unresisted, the only effort necessary being that required to overcome the weight of the limb itself. It is characteristic of tabes dorsalis and of paralysis due to injury of the peripheral motor neurons.

The Reflexes. These were first described by Westphal in connection with the knee. They consist essentially of a rapid twitch or succession of twitches in the muscle when the tendon by which it is attached to some bony part receives a sharp blow. There is some difference of opinion regarding the true nature of the stimulus required to produce them. According to Gowers, it is a simple contraction of the muscle, and he therefore uses the term *myotatic phenomenon*. Sternberg, on the other hand, believes that he has shown that they are the result of vibrations in the tendon, which are communicated by it to the muscle. Strümpell regards them as relics of the complex co-ordinated movements performed by the spinal centres of the lower animals, and explains the great constancy of the reflexes of the lower extremities by the greater amount of automatic action they are obliged to perform. Others contend that they are pure reflexes produced by the mechanical action of the blow upon the nerve-fibres in the tendon itself. It is certain, at any rate, that more factors are required than the mere tone of the muscle, and that afferent impulses to the spinal cord and efferent impulses from it are

[1] Various forms of apparatus have been devised for the purpose of estimating muscle-tone quantitatively. None is sufficiently convenient or accurate to be available for clinical purposes.

necessary to the development of the reflex; and that it is furthermore profoundly influenced by higher centres that usually have an inhibitory action (upper reflex arc). The question is complicated by the fact that in certain cases reflexes may be elicited by tapping the bony parts, such as the periosteal reflexes; by irritating the skin overlying the muscle, or even at a distance from it, as the cutaneous reflexes; or by tapping upon the fascia or the belly of the muscle itself. In general, it may be said that all conditions producing increased muscular tone produce exaggeration of the reflexes, and that all conditions diminishing muscular tone diminish the reflexes. In marked contradiction to this, however, are the facts that attention to the reflex which is being tested may diminish or abolish it completely, whereas distraction of the attention, as by directing the patient to perform some violent muscular effort (*Jendrassik's method*), which ordinarily diminishes muscular tone, increases the force of the reflex. Moreover, in certain forms of profound coma, in which the muscle-tone is greatly reduced, the reflexes often appear to be greatly exaggerated. Thus, in uræmia and diabetic coma, I have been able on several occasions to detect exaggeration of the reflexes when the limbs were flail-like in their relaxation.

The individual reflexes of the head are numerous but of little importance. The *supraorbital reflex* (McCarthy) consists of a slight contraction of the eyelids when the supraorbital nerve is struck. It is lost in facial paralysis and after section of the supraorbital nerve. It may, however, be observed on the sound side in cases of facial paralysis when the nerve on the paralyzed side is struck. The *malar* reflex is elicited by striking upon the malar bone. It consists of an elevation of the angle of the mouth. The *chin-jerk* is elicited by having the patient open his mouth slightly; then a flat object, such as a tongue-depressor, or the handle of a spoon, is placed upon the teeth of the lower jaw and sharply tapped with the finger or hammer. Under normal circumstances there will be a slight upward jerk of the chin. It may also be elicited with less discomfort to the patient by placing the finger beneath the lower lip and upon the mental prominence, and striking it sharply with the hammer. This does not always result in a reflex under normal conditions, but is quite satisfactory for the purpose of testing pathological exaggeration. The chin-jerk is nearly always increased in neurasthenia and hysteria, and is sometimes present in profound coma. It is also usually exaggerated in the conditions of general spasticity that are occasionally met with in severe infectious diseases. Its absence does not appear to be of any pathological significance. Allied to the tendon or periosteal reflexes is the phenomenon known as *Chvostek's sign*. This occurs only in tetany, and consists of a sudden, lightning-like twitching of the muscles of the face, particularly of the elevators of the angles of the lip and the muscles of the eyelids. It is elicited by striking the skin below the zygomatic arch just in front of the ear with the hammer. It was formerly supposed that this was due to mechanical irritation of the trunk of the facial nerve, but the same phenomenon can also be elicited by striking over the malar bone or in the region of the infraorbital foramen.

In the arms the most important reflexes are the *bicipital*, *tricipital*, and

the *supinator reflexes*. The bicipital reflex is best obtained by allowing the patient to rest the perfectly relaxed arm upon some support—for example, the arm of the investigator—in a semiflexed position. The finger or thumb is then placed upon the tendon of the biceps, and struck a sharp blow with the hammer or the finger, as in percussing. In nearly all normal cases a slight twitching or distinct contraction of the biceps can be obtained in this manner. Sometimes it is possible, by resting the arm upon a support, to see the tendon distinctly and to strike it directly, but this is usually much less satisfactory. The tricipital reflex is readily obtained by holding the arm semiflexed and relaxed, and then striking just above the olecranon process of the ulna. The supinator reflex is obtained by striking the radius just above the styloid process. These reflexes are particularly distinct in hemiplegia, upon the paralyzed side. They also occur in the general conditions above mentioned. Their absence is of no pathological significance, as it is often impossible to obtain them in normal individuals. In addition a reflex may be obtained by striking the bodies of the extensor muscles of the forearm, giving rise to extension of the fingers. A form of *wrist-clonus* occasionally occurs that may be elicited by suddenly flexing the wrist-joint either dorsally or ventrally, and holding it in the cramped position. The *hypothenar reflex* is the tonic contraction produced in the palmaris brevis by pressure upon the pisiform bone. It does not appear to be dependent upon any diseased condition.

The *scapulohumeral reflex* was described by von Bechterew in 1899, and appears to be of considerable importance. It is elicited by tapping upon the spinal border of the scapula at or just above the inferior angle. In the majority of normal cases this causes a slight adduction and an external rotation of the arm, apparently due to contraction of the spinati muscles. In functional conditions of exaggerated reflexes, such as neurasthenia, this reflex may also be increased, although the type remains unchanged. In cases of disease of the pyramidal columns above the cervical enlargement it is greatly modified; there is contraction of the posterior fibres of the trapezius, of the deltoid, biceps, and the muscles of the forearm. As a result the shoulder is lifted, the arm thrown from the side, the forearm flexed upon the arm, and the fingers extended. Frequently the muscles of the opposite shoulder also respond (*crossed reflex*). In disease of the brachial plexus the crossed reflex may exceed the reaction upon the affected side.

Tapping upon the bodies of the muscles sometimes gives rise to a sharp contraction. This is particularly observed in connection with the shoulder (Strümpell) and pectoral muscles. An important reflex, the *abdominal reflex*, is elicited by drawing the end of a blunt object obliquely across the skin of the abdomen downward and outward or upward and inward, the object being to make it cross the line of the intercostal nerves as nearly as possible at a right angle. This produces contraction in the muscles innervated by these nerves, and is due to the stimulation of their cutaneous distribution. It may be exaggerated in functional nervous conditions, and is diminished in cases of hemiplegia with anæsthesia on the affected side. Its absence at some particular point occasionally serves

as an additional factor in the localization of lesions of the spinal cord. The *cremasteric reflex* is elicited by irritating the skin on the inner side of the thigh, causing a quick retraction of the testicle, which should not be confused with the slow contraction of the dartos; it is really a part of the abdominal reflex. This phenomenon is not invariably present, is occasionally exaggerated in neurasthenic states, and is usually marked in persons who have voluntary control over the cremaster. It is no indication of sexual vigor, and is of slight clinical significance, but its persistence may serve to exclude lesion of the lumbar segments in which its arc is completed (the second to the fifth).

Various reflexes, probably periosteal or fascial in nature, may be produced by tapping upon the spinous processes of the ilium. As far as is known, they are of no clinical value.

The reflexes of the lower extremities are the most important of all. The first discovered, the *knee-jerk*, is invariably present in health, and by its delicacy and constancy is the most valuable reflex for clinical purposes. It may be elicited in a variety of ways. Perhaps the best method is to have the patient lie upon his back; then placing one hand under the knee it should be lifted several inches from the surface of the bed or table until the leg and thigh form an angle of about 120 degrees. Then with the finger, the side of the hand, the edge of the stethoscope, or the percussion-hammer,[1] the patellar tendon is struck a sharp blow between the lower edge of the patella and the tuberculum of the tibia. The stroke should be delivered with moderate force, and, according to the practice of most clinicians, a single blow is sufficient, but sometimes the reflex is more certainly elicited if several strokes are given in quick succession. The most obvious and vigorous contraction occurs in the quadriceps of the same side, causing the leg to be tipped upward suddenly and giving rise to the name knee-jerk. In addition, the adductors of the same side nearly always contract slightly, and occasionally the flexor muscles—that is, the biceps, the semitendinosus, and the semimembranosus—also contract. Frequently the adductors of the opposite side contract very slightly in health, and sometimes quite vigorously in diseased conditions (*crossed reflex*). Another method of obtaining this reflex is to allow the patient to sit on a low chair with the leg extended forward until it forms a blunt angle with the thigh, the heel being rested upon the ground. The patellar tendon is then struck as before. Clinically, it is usually sufficient when the patient is sitting in an ordinary chair to have one leg thrown over the other, and hanging loosely and freely. Occasionally it is difficult, on account of extreme relaxation of the muscles, to stretch the tendon sufficiently to obtain the reflex by this method, and Gowers suggests that under these circumstances the legs should be completely flexed upon the thighs. Exaggeration of the knee-jerk is characterized by a more vigorous effort or more extensive contraction of the surrounding muscles. The latter, indeed, may, by involvement of the flexors, diminish the excursion of the leg. Sometimes in cases of profound emaciation, as in

[1] There are various forms of these—one with a heavy metal head and short wooden handle, the end of the metal head being covered with leather; another, composed of a wedge-shaped piece of rubber set in a light metal handle; the latter is probably the better.

cachexia, although the knee-jerk is increased and the muscle apparently contracts vigorously, its power is so greatly diminished that it is unable to move the leg. Elaborate apparatus, therefore, that have been devised for measuring the knee-jerk, do measure in fact only the amount of movement of the foot, and are practically worthless. They consist essentially of an arc of a circle whose radius is approximately equal to the length of the leg. Either a pencil or a small readily movable index is placed against the foot, and the knee-jerk is measured by the number of degrees marked off on the scale. It is manifest that comparisons are only valuable when the blow is of exactly the same force, and then only when the experiments are performed upon the same individual within a limited period of time. In order to obtain a constant force of blow, various instruments have been devised, the simplest being weights dropped through a paper cylinder upon the patellar tendon, and the more complicated having springs for their motive power. It is often difficult to discover the tendon, either on account of deformity of the joint or because of an excess of fat tissue. In a case I observed, in which extensive arthropathies existed, the knee-jerk was present, but obtained with great difficulty on account of the distortion of the parts. The patellar tendon reflex, therefore, is a multiple muscular reflex, with phenomena on the opposite side, the so-called *bilateral reflex*. It is said to be invariably present in health, but its intensity varies considerably, and in some apparently healthy persons without any evidence of disease of the spinal cord, it is extremely difficult to elicit. Under these circumstances it is necessary to use various procedures in order to make it evident. These consist either in requesting the patient to look at the ceiling, in order to distract the attention, or to perform some violent muscular effort, such as an attempt to pull the hands apart when they are clasped together, to squeeze the dynamometer, etc. Under these circumstances the knee-jerk, if obtained, is spoken of as *reinforced*. (Jendrassik's method.) It is always important to have the muscles completely relaxed, and to persuade the patient not to think of what is being done. The knee-jerk is sometimes rendered more pronounced by emotion, and sometimes inhibited, as by fright. The arc of the knee-jerk is situated in the first lumbar segment of the cord, but probably occasionally deviates slightly from this position, being either higher or lower. The knee-jerk is therefore increased in any disease of the pyramidal tracts above this point, excepting total transverse lesion of the spinal cord, in which it is lost. It is diminished in disease of the efferent or afferent fibres. Its absence in tabes dorsalis was noted early (Westphal, Erb), and has long been considered evidence of disease of the posterior columns. It is often absent in cerebellar disease. Closely allied to the knee-jerk in its clinical significance and mode of occurrence is the *patellar reflex*. This is elicited usually by placing the finger transversely above the patella, pushing the bone forcibly down, and then striking the finger with the hammer. Ordinarily a distinct, pronounced contraction of the quadriceps alone is produced. In order to elicit this reflex the leg must be extended and relaxed. *Patellar clonus* occasionally occurs, and is obtained by placing the thumb and forefinger on the upper edge of the patella and pushing it forcibly downward and keeping it in that situation.

If clonus occurs, it will be characterized by a series of rapid contractions of the quadriceps, resulting in a vertical oscillation of the patella. It occurs in disease of the spinal cord, and not infrequently in conditions of increased tonicity in general infectious diseases.[1] In general, it may be said that the mechanical effect is dependent upon the condition of the nutrition of the quadriceps and the amount of interference of the opposing muscles. Tendon-reflexes may also be obtained by tapping upon the hamstring tendons. They are of no particular clinical value. Tapping upon the inner condyle of the tibia often produces contraction of the adductor muscles, but this is not, as a rule, as pronounced as the contraction produced by percussion upon the patellar tendon.

Next in importance to the knee-jerk is the *Achilles tendon reflex*, which consists in the contraction of the gastrocnemius and soleus muscles when the Achilles tendon is struck. It is most readily elicited by lifting the entire leg from the bed or table, and holding it by the ball of the foot, which is gently pressed upward. The tendon is thus moderately stretched, and may be struck directly. In nearly all healthy individuals this reflex is present, but is absent in some, and its absence is apparently of no clinical significance. Exaggeration may be indicated in moderate cases by the more forcible extension of the foot. In more pronounced cases it gives rise to a peculiar and characteristic phenomenon, known as *ankle-*

FIG. 49.

Ankle-clonus.

clonus. This may be elicited by vigorously tapping the tendon once or several times in succession while the leg is being held in the manner described; but is more readily produced by slightly flexing the leg and the thigh, then grasping the ball of the foot firmly, flexing it dorsally with considerable force, and holding it in that position. When ankle-clonus exists, there will be violent vibratory oscillations of the foot, as long as the pressure upon the sole is continued, that vary from two to three up to five

[1] C. K. Mills has devised an ingenious instrument, consisting of a metal ring with a curved handle, by which the patella may be drawn downward and the jerk or clonus more certainly elicited.

or ten movements per second. There is usually a rhythmical increase and decrease in the rapidity, without absolute cessation at any time. Occasionally, in very mild cases, the clonus after a few movements becomes weaker, and rapidly disappears. Ankle-clonus is supposed to indicate the existence of a lesion above the second lumbar segment of the spinal cord that seriously interferes with the function of the pyramidal tract. For a long time there has been doubt as to whether it occurs in functional disease, but it seems now to be established that it does. Its occurrence in functional conditions is, however, of such rarity that when it is present organic disease should always be suspected. It is most characteristic in spastic paraplegia, either due to transverse myelitis, to lateral sclerosis, or to syringomyelia, and also occurs after lesions in the motor regions of the brain. Ankle-clonus can sometimes be elicited by supporting the weight of the leg upon the toe. Under these circumstances it develops spontaneously in organic conditions, sometimes after fatigue, exposure to cold, or in states of exhaustion. It may also be produced in normal persons who continue for a sufficient length of time voluntarily oscillatory movements of the foot supported in this manner. A *pseudo-ankle-clonus* has been described as characterized by a few irregular oscillations that soon cease. It occurs in functional disease and occasionally among malingerers.

Tapping upon the tendon of the great toe occasionally produces a slight contraction of that member.

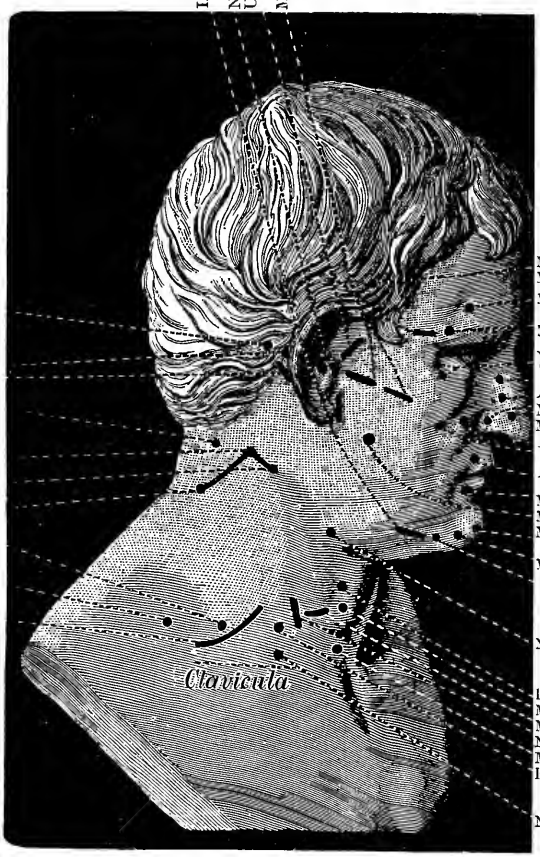
The other reflexes of the lower extremities are *front tap*, dorsal extension of the toes upon percussion of the anterior surface of the tibia, and the *toe-reflexes*. There are two known forms of the latter. One is called *Sinkler's reflex*, and is elicited by flexing the great toe upon the sole. The foot is then dorsally flexed, the leg flexed on the thigh, and the thigh on the abdomen, so that the limb is drawn up. It occurs only in conditions that cause extreme spasticity of the limbs, such as transverse myelitis. The other one is known as *Babinski's phenomenon*, and is elicited by stroking the sole of the foot from the heel toward the toes, preferably on the inner side. In normal states all the toes show plantar flexion ; in disease of the pyramidal columns this is replaced by a slow dorsal flexion of the great toe alone or of all the toes. This occurs in the great majority of cases of pyramidal disease, and is extremely rare in other conditions. It was at first alleged that it was normal in very young infants, but this has not been confirmed. The *plantar reflex* properly belongs to the group of cutaneous reflexes. It is characterized by the involuntary withdrawal of the foot when the sole is irritated. It is, of course, absent in cases of anæsthesia, and is generally exaggerated in functional nervous conditions, occasionally giving rise to a peculiar general tremor of the leg or even of the whole body. It is best elicited by drawing a blunt object (pencil, handle of a stethoscope) across the surface of the foot.

Allied to the reflexes is the so-called *paradoxical contraction* of Westphal. This consists in a persistent spasm of the muscle when its two attachments are suddenly brought closer together. It is most frequently observed in the peroneal muscles of the leg, and may be elicited by suddenly flexing the foot dorsally. It is found most frequently in various functional conditions, and has also been observed in paralysis agitans.

Nutrition of Muscles.

Next to the functional condition of the muscles, which is indicated by the degree of power that they possess, we are interested in the state of their *nutrition*. It may be suspected that this is impaired when fibrillary contractions, atrophy, loss of power, or change in consistency are present.

FIG. 50.



Motor points for the head and neck. (SAHLI.)

Atrophy of the muscles may usually be detected by simple inspection. If only certain groups are involved, the latter will appear more or less distorted. It is always, however, important to measure the injured limb and compare it with the sound side if the affection is unilateral. When due to general conditions, such as the muscular dystrophies or polyneuritis, it is sometimes more difficult to be certain of its existence. A general atrophy of the muscular system also occurs in cachectic states, such

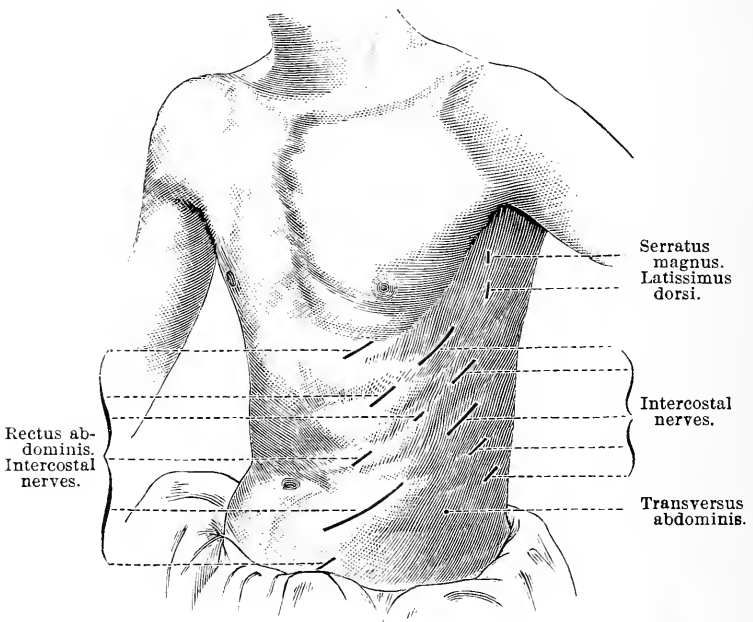
as the cachexia of carcinoma. *Fibrillary twitchings* occur in muscles undergoing degenerative changes. They are characterized by the sudden, spasmodic contraction of individual fibres in the mass of the muscle itself, giving rise to a curious trembling of the overlying skin and a peculiar sensation to the palpating hand, as if minute waves were passing through the muscular substance. They often occur spontaneously, and in degenerating muscles may be elicited by slight mechanical stimuli, such as cold, percussion, or shock. Fibrillary twitchings may also occur in healthy muscles that have either been chilled (tremor or shivering) or subjected to severe fatigue.

The most reliable method of diagnosis is by an *electrical examination*. For this purpose we use two types of apparatus. The galvanic current is produced by the galvanic battery, which consists of a number of cells, each containing an electropositive and an electronegative element and filled with battery fluid. Long wires are attached to the battery, through which the current flows when they are brought into contact or the circuit closed, and ceases when they are kept apart or the circuit opened. The free end of the wire toward which the current flows from the cell is called the *anode*, and the free end from which the current passes to the cell, the *cathode;* then if any substance is introduced between these ends of the wire, closing the circuit, the current passes through it from the anode or positive pole to the cathode or negative pole. It is customary to introduce a galvanometer graduated in milliampères[1] into the circuit for measuring the amount of electricity employed. As it is important to employ a definite number of milliampères, the apparatus is also provided with a rheostat, which renders it possible, by the introduction of a greater or less degree of resistance, to regulate the amount of electricity passing through the body. For medical purposes the free ends of the wires are supplied with electrodes. These consist essentially of metal disks or plates provided with a wooden or hard-rubber non-conducting handle. As the resistance normally offered by the skin is greatly reduced if it be moistened, the ends of the electrodes are covered with cotton or gauze and moistened by immersion in either plain or salt water. The area of the cross-section of the electrode may vary considerably. Ordinarily it is customary to have one very large electrode, from 50 to 100 square centimetres in area, and one having exactly 3 square centimetres of surface. (Stintzing's standard electrode.) In addition, for therapeutic purposes, it is customary to have for the galvanic and faradic apparatus a wire brush and various special electrodes for application to the more inaccessible portions of the body. If a muscle or nerve is to be investigated, the large electrode is thoroughly moistened and placed over the back or the sternum. It is not advisable to place it over the neck nor to allow the patient to hold it in the hand. The current is so arranged that this large electrode is at first the anode and the small electrode the cathode. The cathode is now

[1] 1 milliampère equals 0.001 ampère. The ampère is the unit adopted for the measure of the amount of current. It is determined by dividing the unit of electromotive force, 1 volt—that is, 0.9 of the amount of current liberated by a freshly filled Daniell cell, divided by 1 ohm—that is, the amount of current required to overcome a unit of standard resistance, or a column of mercury 1.06 metre in length and 1 square millimetre in cross-section.

placed over the muscle or the nerve to be stimulated, locating it, if possible, exactly over the most sensitive (electrically) point. This is most readily determined by comparison with the figures on page 308 *et seq.* The circuit should be opened and the rheostat so placed that the minimum amount of current flows through the body. The circuit is now rapidly opened and closed, while the cathode is kept in position and the rheostat gradually moved around until the current is sufficiently strong to produce a slight twitching of the muscle. This will first occur at the making of the circuit, and is spoken of as cathodal closing contraction, or CCC. The current should now be slightly increased, and by means of a switch the small electrode converted into the anode and the

FIG. 51.

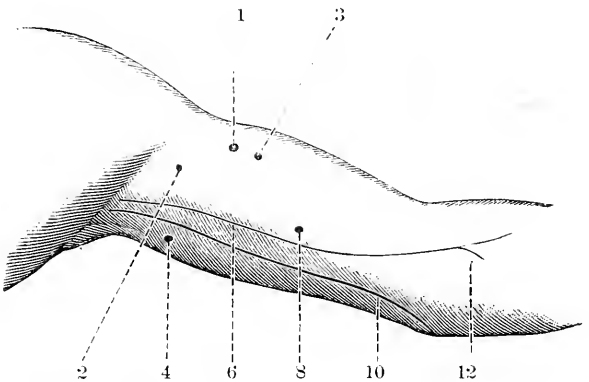


Motor points of the trunk. (From VON ZIEMSSEN.)

other into the cathode. It will soon be observed that a contraction takes place both at opening and closing the current. This is spoken of as the anodal closing contraction, or ACC, and the anodal opening contraction, or AOC. If the small electrode be again made the cathode, it will be found that there is a vigorous contraction when the current is closed, but none when it is opened. Finally, if the current is made still stronger, it will be found that the closure of the current produces at the cathode no longer a simple lightning-like contraction, but a prolonged cramp of the muscle, spoken of as cathodal closing tetanus, or CCTe. The contraction produced by both opening and closing the current at the anode is now much stronger than before, and there will probably appear a slight contraction at the opening of the cathode, the cathodal

opening contraction, or COC—that is to say, with a gradually increasing current the order of contraction is as follows in a normal muscle: cathodal closing contraction, anodal closing contraction, anodal opening contraction, cathodal closing tetanus, cathodal opening contraction. Under ordinary circumstances the healthy muscle contracts suddenly and relaxes

FIG. 52.

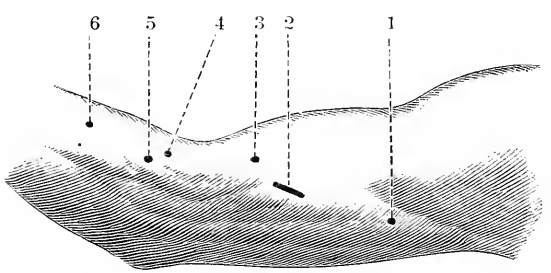


Motor points of the arm, under side. (From VON ZIEMSSEN.)

1. Musculocutaneous nerve. 2. Musculocutaneous nerve. 3. Biceps. 4. Internal nerve of the triceps. 6 Median nerve. 8. Brachialis anticus. 10. Ulnar nerve. 12. Branch of the median nerve to the pronator teres.

almost immediately. Various modifications of these phenomena occur in diseased conditions, and there are considerable quantitative changes in the different muscles in health. Thus, in the muscles of the face contraction is always more rapid than in those of the thigh, and can be elicited

FIG. 53.



Motor points of the arm, outer side. (From VON ZIEMSSEN.)

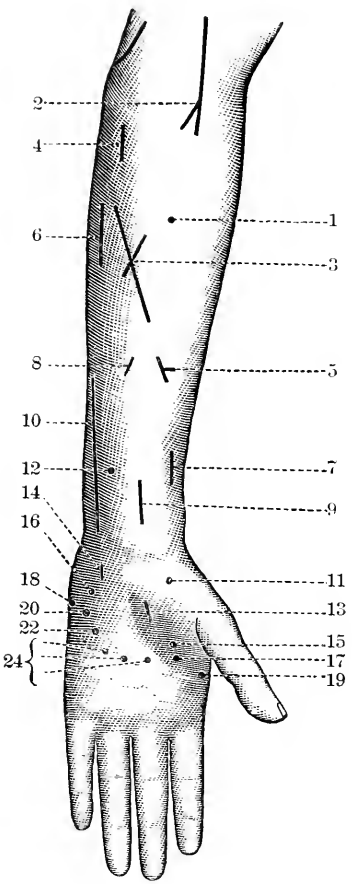
1. External head of the triceps. 2. Musculospiral nerve. 3. Brachialis anticus. 4. Supinator longus. 5. Extensor carpi radialis longior. 6. Extensor carpi radialis brevior.

with much weaker currents. In disease we recognize three types of alteration: (1) quantitative changes; (2) quantitative qualitative changes; (3) purely qualitative changes.

The *faradic apparatus* consists essentially of a coil of wire through which flows an electric current, that forms the core for a second coil not

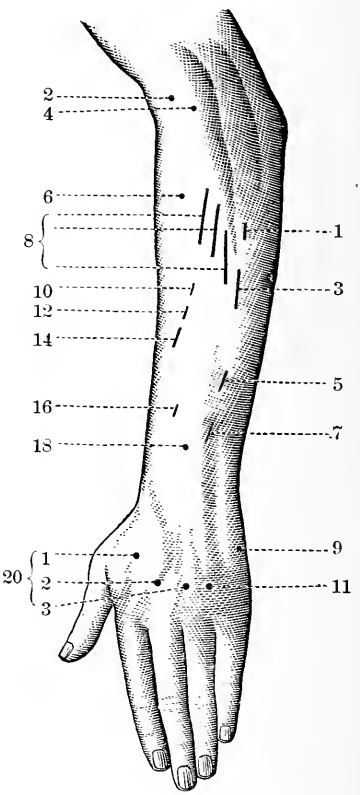
attached to it. If, now, the current passing through the inner or primary

FIG. 54.



Motor points of the forearm, inner surface.

FIG. 55.



Motor points of the forearm, outer surface.

(From VON ZIEMSSEN.)

FIG. 54.—1. Flexor carpi radialis. 2. Branch of the median nerve for the pronator teres. 3. Flexor profundus digitorum. 4. Palmaris longus. 5. Flexor sublimis digitorum. 6. Flexor carpi ulnaris. 7. Flexor longus pollicis. 8. Flexor sublimis digitorum (middle and ring fingers). 9. Median nerve. 10. Ulnar nerve. 11. Abductor pollicis. 12. Flexor sublimis digitorum (index and little fingers). 13. Opponens pollicis. 14. Deep branch of the ulnar nerve. 15. Flexor brevis pollicis. 16. Palmaris brevis. 17. Adductor pollicis. 18. Adductor minimi digiti. 19. Lumbricalis (first). 20. Flexor brevis minimi digiti. 22. Opponens minimi digiti. 24. Lumbricales (second, third, and fourth).

FIG. 55.—1. Extensor carpi ulnaris. 2. Supinator longus. 3. Extensor minimi digiti. 4. Extensor carpi radialis longior. 5. Extensor indicis. 6. Extensor carpi radialis brevior. 7. Extensor secundi internodii pollicis. 8. Extensor communis digitorum. 9. Abductor minimi digiti. 10. Extensor indicis. 11. Dorsal interosseus (fourth). 12. Extensor indicis and extensor ossis metacarpi pollicis. 14. Extensor ossis metacarpi pollicis. 16. Extensor primi internodii pollicis. 18. Flexor longus pollicis. 20. Dorsal interossei.

coil is interrupted, there will be generated at each opening of the current, a current in the outer or secondary coil, going in the opposite direction,

and, at each closure, a current going in the same direction. This is usually the stronger, and, if the interruptions are sufficiently rapid, dominates the reversed current. The ends of the secondary coil are attached to the electrodes. The strength of the current is altered by moving the inner coil away from the secondary coil. This is spoken of as the distance between the coils, and is measured in inches or centimetres. It is manifest that this method for measuring is not absolute, but its value must be determined for each particular machine. This can only be done by the physiological test—that is, measuring the force required to produce contractions in some muscles and then comparing it with the known value

FIG. 56.

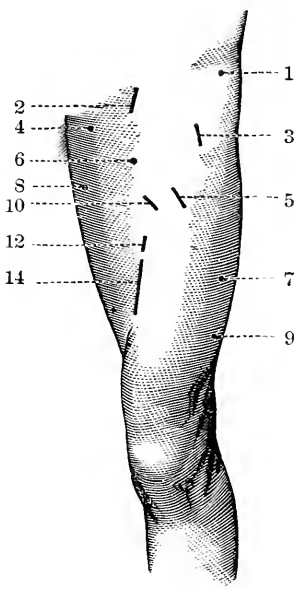
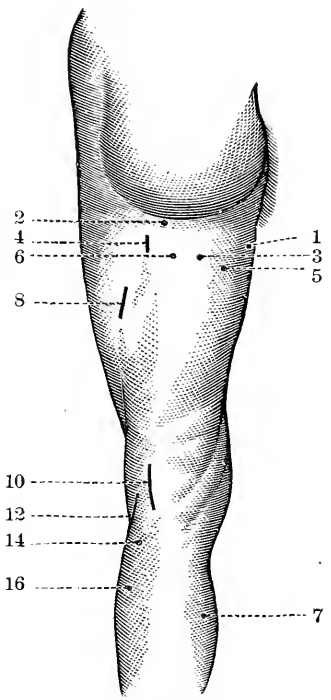


FIG. 57.



Motor points of the thigh, anterior surface. Motor points of the thigh, posterior surface.
(From VON ZIEMSSEN.)

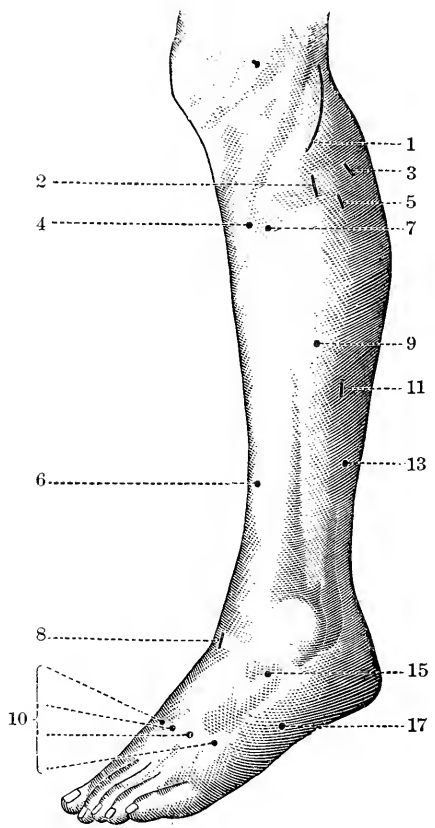
FIG. 56.—1. Tensor vaginæ femoris (braneh of the superior gluteal nerve). 2. Anterior crural nerve. 3. Tensor vaginæ femoris (branch of the crural nerve). 4. Obturator nerve. 5. Rectus femoris. 6. Sartorius. 7. Vastus externus. 8. Adduetor longus. 9. Vastus externus. 10. Branch of the crural nerve to the quadriceps extensor cruris. 12. Crureus. 14. Branch of the crural nerve to the vastus externus.

FIG. 57.—1. Adductor magnus. 2. Inferior gluteal nerve for the gluteus maximus. 3. Semitendinosus. 4. Great sciatic nerve. 5. Semimembranosus. 6. Long head of the biceps. 7. Gastrocnemius (internal head). 8. Short head of the biceps. 10. Posterior tibial nerve. 12. Peroneal nerve. 14. Gastrocnemius (external head). 16. Soleus.

for this muscle obtained by a standard machine, and obtaining in this way the ratio. The current is, of course, increased when the secondary coil is directly over the primary one and diminished when the primary

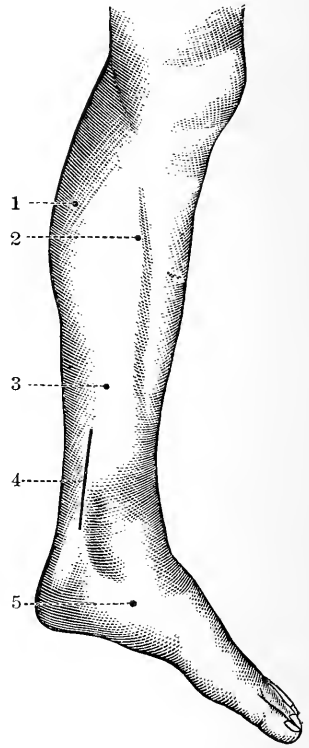
coil is withdrawn. As the current in the secondary coil is oscillatory—that is, going first in one direction and then in the other—it is not theoretically possible to speak of an anode and a cathode. Practically, however, the current going in the same direction as that of the primary coil is the stronger, and a difference does exist between the two ends of the wire, which are usually designated, therefore, as cathode and anode. A

FIG. 58.



Motor points of the leg, outer side.

FIG. 59.



Motor points of the leg, inner side.

(From VON ZIEMSSEN.)

FIG. 58.—1. Peroneal nerve. 2. Peroneus longus. 3. Gastrocnemius (external head). 4. Tibialis anticus. 5. Soleus. 6. Extensor longus pollicis. 7. Extensor communis digitorum longus. 8. Branch of the peroneal nerve for the extensor brevis digitorum. 9. Peroneal brevis. 10. Dorsal interossei. 11. Soleus. 13. Flexor longus pollicis. 15. Extensor brevis digitorum. 17. Abductor minimi digiti.

FIG. 59.—1. Gastrocnemius (internal head). 2. Soleus. 3. Flexor communis digitorum longus. 4. Posterior tibial nerve. 5. Abductor pollicis.

contraction produced by the faradic stream is always tetanic in health, as there are a series of stimulations constantly passing through the muscle. By employing a long and weighted vibrator the intervals between the interruptions may become so great that the muscle has time to relax between

each stimulus. In normal conditions the contraction resembles that of galvanic stimulation.

Alterations in the Reactions of the Muscles and Nerves to Electricity—Reactions of Degeneration. *Quantitative alterations* consist in increase or decrease of the susceptibility of the muscles or nerves to electrical action. When the lesion is unilateral, they may be determined by comparison with the normal side of the body; in the case of bilateral lesions a table of electrical reactions, such as has been furnished by Stintzing, must be used as a standard. If the deviation from the normal is slight, the error has very likely been produced by variation or alteration in the resistance of the skin. Quantitative increase in the electrical reaction occurs chiefly in tetany, for which disease it is almost pathognomonic, and has been spoken of as Erb's sign. It occurs also occasionally in the early stages of hemiplegia, in paralysis of the facial nerve, and has been noted in certain cases of tabes dorsalis. Diminished electrical irritability occurs in all the forms of idiopathic muscular dystrophy; in the forms of atrophy due to lesion of the central motor neuron without involvement of the peripheral neuron; in atrophies secondary to disease of the joints and loss of functional activity on the part of the muscle. Diminished reaction may occur in hysteria and profound neurasthenia, and has been occasionally observed in tabes dorsalis and in some cases of progressive spinal muscular atrophy of exceedingly slow course. It also occurs in certain nervous diseases whose nature is not yet understood, as in Goldflam's periodic paralysis; although it is to be noted that there are other alterations in the electrical reactions in this disease. The *quantitative qualitative reaction* consists in diminution of the reaction of the muscle or nerve to the faradic current, and diminution or exaggeration to the galvanic current, with distinct alteration of the order in which the various forms of galvanic irritation produce contractions. Cohn discriminates three types of this form of degeneration: (1) the complete reaction, mild in character, and terminating in recovery; (2) the complete reaction, severe and incurable; and (3) a partial reaction. He gives the following table illustrating the various stages of these three forms:

Total Reaction of Degeneration.

	Moderate Form.			
	Indirect stimulation (nerve).		Direct stimulation (muscle).[1]	
	F.	G.	F.	G.
1st stage, 1–8 days.	Diminished.	Diminished.	Diminished.	Diminished.
2d stage, 2–15 weeks.	Lost.	Lost.	Lost.	Increased, contraction slow. AOC>CCC.
3d stage, 6–30 weeks.	Returning.	Returning.	Returning.	Diminishing, contraction more rapid. AOC = or >CCC.

[1] By direct stimulation is meant the application of the electrode to the muscle itself. By indirect stimulation is meant the application of the electrode to the motor nerve-trunk. The latter term is employed because irritation of the nerve can only be detected by the activity of the muscle, and the stimulation of the latter is of course indirect in this mode of application.

Moderate Form.

4th stage, later.	Subnormal.	Subnormal.	Subnormal.	Subnormal, no qualitative changes.

Progressive Incurable Form.

1st and 2d stages.	As first and second stages above.			
3d stage, after 6 weeks.	Lost.	Lost.	Lost.	Diminished. or lost. AOC>CCC.

PARTIAL REACTION OF DEGENERATION.

	Indirect stimulation (nerve).		Direct stimulation (muscle).	
	F.	G.	F.	G.
1st stage, 1–8 days.	Normal or diminished.	Normal or diminished.	Normal or diminished.	Normal or diminished.
2d stage, 2–5 weeks.	Normal or diminished.	Normal or diminished.	Normal or diminished.	Increased, contraction slow. AOC<CCC.
3d stage, 6–12 weeks.	All normal or progressively diminishing.			
3d stage, 6 weeks.	Diminished or lost.	Diminished or lost.	Diminished or lost.	Diminished or lost, contraction still slow. AOC<CCC.

The following points in these tables need explanation. The faradic reaction obtained is similar to that which occurs in the normal muscle, but requires a much stronger current to produce it. The slowly interrupted faradic current occasionally produces a sluggish contraction in degenerated muscles. The galvanic reaction of the nerve is similar to that obtained under normal conditions, excepting that a stronger current is required. The contraction is lightning-like and disappears instantly. The direct galvanic stimulation of the muscle, however, produces a worm-like contraction very different from that observed in the normal muscle, and is ascribed to direct stimulation of the muscle itself, not to stimulation of the terminations of the motor nerves. This often occurs with a much weaker current than is normally required to produce contraction in the muscle. It will also be observed that the cathodal closing contraction is no longer the first to appear, but is replaced by the anodal opening contraction; and this is followed by the anodal closing contraction, cathodal closing contraction occurring only with a relatively strong current. If regeneration ensues, muscular contractions occur in response to weaker faradic currents, and by direct galvanic stimulation they become more lightning-like in character. Gradually the cathodal closing contraction appears in response to weaker currents, and finally occurs before the anodal opening contraction. If recovery does not take place, direct galvanic stimulation requires stronger and stronger currents, and there is no increase in the rapidity of the contraction. The cathodal closing contraction disappears, and finally only the anodal contraction remains, which is exceedingly slow and worm-like. When the muscle tissue has been completely replaced by connective tissue, all reactions naturally cease. The partial reaction of degeneration is very similar to the mild, complete form. Recovery proceeds, as a rule, very rapidly.

The voluntary contractions of the muscle as a rule persist after the reaction of degeneration has become manifest, unless there has been total destruction of the peripheral motor neurons. Often in cases of peripheral neuritis it will be observed that the reaction of degeneration is present in muscles that are apparently healthy, but which, when the process is progressive, subsequently atrophy. When regeneration occurs, voluntary motion will have been almost completely restored long before the muscle has become entirely normal, and it may often reappear before any improvement can be detected in the electrical reactions.

In testing for the reactions of degeneration the following points are to be noted: (1) the reaction of the nerve to faradic and galvanic electricity; (2) the reaction of the muscle itself. It is particularly important to be certain that only the muscle under investigation is affected by the electrical current. Sometimes it will be impossible to accomplish this, but ordinarily it can be done sufficiently well to insure positive results. It must be remembered, however, that the reactions of degeneration often occur in the muscles of limbs that have been injured, and may be found in limbs in which some of the groups of muscles have already undergone atrophy and thus altered the anatomical relations. Under these circumstances mistakes are very likely to arise. Sometimes valuable information can be obtained by stimulating a nerve-trunk and observing whether the muscles innervated show normal or impaired contractility.

Quantitative and qualitative reactions of degeneration occur primarily as the result of disease of the peripheral motor neuron. They are therefore found in all diseases of the spinal cord that affect the anterior cornua or the motor roots, and in all diseases of the medulla that affect the motor nuclei or their roots; in acute and chronic anterior poliomyelitis, progressive spinal muscular atrophy, in bulbar palsy, in transverse myelitis, syringomyelia, tumor of the cord, and as a result of chronic forms of meningitis, or disease of the vertebral column pressing upon the roots. They are also found in all forms of peripheral neuritis, whether toxic, infectious, or traumatic, and in all cases of solution of continuity of the nerves. They occur in the so-called idiopathic muscular dystrophies, but in these they are exceptional. They are also found in a few cases after cerebral lesions.

The reactions of degeneration may be used for determining the *prognosis* of the case. When after the sixth week the muscle does not respond as readily as before to direct galvanic stimulation, and the cathodal closing contraction becomes equal to or greater than the anodal opening contraction, the prognosis is exceedingly favorable; the increased rapidity of the contraction particularly is of great significance. If, on the other hand, after from six to twelve weeks no change has occurred, the anodal still precedes the cathodal contraction, and both are worm-like in character, the prognosis is doubtful. Months, however, may elapse before the muscle gradually begins to regain its normal character.

Atypical Forms of the Reactions of Degeneration. Only two of these are important. In the *myotonic reaction* the muscular contraction persists after the electrical stimulus has been removed. This occurs both with the faradic and the galvanic current, but the order of contraction to the

various forms of stimulation of the latter is not altered. Myotonic reaction is pathognomonic of Thomsen's disease—myotonia congenita. It is more likely to occur as a result of stimulation of the muscle itself than of stimulation of the nerve. The *myæsthenic reaction* is characterized by the rapid exhaustion of the muscle or nerve, so that relaxation may take place while the faradic current is still being employed; and if the muscle is stimulated successively several times, it loses its power to contract or requires a stronger current. Myæsthenic reaction occurs in periodic family paralysis and the various forms of myæsthenia. Remak and Marino have described a peculiar form of reaction which they name the *neurotonic reaction*. It consists of the persistence of the contraction only after stimulation of the nerve.

Disorders of Nutrition.

Trophic Changes in the Skin and Subcutaneous Tissues. Disorders of nutrition or trophic changes are lesions produced in the tissues as a result of defective or altered innervation. See Chapter XXIII., Examination of the Skin, page 204.

Trophic Changes in the Bones and Joints. See Chapter XXVII., Examination of the Bones and Joints, page 247.

The Contour of the Body. Alteration of the contour of the body occurs in various nervous diseases. See Chapters XIX., XX., and XXI.

Changes in the Extremities. See Chapter XXII.

Mental Disturbances.

Mental disturbances may be divided into disturbances of consciousness and disturbances of intellect. The first form presents a variety of degrees from *apathy*, in which the patient lies quietly, makes no voluntary attempt to commence a conversation, shows no interest in his surroundings, and only answers when addressed, to *lethargy* or *stupor*, and *coma*. The term coma implies that it is impossible to arouse the patient by any means, and at the same time the condition resembles more or less closely actual sleep. The reflexes are usually preserved, and there is a certain degree of perception to painful impulses, manifested by the withdrawal of the part irritated. *Unconsciousness* is, of course, a condition that can not be sharply differentiated from coma. The term is ordinarily applied to conditions that do not resemble natural sleep. The patient may lie quietly, but the breathing is stertorous, the eyes may be open, all the muscles may be relaxed, or various types of spasm may be present. These conditions occur in the intoxications, infections, and poisonings, and as a result of severe injury to the head. A peculiar type of coma, known as *coma vigil*, is characterized by complete relaxation of the patient, whose eyes, nevertheless, remain open and appear to observe what goes on around the bed.

The mildest form of *disturbance of intellect* consists in impairment of memory, or *amnesia*. This may be restricted to the memory of certain

things only, as the names of certain classes of objects or certain groups of words. It may also be restricted to loss of memory for certain definite periods of time, which may occur as a result of severe injury or disease during or about this period. If the memory is lost for the period preceding the traumatism, the condition is spoken of as antero-active amnesia; if for the period following, retro-active amnesia. Memory is commonly impaired in old age, and often as a result of chronic cerebral disease, particularly in paralytic dementia. General impairment of the intellect is manifested in a great variety of ways. Congenital failure of development is spoken of as imbecility or idiocy. In its milder forms imbecility consists in diminution of the reasoning powers, so that the patient is unable to form accurate judgments. In its severer grades, and particularly in the more pronounced forms of idiocy, intellectual activity may appear to be absolutely abolished, life being merely a mechanical process not under control of the reason. Both conditions are usually associated with alterations in the substance of the brain, either in the form of hydrocephalus or of the various scleroses associated with epilepsy. General impairment of the intellectual powers is spoken of as dementia. In its most typical form this occurs among the aged: the subject has loss of memory for recent events, is confused and querulous, and the reasoning powers are defective. Dementia also occurs as a terminal stage in other forms of insanity, such as periodic insanity, Huntington's chorea, etc. Disturbance of the intellectual functions associated with excitement and more or less violence is usually spoken of as *delirium*. This may be severe or mild. It is characterized by a tendency to talk or to be noisy, and by great restlessness. Delirium occurs in many of the acute infectious diseases, particularly in meningitis, meningismus, in the intoxications, and sometimes in profound cachexia. Among the commoner symptoms of intellectual disorder usually grouped under the term insanity are exaltation or *mania*, depression or *melancholia*, and delusional states or *paranoia*. By *mania* is meant excessive intellectual activity, characterized by a tendency to be noisy, to be active, fondness for singing, shouting, swearing, or punning. There are usually, also, in the acute forms, a rapid loss of weight and decrease in the physical powers, while the patient believes himself to be in the most admirable and exceptional condition. Mania occurs as a result of the inflammation of the brain-substance in *acute delirium*. It occurs in the exacerbations of general paresis and in diseased states of unknown etiology that are known by the term itself. In *melancholia* the expression of the patient is mournful; he is commonly quiet, sits with his head cast down, refuses to speak, to eat, or to take any interest in what goes on about him. Often he weeps or groans, and when persuaded to talk, expresses an acute sense of his sins and the hopelessness of his salvation, or will complain of misfortunes that have not befallen him. Melancholia occasionally occurs in general paresis, and as one of the varieties of insanity. The term *paranoia* is used by different authors in very different senses. In general, it may be said that the majority imply by it the existence of delusions or false ideas that have, among themselves, a certain logical sequence, or, as the term is, are organized. Thus a paranoiac may believe that he is being persecuted by a certain

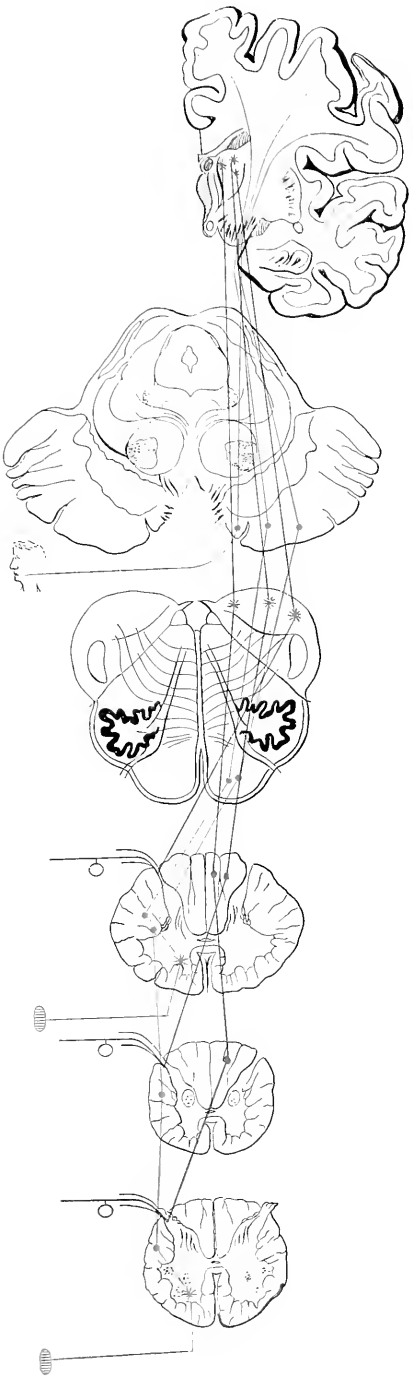
person and be able to give reasons why his persecutor should torment him. It must not be forgotten that occasionally those delusions may be true in fact, although none the less, symptoms of the mental condition. When there is merely a false idea, it is spoken of as a *delusion.* If the person complains of certain physical impressions, such as non-existent sounds, visions, odors, or tastes, the term *hallucination* is generally employed.

Localization of Lesions of the Nervous System.

In a diagnosis of diseases of the nervous system, particularly those that are the result of focal lesions, it is usually far more important to determine the situation of the lesions than the nature of the pathological process. The nervous system may be regarded physiologically as a collection of neurons. By *neuron* is meant a nerve-cell and all its processes to their ultimate ramifications. The processes are of two kinds: the so-called protoplasmic processes, which are relatively short, thick, and branched, and appear to resemble in many respects the protoplasm of the nerve-cell itself; and the axis-cylinder, a long, slender process that in its course gives off at regular intervals still more slender branches, the collaterals, and at its termination usually breaks up into a small tuft of fibres which surround some other ganglion-cell. An exception to the latter rule is formed by the axis-cylinders of the motor cells that run to the muscles, and end in tufts of fibres distributed to peculiar terminations in the muscle-fibres. At a certain distance from the nerve-cell the axis-cylinders usually become surrounded by myelin-sheaths, and constitute the nerve-fibres which make up the greater bulk of the central nervous system (the white substance), and practically all of the peripheral nervous system. Neurons with similar functions are usually grouped together, the aggregation of the cells forming a nucleus, and of the fibres, a bundle, tract, or system. The gray matter is largely composed of these groups of ganglion-cells or nuclei. Physiology has shown, although not conclusively, that the axis-cylinders convey impulses from the cell, and the protoplasmic processes convey impulses or nutriment to the cell. In the cell itself these impulses are modified or altered in some as yet unknown manner. At present the course and functions of comparatively few of the groups of neurons are known. Those which have been most accurately studied may be divided into the sensory neurons, conveying impulses from the peripheral nervous system, and the motor neurons, conveying impulses from the central nervous system to the muscles.

Sensory Neurons. The sensory neurons commence in the various sensory corpuscles, in the skin and organs. They pass through the peripheral nervous system to the posterior roots of the spinal cord, and here each enters a cell in the ganglia of the posterior roots. From these cells a fibre emerges that for a short distance is continuous with the entering fibre, and then leaves it and continues along the posterior root of the spinal cord. Here it divides into two branches, an ascending and a descending branch. Of the function of the latter nothing certain is

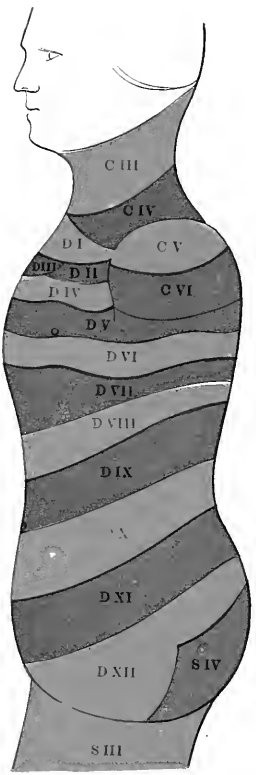
PLATE III-a.



Schematic Representation of the Course of the Motor (red) and the Sensory (blue) Fibres through the Brain and Cord.

The motor fibres arising in one hemisphere pass through the internal capsule, the crus cerebri, and the pyramids of the medulla on the same side, decussate in the first cervical segment, and pass down the cord in the lateral pyramidal tracts. At different levels fibres pass through these tracts to the anterior cornua and terminate around the multipolar ganglion cells. From these cells fibres emerge which pass through the anterior roots and the peripheral nerves to the muscles. The sensory fibres commence in the skin and other peripheral structures of the body, pass through the peripheral nerves of the posterior roots, enter and emerge from the cells in the ganglia of the posterior roots, and continue into the column of Burdach of the cord, decussating almost immediately to the opposite side. They pass up through the column of Burdach, then enter the column of Goll, and terminate about the cells in the ganglia in the dorsal part of the medulla. From these cells fibres emerge which pass through the fillet and terminate about the cells in the optic thalamus on the side opposite to that from which the sensory fibre took its origin. Other fibres (not shown), particularly those for pain and temperature, pass upward through the central gray matter of the cord.

PLATE III.-b.



Heart:
Cervical plexus (=Depressor ?)
1st, 2d, and 3rd Dorsal segments.

Lungs:
Cervical plexus (≒Vagus ?)
1st, 2d, 3rd, 4th and 5th Dorsal segments.

Liver and Gall Bladder:
6th, 7th, 8th, 9th, and 10th, Dorsal.

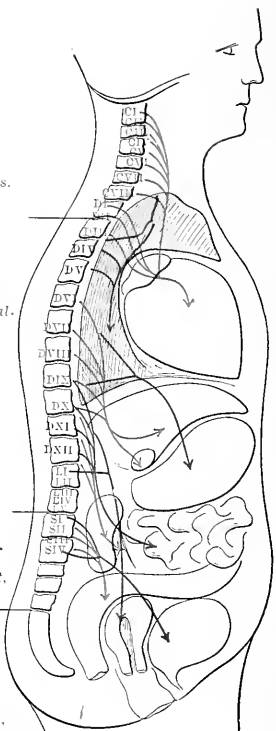
Stomach:
6th, 7th, 8th, and 9th Dorsal
cardiac end=6th, and 7th
Pylorus=9th.

Intestines:
(A)–Down to Rectum
9th, 10th, 11th, and 12th Dorsal.

B. Rectum:
2nd, 3rd, and 4th Sacral.
Kidney and Ureter:
10th, 11th, and 12th Dorsal.

Bladder:
(A)–Mucous membrane and neck.
1st, 2nd, 3rd, and 4th Sacral.
(B)–Over distention and ineffect. contraction 11th, and 12th, Dorsal and 1st Lumbar.
Uterus:
(In contraction) 10th, 11th, 12th, Dorsal and 1st Lumbar.
Os uteri: 1st, 2nd, 3rd, and 4th Sacral.

Ovary: 10th Dorsal
Appendages, etc. 11th, and 12th, Dorsal and 1st Lumbar
Prostate: 10th, 11th, and 12th, Dorsal 5th Lumbar, and 1st, 2d, and 3rd Sacral.
Epididymis: 11th and 12th Dorsal 1st Lumbar.
Testis: 10th Dorsal.



known. Some of the ascending branches pass into the lateral posterior column (Burdach), and at a higher level into the median posterior column (Goll). Those entering the cord in the upper dorsal and cervical regions, however, do not pass into the median posterior column, but continue in the lateral posterior column. Both columns end in the nucleus cuneatus and the nucleus gracilis, respectively. These two nuclei may be looked upon as indicating the termination of the peripheral sensory neurons. These two groups of fibres probably convey only tactile and muscular sensations. The fibres conveying pain and temperature sensations apparently pass up the cord through the central gray matter, but their central terminations are not yet definitely known. From the ganglion-cells in the two nuclei in the medulla, axis-cylinders arise that pass toward the brain and form a mass of fibres known as the fillet. In the medulla these are situated on either side of the median line, lying between the olivary bodies. They continue to occupy the central regions of the pons in its posterior part, but anteriorly they gradually spread out until they form a narrow band, placed horizontally, just below the gray matter surrounding the aqueduct of Sylvius. They then enter the tegmentum of the crus, and the majority lose themselves in the ventral nucleus of the optic thalamus. They constitute the second chain of sensory neurons. It is probable that from the optic thalamus, and from the other nuclei in which perhaps fibres of the fillet terminate, other axis-cylinders arise which pass through the corona radiata to the sensory areas in the cortex. These sensory areas will be discussed in connection with the cortical localization.

Destructive lesions in the peripheral sensory nerves produce total anæsthesia of the part supplied. Partial lesions may produce partial anæsthesia or even dissociation of sensation. *Irritative lesions* of the peripheral nerves

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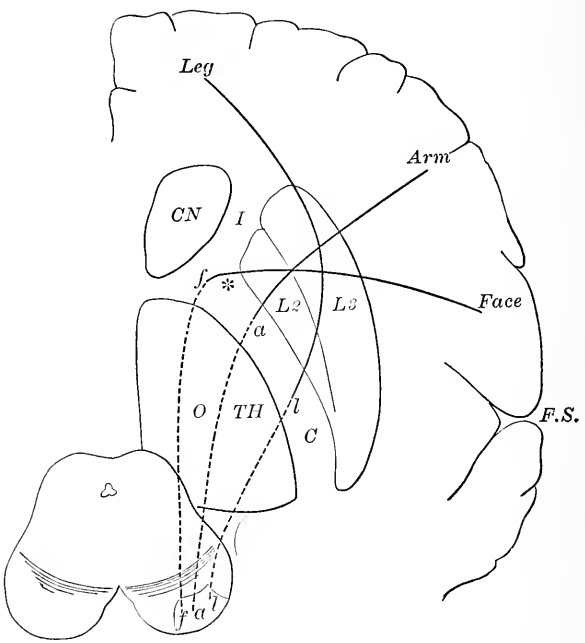
EXPLANATION OF PLATE III., B.—ILLUSTRATING THE COURSE OF THE SENSORY AND MOTOR NEURONS FROM THE CORTEX TO THE PERIPHERY.

MOTOR NEURONS (red). The fibres to the face commence in the lower portion of the motor cortex and pass downward through the internal capsule to the crus. Between the crus and the lower portion of the pons they decussate and pass to the facial nucleus of the opposite side. From these, fibres pass to the half of the face on the same side. The *fibres to the arm* arise in the cortex above the facial area and pass downward to the crus, then to the pyramidal columns in the medulla, and in the first cervical segment cross the median line to the lateral pyramidal tracts in the cord of the opposite side, from which they pass forward to enter into association with the dendrites of the cells of the anterior cornua. From these cells the peripheral neurons arise which innervate the muscles of the arm. The *fibres to the leg* follow the same course, but continue down the cord in the lateral columns until they reach the lumbar enlargement, where they pass into the gray matter and enter into association with the dendrites of the cells of the anterior cornua. From these cells nerve-fibres pass to the muscles of the leg.

The SENSORY NEURONS (blue) arise in the cells of the spinal ganglion. They enter the posterior roots of the spinal cord, cross the median line, and then enter the columns of Burdach and then become more central in the columns of Goll. The same course is followed by fibres entering at higher levels. These fibres terminate in the nucleus gracilis and nucleus cuneatus of the medulla. From these, other sensory neurons arise which pass upward through the tegmentum and internal capsule, and terminate chiefly in the optic thalamus.

produce severe pain, usually referred to the part supplied by the nerve, and there are also sensitive points or general tenderness over the nerve-trunk. Certain forms of irritative lesion produce partial alteration of sensation, which is usually spoken of as paræsthesia (*q. v.*). Trophic changes in the skin often occur. *Destructive lesions of the posterior roots* also produce total anæsthesia. If the lesion is on the peripheral side of the ganglion, there are in addition trophic changes in the part supplied.

FIG. 60.



Showing the relative positions of the several motor tracts in their course from the cortex to the crus.

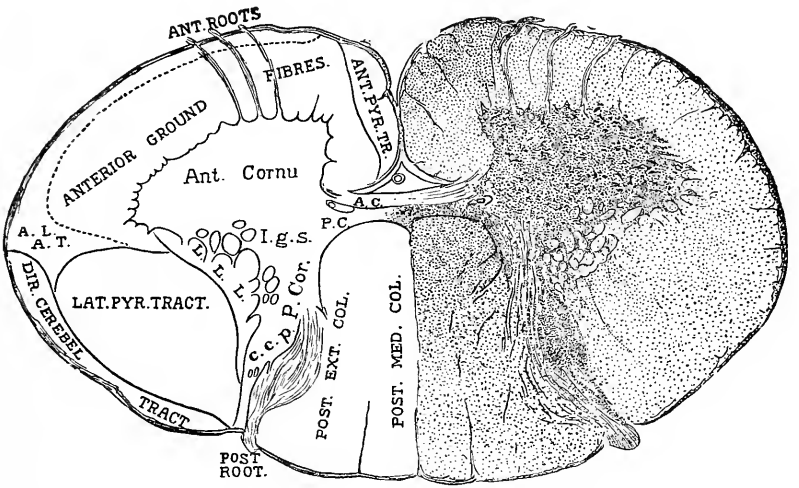
The section through the convolutions is vertical; that through the internal capsules, IC, horizontal; that through the crus is again vertical. CN, caudate nucleus; OTH, optic thalamus; L^2 and L^3, the middle and outer parts of the lenticular nucleus; *f a l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres. (GOWERS.)

If the lesion lies between the ganglion and the spinal cord, the anæsthesia is total, but trophic changes do not occur. Lesion of the ganglion itself usually produces anæsthesia and trophic changes, if complete; if partial, the symptoms are variable. In some cases herpes zoster along the course of the nerve has been observed. *Irritative lesions* of the posterior roots produce fulgurant pains in the limbs, or a feeling of constriction in the trunk. They may also be the cause of visceral crises. *Destructive lesions of the posterior columns* of the spinal cord produce more or less tactile anæsthesia and loss of the muscle sense. As a result of the latter there is ataxia. Lesions of either of the two central sensory neurons produce various forms of anæsthesia, depending upon their extent. According to

our knowledge of this subject, destructive lesions, such as hemorrhage or aneurism, in the posterior portion of the posterior limb of the internal capsule, or destructive lesions of the optic thalamus are usually associated with hemianæsthesia on the opposite side of the body. At times tactile sense is preserved and only the pain sense lost. As a rule, however, all forms of sensation are more or less affected.

Motor Neurons.—The motor neurons consist of two groups, the central and peripheral neurons. The *central motor neurons* commence in the motor portion of the cortex. They then pass through the corona radiata to the internal capsule, where they form a large band of fibres occupying the knee and the anterior two-thirds of the posterior limb. (See Fig. 60.) The fibres for the face occupy the knee and anterior third of this portion. Next come the fibres for the arm, then those for the leg, and, finally, the fibres for the trunk. From the internal capsule the fibres pass into the

FIG. 61.



Showing the different tracts of the cord. (GOWERS.)

crura cerebri, where they lie beneath the substantia nigra, occupying about the middle of each crus. The fibres for the face and cranial nerves lie internal to those for the extremities and trunk. From here they pass to the ventral portion of the pons, where they are broken up into small bundles by the association of fibres of the two cerebellar hemispheres. These reunite and form the pyramids in the anterior portion of the medulla, which decussate in the first cervical segment and pass down the cord as the lateral pyramidal columns. (See Fig. 61.) A few of the other fibres, however, do not decussate at this time, but pass downward in the direct pyramidal columns which decussate through the anterior commissure of the cord at lower levels. The fibres for the cranial nerves decussate, as a rule, in the neighborhood of the nuclei for these nerves, and by this means we are able to locate with considerable accuracy the situation of lesions in the pons and medulla. The fibres for the oculo-

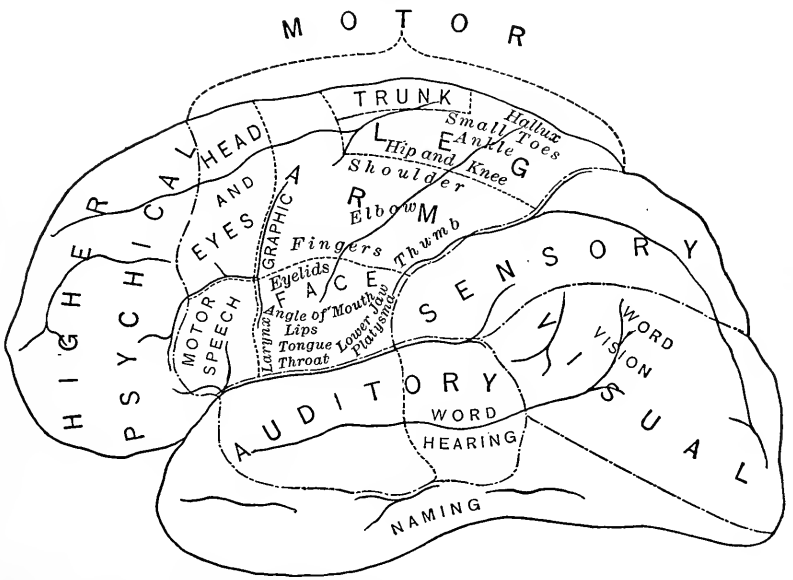
motor nerves decussate in the tegmentum and the nuclei around the aqueduct of Sylvius. The fibres for the facial decussate in the anterior portion of the pons. From this point downward fibres are continually crossing the median raphe to the nuclei of the various motor cranial nerves until the main decussation—that is, in the first cervical segment. It follows, therefore, that if a lesion occurs in such a position that it affects the fibres of one of the cranial nerves after they have crossed the median line, at the same time involving the undecussated fibres of the pyramids, we will have the syndrome known as a *crossed paralysis*—that is, the muscles supplied by the affected cranial nerves will be paralyzed on the same side as the lesion, and the rest of the body on the opposite side. (See Lesions of the Cranial Nerves.) The *peripheral motor neurons* commence in the cells of the anterior cornua of the spinal cord, passing out through the anterior roots, and reach the muscles through the peripheral nerves.

The functions of these two sets of neurons are not identical. The central motor neurons convey impulses from the cortex to the cells of the anterior cornua, by which the latter are stimulated to produce muscular movement. At the same time they seem to possess an inhibitory influence by means of some form of constant activity, so that while they are intact the reflexes are restrained and the muscles do not become spastic. Upon the nutrition of the muscles they apparently have no influence whatever, or at least act only indirectly by causing paralysis. The peripheral motor neurons control directly muscular activity. By their continuous action they maintain muscle-tonus, and when unrestrained by the influence of the upper neurons, produce a condition of spasticity. While they and the sensory neurons forming the arc are intact, reflex action persists. They also control in some unknown way the nutritional changes in the muscles. Destructive lesions of the lower neurons—that is, of the peripheral nerves involving the motor fibres, of the anterior root, and of the ganglion-cells in the cornua—cause paralysis and degenerative changes in the muscles. Irritative lesions cause spasms, usually tonic in character, and either momentary (as in facial tic) or persistent (tetanic). The muscle-tonus is lost, and the paralysis is therefore flaccid in character, while the reflexes are abolished. Destructive lesions in the central motor neurons, on the other hand, produce paralysis of the muscles; but their nutrition is not impaired, their muscle-tonus is increased to spasticity, and the reflexes are exaggerated. Irritative lesions of the central motor neurons produce, as a rule, clonic spasms. These may be limited to the part irritated, as occurs in some forms of central softening in the motor region, or become generalized. (See Convulsions.)

Cortical Localization. The origins of the motor neurons and the terminations of the sensory neurons are, as will be seen from this description, in the cortex of the brain. It is therefore of considerable importance to be able to locate the portions of the cortex that have to do with these functions. As a result of experimental work and of the repeated examination of pathological specimens a considerable amount of knowledge has been acquired upon this subject. The motor regions, indeed, have been marked out with accuracy, and some of the regions for the recep-

tion of impulses from the organs of special sense are also certainly known. The cortex of the brain has been divided into various regions which are referred to certain fissures which are quite constant in position. The most important of these is the fissure of Sylvius, which separates the temporosphenoidal lobe below from the frontal and parietal lobes above. Around its posterior extremity winds the convolution known as the gyrus angularis. Next is the Rolandic fissure, passing from the superior longitudinal fissure to the fissure of Sylvius, with which it forms an acute angle. It separates the frontal from the parietal lobe, and lies in the

FIG. 62.

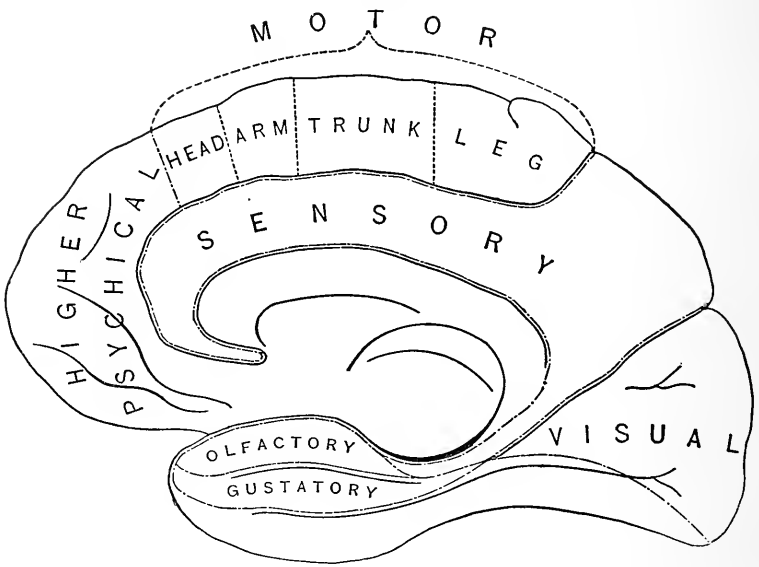


Cortical centres and areas of representation on the lateral aspect of the hemicerebrum. (MILLS.)

midst of the motor region of the cortex. In front of it is the ascending frontal convolution, and behind the ascending parietal convolution. These two contain nearly all the motor centres. The third prominent fissure is the occipitoparietal. It is best defined on the median surface of the brain, but can be traced for a short distance on the convex surface. It separates the parietal from the occipital lobe. On the median surface it unites at an acute angle with the calcarine fissure, the two enclosing between them the triangular convolution known as the cuneus. (See Fig. 62 and Fig. 63.) The motor centres are so arranged that those for the face are in the lowest portion of the motor region, those for the arms just above them, those for the legs above these, and those for the trunk in the posterior termination of the ascending parietal convolution, along the margin of the superior longitudinal fissure. These centres do not represent particular muscles, but particular forms of movement, involving frequently the simultaneous contraction of several muscle-groups. It is not known how sharp their limitations are, but it is supposed that the

central portion of the focus is exclusively devoted to its function, while at the periphery this fades gradually into the surrounding centres. The motor region for speech was discovered by Broca in 1861. It occupies the posterior portion of the third frontal convolution and the lower part of the ascending frontal convolution. The terminations of the sensory neurons have not yet been conclusively determined. It seems likely that some of them terminate in the motor region, and others in the upper portion of the parietal lobe. It is probable that different forms of sensation are represented by different areas upon the cortex, but at present our knowledge of this subject is uncertain. The anæsthetic areas produced by cerebral lesions have, in some cases, a characteristic distribution.

FIG. 63.



Cortical centres and areas of representation on the mesial aspect of the hemicerebrum. (MILLS.)

They are found on the opposite side of the body, and on the limbs are bounded by horizontal lines at right angles to the long axis, the so-called glove or stocking form of anæsthesia. Upon the trunk, the type of anæsthesia must be determined by exclusion—that is, it corresponds neither to the distribution of the cutaneous nerves nor to the segmental innervation of the cord; it may, however, closely resemble the hysterical type. The stereognostic sense appears to be situated in the parietal lobe—that is, lesions in this locality will cause its loss without disturbance of tactile sensation. As it has been shown that this sense is largely dependent upon muscular and localization senses, it is likely that the fibres conveying these terminate in the parietal lobe. It is to be noted that although it is the general rule that fibres from one hemisphere ultimately pass to the opposite side of the body, this is by no means invariably the case. Certain muscles, such as those of the trunk, apparently are innervated from

both sides of the brain—that is, bilaterally—so that if one centre is destroyed, the other assumes its functions, and no paralysis ensues. It also appears possible, in certain instances, for the centre of one hemisphere gradually to learn to perform the functions of the centre of the other hemisphere when the latter has been destroyed. This is seen most clearly in cases of destruction of the speech-centre on the left side, when, if the patient is still young, the speech-centre on the right side may assume all its duties.

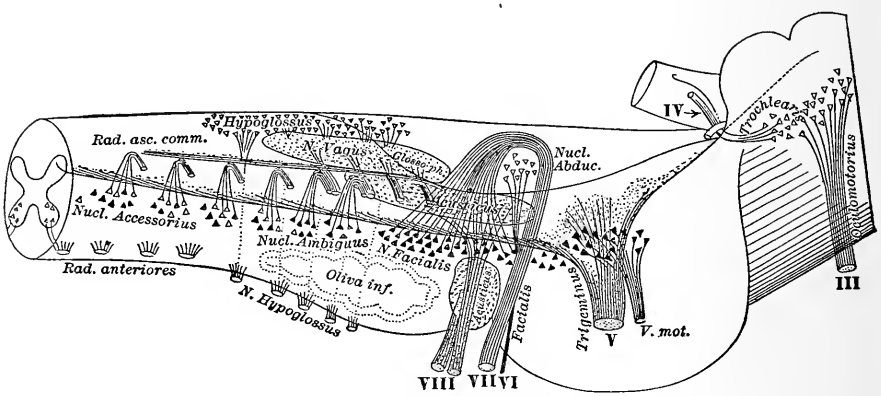
The Centres for Reception of Special Senses. The cuneus of the median surface of the occipital lobe appears to receive directly the fibres from the optic tract. When it is destroyed there is bilateral contralateral hemianopsia. The pupillary reflexes are, however, preserved, so that light-impulses must exert some activity at a point in the chain of neurons between this and the eye, probably in the anterior quadrigeminal bodies. The centre for audition is situated in the temporosphenoidal convolution. Destructive lesions produce deafness in the ear of the opposite side, or at least impairment of hearing which, as a rule, rapidly disappears. The centres for smell and taste have been placed respectively in the uncinate and fornicate convolutions. The evidence for these localizations is very strong, but is not yet absolutely conclusive. It is doubtful whether irritative lesions in any of the centres for special sense are responsible for hallucinations.

The functions of the *frontal lobes* are not well known. It has been supposed that they are the seat of intelligence, but there has never been adequate proof of this. Lesions of the frontal lobes may, therefore, exist without giving rise to any symptoms that might lead to a suspicion of their presence. On the other hand, the patients may exhibit various intellectual disturbances, but, on the whole, none that are characteristic, and perhaps these symptoms do not occur more frequently as a result of disease of this part than when some other part of the brain has been affected. It has been claimed that there is a certain degree of intellectual impairment; that the patient, while not insane or even eccentric, becomes incapable of exercising the same degree of judgment and comprehension that he formerly possessed. It has been claimed, also, that a peculiar form of insanity, characterized by progressive dementia associated with a manifestation of self-contentedness, occurs only in association with lesions of this part, and it has been given the term *moria*. A tendency to make puns has also been ascribed to these lesions; it is not always present, but, on the other hand, it may occur as an early manifestation of insanity without gross lesion or in connection with lesions of other parts of the brain. The most important symptoms, of course, are those due to involvement of the adjacent motor centres. The one most frequently affected is the speech-centre in the third left frontal gyrus, and as a result aphasia is a common associated symptom, particularly if the lesion is situated in the left hemisphere. The other motor centres may be involved and produce characteristic symptoms.

The functions of the *basal ganglia* of the brain are as yet insufficiently known to enable us to diagnosticate lesions situated in them with certainty. Lesions in the *lenticular nucleus* may be latent. In some cases

they appear to have produced sensory disturbances, but even this is doubtful. Ordinarily, the only symptoms they produce are those resulting from pressure upon the surrounding parts, such as the internal capsule. The *optic thalamus* appears to receive fibres from many parts of the cortex. Its relation to the fillet has already been mentioned, and lesions in this region frequently produce sensory disturbances. The pulvinar appears to be one of the three basal ganglia associated with the optic tract, and when it is destroyed there is usually bilateral or contralateral hemianopsia. There is some doubt, however, whether this is not due to involvement of the neighboring structures, either the fibres of the optic tract passing just beneath it or of the geniculate bodies. Nothnagel and von Bechterew have called attention to the fact that certain specialized movements on the part of the muscles of the face, particularly those concerned in the expression of the emotions, are more or less completely abolished by destruction of the optic thalami. The existence of this

FIG. 64.



Relative location of the nuclei of the different cranial nerves. (EDINGER.)

mimic paralysis has, in a few cases, led to the correct diagnosis of thalamic lesion. The *anterior corpora quadrigemina* apparently form one of the intermediate stations for the optic tract, the fibres from the nerves ending in them, and new neurons commencing that possibly form the fibres of the optic radiation. They are apparently the situations in which the arch of the pupillary reflex is completed. The *internal geniculate ganglia* and the *posterior corpora quadrigemina* appear to be associated with hearing.

Lesions in the *pons* and *medulla* produce, as a rule, characteristic symptoms that make it possible to locate them with considerable accuracy. This is due to the fact that the nuclei of nine cranial nerves are situated in these two portions of the brain, and also that they form the great source of communication between the cerebrum and the spinal cord, containing both motor and sensory fibres. (See Fig. 64.) The nucleus of the oculomotor nerve is found surrounding the anterior portion of the aqueduct of Sylvius, just beneath the anterior corpora

quadrigemina. Numerous groups of cells have been separated which are supposed to belong each to a different muscle. Destructive lesions cause partial or complete ophthalmoplegia, according to the extent of the lesion. There is, therefore, abolition of the pupillary reflex. Just behind the nucleus of the third nerve, and beneath the posterior quadrigemina, is a small group of cells for the pathetic nerve. The nucleus of the trigeminus is situated in the anterior portion of the pons, just to the outer side of the fillet, the motor group of cells lying inside the sensory group. The Gasserian ganglion receives the peripheral branches of this nerve and corresponds to the spinal ganglia. In addition the nerve receives a bundle of fibres from the lower portion of the medulla. Disturbances of the nucleus produce anæsthesia on the same side of the face, including the conjunctiva and the mucous membrane of the mouth. There is loss of taste in the anterior two-thirds of the tongue, and there is some disturbance of smell in the nostril on the same side. The pterygoid muscles are paralyzed and mastication is imperfect. Irritative lesions cause tic douloureux. This may also be the result of disease of the ganglion. The nucleus of the *abducens* lies in the posterior portion of the pons, just beneath the floor of the fourth ventricle. Destructive lesions cause internal strabismus. The nucleus of the *facial nerve* is found in the posterior portion of the pons, lying slightly behind and to the median side of the nuclei for the trigeminus. The fibres from this nerve pass out first forward, then downward and backward, and emerge from the lateral surface of the medulla at its anterior extremity, passing forward over the pontine cerebellar peduncles. Destructive lesions cause paralysis of the same side of the face, usually involving the upper branch. (See Hemiplegia.) Irritative lesions cause facial tic. The nucleus of the *acusticus* is found in the anterior portion of the medulla oblongata, just beneath the floor of the fourth ventricle, lying immediately above the superior olivary body. Lesions produce nerve or mental deafness on the same side. The nuclei of the *vagus* and the *glossopharyngeal nerves* are apparently in the jugular and petrosal ganglia—that is to say, they are sensory nerves, and correspond to the sensory fibres entering the spinal cord. From these ganglia fibres pass into the medulla oblongata at its lateral aspect, and end in a nucleus in the floor of the fourth ventricle. The motor nucleus of the vagus is supposed to be the nucleus ambiguus, situated just posteriorly to the olive in the posterior portion of the floor of the fourth ventricle. Close to the median line is the *hypoglossal nucleus.* Its destruction produces paralysis and degenerative atrophy of the corresponding side of the tongue.

The functions of the *pons* are merely those of the centres and tracts it contains, and therefore the symptoms are dependent upon the situation, and greater or less amount of destruction, that the lesions produce. On account of the decussation of the central fibres for the facial nerve in this region, crossed paralysis is usually considered pathognomonic of pontine disease. The functions of the *medulla* are also largely dependent upon the nuclei and tracts it contains. As it contains the centres for the pneumogastric and some of the centres or tracts of fibres for respiration, lesions in it are usually very promptly followed by death. Lesions of

the *restiform bodies*—that is, the lower portion of the medullary peduncle to the cerebellum—are frequently associated with nystagmus, and may cause the symptoms of cerebellar ataxia. As the medulla contains the nuclei of the motor nerves to the pharynx, larynx, and mouth, paralysis of the muscles in this region is spoken of as bulbar palsy.

The *cerebellum* is supposed to be concerned in co-ordination and the maintenance of the equilibrium. The hemispheres may, however, be extensively diseased without giving rise to any symptoms. If the middle lobe is affected, the characteristic manifestations are disturbance of equilibrium and inco-ordination. The gait resembles that of a drunken man. The patient often manifests a tendency to fall to one side, or forward or backward; sometimes there are distinct rotary movements. Nystagmus is frequent, especially in cases of tumor; and choked disks appear early. Giddiness and vomiting sometimes occur, but are, however, of no localizing value. The knee-jerk is often absent, but sometimes increased and sometimes variable. If the pyramidal tracts are pressed upon, it is always increased, and there is then weakness in the extremities. As a result of pressure there may be paralysis of the cranial nerves, difficulty in articulation, and occasionally epileptiform convulsions. If the medullary peduncle is affected by an irritative lesion, quite characteristic symptoms result. There are forced movements—that is to say, the patient may have an irresistible tendency to fall toward or lie upon the side. There are no symptoms diagnostic of disease of the superior or middle peduncles. Disease of one side of the pons may cause symptoms similar to those of cerebellar trouble.

Localization of Spinal Lesions.

The spinal cord may be regarded in two ways: first, as the pathway between the peripheral nervous system and the brain, containing the tracts running from the brain to the motor nerves, and from the sensory nerves to the brain; second, as a number of groups of ganglion-cells arranged in horizontal layers or segments. These segments are usually classified according to the nerve-roots that spring from them. There are, therefore, eight cervical, twelve dorsal, five lumbar, and five sacral segments of the cord. The white matter of the spinal cord is divided into two regions: the anterolateral part, extending from the median fissure to the posterior horns, and the posterior part, lying between the posterior horns. The anterolateral part contains the motor fibres or pyramidal tracts, whose functions have already been described. In addition, there are certain fibres that pass downward, the functions of which are not certainly known. The gray matter of the cord is divided into the anterior and the posterior horns. It is composed of nerve-cells and nerve-fibres. The nerve-cells in the anterior horns form a large group, which send their axis-cylinders into the anterior roots, and comprise the peripheral motor neurons. In the posterior horns, in the dorsal region, there is a group of cells on the inner side known as the column of Clarke, which apparently has something to do with equilibration. Other cells with unknown definite functions are also found in the posterior cornua. The gray matter also contains a

large number of nerve-fibres, some of which pass transversely and apparently are concerned in reflex action; others ascend, and convey to the

TABLE OF MOTOR AND REFLEX FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD. MODIFIED FROM GOWERS, MULLER AND WICHMANN.

Segments.	Motor innervation.	Reflex centres.
C 1	Small rotators of head.	
2	Depressors of hyoid	
3	Diaphragm	
4	Platysma (?)	
5	Deltoid, Biceps, Coracobrachialis, Supinator longus, Spinati, Serratus major	Scaleni. Lev. ang. scapulæ. Cucullaris.
6	Pectoral. maj. (clav.)	Pronators, Triceps
7	Subscapularis, Flexors of wrist and fingers, Pectoralis (costal)	Extensors of wrist and fingers
8	Latissimus dorsi, Teres major	Muscles of hand, Extensors of thumb
D 1		
2		
3		
4		
5		
6	Intercostal muscles	
7		
8		
9	Abdominal muscles	Erectors of spine
10		
11		
12		
L 1	Quadratus lumborum	
2	Iliopsoas, Cremaster	Quadriceps
3	Sartorius, Pectineus, Adductors	
4	Gracilis, Obturator	Gluteal
5	Adductors, Flexors of knee	Extensors of foot, Tibialis anticus, Peroneal muscles, Perineal and anal muscles
S 1		
2	External rotators of thigh	
3		
4		
5		

Dilatation of the pupil, sensory part. (?)

Scapular.

Tendon-reflexes of the muscles of the arms.

Dilatation of pupil, motor part. (?)

Epigastric.

Abdominal.

Cremasteric.

Knee-jerk.

Gluteal reflex.

Achilles tendon reflex. Plantar reflex.

Centres for the bladder and rectum.

brain the sensations of pain, heat, and cold. Each segment of the cord innervates and receives sensory impressions from an approximately corresponding segment of the body, and contains the lower reflex arcs. The motor and reflex functions of the various segments are shown in the table on page 331, and the sensory functions in Plate III.

The following table exhibits the spinal segments involved for each particular muscle. It must be remembered that the limits for the innervation of the muscles are not fixed, but vary in different cases within considerable limits, in some instances as much as two segments. The table is prepared in accordance with the views of Wichmann.

Muscles of the posterior portion of the cranium	C. 1 and 2
Deep muscles of the neck (rectus capitis, etc.)	C. 1 and 2
Muscles of the hyoid bone	C. 1 to 3
Sternocleidomastoid	C. 3
Trapezius	C. 3 and 4
Scalenus anticus	C. 4 to 7
Scalenus medius	C. 3 to 8
Scalenus posticus	C. 6 to 8
Diaphragm	C. 3 to 5
Levator anguli scapulæ	C. 3 to 5
Subscapularis	C. 5 and 6
Teres major	C. 6 and 7
Teres minor	C. 5
Latissimus dorsi	C. 6 to 8
Serratus anticus	C. 5 to 7
Pectoralis major	C. 5 to D. 1
Subclavian	C. 5 and 6
Deltoid	C. 5 and 6
Coracobrachialis	C. 6 and 7
Brachialis anticus	C. 5 and 6
Biceps	C. 5 and 6
Muscles of the forearm	C. 7 to D. 1
Muscles of the hand	C. 8 and D. 1

Intercostal muscles, from the corresponding dorsal segments, excepting the last three, which are innervated respectively from the eighth, ninth, and tenth segments:

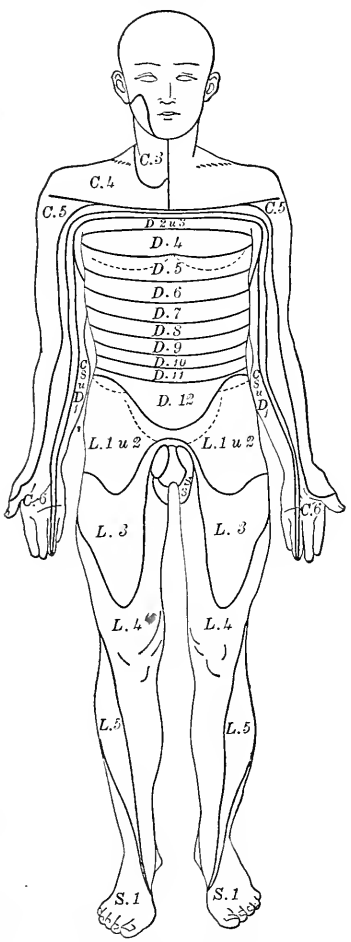
Iliopsoas	L. 1 to 3
Quadriceps extensor	L. 2 and 3
Sartorius	L. 2 and 3
Adductors	L. 4 and 5
Glutei	L. 2 to 4
Gemelli	L. 5 and S. 1
Obturators	L. 5
Semimembranosus, semitendinosus, and biceps	L. 4 to S. 1
Peroneal muscles	L. 4 to S. 1
Tibialis posticus, soleus, gastrocnemius, popliteus, and plantaris	L. 4 to S. 2
Muscles of the foot	S. 1 and 2
Muscles moving the toes	L. 5 to S. 2

General Symptomatology of Lesions of the Brain.

Lesions of the brain may be irritative or destructive. The former, if affecting the motor tract, produce clonic spasms. If destructive, they produce paralysis without atrophy, and cause increase in the muscle-tone by removal of the influence of the superior arc and exaggeration of the reflexes. All these changes occur in the muscles of the opposite side of the body. Irritative lesions are most likely to be extracerebral—that is, pressing upon the cortex. Lesions in the brain-substance are usually

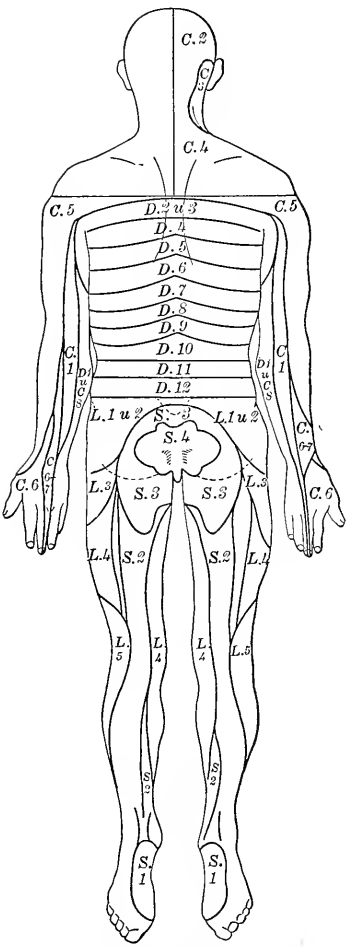
destructive, and therefore cause paralysis. As motor fibres are distributed over a considerable area of the cortex, lesions in this region, if circumscribed, are likely to cause at least monoplegia. If they involve the area for the face, the upper branch of the facial nerve, which is innervated from both sides, is rarely affected. Aphasia occurs only when the left side is diseased. Lesions in the corona radiata near the cortex

FIG. 65.



(From OPPENHEIM.)

FIG. 66.



(From OPPENHEIM.)

usually cause monoplegia ; if the lesion is near the internal capsule, hemiplegia is more common. Lesions in the internal capsule almost invariably cause hemiplegia. If the knee and anterior portion of the posterior limb are involved, hemiplegia without sensory changes results. When the posterior third of the posterior limb is involved, sensory disturbances are present, and there is likely to be hemianopsia. Lesions in the anterior portion of the anterior limb produce no recognizable symptoms, and are

termed latent. Increase of intracranial pressure may be brought about by new growths, traumatism, œdema, or inflammation. There are usually headache, delirium or coma, and vomiting. If the process is of slow development, a certain amount of adaptation may occur, and only the headache and vomiting may be present. The former is occasionally sharply localized. In addition, if the pressure be long continued, there is œdema of the optic nerve. (See Disorders of the Special Senses.)

General Symptoms of Disease of the Spinal Cord.

These depend upon the segment of the cord and upon the nerve-tracts involved. Lesions are spoken of as transverse if they involve the whole cord, unilateral if they involve but one side, and focal if they involve only a circumscribed portion.

Transverse Lesions of the Spinal Cord. Transverse lesions may be produced by inflammation or by pressure either of a tumor or as a result of deformity of the vertebral column (Pott's disease). Lesions above the fifth cervical segment usually cause death by paralysis of the diaphragm. If the patient survives, paralysis of all four extremities and total anæsthesia of the body remain. There are also paralysis of the bladder and rectum and abolition of the cutaneous reflexes, and in nearly all cases, of the tendon-reflexes. Transverse lesions between the fifth cervical and the first dorsal segments produce atrophy and degeneration of certain muscles of the arm, according to their situation. Spastic paralysis of the legs and total anæsthesia of the body as far up as the part that transmits sensation to the lowest intact segment; paralysis of the bladder and rectum; abolition of the reflexes whose arcs are found in the segment involved; and, if the destruction is not complete, exaggeration of all the tendon-reflexes that are completed in the lower segments are present. The cutaneous reflexes are abolished. Lesions of the dorsal region produce spastic paraplegia and paralysis of the bladder and rectum. The arms escape and respiration is not disturbed. The anæsthesia extends up to the segment involved. Lesions in the lumbar region produce atrophy and degeneration of certain groups of muscles in the legs, with paralyses and disturbances of sensation, distributed according to their extent. The situation of a lesion may be roughly determined by a study of the reflexes. If the lesion involves the segments concerned in any of these, they are of course abolished. If the lesion is above them, they are sometimes exaggerated; if below, they are ordinarily not involved. Lesions of the conus terminalis and the cauda, as they involve a large number of nerve-roots, produce a complexity of symptoms. There are irregular areas of anæsthesia corresponding to the posterior roots involved, and atrophy and degeneration of the muscles supplied by the anterior roots. The bladder and rectum usually are affected. If the lesion involves only the lowest roots, there is a characteristic saddle-shaped area of anæsthesia over the sacrum.

Wichmann gives the following table which represents the symptoms produced by total transverse lesion of the different segments of the spinal cord, commencing from below and ascending:

(M = motor disturbances; S = sensory disturbances.)

S. 5.
- M. Nothing.
- S. Anæsthesia in a small area over the coccyx.

S. 4.
- M. Paresis of the levator ani, sphincter ani, and of the detrusor urinæ.
- S. Small anæsthetic area over the lowest portion of the sacrum, about the border of the anus and the adjacent portions of the buttocks.

S. 3.
- M. Paralysis of the sphincter ani, levator ani, and detrusor urinæ, paresis of the rectum, constipation, retention of urine; later dribbling of urine, loss of ejaculatory power; erection still possible, but weaker. Cremasteric reflex preserved.
- S. Anæsthesia over the sacrum, the outer portions of the buttocks, the coccyx, the perineum, the anus, the posterior lower portions of the scrotum (labia) and the penis, and the uppermost portion of the posterior side of the thigh (saddle type). Testicles still sensitive.

S. 2.
- M. Paralysis of the levator ani and sphincter ani and of the detrusor urinæ. Loss of ejaculation and erection. Paresis of the external rotators of the thigh, of the gluteus maximus, of the biceps; difficulty in the plantar flexion of the foot (gastrocnemius and soleus), difficulty in standing on the toes; difficulty in elevating the inner border of the foot (tibialis posticus); paresis of all the small muscles of the foot.
- S. Anæsthesia over the coccyx, sacrum, the gluteal region, the anus and genitalia, and the posterior surface of the thigh as far as the knee. Hypæsthesia of the posterior middle surface of the leg in the region of the Achilles tendon, the lateral half of the sole of the foot, the lateral border of the small toe, and the back of the foot.

S. 1.
- M. Paralysis of the anus, bladder, and genitalia; external rotation of the thigh is difficult. The movements of the toes are imperfectly performed as the result of paralysis of the adductor hallucis, etc. Paresis of the external and internal rotators of the thigh; difficulty in bending the knees, difficulty in plantar flexion of the foot, in elevation of the inner border of the foot, in the dorsal flexion of the outer border of the foot, in extension and flexion of the toes.
- S. Anæsthesia as in S. 2, with the addition of the posterior and median surfaces of the leg, of the lateral half of the sole of the foot and the small toe; hypæsthesia of the external surface of the leg from the knee downward, of the median half of the sole of the foot and of the back of the foot; of the outer side of the anterior surface of the leg; loss of the Achilles tendon reflex, loss of the plantar reflexes.

L. 5.
- M. Bladder, rectum, genitalia as above. Paralysis of the external rotators of the thigh and of the flexors of the leg on the thigh. Extreme paresis of the internal rotators of the thigh, of the plantar flexors of the foot; paralysis of the flexors of the toes; paresis of the extensors of the toes, of the elevator of the inner side of the foot (tibialis anticus). Paralysis of the levators of the outer border of the foot (the perineus).
- S. Anæsthesia over the sacral and gluteal regions, the perineum, the genitalia, the posterior median surface of the thigh and leg, the posterior lateral surface of the leg, the region of the Achilles tendon, the sole of the foot, the back of the foot, and the external lateral surface of the leg and the knee.

L. 4.
- M. Bladder, rectum, and genitalia as above, paralysis of the lower extremities with the exception of the quadriceps extensor and the adductors, which are paretic.
- S. Anæsthesia as above, with the addition of the entire foot; hypæsthesia of the inner surface of the leg on the anterior and posterior sides, as well as the lower halves of the inner surfaces of the thighs.

L. 3.
- M. As above. The paresis of the extensors and adductors of the leg is more pronounced, and there is paresis of the flexors of the thigh. The leg usually is rotated externally, and it is impossible to stretch it.
- S. Anæsthesia as in L. 4; hypæsthesia of the anterior surface of the thigh, of the upper half of the inner surface of the thigh; slight hypæsthesia of the outer surface of the thigh as far as the trochanter major. Loss of the patellar reflex; ankle-clonus may persist.

L. 2.

M. Complete paralysis of all the muscles of the lower extremities with the exception of the psoas, which is markedly paretic.

S. Complete anæsthesia of the leg from the sacrum and from Poupart's ligament, with the exception of the region of the external cutaneous femoris and the region of the lumbo-inguinalis nerves, which are hypæsthetic. Patellar and tendo Achillis reflexes are absent; the cremasteric reflex is lost; the sensation of the testicle is lost.

L. 1.

M. Complete paralysis of all the muscles of the lower extremities, including the psoas.

S. Complete anæsthesia of the lower extremities; patellar reflex preserved or increased—lost in cases of total transverse lesion. Cremasteric reflex is lost. The tendo Achillis reflex is increased or lost.

D. 12 to D. 3.

M. In addition to the paralysis above mentioned there is paralysis of the muscles of the abdomen and the back. The higher the transverse lesion the more the proximal groups of the abdominal and back muscles are affected.

S. Complete anæsthesia of the lower limbs; also anæsthesia of the trunk bounded above by a horizontal line at the level of the spinous process of the vertebra, corresponding to the segment involved. As, however, there is a slight overlapping of the region supplied by adjacent segments, the area of anæsthesia is usually a little bit lower than would be anticipated, and in the lower portions of the abdomen there is a gradual change between two segments.

In total transverse lesion of the spinal cord the reflexes of the lower extremities are lost. In incomplete transverse lesion they are exaggerated. As the muscles of respiration are paralyzed, the function is accomplished entirely by the diaphragm, and there may be dyspnœa. This increases until the fourth cervical segment is reached.

D. 2.

M. As in D. 3.

S. Total anæsthesia bounded by a line at the level of the second interspace and the spinous process of the first dorsal vertebra; also an area of anæsthesia on the inner surface of the upper third of the arm.

D. 1.

M. In addition to the paralysis of the trunk and lower extremities, there are paresis of the muscles of the fingers—that is, the interosseal flexors—and slight weakness of the pronator quadratus. There is also paresis of the lower portions of the pectoralis major and minor.

S. Anæsthesia as in D. 2. Anæsthesia or hypæsthesia to the centre of the inner surface of the arm, and hypæsthesia along the ulnar side of the arm and forearm, the ulnar half of the hand. Disturbances of the pupil.

C. 8.

M. Paralysis of the trunk and lower extremities, loss of power to abduct the fingers, loss of power to adduct the thumb, flexion of the little finger difficult or impossible; paralysis of the interossei and of the lumbrical muscles. Flexion of all the muscles impaired. Ulnar flexion of the hand weakened, paresis of the extensors of the thumb and fingers. Difficulty in extending the arm. Paresis of the lower portion of the latissimus dorsi and of the pectoralis major and minor, and of the scalenus medius and posticus.

S. Anæsthesia as above, and in addition the whole of the ulnar surface of the arm, forearm, and hand, of the fifth, fourth, and third fingers on the back, and of the fifth and fourth fingers on the palm. In total transverse lesions there are disturbances of the pupil which are occasionally also present if the roots alone are involved.

C. 7.

M. Paralysis of the lower extremities and trunk, and of the flexors of the fingers, of the flexors of the hand, of the small muscles of the hand, and the pronator quadratus. Movements of the thumb and extension of the fingers are still possible, but weak. Supination of the forearm is possible; flexion and extension at the elbow are very weak. Adduction and backward movement of the arm are difficult. There is commencing paralysis of the serrati, and wing-like scapulæ.

S. As above, and in addition, the whole of the inner half of the arm and forearm and hand. Hypæsthesia of the radial side of the hand and of the radial side of the arm and forearm. The arm-reflexes are lost.

C. 6.

M. As above, and in addition paralysis of the muscles of the fingers, including the thumb, of the extensors of the forearm, of the adductors of the arm; paresis of the flexors of the forearm, of the elevators of the arm (deltoid). Difficulty in turning the head (scalenus and splenius and the deeper muscles of the neck).

S. As above, and in addition complete anæsthesia of the hand and forearm and of the flexor and extensor surfaces of the arm. Hyperæsthesia of the region supplied by the axillary nerve in the arm and shoulder. The reflexes of the arm are lost. Death usually occurs in a few days or weeks.

C. 5.

M. Paralysis as above, with complete paralysis of the upper extremities, the only motion remaining being slight elevation of the shoulder-blade. Rotation and bending of the head are difficult. There is dyspnœa on account of paresis of the diaphragm, either on account of the origin of the phrenic from the fifth segment, or because there is œdema of the fourth segment.

S. Complete anæsthesia of the body, bounded by a transverse line around the lower portion of the neck. Death occurs in a few hours or days.

C. 4 to 1.

Total transverse lesion in this area causes immediate death as the result of the bilateral paralysis of the diaphragm. In case of unilateral lesion life may persist, and there are, in addition to the symptoms above, paralysis of the trapezius and sternocleidomastoid. The anæsthesia, of course, is bounded by a transverse line at a higher level, and there may be areas of anæsthesia in the face and scalp.

Unilateral Lesion of the Spinal Cord (the syndrome of Brown-Séquard). This produces paralysis of the same side and anæsthesia of the opposite side, both symptoms extending as far upward as the region controlled by the segment that has been affected. Disturbance of sensation is not total. On the side opposite the lesion, tactile, pain, and temperature sense are lost, but the muscular sense persists. On the same side as the lesion there are loss of the muscular sense and complete paralysis. Atrophy and degeneration occur in the muscles supplied by the involved segment; below this there is spastic paralysis, with increase in the reflexes. Above the paralytic area there is a zone of hyperæsthesia, the reason for which has never been satisfactorily explained. The commonest cause of unilateral lesion is traumatism, particularly bullet and stab wounds. Occasionally the symptoms develop in the early stages of syringomyelia or as a result of tumor or hemorrhage of the spinal cord. *Focal lesions in the spinal cord* produce various symptoms, according to their situation. Inflammations involving the gray matter are commonly spoken of as poliomyelitis. They usually attack the anterior cornua and involve only the peripheral motor neuron—that is, they produce paralysis, atrophy, and degeneration of the muscles. Inflammatory lesions in the white matter are spoken of as leukomyelitis. They produce various symptoms according to the tracts they involve.

The Cranial Nerves.

The olfactory, optic, oculomotor, pathetic, abducens, auditory, and glossopharyngeal nerves have been described in connection with the special senses. The *trigeminal nerve* takes its origin from the centres in the pons and medulla already described. Destructive lesions of the motor portion cause paralysis of the pterygoid muscles. If they are unilateral, it is impossible for the patient to move the mouth toward the

opposite side when the lower jaw is protruded. The soft palate is sometimes flattened on the affected side, and occasionally the uvula deviates to the sound side. It is to be assumed that atrophy and degeneration of these muscles occur, but it is practically impossible to test their electrical reactions. Irritative lesions produce cramp known as *trismus*. It is, of course, usually due to central disease. The sensory portion of the trigeminus supplies the skin of the face and the mucous membranes of the cavities of the head. The distribution of the three branches is shown in Fig. 67. Irritative lesions produce tic douloureux; destructive lesions, anæsthesia in the distribution of the part affected. The *Gasserian ganglion*, situated in the floor of the middle fossa of the skull, is the ganglion of the sensory portion of the nerve, and corresponds to a spinal ganglion. Irritative lesions may cause facial neuralgias, usually associated with disturbance of the various secretions, such as the tears, saliva, perspiration, or vasomotor phenomena. Destructive lesions (operative removal) produce, in addition to the areas of anæsthesia, various trophic lesions, particularly ulceration of the cornea, or chronic ulcers in the mucous membrane of the mouth. There is usually diminished secretion of tears and of saliva, although occasionally a paralytic increase of the latter may occur. The *facial nerve* arises from the nuclei in the posterior portion of the pons. These are probably double, each supplying a separate branch of the nerve, and the superior nucleus is innervated from both sides of the cerebrum. It is the motor nerve for the muscles of the face, and supplies the temporal, masseter, the orbicularis palpebrarum, the muscles of the lower part of the face, the muscles of the palate, and the platysma myoides. Unilateral destructive lesions produce paralysis of the muscles of the face (Bell's palsy). This can be recognized by disappearance of the folds, drooping of the corner of the mouth, and inability to close the eye. In addition there may be loss of taste and hyperacusis in the ear on the same side. Occasionally there is deviation of the tongue, the palate is oblique, and the uvula is pulled toward the sound side. Secretion of saliva on the same side is diminished or abolished. This may be tested on the sublingual glands by raising the tip of the tongue, carefully drying the sublingual space, and causing the patient to inhale some pungent substance, such as acetic acid or musk. The saliva will immediately appear on the sound side, but will fail to appear on the other. If the peripheral portion of the nerve is involved, usually both the upper and lower branches are affected, and the paralysis is general. If the lesion is intracranial, other cranial nerves, especially the auditory, are likely to be involved. The muscles give the characteristic reactions of degeneration. If the lesion is central, the upper branch commonly escapes, or, at least, instead of being paralyzed, is only paretic. Moreover, in central lesions lying above the pons, there is also hemiplegia. In facial paralysis it is almost impossible for the patient to masticate on the diseased side, because the food collects between the cheek and the gums. It is also impossible for him to whistle. Saliva freely dribbles from the drooping corner of the mouth, and as it is impossible to contract the orbicularis palpebrarum, the eye remains open even in sleep (*lagophthalmos*); the corneal reflex is abolished or imperfect. When the patient

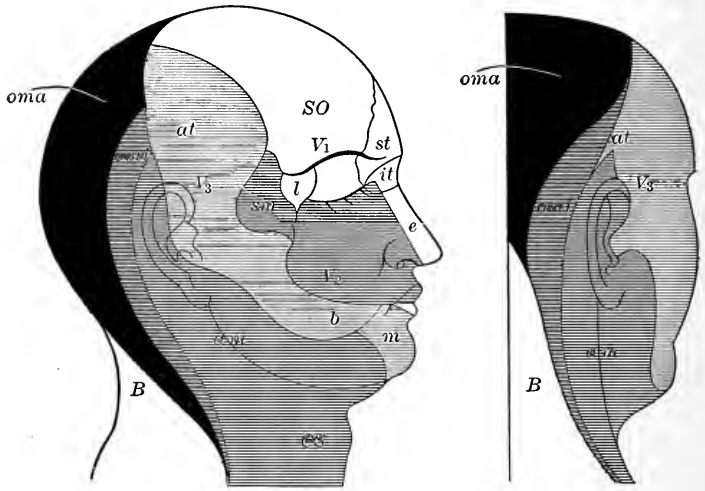
attempts to close the eye, the ball rolls upward and outward. In addition, the palatine reflex also disappears. If the paralysis is of long standing, contractures may occur. Irritative lesions of the facial nerve cause spasm of the facial muscles, usually spoken of as facial tic. The *vagus* nerve supplies motor fibres to the larynx, sensory fibres to the lungs, and inhibitory fibres, probably sensory in nature, to the heart. This nerve also probably sends sensory fibres to the gastro-intestinal tract. Destructive lesions of the vagus produce, if unilateral, paralysis of one vocal cord, interference with deglutition, and transient tachycardia. The laryngeal changes are most characteristic. (See Chapter V., Part II.) Irritative lesions produce spasm of the glottis, with dyspnœa or aphonia. The *spinal accessory* nerve is the motor nerve for the trapezius and part of the sternocleidomastoid. Destructive lesions of this nerve are the chief cause of torticollis. The *hypoglossal* nerve is the motor nerve for the tongue, and is, therefore, concerned in chewing, swallowing, and speaking. Unilateral destructive lesions produce paralysis of one-half of the tongue, which is protruded toward the paralyzed side, with atrophy and degeneration of the muscle. Fibrillary twitchings are usually present. The functional disturbance, however, is slight, and the patient may complain of no discomfort. Bilateral paralysis produces, however, very severe symptoms. The tongue lies flaccid in the mouth; it is impossible to protrude it, or even to move it from side to side. Mastication is impossible and swallowing exceedingly difficult. Speech is at first seriously affected, but, as a rule, the patient in time learns to compensate the lingual palsy. Paralysis of the tongue as a result of central lesion almost never occurs.

General Diagnosis of Nervous Diseases.

In the study of a nervous disease, it is necessary to follow some fixed plan from the beginning of the investigation, otherwise the obscure nature of many of the symptoms will cause them to be overlooked, and their omission may make the diagnosis difficult or impossible. It is true, of course, that in actual practice diseases will be met in which the clinical symptoms are so characteristic that the diagnosis can be made almost by inspection alone, and a prolonged examination will only be useful for the purpose of excluding or detecting possible complications. On the other hand, certain cases will occur that almost defy diagnosis on account of the multiplicity and apparently contradictory character of the symptoms. In general it may be said that, aside from the history and the subjective symptoms, the physician will meet with four groups of signs: disturbances of intelligence, disturbances of sensation, disturbances of motility, and atrophic and degenerative lesions.

1. Disturbances of Intelligence. If the patient is comatose, delirious, or maniacal, the condition can usually be recognized by simple inspection. The more obscure forms of insanity are often difficult to elucidate, and can only be detected by prolonged observation of the patient. It is desirable in these cases to find out whether the patient is capable of realizing his identity and comprehending his surroundings; thus, he should be asked his name and age, the date, and his location.

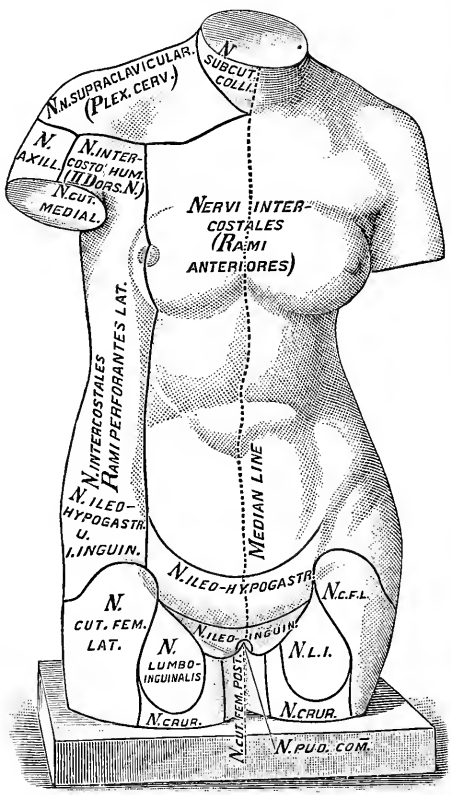
FIG. 67.



Cutaneous nerves of the head and face.

V_1, V_2, V_3, first, second, and third branches of the trigeminus; *SO*, supraorbital; *l*, lachrymal; *st*, supratrochlear; *it*, infratrochlear; *e*, ethmoidal; *sm*, malar; *at*, auriculotemporal; *b*, buccinator; *m*, mental; *am*, auricularis magnus; *oma* and *omi*, occipitalis major and minor.

FIG. 68.

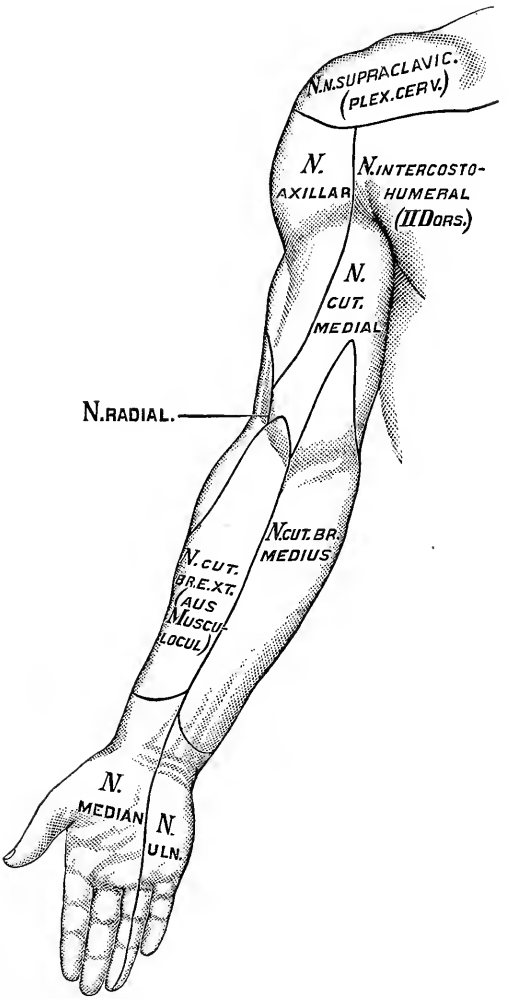


Cutaneous nerves of the anterior surface of the trunk. (SAHLI.)

Memory may be tested by requesting him to describe some recent or more remote event, to give the names and occupations of various friends and relatives. The history and the behavior of the patient may exclude intellectual disturbance altogether.

2. Disturbances of Sensation. There is first obtained a rapid orientation of the sensory condition of the patient. For this purpose it is

FIG. 69.

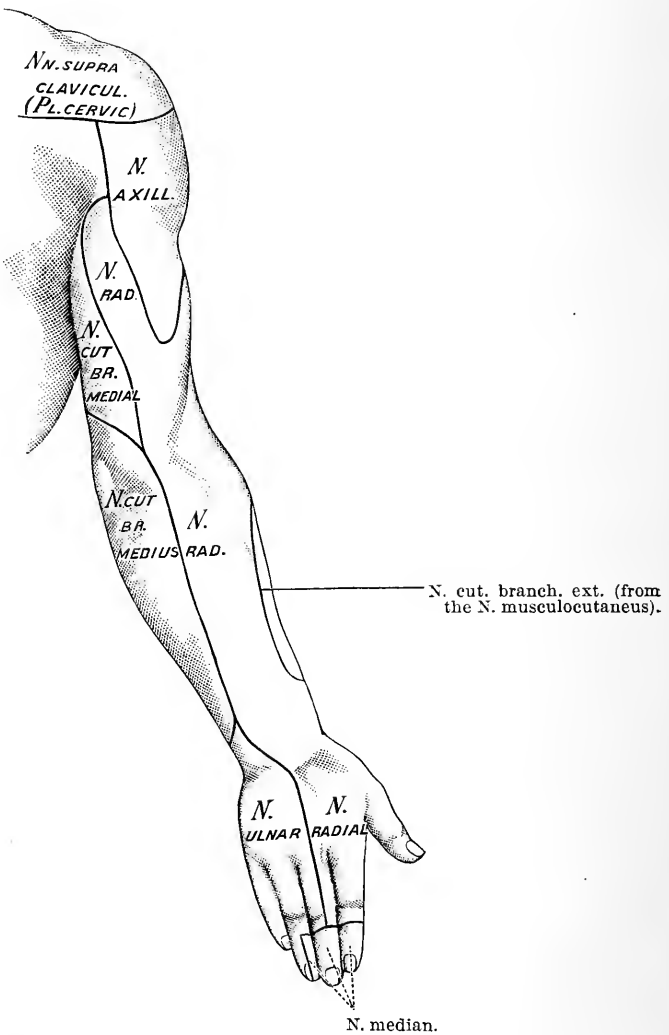


Cutaneous nerves of the anterior surface of the arm. (SAHLI.)

customary to touch with the finger or a blunt object both sides of the face, the arms, the legs, and both sides of the body. If the patient declares that there is no difference in the sensory perceptions, tactile anæsthesia may be temporarily excluded. The same regions are tested for pain and temperature sense, and it is often desirable to test the muscle sense at the same time, although this properly belongs to disturbances of

motility. It is often possible, in testing sensation, to decide whether the lesion is peripheral or central by its distribution. For this purpose it is usually most satisfactory to imagine the body placed upright upon the ground with the arms and legs extending laterally at right angles to the trunk. Sensory disturbances due to cerebral lesions will be bounded by

FIG. 70.



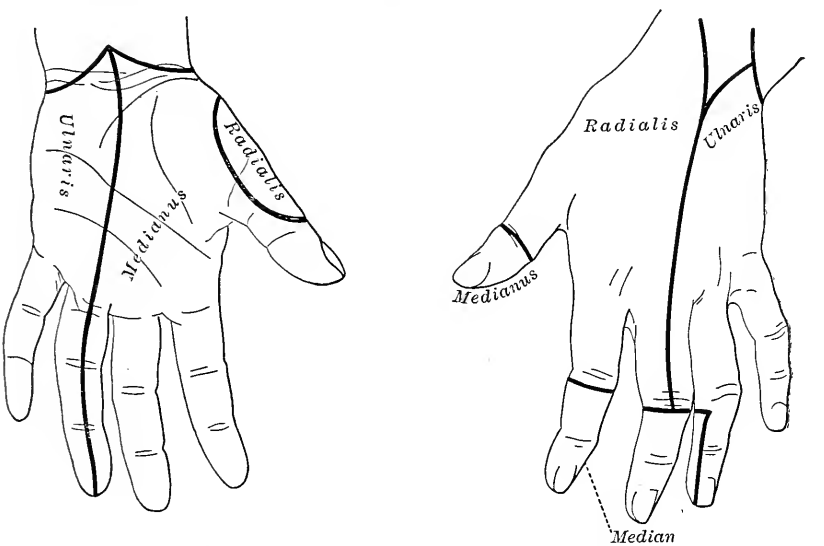
Cutaneous nerves of the posterior surface of the arms. (SAHLI.)

lines parallel to the spinal column—that is to say, there will either be hemianæsthesia or anæsthesia of the limbs bounded by planes passing through them perpendicularly (the glove or stocking type). Sensory disturbances due to spinal lesions will be bounded by lines perpendicular to

the long axis of the body—that is, horizontal lines passing around the body or extending from the shoulders or hips along the limbs, so that in the spinal or segmental type of anæsthesia, the areas of disturbance form long strips upon the limbs or belt-like bands around the body. These statements are not, however, to be taken too absolutely, as the areas of sensory disturbance are apt to be variable. (See Plate III.) If the lesion affects the peripheral nerves, the area or areas will correspond to the cutaneous distribution of the nerve or nerves involved. (See Fig. 67 *et seq.*)

3. **Disturbances of Motion.** It is well to study first the more patent alterations. Thus the patient should be told to move the arms and legs, in order to detect paralyses; he should be requested to walk, in

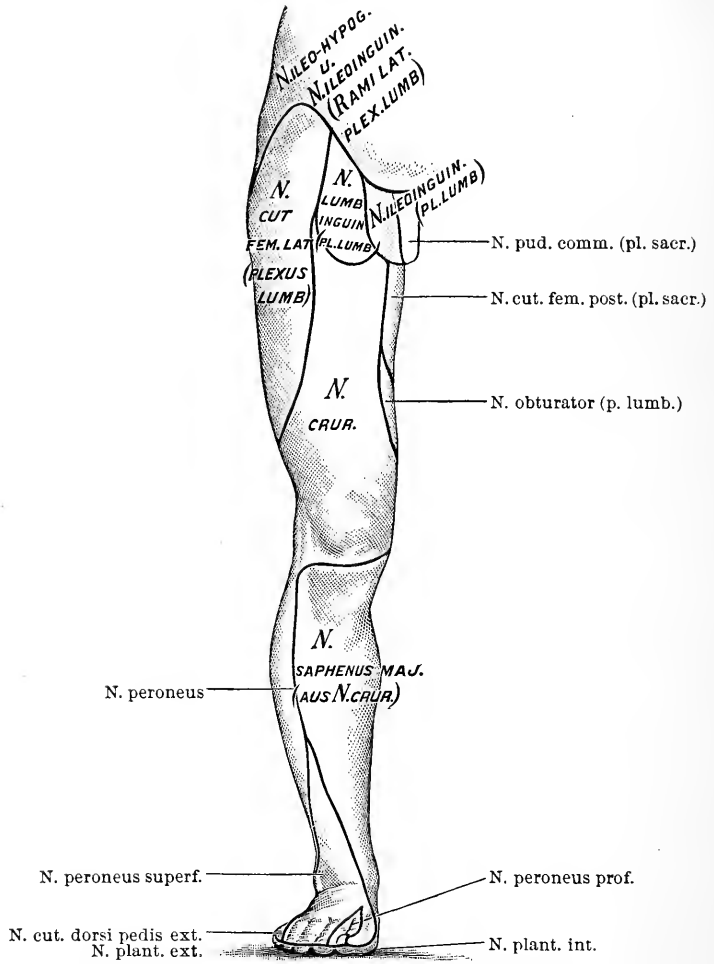
FIG. 71.



Distribution of the cutaneous nerves in the hand.

order to study the gait; he should be directed to perform some delicate, co-ordinated movement, in order to detect possible ataxia; and to put the muscles in a state of tension, in order to exaggerate a possible tremor. Following this the individual movements should be carefully examined. It must be remembered that, whether the lesion is in the central or peripheral nervous system, disturbance of motility is manifested only in the muscles themselves, and the investigation, therefore, should commence with these—that is to say, it is not desirable to test the motor functions of each particular nerve, but rather of each particular group of muscles, and to deduce from the changes found in them the nerve or segment involved. The following table from Sahli gives a classification of the muscles of the extremities, according to their functions, with their nerve-supply:

FIG. 72.



Cutaneous nerves of the anterior surface of the leg. (SAHLI.)

TABLE OF THE VOLUNTARY MUSCLES GROUPED ACCORDING TO THEIR FUNCTIONS, WITH THEIR NERVOUS SUPPLY. (SAHLI.)

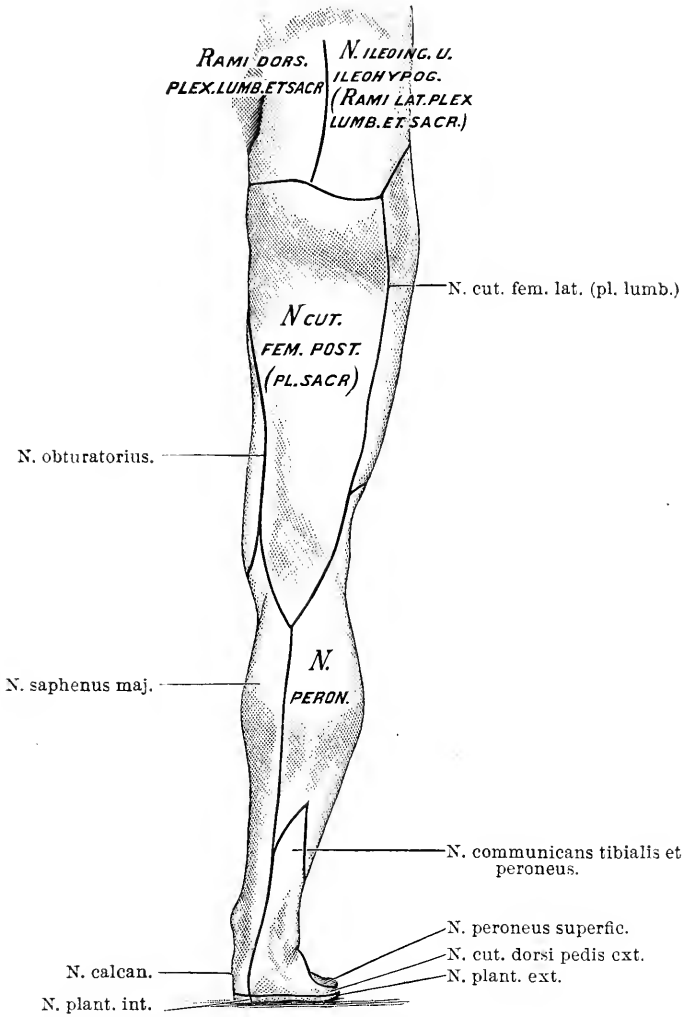
Upper Extremity.

A. MOVEMENTS OF THE SHOULDER-BLADE.

1. *Elevators of the shoulder.*
   Middle part of the cucullaris (N. accessorius).
   Rhomboidei (N. dors. scapul., 5th cervical nerve).
   Levator scapulæ (2d and 3d cerv. nerv. and N. dors. scap.).
   Upper portion of the pectoral major (Nn. thorac. ant., 5th and 6th cerv. nerves).

2. *Depressors of the shoulder.*
   Pectoralis minor (Nn. thorac. anterior).
   Lower portion of the latissimus dorsi (N. subscapularis).
   Lower portion of the pectoralis major (N. thorac. ant.).

FIG. 73.



Cutaneous nerves of the posterior surface of the leg. (SAHLI.)

3. *Adduction of the shoulder.*
   Lower portion of the cucullaris (N. accessor.).
   Upper portion of the latissimus dorsi (N. subscapularis).
4. *Abduction of the shoulder.*
   Upper third of the pectoralis major (N. thor. ant.).
   Serratus anticus major (N. thorac. longus, 6th, 7th, 8th cerv. nerv.).

B. MOVEMENTS OF THE SHOULDER-JOINT.

1. *Elevators of the arm.*
   (*a*) Laterally, deltoid (N. axillaris).
   Vertically, serratus anticus major (N. thorac. longus).
   (*b*) Anteriorly, anterior portion of the deltoid (N. axillaris).
   Coracobrachialis (N. musculocutaneous).
   Biceps (N. musculocutaneous).
   (*c*) Posterior portion of the deltoid (N. axillaris).

2. *Adduction of the arm.*
   Pectoralis major (N. thorac. anticus, 5th and 6th cerv. n.).
   Latissimus dorsi and teres major (N. subscapularis).
   Infraspinatus (N. suprascapular, 5th and 6th cerv. n.).
   Teres minor (N. axillaris).
   These muscles also depress the arm.
3. *Internal rotation.*
   Subscapularis (Nn. subscapulares).
4. *External rotation.*
   Infraspinatus (N. suprascapularis).
   Teres minor (N. axillaris).

C. Movements of the elbow.

1. *Flexion.*
   Biceps (N. musculocutan.).
   Brachialis (N. musculocutan.).
   Supinator longus (N. radialis).
2. *Extension.*
   Triceps (N. radialis).
3. *Supination.*
   Supinator brevis, Supinator longus } (N. radialis).
4. *Pronation.*
   Pronator quadratus, Pronator teres } (N. medianus).
   Supinator longus (N. radialis).

D. Movements of the wrist-joint.

1. *Flexion.*
   Flex. carpi radialis (N. medianus).
   Flex. carpi ulnaris, Palmaris longus } (N. ulnaris).
2. *Extension.*
   Extensor radialis longus and brevis, Extensor ulnaris } (N. radialis).
3. *Abduction.*
   Flexor carpi radialis, Radialis longus and brevis } (Nn. medianus, radialis).
4. *Adduction.*
   Extensor ulnaris and flexor carpi ulnaris (Nn. radial., ulnar.).

E. Movements of the fingers.

1. *Flexion.*
   Flexor digitor. sublim.; flexion of the 2d phalanx (N. medianus).
   Flexor digitor. prof.; flexion of the terminal phalanx (Nn. medianus, ulnar.).
   Interossei and lumbrical muscles, flexion of the proximal phalanx (Nn. ulnaris, medianus).
2. *Extension.*
   Extensor dig. comm. (N. radialis).
   Interossei and lumbrical muscles (Nn. ulnar, medianus).

F. Movements of the thumb.

1. *Flexion.*
   Flexor pollicis longus and brevis (N. medianus).
2. *Extension.*
   Extensor pollicis longus and brevis (N. radialis).
3. *Abduction.*
   Abductor pollicis long. (N. radialis).
   Abductor pollicis brev. (N. medianus).

4. *Adduction.*
   Adductor pollicis (N. ulnaris).

5. *Opposition.*
   Opponens pollicis
   Adductor pollicis brev. } (N. medianus).

G. Movements of the little finger.

1. *Flexion.*
   Flexor communis digitorum profundus and sublimis (Nn. medianus, ulnaris).

2. *Extension.*
   Extensor minimi digiti proprius (N. radialis).

3. *Abduction.*
   Abductor minimi digiti (N. ulnaris).

4. *Opposition.*
   Opponens minimi digiti (N. ulnaris).

Lower Extremity.

A. Movements of the hip-joint.

1. *Elevation of thigh.*
   Iliopsoas (Nn. plexus lumbalis).
   Rectus femoris
   Sartorius } (N. cruralis).

2. *Depression of thigh.*
   Glutæus maximus (Nn. glut. inf. and ischiadicus).
   Flexors of the knee (N. ischiadicus).

3. *Internal flexion.*
   Glutæus med. and minim. (N. glut. super.).

4. *External rotation.*
   Quadratus femoris
   Obturator int. and Gemelli } (N. ischiadicus).
   Obturator ext. (N. obturat.).
   Pyriformis (Plex. ischiad.).
   Iliopsoas (Plex. lumbal.).
   Glutæus max. (N. glutæus inf.).

5. *Adduction.*
   Adductores (N. obturator).
   Pectineus (Nn. crural. and obturat.).
   Gracilis (N. obturator).

6. *Abduction.*
   Glutæus med. and min. (N. glut. sup.).

B. Movements of the knee-joint.

1. *Flexion.*
   Sartorius (N. cruralis).
   Gracilis (N. obturat.).
   Semitendinosus
   Semimembranosus } (N. ischiad.).
   Biceps
   Popliteus (Nn. tibial., ischiad.).

2. *Extension.*
   Quadriceps (N. cruralis).

C. Movements of the ankle-joint.

1. *Dorsal flexion.*
   Tibialis anticus
   Extensor commun. dig. long. } (N. peron. prof.).

2. *Plantar flexion.*
   Gastrocnemius, Soleus } (N. tibialis).
   Peroneus long. (N. peron. superficial.).
3. *Adduction.*
   Tibialis postic. (N. tibialis).
   Tibialis ant. (N. peron. prof.).
4. *Abduction.*
   Peroneus longus, Peroneus brevis, Extens. comm. dig. long. } (N. peron. prof.).
5. *Elevation of the inner side of the foot.*
   Tibialis ant. (N. peron. prof.).
   Tibialis post. (N. tibialis).
6. *Elevation of the outer side of the foot.*
   Peroneus long. and brev., Peroneus tertius } (N. peron. superf.).

D. MOVEMENTS OF THE TOES.

1. *Flexion.*
   Flexor comm. digit. long. and brev., Interossei and lumbricales } (N. tibialis).
2. *Extension.*
   Extensor comm. digit. long. and brev. (N. peron. prof.).
3. *Adduction.*
   Interossei plantares (N. tibialis).
4. *Abduction.*
   Interossei dorsales (N. tibialis).

E. MOVEMENTS OF THE GREAT TOE.

1. *Flexion.*
   Flexor hallucis long. and brev. (N. tibialis).
2. *Extension.*
   Extensor hallucis long. and brev. (N. peron. prof.).
3. *Adduction.*
   Adductor hallucis (N. tibialis).
4. *Abduction.*
   Abductor hallucis (N. tibialis).

F. MOVEMENTS OF THE SMALL TOE.

1. *Flexion.*
   Flexor minimi dig. (N. tibialis).
2. *Abduction.*
   Abductor minimi dig. (N. tibialis).
3. *Opposition.*
   Opponens minimi dig. (N. tibialis).

Each movement should be tested by requesting the patient to perform it first unimpeded, and then against resistance. (For functions of motor cranial nerves, see page 337.)

4. The **cutaneous trophic changes** have already been described. almost invariably indicate some lesion involving the peripheral neurons, or transverse lesion of the spinal cord.

Having obtained a rough idea of the condition of the patient, it is then necessary to make a more minute examination. Although no uniform plan can be used, the following order is often convenient: 1. The various special senses. These should be taken up in order and all their functions tested. 2. The reflexes, especially those of the eye, and the tendon and cutaneous reflexes of the body and extremities. 3. Position, station, and gait. 4. Disturbances of speech. 5. The condition of the individual muscles and nerves of the body. 6. Finally, a general physical examination to determine or exclude the existence of organic disease of the various organs. The diagnosis must then be made by the study of the symptoms elicited. It should, if possible, include both the situation and the nature of the lesion, although it is not always possible to make the latter.

CHAPTER XXXII.

THE TEMPERATURE.

CHILLS.

"CHILLS" vary from a passing "creep" or cold sensation, extending up and down the spine, to the "shake" or true rigor lasting a half hour or even longer. In infectious diseases the milder form is of as much significance as the more severe. The rigor may be so violent and prolonged as to terminate fatally. It must be distinguished from the algid stage of cholera and the coldness of collapse. The chill is attended by general tremor or shaking, chattering teeth, cold extremities, pallid face, often parched, blue lips and finger-tips. Notwithstanding the peripheral coldness and the extreme sensation of cold, the internal temperature rises, and may be 104° to 107° F.

Clinically, a chill or rigor marks the onset of a severe infection, as pneumonia. "Chills" are symptoms of some affections, as malaria. They are seen in the course of many diseases, as typhoid fever, tuberculosis, and septicæmia. In typhoid fever they disclose the occurrence of a secondary infection or a mixed infection; they may be due to antipyretic treatment by coal-tar remedies (Osler) or result from constipation. Endocarditis is attended by daily chills or they occur at irregular intervals. Pyæmia and septicæmia, purulent inflammation (infections), inflammations of the biliary or renal passages, stone in the biliary canal or the pelvis of the kidney (see Intermittent Fever) are frequently attended by chills. The morphine-habit gives rise to chills, with some fever.

FEVER.

In conditions of health the body temperature is maintained constantly at about 98.6° F. (37° C.). This stability of temperature is due to the central regulating apparatus called the thermotaxic mechanism which controls the production and the dissipation of heat. Fever is a condition characterized by an increase of temperature, usually with increased disintegration of nitrogenous tissue. The muscles and large glands, as is well known, are the chief seats of heat-production. Both heat-production and heat-dissipation are believed to be under the control of the nervous system, either through the motor nerves or through special nerves which pass with them to and from definite centres in the brain, called heat-centres, and in conditions of disease this thermotaxic mechanism may be altered. (1) There may be elevation of temperature from diminished dissipation of heat, though not necessarily increased nitrogenous disintegration and disordered function. Or (2) there may be increased production of heat with diminished dissipation; hence the temperature will

naturally be higher than if increased heat-production were accompanied by normal heat-dissipation. (3) There may be increased heat-production and at the same time increased heat-dissipation, in which case there would be the increased waste of fever with or without elevation of temperature. (4) It is possible that heat-dissipation may be greater than heat-production, or that the thermotaxic mechanism may be disturbed so as to promote loss, in which case there will be subnormal temperature.

Mode of Determination of Fever.

The temperature of the body can be roughly estimated by the sense of touch, but this method is open to many sources of error. The skin is at times hot, and gives a deceptive sensation of considerable elevation of temperature, whereas when tested with the thermometer the temperature is found to be but slightly or not at all above normal. So, too, when the skin feels cold and clammy in phthisis and during a chill from any cause, the actual temperature of the body is decidedly above normal, and may be as high as 103° or 104°. To insure accuracy, therefore, it is now almost the universal custom to employ clinical thermometers. They are of a convenient size and shape for insertion under the arm or into the mouth, rectum, or vagina. The better ones are provided with an indestructible index, so that the mercury in the capillary tube remains stationary at the highest level to which it rose when the thermometer was in the mouth or axilla. When the instrument is not provided with such an index, the reading must be made while the thermometer is still in position.

Thermometers vary in the accuracy with which they register temperature. The best ones are compared with an acknowledged standard, and sold with a slip of paper which gives their fractional variations from the standard. When the exact temperature is a matter of great importance, it should be taken in the *rectum* or *vagina*, where the temperature is more nearly that of the body. It is of advantage to take the rectal temperature in the case of children or comatose patients. This situation is also a good one to select when a bath is being administered. If possible, scybalous masses should be removed from the rectum. At least an incorrect reading may be obtained if the thermometer should happen to be plunged into the feces; this must be guarded against. From motives of delicacy, however, the axilla is to be preferred to the rectum and vagina on all ordinary occasions. The axillary temperature is somewhat less than a degree below that of the rectum. The temperature of the *mouth* is above that of the axilla and below that of the rectum. The mouth is preferable to the axilla, as it is more accessible and records the temperature more quickly and more accurately. Nevertheless, as the physician's thermometer is carried from patient to patient, some place should be selected which is less capable of absorbing disease germs than the mouth. The *axilla* is therefore by common consent the usual place for taking the temperature. Observe two precautions: (1) Before introducing the thermometer see that there is no undue moisture; if there is, the axilla should be wiped dry, otherwise a lower than a true reading will be obtained. (2) See that

the instrument is inserted into the armpit and does not project beyond the posterior fold, and that it is not caught in a fold of the undershirt or night-dress. After the thermometer is in position the arm should be brought gently across the chest and kept in that position until the instrument is withdrawn. The arm should not be held rigidly, as such muscular action increases the hollow of the armpit and may keep the sides apart, instead of in contact, as they should be to make a correct reading. The length of time required to take the axillary temperature will depend upon the instrument used; generally from five to eight minutes are required. Some very delicate thermometers register in one minute, but they are too fragile for ordinary use. If the index is in such a position that it can be seen, it is proper to withdraw the thermometer when the mercury has ceased to rise for two minutes.

The index, of course, must be shaken down to normal, or slightly below normal, before the thermometer is again ready for use; and the instrument must be carefully cleansed after use.

In children who are restless the temperature may be taken in the groin, as the folds of fat readily admit of completely enveloping the bulb of the thermometer. The height to which the mercury rises will correspond to the temperature of the axilla. The temperature of the *urine* corresponds exactly with that of the body, if taken when freshly passed and during the act, a method only applicable in the case of males. Sometimes this method of securing the temperature is resorted to, particularly in case of malingerers, when it is desirable to have the temperature taken in the physician's presence.

If the *mouth* is selected as the place in which the temperature is to be taken, care should be exercised that the thermometer is placed under the tongue, or along its side between it and the lower jaw, and retained in position by the lips of the patient. If the teeth are set firmly on the thermometer, it may be broken, or, what is of still greater importance, it will be tilted out of position and a correct reading will not be obtained. The lips should be closed and breathing be carried on through the nostrils. Four to seven minutes is sufficient time to allow it to remain in position. The patient should not have taken ice or anything cold prior to the observation.

Observations of the temperature should be made at least twice a day, in the morning and evening, and, as far as possible, at the same hour on successive days. It is frequently desirable to have the temperature taken every two or three hours, and sometimes at more frequent intervals. This is particularly the case, if observations of the indications for, and the effect of, antipyretic treatment are to be made.

In obscure cases the observations should be repeated at night as well as during the day. In this manner the presence of unsuspected tuberculosis may be revealed, or the occurrence of suppuration in some portion of the body definitely determined. It should not be forgotten, however, that the temperature may be taken too frequently for the patient's good, the disturbance of his needed rest being distinctly harmful.

As the general range of temperature and its diurnal variations are of more importance than the absolute temperature at any one time, ther-

mometers not perfectly accurate in their reading are still good enough for clinical and therapeutic purposes.

Physiological Variations of Temperature.

The temperature is subject to *physiological variations*. 1. It rises from 7 or 8 in the morning until 7 or 8 in the evening, at which time it reaches its maximum. It then begins slowly to fall, reaching its lowest point in the early hours of the morning, between 2 and 4. This *diurnal fluctuation* does not usually amount to more than 1 degree. 2. *Exercise*, etc. Violent exertion raises the temperature, and so does a heated atmosphere, cold having a contrary effect. 3. *Age*. In infants and young children, up to puberty, the temperature has a somewhat higher range, and is subject to greater variations than at a later period. In very old persons the temperature may be subnormal. The *normal* axillary temperature of adults is 98.6° F. The period in the twenty-four hours in which the temperature is at its lowest ebb is from 12 P.M. to 4 A.M. It may then be subnormal. The writer has known an over-cautious parent to make this physiological fall the subject of meddlesome observation and ill-judged treatment.

Pathological Variations of Temperature.

An elevation of temperature above the normal, not to be accounted for by external heat or severe exhaustion, may be considered febrile, and is pathological. The range of febrile temperature varies from above normal to 105° or 106° in ordinary cases. A range above 106° may occur, but it is not usually compatible with life. Certain terms have been applied to various degrees of temperature, to indicate in a general way the degree of fever :

Below	35°	Cent.	= 95.0°	Fah.	Very low or collapse temperature.
	36	"	= 96.8	"	
About	36½	"	= 97.7	"	Subnormal temperature.
Normal	37	"	= 98.6	"	Normal temperature.
About	37½	"	= 99.5	"	Slightly above normal or subfebrile temperatures.
	38	"	=100.4	"	
	38½	"	=101.3	"	
About	39	"	=102.2	"	Moderately febrile temperature.
	39½	"	=103.1	"	
About	40	"	=104.0	"	Highly febrile temperature.
	40½	"	=104.9	"	
Above	41	"	=105.8	"	Hyperpyretic temperature.

(From FINLAYSON.)

The Degree of Danger.

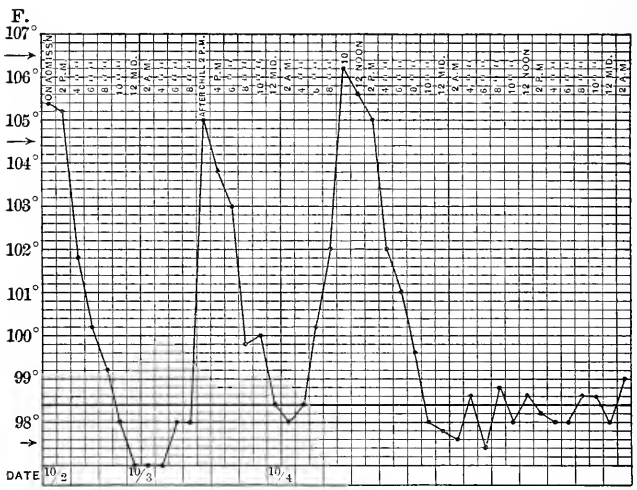
In general the degree of danger to the patient increases with the height of the fever, but the duration of the high fever modifies this greatly. A temperature of 106° on the second or third day of an acute lobar pneumonia is not rare, such cases frequently ending in recovery, while a temperature of 105° in the second or third week of typhoid fever is of much graver significance. Da Costa has reported a case of cerebral rheumatism in which the axillary temperature reached 110°, yet the patient recovered.

In a case of injury to the spine reported by Teale, the extraordinary temperature of 122° was recorded, and the temperature range for days was between 112° and 114°. The patient recovered.

The Types of Fever.

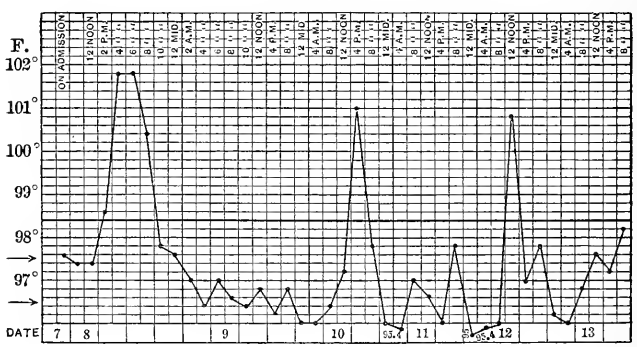
Fevers are divided, in accordance with the character of their range, into certain definite types. The types may be indicative of special processes. It is certain that the recognition of a peculiar type is a positive

FIG. 74.



Malarial intermittent fever, quotidian type. (Original.)

FIG. 75.

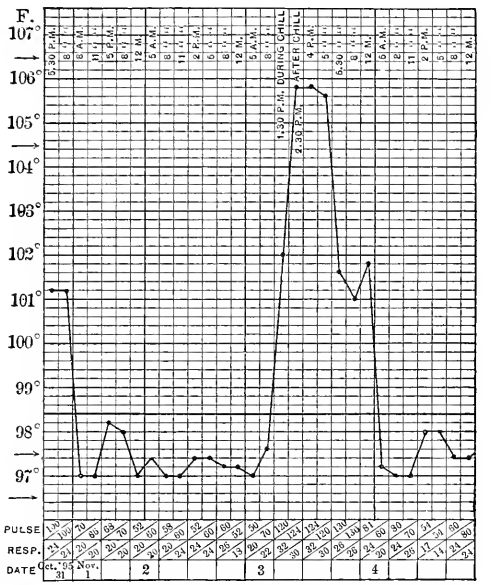


Malarial intermittent fever, tertian type. (Original.)

aid to diagnosis. Fever that continues for more than two days, in which the difference between the daily maximum and minimum of temperature is less than 2 degrees, is known as *continued* fever. (See Fig. 79.)

Fever existing more than two days, in which the daily difference is far greater than 2 degrees, is known as *remittent* fever. Further, a fever in which there is a rise of temperature followed by a fall to or below the normal, occurring periodically, is known as *intermittent* fever. The paroxysms may occur daily, every second or third day, or once a week.

FIG. 76.



Malarial intermittent fever, quartan type. (Original.)

When the paroxysms occur daily, the intermittent fever is of quotidian type (see Figs. 74 and 81); every second day, tertian type, one day intervening without fever (see Fig. 75); every third day, quartan type, two apyretic days intervening (Fig. 76).

The Course of the Fever.

Fevers frequently have a definite course, known as

(1) The initial stage;

(2) The fastigium;

(3) The period of defervescence.

During the *initial* stage the temperature rises higher each hour (or if extended over days, each day) than the preceding hour or day—in this latter instance, interrupted by the daily fluctuations. The stage may last from a few hours, as in a paroxysm of intermittent fever, to four or five days, as in typhoid fever. In this stage we have a chill such as characterizes the onset of an intermittent fever, or the recurrent chills or chilliness with headache and backache that attend the first four or five days of typhoid fever. During this stage, also, the heat-dissipation from the cutaneous surface is diminished and the total heat-dissipation is less.

When the hand is placed upon the patient the surface will be found to be cool, whereas the temperature in the mouth or rectum will be found to be far above the normal. The patient complains of the coldness or chilliness, and the low temperature of the surface is indicated by the shrunken hand and the pallid, pinched face. The peripheral arteries are contracted, and hence cause diminution in the amount of blood available for warming the skin and compensating for the loss by radiation and conduction. This peripheral contraction is the cause of the chilliness and the fall in the temperature of the skin.

The initial stage or onset may be sudden or gradual. 1. The onset may be *sudden* in acute diseases, as tonsillitis, pneumonia, and gastro-intestinal disorders of children, in erysipelas, and in intermittent fever, in all of which the rise of temperature is rapid. The maximum of temperature is reached within a few hours. (See Fig. 80.) 2. The mode of onset may be *gradual*. The initial stage is prolonged under these circumstances, as in cases of typhoid fever. (See chart of Typhoid Fever.)

During the second period of the course of pyrexia—the *fastigium*—the temperature of the body attains the highest point, and remains almost stationary, or may vary but a degree or two between maximum and minimum. It may last a few hours or from two days to three or more weeks, during which time it may oscillate to the maximum point of the first day. The temperature of the surface of the body is about the same as that of the deep parts, particularly in cases of pneumonia, measles, and scarlet fever. In typhoid fever, acute rheumatism, and phthisis, during the fastigium there may be a difference between the external temperature and the temperature taken in the cavities, as the mouth or rectum. More or less antagonism between heat-production and heat-loss exists under these circumstances. The latter may be greater than the former, if the skin perspires freely, as in rheumatism. The temperature then remaining high indicates that the production of heat must be proportionately increased, and hence far greater than in the cases in which the external and internal temperature are nearly the same. (See Fig. 77: the fastigium here occurs in the first three days. In Fig. 80 the fastigium lasts until the crisis.)

During the period of *defervescence* the temperature falls to the normal. In this period an attempt is made by the economy to return to a physiological state, in which heat-production and heat-loss are evenly balanced. The state of pathological pyrexia has come to an end. The termination may be by *crisis*. (See Figs. 75 and 80.) When this takes place, the perturbation of the thermotaxic mechanism must be very great, but the normal state is resumed at once. The crisis is also attended by copious perspiration, a "critical sweat," or by the passage of a large quantity of urine, and sometimes by several large liquid stools. The pulse-rate and respirations fall correspondingly with the temperature. (See Fig. 76.) In other cases the termination is by *lysis*—the temperature falls a degree or two each day until the normal is reached. (See chart of Typhoid Fever.) It seems that the thermotaxic mechanism of health is restored with difficulty. In this case the sweating is less marked, but may recur

for several days. The slowing of the pulse and respiration likewise take place gradually. (See chart of Typhoid Fever.) In some cases, in the period of defervescence, the aberrations are very remarkable, and it seems as if the thermotaxic mechanism which controls heat-loss is in a convulsive state. The temperature rises and falls irregularly, gradually resuming the normal only as the strength of the patient increases.

The defervescence may, however, occupy several days, in which case it is called *lysis*.

Diseases of sudden onset usually terminate with sudden decline, and conversely, in diseases with a prolonged onset the decline is also prolonged. Many cases which naturally terminate by crisis may end by lysis. This irregular termination is usually due to a complication. (See Fig. 77.) For instance, in measles, pneumonia is usually the causal complication, while in pneumonia it is empyema or endocarditis.

The Daily Range of the Prolonged Initial Stage and the Fastigium. The daily range of the temperature in fever generally corresponds to the normal variations—that is, the temperature is higher in the evening than in the morning. The difference in the daily range varies in the different types of fever; generally, as previously noted, the continued fevers show a smaller, the intermitting fevers a larger, difference between morning and evening temperature.

Sometimes there is *inversion* of the normal range. The evening temperature is lower than the morning; although a rare condition, this is of serious import. It is seen in the more severe cases of typhoid fever and occasionally in tuberculosis.

Recrudescence. In many cases the fever returns after the temperature has fallen to the normal. This may be due to a number of causes. Any perturbation of the nervous system from excitement, over-exertion, or loss of sleep; or an attack of indigestion, may send the temperature up. Slight aberrations, which in health would not modify the temperature, cause pronounced oscillations in disease. Recrudescence, further, may be produced by a relapse. After the afebrile period following typhoid fever, for instance, the temperature may rise and a recurrence of the disease take place.

The Symptoms of Fever.

Pyrexia, or increased temperature, is not the only evidence of fever. The production of heat within the body is not due to increased tissue-change alone. It may be due, for instance, to increased oxidation of sugar, which is part of the substance of the body. Physiologists have found that a high temperature may take place, and yet the quantity of urea and of carbon dioxide discharged may not be as great as that of a healthy person who is taking active exercise or who has eaten a large meal. It must be remembered, therefore, that it is not heat-production alone, but *alterations of heat-regulation* that cause pyrexia and its phenomena.

Wasting. Wasting of the body is a striking symptom of fever. There is no doubt that even in fever of moderate duration great wasting of the solid structures takes place. At the same time the blood wastes

(see observations of Thayer) and the various fluids of the body are also diminished; hence the disorders due to diminished secretion of glands are prominent in febrile conditions. Diminution of secretion in the gastro-intestinal tract, causing thirst, loss of appetite, indigestion, and constipation, indicates wasting of the fluids. Scanty urine of high color and specific gravity is due to the same cause.

The Pulse-rate. Acceleration of the pulse is one of the phenomena that attend pyrexia. While increased pulse-frequency is the rule, and is, in all probability, a result of the increase in temperature, other circumstances may cause a change in the pulse-rate in pyrexia. Thus in basilar meningitis, although there may be a high fever, the pulse is not accelerated. On the other hand, some diseases usually accompanied by fever, as diphtheria and peritonitis, may run an afebrile course, and yet the pulse be very much accelerated.

Arterial Tension. The arterial tension and the rapidity with which the blood flows in fever do not bear a due proportion to the acceleration of the pulse. The true febrile pulse is not dicrotic. In the early stages of fever the pulse is large and hard, the arterial tension is high, and the vessels are full. In the later stages arterial relaxation takes place, and the pulse becomes soft, feeble, and often small, with low pressure. The pulse is rapid, and dicrotism or even hyperdicrotism now becomes a prominent feature. The heart beating rapidly empties itself incompletely and discharges less rather than more blood into the arteries. The impairment of the cardiac beat is no doubt due to the degenerations incident to the high temperature, and is not dependent upon any special febrile affection. Such changes also take place in the glands, particularly the liver and kidneys, and are known as parenchymatous degenerations, or cloudy swelling. These changes in the cardiac muscle may induce thrombi during the later stages of fever, and cause death from heart-clot.

The Respiration. The respirations are increased in fever, probably because of the close dependence of the regulating centre of respiration on that of the heart. The heated blood acts as a stimulant to the respiratory centre. As proof of this, the hurried respiration of pneumonia ceases as soon as the temperature falls, notwithstanding the fact that the affected part of the lung remains hepatized.

Cerebral Symptoms. Delirium and other nervous symptoms may attend fever. They are not dependent upon the increased temperature of the blood alone. No relation appears to exist between the intensity of the fever and the severity of the delirium. In relapsing fever a temperature of 106° occurs with the mind clear. In certain cases of typhoid fever a temperature of 103° is attended with marked delirium. If fever persists for a short time, a low asthenic state, so-called *adynamia,* may develop. Because the symptoms resemble those of typhus fever the term typhoid is also applied to them, and the condition about to be described is known as the *typhoid state.* The expression is dull and heavy, the capillaries of the face are congested. There are stupor and sluggishness of mental processes, so that the patient is slow in answering questions. The stupor is attended with low muttering delirium and may be followed by complete unconsciousness. The pupils are con-

tracted, the eyes heavy and dull. The patient is so prostrated that he slips down into the bed from the pillow. There is marked subsultus tendinum. The tremulous tongue is protruded slowly. It is dry and brown, and the mouth and teeth are covered with sordes. The sensibilities are blunted, so that food and drink are not asked for, or relished if given. Involuntary discharges take place from the rectum and bladder, and incontinence of retention of the urine arises. The pulse is small, feeble, and dicrotic, the heart-sounds are weak and feeble. The first sound becomes short and snappy like the second, or may be absent entirely. Venous stases take place in the dependent portions, particularly in the back of the lungs.

As œdema or hypostatic congestion advances the breathing becomes shorter and labored. More or less cyanosis then creeps over the general surface. The urine becomes more and more scanty and high-colored, contains albumin, and sometimes blood.

The typhoid state may continue for many days, or even last two or three weeks, although not in so advanced a degree as has been described. It is more likely to supervene when there is excessively high temperature, but it also occurs in the course of a prolonged illness with a temperature of moderate degree—that is, 103° F. Although it is in all probability due to the direct effects of heat upon the nerve-centres and the organs of the body, yet there are cases in which the temperature is not high, and yet all the symptoms of the typhoid state develop. While the typhoid state is common to *typhoid fever*, it occurs also in *pneumonia* and *septicæmia*, and may even be seen in its most typical form in other conditions in which fever is not a pronounced symptom; thus in *uræmia*, in the later stages of *softening of the brain*, in *paresis*, and in allied nervous diseases the symptoms of the typhoid state are most striking. In this class of cases it certainly cannot be attributed to the fever, but is in all probability due to the depressing effect on the nervous system of material which should be excreted from the body, a view which has been advocated by Murchison, Flint, and others.

Ataxia or the *ataxic state* in fever is a condition the opposite of the adynamic or typhoid state. In the latter there is weakness, while in the former there is exhibition of strength. In the latter the nerve-centres and the vital processes are depressed; in the former they are stimulated. Ataxia is characterized by a strong pulse and by active, violent delirium, so that it is almost impossible to keep the patient in bed and restrain him from exerting himself. The face is flushed bright red; the eyes injected, bright, and active. The tongue is furred, but is not necessarily dry or brown. The delirium may be constant or paroxysmal, and is often maniacal in character. The body temperature is high, and a sensation of intense heat is imparted to the hand when placed on the surface of the trunk. The patient may complain of a bursting, intense headache. If the ataxic state is not controlled after a few days, or at the most a week, the patient becomes exhausted and lapses into stupor, which may proceed to coma. In some forms, particularly in children, convulsions may accompany the excessively high temperature and be followed by coma. The so-called coma vigil may supervene. Ataxia is seen notably

in scarlet fever, "cerebral" pneumonia, and in forms of typhoid fever. The peculiar behavior of the temperature and nervous systems in this affection and in apical pneumonia, or so-called pneumonia of the cerebral type, has led observers to mistake such cases for actual cerebral disease. Frequently such patients have been admitted into insane asylums for supposed mania; the condition is often mistaken, and because of lack of attendants the patients have jumped from the window or done violence to themselves in other ways.

It is as difficult to determine the exact cause of the extreme perturbation of the nervous system in *febrile* ataxia as in adynamia. It may be due to a high temperature acting on nerve-centres; or to a poison, as the special toxin of the infection which has caused the fever.

The presence of fever may be suggested by *flushing* of the face. This may be general or local. The local flush of phthisis and of pneumonia has previously been referred to. *Dryness* and pungency of the skin occur in fever. In former times the sense of heat was given different attributes, said to be distinctive of various affections. Hence the terms *calor mordax*, and the like. Thus the sensation of heat conveyed to the hand in typhus fever was said to be characteristic, and the degree of fever was determined by the sense of touch. *Sweating* is a condition habitual in some fevers. It may occur throughout the course of the disease, or at certain stages only, as instanced by the early morning or night sweats of tuberculosis. In such cases the skin is cold and clammy. Like sweatings are common in the fever of deep-seated suppuration and in diseases of the bones. Sweating in defervescence marks the occurrence of crisis.

Headache. Headache and pain in the back occur in the acute specific fevers during the initial stage. One or both are nearly always present, but in different affections they have diagnostic significance. Thus severe pain in the back is more pronounced in tonsillitis and smallpox, severe headache in cerebrospinal meningitis, and protracted throbbing headache in typhoid fever.

The Diagnostic Significance of Fever.

The presence of fever, its course and its type, are of diagnostic significance. When it is present, *hysteria* and *malingering* can usually be excluded, and one of the four following morbid processes is present:

1. An *infection*, general or local, as seen in any one of the infectious diseases and in the local inflammations induced by micro-organisms, especially those known as pyogenic. When local, the inflammation is known as purulent, suppurative, or septic. The micro-organism, a product of its growth, or the poisons or ferments resulting from the tissue change, disturb the thermotaxic mechanism and cause fever. Any tissue-membrane, or organ of the body may be the seat of an infectious process.

2. An *intoxication*, or *toxæmia*, as caused by albumoses, ferments, toxins, or ptomaïns, generated within the system, the result of impaired functional activity of organs or structures, or of cell metabolism, as seen in tissue-waste; and by food-products, medicines, or toxic substances

introduced from without. Catarrhal inflammations cause a toxic fever. The fever of gout, of anæmia, and of starvation is toxic.

3. A *cerebral lesion* from disease involving the centres controlling heat-production and heat-loss, or in proximity to them. It may arise in cases of brain tumor, in cases of apoplexy and of thrombosis. The centres may also be irritated by direct exposure to external heat alone, or possibly by poisons generated within the system on account of the heat (an intoxication), as in sun-stroke.

4. A pronounced *peripheral irritation* or the occurrence of *pain*, reflexly altering the thermotaxic mechanism. In iritis or orchitis a fever arises out of all proportion to the local inflammation.

Atypical Fevers. It must be remembered that cases of *continued fever* exist that have not thus far been classified. One of the nurses in the Presbyterian Hospital in Philadelphia was under my care for two months with a continued temperature from 100° to 103°. No general or local condition could be found to account for it. The patient was emaciated, and she had had two years of very hard work. Although the fever kept up, the appetite was good. Careful and abundant feeding, with rest for many weeks, caused the temperature to fall to normal, with complete recovery. I looked upon it as a *nervous fever;* an expression of exhaustion. Fagge refers to such cases.

Practically, we must in all cases of fever decide between one of *infectious* and one of *toxic* origin. The mode of determining the occurrence of an *infection* will be considered presently. In the meantime it may be remarked that the poisons which are generated in the gastro-intestinal tract are likely to disturb the cardiac and respiratory as well as the thermotaxic mechanism. Hence, we often see *irregularity* and *intermittency* of the heart—so often as to consider them of diagnostic value in favor of toxic fever.

Significance of the Temperature-curve. Certain clinical features of a febrile course belong, in the main, to special affections, and are quite diagnostic. Hence, the mode of onset, or *initial stage*, the course or *fastigium*, the *decline* and the *type* should be carefully studied. They are most important indications of the nature of the disease.

The Initial Stage. In the initial stage of fever, *sudden* excessive rise of temperature during a condition of apparent health argues against any of the acute specific fevers except scarlet fever. It is of more frequent occurrence in acute gastric or gastro-intestinal catarrh in children than in any other ailment. It may be due to pneumonia, and is significant of this infection in adults if attended by a rigor. In children convulsions may replace the chill. The sudden rise may be due to certain types of malaria, when it is also preceded by a chill and followed by free sweating. It may also be due to affections of the throat, to follicular or phlegmonous inflammation of the tonsils. The throat must always be examined in cases of sudden high temperature.

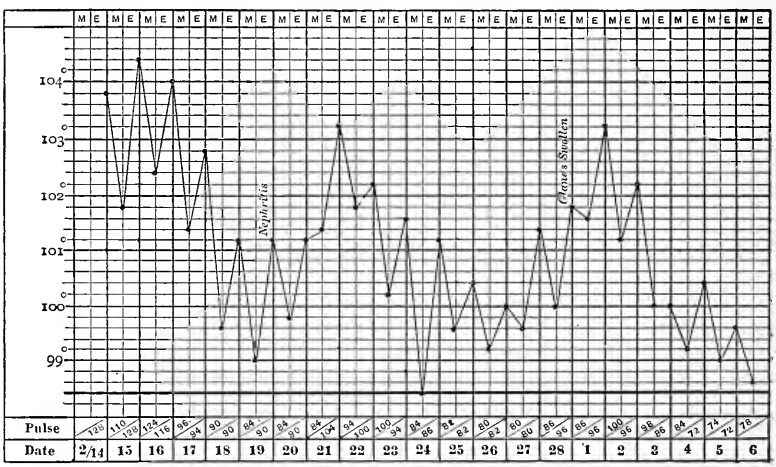
In children, if pain attends any inflammatory affection, the temperature will rise to a greater height than the local process alone would warrant. Suppurative inflammation of the middle ear is a case in point. This must always be borne in mind in sudden rise of temperature. The same

active febrile reaction will take place in osteomyelitis and in mastoid abscess. The associate signs point to the true nature of the affection, although it must be confessed that in both, the symptoms are often obscure in the beginning.

In typhoid fever the temperature rises in a characteristic way. It ascends by *successive evening rises*, followed by morning remissions, until it reaches the maximum at about the end of the first week.

The Fastigium. In typhoid fever the course of the fastigium is of characteristic significance. From the end of the first, throughout the second week, and sometimes longer, the fever is of the continued type. Subsequently during the third week, or later, morning remissions set in, the temperature for a time still rising to the former height in the evening. Then the morning remissions become more decided, the temperature not rising as high in the evening, and so gradually the temperature sinks to

FIG. 77.



Scarlet fever. Modification of temperature by complications. Nephritis on the ninth day. (Original.)

and below normal. This course of the temperature in typhoid fever is far from being invariable; it is modified by indiscretions on the part of the patient or his attendants, and by the necessities of antipyretic or other treatment; nevertheless, the gradual onset of the fever and its long duration are sufficiently common to make them of great value in diagnosis, as, with the exception of tuberculosis, there is hardly any other disease in which a continued fever exists for two or three weeks apart from local inflammation or suppuration.

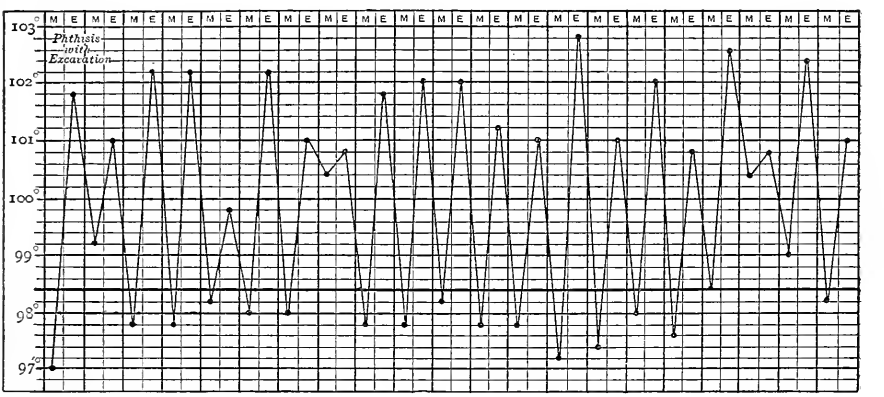
The Decline. In the self-limited diseases there is a period when *defervescence* should take place. A continuance of the fever, the persistence of the fastigium beyond the usual period, indicates that the case is one of a greater degree of gravity than usual, or that there is a complication. It is usually significant of the latter. In measles the complication is usually pneumonia, which may take place after the

disease has developed, and may be the cause of the unusual rise in temperature. In scarlatina it may indicate acute nephritis, or inflammation of any of the serous membranes, particularly the pericardium or endocardium. Persistence of the fastigium of typhoid fever after the period at which it should decline, if the patient is well nursed and properly fed, usually indicates the occurrence of a reinfection, a secondary infection, or the development of tuberculosis. If the latter, the fever is more likely to develop during convalescence. Of the inflammatory complications, phlebitis, glandular and bone infections are likely to cause persistence of fever.

A Sudden Fall. A sudden fall of temperature in a person who has previously had high fever signifies the crisis, if the time for that event has arrived, as in pneumonia; or of a grave complication, which induces shock. In typhoid fever this unusual drop in the temperature will take place if there has been hemorrhage from the bowels or perforation, or if peritonitis has developed. It must not be confounded with the sudden falls of temperature that occur in the typhoid fever of children at the onset of convalescence. They occur earlier in the period of the disease than with adults.

Significance of the Type. **Intermittent Fever.** The representative of this type is seen in *malaria*, but is simulated by a number of conditions: (1) In certain cases of *typhoid* and *relapsing* fever the type is intermitting or paroxysmal. The same type of fever is seen (2) in

FIG. 78.



Intermitting fever of tuberculosis. (Original.)

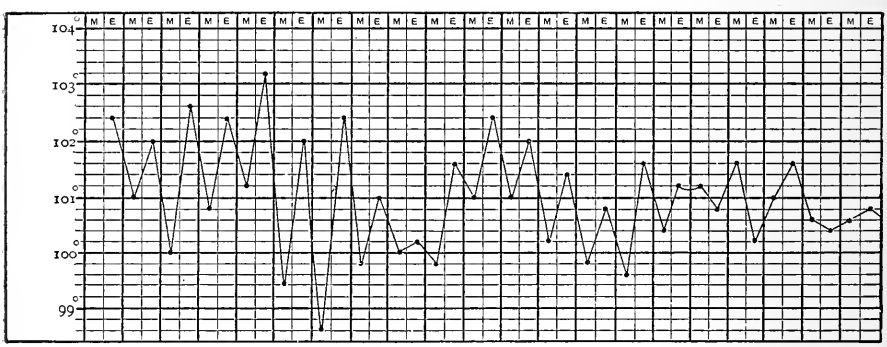
suppuration, particularly if the pus is confined, although in brain abscess the temperature may be normal or subnormal; (3) in *infectious endocarditis;* (4) in *tuberculosis*. *a*. It may occur in the earlier stages of tuberculosis. The primary seat of the lesion may be in the lungs, in the bones, or in the glands. *b*. In pulmonary tuberculosis, after the formation of a cavity, intermitting fever is of common occurrence. It is then of septic origin due to the septic influence of the necrosed tissue and products of putrefaction in the cavity. (See Fig. 78.) (5) In *lymphadenoma* and *anæmia* the fever is at times paroxysmal. (6) In *syphilis* the same

type is often seen. It may be noted (*a*) in the initial fever; (*b*) in the tertiary periods of the disease where gummata have formed or other forms of visceral syphilis have developed. (7) *Urinary intermitting* fever is the form which usually occurs after the passage of a catheter or sound, but it may also occur when there is suppuration in the genito-urinary tract. (8) *Hepatic intermitting* fever is a form of frequent occurrence and of great diagnostic importance. It may be due to (*a*) gallstones somewhere in the biliary ducts, usually with obstruction; (*b*) suppuration in the canal, with or without obstruction; (*c*) obstruction of the biliary passages by external pressure without suppuration; (*d*) inflammatory affections of the liver, as abscess, and forms of cirrhosis. (See Fig. 81.) It occurs rarely in rapidly growing cancer. (9) Intermittent fever may also attend the prolonged use of *morphine*.

Of the above-mentioned varieties of paroxysmal or intermittent fever, those of the most common occurrence are due to suppuration, pyæmia, infectious endocarditis, tuberculosis, or hepatic disorder. In addition to the paroxysmal temperature, rigors precede and sweating follows the paroxysm, as in cases of malarial intermittent fever. The diagnosis from malarial intermittent fever can be established at once by the demonstration of the plasmodia of Laveran in the blood.

Remittent Fever. Fever of a remittent type occurs in many of the conditions in which intermittent fever is present. It is characteristic of one of the forms of malaria. It is most frequently encountered in tuberculosis of the lungs. The remissions usually occur in the mornings, but

FIG. 79.



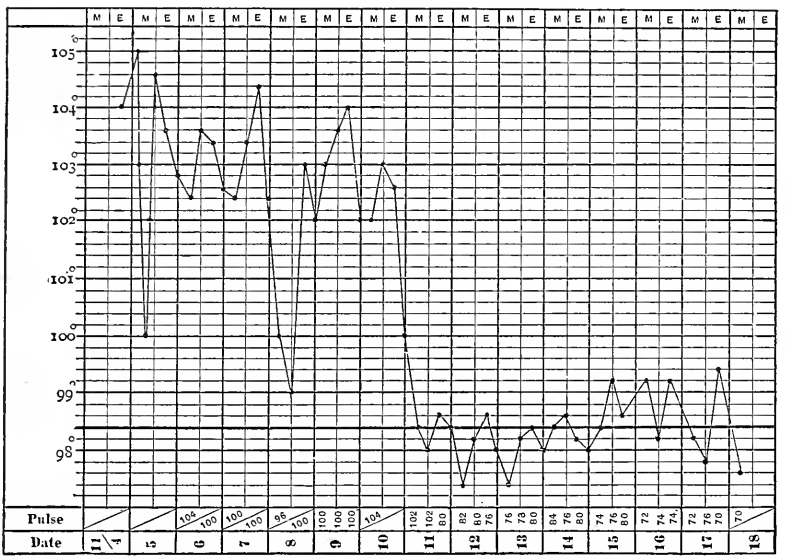
Continued fever of tuberculosis. (Original.)

the order may be reversed. The same type is met with in puerperal fever, pyæmia, and septicæmia, and in local suppurations such as abscess of the liver and empyema. A continued fever may be made to resemble a remittent by antipyretic treatment, which may cause abnormal remissions. Remissions characterize the decline of the continued fevers, particularly typhoid, during the period of lysis.

Continued Fever. Continued fever is met with in lobar pneumonia, typhoid fever, typhus fever, erysipelas and tuberculosis. In acute lobar

pneumonia the temperature rises rapidly, and in a few hours from the initial chill reaches 103° or 105°. The morning and evening temperatures vary but little, usually not more than 1 or 2 degrees, until a crisis occurs in from four to eight days. The temperature then falls to or slightly below normal, and does not rise again. (See Fig. 80.) A marked remission in the fever sometimes occurs on the fourth day, before the actual crisis; the temperature falls to 100° and rises again to 103°

FIG. 80.



Pneumonia. Sudden rise. Termination by crisis. Pseudocrisis also seen. (Original.)

or 104°, remaining at that level for twenty-four or forty-eight hours, when the true *crisis* occurs. The first fall is known as the *pseudocrisis*. The critical fall of temperature (defervescence) may be completed within a few hours. (See Fig. 80.)

The Variation with Age and Sex. The significance of a febrile form is not so great in children as in adults. That is, the high temperature is not so important, inasmuch as children are subject to sudden excessive increase of temperature; and a higher temperature may persist in children without the deleterious effects upon the tissues which are noticed in adults. In women of nervous temperament the temperature is also likely to rise to a great height without adequate cause or serious result.

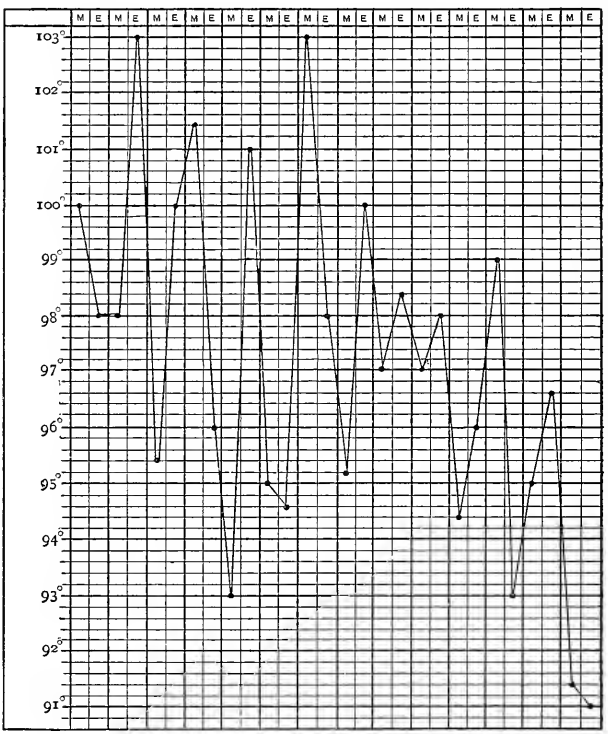
Subnormal Temperature.

A temperature below the normal may occur independently of fever. It may follow as a sequel of the diseases with more or less prolonged pyrexia. It occurs in the course of wasting diseases such as cancer, during starvation, and at times in anæmia. It is seen habitually in myxœdema, and occasionally in diabetes. In certain forms of tubercu-

losis it may extend over a long period of time, as in tubercular peritonitis. (See chart of Tubercular Peritonitis.) In cases of cerebral abscess the temperature is often subnormal.

Sometimes the drop to subnormal temperature may occur suddenly, to be followed by a return to normal or even a rise above normal. The sudden fall may occur in shock, or in hemorrhage from any cause. It may take place from disturbance of the nerve-centres, as from apoplexy,

FIG. 81.



Subnormal temperature. Oscillations in hepatic intermittent fever with jaundice. Catarrh of the ducts with diffused hepatitis. G. W., aged sixty. Philadelphia Hospital, 1877. (Original.)

thrombosis, or embolism of the brain, causing shock or other disturbance of the thermotaxic mechanism. It is characteristic of cholera. In the course of organic heart disease pulmonary embolism is also attended by subnormal temperature. In many of these instances the temperature will rise after the shock if the latter is not too profound (reaction). This is notably the case in apoplexy and in embolism or thrombosis, because of local irritation or a secondary softening. In apoplexy the rise in temperature will occur either from central disturbance of the thermotaxic mechanism or from secondary inflammation about the clot. A subnormal temperature in the course of fever may be due to an accident or complications, as hemorrhage in disease of the lungs, or in typhoid fever, or per-

foration of the intestine in the latter condition. It may attend the crisis of acute disease. More or less collapse usually attends the pathological fall of temperature below the normal. While such fall is the result of some accident in many of the diseases mentioned, in others it is a part of the process.

Fig. 81 represents the effect of a local process in the largest gland of the body upon the general temperature. It is possibly a septic temperature, although the observation was made before the days of bacteriological research. The extreme low temperature is remarkable.

The Causes of Fever.

We have already indicated the diagnostic significance of *fever*. Consequently, when this symptom is present, we examine every organ and structure of the body, in the order suggested by the subjective symptoms. By this examination we will find either: (1) functional disturbance of some organ of the body, (2) an inflammation, (3) an intoxication, (4) evidence of brain disease, (5) a general infection.

1. Any *functional disturbance* of one or more organs—glandular—attended by fever must give rise to the symptom through toxic products and is therefore to be looked upon as an *intoxication*. (See the Intoxications.)

2. We may find local *inflammation* of some part, as an inflammation of the nares, a bronchitis, a gastritis, or an enteritis. The inflammation may be *toxic* or it may be *infectious*. The kidneys may serve as an example. Blood, albumin, and renal casts would show that they are the seat of inflammation. The inflammation may be due (1) to the action, during elimination, of a poisonous substance taken into the intestinal canal; or (2) to the direct effect of a focal bacterial invasion of the kidneys; or (3) to the remote effect of a bacterial toxin formed in some other part of the body and reaching the kidneys through the circulation. In any case the fever is caused by the local process. To determine whether the inflammation is *toxic* (generally catarrhal) or *infectious*, we must rely upon the data obtained by inquiry, the clinical course, and the result of the examination, which discloses the method of determining the presence of an infection.

3. If an *intoxication*, it will be recognized by the symptoms considered later and by excluding other causes of fever. (See the Intoxications, Chapter II., Part II.)

4. Central brain disease must be looked for, as apoplexy, brain tumor, or cerebral thrombosis.

5. If the above are excluded, we proceed with the *bacteriological diagnosis*. By this means we find if a local or general *infection* prevails. Such a method may be necessary to recognize pyæmia and septicæmia.

I. The Infections.

The *infectious diseases* are those that are produced by a living pathogenic germ. The organism is introduced into the body through the skin if the latter is the seat of some lesion, as in syphilis, tuberculosis, and anthrax; through the air-passages, as in diphtheria, scarlet fever,

and other specific fevers; or through the digestive tract, as in typhoid fever, dysentery, and cholera. The virus, as the living cause is named, in many instances produces certain changes at the point of entrance—the *initial phenomena*. If the organism remains *in situ*, multiplies, and produces its toxin, the infection is said to be *local*. The symptoms resulting therefrom are often twofold: local, due to the infection, and general, due to the toxæmia or intoxication. The greater number of inflammations of internal organs and tissues are examples of local infections, as appendicitis or peritonitis. The organisms may be conveyed by the lymphatics or bloodvessels to near-by organs in the related lymph-stream or blood-stream, or transmitted to the whole body. They will be found everywhere, and the infection is said to be *general*. The symptoms are due to occlusion of the capillaries by micro-organisms, to generally distributed areas of infectious inflammation, and to an intoxication. The above are the *phenomena of general distribution* of the virus, or of *infectiveness*. The virus or poison thus distributed may be the living organism, as in tuberculosis or anthrax, or it may be a poison generated by the organism, a *toxin*, as in diphtheria.

Phenomena of a secondary character may be due to local changes in organs affected by the original organisms, or by a new germ (mixed infection). Some germs have a special affinity for certain organs, as in whooping-cough, parotitis, pneumonia, and leprosy.

In some instances the local phenomena are so marked as to give to the disease a corresponding distinctive feature. They are the granulomata. Bearing in mind the above distinctions, specific infectious diseases are divided, from the standpoint of the pathologist, into six classes:

First Class. *Acute Specific Fevers.* Initial phenomena slight. The symptoms of infection are marked; an eruption is one of the most characteristic. The secondary local phenomena are variable. The following are included in this class: typhoid fever, typhus fever, variola, varicella, scarlet fever, measles, relapsing fever, rubella, dengue, the plague, and cholera.

Second Class. *Specific Inflammation.* Initial phenomena indefinite. General phenomena (infectiveness) variable, but no eruption. Specific affinity of poison for one particular structure in the milder forms of the infection. Whooping-cough, mumps, diphtheria, influenza, dysentery, erysipelas, tetanus, hydrophobia, cerebrospinal meningitis, rheumatic fever, and pneumonia belong to this class.

Third Class. *Local Infection. Contagious or Infectious Suppuration.* Initial phenomena marked (suppuration); generalization not marked unless the virus enters the blood; secondary local phenomena decisive. Gonorrhœa is one type; another form includes pyæmia, or any infection from pus-producing micro-organisms, as abscess, carbuncle, etc.

Fourth Class. *Local or General Infection, or both. Infective Granulomata.* Distinct initial phenomena. Phenomena of generalization not marked or like specific fevers. Secondary local phenomena prominent. Examples: tuberculosis, syphilis, leprosy, and glanders.

Fifth Class. *General Infection. Diseases due to Protozoa.* No initial phenomena.

Sixth Class. *Local Infection. Vegetable Parasitic Diseases.*

Unfortunately, the cause of many of the infectious diseases has not been definitely isolated. This group is largely composed of the infectious disorders which are epidemic and contagious. In order to diagnosticate them it is necessary to associate with the mode of onset and clinical course of the disease, the facts and laws pertaining to epidemics and to contagion. Data, therefore, obtained by *inquiry* are quite necessary to establish their diagnosis. Such data are also useful in confirming the results of an objective or bacteriological examination of the patient, even though the diagnosis be at once established by that method.

In the first place, we note the *social history*. It should be personal and general, and should include the age, sex, habits, and occupation. The nature of the prevailing diseases in the community are known or sought for, and all possible unusual circumstances in food, drink, and clothing are inquired for. In short, a history of exposure to influences which attend an intoxication or those which permit infection is to be zealously sought for.

An inquiry for *previous diseases* does not imply a history alone of a previous infectious disease, but a history of such diseases as are often followed by infection. Thus a history of a previous attack of gallstones or of renal calculus may be a clue to the localization of an infectious process. Too much stress cannot be laid upon the diagnostic value of the data obtained in this manner.

The next data obtained by inquiry are the history of the *present disease*. The mode of onset is of itself suggestive. Sudden onset points more closely to an intoxication, though not necessarily, although more likely in children. Otherwise sudden onset usually indicates one of the short infections, of which scarlatina and pneumonia are representative types; while gradual onset points to a long infection, of which typhoid fever is a type.

The *subjective symptoms* are then inquired for, and their site affords a clue as to the steps to be taken in the objective examination. Thus pain in the throat with difficulty in swallowing calls for an examination of the fauces; pain in the chest, of the lungs; in the præcordia, of the heart, etc. Any functional disturbance of an organ should also lead to a study of it.

In a case of fever the problem for solution is: Is the infection local or general?

Local Infections. The appearance of the inflammatory process may be sufficient to decide its nature—a boil, an abscess, a carbuncle, which give rise to more or less fever, are recognized by the sense of touch and the eye. But an appendicitis, a cholangitis, an inflammation of a serous membrane, as well as a boil or carbuncle, may be a *local infection*. In like manner the accidental wound of a surgeon, by which he is inoculated or infected by the micro-organism of pus, may be an infection. The natural or acquired wounds of the puerperium may also be infectious. It must be borne in mind that any local inflammation may be infectious. Some, indeed nearly all, of the streptococcic and staphylococcic infections are local. The general symptoms are produced by a toxæmia, the toxin alone passing into the blood. It is of importance, however, to remember that to determine the infectious nature of a local inflammation and the nature

of the causal micro-organism, we use the same methods that are employed to determine the nature of a general infection.

It is also important to repeat that a local infection is circumscribed and may cause a toxic fever. On the other hand, a small portion of the purulent exudate from the infection may get into the circulation and be carried to distant parts, as the brain, the lungs, the kidneys, the joints, and the spleen. Distant foci of inflammation are set up, giving rise to multiple small abscesses, local infections, in the organs affected. *Pyæmia* is the name of this form of infection. Finally, such local infection may become general and the condition constitute *septicæmia.*

Pyæmia is characterized by rigors, fever, usually intermittent, and sweats. There is exhaustion ; the skin is slightly icteroid, and the odor of the breath is sweet. There are anorexia, nausea, perhaps vomiting, and frequently diarrhœa. Erythematous eruptions are seen. With these general symptoms there are present the physical signs of abscess in the lungs, the spleen, or other organs of the body ; or we may have an endocarditis. When the affection is limited to the portal area, and multiple abscesses of the liver succeed a purulent process in the neighborhood of the portal vein, the general symptoms are combined with deeper jaundice and with enlargement of the liver, which is tender and painful. The micro-organisms which invade the system and cause areas of suppuration are *Streptococcus* and *Staphylococcus pyogenes, Micrococcus lanceolatus,* the gonococcus, *Bacillus coli communis, Bacillus typhi abdominalis, Bacillus proteus, Bacillus pyocyaneus, Bacillus influenzæ,* and *Bacillus aërogenes capsulatus.*

Diagnosis. Pyæmia resembles in many respects tuberculosis of the kidneys and calculous pyelitis, in both of which recurring rigors and sweats are common. In gross aspects it resembles malaria. (See Intermittent Fever.) In prolonged cases of pyæmia the symptoms may resemble typhoid fever, but leucocytosis is present in the former condition. Ulcerative endocarditis and acute miliary tuberculosis usually resemble septicæmia, but may be confounded with pyæmia. Any febrile process associated with chills may be mistaken for pyæmia. These phenomena are seen in grave anæmias, in Hodgkin's disease, in hepatic intermittent fever, and in the intermittent fever of carcinomatosis. Post-febrile arthritis after scarlet fever and gonorrhœa is in all probability pyæmic. Of course, we rely in the diagnosis of pyæmia upon the data obtained by bacteriological methods when their employment is practicable.

Septicæmia. Again, we may find with the above-described wound, or without any apparent local inflammation, *fever* which is more or less continuous. In addition there may be an occasional rigor. The pulse is rapid, and exhaustion, anæmia, and some emaciation are present. Secondary infection of other structures may be present. Microbic infection of the blood usually takes place. The process is a *septicæmia.* If it originates from a local infection, it is known as *progressive septicæmia ;* if independently of any apparent local infection, it is a *cryptogenetic septicæmia.* The former is easily recognized, particularly if there is a history of a primary local infectious process. The micro-organisms which may

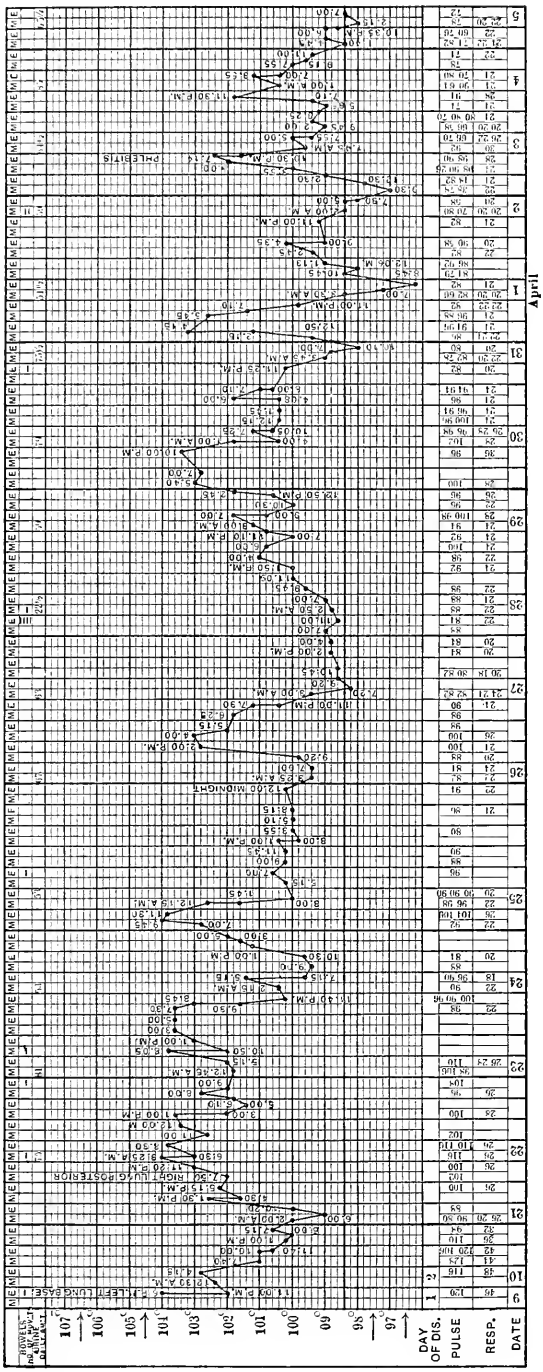


FIG. 82.

General infection or septicæmia, probably pneumococcic. (Original.)

give rise to the latter are *Staphylococcus pyogenes*, *Streptococcus pyogenes*, *Bacillus proteus*, *Bacillus pyocyaneus*, and *Micrococcus lanceolatus*. It is recognized by a bacteriological diagnosis.

The accompanying chart (Fig. 82) represents the course of a general, and various areas of secondary infection in a case of general septicæmia. The illness extended over a period of thirty-five days. During the first five days, as indicated by the chart, there was pneumonia at the base of the left lung. The crisis only is represented. From the tenth to the twenty-first day the fever was continuous, and, to save space, the chart does not give the temperature range. On the twelfth the right pleura was infected; on the nineteenth the femoral vein of the right leg, the temperature not rising above 101°. On the twenty-first, as the chart indicates, a patch of pneumonia was found in the right lung posteriorly. On the twenty-fourth pseudocrisis, and on the twenty-fifth and twenty-sixth the true crisis took place. On the twenty-ninth and thirtieth there was reinfection of the pleura of the left side. On April 3d phlebitis of the femoral vein of the left leg developed. During the course of the disease there was a low-pitched endocardial murmur, which in all probability was anæmic. Sweats, attacks of collapse, and irregular rigors took place. Life was imperilled at the time of the collapse. The spleen was enlarged; the sputa contained pneumococci. The blood examination was negative. The patient recovered.

Fever, varying in type, sweats, emaciation, anæmia, and exhaustion are the common general symptoms of septicæmia. The pulse is increased in frequency, and is dicrotic and compressible. The heart-sounds grow weak, the breathing hurried. There is slight delirium at times. The urine contains albumin and casts, and it is scanty, high colored, and of high specific gravity. In some forms of this disease there is leucocytosis. There also occur anorexia, nausea and vomiting, often diarrhœa. As the case advances the symptoms of the typhoid state develop. (See page 358.)

Objective Signs of Septicæmia. In other instances there is marked evidence of a septic process in the structures which carried the poisoned or infected blood from the primary point of entrance of the infecting material—the *infection atrium*. Hence, we may find the origin of the infection in diseases of the ear, the nose or its accessory cavities, the fauces, the mouth, the genital organs, the bladder, or the rectum.

On further examination we may find *lymphangitis* and *lymphatic adenitis*. The *spleen* is enlarged. There may be *phlebitis*, especially of the femoral vein, inflammation of which is always infectious in character. Other *veins* may be affected. The *endocardium* may be infected, and, indeed, the symptom-complex of *endocarditis* may be the chief indication of the septic process. The serous membranes may be involved, so that septic *pleurisy*, *meningitis*, *pericarditis*, *peritonitis*, or *arthritis*, singly or combined, may be local expressions of the sepsis. *Hemorrhages* from the mucous membranes, or subcutaneously, either because the blood is destroyed (toxic) or because of multiple small infarcts, frequently attend septicæmia. Hemorrhages may be the most pronounced symptom of certain forms of infection, as that due to capsulated bacilli. A slight *jaundice* of toxic origin may prevail.

Toxic Symptoms. In some instances there is a profound *toxæmia*, indicated by delirium, stupor, and later coma and convulsions. The typhoid state may predominate. The intoxication may overwhelm the cardiovascular centres, and the pulse grows rapid and feeble, the respirations hurried and shallow. The urine is diminished in amount and contains albumin.

The clinical course varies with the infective agent. Streptococcus infections are characterized by chills, high fever, and an extreme septic state. Infection by the capsulated bacilli (Howard) gives rise to a hemorrhagic septicæmia. In other infections the greater part of the clinical course may be afebrile. Toxic symptoms, and especially increased frequency of pulse-rate with collapse phenomena, are present, as in forms of infectious peritonitis.

A general infection, or this general expression of *septicæmia*, occurs in diseases in which the clinical course of the infectious process is usually a definite one. Hence, we speak of typhoid septicæmia or a pneumococcus septicæmia when the intoxication or general infection is paramount to the local process. Then in tuberculosis and other prolonged infections, in which septicæmic symptoms arise, the terminal phenomena of the disease are usually due to a *mixed infection*. It must then be understood that *pyæmia* or *septicæmia* or septicopyæmia are not due to special micro-organisms in the sense that typhoid fever is due to *Bacillus typhosus*, malaria to the plasmodium, or pneumonia to the pneumococcus.

General or Systemic Infections. The subjective and objective symptoms gathered by the study of a case will be considered in the discussion of each infection in the following pages. It is absolutely necessary that data of this character should be secured to control the studies of the laboratory. Clinical expression should tally with bacteriological findings. As an example of general infection we have *septicæmia* (see page 370), which may be derived from a local or a general systemic invasion.

The classification of the infectious diseases is based upon the fact that a specific micro-organism is known which gives rise to similar phenomena in the respective infections. In other words, the infection of malaria, of tuberculosis, or of diphtheria follows a recognized clinical course. The period of invasion, the mode of onset, the symptoms throughout the course of the disease, are with notable exceptions practically the same.

It is readily seen that when the definite cause of an infectious disease is isolated, and the morphological and biological properties of the causal micro-organism determined, the clinician has acquired a valuable aid to diagnosis. Indeed, in such affections the bacteriological diagnosis has become an absolute certainty.

Infections of Certain Bacteriology. In our investigation of the cause of the *fever* in a suspected case we have found evidence of an infection as shown by (1) the phenomena of a local inflammation, (2) by the presence of pyæmia or (3) of septicæmia. The clinical course alone frequently enables one to make a diagnosis. At times we may have to resort to more positive methods. While the nature of the process, however, is usually recognized, the nature of the infection must be decided by bacteriological examination.

Infections of Uncertain Bacteriology. The presence of those infections, the bacterial cause of which is not known must be determined somewhat differently. In one group we must be content with the data obtained by inquiry and by observation, comparing the symptoms with the known course of a similar disease. Scarlet fever can only be recognized in this way. In subsequent chapters, therefore, the infections are divided into those recognized by inquiry and observation, and those recognized by supplementary observation by bacteriological methods.

Terminal Infections. At the termination of many chronic diseases, as the various fibroid affections—cirrhosis of the liver, the kidneys, end-arteritis, or spinal cord disease, and in carcinoma—there is *fever*. This is generally due to an infection which the weakly resisting organism invited.

Flexner has studied the terminal infections. In 255 cases of renal and cardiac disease he found 213 infectious, excluding tuberculosis. They were local and general. Infections of the serous membranes are the most common. The old clinical fact that serous inflammations were complications of Bright's disease has been proved by bacteriological methods to be due to an infection, and not, as formerly thought, to a chemical change in the blood. The following micro-organisms are met with: *Streptococcus pyogenes*, the pneumococcus, *Staphylococcus aureus*, *Bacillus proteus*, the gonococcus, *Bacillus pyocyaneus*, and the gas bacillus.

Tuberculous infection is also a terminal process in many diseases. Frequently an acute tuberculosis of serous membranes is found in the course of chronic heart or kidney disease.

Fever in Carcinoma. Fever occurs in the course of carcinoma under two circumstances. It is well known that in rapidly growing cancer of the liver, fever, often intermitting in type, is present. Possibly the fever is the result of the action of the poisonous substances of its own production. It may also be present in general carcinoma, and in all probability in carcinoma of the lungs and of the bones. But fever in the course of cancer may be due to a secondary infection. It can be readily understood that such infection is likely to take place if the malignant disease occurs in the course of any of the tubes or channels. First, there is ulceration of the cancerous mass in the course of the malignant process. Then the ulcerated area is infected by the pathogenic organisms in the vicinity, as the colon bacillus in the intestinal canal. With this area as the infection atrium a general septicæmia may arise.

Afebrile Infections. Although most infections cause such reaction of the system as to produce fever, some few are afebrile. Such is the case with tuberculosis—when it is local—and of syphilis in certain stages. The writer is of the belief that when the syphilitic poison is active—*i. e.*, productive of lesions—fever is present some time during the twenty-four hours. He is fully persuaded that mistakes are made because fever is not considered a part of the syphilitic infection. He has seen all types of fever—intermittent, remittent, and continuous—in all of the arbitrarily called stages of infection. The rise may be moderate or very pronounced. For its detection the thermometer should be employed every two hours.

Typhoid fever is an example of an infection which sometimes runs its course without rise in temperature, though this is very rare.

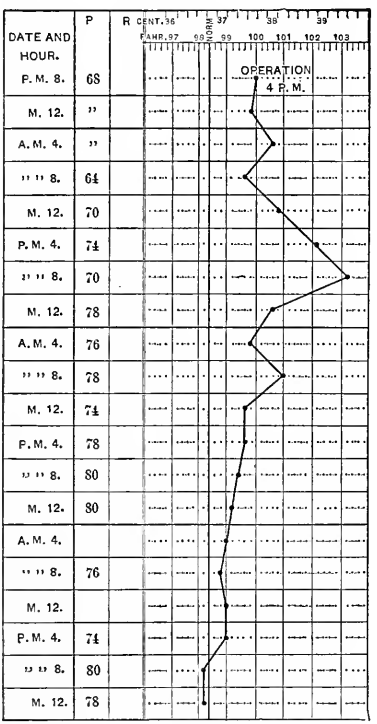
II. The Intoxications.

Catarrhal Fever. The substance which produces fever of this type may be a toxic material, the product of local or general disturbance of tissue-metabolism. Thus in a local catarrhal inflammation, as of the bronchi, the result of the direct action of an irritant vapor, toxic substances are generated which disturb the heat-mechanism and produce fever. Therefore, a simple inflammation of mucous membranes causing an intoxication which is attended by fever, may be styled *catarrhal fever.*

Anæmic Fever. In anæmia, on the other hand, if all infections can be excluded, it may be said the general disturbance of tissue-metabolism possibly gives rise to the formation of a toxic substance which causes the fever to attend this process—*anæmic fever.*

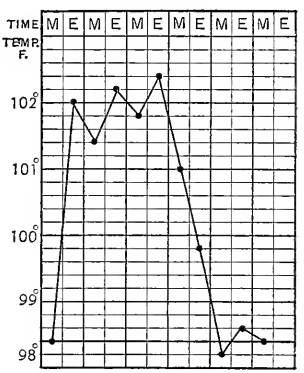
Aseptic Fever. A better example of fever due to a poison is that which J. Collins Warren terms *aseptic fever.* It is also known as *absorption* or *fermentation* fever. The fever follows a perfectly aseptic operation, and no causal factor is present. It

Fig. 83.



Temperature-curve after amputation of the forearm.

Fig. 84.



Aseptic or fermentation fever.

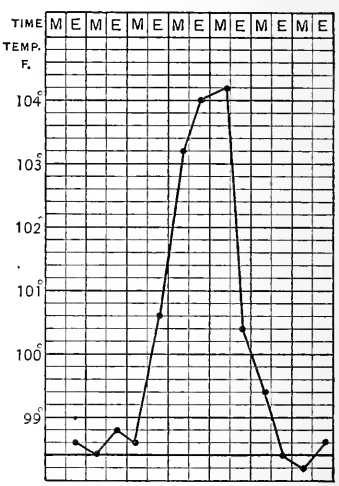
is due to the absorption of ferments, from blood-clot or coagulated serum or tissue-debris. The temperature rises to 102°, and may remain above normal from three days to two weeks. (See Figs. 83 and 84.) There is a striking absence of constitutional symptoms, however. Another peculiarity is that the fever begins immediately after the operation. The urine is not lessened, the body weight remains normal, and the pulse-rate

corresponds to the temperature rise. In some instances an eruption like that of scarlet fever—*surgical scarlet fever*—breaks out.

Traumatic Fever. Should it happen that the retained fluids undergo decomposition and are absorbed, a more intense type of fever is seen, attended by marked constitutional symptoms. We then have *traumatic fever*—a fever which may subside if the poison is liberated from the wound. In the meantime the temperature has been as high as 102° to 103°, the pulse very rapid, delirium has been marked, and there have been furred tongue, thirst, anorexia, restlessness, and malaise.

Sapræmia. It may happen that septic infection of a wound takes place. Thus one of my patients while dressing a suppurating vaccine wound inoculated or infected her finger. Fever developed, and the tender spot was followed by redness along the lymphatics and enlargement of the glands—a lymphangitis. A deep cut in the infected spot released a serous discharge, the fever disappeared, and the lymphatic inflammation subsided at once. Such accidents happen frequently to surgeons. Another patient was infected by a surgeon who had just operated on an osteomyelitis. The temperature rose to 106.5° in twenty-four hours, and the constitutional symptoms were extreme. The wound in the abdominal walls was opened and cleansed, but the peritoneum was not reopened; no peritonitis resulted. The temperature fell 4 degrees at once. The muscles and other tissues of the wound became grayish and almost putrid. Recovery was slow. Such cases are known as septic cases, and the intoxication *sapræmia*. (See Fig. 85.) No bacterial invasion of the body takes place, and there is no local suppuration. No doubt, in each instance micro-organisms infected the wound, but the symptoms arose from the chemical product resulting from their growth.

Fig. 85.



Temperature of septic intoxication (sapræmia).

In obstetric practice the retained putrefying placental fragments will cause such symptoms. In medicine we see similar intoxication take place in infections. Thus in diphtheria systemic intoxication with fever results from the absorption of a toxin from the local point of bacterial growth. In tetanus the same toxic fever and symptoms occur. It is impossible to draw hard-and-fast lines between the toxic fever and the infective, pyogenic, or suppurative fever; and, indeed, such conditions properly belong, because of their origin at least, to the infections, and will be considered under that head.

Toxic Fever. But "fever" may be due to other intoxications. It is well known that pepsin and other digestive ferments injected into the body cause fever. It is supposed products of imperfect assimilation or digestion absorbed into the system from the gastro-intestinal tract give

rise to fever. Ptomaïns or leucomaïns, albumoses or peptones, absorbed from the intestinal tract may thus cause fever. The retention of excretory products, as those of the renal organs, causes a systemic intoxication, with the frequent occurrence of fever. Gout, too, may be considered as an intoxication giving rise to fever.

The fever of auto-intoxication (gastro-intestinal or glandular), so called, therefore, is an entity. The clinician, at least, without proof by the bacteriologist, sweeps the intestinal tract with mercurials and salines, and thereby administers the causal antipyretic.

Poisoning by food-products, as of cheese, meats, sausages, milk, etc., appears to cause fever, although it is possible intestinal bacteria may play some part in the process.

Diagnosis. It is assumed that the student is investigating a case of fever. In keeping in mind an intoxication as a cause of fever, he must first consider all causes of intoxication from within; second, all causes from without the organism.

To the first belong gout, uræmia, cholesteræmia, and the auto-intoxications from the intestinal tract, as well as those from modification or suppression of internal secretions, as of the thyroid and other glands.

To the second belong the following: *sun-stroke*, *morphinism*, and *food-poisoning*. That the fever which attends the so-called *febricula* and *simple continued* or *catarrhal fever* is due to an intoxication is doubtful. Until its true nature is demonstrated it can well have a place in this section.

The evidence of the presence of an intoxication must be based upon (1) the history of exposure to a cause of intoxication; or (2) the occurrence of disease of such organs which give rise to toxic phenomena, as the kidney, the thyroid gland, etc.; (3) the absence of the phenomena of infection; (4) the presence of symptoms of the nervous system known as toxic, and symptoms of the gastro-intestinal tract, the result of irritation of that tract—gastro-enteritis; (5) rarely, local irritations of the respiratory mucous membranes are associated with toxæmia.

Toxic nervous symptoms are, varying in degree, delirium, convulsions, coma, tremor, the typhoid state, vertigo, motor and sensory symptoms due to an encephalomyelopathy, and forms of neuritis.

The Action of the Heart. Increased frequency of cardiac action is a symptom common to all forms of fever. It is more common to see irregularity and intermittency in the fever of intoxication, and especially of auto-intoxication, than in that of infections. Indeed, I should call a fever, not obviously due to an infection, if attended by a cardiac neurosis, one of intoxication, provided cardiac mural disease and cerebral disease could be excluded.

Increased Respiration. The same may be said of the breathing. When a respiratory neurosis prevails in the course of fever, it and the fever attending are due to a common cause, an intoxication. Of course, pulmonary and central brain and medulla disease are excluded. Both the above observations aid in the diagnosis of an intoxication from an infection.

SECTION V.

PHYSICAL DIAGNOSIS.

CHAPTER XXXIII.

GENERAL CONSIDERATIONS.—METHODS.

INSPECTION.

By inspection we judge of the physical condition of the whole or a part of the body, as seen by the shape and size, and by the color; of the vital condition, by the expression of countenance, by the character of the movements of the body as a whole or in part, by the position in bed, and by the gait. The appearance of fluids (blood) and of discharges is also observed. The results of inspection as to size are confirmed by actual *weighing*.

Scope of the Inspection.

In order that the data obtained by inspection may be complete and accurate, every portion of the body, and of its internal cavities which can be seen by the unaided or aided eye, should be inspected. The whole body should be examined. The entire surface, of course, need not be exposed at once, and circumstances may be such that only one portion need be examined. Portions of the body can be covered, or a light gown thrown over the patient from head to foot. Nevertheless, the fact must be insisted upon that patients who have been ill for a considerable time, as well as all grave cases, should be examined all over. It is even more important to do this if the patient is comatose. A node on the tibia, undue prominence of the vertebræ, a special rash about the anus, may afford information which could not be obtained in any other way. It is assumed that the patient has been examined lying down. In nervous diseases and diseases affecting the muscles and bones, the patient's gait, his ability to stand, the method of rising or assuming a sitting posture, and the performance of other customary physiological acts should be observed.

Methods of Inspection.

The patient must be seated, or lying down, in an easy, symmetrical position. The light should fall alternately directly and obliquely on the

surface. He should be viewed by the observer standing, first in front, then behind, and also from the side. To observe the anterior portion it is often well to stand behind the patient and look downward over the shoulders. The arms should fall by the side; the breathing should be quiet and undisturbed by talking or unusual movements.

In an examination of the heart it may be necessary to have the patient lean forward to bring it in contact with the chest-wall.

Order of Procedure.

To secure the data in full, the student should teach himself a method of observation by which all the facts are collated in regular, systematic order. Whether the examination be general or local, whether the whole of the body be inspected or only a part, as, for instance, the nose, the student should accustom himself to make observations in the following order: first, the shape or contour (expression); second, the size; third, the color; fourth, the movability and the physiological condition of the part on movement. If this plan is pursued, nothing will be overlooked.

Inspection of Special Regions. In the inspection of special regions artificial light and special instruments are also required. The artificial light should be secured from an Argand or Welsbach burner, or from a gas-jet with a reflector, or from electricity. To facilitate the examination the room should be darkened and head-mirrors used as reflectors. A number of these have been devised, any one of which is suitable if it fits the head well and can be adjusted with comfort, so that the observer can throw the light on the part he wishes to examine, and at the same time peer through the centre of the mirror. A special arrangement of the patient and the light is required. The patient should sit in an easy, comfortable, erect position, with the light on a level with the part to be examined, a little behind, and to his right or left, according to the convenience of the examiner. Special apparatus is required for the examination of each cavity: mirror, tongue-depressor, and specula for the throat, an ophthalmoscope for the eye, etc. (See respective sections.)

PALPATION.

The results of inspection are confirmed, when possible, by palpation, for the sense of touch supplies additional data. The nutrition of the parts is ascertained. The density, the resistance, the special character of the part, whether solid or liquid, are determined by this method of examination. On examination of the skin, the degree of dryness or moisture, the surface, whether smooth or rough, the density of the part—as thickness and resistance—are all ascertained by the sense of touch. The presence or absence of pitting is observed, and the nature of swellings ascertained. The routine method suggested in inspection should become habitual with the student: (1) the shape and contour; (2) the size; (3) the color, its change on pressure, etc.; (4) the movability of the part,

as determined by the palpating hand, or with respiration or functional movement, as peristalsis of the stomach or intestines, and the character of the normal movements, as when a joint is under observation ; (5) the resistance and density of the part examined, or special characteristics revealed by touch—the elasticity of the skin, firmness of muscles, and, in swellings, the presence or absence of fluctuation.

Other phenomena are detected which are vital, in contrast to the above which are physical. By palpation, alone or with instruments, we determine the sensibility of the part, the presence or absence of tenderness, and the temperature.

In the examination of special regions by means of palpation certain special phenomena are determined which depend upon the function of the part. Thus in palpation of the chest, in addition to its movements, we note the vibrations transmitted to the hand when the patient is asked to speak—*fremitus ;* or detect abnormal vibrations from the friction of two rough surfaces (pleura), or from the agitation of fluids : *fremitus, friction,* and *râles* are thus transmitted.

Knowledge of the action of the heart and of its position is obtained by palpation ; *thrills* are detected, abnormal impulses felt. (For method of procedure, see the respective chapters of Section IV.)

PERCUSSION.

Physical bodies when struck or percussed give forth sounds which vary according to the composition of the body struck. An artisan often strikes an object to determine whether it is hollow or solid. In like manner the physician percusses a portion of the human body in order to learn from the sound elicited the physical condition of the parts beneath. The object of percussion is to estimate the degree of resistance to the percussing finger and to elicit sound. The quality of the sound indicates the proportion of air to the solid tissue contained in the organ. By it can thus be determined the outline of the organ ; the presence or absence of disease causing abnormal physical conditions ; and the presence of tumors or other solid structures.

Method of Procedure.

Percussion may be performed with the fingers, with artificial instruments, or with a combination of both. The blow may be struck upon the part directly (*immediate percussion*), or upon a medium interposed between the part percussed and the percussing finger or instrument (*mediate percussion*). The blow given may be light (*superficial percussion*) or forcible (*deep percussion*). The sounds elicited may be listened to through a stethoscope placed near the part percussed (*stethoscopic* or *auscultatory percussion*).

Due attention should be paid to the presence or absence of tenderness, which necessarily modifies the results obtained by this method of exploration. Definite information can be secured by light percussion even

when there is a good deal of tenderness. In children percussion should be the final step in the examination.

Immediate Percussion. The part may be tapped directly by the finger, hand, or instrument. This was the original method of percussion, and is known as the *immediate* or *direct method.* When the fingers are employed, it is known as *palpatory percussion;* one finger is sufficient. The blow may be given with the finger in the extended position or bent at a right angle. By this method the sense of resistance is better appreciated. Immediate percussion is the method usually employed for bony structures such as the clavicle.

Mediate Percussion. The method now usually employed is that in which a medium is placed between the part percussed and the finger or instrument used for percussing. This medium is called a *pleximeter,* and may be either a plate of ivory, glass, hard rubber, or wood, of suitable size to place between the ribs, or, better still, a finger of the hand not engaged in tapping. The *plessor* is the finger or instrument that strikes the blow. It may be a small hammer. The best instrument is a double cone of caoutchouc inclosed by a metallic ring, to which is attached a rod of metal with a handle of convenient length, weight, and size. (Flint.) For purposes of class demonstration a plessor of this character, with an ivory pleximeter, is of value; but for bedside-work the fingers are better.

Technique. The examiner should stand or sit directly in front or behind the patient. In percussion of the chest, beginning anteriorly with the supraclavicular fossæ, and proceeding downward an interspace at a time, *comparison* should be made with the corresponding place on the other side at each step. The axillary portions of the chest and the posterior portions from the supraspinous fossæ to base should then be examined in the same way. A series of blows (five or seven) should be struck in rapid succession.

The Use of the Pleximeter. The pleximeter must be placed in close contact with the surface of the part percussed. For instance, if the finger is used as a pleximeter in percussing the anterior portion of the chest, it must be placed parallel with the ribs and not across them. If it is not in close contact with the chest, the cushions of air between the two will modify the sound. Interspace after interspace from above downward should be percussed in the manner described. At the same time, if necessary, the pleximeter may be placed over the corresponding ribs, but it must be parallel with them. With a little practice the method of applying the pleximeter can soon be acquired.

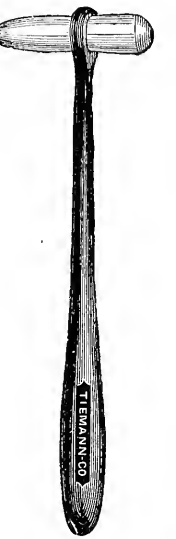
FIG. 86.

Flint's pleximeter.

The Use of the Plessor. This requires considerable practice. If a metal instrument is used, care should be taken to acquire the habit of percussing under all circumstances with the same degree of force. If the finger of the operator is employed as a plessor, several points in the procedure must be remembered. It is better to use one finger, preferably the middle finger. Some operators use more than one finger, but with practice a sufficient degree of force can be given with one, usually the

middle finger. The finger should be bent at a right angle and kept in a fixed position. It must be made to strike the plexímeter perpendicularly; if the blow is given at any other angle to the part percussed, a true sound can not be obtained. The blows must be of regular and even force. The character of the part investigated will determine the degree of force that should be used. (See Method of Percussion, page 381.) The blow must be struck from the wrist; neither the arm nor the forearm comes into play. For light percussion the motion of the finger alone may suffice. The blow should be quick and rebounding like that of a piano hammer, the percussing finger being withdrawn instantly the blow is given.

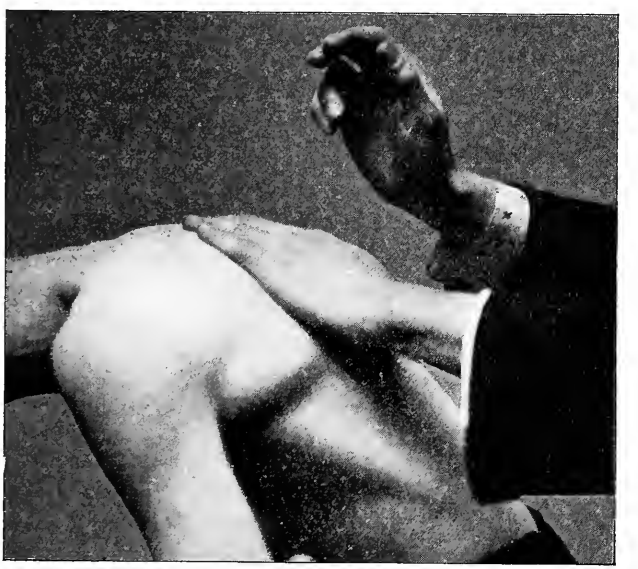
Fig. 87.



Flint's plessor.

Position of the Patient. The best position is the standing one, with the arms hanging loosely at the sides, the head straight and pointing forward but not thrown back, and the shoulders on a level and slightly bent if they are inclined to do so. The muscles must be thoroughly relaxed; any position that throws the chest muscles into contraction produces a muscular tension which modifies the percussion-note. While percussing the posterior portions of the chest it is desirable to have the patient

Fig. 88.



Percussion—showing method of delivering the blow.

stoop forward with his arms folded. While this renders the muscles more tense, it has the advantage of exposing a larger portion of the

chest. When the patient is confined to bed, if not too ill, he should be allowed to sit up during percussion, as contact with the bed-clothes or with the pillows deadens the sounds elicited. This fact should be borne in mind when from any cause it is not desirable to have the patient sit up. If the body be recumbent, it should be as level as possible. The clothing should be removed if possible; although a thin undershirt may be permitted from motives of delicacy, and only part of the chest need be exposed at a time if there be danger of a chill. Percussion over the anterior portion of the chest while the patient leans against a door, a board partition, or a lathed wall, intensifies the resonance. (Flint.) More delicate differentiation of the notes obtained on percussion can be made if the patient stands on a box which has an end out and which thus acts as a resonator. (J. Solis-Cohen.)

Combined Hearing and Feeling. By another excellent plan information is secured by the sense of touch as well as by the sound. The second, third, and fourth fingers of the percussing hand are flexed at an angle of 45 degrees and their tips are brought down on the pleximeter finger and kept there for a few seconds, when the blow may be repeated. The perpendicular blow is not used and the sound produced is not loud. This plan is most useful in diseases of the lungs, spleen, and liver, and where strong percussion cannot be used, as in perityphlitis and cholecystitis.

Superficial and Deep Percussion. The depth and extent of tissue thrown into vibration by percussion depends usually upon the force of the blow. In *superficial percussion* the blows are directed lightly over the part, so as to bring out the sound yielded by the portion directly underneath the surface. Hence, superficial percussion over the thinner portions of the lung covering the heart will give resonance, known as superficial resonance; while percussion over that portion of the heart not covered by the lung will give dulness, called superficial or *absolute dulness.* Light percussion is necessary in children and in patients with sore chest-walls or who have just had a hemorrhage. In *deep percussion* the blows are given with sufficient force to throw the more deeply situated parts into vibration. Deep percussion over the portion of the heart overlapped by the edges of the lung therefore will give dulness, known as deep or *relative dulness.* Deep percussion consequently is necessary in defining the limits of a deep-seated consolidation or of an aneurism covered by the lung, and in determining the upper limit of the liver-dulness and the relative area of dulness of the heart.

Auscultatory or Stethoscopic Percussion.—This consists in placing the stethoscope over the organ the border of which is to be defined, and beginning to percuss some distance from it, but moving toward it. The dull sound given forth by the non-resonant structure is transmitted to the ear beyond the limits determined by ordinary methods. Auscultatory percussion is a valuable means of defining the exact outline of a dull area, as an aneurism or tumor within the chest, or of determining the limits of organs even of similar physical structure. To distinguish the tympany of the stomach from the tympany of the colon the stethoscope is placed over either of the organs and immediate percussion is made with the

ness implies the interposition of an air-containing structure between an airless structure and the chest-wall. The portion of the heart or liver in contact with the chest-wall yields absolute dulness when percussed; that portion overlapped by lung yields relative dulness. Absolute dulness is readily elicited, and all observers will usually obtain the same area of absolute cardiac dulness. Relative dulness, however, depends so much upon the method of percussion, whether light or strong, and upon the examiner's ear, that its extent will be determined differently by each observer, the personal equation being a disturbing factor. In disease of the lungs, of the bloodvessels, and of the mediastinum the location of the lesion is usually made out by the detection of relative dulness, or of changes in the pitch, quality, and duration of the sound, all of which indicate less air in the part percussed.

Tympany. This is the term applied to the sound that is produced when a single air-containing cavity with smooth walls is percussed. In health it can be elicited over the empty stomach, over the large intestine, and at times over the small intestine. In addition to a variable pitch and large volume, it possesses a peculiar metallic quality which is best described as "hollow." It is a quality of sound with which the student should become familiar, for variations are characteristic of abnormal physical conditions in the lungs and in the abdomen. It must be remembered that tympany can be developed normally over the posterior portions of the lungs of infants and children.

The *tracheal tone* is a clear tone produced over the trachea when the mouth is moderately open. It is clear, higher in pitch than resonance, and of a tympanitic or tubular quality.

The Degree of Resistance.

This is estimated by the sense of touch. When air-containing organs are percussed, the resistance appreciated by the finger percussed is small, or indeed may be said to be absent entirely. The sensation to the finger is as if the parts underneath bound away. With a decrease in the air and an increase in the proportion of solid structure more resistance is felt. It is of the greatest importance to educate the finger carefully to this sense of resistance. It is often difficult to determine the pitch exactly, and the sense of resistance furnishes an additional means of detecting the presence or absence of solid structure. Palpatory percussion indicates the sense of resistance better than any other method.

Percussion of the Healthy Lungs and Pleuræ.

Modifications of the Sounds in Health. The degree of clearness or resonance differs in various parts of the thorax. It is purer in the upper axillary region, at the angle of the scapula, and on the anterior surface of the chest, in the second interspace. It is slightly higher in pitch at the right apex than at the left. It is modified by the condition of the chest-wall. Thick chest-walls, accumulations of fat, the mammary gland, and the scapulæ impair the resonance and necessitate deep percussion to bring

out the true sounds. In persons with thin chest-walls the resonance is clear and more pronounced. The percussion-note is also modified by the elasticity of the chest-walls. In the aged, because of rigid chest-walls, it is less clear. In children, in whom the chest-walls are elastic, the resonance is much fuller or clearer. The sounds therefore vary, within certain limits, in different individuals with perfectly healthy, normal chests. Moreover, a sound normal in one part of the chest may in another part indicate disease.

Percussion-sounds have consequently no absolute value; their significance depends upon the individual and upon the part of the chest examined. The student should learn from the outset to compare the sounds developed by percussion of symmetrical portions of the chest, and thus determine the normal for the individual. Below the third rib on the left side, however, the dulness of the heart destroys the value of comparative percussion. Under the right clavicle in health the pitch is higher than under the left, but the sound is not dull in character. The student may become familiar with the pitch, and with alterations in it, by percussing over a clearly resonant portion of the lung, as in the third interspace, and thence downward on the right side. As the interspaces in apposition with the liver are reached the pitch changes. The sound is lessened in fulness; it becomes more shallow. The increase in the rapidity of the vibrations can almost be appreciated, and with it the heightened pitch caused by it is recognized. This normal increase in pitch is due to a thin layer of lung tucked in behind the solid liver. Change in pitch makes it possible to outline organs and pursue *topographical percussion.*

AUSCULTATION.

In the act of breathing sounds are produced which are heard by the ear applied directly to the chest-wall or through some medium. They are created both in inspiration and in expiration. They vary in character in accordance with the situation and with the conditions producing them.

Methods of Auscultation.

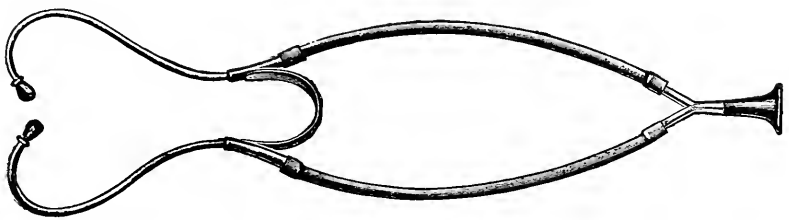
If possible, the patient should stand or sit upright in an easy, unrestrained position. During ascultation in front the arms should hang easily by the side. During auscultation behind, the patient should fold the arms and lean forward. For comparison, both sides should have the same freedom of movement, which would not be attained if the patient assumed a twisted or constrained attitude. Auscultation should be practised during quiet, full, and forced inspiration and expiration.

Auscultation may be practised by one of two methods. In the *immediate* or *direct* method the ear is applied directly to the chest, a thin towel or napkin free from starch alone intervening. This method is of service in ascertaining the general character of the sounds, but has the disadvantage of imperfect localization. The *mediate* or *indirect* method is practised by means of the stethoscope and phonendoscope; but it is disadvantageous in infants, because they can not be kept quiet or are sensi-

tive to its pressure, and in children, because to them instruments are alarming.

The disadvantages of the indirect over the direct method of auscultation are appreciated when it is necessary to localize sounds. By means of the stethoscope it is possible to ascertain the definite localized area in which the sound is produced, and to differentiate sounds in close proximity. In addition, the operator is more likely to escape from contagious diseases and vermin. Moreover, the stethoscope is preferable on the score of delicacy.

FIG. 91.



Binaural stethoscope.

The Stethoscope. The stethoscope consists of a bell which is placed on the patient's chest, of one or two ear-pieces, and of a tube connecting the bell with the ear-piece or pieces. The tube may be rigid or flexible. Stethoscopes may be single and double, and vary in form with the preference of the operator. It should be an absolute rule with the student to become familiar with and to use but one form of stethoscope.

FIG. 92.

Stokes' single stethoscope. (One piece.)

FIG. 93.

Arnold's flexible single stethoscope.

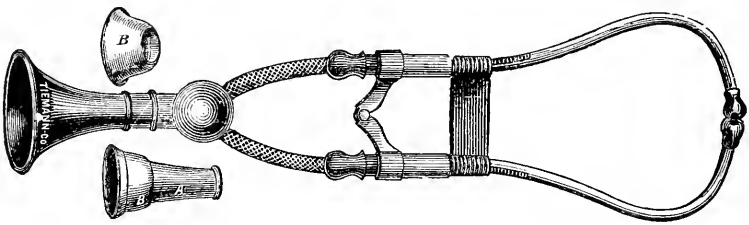
FIG. 94.

Hawksley's single stethoscope. (Two pieces.)

The *single stethoscope* has but one ear-piece. It is very good to localize and determine the relation of sounds. When stiff it also transmits the shock of an aneurismal vessel or of the heart. The objection to the stiff single stethoscope is that the weight of the head causes pain if the

chest is sore, and the pressure of the instrument may modify the sound in auscultation of bloodvessels or structures in close proximity to the ear, as the pleura. In the use of it the student should be particular to see that the portion applied to the chest is perpendicular to the plane of the area over which auscultation is practised, as slight tilting of the instrument will transmit outside noises through the tube. The operator should

FIG. 95.

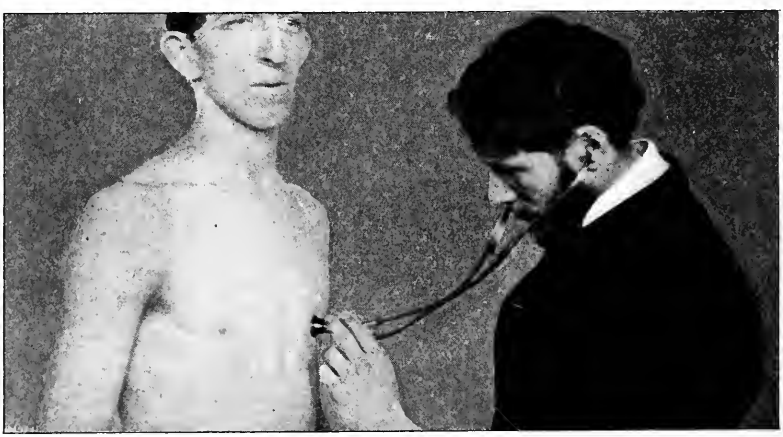


Cammann's double stethoscope and Snelling's rubber bell.

place himself in an unconstrained position and see that his head is accommodated to the position of the instrument, not the latter to the head. If the parts over which auscultation is practised are covered with hair, an extraneous sound from friction is produced. This may be obviated by the application of oil.

The *double* or *binaural stethoscope* has two ear-pieces. It is the most suitable for class instruction. It can even be applied over parts that are

FIG. 96.



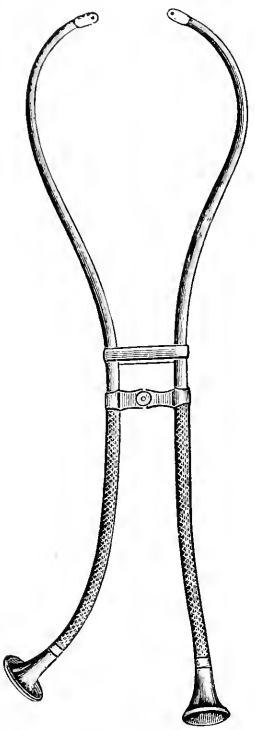
Listening to the heart-sounds.

quite tender. The rule of application to the chest is the same as for the single stethoscope. The ear-pieces should fit comfortably. The humming sound in the tube is confusing at first.

The *differential double stethoscope* has two bells, the sound from each being conveyed to a different ear.

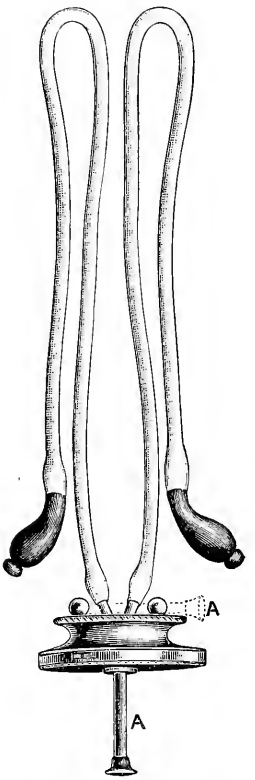
The *phonendoscope* consists of a flat circular metal box enclosing an air-space which communicates by means of tubes with the ears of the observer. The bottom of the box is formed by a hard-rubber diaphragm capable of vibrating easily. A stouter disk, to which a solid metallic

Fig. 97.



Allison's differential stethoscope.

Fig. 98.



Phonendoscope: A, metallic rod.

rod may be screwed, can be slipped over the inner rod disk. With the phonendoscope the intensity of the sounds is increased.

In the *multiple stethoscope* or phonendoscope, for the use of classes, several sets of ear-pieces are attached to one chest-piece by means of branched tubing.

Errors to be Avoided in Ausculting. In ausculting it is necessary to disregard external noises, sounds produced by friction of the stethoscope or towel upon the skin or of the fingers upon the stethoscope, the creaking of a starched towel, noises produced in the stethoscope itself, and muscle-sounds. One must avoid tilting the bell, allowing the tubes to become blocked, kinked, or disconnected, or inserting the ear-pieces upside down.

Graphic Records of Physical Signs.

In order to draw accurate conclusions from the various data obtained during the physical examination of a patient the physician must carry in his mind the results of the inspection, the palpation and percussion, and the auscultation of each individual part of the thorax and abdomen. For the beginner the grouping together of these phenomena according to regions of the body, instead of by methods of examination, is extremely difficult. He is taught to examine the thorax first by inspection, then by palpation and percussion, and finally by auscultation; and in following this routine the results of the examination naturally divide themselves into the signs obtained by this or that method. In making the diagnosis, however, the grouping must be rearranged, for in order to determine the condition of a certain organ or part of an organ *all* the local phenomena, by whatever method recognized, must be considered in their relation to one another and not merely as isolated facts. By weighing all the evidence obtained by the various methods of examination, and by balancing the relative importance of this sign or that, a verdict is finally reached in regard to the condition of the part in question. Only after the status of each organ has been thus separately determined can a complete diagnosis of the case be made with certainty.

In describing in the text the physical signs of the various diseases of the internal organs it is necessary, in order to avoid endless confusion, to consider data in the order in which they are elicited—*i. e.*, grouped according to the method of their recognition. To redescribe them grouped according to regions would involve constant repetition, and would still fail to give a clear picture of the sign-complex of the part. And yet it is essential that this picture should be so clear and well defined that in summing up the examination the physician has but to glance at the part in order to call to mind all the various data obtained by its examination. Experience adds daily to the facility with which this piece of mental gymnastics is performed, and it finally becomes half-automatic, but for the beginner it is most discouragingly difficult. He may, however, obtain great assistance in acquiring the right habit of thought by systematically writing down each sign *as it is perceived*, and by grouping with it the other signs belonging to the same region. This he may do by means of short descriptions; or, better still, he may employ symbols to represent the various sounds, etc., and may mark them directly on the patient's body, or may fill them in on blank diagrams of the thorax and abdomen, and thus obtain a complete and vivid picture of the results of the examination of each separate region. The practical value of this method, both as an aid to the beginner and as an easy and accurate means for preserving records, has been widely recognized, and numerous symbols have been devised to represent graphically the various physical signs. Those suggested by Wyllie, of Edinburgh, and by Sahli, of Bern, are among the best. Many of the symbols used in the following pages will be recognized as borrowed from the above-named authors.

PLATE IV.

FIG. 1.—Anterior Aspect.

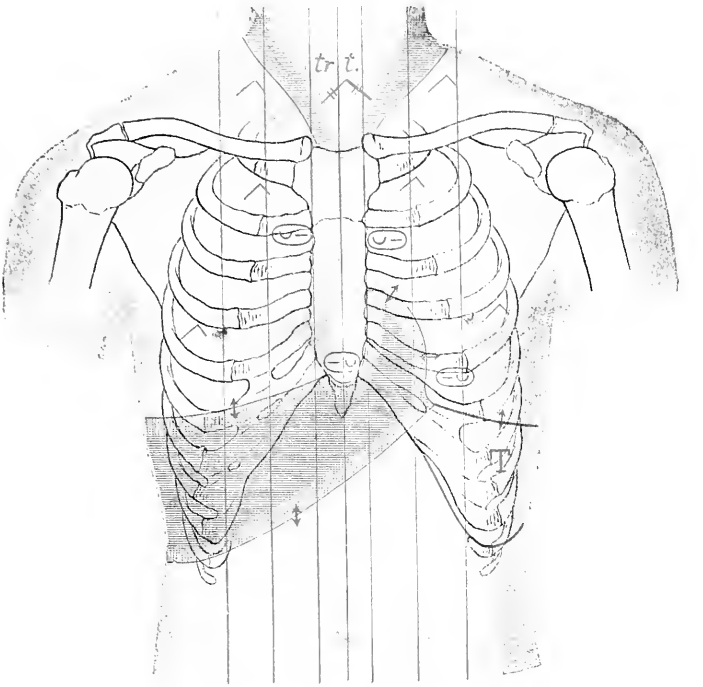
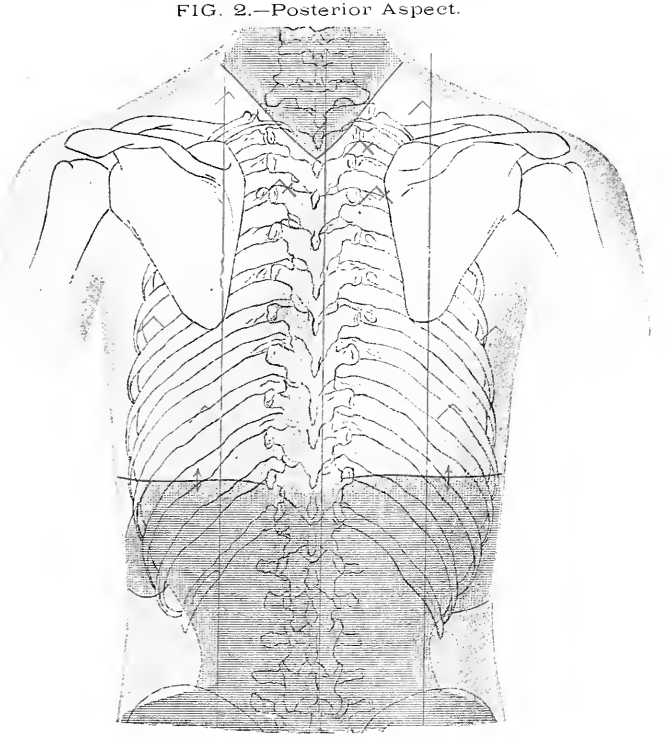


FIG. 2.—Posterior Aspect.



Physical Signs in Health.

Normal percussion outlines of the Viscera. Normal heart and breath sounds. Vertical lines for localization.

Explanation of the Symbols Used in the Plates Illustrating Special Diseases.

Percussion-sounds. Superficial dulness (also called absolute dulness) is alone indicated in the following illustrations. As has been stated, the personal equation enters so largely into the determination of the extent of deep (relative) dulness that it is scarcely possible to make any positive statements in regard to the areas over which it is obtained in health and in disease. Absolute dulness is, on the other hand, easily recognized, and it is, therefore, far better that the student first become thoroughly familiar with this, about which there can be little or no question, before being taught what, in the case of relative dulness, is after all merely the expression of the individual skill and acuteness of ear of the instructor. With a clear picture of the areas of superficial dulness once firmly fixed in the mind, the student should determine for himself just how far he individually is able to rely upon his perception of deep dulness. As his skill in percussion increases and his ear becomes better trained, he will find himself progressively better able to make use of deep dulness as an aid in diagnosis. He should, however, remember that many skilled diagnosticians are content to rely almost exclusively upon superficial dulness.

Blue shading = Areas of superficial dulness; the intensity of the color expresses the intensity of the dulness.

HR = Hyper-resonance.

T = Tympany; the pitch is indicated by a dot above or below the letter.

Breath-sounds. An ascending line indicates inspiration; a descending line, expiration. The length of the line shows the length of the sound; the thickness, its intensity. A dot above or below the line indicates high or low pitch. Two cross lines are used to designate bronchial breathing; a single cross line indicates bronchovesicular breathing. An interrupted line stands for cog-wheel or interrupted breath-sounds.

 = Normal vesicular breath-sounds.

 = Weak vesicular breath-sounds.

 = Harsh vesicular breath-sounds (puerile breathing).

 = Harsh vesicular inspiration, prolonged vesicular expiration.

 = Sharp vesicular inspiration, slightly prolonged vesicular expiration.

 = Interrupted (cog-wheel) breath-sounds.

 = Bronchial breath-sounds (bronchial breathing), inspiratory and expiratory.

 = Bronchovesicular inspiration, low-pitched bronchial expiration.

Râles. **Dry râles** are represented by undulating lines, the length corresponding to the duration, while a dot above or below the line indicates the pitch.

 = Sonorous râles.

 = Sibilant râles.

Moist râles are represented by circles the diameter of which indicates the size of the râles. An ascending line drawn through the circle shows that the râle is heard during inspiration, a descending line that it is heard during expiration. The clear, sharp, moist râles heard over consolidated areas—râles with over-tones—are indicated by large or small dots, according to their size.

 = Small, moist (subcrepitant) râles.

 = Medium-sized moist râles.

 = Large moist râles heard during both inspiration and expiration.

 = Large and small moist râles.

 = Small moist râles heard over consolidated areas.

 = Medium-sized moist râles heard over consolidated areas.

 = Large moist râles heard over consolidated areas.

 = Large and small moist râles heard over consolidated areas.

Crepitation.

 = Crepitant râles, to be heard only during inspiration.

Friction-rub.

 = Friction-rub, as heard over any serous surface.

Heart-sounds. The symbols used to indicate the feet in Latin poetry are made to represent the heart-sounds. The straight line indicates the first or systolic, the curved lines the second or diastolic sound. The thickness of the line shows the relative as well as the absolute loudness. The length of the line and the extent of the curve indicate the length of the sound.

 = Normal heart-sounds.

 = Long loud first sound.

 = Normal first sound, accentuated second sound.

 = Loud first sound, reduplicated second sound.

 = Reduplicated first sound, accentuated second sound.

Murmurs. Murmurs are represented by short parallel lines either increasing or diminishing in length according as the murmur increases or diminishes in intensity. The thickness of the lines shows the loudness of the murmur; the number of lines shows its duration.

 = A soft murmur, commencing distinctly and gradually fading away.

 = A loud murmur of the same character.

 = A short loud murmur, increasing in intensity (type of presystolic murmur).

 = Loud first sound, slightly accentuated second sound; short loud presystolic murmur, increasing in intensity to end with the first sound; long, soft, systolic murmur.

Fremitus.

F+ = Increased fremitus.

F— = Diminished fremitus.

No**F** = Absent fremitus.

Other Symbols.

X = Impulse.

M = Margin (of an organ).

R = Retraction.

B = Bulging.

v = Visible.

p = Palpable.

Xvp = Visible and palpable impulse.

Mvp = Visible and palpable margin.

CHAPTER XXXIV.

PHYSICAL DIAGNOSIS OF DISEASES OF THE HEART AND BLOOD-VESSELS.

Topographical Anatomy. Outline of Heart on Chest-wall.[1] (See Plate V.) To have a general idea of the form and position of the heart, map its outline on the wall of the chest as follows:

a. To define the base—*i. e.*, the part to which its great vessels are attached—draw a transverse line across the sternum corresponding with the upper borders of the third costal cartilages; continue the line half an inch to the right of the sternum and one inch to the left.

b. To find the apex, mark a point about two inches below the left nipple, and one inch to its sternal side. This point will be between the fifth and sixth ribs.

c. To find the lower border (which lies on the central tendon of the diaphragm, draw a line, slightly curved downward, from the apex across the bottom of the sternum (not the ensiform cartilage) as far as its right edge.

d. To define the right border (formed by the right auricle), continue the last line upward with an outward curve, so as to join the right end of the base.

e. To define the left border (formed by the left ventricle), draw a line curving to the left, but not including the nipple, from the left end of the base to the apex.

Such an outline shows that the apex of the heart points downward and toward the left, the base a little upward and toward the right; that the greater part of it lies in the left half of the chest, and that the only part which lies to the right of the sternum is the right auricle. A needle introduced in the third, fourth, or fifth right intercostal space close to the sternum would penetrate the lung and the right auricle. A needle passed through the first intercostal space close to the right side of the sternum would pass through the lung and enter the superior vena cava above the pericardium.

The best definition of that part of the præcordial region which is less resonant on percussion was given by Latham years ago in his "Clinical Lectures": "Make a circle of two inches in diameter round a point midway between the nipple and the end of the sternum. This circle will define sufficiently, or for all practical purposes, that part of the heart which lies immediately behind the wall of the chest and is not covered by lung or pleura."

Valves of the Heart. The aortic valve lies behind the third intercostal space, close to the left side of the sternum.

[1] Holden. Landmarks, Medical and Surgical.

The pulmonary valve lies in front of the aortic behind the junction of the third costal cartilage with the sternum, on the left side.

The tricuspid valve lies behind the middle of the sternum, about the level of the fourth costal cartilage.

The mitral valve (the deepest of all) lies behind the third intercostal space, about one inch to the left of the sternum.

Thus these valves are so situated that the mouth of an ordinary-sized stethoscope will cover a portion of them all if placed over the sternal end of the third intercostal space on the left side. All are covered by a thin layer of lung; therefore we hear their action better when the breathing is suspended for a moment.

Physiology. Action of the Heart. The heart beats—that is, alternately contracts and dilates or relaxes—65 to 85 times per minute in an adult. In females the frequency varies from 75 to 85; in males, from 65 to 75. With each beat blood is propelled through the vascular channels of the body and drawn from them to the heart-chamber. The first effect is produced by the contraction of the heart, or the *systole;* the second by the relaxation, or *diastole.* Other events, as the act of respiration, contribute to the completion of the outflow and inflow of blood, particularly to the latter.

The completion of the act of contraction and of the act of dilatation make up one revolution of cardiac action or, as it is termed, a *cycle.*

Events of the Cardiac Cycle. The following events make up the cardiac cycle. The act of contraction is the systolic period of the cycle; that of relaxation is the diastolic period. During the *systole* (1) the ventricles contract; (2) the auriculoventricular valves close; (3) the blood is propelled from the ventricles into the arteries, the columns of blood in the aorta and pulmonary artery receive a shock from the impact of the new volume of blood, and their bulk increases. The movement of the blood-wave from this cause and from the contraction of the large vascular trunks produces pulsation of the peripheral arteries, which is known as the *pulse.* The contraction is immediately followed by *relaxation*—the *diastole.* (1) The blood-columns in the aorta and in the pulmonary artery fall back upon the valves guarding their outlets, the aortic and pulmonary valves, and lead to their closure. At the same time (2) the auricles are filled by the blood pouring in from the veins. (3) The auricular muscles contract upon the blood in the chambers, driving it into the ventricles.

The systolic and the diastolic periods of a cardiac cycle are nearly equal in length of time occupied in their occurrence. The systolic period occurs at the same time, or is synchronous with the apex-beat and the carotid pulse, and precedes by a fraction of a second the radial pulse. It is immediately followed by the diastolic period, which therefore follows the carotid and radial pulse.

Inspection.

The Heart. The Method of Examination. The patient should be stripped, and good light should fall directly as well as obliquely on the

surface. The patient can be examined in any position, and indeed for accuracy should be examined both in the upright and in the recumbent posture, particularly when the pulse-rate is taken and when auscultation is practised. The sounds vary frequently in different positions. Some diagnostic significance is attached to these variations. It is necessary sometimes to have the patient lean forward to bring the heart into closer contact with the chest-wall.

The examination should not be confined to the heart and vessels. The reader will remember that in the account of the exterior and of local areas it was pointed out that various abnormal conditions may be due to disease of the heart. In the examination, therefore, of a case of suspected heart disease observation is made of the general and of the local color, as of the lips, the fingers, and the conjunctivæ, to determine the presence of *cyanosis, pallor*, or *jaundice;* of the feet, to discover *dropsy;* the face, to note the appearance of the countenance; the neck, to note the state of the vessels—the veins as well as the arteries; the eyes, to note their prominence and any retinal changes; the thorax, to ascertain the presence of dyspnœa.

The Præcordia. The præcordia is the region of the chest which overlies the heart. In the study of the appearance of the præcordia we observe: 1. The degree of *prominence* or swelling. 2. The *impulse* and other pulsations. 3. The *interspaces*. 4. The *color* of the surface.

The Prominence. The præcordia may be unduly *prominent* in children who have had rickets and possibly some cardiac hypertrophy in childhood. The prominence persists in later life. The ribs as well as the soft tissues are prominent. The lower end of the sternum may project. Swelling also occurs in hypertrophy or dilated hypertrophy of the heart, in pericardial effusions, localized pleural effusions and pointing empyema, and in aneurisms in the region of the heart. In pericardial effusion the ribs and interspaces project. The latter are full or even with the surface. The prominence of cardiac disease is observed between the third and seventh ribs on the left side, and extends from the left nipple to the sternum, and even as far as the right nipple. The distance from the middle of the sternum to the midaxilla is greater on the left than on the right side. Local bulging may be seen at the apex in cases of aneurism of the heart.

The præcordia may be *sunken*. Old pericarditis, but more frequently old empyema, causes sinking in of the region. It may be a result of *rickets* or of spinal curvature, and occurs in the condition known as "funnel-chest."

The Cardiac Impulse. THE APEX-BEAT AND THE DIFFUSE HEART-BEAT. The normal impulse produced by the heart striking against the chest-wall may be studied as the apex-beat and the diffuse heart-beat. By the latter is understood the visible and palpable shock transmitted to the entire præcordia by the movement of the heart. This is usually not to be observed in health. With each contraction of the heart, however, there is noticeable in the fifth interspace, just inside the midclavicular line, a circumscribed area, from three-fourths to one inch in breadth, that rhythmically rises and falls. This is known as the apex-beat,

although it is a portion of the heart inside the true apex that strikes the chest-wall. It can readily be detected by inspection with a good light, even in patients with moderately thick chest-walls. It occurs coincidently with the contraction of the ventricles, and is therefore systolic in time. Both the apex-beat and the diffuse heart-beat may be conveniently studied together as the *cardiac impulse*. In general that which alters the apex-beat alters also the diffuse heart-beat. In studying the cardiac impulse we note (1) its position, (2) its extent, (3) its strength, and (4) its rhythm.

The Position of the Cardiac Impulse. *Changes of Position in Health.* The apex-beat is not a fixed point in health. It moves with the movements of the body, and hence when the trunk is inclined to the left it falls toward the left axilla as far outward as the midclavicular line or even beyond that point. It moves toward the right and downward in full inspiration, or may disappear entirely toward the completion of that act. It may not be observed if there is a large amount of subcutaneous fat, or if the mammary gland intervenes. It becomes more conspicuous at the end of expiration or when the body is inclined forward. In children it is higher (fourth interspace) and more to the left. It is depressed in old people. It must be remembered that in transposition of the viscera the position of the impulse is changed.

Change of Position in Disease. The apex-beat, or the lowest point of impulse, may be *displaced* to the right or left, and upward or downward. These changes are due either to (1) *disease outside of the pericardium*, to (2) *disease within the pericardium*, or to (3) *disease of the heart itself.*

1. *Displacement to the Left.* This occurs from (*a*) *Alterations outside of the Pericardium.* When the right lung is the seat of extensive compensatory emphysema, or the right pleura is filled with a large effusion, the impulse is displaced to the left. On the other hand, fibroid phthisis localized at the apex of the left lung, or pleural adhesions which have become attached to the pericardial sac, with probably coincident pericarditis, pull the heart to the left, thereby changing the position of the impulse. In disease of the mediastinum the heart is pushed downward and toward the left. An aneurism, an abscess, or enlarged glands in this situation may invade the normal cardiac territory and cause dislocation of the heart.

In disease of the abdomen the impulse is displaced. If the liver and spleen are enlarged, or if the abdomen is distended by ascites, the diaphragm is raised, and, therefore, also the heart. The impulse is then seen to the left of the normal position, and may be one or two interspaces higher than normal. A common physical change in the stomach—dilatation—is a frequent cause of displacement of the impulse. The dilatation may be temporary from flatulence or may be due to organic disease.

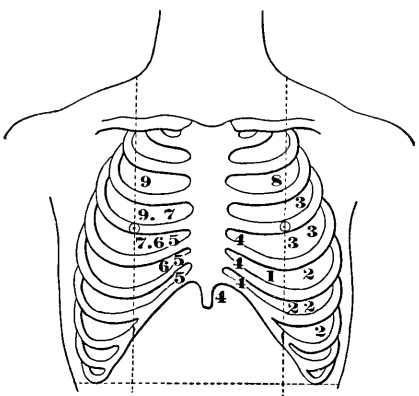
(*b*) *Alterations within the Pericardium.* In cases of pericardial effusion the impulse is shifted to the left and upward. It is seen in the fourth and even as high as the third interspace, and sometimes an impulse is noted only in the second interspace. This, however, is not the true apex. Instead, we undoubtedly see in pericardial effusions the

impulse of the right auricle and the conus arteriosus against the chest-wall.

(*c*) *Diseases of the Heart.* The apex-beat is displaced to the left in dilatation and hypertrophy of the heart. In the latter it is also displaced downward. It may be as low as the sixth or seventh interspace and extend as far to the left as the anterior axillary or the midaxillary line.

2. *Displacement to the Right.* (*a*) *Alterations outside of the Pericardium.* The heart is pushed to the right in left pleural effusion, and in emphysema of the left lung. We find, moreover, in pleural contractions and fibroid phthisis of the right lung the heart drawn to that side.

FIG. 99.



Normal and abnormal impulses.

1. Normal position of apex-impulse. 2. Various points of displacement to left and downward. 3. Various points of displacement to left and upward. 4. Impulse from enlarged right ventricle. 5. Displacement to right. 6. Dilated right auricle. 7. Displacement in fibroid phthisis. 8. Impulse of conus arteriosus. 9. Fibroid phthisis, right lung.

Under these circumstances the impulse is noted either in the epigastric region along the margin of the ribs, or even to the right nipple-line, or in any interspace from the third to the sixth, along the right edge of the sternum. The impulse in the epigastric region usually represents the hypertrophied right ventricle which commonly attends the lung changes that cause displacement of the apex-beat. The impulse along the right edge of the sternum may be the apex-beat, or the right auricle and the right ventricle brought into apposition with the chest-wall by the cardiac dislocation. The apex or the tip of the heart is in all probability displaced but little beyond the midsternal line. (*b*) The impulse is not displaced to the right in *alterations within the pericardium*, or (*c*) in *disease of the heart.*

THE EXTENT OF THE CARDIAC IMPULSE. In *health* the apex-beat is limited to an area from three-fourths to one inch in diameter. The area may be increased when the individual leans forward, at the end of expiration, and during states of bodily or emotional excitement. It is more evident when the chest-walls are thin, and less when they are thick.

Extent of the Cardiac Impulse in Disease. The area of impulse may be *increased.* The causes are: (*a*) *Disease outside of the pericardium.* The area is increased in chronic phthisis with fibrous adhesions, and in pleural adhesions when the lung is drawn away from the surface of the heart. It is increased when the heart is pushed against the chest-wall, as in aneurism or in diseases of the mediastinum, from inflammation, cancer or other mediastinal growth. The impulse is seen not only in the third and fourth interspaces, but also as high as the second; is not limited to the spaces between the sternum and parasternal lines, but may extend beyond the midclavicular line. It may be not only systolic in time, but also diastolic, presystolic, and systolic, and have the appearance of a peristaltic wave from the base to the apex. In time it coincides not only with contraction of the ventricles, but also with contraction of the auricles and with closure of the semilunar valves. (*b*) *Disease of the pericardium* tends to increase the area of impulse if moderate effusion is present. It will be seen as a diffuse wave occupying the second, third, and fourth interspaces. It is also increased in the presence of pericardial adhesions, without increase in strength. (*c*) *Disease of the heart.* The heart must be enlarged, and hence must either be hypertrophied or dilated. The extent of the impulse varies. In hypertrophy the impulse may be communicated to the sternum, so that the lower part heaves with each contraction. The apex-beat falls below the fifth interspace and toward the left, particularly if the left ventricle is the seat of enlargement. If the right ventricle is hypertrophied, the impulse is very marked in the third, fourth, fifth, and even the sixth interspaces near the termination of the cartilages, or in the epigastrium along the border of the ribs of the left side. It may be seen in this situation in anæmia, particularly in persons whose respirations are habitually shallow. Sometimes, when associated with and displaced by lung disease, it is seen to the right of the xiphoid cartilage.

The Strength of the Cardiac Impulse. The cardiac impulse may be increased or diminished in strength, or entirely absent. The force of the beat varies much in health. It is increased in strength in psychical disturbances, bodily exertion, so-called cardiac neuroses (hysterical tachycardia), in most conditions that increase the rapidity of the heart's action, such as fevers, and in the conditions that have already been detailed as increasing the extent of the impulse. It may be diminished in strength or entirely absent: (*a*) In *disease outside of the pericardium,* on account of which something intervenes between the heart and the chest-wall; hence in emphysema of the lungs and in compensatory emphysema of the left lung the impulse is entirely effaced. (*b*) In *disease of the pericardium* the impulse is absent when there is large effusion; the absence here succeeds the dislocation to the left, and with its effacement the impulse in the second and third interspaces disappears. (*c*) In *disease of the heart* the impulse is absent when the heart is diminished in size, as in atrophy, or in myocarditis, or when weakened by fatty degeneration or dilatation.

We must bear in mind that the cardiac impulse may be entirely absent in health, and too much importance should therefore not be attached to its absence.

The Rhythm of the Cardiac Impulse. Usually the apex-beat is appreciated as a single systolic elevation. Rarely, however, it may be doubled or tripled—that is, to a single arterial pulse there may occur two or three apex-beats (cardiac bigeminia, hemisystole, alternate systole). These conditions require for their proper study inspection, palpation, auscultation, and sphygmographic and cardiographic examinations.

New Impulse. New areas of impulse, the heart not being dislocated, arise from enlargement of one of the cardiac chambers or from disease of the bloodvessels. A new area of impulse in the second or third left interspace may be caused by pulsation of the conus arteriosus or the pulmonary artery; it may be due to exaggerated pulsation of the left auricle such as occurs in mitral stenosis, for instance; it may be occasioned by retraction of the lung in this region; it may be due to hypertrophy and dilatation of the right ventricle; or it may be due to aneurism of the aorta. A dilated right auricle may give rise to a new impulse to the right of the sternum, in the fourth or fifth interspace. Most new impulses to the right of the sternum, however, are due to aneurism of the aorta or of the innominate artery, and they are usually present in the second or third interspace.

The Interspaces. They are retracted possibly from pericardial adhesions; they are full or bulging in effusion. This retraction may be limited to the apex or may occur in each interspace over the præcordial region. It may occur with the systole or with the diastole. It may occur in hypertrophy of the heart, and is then systolic in time. It is of some, although doubtful, diagnostic significance when it is systolic in time, as it is said to indicate adhesions of the pericardium. The traction at the systole of the heart causes the interspaces to be drawn in.

On inspection behind, a systolic retraction of the region corresponding to the left eleventh and twelfth interspaces is seen in adherent pericardium. This is known as *Broadbent's sign*.

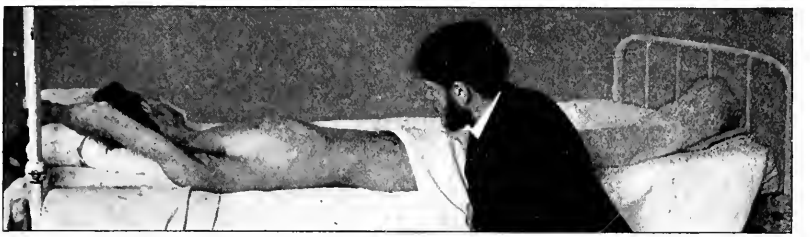
Color of Surface. Only when purulent pericardial effusion is about to rupture or an empyema to discharge do we note redness or other change in hue of the surface of the præcordia not observed over the remainder of the thoracic surface.

The Arteries. By inspection we may be able to determine pulsation or any undue swelling or other change in the course of the vessels. With the exception of pulsation in the carotids, which may temporarily increase under excitement, pulsation of the vessels is not usually seen in health. In old people we can see the pulsation of the aorta (rarely) at the episternal notch, and often in other vessels, the temporals, the innominate, the carotids, the subclavians, the brachial and radial arteries, the abdominal aorta in thin subjects, the femoral arteries, and the posterior tibials.

The Arteries in the Neck. Temporary pulsation of the carotid arteries from excitement has been mentioned. It is commonly seen in anæmia, and is quite marked in exophthalmic goitre. It is striking in aortic regurgitation. It often attends the vascular changes of old age. It is common in conditions of low vascular tension, such as fever states. It may be due to atheroma or aneurism. It is always suggestive of aortic valvular disease. The innominate artery, as well as the carotids, often

pulsates visibly in the neck, and may be so large as to simulate aneurism. The subclavians may pulsate for the same reason; they may also be seen to pulsate if the lungs are consolidated or shrunken by disease. If the patient is young, the throbbing is more likely to be of neurosal or hæmic origin. In later life, if such pulsation is associated with a more or less defined swelling or tumor, with other physical signs of aneurism, that disease is doubtless present.

FIG. 100.



Studying pulsations.

The Thoracic Aorta. An impulse of the *thoracic aorta* is usually from aneurism. The pulsation is not always due to disease; the aorta may be pushed against the chest-wall or the lung structure which overlaps it normally may be withdrawn.

Pulsation due to aneurism must not be confounded with pulsations of the thoracic wall due to pulsating pleural effusion or to those diffused pulsations occurring in anæmia and exophthalmic goitre.

TUMOR. An enlargement or swelling in the course of the aorta may be due to aneurism of that vessel. It must be distinguished from the tumor of mediastinal disease and of empyema.

The Abdominal Aorta. Pulsation of the abdominal aorta is often the cause of serious distress. The violent throbbing keeps the patient awake at night and makes him more and more nervous and irritable. The pulsation is usually seen in the epigastrium. It is more frequent when the vessel is not diseased, in neurasthenic subjects. It occurs reflexly in patients with dyspepsia or organic disease in the upper abdominal tract. The shock of the pulsation is transmitted to the hand with considerable force. The impulse is diffused, but not expansile.

Epigastric pulsation also may be due to transmission of the impulse of the aorta by enlargement of the pancreas or tumors of the stomach or the omentum. The transmitted pulsation is distinct. The impulse is a transmitted one when the tumor can be defined and when a sensation of lifting is transmitted to the hand. The physical signs of aneurism are absent. If the patient lies on the abdomen or in the knee-chest position, the tumor falls away from the aorta and the impulse is not readily transmitted. Epigastric pulsation is also caused by aneurism of the abdominal aorta. The pulsation is distensile or expansile, and the aneurismal sac can be defined at times. The other physical signs of aneurism are usually present—namely: thrill, dulness over the tumor, a murmur on auscultation. In these conditions, however, we can not always rely on the physical signs alone; the history of the subjective symptoms and of

disease of other structures must be carefully inquired into. Aneurism rarely occurs without some evidence of arterial sclerosis or some physical effect upon the circulation. Accentuation of the aortic second sound, variations in the femoral pulse, high arterial tension, and the usual evidences of sclerosis favor aneurism. While functional epigastric pulsation usually occurs in neurotic subjects, and hence in the earlier periods of life, yet such pulsation is frequently seen at the climacteric and in the neurasthenia of old age. Late in life, with such impulse, fibrous thickening about the pylorus or contraction of the omentum may easily be confounded with malignant disease. Cancer of the stomach has been diagnosticated under these circumstances when the pulsation was simply reflex from chronic gastritis.

The diagnosis must be made by carefully weighing all concomitant circumstances and phenomena that surround cancer. (See Symptomatology of Morbid Processes.) *Fecal accumulations* in the colon may be made to heave by the beat of the aorta and cause exaggerated epigastric impulse. The bowels must be emptied before definite conclusions are arrived at.

Some time ago a patient in the Presbyterian Hospital had extreme pulsation of the abdominal aorta, with great local discomfort on account of the throbbing. She was sixty-five years of age, and had within the preceding two years nursed her son through tuberculosis. She failed in health, and came to the hospital emaciated, with chronic gastritis and diarrhœa. On examination a distinct tumor was felt above the umbilicus, which she had been told was due to carcinoma. It was hard and painless; the physical signs of aneurism were not present; the pulsation was extreme. A second tumor, not so large, was felt in the right hypochondriac region. Both tumors were movable, dull upon percussion, and surrounded by tympanitic areas. While it was impossible to be sure of the nature of the tumors, it seemed to me they were tuberculous or simply fibrous, and would not influence the patient's immediate welfare. Under treatment the pulsation disappeared; the gastro-intestinal symptoms were relieved entirely; the patient rapidly gained in weight and strength; the tumors remained, but the outlines became less distinct because the previously scaphoid abdomen had become distended (two years under observation). The question arose: Was the epigastric pulsation due to a throbbing aorta or transmitted by an obscurely defined probably tuberculous mass in that region? No doubt it was the vessel alone that caused the impulse.

An epigastric impulse due to one of the above-mentioned causes must not be confounded with the impulse of hypertrophy of the right ventricle, or with the shock of an hypertrophied or over-acting heart transmitted to the left lobe of the liver. In hypertrophy of the right ventricle or dislocation of the heart from disease within the chest the impulse may be seen to the right or left of the xiphoid cartilage. The symptoms and signs of right-ventricle hypertrophy explain the pulsation.

The Smaller Arteries. By inspection of the arteries beyond the aorta we can often recognize more distinctly the condition known as *arteriosclerosis*. In endarteritis examination of the femoral, popliteal, tibial, brachial, radial, and temporal arteries reveals dilated, tortuous, hard, often

pulsating vessels. Elongation of the artery is seen, so that instead of a straight tube it becomes a sinuous canal, turning and twisting at short intervals. (See Arteriosclerosis.) But pulsation of the above-mentioned peripheral arteries may be due to other causes. In hypertrophy of the left ventricle arterial pulsation is a prominent sign, although more marked in the vessels near the heart, as the carotids. In regurgitation at the aortic orifice pulsation is also frequently seen.

Capillary Pulse. The capillary pulse is seen under the finger-nails or in the skin after hyperæmia is induced by firmly stroking the skin with the nail. It may be seen inside the lips, if a piece of glass is pressed against them. There is rhythmical pulsation of the capillaries, causing alternate flushing and pallor of the surface. When marked, it is significant of aortic insufficiency. An indistinct capillary pulsation may sometimes be observed in apparently healthy persons, and is not rare in fever states and other conditions of low vascular tension.

The Veins. Diseases of the veins are largely surgical and do not frequently come under the notice of the physician. Alterations in the veins from physical causes in the local or general circulation are of frequent occurrence and of the greatest diagnostic significance. The "venous phenomena" are physiological and pathological evidences of the circulation of the blood in the veins.

Examination is limited largely to the jugular veins in general affections of the circulation; to other subcutaneous veins, in addition, in local affections. The examination is made by inspection, to determine the size and degree of pulsation of the veins; by palpation, to confirm the results of inspection and to determine the presence of a thrill; by auscultation, to determine the presence of murmurs.

By *inspection* we note the presence of:

A. Enlargement of the Veins. The change in size may be general or local. In both instances there is interference with the venous return of blood.

1. General Enlargement. General enlargement may be observed in all the veins, but is more readily studied in the *jugular veins* of the neck. Associated with the enlargement, general venous engorgement is observed, and hence œdema (which obscures external veins), cyanosis, effusions in serous cavities, and congestion of internal organs attend the pathological venous phenomena. It must follow that a central disturbing influence upon the circulation is present, and so we find interference with the circulation in the right heart to be the causal factor. This interference is due to dilatation of the right auricle and ventricle, which in turn may have arisen from valvulitis, myocarditis, pericarditis, or, on account of increased pulmonic blood-pressure, from emphysema and other pulmonary obstructions. In rare instances pressure upon the cavæ by a mediastinal tumor may cause general over-fulness of the veins.

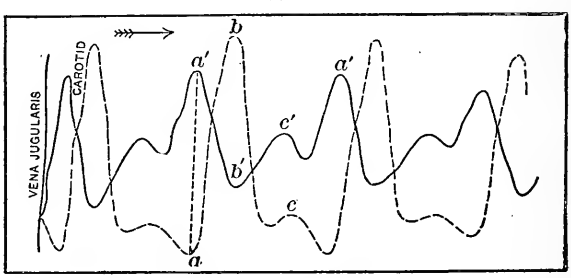
The jugular veins, both internal and external, are seen to be distended even in stout people. The observation can be made better by viewing the head when it is turned away from the side of the vein under examination. The external jugular can almost always be seen; the internal jugular frequently when engorged. They may also be felt under these

circumstances. The position of the veins can be distinguished more readily by observing their relation to the sternocleidomastoid muscle. The internal jugular vein is seen in the inter-sternocleidomastoid fossa, just behind the sternoclavicular articulation. Here the jugular bulb is seen, and at this point in the veins the bulbar valves are situated. When abnormally full, the bulb may project beyond the surface and rise one-fourth or one-half inch above the articulation. The over-fulness is more marked in the dorsal than in the upright posture.

2. Local Enlargements. Local increase in fulness of the veins is due to narrowing or closure of the venous trunk by pressure or by thrombosis. A mediastinal tumor pressing upon the cava will cause abnormal fulness of the *jugulars*. The veins of the *scalp* become distended and tortuous in thrombosis of the longitudinal sinus. Enlargement of the veins of the *arm* or *leg* points to compression or thrombosis of the axillary or femoral vein, respectively. The enlargement is associated with œdema of the respective extremity. Enlargement of the superficial veins of the *thorax* is seen in intrathoracic pressure from tumor or aneurism, rarely in dilatation of the heart. Enlargement of the veins of both *legs* may be due to obstruction of the vena cava or both iliac veins. The latter is liable to occur in pelvic tumors. When there is engorgement of the portal vein, collateral circulation is frequently carried on through the *abdominal veins*. The veins are enlarged; and in some instances the veins about the navel become enormously distended because of a permanently patulous umbilical vein. The crown of veins—*caput Medusæ*—is significant of cirrhosis of the liver and of pyelothrombosis. Enlargement of the veins of the extremities from the causes above mentioned must not be confounded with the unilateral or bilateral varicosity that occurs during and after pregnancy, after prolonged intra-abdominal pressure from other causes, and in inflammation of the veins in the course of septic diseases, as typhoid fever.

B. Pulsation of the Veins. The circulation in the veins differs from that in the arteries. The blood-flow is continuous. Two circumstances modify it—respiratory movements and cardiac action.

Fig. 101.

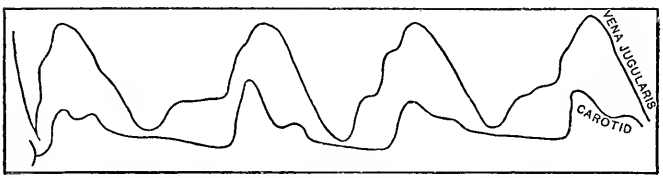


Negative (diastolic-presystolic) venous pulse. (After Riegel and Sahli.)

Pulsation due to Respiratory Movements. The modification is particularly seen in the veins of the neck. During inspiration all of the veins empty rapidly, while in forced expiration, or with strong effort, as

seen in coughing, the discharge from the veins is checked and they become full and even over-distended. When the fulness of the veins is normal, the respiratory alterations are not observed, except the swelling that occurs in severe coughing, as in whooping-cough. When they are abnormal, as

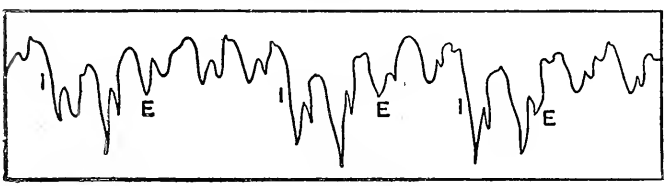
FIG. 102.



Positive (presystolic-systolic) venous pulse. (After RIEGEL.)

from right-sided cardiac dilatation (*q. v.*), they show a corresponding to-and-fro swelling synchronous with respiratory movements. Upon coughing, the jugular bulb may appear as a rounded pulsating bunch between the heads of the sternomastoid muscle. The internal jugular may also swell and contract. Increased pulsation with fulness of the veins is seen during the labored expiration of asthma and emphysema.

FIG. 103.



Illustrating the influence of respiration on the positive (presystolic-systolic) venous pulse of the jugular vein. (After KOVÁCS.)

Alteration of the respiratory movements of the veins is observed in cases of pericarditis or of mediastinopericarditis. Normally the vessels are drawn upon and bent during the act of inspiration—inspiratory collapse. In the above-mentioned pathological conditions they swell in inspiration and empty during expiration, directly opposite to the normal state.

PULSATION DUE TO CARDIAC MOVEMENTS. *The Venous Pulse.* The cardiac movements also modify the movements of the blood in the veins. They cause rhythmical pulsation, or the venous pulse. This may be communicated from the carotids underneath or occur in the veins. The so-called *true* and *false* pulses are thus produced. The true venous pulse is divided into the (1) *negative* and the (2) *positive* pulse, the former being the pulse of health, the latter the pathological venous pulse.

1. *The normal or negative venous pulse* is so designated because it is not due to positive action of the heart causing retrogression of blood. It can be demonstrated by pressure of the finger on the middle of the veins. Pulsation ceases to the proximal side of the compressing finger because the blood does not regurgitate from the heart; there is either no pulsa-

tion to the distal side or the pulsation lessens materially, indicating non-transmission from the carotid. The negative venous pulse is presystolic in time and can only be seen in the external jugulars. The vein collapses during the systole and distends or pulsates before the systole, hence is presystolic or diastolic presystolic. This may be observed by inspection, keeping in view at the same time the apex or carotid pulse. The systolic collapse occurs quickly. The presystolic pulsation follows slowly, with an appreciable interval between the two. The presystolic distention occurs during the time that the auricle is filled with blood; the collapse occurs when the auricle is empty—that is, during the ventricular systole. When the auricle is distended, the flow of blood from the veins is impeded, and hence the jugulars are over-filled. When the auricle is empty, the flow of blood from the veins is favored, hence the vein collapses (the systole).

Diagnosis. The negative venous pulse may be distinguished from pulsation in the artery by the time, by the greater size of the surface-pulsation on account of the greater size of the vein, by the impression of undulation rather than shock received by the finger, by the impression of passive force rather than of active power. Sometimes it is extremely difficult to recognize the normal or negative venous pulse on account of undulations in the veins produced by the blood-flow and transmitted carotid impulse.

2. *The positive venous pulse* is systolic in time. It is sometimes spoken of as being presystolic-systolic in time. It is due to positive action of the heart. It is pathognomonic of tricuspid regurgitation (*q. v.*). When the right ventricle contracts, the regurgitant blood-wave is transmitted into the cava through the incompetent valves. It appears first in the internal jugulars or their bulbs, because of the direct course of the innominate and right jugular from the cava. Subsequently the left may become affected. If the valve in the jugular bulb is competent, the systolic regurgitant wave is seen there only. The pulsation of the enlarged bulb is seen in the inter-sternocleidomastoid fossa. Usually the valve is insufficient or rapidly becomes so, and the systolic back-wave therefore extends upward. The same wave is transmitted to the veins of the liver, causing systolic swelling and diastolic collapse of the liver. These conditions are produced, as previously mentioned, in right-sided dilatation of the heart, providing there are moderate force and slowness of the heart's action. When the heart becomes very weak and rapid, the pulsations disappear.

Diagnosis. 1. The negative, true, or normal pulse is distinguished from the pathological or positive pulse and from the transmitted pulsation by its time. It is timed by the apex-beat or the carotid pulse of the opposite side. The negative pulse (normal) is presystolic, the collapse of the vein systolic; the positive pulse (pathological) is systolic in time. The patient should hold his breath, as increased respiratory movement will modify the venous pulsation. 2. The imparted or *false pulse* is transmitted from the carotids, and can be recognized by stopping the flow of blood by pressing the finger or barrel of the stethoscope on the vein in the middle of the neck after it has been emptied by pressure

upward. If the pulsation is communicated (false pulse), the vein remains empty in the portion nearest the heart and fills up in the peripheral portion, while the pulsation ceases toward the centre (below) and increases in the periphery (above the finger). If the carotid artery is pressed upon as near the heart as possible, the transmitted pulse will cease. In the positive pulse the portion near the heart slowly fills from below upward.

Though a positive venous pulse is properly considered pathognomonic of tricuspid insufficiency, there are two other conditions which at least theoretically may give rise to it: 1. Coincident mitral insufficiency and patulous foramen ovale, of which Ritter has reported a case. 2. Varicose aneurism of the aorta and superior vena cava. Both conditions, however, are of extreme rarity.

Diastolic venous collapse is seen in pericarditis, as observed by Friedreich. The collapse occurs at the time of the cardiac diastole. It is distinguished from the true pulse as follows: compress the jugular vein —pulsation ceases above and below the seat of compression.

Pulsation of other Veins. Quincke has described a venous pulse in the hand and back of the foot in association with the capillary pulse and the *pulsus celer* of aortic regurgitation; it has also been observed in anæmia. It is probably only the arterial pulse propagated through the capillaries. The positive pulse may be seen in the veins of the face, in the cutaneous veins of the arm and hand, in the superficial mammary veins, and in the veins of the legs.

Palpation.

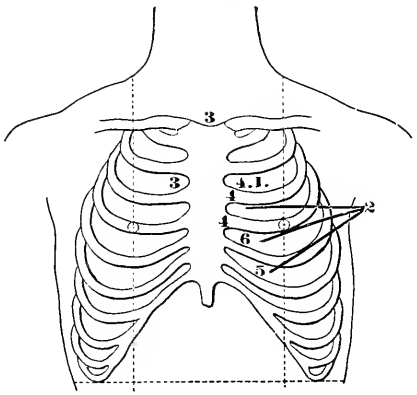
The Heart. Palpation confirms inspection as to the shape of the præcordia, the position and the extent of the impulse, and the condition of the intercostal spaces. In addition, we determine by palpation the character and strength of the *impulse* and the presence or absence of *valve-shock, thrills,* or *friction.* Palpation also reveals *œdema* of the surface and *fluctuation.*

The Cardiac Impulse. In a normal chest with moderately thick walls a slightly prolonged, moderately strong shock is transmitted to the hand when placed over the præcordia. It is synchronous with the cardiac systole and precedes the radial pulse. It is therefore systolic in time. It is stronger when the patient leans forward, and exhales freely, removing the lung from the surface, and when the chest-walls are thin; it is weaker in opposite conditions.

Character and Strength of the Cardiac Impulse. A. *Strength increased.* 1. Overaction. In the violent action of the heart that attends palpitation, and in the increased action in the early stages of fevers or of inflammation, the force of the cardiac impulse is much increased. 2. Disease. (*a*) *Alterations outside of the pericardium.* Increase in the extent of the impulse is attended by increased strength when the heart is hypertrophied or the lung retracted. (*b*) *Alterations within the pericardium.* In pericardial adhesions the heart is held more firmly against the wall and may give the appearance of strength to the impulse.

(*c*) *Disease of the heart.* True increase in force of the impulse is seen in disease of the heart. When the organ is *hypertrophied* or the seat of dilated hypertrophy, the force of the impulse is increased, sometimes to an almost unbearable degree. Uplifting of the præcordial area or even of the lower half the anterior part of the chest is seen. The hand or the head laid over the heart is forcibly lifted with each systolic contraction.

FIG. 104.



Abnormal palpable impulse and thrills.

1. Diastolic impulse palpable from closure of pulmonic valve. 2. Presystolic impulse often seen in third, fourth, and fifth interspaces in mitral obstruction. 3. Thrill at aortic orifice: systolic, obstruction; diastolic, regurgitation. 4. Thrill at pulmonary orifice: systolic, obstruction; diastolic-regurgitation. 5. Thrill at mitral orifice: systolic, regurgitation; diastolic, obstruction; presystolic, obstruction. 6. Thrill at tricuspid orifice.

This great force is most pronounced in the enormous hypertrophy that occurs in cases of aortic regurgitation or in obstruction with endarteritis. It is the impulse and force of the so-called *cor bovinum.* In *dilatation* the impulse is diffused and wavy.

B. *Strength lessened.* This occurs from causes which diminish the extent of the impulse or cause it to be absent entirely, as when some adventitious material intervenes between the heart and the chest-wall, or when the heart is weakened by disease. Hence (following the classification above): (*a*) in emphysema of the lung; (*b*) in pericardial effusions; (*c*) in fatty heart or myocarditis, in dilatation and simple weakness of the heart, the strength of impulse is lessened.

Valve-shock. The shock of the closure of the valves can be felt by the hand when placed evenly over the præcordia. The shock from the pulmonary and aortic valves is best transmitted. It is felt most distinctly in persons with thin chest-walls, and when there is heightened tension either in the aorta or in the pulmonary artery. The shock follows the impulse. It may be localized more accurately with the finger-tips in the third or fourth interspace along the left edge of the sternum. The shock of the auriculoventricular flaps is also transmitted. The shock is synchronous with the first sound. It is felt in the left fourth interspace near the sternum, sometimes over it. It is due to dilatation of the heart, and is felt more readily in thin-chested persons.

Thrills. A thrill is produced when the blood is thrown into vibration by passing over a rough surface. It may be created with the systole or during the diastole. It can be created only at the time blood is passing through the orifices. 1. The most common seat of the thrill is the apex. If the hand is placed in close proximity to the surface of the chest at this point, a vibration or tremor is transmitted to it in most cases of *mitral obstruction.* The blood is passing from the auricle to the ventricle; as this takes place before the systole, the thrill is felt before the impulse or carotid pulse. It is *presystolic* in time. It is sometimes difficult, however, to distinguish it from the impulse. Its character can not well be described. The hesitating, jogging manner of the vibrations or the thrill is clearly transmitted to the hand. Sometimes the thrill continues throughout the entire diastole—the diastolic thrill of mitral stenosis. 2. The next most frequent seat of thrill is the second costal cartilage on the right. Here the thrill or vibration is *systolic* in time and is caused by *obstruction* at the *aortic orifice.* It may be felt away from the heart, in the aorta, or in the carotids. The aortic cusps are thickened, contracted, and stiffened by a sclerotic endocarditis, or the orifice is occluded by valvulitis. The systolic thrill must not be confounded with the thrill elicited over the aorta or at the aortic cartilage in aneurism. 3. Sometimes a thrill is felt at the apex with the systole—*first sound.* This must not be confounded with the before-first-sound thrill. It is never so distinct, and is not made up of a series of vibrations. It is due to regurgitation at the mitral orifice. 4. Rarely a thrill is felt at the second cartilage on the right, with the *second sound.* It may be felt along the course of the sternum also, and is due to regurgitation through the aortic orifice. 5. At the second costal cartilage on the left a thrill is sometimes felt. It is *systolic* in time and is not transmitted. It is due to obstruction at the pulmonary orifice. 6. At the lower portion of the sternum a thrill, systolic in time may also be felt, due to tricuspid regurgitation. Care must be taken not to confound the above-mentioned thrills with those due to aneurism. (See Aneurism.)

Pericardial Friction. In addition to the thrills, a friction or to-and-fro rubbing is transmitted to the hand in cases of pericarditis in the first stage. The friction may be felt all over the heart region, but is pronounced in the third or fourth interspace. It may be detected on slight pressure or only when the tips of the fingers are pressed firmly against the interspaces.

It is important to remember that the *position* of the patient weakens or modifies the thrill or friction. When the patient is lying down, it may not be felt. The upright posture or leaning forward makes it evident, and hence the patient should be instructed to assume this position in the examination.

The Arteries. The results of inspection are confirmed. In addition the artery is examined to determine its tension, the condition of the coats, and the presence of thrills.

Pulsation of Organs. It is said that in aortic regurgitation an arterial liver-pulse similar to the venous liver-pulse can be felt when the hands are placed over that organ. Similar pulsation may be felt over the spleen.

In examining the arteries it is important, as will be detailed in the section devoted to the pulse, to compare the arteries of the two sides. Often the pulse-wave is found to be unequal in force, in volume, and in time. This is almost always due to obstruction to the passage of the blood. When not due to *endarteritis* or *aneurism*, it is due to the pressure of a tumor on the vessel somewhere in its course or to a thrombus or embolus in the artery. A difference between the radial and the femoral pulse points to obstruction in the thoracic or abdominal aorta. Anatomical variations must be remembered.

The Pulse. The pulse is an index of the force, frequency, and rhythm of the heart's action and of the pressure, or tension, which is maintained in the arteries.

General Observations. The *frequency* of the pulse before birth is from 120 to 140 beats per minute. From this time it is diminished in frequency up to adult life, 72 being then accepted as an average; the number of beats, however, is often more and sometimes less than 72. In old age the pulse-rate is again increased. Sex has some influence. The rate is slightly higher in females than in males of the same age.

The frequency of the pulse is subject to diurnal variations, at times corresponding with the diurnal rise and fall of temperature. The rate will, therefore, be highest in the afternoon and evening and lowest in the early morning hours.

The position of the body also has a modifying influence. The pulse is more frequent when a person is standing than when he is sitting, and more frequent when he is sitting than when he is lying down. Walking, running, bodily and mental exertion, fear, and excitement all tend to accelerate the pulse.

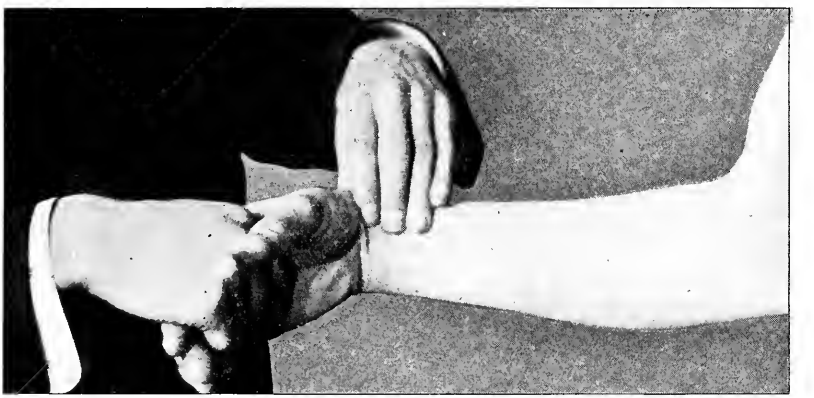
During and for one or two hours after a meal the pulse-rate is higher, especially if an alcoholic or other stimulant, such as coffee, has been taken.

How to Take the Pulse. To make a correct count of the frequency of the pulse, the conditions just mentioned as normally modifying its rate should be borne in mind. If the object of the count is to determine the rate which is normal for a particular individual, several counts will be necessary at different times and under different conditions, such as sitting and standing. The best time for the physician to take the pulse will have to be determined by his own judgment in each case. If the patient comes to his office and is excited by the prospect of an examination, it will be well to wait until he becomes calm. On the other hand, if he is calm at first, a count at that time is to be preferred to one made after he has been disturbed by a physical examination. In the same manner, on visiting a patient at his house, the judgment of the physician must decide whether to count the pulse immediately on his arrival or to postpone it until by general conversation all apprehension and alarm on the part of the patient have been allayed. In general, it may be said that if the physician finds upon his arrival that the pulse is more frequent than the condition of the patient would lead him to expect, he should wait a while, endeavor to find out whether anything has served temporarily to disturb the circulation, and then make the count when the conditions are more favorable. Some patients are so nervous that the

mere act of placing the finger upon the wrist sends the pulse-rate up 10 or 20 beats in a minute. In such cases an effort should be made to obtain a count without the patient's knowledge by observing the pulsations of the temporal or carotid. In other cases it may be well to entrust the counting of the pulse to the nurse or to a member of the family. In infants and young children the pulse may be counted while they are asleep. In febrile conditions the count is more likely to be too high than too low.

In hospital practice, or when a nurse is constantly in attendance, the pulse and respiration should be taken at the same time as the temperature. But the nurse must be warned against taking them under dissimilar conditions upon successive days. For example, the pulse should not be taken one day while the patient is lying down, quiet and comfortable, and compared with the count of the next day when he is sitting up or has just taken some hot liquids, or has had a spell of coughing, or been subjected to some other disturbing influence.

FIG. 105.



Taking the pulse.

The preferable position is the recumbent one in the case of bedfast patients, and the sitting posture in those not confined to bed. Care should be exercised in all cases to see that the patient's position is comfortable, and that nothing obstructs the artery or interferes with the flow of the blood.

The wrist is the place usually selected for feeling the pulse. At this point the radial artery passes over the radius, and can readily be compressed and its character made out. In particular cases it may be advisable to count the pulse at the temporal or carotid artery. The fingers should be applied so that the beats can be most distinctly felt. The beats are counted for fifteen seconds by the second hand of a watch when only an approximate count is desired or when time is a factor, and then multiplied by 4. It is better, however, to count the pulse for half a minute, and still better for a full minute.

The arteries of the two sides must be compared. Differences in force, volume, and time may be due to anomalous distribution of arteries. In

disease such differences may occur in aneurism and atheroma, in pressure on the trunk from external disease, and in embolism and thrombosis.

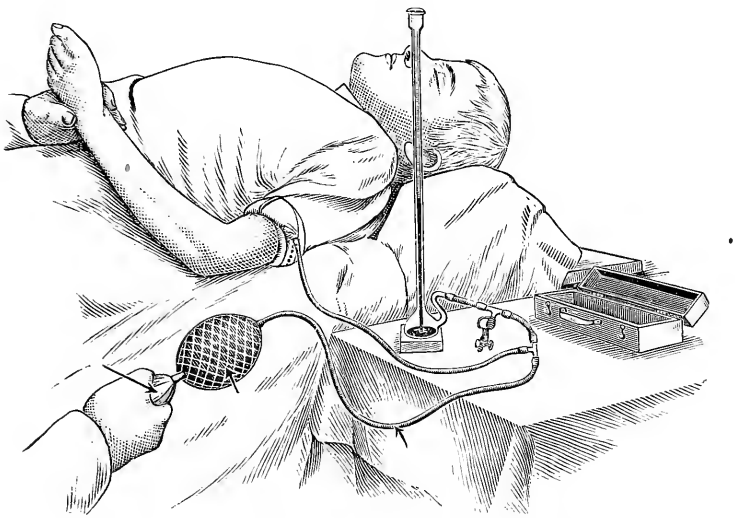
Condition of the Walls of the Artery. The condition of the artery is often of more importance than the pulse-rate. A healthy radial artery, in a person not advanced in years, can be compressed easily against the radius without the finger being able to differentiate the artery from the other tissues. But as age advances, and also as the result of certain constitutional diseases—syphilis, gout, chronic endarteritis, alcoholism, and others—the artery tends to become thicker, so that in pronounced cases it cannot be obliterated, but is rolled like a cord or pipe-stem between the compressing fingers and the bone. Small atheromatous specks or plates, feeling like hard particles in the coats of the artery, may be detected. The artery has a beaded feeling, and is usually very tortuous. Fatty degeneration of the organs is likely to occur when the arteries are in this condition, and apoplexy is to be feared.

Blood-pressure as a Factor in Diagnosis. The clinical estimation of blood-pressure by means of instruments of precision is a comparatively recent procedure in physical diagnosis, and while the results of such observation cannot rival in accuracy the information obtained in the physiological laboratory, the method nevertheless has a distinct practical value. As the clinical thermometer has made a much more accurate study of the temperature possible, so the substitution of an instrument for the sense of touch in the estimation of the blood-pressure ought to prove useful both directly by the information gained in a given case, and indirectly by developing accuracy in observation and adding to our knowledge of the effects of disease on arterial tension. It is difficult to distinguish between the resistance offered to the finger by the pressure of the blood in the artery from the resistance depending on changes in the vessel-wall, or that due to an increased volume of blood within the vessel; but an instrument impartially records the physical impression received and its findings are not disturbed by allied subjective sensations.

Apparatus. A perfectly satisfactory instrument for the determination of blood-pressure is still to be invented. Of those at present in use, the *sphygmomanometer of Riva Rocci* appears on the whole to be the most accurate; that is, its readings are nearly uniform under conditions as nearly as possible identical. The question whether the height of the mercurial column in the manometer corresponds exactly to the actual pressure of the blood in the artery, expressed in millimetres of mercury, does not affect the practical value of the method, since the value of all instrumental clinical observations is at best only comparative. The apparatus in question consists essentially of a pneumatic band or arm-piece, like a section of a bicyle-tire, connected by means of rubber tubing with a mercurial manometer and a rubber hand-bulb, the whole forming a closed system. The arm-piece having been applied to the upper arm midway between the shoulder and the elbow, the pressure is raised by compressing the bulb until the radial pulse can no longer be felt, when the height of the column of mercury in the manometer will represent the pressure necessary to overcome the pressure of the blood in the artery, or, in other words, the blood-pressure. The reading can also be

taken by first causing the pulse to disappear and allowing the air to escape through a short branch-tube, provided for the purpose and controlled by a pinch-cock, until the pulse reappears at the wrist, when the number of millimetres of mercury is read off as before. As the only personal factor involved in this procedure is the observer's ability to detect a more or less feeble pulse, the results are fairly accurate; the disadvantages of the instrument are that it is cumbersome, and that the observation requires some little time and some preparation of the patient, as the arm-piece must be applied to the bared limb. The inventor has

FIG. 106.



Cook's modification of the Riva-Rocci sphygmomanometer.

had a number of imitators, who have made more or less useful changes in the original apparatus, a modification of which is now on the market in this country. There is also a portable form, which, however, suffers from the serious disadvantage that the jointed glass manometer is easily broken. A very good instrument can be constructed by any one having the necessary manual skill with the ordinary appliances available in any clinical laboratory.

An instrument that is much used in Germany is *Gärtner's tonometer*, which is constructed on a somewhat different principle. Instead of compressing the trunk of the artery at some point proximal to the radial pulse, the finger is compressed by means of a pneumatic ring, the tissues having first been rendered bloodless by slipping a tightly fitting circular rubber band over the tip of the finger. The reading is taken at the moment when the finger begins to blush or the patient feels the throb of the returning blood, the subjective sensation usually preceding by a few seconds the visible evidence of the returning blood-flow. A metal aneroid accompanies this instrument, which therefore has the advantage of being readily carried in the pocket, although for accurate work the standard mercurial manometer is to be preferred. Gärtner's tonometer is a con-

venient instrument for office practice, as the observation can be made in a few moments and no preparation of the patient's person is required. Unfortunately the accuracy of the readings depends on absolute uniformity in technic; differences in the relation between the size of the compressing ring or circular rubber band and the size of the finger, as well as other differences in technic, have a considerable effect on the readings; and the fact that the observation is based on the change of color in the skin of the finger renders the instrument practically inapplicable to individuals of the colored race.

Normal Blood-pressure. The pressure of the blood varies widely in health, being influenced by a number of factors, such as the position of the body, exercise, rest, emotional excitement, the time of day, the taking of food, and the like; and it is accordingly difficult to assign a figure for the *average normal blood-pressure.* Other things being equal, it is somewhat higher in men than in women, and in hard-working men than in those who lead a sedentary and indolent life; children as a rule have a lower pressure than adults, but in the aged the reading is usually above the average, the heightened tension being accounted for by the physiological arteriosclerosis and the tendency to emphysema and fibroid changes in the respiratory apparatus incident to advanced age. The pressure is higher in the erect than in the sitting posture, and lowest when the individual is lying down; but in states of fatigue and debility the pressure falls when the patient sits up or assumes the standing posture, thus explaining the tendency to syncope under those conditions. As the readings obtained with different instruments are apt to show great variations, it would be advisable, in order to obtain comparable results, for every observer to report his readings in the form of percentages based on a standard determined by himself with the particular instrument of his choice. Most authorities agree that a blood-pressure *below 120 and above 160 millimetres of mercury,* when the patient is at rest, must be regarded as pathological.

General Conditions Affecting the Pressure of the Blood. *Age, sex,* and *heredity* are predisposing factors in the production of high arterial tension. The tendency to apoplexy and paralysis observed in certain families finds its explanation in a hereditary predisposition to excessive blood-pressure. The diseases associated with high pressure are more common in middle and advanced age than in youth; and men are more frequently attacked than women, because they are more exposed by their occupations and mode of life to the exciting causes. Three factors contribute to the production of high arterial tension: *increased peripheral resistance, increase in the strength of the heart,* and *increase in the volume of blood.* Thus, deficient oxygenation of the tissues, as in emphysema and certain other affections of the respiratory apparatus, and imperfect elimination of waste-products, as in renal disease and in gout, result in a toxæmic condition in which the altered blood, acting on the capillary walls, keeps them in a constant state of contraction. Plethora and hypertrophy of the cardiac muscle bring about the same result each in its own way. The conditions which bring about obstruction in the capillaries, in the order in which they are enumerated by Broadbent, are: 1. Age. The liability to high arterial

tension increases with the age, especially after middle life. 2. Heredity. There is in some families a marked tendency to high tension. The younger members show its effects in headaches and bilious attacks, while the older ones develop chronic heart disease and apoplexy. 3. Disease of the kidney. Parenchymatous, but especially interstitial, nephritis is associated with high arterial tension; this, with accentuation of the aortic second sound, is one of the early and, therefore, one of the most valuable indications of chronic Bright's disease. 4. Gout. Gout and lithæmia are almost always accompanied by high arterial tension. 5. Diabetes in old persons associated with gout. 6. Lead-poisoning. 7. Pregnancy. 8. Anæmia. 9. Emphysema and chronic bronchitis. 10. Mitral stenosis.

As regards arterial tension in persons presenting signs of *angina pectoris*, Sansom asserts that if the tension is increased, even though the signs are not typical, the fear, present or remote, of true angina is justified. On the other hand, if there is persistent low tension, especially during the painful crises, it is almost certain the affection is a false angina. On the other hand, the pressure of the blood is lowered in conditions of general debility or exhaustion, during convalescence from acute disease, in shock, in anæmia, in obesity, in neurasthenia, and whenever the heart-action is weakened either from local or from constitutional causes. Low tension, like the opposite condition, may be a family peculiarity. Fat persons are apt to have a low-tension pulse, and it may occur in any person temporarily under the influence of external warmth and moisture, such as a hot bath, or after taking hot drinks, or under the influence of depressing emotions, and after diarrhœa or copious urination.

Fever. There is no direct relation between temperature and blood-pressure. In the sthenic fevers with full, bounding pulse and excessive heart action the blood-pressure is high; but in typhoid fever—in which also an accompaniment of low-tension pulse, namely, dicrotism, is marked—peritonitis, and in the pyrexia of pulmonary tuberculosis the arterial tension is below normal.

Cardiovascular Disease. The blood-pressure is diminished in nearly all forms of valvular disease with the exception of *mitral stenosis*, in which the increase may be explained by the hypertrophy of the right ventricle. The structural condition and functional power of the heart muscle—in other words, the presence or absence of *hypertrophy*—are the determining factors in the production of high pressure, irrespective of the form of organic lesion present. Thus in *aortic insufficiency*, the typical lesion of low arterial tension, the sphygmomanometer not infrequently shows a high reading. In the so-called "irritable heart" the blood-pressure is high, and unless it is corrected by absolute rest the condition may become chronic and lead to hypertrophy of the heart and of the bloodvessels—"athlete's heart."

Careful observation with the sphygmomanometer has shown that in *arteriosclerosis* high blood-pressure is not constant, although it is difficult to distinguish with the finger between rigidity of the wall and failure of the artery to collapse between systolic expansions. When in a case of arteriosclerosis the blood-pressure is found to be normal or diminished,

it indicates failing power of the heart; and if treatment has been given to correct an existing high tension, the height of the mercurial column in the manometer affords a measure of the therapeutic success, and at the same time an indication for continuing or suspending the treatment.

The well-known difference in the strength and volume of the radial pulses in cases of *aneurism* situated to the proximal side of the origin of the subclavian arteries is fully borne out by observations with the sphygmomanometer, and in doubtful cases the instrument may be of value in bringing out differences not appreciable by the sense of touch. When the pupils are unequal, the blood-pressure is found to be lower on the side corresponding to the larger pupil, a phenomenon that has been explained on the theory that contraction of the iris depends on an increase in the volume of blood in the bloodvessels of the membrane as well as on irritation of the sympathetic nerve.

Renal Disease. The blood-pressure is raised in acute and chronic nephritis. Excessive blood-pressure, as is to be expected, is a constant feature of *chronic interstitial nephritis*, the manometer often registering 200 millimetres and more; the second aortic sound is accentuated, and sooner or later the left ventricle becomes hypertrophied and the arteries thickened.

Pneumonia. As in all sthenic fevers, the pressure is high during the active stage of the disease, an excessive increase being often observed at the height of consolidation. During the later stages, when the heart begins to flag, the pressure is, of course, proportionately reduced.

Pulmonary Tuberculosis. A soft, feeble pulse, general weakness, dilatation of the pupil, and other signs of lowered vital force have always been regarded as characteristic features of pulmonary phthisis; and if the blood-pressure is found to be high in this disease, some other cause will be found to account for the condition. The dilatation of the pupils, which is practically constant in typhoid fever and tuberculosis, has been adduced in support of the theory already referred to in connection with aneurism, that the relaxation of the iris is due to anæmia of its bloodvessels.

Emphysema is characterized by high blood-pressure, the increase in this disease being probably attributable to the deficient oxygenation of the tissues and in part also to an associated hypertrophy of the right heart. *Chronic bronchitis* if long continued may present the same feature.

Typhoid Fever. The blood-pressure is low throughout the disease, and, as already stated, there appears to be no relation between temperature and blood-pressure. The diminished tension persists well into the period of convalescence.

Diabetes. The usual form of the disease, which occurs in persons past middle life, is characterized by heightened blood-pressure; when the disease attacks younger subjects, however, the pressure sometimes is diminished, a sign that is said to be a grave prognostic omen.

In conditions associated with *increased intracranial tension* or with *vasomotor spasm*, such as *cerebral hemorrhage, migraine* and certain other varieties of headache, and in certain neurovascular diseases, notably *Raynaud's disease*, there is constant high arterial tension. In these cases the

blood-pressure can be utilized as a therapeutic index, or in a surgical case to determine the propriety of operative intervention, the subjective symptoms often subsiding at once when the excessive tension has been relieved.

Volume. The volume of the pulse should be noted. It is usually large in conditions of pyrexia and when the tension is low. A small pulse is met with in many conditions other than weakness of the heart muscle. In aortic stenosis the pulse is small, and in mitral stenosis it is small, of high tension, and frequently irregular. In general contraction of the arterioles, as happens under the influence of a chill, the pulse is small. In Bright's disease it is sometimes very small, slow, and hard. Some care will be required to differentiate such a pulse from a weak pulse. In acute peritonitis the pulse is apt to be small and hard.

Rhythm. The rhythm of the pulse is of diagnostic importance. In health one beat succeeds another at equal intervals of time, and the successive beats are of equal force and like quality. Here, also, as in other conditions, there are variations within physiological limits. In some persons the pulse-rate is somewhat accelerated during respiration and becomes slower in the pauses which follow breathing. In disease, disturbance of the rhythm occurs as intermission or as irregularity.

INTERMISSION signifies a dropping of a pulse-beat; several normal pulse-beats succeed each other, and then the pulse is absent during the time that should be occupied by one or two beats. The intermission may occur at regular or at irregular intervals—that is to say, every third, fifth, or sixth beat may be wanting, or the intermission may be irregular—now a second, the next time a fifth or a third beat being absent. Moreover, the intermittent pulse may be constant, or it may and more frequently is only occasional. It is not characteristic of any one disease or condition, and it may exist without the patient's knowledge and without producing any perceptible effect upon his health. Sometimes it is met with in a fatty heart, and this condition may be suspected if the intermittent pulse is associated with a weak first sound of the heart without valvular lesion, and evidences of failing circulation such as œdema of the feet. More frequently, however, the intermittency is a symptom of nervous depression, or is caused by tea, coffee, tobacco, or digitalis. So far as prognosis is concerned, it is much less serious than irregularity. Broadbent says he has met with it at the age of eighty, when it was known to have existed for forty years.

IRREGULARITY is characterized by differences in time, force, or volume of successive beats. A full beat is succeeded by another which is smaller and weaker, or successive beats occur at irregular intervals of time. Irregularity may or may not be associated with intermission. In advanced cases of mitral stenosis the pulse is both irregular and intermittent. The irregularity may be habitual or occasional; the former is due most frequently to mitral lesions, but sometimes occurs without assignable cause, and is attributed to disturbance of the nerve-supply; the latter is due to digestive disturbances and to the effect of nicotine and digitalis. Irregularity is not incompatible with health, but is much more likely to be of serious import than intermission. It occurs in disease of the brain, in degeneration of the heart as well as in valvular lesions, and in grave

cases of febrile diseases, such as typhus and typhoid fevers, when the heart muscle is involved. Some cases of Graves' disease are characterized by great irregularity instead of excessive rapidity of the pulse. Irregularity may occur in rheumatoid arthritis also, though increased frequency is the rule.

Frequency. The frequency of the pulse is of aid in diagnosis.

Increased Frequency. 1. *Infections.* The pulse is *increased* in frequency in all the febrile diseases, and generally in the proportion of 8 to 10 beats for each degree of rise in temperature above 98.6°. But there are important exceptions. In *typhoid fever* the pulse is slower in proportion to the temperature and the gravity of the disease than in most of the other acute febrile diseases. It may not beat above 85 in mild cases, and in severe cases frequently does not rise above 100. Consequently a pulse of 120 is of much graver import than it would be in other diseases. It may be more frequent during convalescence than during the febrile stage. This pulse-rate helps to differentiate it from tuberculosis, malignant endocarditis, and septicæmia. The pulse of *scarlet fever* often aids materially in diagnosis. A pulse of 120 to 160 is the rule from the time of development of the sore throat to the completion of the eruption. In measles, rubella, diphtheria, and follicular tonsillitis the pulse is much slower during the early stages. In the *puerperium* increased frequency with irregularity of the pulse is a surer indication of intra-uterine mischief than is the temperature. So, too, in all cases of inflammation so situated that the products are absorbed into the circulation and not discharged externally, the pulse shows by its increased frequency that a septic process is going on.

2. In *Graves' disease* great frequency of the pulse is the essential and most constant symptom. The pulse may be constantly much more frequent than 100, and in attacks of palpitation the number of beats may reach 200 or more. In these attacks there may or may not be præcordial distress and mental anxiety. Here belong the cases described as paroxysmal hurry of the heart, etc., the thyroid and ophthalmic symptoms being absent.

3. Cases have been reported of extreme frequency of the pulse (160 to 240) without palpitation, dyspnœa, or any signs of Graves' disease. Some of the patients have been able to perform much bodily and mental labor notwithstanding that the rate mentioned was maintained persistently for weeks. To this class of cases the name *tachycardia* has been provisionally applied until their pathology shall be understood. When the condition manifests itself by paroxysmal attacks of great frequency, it is known as *paroxysmal tachycardia.*

4. In all forms of valvular disease, except aortic stenosis with failing compensation, the pulse may be increased in frequency. In collapse, in cardiac weakness, and in central or peripheral vagus disease the pulse is increased. *Mitral stenosis* may be latent until great excitement, overexertion, and particularly running or rapid walking bring on palpitation, or simply abnormal and persistent frequency of the heart's action, with or without dyspnœa.

5. Attention has been called, especially by J. Kent Spender, to accel-

eration of the pulse as an early symptom of *rheumatoid arthritis*. The pulse increases gradually until it reaches a range of 110 to 120 or more, and it may persist at that rate with little diurnal variation even after the arthritic symptoms have subsided.

6. In *locomotor ataxia* permanent moderate acceleration of the pulse (90 to 100) is a frequent symptom.

Diminished Frequency. A *slow* pulse (bradycardia), under 60, like a frequent pulse, is sometimes habitual and sometimes a family characteristic. Pathologically, it is met with in conditions which increase the resistance in the arteries, such as Bright's disease, especially acute glomerulonephritis, and is especially common in jaundice. The bile-acids have the effect of retarding the action of the heart.

A slow pulse is met with in certain forms of *heart disease*, as aortic stenosis, but it is not constant in any of them. It occurs in fatty degeneration, especially when due to obstruction, by atheroma or otherwise, of the coronary arteries. W. J. Pettus has reported a case of bradycardia associated with aneurism of the right sinus of Valsalva, involving the orifice of the right coronary artery. When retardation appears in the late stages of valvular affections or specific diseases with cerebral symptoms, it is usually a sign of danger. It is seen in articular rheumatism. (Atkinson.) According to Riegel, it is most common in *convalescence from acute disease*, particularly pneumonia, typhoid fever, erysipelas, and rheumatic fever. It is also frequently encountered in diseases of the *digestive organs* and of the urinary organs, particularly *acute nephritis*. Moreover, the pulse is generally slow in *myxœdema*, and both slow and irregular in *epilepsy*. Not uncommonly it is slow also in *melancholia* during the early stages of *cerebral meningitis*, and in tumors and cerebral hemorrhage.

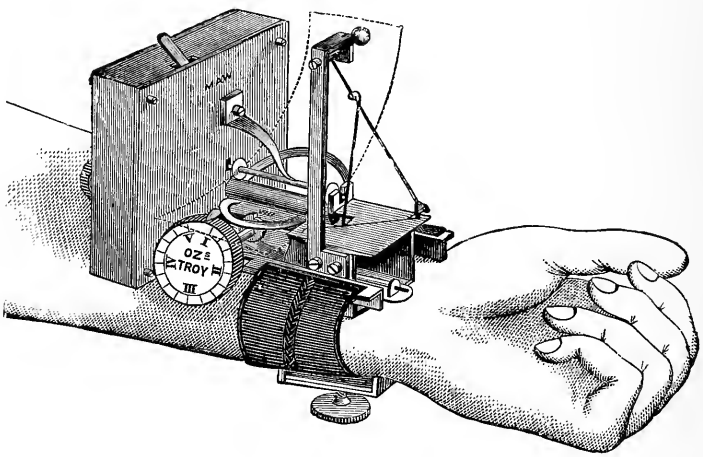
The Sphygmograph. The sphygmograph, as its name implies, is an instrument for recording the volume, force, frequency, tension, and general characteristics of the pulse. The expansile pulsation of the artery is communicated by a system of levers to a needle which graphically records upon smoked paper the qualities of the pulse. Many forms of the instrument have been devised since the original one of Marey. The later models have the advantage of simplicity and ease of application. One of the most convenient is Dudgeon's. It has its faults, particularly in exaggerating the vibrations when the pulse is large and the heart is acting violently; nevertheless, with care, trustworthy tracings can be obtained in all ordinary cases. No matter what instrument is used, the value of the tracing depends largely upon the skill and experience of the operator; hence, the sphygmograph occupies a position very different from that of the thermometer or of other instruments of precision. While it is true that a person can learn to detect nearly all the variations of the pulse by palpation alone, yet the tracing has the great advantage of permanency, and many persons are led to palpate the pulse more carefully when they see in a sphygmographic tracing a dicrotism or irregularity which had escaped their attention.

Directions for Using Dudgeon's Sphygmograph. 1. Wind up, by the button, the clockwork contained in the box. The clockwork carries the smoked paper under the writing-needle.

2. See that the patient is in a comfortable position, and have him hold toward you either hand with wrist exposed, fingers gently flexed, and muscles relaxed.

3. Apply the instrument by slipping the band over the hand, the free end of the band being passed through the retaining clamp. The metal box should be placed toward the elbow.

FIG. 107.



Dudgeon's sphygmograph.

4. Now adjust the instrument by placing the bulging button which connects the levers directly over the radial artery at its most accessible point.

5. Keep the instrument accurately in place with the left hand, and draw the band through the clamp with the right until the writing-needle plays freely with each pulsation of the radial artery, then fasten the band by screwing up the clamp.

6. Introduce the smoked paper between the rollers and under the writing-needle.

7. Vary the pressure by means of the thumb-screw, which connects with an eccentric, until the best apparent amplitude of vibration is obtained.

8. Instruct the patient not to move the fingers or hand, and further steady them for him with your own right hand.

9. Start the clockwork by pushing the bar at the top of the clockwork box.

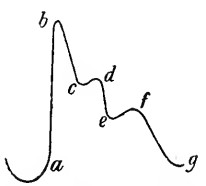
10. Allow the paper to run through, and then stop the clockwork.

The clockwork is so regulated that five inches of smoked paper pass through in ten seconds, so that six times the number of pulsations recorded on the paper represent the pulse-rate per minute. Each instrument, however, should be tested and its time determined. The clockwork should be wound up for every tracing.

The *technic* of sphygmography needs a few words. Smoked paper is generally used for the tracings. A paper glazed upon one surface and

rough upon the other has some advantages. This paper has to be cut in strips about seven-eighths inch wide and six inches or more long. The cutting should be done with care so that the edges are smooth and even, otherwise the paper sticks in the instrument and the tracing is spoiled. The glazed surface is blackened by holding it above the flame of a small piece of burning gum camphor. For convenience, a strip of tin, bent upon itself at each end, so as to catch and hold about an inch of the ends of the paper, may be used to prevent the fingers from becoming blackened and to preserve the ends of the paper unblackened for memoranda. The blacking should not be too thick, otherwise the needle will not plough through it easily, and the white line of the tracing will not be distinct. After the tracing has been made, the name of the patient, the diagnosis of his disease, the date of the tracing, the pulse-rate, and the degree of pressure employed should at once be scratched with a fine-pointed pen upon the blackened surface beneath the tracing, or written in ink upon the unblackened end of the paper. The tracing is then ready for preservation. This is done by dipping it into a solution of shellac or in tincture of benzoin (gum benzoin, ℥j ; alcohol, f℥vj) ; the alcohol evaporates and leaves a smooth glazed surface. Dudgeon recommends as a

FIG. 108.



ab, percussion up-stroke; *abc*, percussion-wave; *cde*, tidal wave; *efg*, dicrotic wave; *def*, aortic notch; *fg*, diastolic period.

varnish a solution of gum damar (℥j) in rectified benzoline (f℥vj). When the tracing is likely to be subjected to friction, a second or third coat should be applied. Considerable practice will be required to take a tracing rapidly and accurately in spite of the simplicity of the mechanism. Several tracings should be taken at different pressures and compared ; or, what is better, as suggested by Sansom, stop the clockwork and alter the pressure two or three times, so as to have the effect of varying pressures on one tracing.

Explanation of the Normal Pulse-tracing. With each contraction of the left ventricle there is forced into the aorta a volume of blood which distends the vessel. The distending impulse of this volume of blood is transmitted by a wave-like motion to remote arteries. This distending impulse lifts the button of the lever sharply upward, forming the so-called percussion up-stroke *ab ;* but the distending impulse is exaggerated by the system of levers, and having been thrown up too high, the lever falls by its own weight too low, so that it is again caught and lifted by the tidal blood, forming the tidal-wave *cde*. The gradual descent of the lever is again interrupted at *efg*, forming a wave called the dicrotic wave, which is due to the recoil of the blood from the closure of the aortic valves. (Fig. 108.)

Roy and Adami believe that the apex (*abc*) of the percussion-wave is due to the sudden pulling down of the auriculoventricular valves by the papillary muscles during the first rapid part of their contraction; hence they called the wave the "papillary wave." The second wave (*cde*) corresponds in time, they say, with the outflow from the ventricle due to the continued contraction of the heart-wall and papillary muscles after the flaps have been pulled down; hence they prefer to call this wave the "outflow remainder," instead of "tidal" wave.

Interpretation of Pulse-tracings. Sphygmographic tracings must be interpreted in accordance with the known peculiarities of the patient, his history, and the associated physical signs.

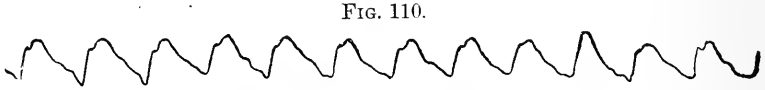
1. The Amplitude. The height of the percussion-stroke varies considerably in health. It is increased in conditions which bring about low tension and rapid systolic contraction of the heart; hence the febrile pulse is usually one of considerable amplitude. It is increased also very markedly in aortic regurgitation. Suddenness of systole rather than force determines the height of the up-stroke. (Fig. 109.)

Fig. 109.

Tracing from a case of aortic regurgitation. (Original.)

2. Obliquity of the Percussion-stroke. Normally the percussion-stroke ascends vertically from the base-line. A tendency to incline forward indicates a weak and laboring heart or an aneurism interposed between the radial artery and the heart. In the latter case there is also a tendency to rounding of the summit of the percussion-wave, and the up-stroke is generally short. There is usually also regularity in successive pulsations, some showing the gradual ascent and rounded summit much better than others. Sometimes, however, when aneurism exists, there is no evidence of it in the tracing, and differences upon the two sides are not always significant. (Fig. 110.)

Fig. 110.



Tracing from a case of aneurism of the aorta. (Original.)

Disease at the aortic orifice and the intervention of a considerable quantity of subcutaneous fat or of any growth superficial to the vessel may cause a marked obliquity of the percussion-stroke. Sansom asserts that, such causes excluded, as well as aneurism and organic disease of the aorta and its valves, a sloping line of ascent, observed under various gradations of pressure, indicates feebleness of the left ventricle. He considers it of higher diagnostic value than irregularity, which he says is often neurotic.

3. Increased Breadth of the Apex of the Percussion-wave. The breadth of the apex of the percussion-wave indicates the time during which the artery is kept full by the systole of the left ventricle. When the left ventricle acts slowly and forcibly, the arteries will be kept distended for a longer time, and this distention will be manifest in broadening of the apex of the tracing. (See Fig. 111.) The degree of distention of the artery is called tension, hence a broadening of the apex is an evidence of high tension. As the word "high" does not indicate the duration of the

Fig. 111.

From a case of aortic stenosis, showing increased tension and the *pulsus bisferiens* (Original.)

tension, Sansom has very properly suggested that we should speak of persistent high tension as "prolonged" tension. This, then, is the significance of the broad top of the tracing. (See Fig. 112.)

Prolonged arterial tension occurs when there is a strong heart acting slowly, a large volume of blood, or obstruction in the capillary circulation. (For specific causes, see under Blood-pressure.)

The degree of pressure required to develop the characteristics of a pulse, and, still more, the degree required to obliterate it, are good indications of the degree of tension present. Some pulses, however, appear to

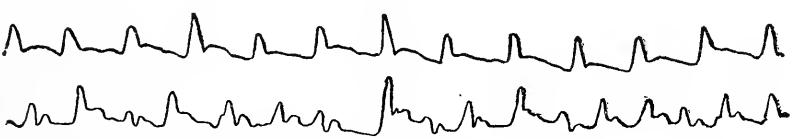
Fig. 112.

From a case of mitral stenosis, showing increased tension and some irregularity. (Original.)

the touch to be of prolonged tension, although a sphygmogram does not show it. Such cases are often explained by the fact that the heart has begun to fail under the strain put upon it by prolonged obstruction in the capillaries. There may be regurgitation also from the mitral or aortic orifice.

4. Acute Angle of the Percussion-wave. When the heart's action is feeble or sudden, the volume of blood small, or the resistance in the

Fig. 113



Low tension with irregularity, from cases of mitral regurgitation. (Original.)

capillaries much diminished, the up-stroke of the tracing is vertical, and the down-stroke forms an acute angle with it. The dicrotic wave is pronounced, and often descends unduly low, sometimes to the base-line. These are the characteristics of low tension. (See Fig. 113.) When the

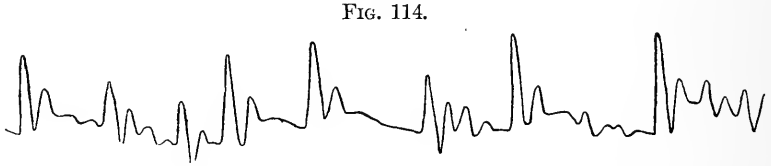
dicrotic wave springs from a lower level than the base-line of the tracing, it is *hyperdicrotic*. When the dicrotic wave is wholly effaced in the succeeding up-stroke, it is *monocrotic*.

While dicrotism is commonly associated with low-tension pulses, it is occasionally met with also in high-tension pulses. Sansom says, however, that he has seldom observed conjunction of broad summit and marked dicrotism without the patient's manifesting the signs of failing heart.

5. Irregularity of the Base-line. This occurs normally in some persons as the result of respiration, especially deep breathing. It also occurs in respiratory diseases and in affections causing dyspnœa. Decided undulation of the base-line, the curves being irregular, occurs in tubercular meningitis.

6. Differences in the Height of Successive Percussion-waves or in their Distance from each Other. These are graphic evidences of disturbance in the rhythm of the heart. The former expresses irregularity in the volume of successive beats; the latter, irregularity in time, and when it amounts to the omission of a beat, it is called intermission. All these changes are shown in Fig. 114.

Fig. 114.



From a case of advanced mitral stenosis, showing extreme irregularity and intermission. (Original.)

The Veins. **Thrombosis.** This is usually detected by palpation and occurs most frequently in the femoral vein. The vein is transformed into a firm round cord, and is distinguished from the artery by the absence of pulsation. Thrombosis in these veins and in the iliac veins higher up occurs in acute infectious diseases and in the debility of the aged, and leads to dropsy in the area of distribution of the veins.

Percussion.

By means of percussion the shape and size of the heart and changes in the area of cardiac dulness are determined. (See the Lungs for discussion on percussion.) To determine the size of the heart, both superficial or light, and deep or strong percussion must be employed. By the former we determine the area of superficial or absolute cardiac dulness; by the latter, the area of deep or relative cardiac dulness.

The Area of Superficial or Absolute Cardiac Dulness. (See Plate IV.) This corresponds with that portion of the heart not covered by the lung at the time of inspiration. The lungs overlap the heart, and in inspiration allow a small area to be in contact with the chest-wall. The percussion-force employed must be light, so as not to elicit the resonance of the extreme thin edge of the lung. The area is irregularly triangular in shape and extends from the fourth to the sixth costal cartilages. The right border may be roughly defined by a line

drawn along the left edge of the sternum from the upper border of the fourth rib downward; the left border by a line extending from the upper border of the fourth rib at the left edge of the sternum to a point midway between the parasternal and the mammillary lines in the fifth interspace. The lower border is continuous with liver-dulness.

Technic. The right border is determined by percussing from right to left toward the median line. Always begin to percuss sufficiently far from the heart to get the clear pulmonary note. To insure uniformity, select in all cases a definite area from which to start. Apply the finger vertically at first. The right border may correspond with a line outside of or along the right edge of the sternum, with the median line, or the left edge of the sternum, or even beyond the latter. After the edge of absolute resonance is reached, percuss with the finger parallel to the ribs to control the result previously secured; and as each interspace is percussed, the upper limit of liver-dulness and the triangle (Ebstein's) between the liver and heart may be determined.

The left edge is determined by percussing in vertical lines from a point near the axilla toward the heart. Opposite the second and third interspaces the aorta on the right side and the pulmonary artery on the left will cause impairment of the normal pulmonary resonance. The student should acquire the habit of proceeding from definite fixed positions toward the heart, and to observe the changes during inspiration and expiration. The lower border and rounded apex of an enlarged heart can not be defined if the stomach contains food or fluid. The hypertrophied heart is triangular in shape with the apex pointing downward.

The *cardiohepatic* (or *Ebstein's*) *triangle* is the more or less resonant area in the right fifth interspace which separates the right heart from the liver. The apex of the triangle points to the right sternal edge, the base to the axilla. The upper side corresponds to the right border of the heart; the lower is the upper limit of the liver.

Changes in Size. The superficial area of dulness or absolute dulness may be increased or diminished in size.

Dulness Decreased or Absent. It is diminished or replaced by resonance in emphysema, and hence *absent* entirely if the lung completely covers the heart. It is absent when the heart is drawn under the lungs by adhesions and when there is air in the pleural or pericardial sac. It may be increased in size in all directions, or especially in one or another direction.

Absolute Dulness Increased. Increase in the area of absolute dulness in all directions occurs in hypertrophy and dilatation of the heart, in pericardial effusions, in retraction of the lung, when the heart is pushed against the chest-wall, as, for instance, by an aneurism or a mediastinal tumor, and when the patient leans forward or is in the knee-chest position. Increase in width of the dulness at the base of the heart occurs in dilatation, pericardial effusion, and aneurism of the aorta. Change in the position of the heart, a general idea of which is obtained by inspection and palpation, always changes the shape and extent of the dulness. The heart should be accurately delimited when displacements have taken place.

Increase of Dulness Upward. In addition to a general increase in cardiac dulness, one of the boundaries or a portion of the boundary may be increased or extended beyond the normal line. Thus in pericardial effusion the area of dulness may extend *upward*, and this may be followed by extension of the right and left boundaries. The relative area of dulness becomes abolished; the change from pulmonary resonance to dulness becomes abrupt and decided; and the area of dulness becomes pyramidal or pyriform in shape. Upward increase of dulness may be due to disease of the vessels, especially the aorta. Increase in the area of dulness over the bloodvessels is usually due to aneurism. It may be general, as in dilatation or fusiform aneurism of the aorta, or local, as in saccular aneurism. Extension of the dulness outward or upward from the normal line may be found at the right of the sternum (aneurism of the ascending aorta), or over the first bone of the sternum (aneurism of the transverse aorta), or to the left just above the cardiac area. In the last case the dulness is an extension upward of the normal area of cardiac dulness with rounding of the area affected, and is usually indicative of an aneurism situated at the beginning of the descending portion of the aorta.

Increase of Dulness to the Left. Increase in dulness to the *left* occurs in enlargement of the heart from hypertrophy or dilatation. If the dulness extends outward to the left and retains the triangular shape, with the apex pointed, it is due to hypertrophy of the left ventricle. If, on the other hand, it becomes quadrilateral in shape with the apex rounded, it is due to dilatation of the left ventricle. In other cases increase in the dulness to the left occurs in displacement of the heart due to aortic aneurism or mediastinal growth. In many of these cases the dulness due to the aneurism or tumor is continuous with that due to the heart, and an effort must be made to discriminate between them. The results of palpation and inspection aid in detecting the presence of one or the other of these conditions.

Increase of Dulness to the Right. The area of dulness may extend farther to the right than normal, in which case it is due to hypertrophy and dilatation of the right auricle and ventricle. If the auricle is dilated, the right edge of dulness is extended beyond the normal in the third, fourth, and fifth, or as high as the second interspace. With this increase in dulness there is also seen, although not necessarily because of the cardiac enlargement, an epigastric impulse. Venous turgescence and pulsation of the veins of the neck or of the liver are likely to be present.

Deep Cardiac Dulness. Many authorities consider the deep or *relative area* of cardiac dulness of importance in diagnosis. To elicit it the percussion must be strong. The best method is that advised by Gibson and Russell. Their directions are as follows: "Begin in the upper left interspaces sufficiently far out from the sternum to secure pulmonary resonance. For instance, in the second interspace begin in the midclavicular line and percuss strongly. As soon as a slight alteration in that sound is noted, the point is indicated by a mark. The second or third and succeeding interspaces are percussed in like manner, bearing in mind that the percussion must begin farther out in each interspace in order to get pure resonance. As dulness is secured in each space a mark is made.

This is continued to the apex if that is visible, or to the base of the chest. By joining the marks in each interspace with the line at the base of the heart, the left border of the cardiac dulness can be fixed." The authors correctly point out that in this way the true apex of the heart is found, enabling auscultation to be conducted more accurately.

The right edge of the vessels and of the heart is defined in the same way. In passing from the lung to the heart the difference in the sound is not so distinct along the right border as along the left. The authors include the dulness which is due to the vessels at the base of the heart, and hence begin percussion in the higher interspaces. This they deem proper because it is impossible to delimit the two. The dulness of the vessels is not so marked, however, and may be indicated by simple change in pitch in the percussion-note. The lower border of cardiac dulness is ascertained with difficulty because of its close apposition with the liver. At times there is a difference in the character of the dulness between the two organs. It can be well made out by stethoscopic (or auscultatory) percussion. It may not be so pronounced as we pass from the heart to the liver in the median and parasternal lines. Toward the apex the difference is more apparent.

Pleximetric Percussion. For more accurate cardiac percussion Sansom recommends the use of a pleximeter designed by himself, by which delicate shades in dulness can be readily heard. The pleximeter is a thin, flat, oblong plate one inch by half an inch, which has on its upper surface a column rising from the middle, one and a half inches in height, which is surmounted by a second plate three-eighths to three-fourths of an inch, set parallel with the lower plate. The instrument is held between the forefinger and middle finger of the left hand, the sensitive tips of the fingers resting on the upper surface of the larger horizontal plate. The lower surface of this latter is held close to the wall of the chest, and percussion with one or two fingers of the right hand with an even and not too forcible stroke from the wrist is made upon the upper plate. The resulting vibrations are transmitted to the ear and are also appreciated by the digital sense of touch, so that both senses aid in the determination of the nature of the sound produced.

Method. The pleximeter is placed with its long diameter parallel with the sternum, about midway between the axilla and the right sternal border. Percussion is made upon the summit of the column by one or two fingers, and the pleximeter is moved, always in parallel lines, nearer and nearer to the sternum. A line is reached where the vibrations are modified. Incline the pleximeter so that the vibrations come from its left edge. This edge, or line, is practically the line of demarcation of the dulness, and should be indicated with an aniline pencil. It corresponds to the outline of the right border of the heart. The process must be repeated at higher and lower levels until the entire right border of cardiac or aortic dulness is ascertained. In passing, it may be stated that percussing from above downward with the long diameter of the pleximeter horizontal instead of vertical, leads to the upper limit of the liver, as indicated by modified vibrations. At about the fifth right intercostal space a short curved line is thus made out along the right edge of the sternum,

which indicates the outline of the right auricle at the point where it joins the liver-dulness. Above this, as far as the second rib, the line indicates the outline of the right border of the auricle and the aorta. The outline of the auricle may be in the midsternum; of the aorta, at the right edge. In percussing the left side of the chest the same method is adopted. Begin at the level of the second rib, two or three inches beyond the left edge of the sternum, and move to the right. Join the lines of modified vibrations, and in this manner the left border of cardiac and aortic dulness is secured. The outline of the apex of the heart is readily mapped out. Over the tympanitic stomach light percussion is necessary. To narrow the area of percussion about the apex, the percussion may be performed on the larger plate, while the smaller is applied to the chest. The vibrations over the liver and over the right ventricle are difficult to distinguish, although sometimes so different that demarcation of the border of the ventricle presents no difficulty. Between the apex of the left ventricle and the left lobe of the liver the space is easily marked out.

A correct outline of the heart and of the vessels is thus obtained. The upper limit of dulness is formed by the right auricle, the aorta, and the pulmonary artery. Any bulging or undue expansion is due to aneurism or aneurismal dilatation of the aorta. The space between the apex and the left lobe of the liver defines the lower border. Sansom points out that by this method of percussion the following absolute data can be obtained: "A projection to the right of the area of the upper part over the second and third interspaces points to aneurism of the aorta or of the innominate artery. It may be traced to the left side of the sternum on account of saccular dilatation of the aorta. If the dulness at the upper part extend greatly to the left, an increase in size of the pulmonary artery may be suspected. Along the midsternal region, extension beyond the right side joining the line indicating the upper border of the liver indicates distended inferior cava. This distention occurs in right-sided dilatation of the heart, and the dulness may also be due to dilatation of the adjoining auricle. The outline of dulness obtained over the apex of the heart, if pointed, indicates hypertrophy; a more rounded outline shows dilatation. In uncomplicated hypertrophy the line of the right ventricle forms a much less obtuse angle with the liver-dulness than in dilatation. Of great diagnostic value is the diminution of the area of dulness from atrophy of the heart as observed in wasting, as in cancer, and in tuberculosis; it may also be observed in typhoid fever. In the above-mentioned conditions it is a bad prognostic sign."

Adjacent Dulness Care must be taken not to confound the dulness of pleural effusion, consolidated lung, mediastinal tumor, or aneurism with the cardiac dulness.

Repercussion. Modifications of the vibrations felt by the fingers on the pleximeter, as pointed out by Sansom, may indicate an abnormal change in physical condition impossible to detect in any other way. It is to be remembered that over the lungs the vibrations are excessive; over solid structures they are modified or lessened. Now, the change from vibrations to absence of vibrations may be *gradual* or *abrupt.*

Sansom determines this by percussion after the heart has been outlined in the above-mentioned manner. In percussing from the lung to the heart-area, if the modified vibrations occur abruptly, it is very probable that there is pericarditis with effusion or thickened pericardium; or if on percussing from above downward there is pericardial effusion, no vibrations are to be elicited over the area delimited—that is, the absence of vibrations is noted over the whole area—whereas in ordinary conditions, when the pericardium is unaffected, in percussing from above downward over the area which had been delimited on the right and left sides, respectively, a line will be reached where the vibrations become modified. This line commences a little above the ensiform cartilage and inclines toward the left border of the cardiac dulness at the level of the fourth rib and third interspace. Vibrations are more marked above than below the line. The line at which the lessened vibrations begin points out the commencement of the thick wall of the ventricles; the portion above (more vibratory) indicates the position of the right auricle and vessels. By the employment of pleximetric percussion it is not necessary to determine superficial and deep areas of dulness.

The Apex-impulse. Whichever method of percussion is employed, it will be often observed that the spot marked by inspection and palpation as the apex-impulse, is far outside of the left border of cardiac dulness. In hypertrophy of the left ventricle it may be a considerable distance to the left. In dilatation the difference is not so marked. The percussion-lines are made when the heart is away from the chest, and hence are within the systolic apex-beat.

Method of Graphic Record. (See also page 392.) We are indebted to Sansom and Ewart for a method of recording the cardiac outlines. The points of pulsation and the border-lines of dulness are marked by a dermatographic pencil. Various colors may be used in order to indicate the different data. The landmarks are outlined by a camel's-hair pencil dipped in olive oil. The episternal notch, the clavicles, the intercostal spaces, the ensiform cartilage, the nipples, the percussion-outlines, and other recorded marks are passed over with the oiled pencil. A sheet of tissue-paper or of copying-paper is then gently placed over the whole, so that the oil-marks are imprinted. After the paper is removed, the oil-outline is colored with the dermatographic pencil and a permanent record is secured. By this plan of recording, a maximum of precision is attained. Outlines can be measured and positions defined by mathematical data. The name of the patient, the date of observation, with a brief history of the case, should be attached to the chart. If the colored pencil-marks on the patient's chest are objectionable, the outline may be made with the colorless oil-pencil at the various steps of the examination. After it has been transferred to the paper it may be made more distinct with the colored pencils. Packard fits to the chest a square of coarsely woven muslin and outlines the ribs and sternum, etc., which are seen through the meshes. With colored pencils the dull areas, the site of organs, and the position of murmurs are then designated. Ewart has shown that after long intervals the size of the chest and abdomen is apt to alter from various circumstances—growth, muscular development, habit of sitting,

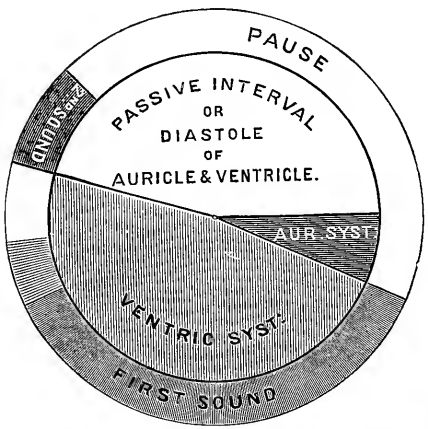
and the like. He therefore points out the advisability of using the *sternum*, which is immovable, for the sake of future comparison.

Sense of Resistance. Ebstein delimits the heart by the sense of resistance, change in size being indicated by increase or diminution of the area, which in health gives a sense of resistance to the percussing finger.

Auscultation.

Method. Either method of auscultation may be employed. By the immediate method we may form a general notion as to the condition of the heart-sounds. The mediate, however, is preferable, because it is essential to localize the sounds that are heard, and because, if the double stethoscope is used, we can percuss the cardiac area. The patient should be in a comfortable position. The muscles should not be strained. The general directions for performing auscultation must be followed. Before he begins, the observer has, if possible, determined the presence of the

FIG. 115.



Diagrammatic representation of the movements and sounds of the heart. (After SHARPEY.) This diagram shows merely the general relations of the several events, and does not represent exact measurements.

In a heart beating 72 times a minute, Foster estimates each entire cardiac cycle as occupying about 0.8 second, of which 0.3 second represents the duration of the systole of the ventricle, 0.4 second the diastole of both auricle and ventricle, or the "passive interval," and 0.1 second the systole of the auricle.

Only one "pause" is marked here—sometimes called the "long pause"; some writers describe a "short pause" also—indicated in the diagram by the small space between the first and the second sound.

apex-beat or found the carotid pulse. By this means the time of the heart is taken and the relation of the events of the cardiac cycle to each other is ascertained. With each normal impulse or carotid pulse a systole takes place; hence they are synchronous. The systole occurs just before the radial pulse.

By auscultation we determine: (1) the normal sounds of the heart, including their rhythm, their character, and the seat of maximum intensity; (2) modifications of the normal sounds as regards (*a*) loudness and (*b*) rhythm; (3) the presence of abnormal sounds or murmurs.

The Normal Sounds. The stethoscope is placed over the heart and the finger on the apex-beat or the carotid pulse; a sound will be noted at the time of the apex-beat or carotid pulse—the systole. This is followed almost immediately by another sound (occurring with the diastole), and then by a period of silence.

The sounds that attend the systole are known as the systolic, or *first sounds*. The sounds that follow are known as the diastolic, or *second sounds*. The sounds and silence mark the completion of a cardiac cycle as far as the ear is concerned. (Fig. 115.) A definite relationship in time exists in the cardiac cycle. *Cause*. Four sounds are created during a cycle, one at each valve. The sounds created with the systole (systolic sounds) are due to contraction of the right ventricle and closure of the tricuspid valve, and to contraction of the left ventricle and closure of the mitral valve. The rush of blood along the course of the vessels and the impact of the heart against the chest-wall may contribute somewhat to the systolic sounds. The sounds heard in the beginning of the diastole (diastolic sounds) are due to closure of the aortic and the pulmonary valves. They are due to the tension produced on the valves as the respective arteries contract upon the columns of blood. The closure of the valves makes up most, if not all, of the sounds. To review: two sounds occur with the systole, one from closure of the *mitral*, another from closure of the *tricuspid* valve; two with the diastole from closure of the *aortic* and the *pulmonary* valves respectively. In health the sounds of the systole blend because they are synchronous, and give the impression at a common point of one sound. Analysis of the sound in the respective valve-areas will show that the systolic sound is made up of two sounds. The sounds of the diastole may or may not blend. Usually in health they do blend; often, however, there is an appreciable difference between the two, becoming more marked on altering the respiratory rhythm, as by a full breath.

Recognition of the Respective Sounds. To distinguish the sounds we study their *rhythm* or *time*, their *character*, their position of *maximum intensity*, and their *direction* of *transmission*. We distinguish the first from the second sounds by their rhythm and character, and then differentiate the elemental sounds of the systole and of the diastole by their points of maximum intensity.

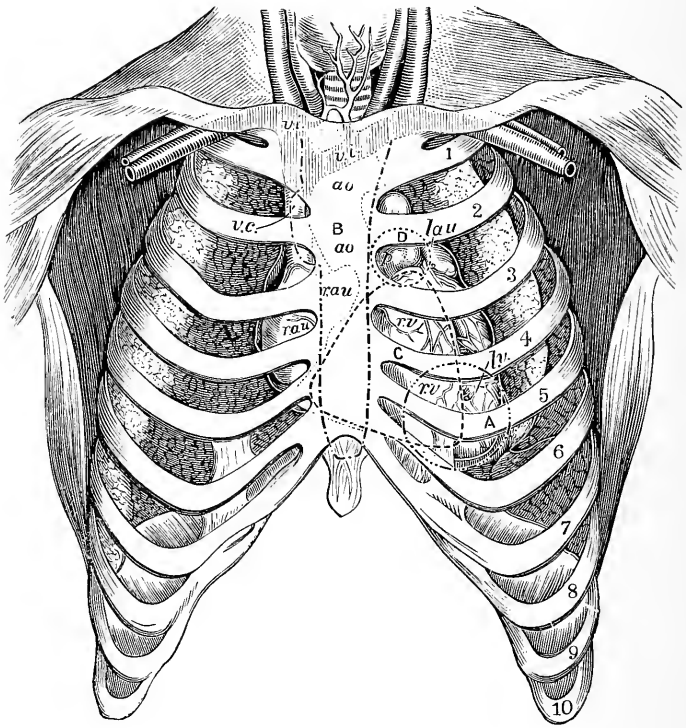
The Rhythm or Time. The sounds that are heard at the time of the normal cardiac impulse or carotid pulse or just before the radial pulse are the systolic or first sounds; the sounds that follow the impulse are the second sounds. The sounds that follow the long silence are the systolic or first sounds; those that precede the long silence are diastolic or second sounds.

Character of the Sounds. The systolic sounds are prolonged, somewhat dull in character, low in pitch, and resemble the sound produced by pronouncing the syllable "*ubb*." The diastolic sounds are short, sharp, and quick, and resemble the sound produced by pronouncing the syllable "*dupp*." The syllables *ubb* and *dupp* indicate the character of the sounds in health. Modifications in the intensity of the sounds are due to changes in the tension of the valve-curtains, and are dependent upon the force of

muscular contraction, which, if strong, renders the valves more tense. Experiment and the results of disease have aided in proving these points.

Position of Maximum Intensity. In general the first sounds are loudest at the lower part of the præcordia, the second at the upper. But we especially distinguish the independent valve-elements that make up the systolic and the diastolic sounds in the following manner: The

Fig. 116.



Areas of cardiac murmurs (Gairdner for the areas; and Luschka for the anatomy). The outlines of organs, which are partialy invisible in the dissection, are indicated by very fine dotted lines; while the areas of propagation of valvular murmurs, as described in the text, have been roughly marked by additional much coarser dotted and interrupted lines—the character of the dots being different in each of the four areas. A capital letter marks each area—viz., A, the circle of mitral murmurs corresponding with the left apex; B, the irregular space indicating the ordinary limits of diffusion of aortic murmurs, corresponding mainly with the whole sternum, and extending into the neck along the course of the arteries; C, the broad and somewhat diffused area occupied by tricuspid murmurs, and corresponding generally with the right ventricle; D, the circumscribed circular area over which pulmonic murmurs are commonly heard loudest.

Reference letters: r.au. = right auricle; a.o. = arch of aorta; v.i. = the two innominate veins; v.c. = vena cava descendens; p. = pulmonary artery; l.au. = left auricle; l.v. = left ventricle; r.v. = right ventricle. (Finlayson.)

sounds produced by the closure of the valves are created, as the topography of the heart shows, quite near to one another, but by conduction of the sound they are transmitted away from the respective valves in particular directions, and heard loudest in definite areas on the chest.

The Systolic or First Sounds. Two sounds are created. The valves

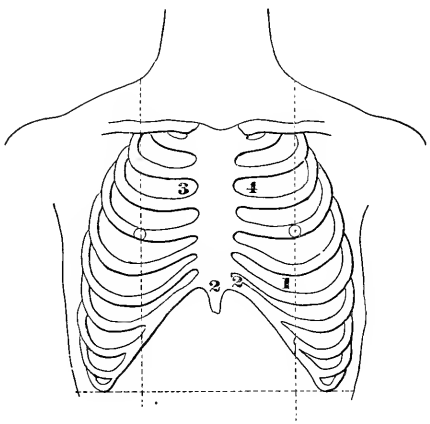
which cause the sound are near to each other. Because of their anatomical relations the sounds are conducted into different areas, by virtue of which they are differentiated.

The Mitral Valve Sound. The sound produced by closure of the mitral valve is created opposite the third interspace one inch to the left of the sternum. It is transmitted to the surface of the chest by the thick left ventricle, and hence is heard *loudest* where that is nearest the chest, namely, at the *apex*—the *mitral area.*

The Tricuspid Valve Sound. The sound produced by the closure of the tricuspid valve is transmitted by the right ventricle, and is heard loudest over the lower portion of the sternum—the *tricuspid area.*

The Diastolic or Second Sounds. Two sounds are created. The valves at which they are produced are also in close proximity. To distinguish the two sounds it is necessary to auscult over areas into which they are transmitted. They may often, especially in diseased conditions, be distinguished by their slight difference in time. In apparent health the aortic sound sometimes precedes the pulmonic by a fraction of a second, but this can scarcely be appreciated clinically.

FIG. 117.



The valve-areas: 1, mitral area; 2, tricuspid area; 3, aortic area; 4, pulmonary area.

The Aortic Valve Sound. The sound produced by the closure of the aortic valve is heard loudest at the second costal cartilage on the right, because the aorta which conducts the sound is nearest the surface of the chest at this point—the *aortic area.* This cartilage is known as the *aortic cartilage.* Cabot shows that it is heard loudest to the left.

The Pulmonary Valve Sound. The sound produced by the closure of the pulmonary valve is conducted to the left and heard loudest in the second interspace near the left edge of the sternum—the *pulmonary area.*

THE DIRECTION OF TRANSMISSION. The first sounds are transmitted toward the left axilla. They may be heard all over the cardiac area, but the position of maximum intensity is in the lower portion and toward the left. The second sounds are loudest at the base of the heart.

They may be propagated beyond the præcordia toward the neck, and be heard loudest in the vessels of the neck.

Precise Location and Differentiation of Each Sound. This may be determined by listening with the bell of the stethoscope over each area. Then move the bell gradually from one area into the other. As the sound of the original area lessens, the sound of the approached area is observed. The change from one to the other is often very marked. 1. *Mitral* first or systolic sound, heard loudest at the apex, inward to the parasternal line, upward to the third interspace. 2. *Tricuspid* first or systolic sound, heard loudest at the lower part of the sternum and toward the left to the parasternal line as high as the third rib. 3. *Aortic* second or diastolic sound, heard loudest at the aortic cartilage, propagated into the vessels of the neck, and also heard at and outside of the apex-beat. It is louder than the pulmonary second sound in health. 4. *Pulmonary* second or diastolic sound, localized to the left interspace and the third rib.

Modifications of the Sounds. The sounds, singly or combined, may be increased or diminished in intensity or accentuation. They may be altered in rhythm.

Sounds Increased. *a. Causes outside of the pericardium.* 1. Anything that brings the heart closer to the ear of the observer. Thus, in patients with thin chest-walls, when the heart is pushed to the surface of the chest (mediastinal tumor) or the lung removed (pleural contraction). 2. Anything that conducts the sounds, as consolidated lung in the vicinity, or a pneumothorax, or pulmonary cavities. *b. Affections of the pericardium,* as pericardial adhesions. *c. Conditions of the heart.* 1. Hypertrophy. 2. Over-action, as in palpitation, fevers, anæmia, exophthalmic goitre.

Sounds Weakened. *a. Causes outside of the pericardium.* 1. Thick chest-walls, large mammary gland. 2. Emphysema of the lungs overlapping the heart. *b. Affections of the pericardium,* as fluid or air in the pericardial sac. *c. Conditions of the heart.* Atrophy; myocarditis; some cases of dilatation; cardiac weakness from general exhaustion.

Modifications of Individual Sounds. The above applies to all the sounds. Increase or diminution of the systolic or of the diastolic sounds, or of any one of the four sounds, may be present.

Increase in Loudness of the Systolic Sound. Increased loudness of the first sound is noted when the muscle is hypertrophied and the tension on the valves thereby increased. The increase is most marked in

Fig. 118.

Normal first and second sounds. Accentuated first sound.

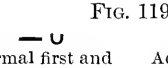
hypertrophy of the left ventricle. The sound is duller and has a prolongation which is very characteristic. In hypertrophy of the right ventricle the sound is dull and prolonged over the sternum, but not to the same degree as when the left is hypertrophied. (Fig. 118.)

Increase in Loudness of the Diastolic Sound. Either of the second or diastolic sounds may be increased in loudness or accentuated.

1. *The Aortic Diastolic Sound.* Anything that causes increased tension in the aortic circulation, and hence increased contractile force of the aorta, will increase the intensity or accentuation of the second sound. In hypertrophy of the heart the aortic sound is accentuated because there is corresponding increased contraction of the aorta following the forcible expulsion of the blood from the ventricle. Increase in arterial tension is also due to increased contraction of the aorta when there is peripheral resistance to the outflow of blood. (Fig. 119.) It is associated with the following conditions which cause accentuation of the second sound: atheroma of the aorta or of the arteries in general; aneurism of the aorta; disease of the kidneys, and particularly that form in which there are also general arterial changes, namely, chronic interstitial nephritis. It is true that the accentuation may be partly due to the coexisting hypertrophy of the heart.

Accentuation of the aortic second sound occurs independently of permanent change in the arteries. If for any reason there is spasm of the peripheral capillaries, as from a chill, from epilepsy, from nervousness due to hysteria, tension in the arteries is heightened, and hence the second sound is accentuated. Accentuation of the second sound is therefore a valuable index of the state of the vascular system in general; it is not an evidence of disease of the heart alone. In certain fevers and in states of the blood in which the vasomotor nerves are irritated and cause peripheral contraction, as in scarlatina, accentuation of the second sound

Fig. 119.



Normal first and second sounds. Accentuated second sound.

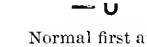
is observed. It may often be heard before there is other evidence of the development of local inflammatory diseases due to the same cause, as nephritis in scarlatina. The occurrence of this complication may be suspected when accentuation of the aortic second sound is heard.

2. *The Pulmonary Diastolic Sound.* Accentuation of this is due to a physical condition similar to that which causes accentuation of the aortic second sound. Anything that heightens the tension in the pulmonary artery will cause increased loudness. In health the pulmonary second sound is not so loud as the corresponding aortic sound. If, therefore, we find in the second or third left interspace the sound as loud as an aortic sound, or louder, it can be said that the pulmonary second sound is accentuated. (Fig. 119.) The causes are: 1. Any condition that causes congestion within the lungs, the right ventricle being at the same time of normal or increased strength. There is accentuation in the early stages of pneumonia, and, if the course of the disease continues favorable, the sound may remain accentuated to the end; if, on the other hand, the circulation is embarrassed, and the right heart is failing, the sound will become fainter, and may be scarcely recognizable. Such change in the sound accompanies increase of respiratory

distress, and indicates that the right heart is becoming exhausted. It is, therefore, an ominous sign in acute pulmonary disease. If the case is unfavorable, the signs of right-sided dilatation will subsequently occur. 2. Emphysema of the lungs. Notwithstanding the lung overlaps the heart, the sound can be heard and may be the only one of the four sounds that can be distinguished. 3. Valvular disease of the heart affecting the mitral orifice. The accentuation is due to increased tension in the pulmonary artery. In *mitral obstruction* the blood is retained in the auricle and pulmonary veins, causing resistance to the contraction of the right ventricle. Increased tension in the pulmonary artery is the result, with exaggerated strain upon the valves. In *mitral regurgitation* the blood is thrown back into the auricle with the systole, and consequently meets with blood coming from the lungs. This in time increases the amount of blood and the blood-pressure in the pulmonary artery. A heightened tension results. Skoda pointed out the significance of this association. Sometimes in doubtful cases, either in the presence or in the absence of a murmur at the mitral orifice, the occurrence of this sign makes it more than probable that there is mitral valvulitis.

Diminished Intensity or Feebleness of the Sounds. 1. *Feebleness of the mitral sound*, observed at the apex of the heart, may be an indication of weakness of the muscle from dilatation, atrophy, or myocarditis. It must be remembered, however, that weakness of the ventricle is not attended by enfeeblement of sound alone, but that when the right

Fig. 120.



Normal first and second sounds. Diminished first sound.

or left ventricle is weakened, the duration of the sound is lessened. The loudness remains the same or may be increased. Note, then, that a short systolic sound, loud, sharp, flapping, and sometimes reverberating, heard at the apex, indicates dilatation or feebleness. The tension of the ventricles and valves creating the sound is increased by internal pressure. The systolic sounds become like the diastolic and may be distinguished by the ear with difficulty; but if the time is taken with the finger on the apex-beat or carotid artery, if the heart's action is slow, the distinction can readily be made. (Fig. 120.)

2. *Diminished intensity of the aortic sound* is an indication of cardiac weakness and is apt to ensue in the course of fevers when exhaustion takes place. It is a sign of myocarditis and of degeneration of the muscular walls of the heart. Under these circumstances the systole of the ventricle is also weakened.

Feebleness of the aortic second sound, with hypertrophy and hence strong contraction of the ventricle, occurs when the aortic leaflets are swollen or enlarged and thickened. This condition of the valves is due to atheroma and is in all probability associated with atheroma of adjacent vessels, as the coronary arteries. It is therefore a sign of serious import.

3. *Diminished intensity of the pulmonary sound* is of importance in the

course of valvular disease of the heart, providing previous accentuation has been observed. If the marked loudness gives way to feebleness, there is strong probability that the right heart is undergoing dilatation with regurgitation at the tricuspid orifice. While accentuation of the pulmonary second sound in valvular disease is of good omen, enfeeblement of the sound is of bad prognostic omen, indicating weakness of the right ventricle.

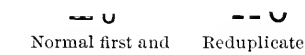
Alterations in the Rhythm. Fœtal Rhythm of the Heart. Embryocardia—a term first used by Huchard to designate a condition of rapid heart action in which the pauses between the heart-sounds are of equal length. The first and second sounds are exactly alike, resembling the beat of the fœtal heart. The sign is of importance in prognosis. In acute disease and in fever it indicates enfeeblement of the heart and reduction of arterial tension. In the later stages of Graves' disease it is a forerunner of death. It is distinguished from the rapid beat of the heart in tachycardia by the fact that in the latter condition the normal rhythm is preserved.

Cantering Rhythm of the Heart. The ear recognizes three sounds. The usual sounds may or may not be attended by murmur, and the interpolated sound may be dull, or short and sudden. It may occur at various periods in the cardiac cycle, either just before the systolic sound, just after the diastolic sound, or during the diastolic pause. The rhythm recalls the sound of a horse cantering. It was termed by Bouillaud the *bruit de galop*. When the interpolated sound resembles the first or second, it is similar to reduplication of the sounds. It has been observed in hypertrophy of the heart, especially of the left ventricle; in dilatation of the heart; in adherent pericardium with dilated hypertrophy; in myocarditis, in the course of fevers; and in excessive anæmia. It is heard loudest over the right and left ventricles. Potain thinks it is due to tension communicated to the wall of the ventricle by the entrance of blood into its cavity, and is more marked when the wall is least distensible, as in hypertrophy or after the muscle has become exhausted; in either of the two the walls vibrate more readily. The triple rhythm is of bad prognostic omen in chronic Bright's disease.

Reduplication of the Sounds. Reduplication, or apparent doubling, of the heart-sounds occurs in various forms. In health the systolic sounds are created synchronously; a fraction of a second, not appreciated by the ear, separates the diastolic sounds. In so-called reduplication, one systolic sound may follow the other, or the aortic and pulmonary diastolic sounds may be created at distinct intervals. As has been stated, in galloping rhythm the effect of reduplication is sometimes transmitted to the ear. Reduplication may take place in *health* under the influence of respiratory movements. The systolic sounds may be doubled at the end of expiration and the commencement of inspiration, while the diastolic sounds are doubled at the end of inspiration and the commencement of expiration. In mitral disease, reduplication, or want of synchronous closure of the aortic and pulmonary valves, is of frequent occurrence. The heart-sounds are doubled and heard over the base of the heart. Reduplication of the systolic sounds occurs in chronic Bright's disease.

Reduplication of Systolic Sounds. Reduplication or doubling of the systolic sounds is heard over the apex or the right ventricle. (See Fig. 121.) Several explanations have been given for the cause of the reduplication. At first it was thought to be due to want of synchronism in the action of the ventricles—that one ventricle contracted before the other, due to the fact, of course, that the presence of blood stimulates one but not the other. By Hayden it was thought that reduplication of the first sound was due to the two major elements of the sound acting synchronously, the muscular sound taking place before the sound produced by the tension of the valves. George Johnson took the view that the reduplication was due to the contraction of the auricle and ventricle; that the sound produced by the former was heard on account of hypertrophy of the auricle, and heard first because of the natural order of precedence. Thus far the reasons for each view have not been fully established.

Fig. 121.



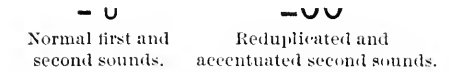
Normal first and second sounds. Reduplicated first sound.

Sansom believes that reduplication of the first sound is due to the shock communicated to the contents of the ventricle just before systole—that is, during the auricular systolic period; that, in other words, it is due to the indirect effect of the auricular systole. The contraction of the auricle makes tense the auriculoventricular valve of the left side. If it occurs late in the diastole, or just before the systole, reduplication of the first sound is produced; if early in the diastole, reduplication of the second sound is created.

Reduplication of the Diastolic Sounds. (See Fig. 122.) While held by some authorities to occur in a large proportion of healthy individuals at the end of inspiration and the commencement of expiration, other observers, equally careful, think that it is extremely rare. It is of frequent occurrence in the patients of the Philadelphia Hospital. This is no doubt due to the fact that so many of the inmates are the subjects of all forms of lung disease, or disease of the vascular system, with muscular degeneration of the heart, that the equability of the pulmonic circulation is disturbed. There is no doubt that it can be modified or induced by inspiration. It is usually heard at the end of inspiration and commencement of expiration. Actual reduplication of the second sound occurs when the normal asynchronism in the closure of the aortic and pulmonary valves is exaggerated. It has been found that the valve of the pulmonary artery closes a fraction of a second after the aortic valve. The ear usually fails to appreciate the difference unless there are differences of blood-pressure; when doubled, and therefore appreciated, it is indicative of a difference in blood-pressure between the two sides of the circulation. Increased resistance in either will lead to increased tension, quickened recoil, and hence quickened closure of the valve. The conditions that are associated with the doubling of the second sound are (1) and most frequently, mitral stenosis; (2) obstruction of the circulation in the lung—tuberculosis, emphysema, and bronchopneumonia; (3) dilata-

tion of the right ventricle; (4) myocarditis. The sound is heard at the second and third costal cartilages along the left edge of the sternum. It is frequently heard at the fourth and fifth cartilages on the left side. In cases of mitral stenosis it is heard near the apex.

FIG. 122.



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

FALSE REDUPLICATION. Simulated doubling, or false reduplication, is a sound produced at the mitral orifice. It is difficult to tell it from true doubling or reduplication. It is most distinct at the base of the heart along the left edge of the sternum. Occasionally it is more distinct near the apex than elsewhere. It occurs with the conditions found in true doubling and in mitral obstruction. *Cause:* Sansom, Cheadle, and others distinctly point out that this double second sound is of frequent occurrence and that it is heard most frequently at the apex. Sansom thinks that the cause of simulated doubling of the second sound is the same as that of doubling of the first. The first sound heard is the normal second sound, while the simulated second sound is produced by tension of the mitral curtain. This tension is due to the shock of the blood coming from the auricle to the ventricle.

III. Abnormal Sounds or Murmurs. Adventitious sounds may accompany or replace the normal sounds. These adventitious sounds are produced in the *pericardium*, in the *heart*, or in the *bloodvessels*. They are divided into friction-sounds and murmurs. They are recognized because they are a departure from the normal sounds or because they are superadded sounds.

Abnormal Sounds in the Pericardium. They are known as *friction-sounds* and *splashing* or *bubbling sounds*. (See Plate XXX.) The former occur in the first stage of pericarditis, and are due to the rubbing together of the inflamed surfaces, either the congested, vascular pericardium, or the membrane bathed in exudation or covered with lymph. The friction-sound is recognized by (1) its character, (2) time, (3) position, (4) transmission, (5) movability, (6) modification by position of patient, pressure, course of disease, etc.

1. The pericardial friction is usually of a to-and-fro character, and can be recognized as distinct from the heart-sounds. It resembles the rubbing or scraping together of two roughened surfaces.

2. It is not necessarily synchronous with the normal heart-sounds. It is a to-and-fro sound, systolic and diastolic in time. It may, however, be only systolic or only diastolic.

3. It is heard over the body of the heart, usually in the third and fourth interspaces, or even over the right ventricle.

4. It is not transmitted away from the heart. Its location may shift from day to day in the præcordial area.

5 and 6. It may be modified by pressure or by respiratory movement, or be influenced by the position of the patient. It may disappear entirely in the upright posture. An impression of nearness to the ear is given

by the sound observed in the first stage of pericarditis. It may be increased or lessened in loudness by a deep inspiration. It disappears during the period of effusion, to return after that is absorbed.

Diagnosis. It must be distinguished from *pleural friction*, which disappears when the patient is asked to hold his breath. The pericardial friction is of cardiac rhythm, the pleural friction of respiratory rhythm. It must also be distinguished from the so-called exocardial friction-sounds. The pleura adjacent to the pericardium may be inflamed. With each beat of the heart the rough surfaces of the pleura are agitated and generate a friction. This murmur is seated along the edges of the right auricle or left ventricle. It is systolic in rhythm, but has the special characteristic that it is modified by respiration. It may cease when the patient holds his breath. It is increased by inspiration, or diminished in expiration when the lungs recede from the heart in expiration. Pericardial friction must be distinguished from the crepitations and râles of cardiac rhythm produced by the impact of the heart against the lung. They disappear when the breath is held. The distinctions between pericardial frictions and cardiac murmurs will be considered later.

Splashing sounds are heard when the pericardium contains air and fluid. They may be bubbling or gurgling, or resemble the sound of a water-wheel. They continue when the breath is held.

Abnormal Sounds in the Heart and Vessels. Murmurs. If the student listens with the stethoscope over a large superficial vessel, and does not employ pressure, he will not detect any sound. If, however, pressure is employed, a sound or murmur is produced. The passage of the blood through the vessel produces no sound because the vessel or tube is of uniform calibre throughout. The pressure of the stethoscope alters the calibre and compels the fluid to pass through a narrow orifice into a wider space. In this manner a *fluid vein* is produced. The vibration of the molecules of the agitated fluid vein produces a sound or murmur. The loudness of the sound depends upon the swiftness of the flow. The sound in this instance is carried in the direction of the blood-current, hence the murmur is known as an *onward murmur*.

The reverse may take place. The fluid may flow backward from a wider into a narrower space without the production of sound; if, however, the fluid breaks on bevelled edges, as the leaflets of heart-valves projecting into the current, the fluid is again thrown into vibration and produces noise. If there is considerable constriction by the bevelled edge, the sound is carried farthest against the natural flow of the fluid—hence the term *backward murmur*. Some authors hold that murmurs are also due to lateral vibrations of the walls of the heart or of the vessels. Some murmurs may resemble tones, and are called *musical murmurs*. Such murmurs are due either to the vibrations of the solids set up by the vibrating fluid vein or to the vibrations of the fluid vein alone.

Murmurs are divided into two classes in accordance with their seat of development. Murmurs originating in the heart are known as cardiac murmurs. Murmurs originating in the bloodvessels are vascular murmurs. (See the Arteries.) Cardiac and vascular murmurs are divided into (1) *organic* murmurs, if due to physical changes of the heart or

vessels; (2) *inorganic, functional,* or *hæmic,* if due to changes in the quality of the blood. (See Functional Murmurs.) Cardiac murmurs are always generated at the orifices from disease or from incompetency of the valves, or from a patulous non-valvular opening. The orifices are *valvular* and *non-valvular.*

Murmurs at Valvular Orifices. The valvular orifices and their anatomical relations have been described. Murmurs are produced at these orifices when they are open or when normally they should be closed. If the murmur is produced when the orifice is open, it is because there is narrowing of the orifice or dilatation of the cavity (relative narrowing). The murmur, then, is always produced *with* the natural current of blood, and hence is known as an *onward* or *obstructive* murmur. It always or nearly always implies organic disease at the valve-orifice, hæmic murmurs excluded. If the murmur is produced when the orifice should be closed, and hence when the valve leaks, it is because the valves are diseased and can not shut the orifice, or because they are too small—incompetent—to shut it. Such murmurs are produced *against* the natural current of blood, and are known as *backward* or *regurgitant* murmurs.

Murmurs at Non-valvular Orifices. The orifices of the vena cava and of the pulmonary veins, and of the perforations of the septa in congenital heart disease, are non-valvular. They are at times the seat of murmurs—as in open foramen ovale or perforated ventricular septum. A patulous ductus arteriosus may also be the cause of a murmur.

Diagnosis of Murmurs. The student has learned that an abnormal sound or a murmur is present. It is necessary then to determine, *first,* at which orifice the murmur is produced (the seat of the murmur), and, *second,* the kind of murmur—obstructive or regurgitant. Murmurs are therefore studied as heart-sounds are studied, as to their *position* of maximum intensity, their *time,* and the direction of their *transmission.* The position of the murmur indicates which valve-orifice is affected; the time and the direction of transmission of the murmur indicate the nature of the lesion and the kind of murmur—whether obstructive or regurgitant.

The Position of Maximum Intensity of the Murmur. *The Orifice Affected.* We are enabled accurately to determine the orifice at which the murmur is generated by noting the position of maximum intensity of the murmur. This position corresponds usually to the area at which the normal sound of the affected valve is heard loudest. It must be remembered that the cardiac orifices are closely situated, and that, therefore, the murmurs must be generated within a small area, so small that it would be impossible to ascertain at which valve-orifice the murmur is created, were it not for the fact that under the laws of conduction of sound, the murmurs are conducted away from their point of origin to certain definite stations, where in health the respective valve-sound is also heard loudest.

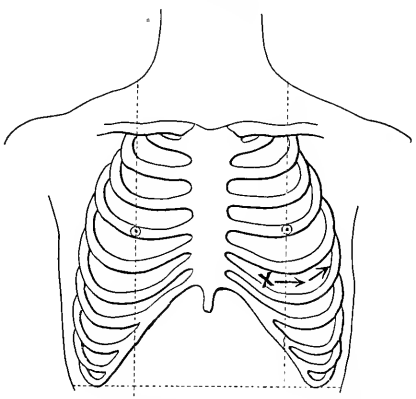
1. *Murmurs at the Apex—the Mitral Area.* A murmur heard loudest, or with the greatest intensity, at the apex is known as a mitral murmur. It is created at the mitral orifice, but is conducted to the apex by the left ventricle, which is nearest the chest-wall at this point. (See 1, Fig. 117, and Plate XXXII.)

2. *Murmurs at the Xiphoid Cartilage—the Tricuspid Area.* The murmur is heard loudest at the xiphoid cartilage or the head of the fourth or fifth rib. It is created at the tricuspid orifice, and is heard most distinctly over the lower portion of the sternum, and along the left edge, because the right ventricle is in apposition with the chest-wall at this spot. (See 2, Fig. 117, and Plate XXXIII.)

3. *Murmurs at the Second Costal Cartilage or Second Interspace on the Right—the Aortic Area.* When a murmur is heard with greatest intensity at this point, it is usually generated at the aortic orifice, and is conducted to this region by the aorta, which comes nearest to the surface of the chest at this point. (See 3, Fig. 117, and Plates XXXI. and XXXVI.)

4. *Murmurs in the Second Left Interspace—the Pulmonic Area.* A murmur heard loudest at the second interspace along the left edge of the sternum is generated at the pulmonary orifice; it is heard loudest in this area because the pulmonary artery is nearest the chest at this point. (See 4, Fig. 117, and Plate XXXIV.)

FIG. 123.



Maximum intensity of murmur of mitral regurgitation; systolic; transmitted to the left.

THE RHYTHM OR TIME OF THE MURMUR. *The Kind of Murmur.* Having determined the point of maximum intensity of the murmur, hence the valve at which it has its origin, we next wish to determine the kind of murmur. A murmur which is produced at orifices when they should be closed is known as the murmur of regurgitation, as the valve permits the blood to flow backward. A murmur that occurs when the blood should in health be passing through an orifice is known as a murmur of obstruction, as the flow of blood is obstructed. We have to determine whether the murmur at an orifice is due to *regurgitation* or to *obstruction*. This is ascertained by the *time* of the murmur.

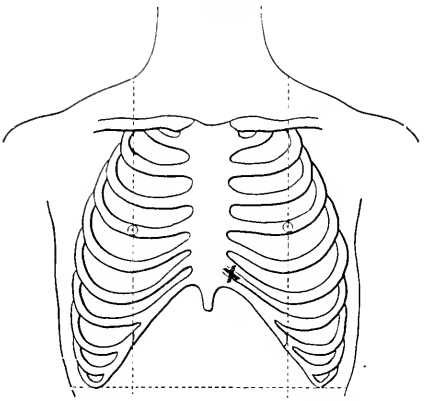
The time of the murmur is determined by comparing it with the heart-sounds, the apex-beat, or the carotid pulse.

Murmurs with the Systole.

1. *In the Mitral Area.* In health during systole the auriculoventricular valve is closed. The murmur indicates that there is such disease

as to permit of a backward flow of blood or of regurgitation into the auricle. It is the murmur of *mitral regurgitation.* It may be due to disease of the valves (organic incompetency) or to relative incompetency. (See Fig. 123, and Plate XXXII., Fig. 1.)

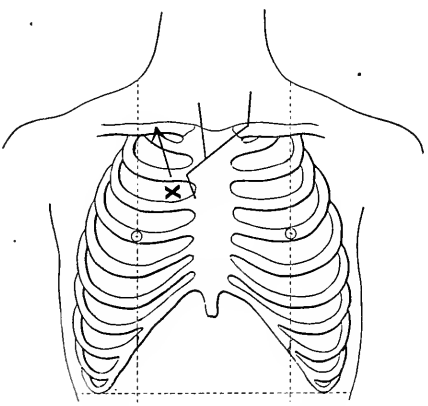
Fig. 124.



Maximum intensity of murmur of tricuspid regurgitation: systolic.

2. *In the Tricuspid Area.* As on the left side, the murmur in this area is due to valvular disease, organic incompetency or relative incompetency, which permits of regurgitation, *tricuspid regurgitation.* (See Fig. 124, and Plate XXXIII., Fig. 1.)

Fig. 125.



Position of maximum intensity and directions of transmission of murmur of aortic obstruction.

3. *In the Aortic Area.* During this time the blood is flowing from the ventricle into the aorta. If there is disease that causes obstruction at the orifice, the murmur of *aortic obstruction* is produced. The murmur may be due to anæmia, disease or malposition of the aorta. (See Fig. 125, and Plate XXXI., Fig. 2.)

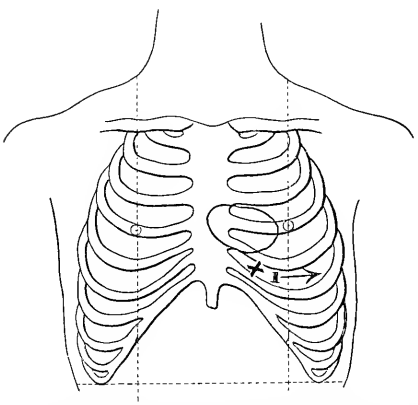
4. *In the Pulmonary Area.* The pulmonary orifice is affected in the

same way as the aortic orifice under the same circumstances. The murmur is due to *pulmonary obstruction*. It is exceedingly rare. A murmur here is more frequently hæmic. (See Fig. 128, and Plate XXXIV., Fig. 2.)

Murmurs with the Diastole.

1. *In the Mitral Area.* The blood is flowing from the left auricle to the left ventricle. Disease of the valves obstructs the flow. The murmur occurs in the beginning, in the middle, or at the end of the long silence. Mid-diastolic and late diastolic, or, because it occurs before the systole, presystolic are the terms applied to this murmur. It is the murmur of *mitral obstruction*. (See Fig. 126, and Plate XXXII., Fig. 2.)

FIG. 126.



Maximum intensity of murmur of mitral obstruction; presystolic, localized, or transmitted as area shows. 1. Normal impulse. ○. Area of reduplication of second sound.

2. *In the Tricuspid Area.* It occurs for the same reason and at the same time as the diastolic murmurs generated at the mitral orifice. It is rare; although it is more common to find *tricuspid obstruction* than is usually supposed. (Plate XXXIII., Fig. 2.)

3. *In the Aortic Area.* The aortic valve closes in the diastole. A murmur indicates that it is so diseased that it can not prevent blood flowing backward or regurgitating into the ventricle. It is the murmur of *aortic regurgitation*. A murmur of the same time and in the same situation may be due to dilatation or aneurism of the aorta. (See Fig. 127, and Plate XXXI., Fig. 1.)

4. *In the Pulmonary Area.* A diastolic murmur in this area is due to *regurgitation* at the *pulmonary orifice*. (See Fig. 128, and Plate XXXIV., Fig. 1.)

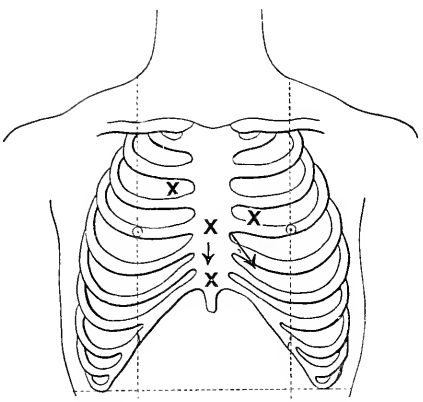
Murmurs are divided as to time into systolic and diastolic murmurs. The above shows that we may have practically only three systolic and two diastolic murmurs. The systolic murmurs are *aortic obstruction* and *mitral* and *tricuspid regurgitation*. The diastolic murmurs are *aortic regurgitation* and *mitral obstruction*.

THE DIRECTION OF TRANSMISSION. It depends upon the situation of the murmur and the time at which it is produced. Some murmurs are not

transmitted. The transmission is usually in the direction of the currents that produce them.

Murmurs in the Mitral Area. To the Axilla. A murmur heard at the apex with the systole and caused by *regurgitation* at the mitral orifice, is transmitted into the axilla and may be heard at the angle of the scapula. The murmur produced in the same area before the systole—*obstruction*—is usually not transmitted. It is heard at the apex, or a little inside of the apex, or may rarely have its point of maximum intensity in the third interspace. Sometimes it is transmitted to the axilla and to the angle of the scapula. (See Figs. 123 and 126, and Plate XXXII.)

FIG. 127.



Positions of maximum intensity and directions of transmission of murmur of aortic regurgitation.

Murmurs in the Tricuspid Area. The murmur of tricuspid regurgitation is not transmitted. It is heard over a relatively large area, depending upon the intensity of the sounds. (Plate XXXIII.)

Murmurs in the Aortic Area. Upward and along the Vessels. The murmur, *systolic* in time, heard at the second costal cartilage at the right, due to aortic obstruction, is transmitted in the direction of the blood-current. The sound is conducted by the vessels and by the fluid; it is therefore heard along the course of the aorta and in the carotid arteries. *Downward along the Sternum and to the Apex:* The murmur of aortic regurgitation, heard in the same way, is transmitted downward along the course of the sternum. It may be transmitted to the apex, or may be heard along the sternum only. The left ventricle conducts this murmur. (See Figs. 125 and 127, and Plate XXXI.)

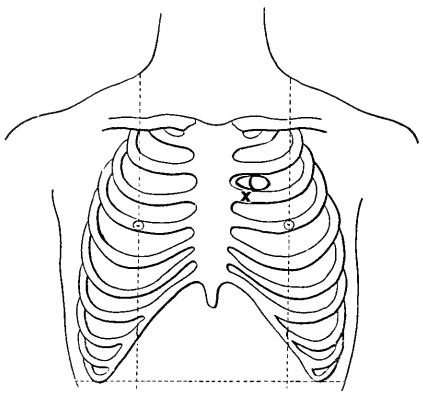
CHARACTER OF THE MURMURS. Murmurs are further distinguished by their character and the degree of *loudness.* By the character of the murmurs we are aided (1) in distinguishing them from heart-sounds; (2) in estimating the nature of the lesion that produces the murmur; (3) in judging, in the case of the murmur of mitral obstruction, of the presence or absence of that disease.

DISTINCTION FROM NORMAL SOUNDS. Normal sounds are sounds of tension; murmurs are sounds of rhythmical vibration. The normal sounds of the heart have been described by the syllables "*ubb,*" "*dupp,*"

"*od*," and abnormal sounds of endocardial origin by "*uf*," "*uv*," "*us*," "*ush*," or by full vowel-sounds as "*oo*," "*u*," "*ah*," and "*aw*," by musical tones, or by interrupted tones, or by general sounds as "*urr*" or "*orr*."

The Nature of the Lesion. The murmurs may be soft or blowing, rough or rasping, musical or whistling. They may be high or low in pitch. Murmurs that are rough and high in pitch are usually due to disease of the valves, causing thickening or stiffening of the leaflets, or to the projection of an atheromatous plate into the lumen of the

Fig. 128.



×, Maximum intensity of pulmonary systolic murmur. ⊃, Area of murmur of anæmia.

orifice. Such conditions occur in chronic endarteritis and chronic endocarditis or valvulitis. On the other hand, murmurs that are soft and low in pitch are usually due to a physical condition which causes swelling of the valve or occlusion by soft exudations; they are heard in endocarditis of rheumatic origin, or the malignant form of endocarditis. The only murmur that has special characteristics is the murmur of *mitral obstruction*. It is a prolonged murmur of a churning or grinding character, sometimes rippling as if fluid were being forced through a narrow channel. It is usually presystolic, but may occur in the middle of the diastole.

Loudness. The *loudness* of the murmur is not of special significance, although, in general, it may be said that it indicates good compensation, and that the heart-muscle is sufficiently strong to meet the demands of the circulation. Murmurs are louder in the recumbent than in the erect posture in some instances, especially mitral and tricuspid murmurs. Murmurs are often more distinct after exertion. Loud murmurs may become weak, and this change in the character of the sound is of serious omen. They may disappear in the course of fevers and during the dying state.

Disappearance of Murmur. The student will often find that after a patient has been under treatment for a short time the murmurs disappear. This is probably due to the fact that there is complete compensation. In the terminal stages of cardiac disease they disappear because

of weakness of the heart muscle. Rarely they disappear because the roughened valve causing them has been repaired.[1] In other cases it may be necessary to bring out a faint murmur or increase its intensity by having the patient move about; this renders it most distinct by inducing more rapid action of the heart.

THE SIGNIFICANCE OF MURMURS. Murmurs heard at the various orifices indicate either (1) disease of the valves; (2) incompetency of the valves; (3) disease of the blood; or (4) disease of the vessels in intimate relation with the heart. The systolic murmur at the second costal cartilage on the right may be heard when there is disease at the aortic orifice, causing obstruction; in atheroma of the aorta; in cases of aneurism just above the valves; in anæmia and chlorosis, and in some vasomotor neuroses, as Graves' disease. Before concluding that the murmur is due to disease of the valves we must be able to exclude the other conditions. *Atheroma of the aorta* is most difficult to distinguish from obstruction, because the character of the murmur is the same and the associated conditions are similar. In both there may be a previous history of gout, rheumatism, syphilis, or alcoholism. The latter diseases are associated with atheroma in other arteries of the body, and with the degenerative changes that accompany atheroma. In young subjects with a direct history of rheumatism, or when the process has followed septicæmia, the probabilities are in nearly all the cases that the murmur is due to aortic obstruction. To recognize the murmur of anæmia, chlorosis, or Graves' disease is often difficult. The associate symptoms in each case are different, however, and with the changes in the blood, indicate the significance of the murmur.

In other valve-areas the chief task is to decide whether the murmur is *organic*, due to valvulitis or relative incompetency of the valve, or functional, due to anæmia or other exocardial causes.

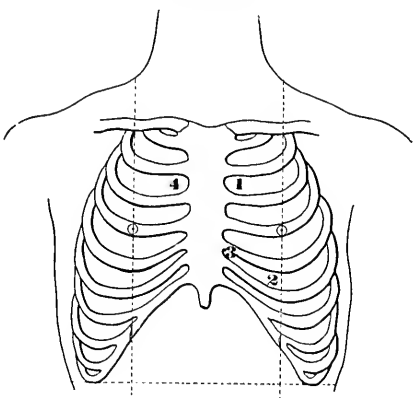
Murmurs due to Incompetency. The valves are sometimes unable to close properly. The cavity of the ventricles may increase in size, so that the valves do not coaptate to close the widened orifice. The tricuspid and mitral valve leaflets often become thus incompetent. Mitral and tricuspid regurgitation ensue. The murmurs are soft and low in pitch and not widely transmitted; the heart is dilated.

Murmurs of Anæmia. The murmurs of anæmia have some characteristics that aid in distinguishing them from true organic murmurs. The most important of these are: (1) the situation of the murmur; (2) its character; (3) the direction in which it is transmitted; (4) the time; (5) the associate signs; (6) the secondary changes in the heart muscle. 1. The murmurs of anæmia may be heard at any orifice, but are usually heard at the second costal cartilage or the third interspace on the left side. They are generated at the pulmonary orifice, or in the cone of the right ventricle. The murmur at the pulmonary orifice may be heard as high as the second interspace, but otherwise is not transmitted. Murmurs of anæmia are also heard at the apex, at the aortic cartilage, and over the tricuspid area. They are comparatively infrequent in these situations, but partake of the same nature as the murmur heard at the pulmonary orifice.

[1] See "Disappearance of Murmurs," by the author. British Medical Journal, 1897.

2. They are soft in character and low in pitch. They are louder in the recumbent than in the upright position. Their loudness is increased by violent cardiac action. They are loudest just at the end of expiration or beginning of inspiration. 3. They are not transmitted away from the heart. 4. They are systolic in time. 5. They are associated with murmurs in other parts of the vascular system, as the murmur in the jugular veins. Its characteristics and mode of recognition will be described elsewhere. 6. Mural changes, as general dilatation, fatty degeneration, or hypertrophy, may be present; but single chambers do not undergo change. The murmur of anæmia may usually be considered to be temporary.

Fig. 129.



Maximum intensity of murmurs of anæmia, systolic. 1. Pulmonary artery, 59 per cent. 2. Apex, 7 per cent. 3. Right ventricle and conus, 11 per cent. 4. Aortic area, 11 per cent. 1 and 2. Pulmonary and apex coexisting, 9 per cent. (After Sansom.)

Functional Murmurs not Anæmic. Drummond divides functional murmurs into three classes: cardiohæmic or anæmic; cardiomuscular or neurotyptic, and cardiorespiratory. The first has been considered above. The *cardiomuscular murmur* attends excited action of the heart. It is heard loudest at the fourth left interspace close to the sternum; loudest in the upright posture; loudest at the end of expiration. It disappears at the end of inspiration or when the patient lies on the side. It is, of course, increased by exertion and excitement. It is rough or whizzing in character. The *cardiorespiratory murmur* is fairly common. It is most marked in inspiration, but may be heard in both acts. It is systolic in time, and is heard loudest at the apex, but I have often heard it along the left border of the heart as high as the second rib, and in the axilla and at the angle of the scapula. It is short and whiffing, and the sound gives one the impression that the heart is striking the lung.

Influence of Pressure. Pressure exerted, Sewall says, while using the flexible stethoscope over the second costal interspace annuls in part or wholly the second sound of the heart; but if the ascending aorta be dilated or the site of an aneurism, the second sound persists strongly notwithstanding firm pressure.

Further, firm pressure removes (more or less)—

(*A*) 1. Hæmic murmurs over the base of the heart (save Jenner's pulmonary murmurs).
2. An aortic obstructive murmur at the apex.
3. When mitral and aortic regurgitant murmurs coexist, the aortic murmur is diminished in the greater degree.
4. Aortic regurgitant murmurs over the second right intercostal space.

while it does not markedly affect—

(*B*) 1. Mitral regurgitant murmurs heard over the apex ; or
2. Mitral obstructive murmurs over the same spot.
3. Tricuspid regurgitant murmurs over the area of greatest intensity.
4. Aortic regurgitant murmurs over the apex (see (*A*), No. 3).

Secondary Effect of Valve-lesions on the Heart and Pulse. The secondary effect of valve-lesions on the heart and pulse aid in the diagnosis. While we are enabled by the time of the murmur, its position, and direction of transmission, to affirm the nature of the disease at the respective valve-orifices, other physical signs further aid us in determining more precisely the lesion and its seat. They are derived from the heart and the pulse. They depend upon the secondary effect of the lesion upon the heart and upon the circulation.

In *aortic obstruction*, on account of obstruction to the flow of blood, the left ventricle hypertrophies ; moreover, the blood-stream is lessened in volume, and hence the pulse is small and of high tension. The physical signs of hypertrophy of the left ventricle and a small slow pulse are corroborative evidence of this lesion at the aortic orifice.

In *aortic regurgitation* the blood flows back into the ventricle. On this account, therefore, some dilatation takes place, a dilatation which, if compensation is perfect, is overcome by hypertrophy. The signs of enlarged left heart are present, however, as shown by inspection, palpation, and percussion. But the pulse of aortic regurgitation is of the greatest diagnostic significance. With the finger on the radial, the impression is at once received of recedence of the pulse-wave as soon as it strikes the finger. This is more marked if the hand is elevated. It is the water-hammer, or Corrigan's pulse.

In *mitral regurgitation* the left auricle does not change, but the stress is thrown upon the right side of the heart, and we have the signs of right-sided hypertrophy and dilatation ; but more marked than this is the evidence of high tension of the pulmonary artery, shown by accentuation of the second sound. (See page 437.) In mitral regurgitation the blood flows back into the auricle, and when the right heart weakens *engorges* the venous system. The arterial system is in consequence devoid of blood, and hence the arteries are empty. The pulse is small and feeble. The depleted coronary arteries do not nourish the ventricle, hence dilatation or failure in nutrition soon ensues, and the heart is further weakened. In addition to being small and feeble, the pulse, on account of inefficient and hurried contractions of the ventricle, is irregular and intermittent.

In *mitral obstruction*, in addition to the characteristic murmur, the thrill is of great significance. Moreover, the left auricle hypertrophies, and shortly afterward the right heart. The pulmonary second sound is accentuated and frequently doubled. The pulse is small and feeble.

Multiple Cardiac Murmurs. More than one murmur may be heard over the heart. The number depends upon the number of valves that are the seat of disease and the lesions at the orifices. We may have valvulitis of the aortic, mitral, and tricuspid valves conjoined. More commonly one valve is diseased, giving rise to a murmur, while another valve is relatively incompetent, on account of dilatation of one or more of the cavities of the heart, and a murmur is thus generated at its orifice. It is common to see aortic obstruction from valvulitis and mitral regurgitation from incompetency; mitral obstruction or regurgitation from valvulitis, and tricuspid regurgitation from incompetency. I have seen double aortic disease (combined obstruction and regurgitation), double mitral disease, and tricuspid regurgitation. The diagnosis of the various murmurs will be discussed in the section on Valvulitis. (See Plate XXXV.)

The Arteries. The stethoscope should always be used in examining the arteries. The double stethoscope is preferable, as strong pressure must be avoided upon the vessels. When the single stethoscope is used, some diagnostic value attaches to the character of the shock that is transmitted to the head. The arteries open to auscultation are the carotids when the head is slightly extended; the subclavian; the innominate above the sternoclavicular articulation; the brachial artery in the bend of the elbow, with the arm slightly extended; and the crural artery just below Poupart's ligament. The normal systolic and diastolic heart-sounds are often heard in the carotid and subclavian arteries. The systolic sounds may be heard over the abdominal aorta, due to tension of the vessels. The diastolic sound is rarely heard in this situation. In the other vessels no sounds are heard.

Induced or Pressure-murmur. By pressure with the stethoscope over one of the vessels its calibre is modified and a murmur created. This murmur corresponds in time with the pulse, hence it is systolic, and increases or diminishes in intensity, depending upon the amount of pressure placed upon it. Just here may be mentioned the systolic humming which is heard in children between the third month and the sixth year over the fontanelles and sometimes over the rest of the head. (See the Head.)

Diseases outside of the bloodvessels may give rise to what may also be called pressure-murmurs. When heard over the subclavian artery, the pressure-murmur may be due to adhesions or consolidation at the apex of the lung. It is more frequently heard at the left, and may only be present during full expansion of the lung. It is due to temporary pulling or bending of the artery during deep breathing. When it occurs on both sides, it is not of much significance. Murmurs in the axillary artery, or in any arteries surrounded by enlarged lymphatic glands, are created by their pressure. Murmurs in the thyroid gland have been referred to. (See Goitre.)

Abnormal Murmurs. Abnormal sounds or murmurs are due to altera-

tions of the blood, disease outside of the vessels causing pressure, and disease of the vessels. Murmurs from disease of the vessels, as the aorta, are discussed under the head of arteriosclerosis or aneurism.

Conduction-murmurs. Murmurs may be propagated into the arteries. A systolic murmur created at the aortic orifice may be heard in the vessels of the neck and along the aorta. On the other hand, in aortic regurgitation, the diastolic sound normal in the carotid and subclavian disappears, and the diastolic murmur is not heard. *Double Sounds of the Vessels:* Double sounds are sometimes heard in the crural artery under the following circumstances: (1) in aortic insufficiency; (2) in mitral stenosis; (3) in lead-poisoning; (4) in pregnancy. Duroziez's double murmur, heard when great pressure is used by the stethoscope, occurs in aortic regurgitation when there is good compensation. Many authorities refer to this as a valuable diagnostic sign in this affection. The double sound in all instances occurs with a large and quick pulse. It is probably caused by sudden collapse of the artery and the reflux blood-current which is possibly an aortic regurgitation.

Murmurs due to Alterations of the Blood. They are generated in anæmia and chlorosis. They are called functional murmurs, to distinguish them from murmurs due to disease of the vessels. They are systolic in time. They are soft and low in pitch, often of a musical character. The degree of loudness may vary with the position of the patient. They are increased by excitement. The intensity of the murmur increases in the course of fevers.

Murmurs in Relaxed Vessels. Murmurs in the vessels, apparently of functional origin, are sometimes heard. The vessels are dilated from actual disease. The increased calibre favors the development of a murmur by the creation of a fluid vein. Dilatation of the innominate artery sometimes takes place, giving rise to a murmur, which in loudness and character simulates the murmur of aneurism. A functional murmur is sometimes heard in the vessels, independently of disease, in cases of aortic regurgitation. The murmur is systolic in time.

Murmurs due to Disease of the Arteries. In the aorta the murmurs are due to aneurism or atheroma, or both. They may be systolic or diastolic. In the smaller vessels both conditions may be present, although atheroma is the usual one. The murmur is systolic in time, rough in character, strong or weak. It is associated with other signs of atheroma.

The Veins. In health no sounds are heard. Two conditions contribute to the creation of a *murmur* in the veins: (1) change in the character of the blood; (2) dilatation with the occurrence of positive venous pulse.

The Venous Hum. In anæmia and chlorosis, and sometimes in health a hum or murmur, or buzzing sound is heard over the jugular veins. It is louder on the right side than on the left. It is soft and low in pitch, and may be musical; it has been described as humming or whizzing. It is continuous. For its detection a double stethoscope should be used, as pressure increases it, and the patient should not turn the head to one side, as it is increased when this position is taken. The murmur is modified by the respiration and by the cardiac action. It is louder in deep inspiration when the blood is going more rapidly to the

thorax. It is also louder in the upright position. It is frequently louder during the diastole. The increased loudness at these periods occurs because, from the sucking action during inspiration and during the diastole, the blood is drawn more rapidly toward the heart. The murmur is caused by the flow of blood from the narrow jugular into its wider bulb, producing a fluid vein. Recent authorities believe it to be due to lateral vibration of the walls of the veins. Similar murmurs are heard in other veins, as in those of the extremities when the anæmia is profound. They are stronger during the diastole of the heart. The venous hum is sometimes heard at the lower border of the liver, to the right of the median line, in cirrhosis of the liver. It is created in the enlarged collateral veins. It may be modified by pressure of the stethoscope. It may be heard in this situation in emaciated and cachectic individuals not the subject of cirrhosis. The venous hum may be heard in the innominate veins (first and second interspaces and right costoclavicular articulation) and in the subclavian and axillary veins.

CHAPTER XXXV.

PHYSICAL DIAGNOSIS OF DISEASES OF THE LUNG.

Anatomy. The *thorax* is a conical-shaped bony case, formed by the sternum, costal cartilages, ribs, and vertebræ, covered by the muscles of the chest and back and by the mammary glands, and enclosing the trachea, the bronchi, the lungs, the heart, and the great vessels. Its floor is the diaphragm.

The *lungs* are irregularly pyramidal in shape. The apices are directed upward and extend above the clavicles. The bases are deeply concave and rest on the diaphragm. The convex external surfaces are in close contact with the ribs. The concave internal surfaces are in contact with the heart and the great vessels. The left lung is divided into two lobes; the right lung into three. The lungs are composed of a relatively small amount of tissue. They are made up of tubes and canals and of connective tissue. There are three sets of channels: (*a*) for the passage of air, (*b*) for the flow of blood, and (*c*) for the flow of lymph. The air-tubes are lined with mucous membrane.

The *pleuræ* are closed sacs containing a small amount of serum. Each sac is flattened out so that the internal surface of the walls are in contact. As a two-layer covering it encloses the lung, the external surface of the inner layer being in contact with the lung and the external surface of the outer layer being in contact with the ribs. The former is called the visceral layer of the pleura; the latter, the parietal layer.

The *muscles* concerned in ordinary respiration are the diaphragm, the quadrati lumborum, the serrati postici inferiores, the scaleni, the intercostales externi, and the levatores costarum. Those called into play in addition during forced respiration are the sternocleidomastoides, the infrahyoides, the pectorales majores and minores, the trapezii, the rhomboides, the erectores spinæ, the intercostales interni, and the abdominal muscles.

Nerves are supplied to the muscles of the chest and of the tubes, to the mucous lining of the respiratory tract, and to the various structures concerned in respiration. They include fibres from both the cerebro-spinal and the sympathetic systems. The principal ones are the pneumogastric, including the inferior or recurrent laryngeal branch, the glossopharyngeal, the trigeminal, the phrenic, and other muscular and cutaneous nerves.

Physiology. Respiration is a reflex act in response to a stimulus received in a nervous centre and transmitted by efferent fibres to the muscles of the thorax. The expansion of the chest causes air to fill the alveoli of the lungs between which and the pulmonary capillaries the respiratory exchange of gases takes place. The excitation for *inspiration*

is supplied by the carbon dioxide in the circulating blood and by stimulus derived from collapse of the lung. The impulses are conveyed by efferent nerve-fibres to the inspiratory centre in the medulla, which is connected by efferent fibres with the muscles of inspiration. The presence of oxygen in the blood and the expansion of the lung excite the rhythmic action of the expiratory or inhibitory centre in the medulla controlling the expiratory muscles in forced *expiration*.

The Value of Physical Diagnosis.

By physical examination of the lungs (1) the degree of activity (movement) and (2) the physical condition of the parts subjected to examination are ascertained; but the disease itself is not diagnosticated. Abnormal signs simply indicate an abnormal condition, which may be due to any number of diseases. As the lungs in health contain air, any physical change that takes place in them causes either an increase or a diminution in the amount of air they contain. This may be general (bilateral), or it may be limited to one side (unilateral), or to a smaller area (local). An increase or diminution in the amount of air in a part suspected to be the seat of disease is determined by percussion. As adjuncts are the phenomena that can be elicited by means of inspection, palpation, and auscultation, which depend upon the movements of the lungs and upon the sounds produced in breathing and speaking.

In the study of lung disease too much emphasis has been laid in the past on auscultation and percussion. It is the habit to rely too much on these methods, to the exclusion of the simpler and quite as valuable methods—inspection and palpation. The latter have been employed for a long time in the study of the objective phenomena of disease. The former are comparatively modern methods, and, to put the findings on an accurate basis, have required in addition to exhaustive comparative research, special cultivation of senses not usually employed in observation. The pernicious habit of examining a patient without removing the clothing, either through haste upon the part of the physician or through false modesty upon the part of the patient, has unfortunately also led to the neglect of inspection and palpation. It is proper to insist that the data obtained by inspection and palpation are as important and valuable as those obtained by other means. They are even more suggestive or diagnostic of physical conditions. The phenomena observed through them are more positive and surrounded by fewer qualifications.

Topographical Anatomy. In order that the condition of the underlying parts may be learned from the physical signs elicited upon the chest, a knowledge is required of the relations of the various organs with the chest-wall.

Diaphragm. The apex of the diaphragm is on a level with the eighth dorsal vertebra.

Trachea. The trachea bifurcates at the plane which passes horizontally from the junction of the first and second portions of the sternum to the body of the fourth dorsal vertebra.

PLATE V.

FIG. 1.—Anterior Aspect.

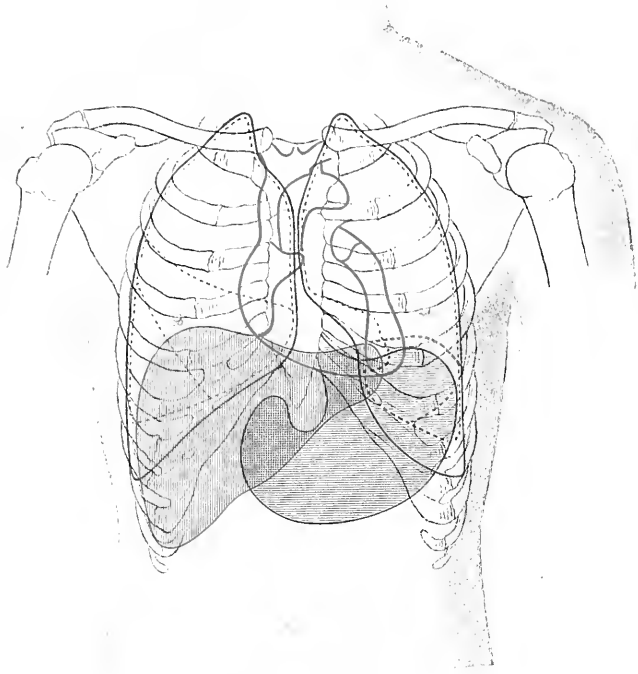
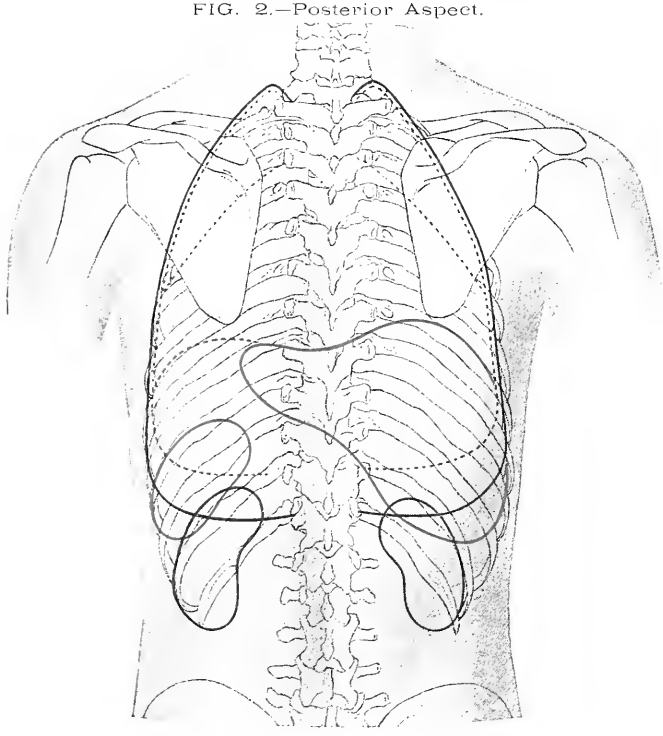


FIG. 2.—Posterior Aspect.



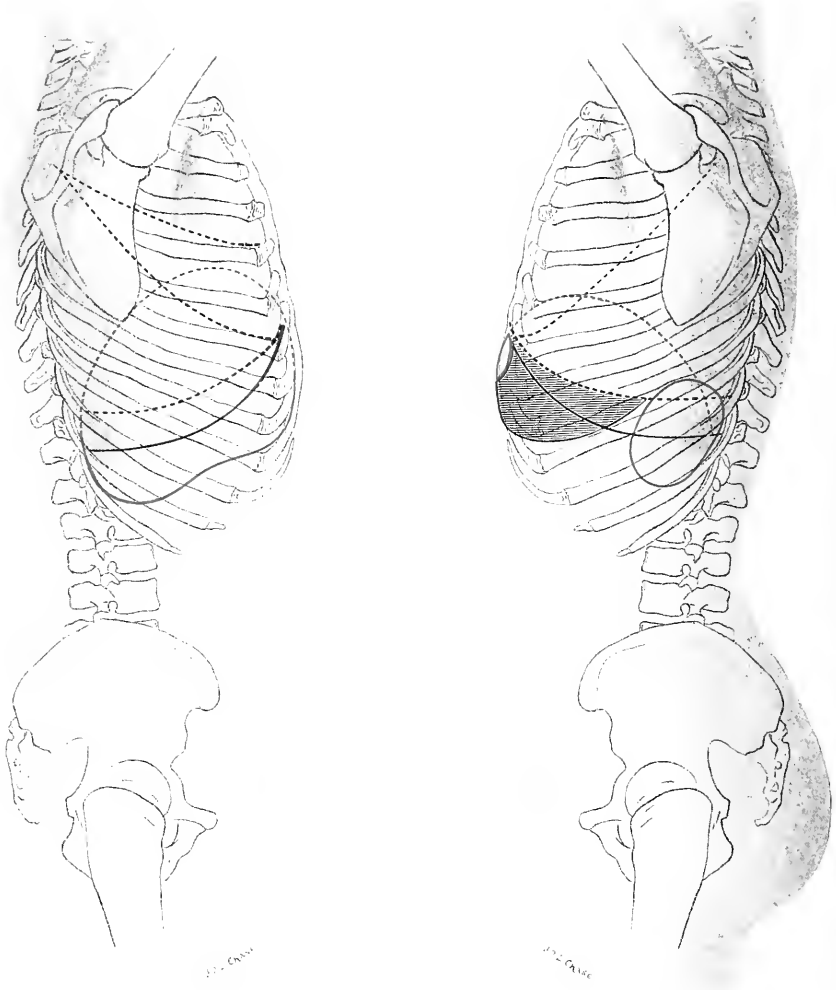
Situation of the Viscera.

Outlines of heart and vessels—broad red lines. Margins of lungs and individual lobes—dotted green lines. Limits of pleural sacs—solid green lines. Liver—red shading. Stomach—green shading.
(In part after His-Spalteholz and Luschka.)

PLATE VI.

Fig. 1. Right Lateral Aspect.

Fig. 2. Left Lateral Aspect.



Situation of the Viscera.

Margins of lungs and of individual lobes—dotted green lines. Limits of pleural sacs—solid green lines. Liver and spleen—solid red lines. Diaphragm—dotted red lines. Stomach (portion not covered by lung)—green shading.
(In part after Luschka.)

Lungs. The apices of the lungs extend in front 3 to 7 centimetres ($1\frac{1}{5}$ to $2\frac{3}{4}$ inches) above the clavicles; behind, to a line drawn transversely through the spinous process of the seventh cervical vertebra. The lower margin of the right lung, when the chest is passive, commences in front at the insertion of the sixth costal cartilage into the sternum, runs parallel with the upper border of the sixth rib to the axillary line, and then descends to the upper margin of the seventh rib. The lower margin of the left lung begins at the sixth costal cartilage. Posteriorly the lower border of both lungs extends to the tenth rib. With full inspiration the lungs descend both in front and behind almost the extent of one interspace.

The position of the lungs and of their lobes is shown in Plates V. and VI. The *upper lobe* of the *right* lung extends in front to the fourth rib, laterally to the third, and behind to the spine of the scapula. The *lower lobe* extends to the tenth rib behind, and, when fully expanded, in the axillary region as well. The *middle lobe* is not seen behind; in the axillary region it extends from the third to the fourth rib in inspiration; in front, from the lower margin of the upper lobe to the sixth rib. The *upper lobe* of the *left* lung extends to the sixth rib in front, to the fourth interspace at the side, and a little above the spine of the scapula behind. The *lower lobe* extends to the base of the lung behind and to the level of the eighth rib at the side.

Pleuræ. The pleural sacs extend below the lower border of the lungs: 2 inches in the midclavicular line, $3\frac{1}{2}$ inches in the midaxillary line, and $1\frac{1}{2}$ inches in the scapular line.

The Landmarks of the Chest. For the purpose of bearing in mind the relations of the organs to the surface of the chest, and the localizing and proper recording of the seat of the disease, certain landmarks are employed, consisting of anatomical points on the surface of the chest and artificial vertical lines. Knowledge of the landmarks which indicate on the surface the position of the parts beneath is of great importance in diagnosis. By means of them the exact location of a diseased area can be indicated. For accuracy in localization, knowledge of the methods of determining the landmarks, and especially of counting the ribs, is essential.

The Imaginary Vertical Lines. Of these, there are six drawn to the right and to the left of the median line: (1) the *parasternal line*, drawn parallel with the sternum and midway between its border and the midclavicular line; (2) the *midclavicular line*, drawn from the middle of the clavicle, generally passing through the nipple in males; (3) the *anterior axillary line*, drawn from the anterior fold of the axilla; (4) the *midaxillary line*, drawn from the centre of the axilla; (5) the *posterior axillary line*, drawn from the posterior fold of the axilla; (6) the *scapular line*, drawn through the angle of the scapula when the arm is at rest at the side of the patient. (See Figs. 130, 131, 132.)

The Ribs and Interspaces. These are used as transverse lines. The *first rib* corresponds to the clavicle; the first interspace is the region between the clavicle, or first rib, and the second rib; the number of an interspace corresponds to the number of the rib above it. The following,

FIG. 130.

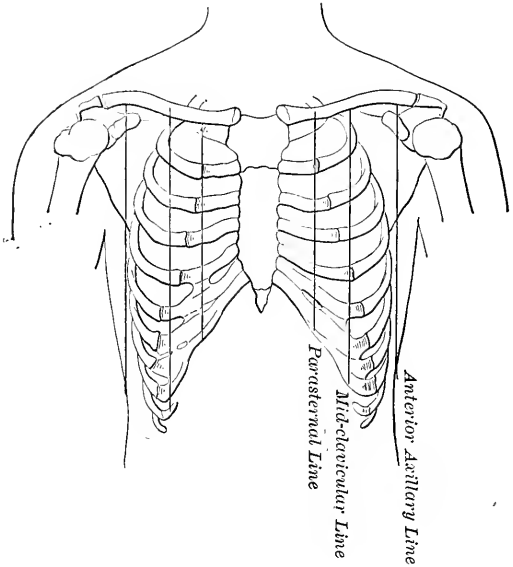


FIG. 131.

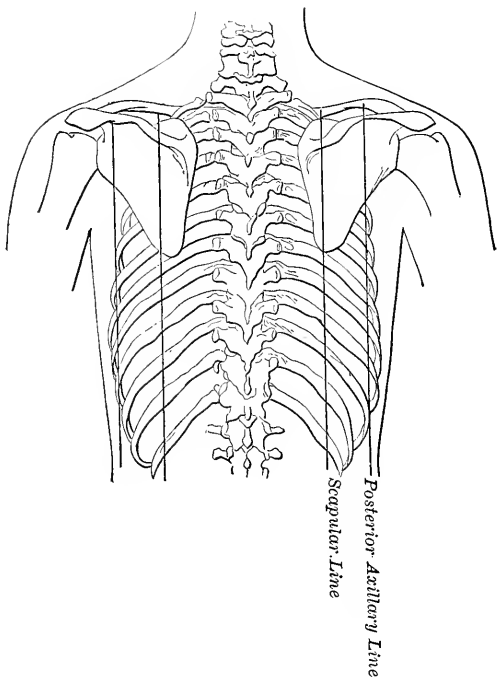
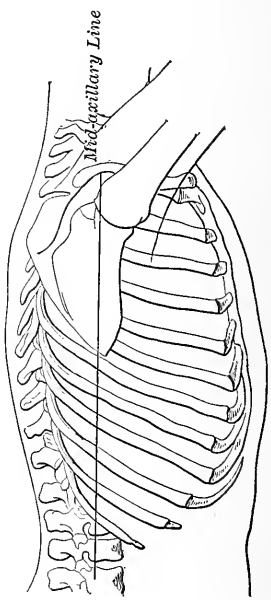


FIG. 132.



Imaginary lines for purposes of localization and recording.

from Holden, is important to remember, particularly when *counting the ribs of fat persons.*

a. The finger passed down from the top of the sternum soon comes to a transverse projection, slight, but always to be felt, at the junction of the first with the second bone of the sternum. This corresponds with the middle of the cartilage of the second rib.

b. The nipple of the male is placed in the great majority of cases between the fourth and fifth ribs, about three-quarters of an inch external to their cartilages.

c. The lower external border of the pectoralis major corresponds with the direction of the fifth rib.

d. A line drawn horizontally from the nipple round the chest cuts the sixth intercostal space midway between the sternum and the spine. This is a useful rule for localization in tapping the chest.

e. When the arm is raised, the highest visible digitation of the serratus magnus corresponds with the sixth rib. The digitations below this correspond respectively with the seventh and eighth ribs.

f. The scapula lies on the ribs from the second to the seventh, inclusive.

g. The eleventh and twelfth ribs can be felt, even in corpulent persons, outside the erector spinæ, sloping downward.

h. One should remember the fact that the sternal end of each rib is on a lower level than its corresponding vertebra. For instance, a line drawn horizontally backward from the middle of the third costal cartilage, at its junction with the sternum, to the spine would touch the body, not of the third dorsal vertebra but of the sixth. Again, the end of the sternum would be at about the level of the tenth dorsal vertebra. Much latitude must be allowed here for variations in the length of the sternum, especially in women.

It is important to recognize the *relation of the ribs to the vertebræ.* The first rib articulates with the first dorsal vertebra, which can be located by the position of the prominent spine of the seventh cervical vertebra; even in very fat people this prominence can be recognized. The remaining ribs, except the tenth, eleventh, and twelfth, have facets of articulation on two vertebræ; as the second rib with the first and second thoracic vertebræ. The eleventh and twelfth articulate with the eleventh and twelfth thoracic vertebræ.

THE ANGLES OF THE THORAX. The *costal angle* is the angle the costal cartilage makes with the sternum. It varies during the act of respiration. In inspiration the rib rises as the sternum projects and apparently elongates, the angle becoming more obtuse; in expiration as the sternum falls the rib becomes more slanting and the angle more acute.

The *epigastric angle* is formed by the meeting of the costal margins of both sides at the xiphoid cartilage. On inspiration it is obtuse, increasing as the ribs rise: in expiration it is more acute.

The junction of the first and second portions of the sternum is known as the *angle of Ludwig.* It is opposite the middle of the second rib, and is on a plane with the lower border of the fourth dorsal vertebra.

Other Topographical Landmarks. The *top of the sternum* is on a plane with the lower border of the second dorsal vertebra. The *junction* of the body *of the sternum with the xiphoid cartilage* is on a plane with the lower border of the eighth dorsal vertebra.

The Regions of the Chest. In the anterior portions of the chest the regions are: the *supraclavicular region*, above the clavicle; the *infraclavicular region*, extending from the clavicle to the third rib; the *mammary region*, between the third and the sixth ribs. Laterally, there are two regions, the *axillary* and the *infra-axillary*, separated by the sixth rib. Posteriorly the regions are the *suprascapular*, the *scapular*, the *infrascapular*, and the *interscapular*, the last being the region between the scapula and the spine.

Inspection of the Chest.

Object of Inspection. By inspection of the chest we learn (1) the appearance of the external surface, (2) the shape and size, and (3) the movements. The first indicates the general state of nutrition; the second, the capacity of the lungs; the third, the degree of functional activity. Inspection may also be conducted by means of the fluoroscope. (See *X*-ray Diagnosis.)

Method of Procedure. The examination may be made with the patient lying down, but preferably standing or sitting in an easy position. All the clothing should be removed from the chest. The light should fall directly upon the chest or obliquely across it. The observer should stand first in front, then behind, and then at the side. In viewing the anterior surface of the chest it is often well to stand behind the patient and look downward over the shoulders. The patient's arms should fall by his sides; his breathing should be quiet, and should not be disturbed by talking or by unusual movements.

The Appearance of the External Surface of the Chest. In many diseases variations are observed from the normal appearance of the different structures seen on inspection.

The Skin. *In health:* the skin normally should be supple, elastic, and of the color described in Chapter XXIII. *In disease:* it is pale in anæmia and wasting diseases; yellow in jaundice; and it may be pigmented generally or locally from causes mentioned in Chapter XXIII. It is the particular seat for the parasitic disease tinea versicolor, and for sudamina as well as for other non-specific eruptions.

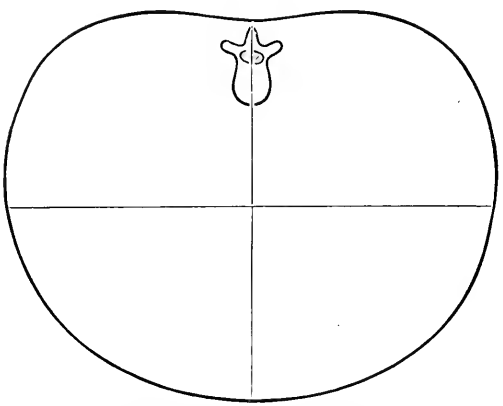
The Veins. *In health:* the veins over the surface of the chest should not be very distinct. *In disease:* they are distinct when the circulation in the mediastinum is interfered with by the pressure of an aneurism or of a morbid growth obstructing the veins. They also, along with the cervical veins, may be enlarged in dilatation of the right heart.

The Capillaries. *In disease:* The capillaries along the base of the chest are often enlarged or more distinct than usual, and arranged in a bow corresponding to the attachment of the diaphragm. The bow is frequently seen in intrathoracic obstruction.

The Subcutaneous Tissue and the Muscles. *In disease:* œdema or subcutaneous emphysema may occur in the conditions mentioned under general inspection. If there is too much fat over the surface of the chest, the muscles may be wanting in tone, thus preventing an estimation of the respiratory capacity.

The Ribs. *In disease:* wasting of the fat and muscles is seen in phthisis, carcinoma, diabetes, muscular atrophy, and paralysis. The degree of softness of the ribs can be diagnosticated in a measure by undue depression of the ribs at the costocartilaginous articulations and at the base of the chest (about the sixth rib) during the act of inspiration. It is an indication of rhachitis. Rigidity of the thorax, equal to the senile fixation, occurs in some adults in middle life, and, as Roberts points out, in young subjects may be due to congenital syphilis.

FIG. 133.



Transverse section of healthy male adult chest. Semicircumference, right side, 16¾ inches; left side, 16½ inches; expansion, 3½ inches. (Ward 6, Philadelphia Hospital.)

The Shape and Size of the Chest. An acquaintance with the normal shape and size is a necessary preliminary to an understanding of the alterations in disease.

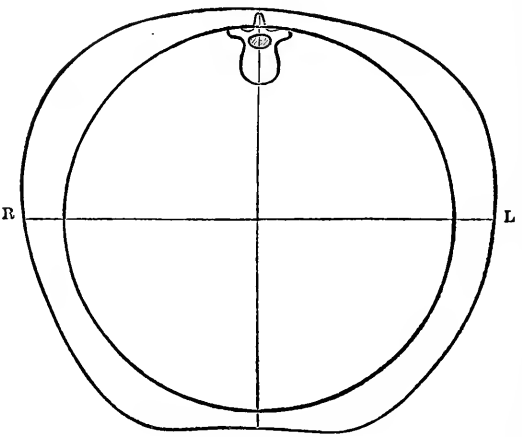
In Health. The shape of the chest is the shape of its transverse section, which depends upon the relations of the anteroposterior and the transverse diameters. It is that of an ellipse, and has been described as reniform. (See Fig. 133.) The anteroposterior diameter is about one-fourth less than the transverse. In children the transverse section is more circular, the anteroposterior and transverse diameters being almost equal. (See Fig. 134.)

In health the chest should be symmetrical, the right side probably a little larger than the left. The muscles of respiration should be well developed, and there should be a moderate amount of subcutaneous fat. The sternum should project forward from above downward, and the portion where the manubrium and the gladiolus join should be a little more prominent than any other part. It is not unusual to see a clearly marked

demarcation between the upper and middle portions of the sternum, or an undue projection of one or more of the upper ribs, or some striking changes about the xiphoid cartilage; none of which are indications of disease. The xiphoid may be depressed, causing a crater-form or funnel-shaped depression. The tip of the cartilage is sometimes retracted, but more frequently thrust forward.

In Disease. The chest may be (1) enlarged or (2) diminished in size. Such change may be (*a*) general or bilateral, (*b*) unilateral, or (*c*) local.

FIG. 134.



Transverse section of an infant's chest, aged nine months, showing the approximately circular outline.

It must not be forgotten that, with the exception of unilateral enlargement, the element of time is necessary to produce changes in the shape and size of the chest. In emphysema the change in shape takes a long time to develop. Unilateral and local contractions are of slow progress, and hence require more or less chronic disease for their development. The occurrence of pleural effusion may cause unilateral enlargement very rapidly.

General or Bilateral Changes in Shape. ENLARGEMENT. In the "barrel-shaped" chest, the type of bilateral enlargement of the chest, all the diameters are increased, particularly the anteroposterior, and the length is diminished. The diameters are almost equal, the transverse section approaching a circle. (See Figs. 135 and 136.) The ribs are elevated and almost horizontal, making the epigastric angle obtuse. The sternum and the spine are arched, the former at the angle of Ludwig. The shoulders are rounded and elevated, and the scapulæ lie flat against the thorax. All the muscles of respiration stand out prominently, particularly the neck and upper trunk muscles. The individual with bilateral enlargement of the chest presents a striking appearance. The neck and arms are short, and there is undue fulness above the clavicles. As this enlargement is attended with dyspnœa, the face is drawn and anxious, and the lips usually faintly livid, or purple.

The Movement of the Chest in Bilateral Enlargement. Expansion may

be lessened to one and one-half inches, or even to half an inch. The respiratory capacity consequently is diminished. The chest is in a state of full inspiration, expiration being three or four times as long as inspiration, and the attendant dyspnœa is known as expiratory dyspnœa. The respirations are hurried, the inspirations short, the expirations prolonged. Both the costal and the diaphragmatic types of breathing are seen in a state of exaggeration. In men the diaphragm acts very vigorously at times.

FIG. 135.

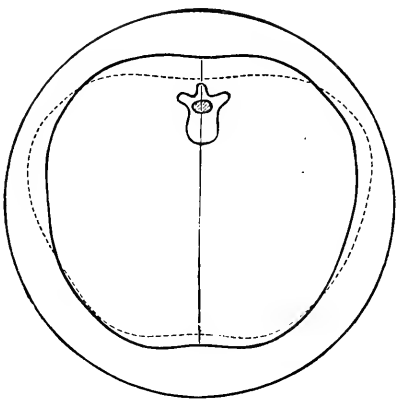
Emphysema with enlargement of the chest: the anteroposterior diameter is much increased. (Ward 6, Philadelphia Hospital.) (Original.)

The Causes of Bilateral Enlargement. The increase in size is due to the enlargement of the normal contents of the chest or to the presence of abnormal contents. In nearly every case it is due to an increased amount of air within the thorax, as in emphysema. In a few instances, in cases of bilateral pleural effusion, enlargement of both sides is seen; but, as considerable effusion would be incompatible with life, the enlargement from this cause is never very great. It is said that such enlargement may occur in rapidly growing cancer of the lungs.

It must be remembered that emphysema can exist without bilateral enlargement of the chest.

Bilateral Diminution in Size. This is represented by two types, the phthisical chest and the rhachitic chest. Certain deformities may also produce bilateral alterations in shape.

Fig. 136.



Bilateral enlargement of emphysema.

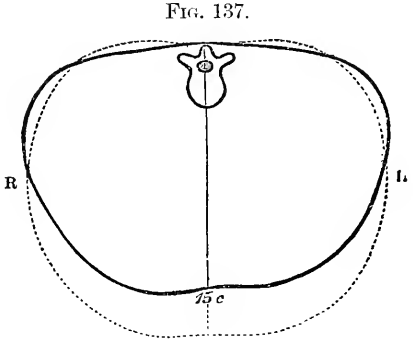
Inner line = emphysematous chest.
Outer line = a circle drawn to show how nearly the emphysematous approaches the circular shape.
Dotted line = normal adult chest.

Actual measurement in centimetres.

Circumference	= natural,	89.0	emphysematous,	87.75
Transverse	= "	29.6	"	27.25
Anteroposterior	= "	22.25	"	25.4 (Gee.)

The phthisical or tuberculous chest is long, the anteroposterior diameter being small (see Fig. 137), and the transverse diameter relatively very much increased. The costal angles are acute and the ribs are

Fig. 137.



The flat or phthisical chest: short anteroposterior, long transverse diameter. (Gee.)

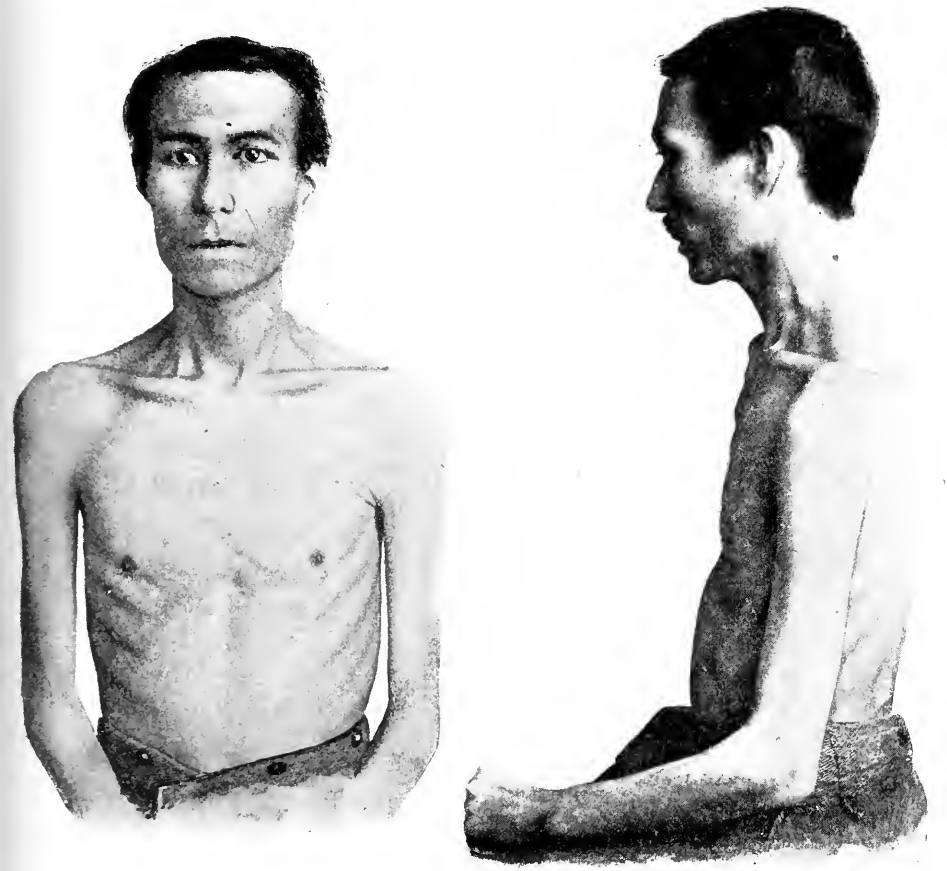
slanting, making the epigastric angle particularly sharp. The shoulders fall; hence the scapulæ are prominent, so markedly so in many cases that the term alar or "winged" chest has been given to this type. The an-

terior plane is often flattened; whence the term "flat" chest is employed. This change occurs through the curve in the cartilage of the true ribs becoming straight.

With this type of chest the neck is long, the larynx (Adam's apple) very prominent, and the arms long; the patient seems loosely put together, the length of the long bones being increased.

It is known as the phthisical, phthisinoid, or tuberculous chest (see Figs. 138 and 139), but this does not necessarily imply that an individual with such a chest has, or will have, tuberculosis. It is true that

FIG. 138. FIG. 139.



The phthisical chest. Full-blooded Indian, Philadelphia Hospital. (Original.)

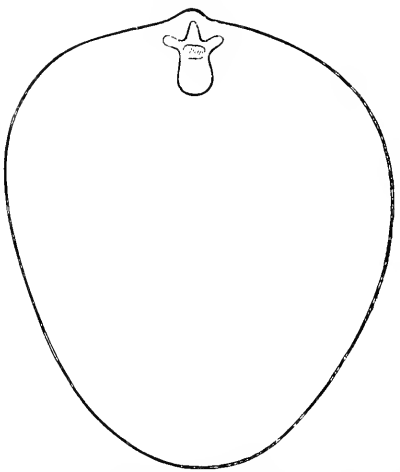
individuals with such a type of chest are more vulnerable to the action of the tubercle bacillus, and are more liable to contract the disease. Nevertheless a very large number of individuals go through life with such chests and die of other diseases. If they are not exposed to the infection, they will certainly escape the disease.

The *movement* or expansion is lessened, and the respiratory capacity is diminished.

The *cause of the phthisical chest* may be diminution of contents, with lessening of the extent of air-surface.

The Rhachitic Chest. Another type of bilateral diminution in size is known as the rhachitic chest (see Fig. 140), which arises in infancy on

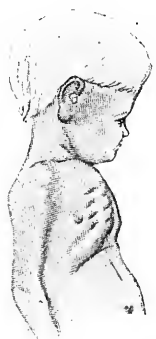
Fig. 140.



Transverse section of a rhachitic chest at the level of the sixth thoracic vertebra. Circumference, 32⅛ inches; right half, 16⅛ inches; expansion, 2 inches.

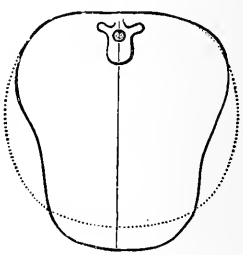
account of rhachitis. Many shapes are seen, to which various names have been given. Among the more common is what is known as the "pigeon-breast." (See Rhachitis, and the Head.) This chest is usually shortened, and the sternum is much more prominent than in health, the lower

Fig. 141.



Chest of rhachitis. (Eichhorst.)

Fig. 142.



Circumference = 42.75 centimetres.

Pigeon-breast of rhachitis. Dotted line indicates the shape of the chest in a normal infant about the same age. (Gee.)

portion projecting to an unusual degree. This is due to a depression of the portion of the chest at the junction of the cartilages with the ribs which tends to throw the sternum farther outward. The transverse section of the anterior portion of such a chest resembles a triangle with

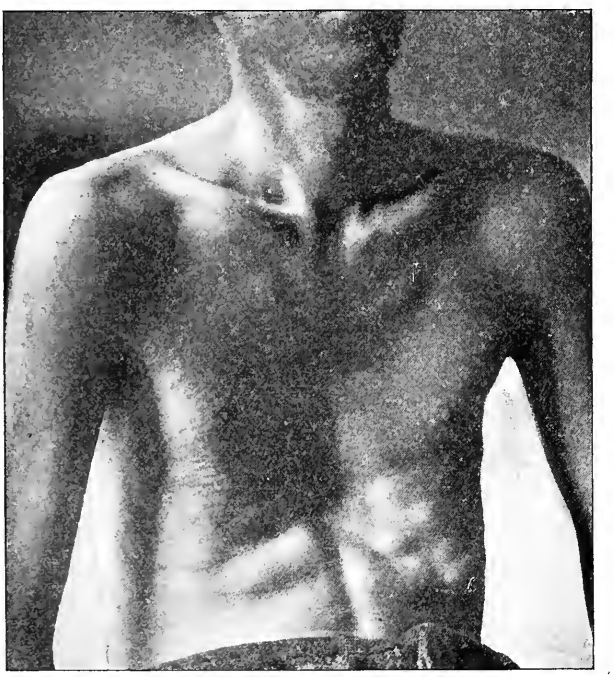
rounded corners. (See Fig. 142.) In some forms of the rhachitic chest the sternum is depressed and the osteocartilaginous articulations are more prominent. In others the ribs and sternum are prominent from above to the fifth rib, and are retracted from the fifth rib downward to the base. In the chest of rhachitis the costal angle is usually very acute. (See Fig. 141.) It often looks as if pressure, as by the hands, had been applied to the sides of the chest about the anterior axillary line, causing the anterolateral portion to sink inward and the anteromedian portion to project forward.

The chest of rhachitis is attended by enlargement of the osteocartilaginous articulations of the ribs called the rhachitic rosary.

The rhachitic chest must not be confounded with chests exhibiting similar changes in shape due to abnormal conditions of the upper respiratory apparatus in early childhood. In cases of adenoid disease of the pharynx (see Diseases of the Pharynx) the change in shape of the chest has been noted.

The rhachitic shape of the chest does not indicate any disease of the lungs, but does indicate deficient respiratory capacity, and is the tell-tale sign by which rhachitis of early life or early laryngeal and nasal obstruction is recognized.

FIG. 143.



Funnel-breast (Trichterbrust). (Original.)

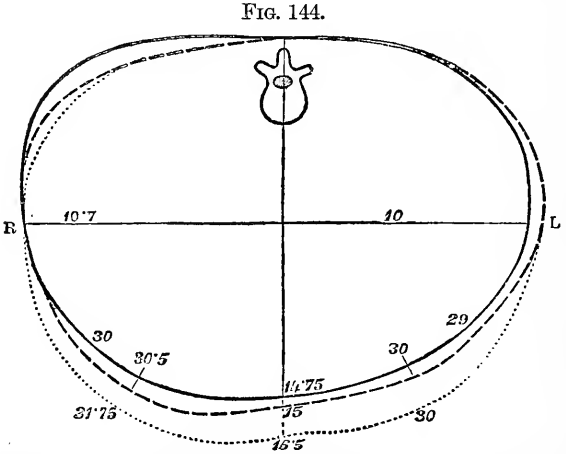
The Transverse or Harrison's Groove. This is a depression or constriction observed in many individuals, especially in those who have had rhachitis. It extends from the xiphoid cartilage, sloping downward along

the base of the thorax toward the axilla. It is caused in early life by the pressure of the external columns of air on the soft bony thorax whose lungs are not completely filled with air. Hence, it indicates the occurrence in early life of nasal, faucial, or bronchial obstruction from adenoid disease, from bronchial catarrh, or from other causes. It may mark the upper limit in infancy of the liver on the right side.

Deformities must not be confounded with the rhachitic chest. Certain deformities of the chest may be congenital in origin and other deformities may be the result of occupation (shoemaking), or of vertebral disease (Pott's disease). The funnel-breast (Trichterbrust) is congenital, and is often seen in several members of a family (Warthin), being associated with other stigmata of degeneration. The lower sternum forms a deep concavity. (See Fig. 143.)

Unilateral Changes in Shape. UNILATERAL ENLARGEMENT. This usually is prominent at the base. The length of the chest is increased, the ribs are elevated, the side is more rounded, and the costal angle is more obtuse. The interspaces are frequently effaced, or fuller than on the corresponding side. The nipple is displaced outward. The scapula of the affected side is also displaced outward; hence the distance from it to the spine is greater than on the opposite side. (See Fig. 144.)

FIG. 144.



Unilateral enlargement of chest (right side) artificially produced by injecting air into the right pleural cavity. Unbroken line: outline before injection. Broken line: outline after moderate distention. Dotted line: outline after extreme distention. Figures at bottom of vertical line indicate the anteroposterior diameter; along horizontal line, transverse semidiameter; remaining figures, right and left semicircumferences. (GEE.)

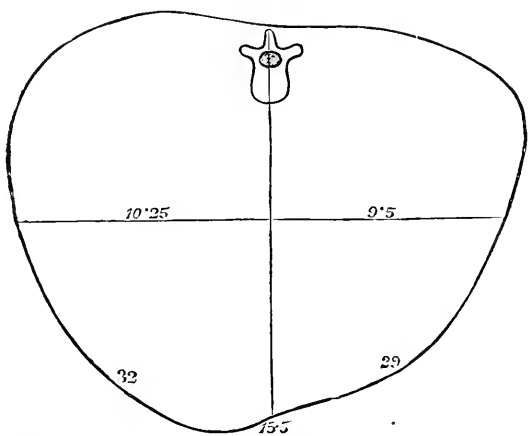
The *movement* may be increased or diminished, depending upon the cause. When the normal contents are increased, the movement is increased; when the pleural cavity is filled, it is diminished.

Cause. Enlargement of one side signifies enlargement of contents. It may be due (1) to an increase of the normal contents as in compensatory emphysema, in which there is an increased amount of air in the lung; or (2) to the presence of abnormal contents as fluid or air in the pleural sac. It is the most characteristic sign of pleural effusion.

Unilateral Contraction or Diminution in Size. The affected side looks flat before and behind, the anterior or the posterior portion, or both being depressed and approaching the transverse median plane of the chest. (See Fig. 145.) The semicircumference is lessened, as is the diameter through the nipple or any other fixed point. The costal angles are sharper. The ribs are closer together, and may almost overlap. The interspaces are lessened in width and may be drawn in.

The *movement* of the side is lessened.

Fig. 145.



Unilateral retraction of chest, consequent upon cirrhosis of the left lung, in a girl of fourteen years. The figures indicate anteroposterior and transverse diameters and semicircumferences of right and left half of the chest. (Gee.)

Cause. Any diminution of contents will cause diminution of the affected side. It may occur from obstruction or compression of the bronchi of that side, lessening the amount of air in that portion of the thorax. Theoretically, it may occur in a case where there is complete occlusion of the main bronchus. This condition is rare, and is accompanied by marked associate emphysema of the other lungs. Unilateral contraction is most frequently seen in cases of chronic pleurisy and of fibroid phthisis. A large portion or even the whole of the lung may be bound down and compressed by thickened adhesions. The pleural cavity of the side thus affected is completely obliterated, save where encroached upon by the heart or by an emphysematous portion of the lung of the same side.

Local Changes in Size and Shape. Enlargement and diminution are also seen in localized areas.

Local Enlargement. That occurring in the region of the heart and great vessels, where it is particularly common, will be considered in the chapter on Diseases of the Heart, the Bloodvessels, and the Mediastinum. A local enlargement in the lower anterior or lateral region of the chest may occur in cases of empyema, in which the pus tends to point externally, or in cases of pulsating pleurisy.

Local Contraction. This may be seen either at the apex or at the

base of the lung. At the apex it is seen above and below the clavicle. The interspaces are sunken and the arc ribs depressed. The term flattening is applied to this condition, which may be more readily seen when looked at from behind. Flattening may also occur either in the lateral or in the posterior region at the base.

Cause. The physical condition causing local contraction is the same as that causing unilateral or general contraction, or diminution in size of the structures within. Anything that lessens the amount of air will cause local diminution in size, or flattening of the surface. This is notably seen in tuberculosis, in which affection three processes, alone or in combination, lessen the amount of air: first, the occlusion of the bronchioles by tubercles and by inflammatory products, causing collapse of the alveoli; second, the overgrowth of connective tissue that attends the more chronic forms of tuberculosis; third, a localized pleurisy. Local pleurisy, with organization and contraction of the inflammatory exudate, may also cause diminution of the amount of air, or diminution of the contents, from compression of the adjacent lung structure.

In local contractions *movement* of the part is generally diminished.

The Movements of the Chest. The frequency, the rhythm, the type, and the character of the respirations, the degree of expansion, and the so-called diaphragm-phenomenon are studied.

The Movements in Health. A complete respiratory act consists of two events, inspiration and expiration, inspiration being active and expiration passive. The latter is a trifle longer than the former, as may be illustrated by the following proportion—Insp. : Exp. : : 5 : 6. A pause follows the act of expiration. In inspiration, as the lung expands with air the chest increases in circumference and in length. The *degree of expansion* varies.

The Frequency and Character of the Movements in Health. These vary in the two sexes. In a healthy male adult the respirations are from 16 to 24 in the minute. In the female they may be 20 to 22. In children the frequency of respiration is much greater. In those under one year it is 44 per minute; at five years it is 26. The respirations are increased in frequency in the standing position, during bodily exertion, with increased temperature of the air, and during digestion. They are lessened in the horizontal position. The hand placed on the epigastrium facilitates counting of the respirations.

Normal Types of Breathing. Three types of normal respiration are recognized.

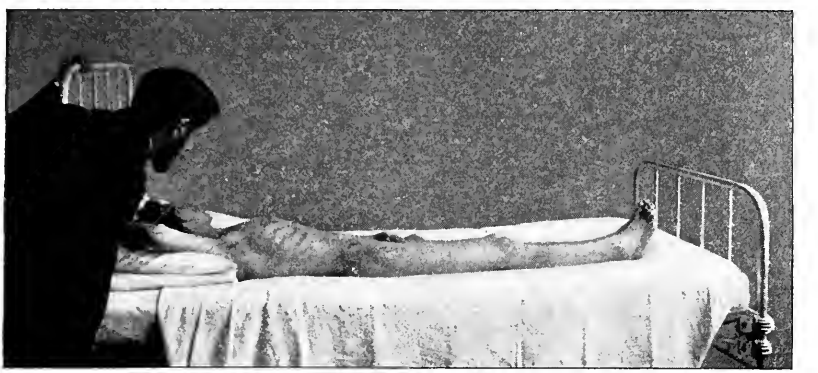
In the *costo-abdominal* or *diaphragmatic type*, seen in male adults during quiet breathing, the movements of the chest are more marked in the lower half of the thorax: the sternum rises; and the ribs are elevated and at the same time drawn forward and outward. The anteroposterior and vertical diameters of the chest increase, the costal and epigastric angles become more obtuse, and the diaphragm acts conjointly with the external muscles of the thorax, the epigastric region swelling with each inspiratory effort as the muscle descends. In expiration the sternum falls, the ribs become more slanting instead of horizontal, the epigastrium retracts, the angles become acute, and the anteroposterior and transverse diameters lessen.

The *costal* or *upper thoracic type of breathing* is seen in women, in whom the upper half of the chest moves more actively. The areas below the clavicles and the upper portion of the sternum swell more distinctly during inspiration, but the movements of the lower portion, and especially of the diaphragm, are limited.

The *costal type* occurs most frequently in children. It is the type of breathing in both sexes during sleep; and is observed during deep respiration.

LITTEN'S DIAPHRAGM-PHENOMENON. The diaphragm and walls of the thorax approach each other during expiration, coming in apposition at the end of this act, and become separated during inspiration. In persons whose chest-walls are not too thick the movements of the diaphragm are indicated on the surface of the chest by the rise and fall of a shadowy line. The patient must lie on his back with his face turned away from the light and with the head slightly elevated. The light should fall from the head or foot of the bed. The observer stands a distance of three or

FIG. 146.



Litten's diaphragm-phenomenon.

four feet with his back to the light, scanning the chest at an angle of about 45 degrees. In the act of inspiration a horizontal shadow or undulation is seen to start on either side about the sixth interspace and to pass during inspiration downward over a distance of two or more interspaces, even to the margin of the ribs. In expiration the shadow begins below and moves upward to the starting-point. By this phenomenon the volume or vital capacity of the lungs can be estimated. In normal individuals the shadow should move more than $2\frac{1}{2}$ inches.

The Movements of the Chest in Disease. In disease, as in health, the type, character, rate, and rhythm of the respirations, the degree of expansion, and Litten's phenomenon must be observed.

THE TYPES OF RESPIRATION IN DISEASE. Any of the normal types of respiration may predominate abnormally or may be abnormally increased.

Abnormal costal breathing may be caused by œdema or inflammation of the lower lobes of the lungs; double pleurisy with effusion; diseases of

the bony thorax; painful affections of the soft parts; paralysis of the diaphragm; pressure on the diaphragm from ascites, tympanites, or from abdominal tumors or enlargements; diseases below the diaphragm, such as peritonitis; emphysema; and hysteria.

Abnormal abdominal breathing may occur in pleurisy, pleurodynia, fracture of a rib, calcification of the costal cartilages, scleroderma of the chest-wall, ossifying myositis, and paralysis or spasm of the muscles of inspiration.

The Respiratory Rate in Disease. The rate of the movements is *increased* in nearly all forms of dyspnœa. (See Dyspnœa.) The frequency of movement varies in many affections. It is more markedly increased in the acute lung affections attended by fever, especially in children. Increased frequency of respiration does not necessarily indicate pulmonary disease. It is always seen in fever, and is a marked phenomenon of hysteria. It may be caused by conditions outside of the chest encroaching upon its capacity, such as enlargement of the abdomen from any cause. The respirations are *lessened* in frequency in cases of disease of the medulla in which there is pressure upon the respiratory centre, and in some forms of poisoning, such as that due to opium.

Alterations in the Character and the Rhythm of the Movements. The ratio in health of the act of inspiration to that of expiration is 5 : 6; in women, in children, and in the aged, it is 6 : 8. The expiration is the longer and may be prolonged, so that it is far greater in length than inspiration. The normal ratio may be disturbed by an increase in the length of either inspiration or expiration.

Inspiration increased. The duration of inspiration is increased when there is an obstruction in the trachea or larynx. Such increased expansion of the upper chest is usually associated with retraction of the soft parts of the thorax, especially at the base. The ribs and the tissues along the margins of the thorax are drawn in with each inspiration. The space above the clavicle occupied by the lung may also be retracted. The transverse groove is more pronounced. If the difficulty in breathing continues, the indrawing becomes very marked, and, if the ribs are soft, may become permanent. The character of the breathing in such a case is usually stridulous. (See below.)

Expiration prolonged. Inspiration is short and quick in cases of emphysema. The expiration is correspondingly prolonged, and the muscles of expiration are seen to be brought into full action.

In the consideration of dyspnœa the appearance and posture of the patient and the action of the muscles of respiration have been described. (See Subjective Symptoms.)

In *Cheyne-Stokes breathing* the acts of respiration are carried on by an alternation of pauses and of periods of modified or "tidal" breathing. A respiratory pause of half to three-quarters of a minute's duration alternates with a short period of increased activity, during which time twenty to thirty respirations occur. The respirations constituting this series are shallow at first, but gradually become deeper and more dyspnœic, and then gradually become shallow or superficial again. Sometimes consciousness is abolished during the pause, and often the pupils are contracted and

inactive. When the respirations begin, the pupils dilate. This type of breathing usually indicates grave cardiac, renal, or cerebral disease.

Jerking or *catchy respiration* may occur in pleurodynia, fractured rib, the early stage of acute pleurisy, and sometimes in asthma, hysteria, and hydrophobia.

Stertorous breathing, snoring, is seen often in the unconsciousness of apoplectic, uræmic, or diabetic coma and of narcotic poisoning; also in paralysis of the palate and in cases of enlarged faucial or pharyngeal tonsils.

Stridulous breathing is a sign of obstruction to the entrance of air at or near the glottis. It occurs in laryngeal or tracheal obstruction by foreign bodies or tumors or by pressure from without, and in spasm, œdema, or paralysis of the glottis.

ALTERATIONS IN THE DEGREE OF RESPIRATORY EXPRESSION. These may be (1) bilateral, (2) unilateral, or (3) local.

1. *Bilateral Changes in Movements. Increased general expansion* occurs normally after exercise or mental excitement, and is often seen in hysteria and in some forms of dyspnœa.

Diminished general expansion may be a part of a condition of general muscular weakness, or it may be due to paralysis or spasm of the respiratory muscles, to obstruction of the upper air-passages, or to emphysema, or it may be caused by an effort to limit the pain of pleurisy, pleurodynia, or fractured rib.

2. *Unilateral Changes in Movements. Increased unilateral movement* is seen when the lung of one side is acting vigorously to compensate for the other lung which is disabled by disease. The whole side moves more rapidly and vigorously. The increased movement is associated with enlargement of the affected side and hyper-resonance on percussion.

Unilateral diminution in movement occurs from diminution of the respiratory surface, from occlusion of the bronchial tubes, or from causes outside of the lung. The air-space is lessened in cases of pneumonia, tuberculosis, or any affection which fills bronchioles and alveoli with inflammatory exudation or fluid. It is diminished particularly by the compression of effusions in the pleura, of contracted and thickened exudations, and of adhesions. The various conditions giving rise to unilateral deficient expansion must be differentiated.

Pleural Effusion. Impaired motion due to this condition is almost always unilateral, it develops gradually after an attack of acute pleurisy, it is unattended by pain on respiration, but it is attended frequently by great embarrassment of the respiration and sometimes by orthopnœa. Fever is usually moderate in uncomplicated cases. These clinical signs and the physical signs of fluid in the pleura will leave no doubt as to the diagnosis.

Chronic Pleurisy. Impaired motion from this cause is of long standing and of gradual development. The chest-wall upon the affected side is retracted and may be very markedly sunken. In the absence of accompanying lung trouble there is no pain and no fever. The diagnostic signs of this type are: the sinking in of the affected side, in sharp contrast to the hypertrophy of the other side; the absence of fever and pain; the

chronicity of the affection; and the physical signs of thickened pleura and of compressed lung.

Pneumothorax. Impaired motion from pneumothorax develops suddenly, as a rule in a person with tuberculosis of the lungs. Its sudden development is marked by coughing, intense pain, distention of the affected side, great difficulty in breathing, and a very anxious expression of countenance. The escape of air into the pleural cavity is followed by the development of pleurisy with effusion, so that the affection presents the physical signs of air and fluid in the pleural cavity.

Pneumonia. The motion of the affected side is greatly impaired in this disease when a large portion or the whole of one lung is involved, the air-vesicles being so occluded that very little air can get in. The diagnosis can be made by noting the acute onset of the disease with high temperature and frequent respiration but without antecedent pleurisy, the presence of cough with expectoration containing the pneumococcus, and the physical signs.

Occlusion of a Bronchus. Diminution of the movement of the corresponding side is seen in the rare cases in which a foreign body fills the lumen of the tube, or in the more common cases of pressure externally upon the bronchus by an aneurism or mediastinal tumor.

Pressure on a Bronchus. An aneurism or enlarged lymph-gland pressing on a bronchus produces the physical signs of collapse of the lung, coupled with those peculiar to the cause in each particular case. The condition develops gradually, the patient having no pain in the lung.

Rheumatism of the Intercostal or Respiratory Muscles. This may cause unilateral lessened movement by interfering with the muscular activity of that side. It is to be distinguished by the presence of tender muscles and of a more constant and less stabbing pain, and by the absence of fever, cough, and râles.

Acute Pleurisy. In this affection the patient checks the motion of the affected side as much as possible, and breathes with the abdominal muscles, because chest respiration causes acute pain. The pain comes on suddenly and is usually depicted in the face. The absence of local tenderness and the presence of fine, dry, or coarse râles on inspiration, and of cough and fever complete the diagnosis.

Pain in the Ribs. This may be due to fracture, inflammation, or tumor.

3. *Local Diminution of the Respiratory Movement or Deficient Expansion.* This is the only local alteration in the degree of respiratory expansion. It occurs under the same conditions that produce flattened and local contraction and for the same reason; hence deficient expansion is observed in the early stages of phthisis and in local pleurisies.

Impaired motion due to tuberculous consolidation of the lung is usually limited to one of the apices, and is accompanied by flattening of the affected apex and by emaciation. The condition is of gradual development, and presents the usual signs of tuberculous consolidation of the lungs (*q. v.*).

Sometimes the impaired motion and flattening are due to a superficial cavity from tuberculosis or from an abscess. When the walls of

the cavity are very thin, they may be seen to flap feebly with respiration.

Rarer causes of local impaired motion of the lung are cancer and hydatid cyst (*q. v.*).

The Change in Litten's Phenomenon. Absence of the phenomenon is noted when the pleural cavity contains fluid or air, and when it is obliterated by adhesions; in pneumonia of the lower lobe; in emphysema of the lungs; and in intrathoracic tumors low down in the chest. Tumors or fluid accumulations below the diaphragm do not lessen the phenomenon. With lessening of the extent of movement the respiratory capacity is diminished and tuberculosis may be suspected. Limitation of the excursion of the diaphragm—*x*-ray investigations have forcibly taught us—is one of the earliest signs of tuberculosis. This limited excursion can be detected in proper subjects by Litten's method, although it must be remembered that general debility and emphysema lessen the excursion on both sides. In splenic and hepatic enlargements the normal shadow persists, but in a large collection of ascitic fluid it may be detected with difficulty or it may be absent.

Thoracometry, Cyrtometry, Spirometry, Pneumatometry, and Stethography.

The results obtained by inspection can be confirmed by certain instruments of precision.

Thoracometry or Mensuration. By this means the size and the degree of expansion of the chest are ascertained and the circumference and diameter of the chest are determined.

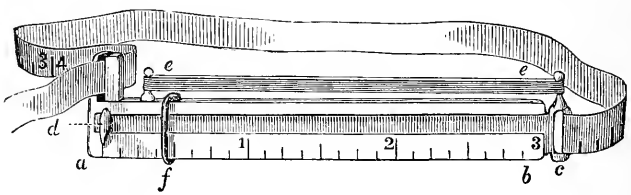
The *diameter* of the thorax is measured by means of caliper compasses. The anteroposterior diameter should be taken on a level with the nipple and without the insertion of the seventh rib behind; the transverse diameter, at the highest points of the axillæ. The length of the chest may be ascertained by measuring in the midclavicular line from the clavicle to the border of the ribs. It is important to remember that in right-handed people the right side of the chest measures a little more than the left.

The *respiratory capacity* is estimated by measuring the circumference of the chest. It is secured by taking the measurement at the end of complete expiration and again at the end of complete inspiration. In taking the measurement the observer must be particular to keep the terminal portion of a tape-measure fixed in the median line; the other portion is to be held in the hand, so as to move with inspiration and expiration. The anterior mesial line should always be marked in advance and when measurements are taken daily the exact level at which they are made should be noted. In health the difference between the two measurements should be from 5 to 10 centimetres (2 to 4 inches). If the expansion is less than 2 inches, it is considered deficient by insurance companies, who then do not regard the risk as first class. The expansion is less in women. Deficiency of chest expansion not only indicates the presence of a local morbid process (notably incipient tuber-

culosis), but it also indicates lack of strength and of muscular development, physiological rather than physical deficiencies, and is an unerring guide to the need of respiratory gymnastics.

Carroll's stethometer consists of a slide (*c–d*) bearing an indicator (*d*) and attached to one end of an ordinary measuring-tape, and moving within a case (*a–b*) marked with a scale and provided with a catch into which the other end of the tape is fastened after it has been passed around the chest. (Fig. 147.)

FIG. 147.



Carroll's stethometer.

The following measurements, secured by laborious investigation, are excellent criteria from which pathological inductions can be made.

MEASUREMENTS OF THE CHEST AND LUNG CAPACITY.

(OTIS, *Boston Medical and Surgical Journal*, 1895.)

TABLE I.—*Chest Measurements.*

	Repose, inches.	*Inflated, inches.*	*Difference, inches.*
Girth, muscular—Men:			
Average of Dr. E. O. Otis, 1000 measurements, between sixteen and forty years of age	34.0	36.1	2.1
Average of Dr. Hitchcock, of Amherst College, 8000 measurements	34.6	36.5	1.9
Average of E. Hitchcock, Jr., of Cornell College, 15,000 measurements	34.5	36.3	1.8
Girth, muscular—Women:			
Mt. Holyoke and Wellesley students. Measurements by Miss Wood and Dr. Mary Colton	29.5	31.5	3.0
Chest, respiratory—Men:			
Average of Dr. E. O. Otis, 1000 measurements	31.1	33.1	2.0
Chest, respiratory—Women:			
50 per cent. of 1500 of Wellesley students, Miss Wood	24.6	27.2	2.6
Depth of chest—Men:			
Average of Dr. E. O. Otis, 1250 measurements in repose and 362 inflated	7.5	8.3	0.8
Depth of chest—Women:			
50 per cent. of 1500 students at Wellesley, Miss Wood	6.9	. .	. .
Breadth of chest—Men:			
Average of Dr. E. O. Otis, 400 measurements	9.9	10.8	0.9

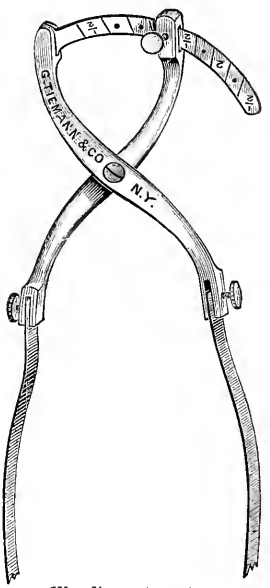
TABLE II.—*Capacity of Lungs.*

	Cubic inches.
Men:	
Average of Dr. E. O. Otis, 1000 measurements	240.6
Hitchcock, 800 measurements	230.0
Hitchcock, Jr., 15,000 measurements	236.6
Women:	
Mt. Holyoke and Wellesley students, measurements by Miss Wood and Dr. Mary Colton	145.8
50 per cent. of 1500 Wellesley students, Miss Wood	150.3

Cyrtometry. This is the determination of the shape of the chest. The outline of a transverse section of the chest is taken at any desired level and may then be traced on paper.

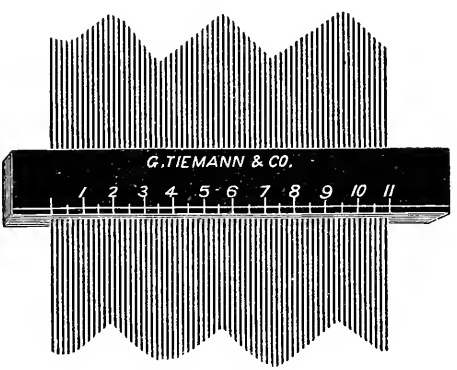
By this means differences in the shape and in the movements of the two sides are made manifest. If the measurement is taken from day to day and graphic records are made, delicate changes can be definitely ascertained. The circumference of the chest is measured by means of the cyrtometer; Woillez's cyrtometer is a chain with links; Weed's consists of two soft metal tapes or strips joined by a hinge. The tapes are made to fit the circumference of the chest accurately, the middle of the hinge being held firmly over the spinous process of the vertebra, while the two limbs are carried around the chest, moulded to all inequalities, and crossed in front, one above the other. A mark is then made on each where it crosses the middle line. Measurements should be taken at about the level of the nipples, and 2 inches below them, care being taken to have the level the same in front and behind. They should be taken in full inspiration, in full expiration, and in repose. The outline secured by this method need not be disturbed, as by flexion on the hinges we are enabled to remove it intact. The tapes are carefully transferred to a sheet of paper, on which imaginary diameters have been marked. After fixing the corresponding points of the tapes on the lines of the respective diameters the outline can be traced.

FIG. 148.



Weed's cyrtometer.

FIG. 149.



Warren's conformator.

The shape of the parts of the chest may be estimated by means of the *conformator*. Warren's conformator consists of two hard-rubber or metal braces which have been grooved to receive a number of blunt-pointed

needles. These needles move up and down easily and when applied to an undulating surface will conform to that surface, preserving the exact shape of the depressions and elevations. (Fig. 149.)

Spirometry. The object of spirometry is to ascertain the respiratory or vital capacity of the lung, the quantity of air taken in with each inspiration and discharged with each expiration. The estimation is made by means of the spirometer. Hutchinson's spirometer is constructed on the gasometer principle; Barnes' is much simpler. The data ascertained

FIG. 150.

Barnes' spirometer.

are not of much diagnostic significance. If measurements are made from day to day in a lung which was incapacitated, we may be able to estimate the extent of recovery from disease. When there is an important diminution of lung-capacity, tuberculosis may be suspected even before subjective and objective signs warrant a diagnosis. We can estimate the degree of interference with breathing caused by disease below the diaphragm. Spirometry is of particular value because it shows in a graphic manner the need for respiratory gymnastics.

TABLE III.—*Comparison of the "vital" or lung capacity and the amount of air expelled after an ordinary quiet respiration.*

(Average of E. O. Otis, 150 measurements.)

	Cubic inches.
Vital capacity or the amount of air exhaled after a full inspiration	230.5
Amount of air exhaled after an ordinary quiet respiration	129.3
Difference of "complemental" or "reserve" air	101.2
Difference as given by Hermann	97.6

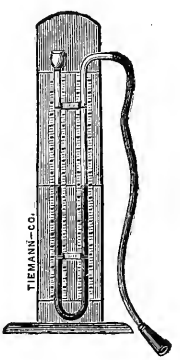
Average Lung Capacity for Height (Otis).

Height.	*Lung Capacity.*	*Average for each inch or centimetre in height.*
66 to 67 inches inclusive.	231.62 cubic inches.	3.4 + cubic inches.
167.7 to 170.3 centimetres.	3797 cubic centimetres.	22.4 cubic centimetres.
67 to 68 inches inclusive.	237.10 cubic inches.	3.46 cubic inches.
170.3 to 172.8 centimetres.	3903 cubic centimetres.	22.7 cubic centimetres.
68 to 69 inches inclusive.	244.44 cubic inches.	3.5 cubic inches.
172.8 to 175.4 centimetres.	4007 cubic centimetres.	23.06 cubic centimetres.
69 to 70 inches inclusive.	259.34 cubic inches.	3.66 cubic inches.
175.4 to 177.9 centimetres.	4250 cubic centimetres.	24.06 cubic centimetres.
70 to 71 inches inclusive.	261.38 cubic inches.	3.64 cubic inches.
177.9 to 180.5 centimetres.	4284 cubic centimetres.	23.9 cubic centimetres.
71 to 72 inches inclusive.	261.34 cubic inches.	3.5 cubic inches.
180.5 to 183 centimetres.	4284 cubic centimetres.	23.03 cubic centimetres.
General average . . .	3.25 cubic inches, for each inch of height.	
	23.19 cubic centimetres, for each centimetre of height.	

Powel lays great stress upon the fact that in phthisis the inspiratory capacity is diminished, while the expiratory power remains normal.

Pneumatometry. This consists in measuring the force in respiration. By means of a Waldenburg's pneumotometer the pressure of the respiratory air on inspiration and expiration is determined. Expiratory pressure is diminished in emphysema, and the degree of diminution may furnish a clue to the severity of the disease or to the degree of the improvement. It is to be remembered that, according to Waldenburg, the expiratory pressure in health always exceeds the inspiratory pressure by as much as 20 to 30 millimetres. Inspiratory pressure is lessened in stenosis of the air-passages, in phthisis and in pleural effusions, but is not of diagnostic significance.

Fig. 151.



Waldenburg's pneumotometer.

Stethography. This is the graphic recording of the movements of the chest and diaphragm during respiration. Stethography is of service only when it is desired to make a record of the respiratory movements.

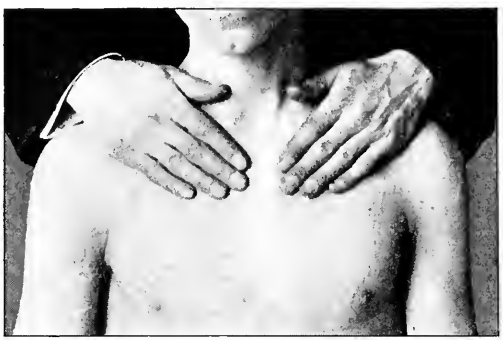
Palpation of the Chest.

Palpation in diseases of the lungs and pleura is employed: (1) to confirm the results obtained by inspection, mensuration, and cyrtometry as to the size, form, and movements of the chest; (2) to elicit tenderness; (3) to determine the resistance of the chest-wall and of tumors; (4) to distinguish the vibrations produced by the voice (vocal fremitus), by bronchial rhonchi (rhonchal fremitus), and by pleural friction (friction-fremitus); and (5) to detect the fluctuation caused by fluid and the succussion due to the presence of both fluid and air.

Method. The surface should be bared, although the fremitus can be detected through a thin layer of linen or gauze. To estimate the degree of expansion and to detect the fremitus in front, it is often well to stand

behind the patient with the palms of the hands placed over the surface of the chest in front. The opposite position is taken to detect the fremitus behind. The axillary region must also be investigated. The

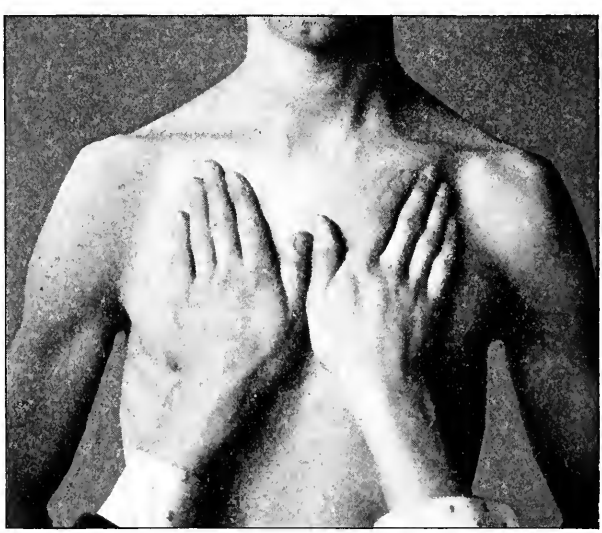
FIG. 152.



Noting expansion and vocal fremitus.

hands should be warmed and applied evenly to the surface without much pressure. The two sides must constantly be compared, either by placing the hands simultaneously on the two sides or by applying the hand first to one side and then to the other.

FIG. 153.

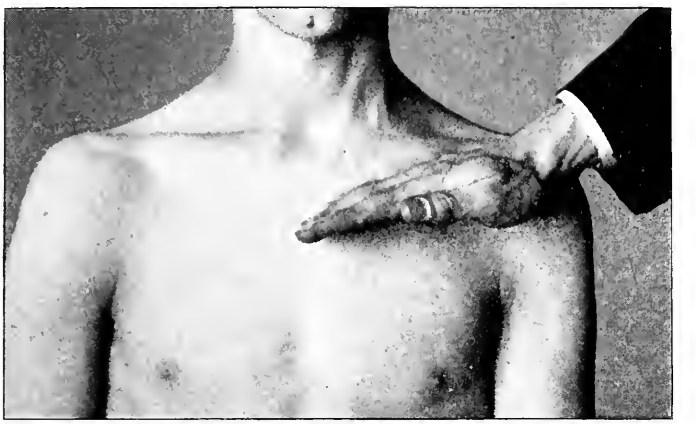


Vocal fremitus.

Tenderness or Pain upon Pressure. This is sometimes present in *pleurisy*, in *croupous pneumonia*, and in *phthisis*. It may be due to *intercostal neuralgia*, to *intercostal myalgia*, to *herpes zoster*, to *erythema nodosum*, to *hysteria*, to *disease* or *fracture of the bones*, and to *injury*, *inflammation*, or *abscess of the soft parts*.

Vocal Fremitus. During the act of speaking the columns of air in the bronchial tubes are thrown into vibration. The vibrations are transmitted to the hand placed on the surface of the chest, constituting

FIG. 154.

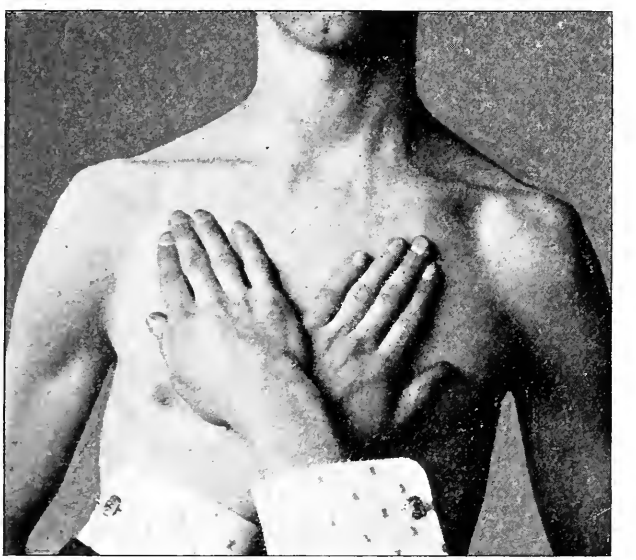


Testing the vocal fremitus with the edge of the hand.

the vocal fremitus. In infants the cry is used instead of the spoken voice.

It is necessary to be familiar with the vibrations produced by fixed

FIG. 155.



Vocal fremitus.

monotones, in order to appreciate the fremitus. The patient is asked to count 1, 2, 3, or to repeat 99 three or four times. It is well to observe

a fixed rule as to the words used, in order to have definitely in the mind the character of the vibrations in health and the departures from the normal in disease.

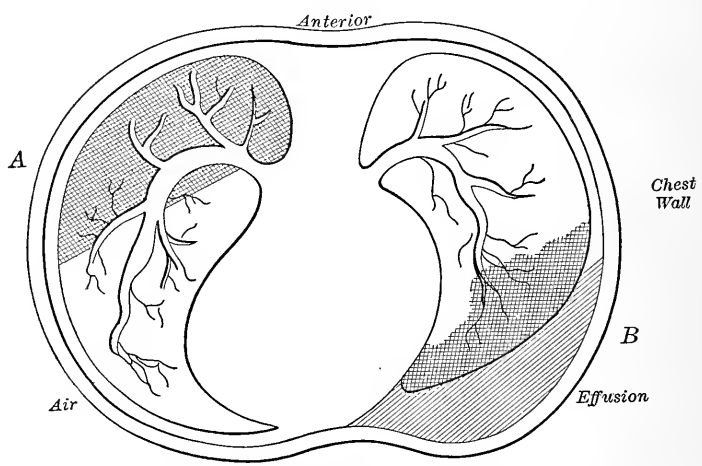
Vocal Fremitus in Health. The fremitus is *stronger* on the right side than on the left because the right bronchus is larger than the left, makes a more acute angle with the trachea, and is $2\frac{1}{2}$ inches nearer the larynx than is the left. (Cary, Ewart.) It is stronger in persons with deep, low-pitched voices, the vibrations not being so rapid, and is more distinct therefore in males than in females. It is felt more distinctly in persons with thin chest-walls.

Fremitus is *weaker* in people with thick chest-walls and large mammary glands which interfere with the transmission of fremitus. The fremitus is not distinct in children because the vibrations are too rapid.

Vocal Fremitus in Disease. The vocal fremitus may be increased; it may be diminished; or it may be absent.

Increased Vocal Fremitus. Vibrations are transmitted to the hand with greater force by a consolidated lung than by one containing air. Fremitus is increased consequently in pneumonia, tuberculosis,

Fig. 156.



Transverse section of thorax: *A*, consolidation—pneumonia, vocal fremitus increased; *B*, pleural effusion, vocal fremitus absent. (Original.)

hemorrhagic infarct, and tumor. (See Fig. 156.) The fremitus, however, may be absent in the rare cases of pneumonia in which the large tubes are occluded by exudate. Fremitus is increased when the lung is compressed above a pleuritic exudate. It is also increased over a cavity, which acts as a resonator.

Diminished Vocal Fremitus. Anything intervening between the lung and the surface of the chest and interfering with the conduction of the vibrations diminishes the fremitus. The fremitus therefore is diminished over a thickened pleura and over a thin layer of pleural effusion. The fremitus is also lessened when on account of diminution in

the calibre of the bronchi the columns of air in them are smaller, as in bronchitis, in emphysema, and in asthma. The fremitus is lessened over cavities filled with fluid and over a lung whose bronchus is occluded.

Absent Vocal Fremitus. The vocal fremitus is absent when the columns of air are obstructed entirely by occlusion of the bronchus, as by the external pressure of a tumor, aneurism, or enlarged gland; and when the pleura contains air or fluid, causing interference with the vibrations (see Fig. 156), as in pneumothorax, hydrothorax, pyothorax, and hæmothorax. When the pleura is greatly thickened, the fremitus is absent.

Rhonchal Fremitus. The vibrations produced by the passage of air through mucus or fluid in the bronchial tubes may be transmitted to the hand when it is laid on the surface of the chest, and constitute the *rhonchal fremitus.* They are felt during inspiration in bronchitis and asthma. They may be felt all over the chest as distinct vibrations, coarse if generated in the large tubes, fine if in the small tubes. In phthisis, air passing through fluid in a cavity may produce vibrations over a localized area. In children with bronchitis rhonchi are distinct, and are often the source of much alarm to parents.

Friction Fremitus. A vibration which is transmitted to the hand is often caused by an exudation of lymph on the surface of the pleura. It is known as a friction fremitus, and is felt in inspiration usually at the base of the chest. It is not modified by coughing, but it is increased by full breathing. Rhonchal fremitus, on the other hand, is influenced by cough and by breathing.

Fluctuation. This may be detected by palpation in some cases of effusion, particularly if the intercostal spaces are swollen and tense, and in the case of an empyema about to point. In rare instances it may be detected by striking with the other hand the side of the chest opposite the palpating hand.

Splashing or Succussion. When both air and fluid are present in the pleural cavity, or in a large pulmonary cavity situated close to the thoracic wall, on shaking the chest the motion of the liquid may be felt by the palpating hand. Agitation of the liquid by violent coughing may also give rise to the succussive phenomenon.

Percussion in Diseases of the Lungs and Pleuræ.

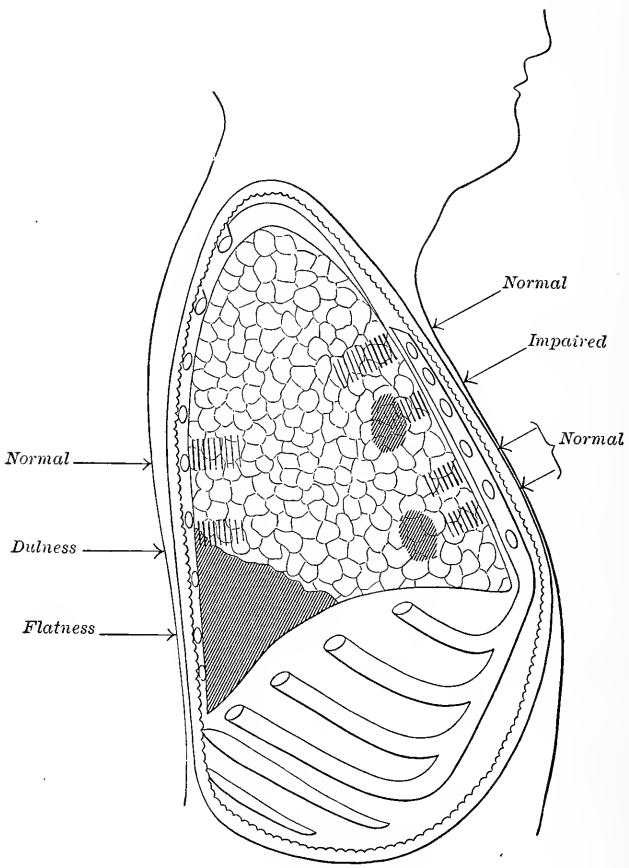
Percussion is employed in disease (1) to elicit abnormal sounds and (2) to define abnormal boundaries.

Abnormal Percussion-sounds. The percussion-sound elicited in disease may be (*a*) *duller* than normal, (*b*) *higher pitched* than normal, (*c*) *clearer* than normal, or (*d*) of a *different character* from the normal sound. To the first group belong *diminished* or *impaired resonance, dulness,* and *flatness;* to the second, *hyper-resonance* and *tympany;* and to the third, *amphoric resonance,* the *cracked-pot sound, Williams' tracheal tone,* the *bell* or *anvil sound, Wintrich's phenomenon, interrupted Wintrich's phenomenon, Friedreich's phenomenon, Gerhardt's phenomenon,* and *Biermer's phenomenon.*

It may be said in general that when a percussion-sound is produced in the thorax which varies from the normal resonant tone, it indicates an abnormal physical condition. In percussing the chest, however, exactly corresponding portions of the two sides must be compared.

Change in tone may be general or local: (1) the areas over both lungs may yield a different percussion-note from the normal (*bilateral*); (2) the change may be limited to one side (*unilateral*); or (3) it may be found in small areas (*local*).

FIG. 157.



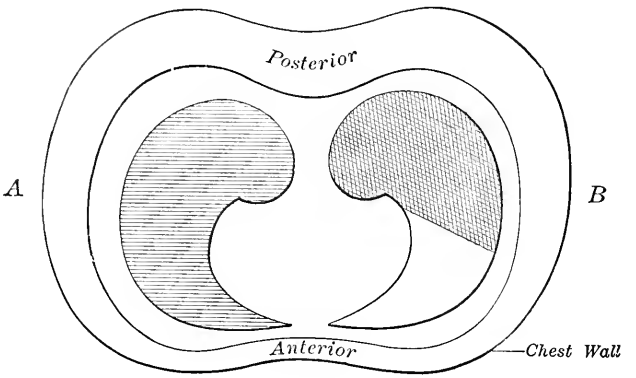
Changes in percussion-note. (LE FEVRE.)

Diminished or Impaired Resonance. The normal tone or resonance is impaired or muffled in cases of beginning consolidation of the lung, in slight thickening of the pleura, and in small pleural effusions which overlap the lung as a thin layer. It is the first change toward dulness. It is particularly noted in the early stages of phthisis, when the lung usually is the seat of small areas of tuberculous infiltration at the apex.

Dulness. The dull note signifies a relatively small amount of air in proportion to solid structure, the extent and the degree of dulness de-

pending upon the proportion of solid to air-containing material. Moderate dulness is seen in tubercular disease, with moderate infiltration of the lung (see Fig. 158), and in small patches of catarrhal pneumonia, in pulmonary congestion and œdema, in phthisis, in condensation of the lung

FIG. 158.



Transverse section of thorax: *A*, moderate dulness over tuberculous infiltration; *B*, heightening of pitch or Skodaic resonance anteriorly from consolidation (shaded portion) posteriorly. (Original.)

from pressure, in carcinomatous infiltration, in atelectasis, and in the presence within the pleural sac of serum, pus, or lymph not sufficient to cause flatness.

Flatness. Absolute or complete dulness, flatness, or deadness, occurs when air is completely absent, as in the stage of hepatization of acute pneumonia, in hemorrhagic infarction, in phthisis, in interstitial pneumonia, in carcinoma of the lung, in pulmonary œdema, in pulmonary abscess, in filled phthisical cavities or circumscribed gangrene, in condensation from pressure, in pleurisy with large effusion, in empyema, in hydrothorax, in great thickening of the pleura, and in tumors.

Hyper-resonance. When the resonance is increased, the sound is abnormally clear. If it is fuller and clearer than in health, without the characteristics of the tympanitic note, it is known as hyper-resonance or exaggerated resonance. The physical condition that causes exaggerated or hyper-resonance is increase in the amount of air. This increased amount of air may be *general*, *unilateral*, or *local*. When general (*bilateral*), it gives the characteristic sound heard in emphysema. In this affection the amount of air is so great and the tension of the chest-walls so exaggerated that hyper-resonance and sometimes a pure tympanitic sound ("band-box" resonance) are produced over the entire thorax. At the same time normally dull areas are encroached upon. The heart-dulness is effaced, the liver-dulness lowered. A like increase in resonance may be present in acute miliary tuberculosis. *Unilateral* increase in resonance occurs when there is an increased amount of air in one lung, on account of compensatory enlargement (vicarious or compensatory emphysema), or on account of an increase of air in the pleura. *Local* increase of resonance occurs when a local area of the lung is acting

in a compensatory manner. This is seen in cases of phthisis in which the alveoli or lobules surrounding small areas of consolidation are greatly distended. The exaggerated note may aid in the recognition of a deep consolidated area. *Skodaic resonance* is obtained over a portion of the lung above the line of pleural effusion, and above the line of consolidation in pneumonia, being due to relaxation of the lung tissue.

Tympany in Disease. If a tympanitic note is elicited over a part where in health resonance should be found, it is an indication of disease. It signifies that air is confined in a large space (cavity) or that there is an excess of air in many sacs (emphysema). A tympanitic sound from the chest occurs: (1) *bilaterally*, in cases of marked emphysema, and (2) *unilaterally*, in cases of pneumothorax and marked compensatory emphysema. In pneumothorax the pitch may be raised if there is much tension, the note then being known as dull tympany. (3) Tympany may also occur *locally*. (*a*) It is limited to the lobe of the lung in some cases of compensatory emphysema; (*b*) it may occur in the early stages of pneumonia, or in the later stages of complete consolidation. In the former it is due to relaxed tension; in the latter, to the presence of air in the bronchus, the lumen of which is free; (*c*) in cases of pleural effusion, owing to alteration in the tension of the lung, a tympanitic note is present above the layer of fluid; (*d*) in phthisical cavities at the base or the apex, and in bronchial dilatation, if the cavity communicates with the air, has moderately thin, elastic walls, and is at the same time empty, a tympanitic note is produced. The musical pitch of the note depends upon the volume of air, the size of the opening, and the tension of the wall. Large volume of air, low pitch; small volume, high pitch. Large opening, low pitch; small opening, high pitch. Greater tension, higher pitch; less tension, low pitch.

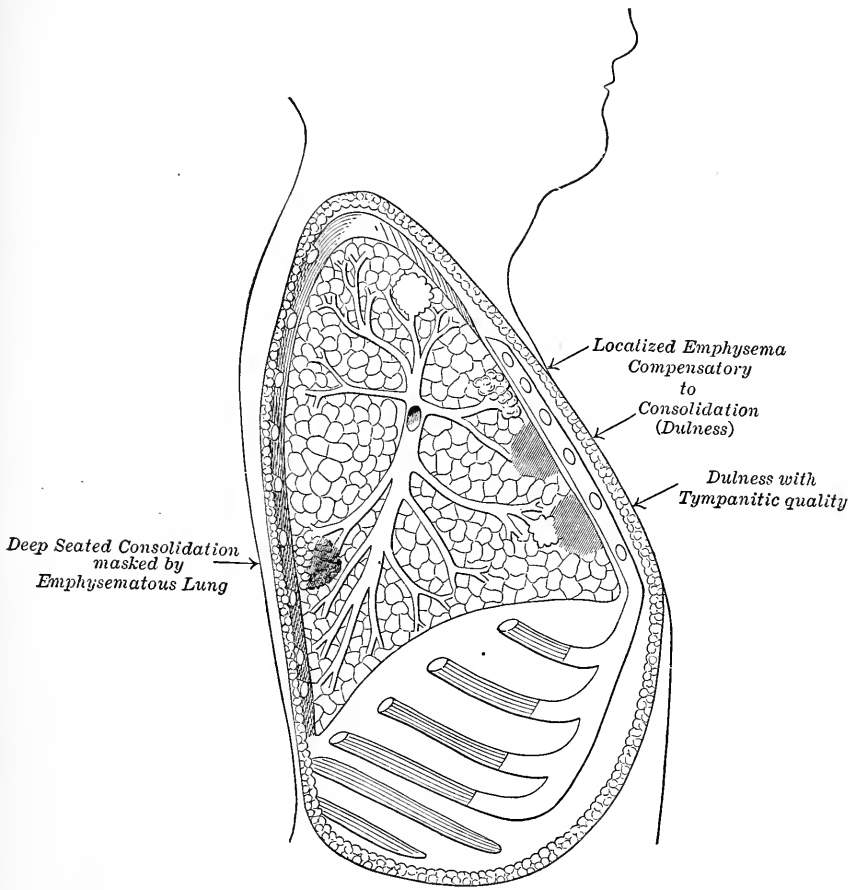
Special Sounds. Amphoric Resonance. The amphoric sound is tympanitic, but has a metallic clang, or echo, which is an over-tone, the prolongation of the sound being compared to an echo. It is like the sonorous ring of the voice when one utters a tone in an empty hall. It can be imitated by percussing an empty vessel. It is heard best in cases of pneumothorax and in large, superficial, phthisical cavities with smooth walls and having open communication with a bronchus. It is elicited by one or two rather forcible blows made when the patient's mouth is open.

Cracked-pot Sound. The cracked-pot sound, as the name indicates, resembles that produced when a cracked metal vessel is tapped; it may be simulated by clasping the hands loosely and striking them over the knee. It is heard best over a cavity that communicates directly with a bronchus, especially if the chest-wall is thin and yields to the percussion-stroke. In order to elicit the sound, the patient should be asked to open his mouth. The sound should be created at the time of expiration, and the percussing finger should be held in place instead of elevated after striking the plexímeter. Both the cracked-pot and the amphoric sounds often may be distinctly perceived if the ear be brought in close proximity to the patient's open mouth, when otherwise they are not appreciable.

In some rare cases this sound can be elicited in health. It may be generated if the chest of a healthy, screaming infant is percussed. In

this instance it is due to the compressed air forcibly throwing the vocal cords into vibration. The other pathological conditions in which the sound occurs rarely are: pleurisy when the chest is percussed above the effusion; pneumonia before consolidation has taken place; and pneumothorax if there is a free communication between the cavity and a bronchus. In the latter instance the sudden rush of air into the bronchus produces this sound. This is proved by the fact that it can be created when the

FIG. 159.



The physical causes of various tones. (LE FEVRE.)

chest is percussed in a case of empyema after the fluid has been evacuated by a free incision. While this sound is corroborative, it is not of itself positive evidence of any single condition.

WILLIAMS' TRACHEAL TONE. This is a tympanitic sound elicited over the apex of a consolidated, shrunken, or thickened lung when it is percussed in front. It is due to the vibrations produced by the percussion-blow in the trachea and primary bronchi, which vibrations are conducted by the solid lung tissue.

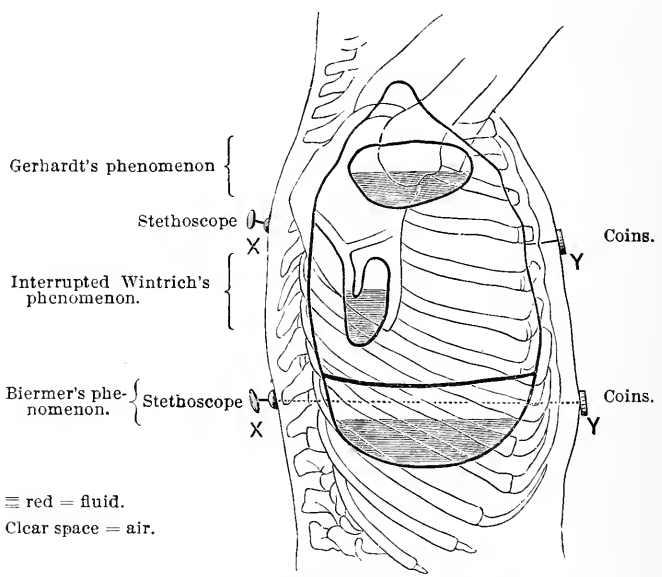
Wintrich's Change of Sound. The sound elicited by percussion over a cavity communicating with a large bronchus changes when the patient alternately opens and closes his mouth, becoming louder, more distinctly tympanitic, and higher in pitch when the mouth is open.

Interrupted Wintrich's Change of Sound. Wintrich's phenomenon may be distinct in some positions of the body, but indistinct or absent in others. This is a positive sign of a cavity containing fluid, which occludes the communicating bronchus in one position but leaves it open in the other.

Friedreich's Respiratory Change of Sound. The note over a cavity is higher in pitch at the end of inspiration than after expiration. This is due to increased tension of the chest-wall, of the lung tissue, and of the walls of the cavity, and to the widening of the glottis during inspiration.

Gerhardt's Change of Sound. The tympanitic sound elicited over a cavity containing fluid may change its pitch with change of the

Fig. 160.



Illustrating Gerhardt's and Biermer's phenomena, interrupted Wintrich's phenomenon and coin-percussion.

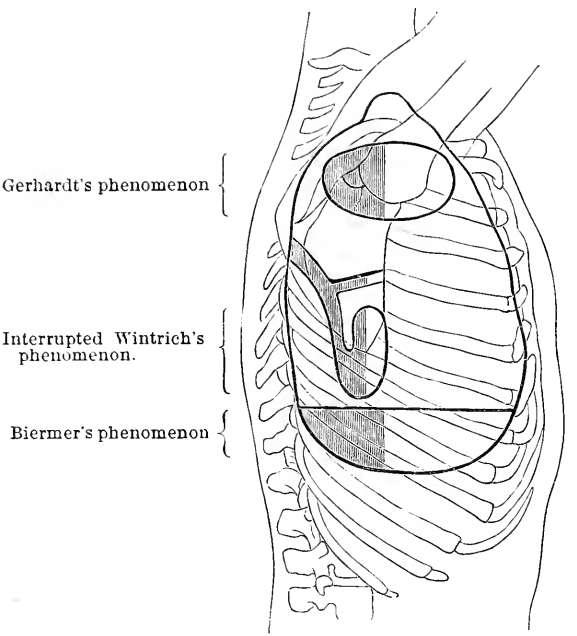
position of the patient, especially if the cavity be longer in one diameter than in the others. This is due to the change in the relative positions of the air and the fluid contained in the cavity.

Biermer's Change of Sound. The percussion-note over a pneumo-hydrothorax changes in pitch with alteration of the patient's position, on account of the change in the relative position of the air and the fluid.

Bell Tympany. This is elicited in pneumothorax by *coin-percussion.* One person listens at the back of the chest while a second person percusses the front of the chest with two large coins, using one as a pleximeter and

the edge of the other as a plessor. Air in the pleural cavity will conduct the sound so that it is heard on the opposite side of the chest as a soft, musical, metallic, echoing sound, like the chiming of a distant church bell or the ring of a hammer on an anvil far off.

FIG. 161.



Illustrating Gerhardt's and Biermer's phenomena, and interrupted Wintrich's phenomenon.

Abnormal Boundaries of the Lungs. The lungs are *increased* in size when the resonance extends beyond the normal boundaries. This is seen in emphysema, in which the area of resonance extends above the clavicles to a greater height than in health, and encroaches upon and may altogether displace the normal area of cardiac dulness; it extends $1\frac{1}{2}$ to 2 inches beyond the lower limits of the healthy lung, making the upper border of liver-dulness lower—instead of the fifth or sixth space making it an inch or two below. *Diminution* in size may also occur. Shrinkage of the apices (one or both) takes place in phthisis, hence the resonance does not extend so high up in the neck. Shrinkage or contraction may take place along the lateral borders of the lower edges in phthisis or retracting pleurisy, causing diminution in size of the lung and spurious enlargement of the heart or liver. In diseases below the diaphragm, effusion or enlarged liver, the size of the lung varies.

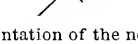
The Lung-reflex. It sometimes occurs that the more a dull area is percussed, the more resonant it becomes. Cabot believes this is part of the "lung-reflex" described by Abrams, who found that if an irritant such as cold or mustard be applied to any part of the skin covering the chest, the underlying lung expands in response to the irritation, producing a temporary localized emphysema.

Auscultation in Diseases of the Lungs and Pleuræ.

In auscultating the lungs we listen for (1) the sounds produced by respiration as heard over the larynx, trachea, and chest (the *breath-sounds*); (2) the new or *adventitious sounds* present only in disease; and (3) the resonance of the spoken voice as heard over the chest (*vocal resonance*).

It may be well to call attention to the confusion that always arises when the student is examining the chest for the first time. The coincidence of heart-sounds and lung-sounds in the chest often prevents the discrimination of the latter sounds. If attention be paid to the respiratory rhythm, the breath-sounds can be distinctly isolated. In auscultating the lungs the student should place his hand on the thorax or on the epigastrium and fix his attention upon the two acts of respiration—inspiration and expiration. Noting the occurrence of each movement, the expansion of inspiration and the contraction of expiration, he should then analyze carefully the sounds during each event of the respiratory act. Having fixed his attention on the respiratory movement and its divisions, and having excluded cardiac rhythm, the observer should note (*a*) the character of the sound in inspiration; (*b*) the character of the sound in expiration; (*c*) the relative length of the two. By means of the first two precautions the sounds of respiration are accurately ascertained, and confusing extraneous sounds, as from the heart, distinctly eliminated. By attending to the third, cognizance is taken of the rhythm of the sounds. In health the movements of inspiration and of expiration are almost equal; but the sound of inspiration is heard during the entire inspiratory movement, while that of expiration occupies only the first third or so of the expiratory act. The sound produced during expiration may even be less than half the length of that produced during inspiration. The following proportion represents relative lengths—Ins. : Exp. :: 3 : 1.

FIG. 162.



Diagrammatic representation of the normal breath-sounds.

The Breath-sounds in Health. These include the two normal sounds and their modifications in health.

Bronchial Breathing, or Normal Laryngeal or Tracheal Respiration. If the stethoscope be placed over the trachea at the top of the sternum, a sound will be heard characterized as follows: (*a*) it attends both inspiration and expiration with a definite pause between; (*b*) the inspiration and the expiration are nearly equal in length, and (*c*) they are of a tubular, blowing character. The expiration is perhaps a little stronger and longer than the inspiration. If the mouth is closed, there is no change except that both inspiration and expiration are harsher and sharper. The sound that is heard in this situation is known as *bronchial* or *tubular breathing*, and is one of the normal sounds of the chest. It may be heard behind, at or a little below the seventh cervical vertebra, where it is feebler in quality than when heard over the trachea in front. It is also heard in

the interscapular space over the large bronchi as they leave the trachea. Bronchial breathing is normal when heard in these areas.

The sound may be imitated by breathing deeply with the back of the tongue and the soft palate in the position to pronounce the guttural "ch" or "h." The tubular character of the sound and the relative length of the sounds heard during inspiration and expiration are diagrammatically represented in Fig. 163.

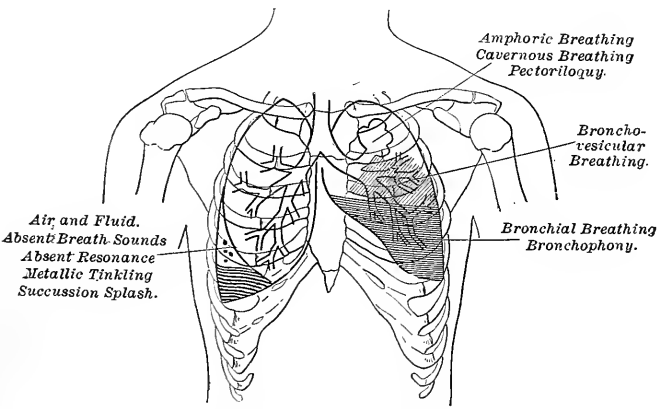
FIG. 163.

Bronchial breathing.

CAUSE OF BRONCHIAL BREATHING. The sound is caused by the passage of air through the nares into the wider pharynx when the mouth is closed, and through the trachea and large bronchial tubes.

Vesicular Breathing, or the Normal Respiratory Murmur. If the ear be applied over the anterior portion of the chest, or, better still, in the upper axilla or below the angle of the scapula behind, a sound is heard both on inspiration and expiration, differing from bronchial breathing, however, in that inspiration and expiration are changed in length. The sound of inspiration is twice or three times as long as the sound of expiration. The sound of inspiration is soft, breezy, or sighing in character, increasing in intensity to the end of full inspiration. It is immediately followed by expiration, which diminishes in intensity as the air is expelled, and terminates when one-half or two-thirds of the expiratory act is completed. The sounds can be imitated by breathing with the lips in the position required to pronounce "f" or "v."

FIG. 164.



Percutory and auscultatory signs.

The vesicular murmur is produced partly in the finest bronchial tubes and air-cells by their expansion and contraction, and partly in the upper air-passages, the latter sound being modified on account of the intervention of the air-vesicles between the ear and the larger bronchi. Normal vesicular breathing is shown diagrammatically in Fig. 164.

Modifications of the Breath-sounds in Health. The normal breath-sounds may be exaggerated or may be diminished in intensity.

Exaggerated Breath-sounds. Bronchial breathing and vesicular breathing are increased in loudness and sharpness by strong, rapid breathing. In some persons a sound is heard which partakes of the qualities of both bronchial breathing and the vesicular sound. It is noticed in the interscapular region about the level of the spines of the scapulæ, replacing the pure bronchial breathing which is heard in other individuals. Its characters are: soft, blowing or loud, harsh inspiration, and slightly prolonged blowing expiration, more exaggerated and louder, but not harsher, than in health. The term *bronchovesicular* is applied to this kind of breathing. It is due to the fact that the sound produced in the upper air-passages is conducted to the ear less dampened or modified, because the air-vesicles which surround the bronchus are here smaller in number than in the remainder of the lung.

The sounds are increased in children, in whom there are combined greater elasticity of the chest-wall and greater friction throughout the smaller bronchi, which are relatively larger. So distinct and characteristic is the sound in children that the term *puerile* respiration is applied to it. The sounds of inspiration and expiration are both intensified or sharper than in healthy adults, that of expiration being relatively prolonged.

Feeble Breath-sounds. The sounds are modified by the condition of the chest-walls. If they are thick, or there is an abundance of fat, the sounds are fainter or lessened in intensity. In wasting and exhausting diseases, feeble respiratory power causes feeble breath-sounds. The condition of the upper air-passages, even if not pathological, modifies the sound. If the glottis is small, or the relationship between the nose and pharynx is disturbed, the sounds will be modified—they are usually weakened.

Fig. 165.

Bronchovesicular breathing.

Fig. 166.

Puerile breathing.

Fig. 167.

Feeble breathing.

Diagrammatic representation of modified breath-sounds.

The Breath-sounds in Disease. It is well for the student to bear in mind that sounds heard in the chest which are departures from the normal sounds always indicate disease.

The normal vesicular murmur may be altered (1) in intensity, (2) in rhythm, and (3) in character.

Alteration in Intensity. The breath-sounds may be (*a*) increased, (*b*) diminished, or (*c*) absent.

Increased Vesicular Breathing. This may be bilateral, unilateral, or local.

Bilaterally the vesicular breathing or respiratory murmur is increased when there is increase in the force of breathing, when normal respiration is increased and the patient takes full, deep breaths; also in some forms of dyspnœa, as at the acme of the Cheyne-Stokes breathing, or in the

dyspnœa of diabetic coma; and in certain forms of bronchitis, particularly when the small tubes are narrowed by inflammatory swelling.

Unilateral exaggeration or increase of vesicular breathing is heard when one lung is acting vigorously or in a compensatory manner. A strong inspiratory sound followed by a strong and relatively prolonged expiratory sound of an actively moving lung almost certainly signifies disease of the opposite lung.

Local exaggeration of vesicular breathing, with the inspiration harsh, is noted in cases of phthisis in its earliest stages. It should be compared with the sound of the opposite side, whereupon the difference can easily be ascertained. It is heard over the apex when pneumonia or pleurisy is affecting the base of the lung, and *vice versa*.

DIMINISHED OR ABSENT VESICULAR BREATHING. This also may be bilateral, unilateral, or local.

It occurs *bilaterally* in a number of conditions:

1. The normal vesicular murmur is lessened in all cases in which expansion is interfered with. It is weak in feeble persons, particularly at the bases posteriorly. If the muscles of respiration are paralyzed or enfeebled, the murmur is also lessened. If the expansion is interfered with on account of disease of the diaphragm or by pressure upward by accumulations in the abdomen, it is likewise weakened.

2. Vesicular breathing is diminished by anything that lessens the amount of air supplied to the chest, such as occlusion or obstruction of the nares, of the pharynx, or of the larynx.

3. Chest-walls thickened from disease, as in œdema, weaken the respiratory sound.

4. The vesicular breathing is weakened throughout the entire extent of the lung in emphysema, the enfeebled respiratory forces and the short act of inspiration in this affection causing less air to enter the already over-filled chest. Moreover, in the bronchitis that attends emphysema the bronchioles are all more or less occluded, and hence the air-supply is diminished. These conditions cause a feeble respiratory murmur except at the anterior margins of the lungs.

Unilateral diminution of breath-sounds occurs:

1. When there is narrowing of the bronchus, as from an aneurism or a mediastinal tumor.

2. When there is pleural effusion, which (*a*) lessens the amount of air-pressure by compressing the lung and (*b*) interferes as a different conducting medium. (See Fig. 156.) If pain in pleurisy, pleurodynia, or neuralgia is present on one side, the breath-sounds of the affected side will be lessened. Not only in pleural effusions from serum, blood, pus, or air, but also in thickened pleura there is weakness or faintness of the respiratory murmur. It should not be forgotten that effusions and thickenings of the pleura rarely take place bilaterally; when they do occur, the breath-sounds are weakened, but not to the same extent as when an effusion is limited to one side.

Local diminution of breath-sounds occurs in the early stages of phthisis or in the earliest stages of pneumonia.

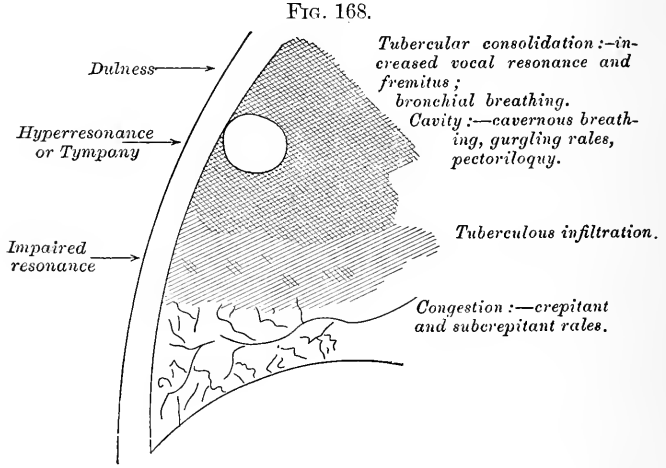
Alteration in Rhythm. The rhythm of the breath-sounds may be

altered. The inspiratory or the expiratory murmur may be lengthened or shortened and the inspiratory sound may be interrupted. The only changes in rhythm, however, having a distinct clinical value are prolonged expiration and jerking inspiration.

Prolonged Expiration. By this is meant prolongation of the expiratory murmur. When the expiration is prolonged, it equals inspiration, or may even be longer. This is due to the difficulty of the air in getting out of the chest in consequence of loss of elasticity of the cells, or of an obstruction in the bronchi, or of conduction of the sound created in the bronchial tubes. Loss of elasticity may be caused by over-distention of the air-vesicles. Hence, prolongation of expiration all over the chest is seen in emphysema and asthma. In this condition the inspiration is short, the expiration prolonged. Although distinct throughout the chest, it is more pronounced above the clavicles and along the free margins of the lung anteriorly. This is one of the first physical signs of tuberculosis. Swelling of the bronchial mucous membrane may prove an obstacle to the exit of air. When prolonged expiration is a part of bronchial or bronchovesicular breathing, it is usually due to the conduction by consolidated lung tissue of a portion of the sound generated in the bronchial tube.

Jerking or Interrupted Inspiration. Instead of the smooth, even, sighing, or breezy inspiration, the sound may be created in puffs or jerks, so that during the act of inspiration as the chest expands, a number of successive vesicular sounds are heard until the act is completed. The physical condition which causes jerking inspiration, or cog-wheel breathing, occurs in the earlier stages of tuberculosis, when the

Fig. 168.



Various stages of phthisis.

various bronchioles are more or less occluded by outgrowths of tubercles. The air, entering different lobules at different periods of time, thereby gives rise to this peculiar broken sound. This must not be confounded with the same character of breathing heard near the heart, due to press-

ure exerted upon portions of the lung by the heart or by structures in intimate relation with it, on account of which air enters various areas in puffs. Jerking inspiration sometimes occurs in health, and is simulated by the jerky act of inspiration in nervous patients. It is of no significance unless attended by other physical signs.

In cases of adhesion at the apex, particularly of the left lung, the same puffing or jerking inspiration is often heard. It is also present when an aneurism presses upon a bronchus, causing the air to enter the part in an intermittent manner. When pathological jerking breathing is present, the expiration is prolonged; and if the case is under observation a sufficiently long time, bronchial breathing will usually replace the jerky respiratory murmur in progressive consolidations. Small moist râles, excited by coughing or a full breath, usually attend jerking breathing when it is pathological.

Alteration in Character. The normal vesicular murmur may be altered in character, becoming harsher, or it may be replaced by bronchial breathing.

BRONCHOVESICULAR BREATHING. In this type of respiration are combined in varying proportions the characters of the bronchial and of normal vesicular respiration. The physicial condition producing bronchovesicular breathing is more or less consolidation surrounded by vesicular structure, as in the early stages of tuberculosis. It is found midway in change from respiratory murmur to bronchial breathing in progressive consolidations, and is observed in the early stages of lobar pneumonia and during resolution, in phthisis, interstitial pneumonia, hemorrhagic infarcts, and in condensation of the lung from pressure or from collapse. It also exists when the bronchial mucous membrane is swollen, as in bronchitis, and is heard over small consolidated areas in capillary bronchitis and catarrhal pneumonia, with collapse of lobules.

Bronchial Breathing. The normal situation of bronchial breathing in health has been stated. If the same kind of breathing is heard in any other portion of the lung, it is pathological. It is generally indicative of the presence of consolidation, the spongy lung tissue being replaced by solid conducting material, which conducts the bronchial sound to the ear. It is heard therefore in all pathological conditions in which consolidation takes place. It is the typical form of breathing heard in consolidation of the lung due to pneumonia or tuberculosis, in hemorrhagic infarcts, and in lung syphilis. It must not be forgotten, however, that pneumonia may exist without producing this type of breathing. This is the case when a large bronchus, or bronchioles, are occluded by inflammatory exudate. In tuberculous consolidation it may be absent for similar reasons. In central pneumonia where the consolidation is deep-seated and surrounded by lung tissue bronchial breathing may not be heard, or it may be postponed until the third or fourth day of the disease, by which time consolidation will have reached the surface of the lung.

In certain cases of pleurisy with effusion bronchial breathing exists. The accumulation not being sufficiently great to compress the lung completely, the bronchial tubes remain patent while the vesicular structure is compressed. Under these circumstances low-pitched bronchial breath-

ing is heard, more pronounced over the upper layer of the effusion. It is always heard close to the spine posteriorly, where the lung is compressed. Sometimes it is heard above the limit of the effusion, in all probability because of relaxed tension of the lung.

Bronchial breathing also occurs in tumor of the lungs, as in pulmonary carcinoma.

Modifications of Bronchial Breathing. While its special characteristics must be borne in mind, it must not be forgotten that bronchial breathing is not represented accurately in every instance by the sounds heard over the trachea. Its character may be modified and yet approach that type of breathing. The modification occurs in one or both of the two portions that go to make up the sound:

1. The blowing element may not be so distinct in inspiration as in expiration.

2. The characteristic blowing sound, in rare cases, may continue so long during expiration as to equal in length the inspiratory sound.

3. Bronchial breathing may vary in pitch. (*a*) At times the sound is high in pitch in both inspiration and expiration, but with a pure harsh blowing quality attending each. (*b*) It may be soft and low in pitch during both acts. The strong, high-pitched sound emitted by breathing deeply when the lips and tongue are placed in position to pronounce "ch," is termed *tubular breathing*. It is the characteristic sound of croupous pneumonia.

4. The loudness of the sound may also vary. This depends largely upon physical peculiarities of the individual, and is determined by the condition of the chest-walls and the force of the breathing.

When pleurisy with effusion coexists with pneumonia, the bronchial breathing, which should be audible, is feeble and distant.

To Elicit Bronchial Breathing. Breathing which, during very quiet respiration, may appear to be normal, is sometimes discovered to be bronchial when the patient has a spell of coughing and then takes, in rather quick succession, several deeper breaths than usual. Sometimes the noise made in nasal respiration obscures the pulmonary sounds. The patient should be instructed to breathe with the mouth open, to take somewhat deeper breaths than usual, and to let expiration follow at once upon the close of inspiration. Many patients when told to take deep breaths, expand their lungs to the utmost, and then hold the air in a while and allow it to pass out slowly. Such a method usually defeats the purpose of the examiner, which is first to note the relative length of inspiration and expiration, and then the quality of the two sounds, (*a*) as compared with each other, and (*b*) as compared with the normal. In listening for bronchial breathing the attention should be fixed more upon the length and quality of the expiratory sound; it is therefore important that the patient breathe so as to bring out its characteristics most clearly. This he can do by taking several moderately deep breaths in quick succession and with the mouth open.

Varieties of Bronchial Breathing. The quality of bronchial breathing is altered in the presence of a cavity, several varieties being observed.

Cavernous Breathing. If a case of tuberculous consolidation is watched, it will be found after a time that the bronchial breathing becomes lower in pitch. It is heard in inspiration and expiration, but a more hollow quality attends the sound. From the hollowness of the tone the word cavernous has been applied to the breath-sound; it is due to the formation of a cavity in the consolidation or to a dilated bronchus.

Fig. 169.

Prolonged expiration.

Fig. 170.

Jerking inspiration.

Amphoric Breathing. Cavernous breathing may have a metallic quality and is then called amphoric. It resembles the sound produced by blowing across the open mouth of a jar. A large cavity with smooth walls that communicates with the air through a small opening is the cause of the development of such a sound. It is heard also in pneumothorax when such communication exists. The metallic tone is analogous to the metallic percussion-sound. It occurs under the same physical circumstances. The physical condition which causes it may be so marked that the same character of tone is imparted to râles produced in the cavity, or to the heart-sounds which are transmitted by the solidified area surrounding the excavation.

Metamorphosing Breathing. Seitz has called attention to a form of breathing heard in connection with cavities, called the metamorphosing breath-sound. In this type inspiration begins harshly bronchial and then becomes faintly bronchial, the bronchial sound being heard also in expiration. It is said to be a sure sign of cavity.

Fig. 171.

Diagrammatic representation of cavernous breathing.

New or Adventitious Sounds. The foregoing sounds are modifications of the normal sounds heard during the act of breathing. New or

Fig. 172.

Diagrammatic representation of amphoric breathing.

adventitious sounds are also created in the lungs or in the pleura. They are rhonchi, râles, friction-sounds, the succussion-sound, and metallic tinkling.

Dry Râles or Rhonchi. These are musical sounds due to the passage of air through bronchial tubes which are narrowed either on account of swelling of the mucous membrane or on account of spasm.

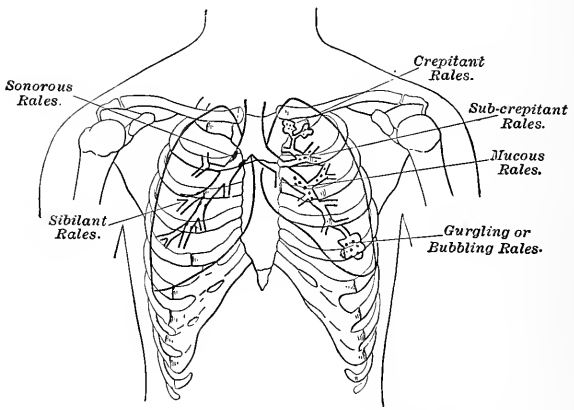
Dry râles may be (*a*) sonorous or (*b*) sibilant. *Sonorous râles* are created in the large bronchial tubes and are coarse, low-pitched musical sounds. *Sibilant râles* are created in small tubes, and are high-pitched whistling sounds. Both are heard over the areas of their creation, but

the sonorous râle may be transmitted all over the chest. They both may be heard at the same time. The dry râles are heard in the early stages of bronchitis, when the mucous membrane is swollen and thickened, but has not begun to secrete mucus or mucopurulent matter. They are also heard in asthma, in which there is spasm of the bronchial tubes, and in the chonic bronchitis of emphysema. In the latter the smaller râles are more common.

Moist Râles. These are due to the vibrations produced by the passage of air through fluid (mucus, serum, pus, and blood), and to the forcing open of air-cells agglutinated by lymph. They are produced in the air-vesicles, bronchioles, bronchi, and cavities. They may be (1) fine or *crepitant;* (2) small or *subcrepitant;* (3) large, coarse, or *mucous* (*bubbling or gurgling*).

Crepitant Râles. The crepitant râle is a fine râle, created in the alveoli, due to inflation of the cells whose walls have been held together by exuded lymph, or to agitation of fluid. It is a fine râle distinctly

Fig. 173.



Showing origins of the different kinds of râles.

localized, resembling the sound produced by rubbing a lock of hair between the fingers or by putting salt on a hot plate. In the early stages of pneumonia and in œdema of the lungs it is said to be pathognomonic. It may, however, be heard whenever there is a small amount of fluid in the alveoli and respiratory action is feeble.

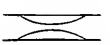
Subcrepitant Râles. The small, moist, or subcrepitant râles are created in the smaller bronchioles and in the alveoli. They may be general or local. If general, they are due to bronchitis in the second stage, there being an abundance of secretion in the terminal air-passages which is thrown into vibration by the current of air during the act of breathing. The element of moisture is pronounced and gives to these râles their quality, to which the term "crackling" is sometimes applied. Subcrepitant râles are found in congestion with outpouring and stagnation of secretion; in œdema; and whenever fluid is drawn into the bronchi, as in hemorrhage of the upper air-passages. Small moist râles in local areas

are found in phthisis, particularly at the end of the first stage and in the second stage, on account of the local bronchial catarrh. They occur in the early stage of pneumonia, particularly in the area of the lung that is adjacent to the consolidation and which is the seat of collateral œdema. They are also heard in the later stages of pneumonia when resolution has taken place, although here they may be replaced by large râles. They may be heard around any consolidation with an attending congestion, œdema, or catarrh. It must not be forgotten that cough or forced inspiration will often cause râles to be heard which otherwise would be missed.

Large, Moist, or Mucous Râles. These occur in the larger bronchial tubes, or in cavities, from the same causes that produce small râles. The fluid, however, is larger in amount, the air-current stronger, and the space for vibration is greater. While sometimes present in bronchitis and bronchiectasis, they are heard in their most marked form in the third stage of phthisis, when softening occurs. They are described as *bubbling* and *gurgling râles*, and are very characteristic after a full breath or cough.

Diagnosis of Râles. Râles are to be distinguished from other adventitious sounds. Although in some instances it is almost impossible to distinguish them from friction-sounds, as when râles are heard over the

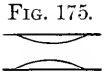
Fig. 174.



Diagrammatic representation of the thickening in the small bronchial tubes producing the sibilant râle or rhonchus.

bases of the lungs, they nevertheless have certain marked characteristics. Râles are recognized: (1) by the *qualities* previously mentioned; (2) by their *location;* if the adventitious sounds are general, they are due to râles; (3) râles are *modified* by *cough* and *breathing*. They may be

Fig. 175.



Diagrammatic representation of the sonorous râle or rhonchus.

intensified by either act, or after the completion of the act may disappear entirely. On quiet breathing, in the early stages of tuberculosis, for instance, they may not be heard at all. Before excluding them it is absolutely necessary to have the patient cough and then take a full

Fig. 176.

Diagrammatic representation of the crepitant râle produced in the air-vesicle.

breath; (4) they *vary* in *position*. This may occur from hour to hour. If the chest is examined in the morning, they may be more pronounced at the base for instance. At another time in the twenty-four hours they may be more distinct at the apex. They are more likely to be present at the base if the patient is kept in the recumbent posture. They *vary* in

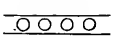
character. At one time small, moist rales may be heard, and in a short time they may be replaced by larger râles. Dry râles are regularly followed by moist râles in the course of bronchitis. In a case of bronchial asthma all sorts of râles may be heard in a few hours. Râles are distant to the listening ear—they seem to be farther away than do the friction-sounds.

Râles in the bronchi must not be confounded with the crepitant or fine crackling sound which is heard at the base of the lung in patients who have been ill with exhausting fevers and who have not taken full breaths for some time. The latter disappear after the patient has inspired deeply half a dozen times.

Râles throughout the lung in themselves are not diagnostic of any affection save bronchitis, but their occurrence all over the chest is significant in the absence of other physical signs. In the absence of bronchitis, râles at the bases of both lungs usually are due to congestion. Râles at one apex with failing health point to the onset of tuberculosis.

Pleural Friction-sound. In health the two surfaces of the pleura rub together without making any sound. When inflamed, as in *acute pleurisy*, the surfaces are roughened, due to the swelling and dilatation of

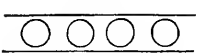
FIG. 177.



Diagrammatic representation of the small moist or subcrepitant râle.

the capillaries, or to the transudation of fluid or lymph. When the two surfaces are rubbing together under these circumstances, a sound is created to which the term friction is applied. It is heard at the end of inspiration and may continue during expiration. The sound is localized usually at the seat of pain ; it is *superficial*, being heard near the

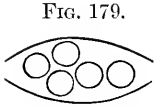
FIG. 178.



Diagrammatic representation of the large moist or mucous râle.

ear, and is not modified by cough or by full breathing, except occasionally by the latter when it is repeated. It may be *increased by the pressure of the stethoscope*. It is a *fixed* sound in that it does not disappear until effusion takes place. It may reappear when the fluid subsides.

FIG. 179.



Diagrammatic representation of the bubbling or gurgling râles that occurs in cavities.

The above characteristics distinguish it from râles. Both, however, may occur together. Although the friction almost always is of respiratory rhythm, when the pleurisy is in the neighborhood of the heart the friction may be of cardiac rhythm. Under these circumstances it is more distinct

during the act of inspiration. It is heard along the borders of the heart as a systolic rubbing, often of respiratory rhythm.

We not only distinguish the friction-sound by the characters just indicated, but also by the presence of pain, which renders its existence more probable. Usually it is heard at the base, in the nipple-line in front, or at the angle of the scapula behind, and frequently in the axillary region.

In cases of old *pleurisy*, dry, creaking sounds are heard not unlike the sounds produced when an old door is swung on rusty hinges or when new leather is bent. Other physical signs of pleural adhesions are present, and a friction-fremitus is often transmitted to the hand.

An old or dry friction is often heard at the apex in the neighborhood of *old cavities*. It attends both inspiration and expiration, is not modified by cough, and has none of the elements of the moisture that attends moist râles. The patient may be cognizant of the grating or rubbing sensation, and be able to describe the sensation felt during each breath. It may continue a long time after an acute pleural effusion has disappeared, and is sometimes the source of anxiety upon the part of the patient.

Pyæmic deposits in the lungs, *infarction*, *bronchiectasis with reactive pneumonia*, and *pleurisy with emphysema* are first revealed by pleuritic frictions. (Vierordt.)

At the base of the right lung a friction-sound may be the first indication, or at least an early one, of *hepatic abscess*. (Clark.) The pleural friction in the hepatic region must not be confounded with peritoneal friction of respiratory rhythm. In a case of secondary cancer of the liver a friction-sound was heard in the seventh interspace from *perihepatitis* over a cancerous nodule.

FIG. 180.

Diagrammatic representation of the friction-sound.

Metallic Tinkling. The impression imparted to the listener by this phenomenon is that of the falling of some material into fluid in a hollow space. The physical condition is that of a cavity partly filled with fluid, partly filled with air, into which fluid is dropping from an opening above. It is seen in hydropneumothorax, in pyopneumothorax, and in a few cases of large cavities. The air-chamber acts as a consonance-box and resonator, and gives a metallic quality to the sound. Other physical signs of cavity and fluid are associated. The sound may be heard when the patient is breathing quietly or only after coughing.

Splashing or the Succussion-sound. When the ear is placed to the side of the chest and the patient's body is moved suddenly by himself or by the observer, a splashing sound sometimes is heard. It can be produced only when air as well as fluid is present in a cavity. It was first described by Hippocrates, and the term "Hippocratic succussion" has been given to it. It is characteristic of hydropneumothorax and pyopneumothorax, although not present in all cases of these diseases. The sound may be audible at a distance. Metallic tinkling can usually be heard at the same time.

Auscultation of the Voice. When the ear or stethoscope is applied to the surface of the chest and the patient is asked to speak, the vibrations of the air in the trachea and bronchial tubes, set up by the act of phonation and transmitted to the chest-wall, become audible. The sound is known as the *vocal resonance.* It is a sign which goes hand-in-hand with *vocal* or *tactile fremitus,* both being modified by the same conditions. While, in general, conditions that increase the fremitus increase the vocal resonance also, this is not invariably the case. Sometimes one is increased and not the other, without there being any evident reason for it.

Method of Procedure. It is immaterial what words are selected by the patient to create the resonance. It is important, however, for the student to become familiar with the resonance of a definite series of words which when pronounced do not need any marked change in inflection of the voice. The words "one," "two," "three," or "ninety-nine," spoken repeatedly, are usually selected. The patient should not raise or lower his voice during the act of speaking. Symmetrical portions of the two sides of the chest must be examined successively.

Vocal Resonance in Health. Vocal resonance varies in health conjointly with the fremitus. The sound normally is purring or buzzing. It is heard more pronouncedly: at the right apex than at the left; in persons with thin chest-walls; and in individuals in whom the voice is low in pitch and strong. It is lessened therefore in females and children. It diminishes the farther away the ear gets from the larynx, and hence is feebler at the bases.

Vocal Resonance in Disease. In pathological conditions the vocal resonance may be (1) increased, (2) diminished, or (3) modified.

Increased Vocal Resonance. The degree of increased vocal resonance depends upon the intensity or extent of the cause. When slightly above normal, it is referred to as a slight increase. This usually is due to slight consolidation or to consolidation covered with a thickened pleura. The vocal resonance is also increased in cavities.

Bronchophony is the name given the sound when the voice is transmitted comparatively distinctly to the ear. This may be heard in health over the trachea or over the bronchi behind. When heard over the vesicular structures of the lung, it indicates that the vibrations are transmitted to the ear by some better conducting material, usually a consolidated lung. In all cases of consolidation bronchophony is present, but in pneumonia, if the bronchus is occluded by exudate, it is absent. If the lung is collapsed but the bronchi open, bronchophony may exist; sometimes the sound is even more pronounced than when heard over the trachea.

Pectoriloquy. The voice may be as distinctly transmitted as if the patient were speaking into the mouth of the stethoscope. If the patient speaks slowly, the words may be distinctly made out. It is more striking when the patient whispers. The term "*whispering pectoriloquy*" is then applied to it. It is detected over a cavity communicating with a large bronchus, and sometimes in consolidation of the lung.

Vocal Resonance Diminished. Vocal resonance is diminished or absent when anything cuts off the supply of air or intercepts the vibrations at the part the observer is auscultating. Vocal resonance is absent

over the area supplied by a bronchus which is occluded by external pressure, such as that of an aneurism. Diminution or absence of vocal resonance is marked in cases of pleural effusion (serum, blood, pus, or air) or of thickened pleura, the vibrations being impeded because of the difference of conducting material. The degree of diminution depends upon the amount of effusion.

Modifications of Vocal Resonance. Certain modifications of vocal resonance may occur.

At the uppermost limit of the pleural effusions, at which point the layer of fluid is thin, the resonance is transmitted in a modified form. It is tremulous and bleating in character, and is known as *ægophony* because it resembles the bleat of a goat. It is heard especially at the angle of the scapula, or below it in cases of moderate effusion. It is due to the fact that the fundamental tones are intercepted by the fluid, while the other tones are allowed to pass through and give the peculiar bleating sound. (Gee.)

The vocal resonance may have a *metallic* character in pneumothorax when there is free communication with the bronchus.

CHAPTER XXXVI.

PHYSICAL DIAGNOSIS OF DISEASES WITHIN THE ABDOMEN.

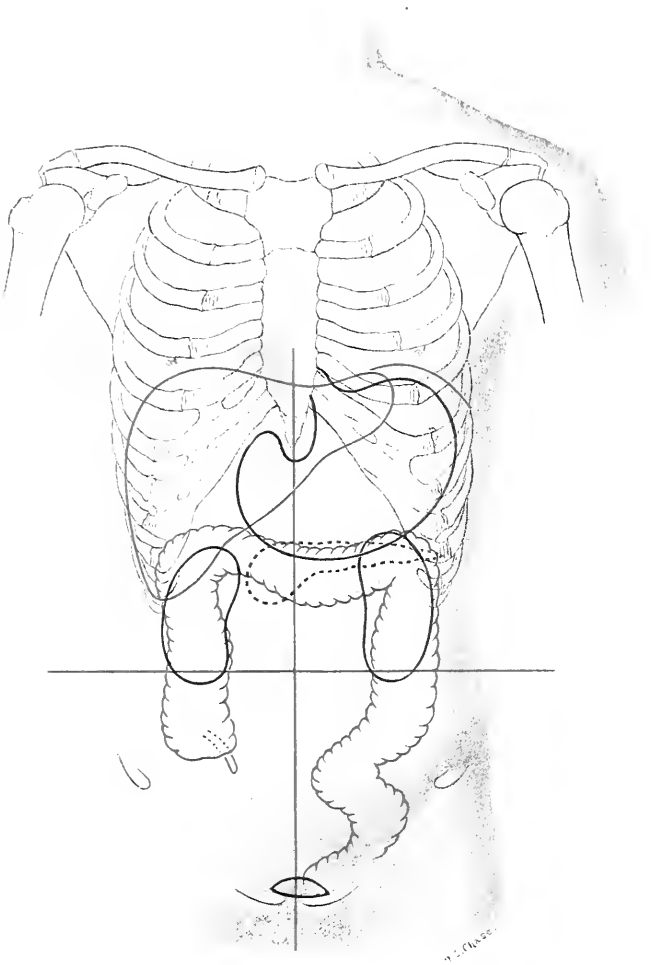
THE ABDOMEN.

THE abdomen is divided arbitrarily into regions to enable us to locate the various organs in health and in disease. Simplicity is essential, and a method of delimitation that is commonly used in the subdivision of other regions should be adopted for the sake of uniformity of description and to assist the memory of the learner. For these reasons Ballance's method of dividing the surface is the best. This author includes the abdomen within a circle having the umbilicus as its centre. The circle is divided into quadrants by diameters drawn at right angles, corresponding to the median and transverse umbilical lines. The portions to the right of the median line are the right upper and lower quadrants, respectively; the portions to the left are the left upper and lower quadrants. (See Plate VII.)

With the abdomen thus divided, measurements may be made from the umbilicus and the fixed bony points in the periphery of the circle to indicate the exact position of the structure under consideration. To locate a tumor in the right lower quadrant, for instance, the umbilicus, pubic bone, and anterosuperior spine of the ilium may be utilized. Measurements may also be made along radii extending from the umbilicus to other fixed points. For example, a tumor is situated in the right lower quadrant; the centre of the tumor is 2 inches below a point on the transverse umbilical line, 3 inches from the umbilicus; it is also 3 inches to the right of a point on the median line, 2 inches from the umbilicus. The size of the tumor can be defined by measurements from its centre. Organs bisected by the median line, as the bladder and uterus, may be described as situated in the median line so many inches to the right and left, as the case may be, and so many inches from the pubis. It is often convenient to describe a structure or morbid condition as being in the *epigastric*, *umbilical*, or *suprapubic* region when it occupies the median line of the abdomen, instead of describing it as being partly in the right and partly in the left upper or lower quadrant, as the case may be.

The *right upper quadrant* contains the right lobe of the liver; the gall-bladder; the hepatic flexure and part of the transverse colon; a portion of the pancreas; the pylorus, near the median line; and, deeper, the upper half of the kidney. The *left upper quadrant* contains the left lobe of the liver; the stomach; part of the transverse colon and the splenic flexure; the pancreas; the upper portion of the kidney; and the spleen. The *right lower quadrant* contains the cæcum; the ascending colon; the

PLATE VII.

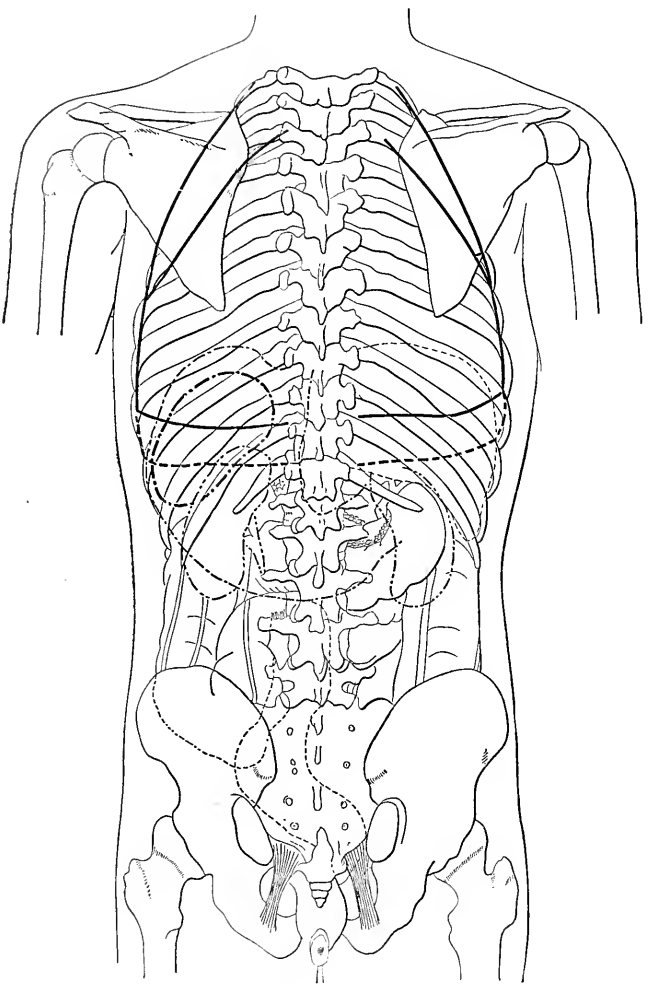


Quadrants of the Abdomen. Position of the Viscera.

Liver and colon—red lines.
Stomach, kidneys and bladder—solid green lines.
Pancreas—dotted green lines.

appendix vermiformis; the right tube and ovary; portions of the bladder and uterus; and above, at the end of full inspiration, the lower portion of the kidney. The *left lower quadrant* contains the corresponding tube, ovary, and portions of the bladder and uterus; the descending colon; and the sigmoid flexure. It does not usually contain the lower portion

FIG. 181.



Viscera of thorax and abdomen, as seen from behind. (After LUSCHKA.)

of the left kidney, which is one-half inch or more higher than the right. (Holden.) About the centre and extending to the periphery on all sides are the small and large intestines.

It must be remembered that objective abdominal symptoms are due not only to disease of the abdominal contents, but also to disease elsewhere. Thus the abdomen may be enlarged from the ascites of cardiac or renal disease, or contracted in tubercular meningitis.

Paralysis or other disease of the diaphragm alters the appearance of the upper half of the abdomen and modifies its movements in respiration. Fluctuating changes in size occur in hysteria and gastric neurasthenia; while the abdomen may be persistently retracted in tubercular meningitis.

Inspection.

We note the appearance of the abdominal walls, the movements of the abdomen, its shape and size (general enlargement), and the presence of local enlargements.

The Abdominal Walls. A glance suffices to determine the thickness of the abdominal walls. *Thin* walls are due to deficiency of adipose tissue and of muscular structure, either associated with general atrophy (see Emaciation), or sometimes caused by intra-abdominal pressure. Frequent pregnancies, previous ascites, or antecedent growths (ovarian tumor) lead to atrophy of the muscles; the recti separate and a hernia-like protrusion of abdominal contents results. Furthermore, a conical projection of the lower median portion of the abdomen is brought about, especially when ascites is present. Such projections are often confusing when tapping is to be resorted to. *Thick* walls are due to œdema or to increase in fat.

Color. The abdomen in general partakes of the hue of the skin; around the umbilicus it is darker, and in Addison's disease a distinct areola is often present. The median line, from the umbilicus to the pubis, darkens in pregnancy. This "brown line" as it is called, is sometimes seen in men. The skin of the abdomen may be the seat of specific eruptions, as in typhoid fever, and of sudamina. The walls are pale and glistening in œdema.

Markings. In first pregnancies and when there is marked ascites, less frequently in the presence of obesity and tumors, striæ are produced in the skin where the tension has been greatest. In pregnancy they form sinuous lines upon the lower lateral portions of the abdominal wall and upon the upper inner portions of the thighs. When first developed, they are reddish, but subsequently by a process of fading become more glistening and white than the rest of the skin. They are also known as "water lines" and *linea albicantes*. (See Fig. 182.) They may be seen after typhoid fever if the distention has been excessive.

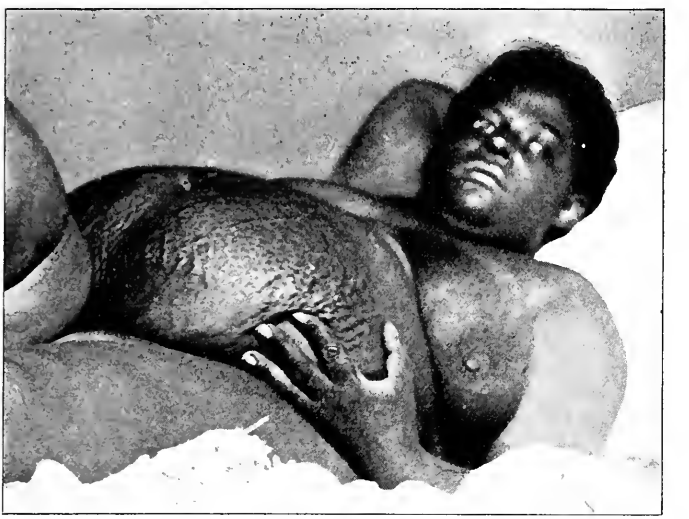
Pouting of the umbilicus occurs in pregnancy after the sixth month, with hernia, and when there is ascites. Not infrequently the walls around the umbilicus are infiltrated with carcinoma secondary to cancer of the stomach, and by excising a small portion of the tissue and examining it under the microscope a correct diagnosis of the internal disease can often be made. A similar but more inflammatory infiltration has been observed by Henry in tuberculous peritonitis.

Glands. Isolated lymphatic glands are sometimes observed in the abdominal wall, and may be examined microscopically to confirm any suspicion of malignant disease.

Veins. Enlargement of the superficial veins is a common accompaniment of cirrhosis of the liver, adhesive pyelophlebitis, and of any con-

dition causing obstruction to the free circulation of the blood in the inferior vena cava. To complete the collateral circulation the veins of the abdomen may anastomose with the mammary veins above or the epigastric veins below; and when the venous distention around the umbilicus is excessive, a characteristic radiating tumor, known as *caput Medusæ*, results. (See Plate XLIII.)

Fig. 182.



Pendulous abdomen and lineæ albicantes.

Movements. (See the Lungs—Dyspnœa.) The *movements* of the abdomen are of respiratory, vascular, gastric, and intestinal origin.

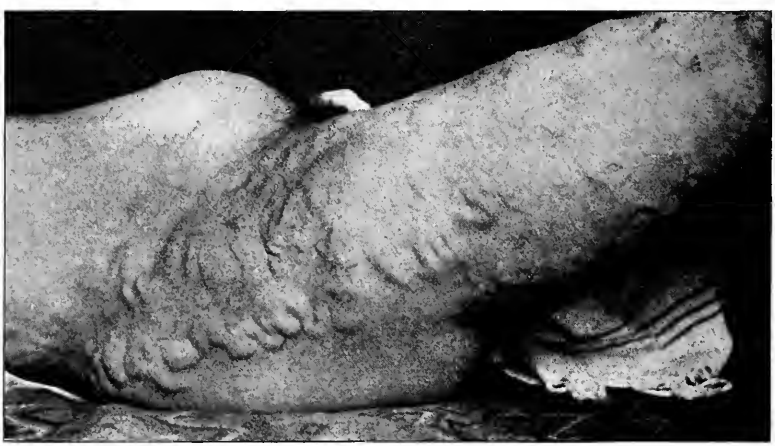
Respiratory Movements. The upper half of the abdomen in health swells or rises synchronously with inspiration; but when the abdomen is enlarged or contains a tumor in its upper half, the movement is restricted. In paralysis of the diaphragm the abdomen retracts during inspiration and the normal respiratory movement is reversed. When the paralysis is limited to one side, as in large pleural effusions, the inspiratory collapse is unilateral. In laryngeal and tracheal obstruction inspiratory retraction is noteworthy, and its extent affords an index of the degree of obstruction. Respiration causes the liver to rise and fall; in persons with thin walls its shadow can be seen to descend with inspiration, the extent indicating the degree of *respiratory expansion* and the size and position of the liver. Such information is of great value. A tumor connected with the liver and an enlarged gall-bladder will move synchronously with respiration. Other tumors, unless adherent to the liver, are fixed, excepting pyloric growths, which show moderate respiratory mobility. A rare exception is found in movable right kidney.

Vascular Movements. These are noted in the median line and usually in the upper half of the abdomen. In moderately thin subjects *epigastric pulsation* (*q. v.*) may be directly caused by the aorta. If the pulsation extends some distance to the right or left of the median line, an

aneurism may be suspected; or a growth, such as carcinoma of the stomach, overlying the aorta and transmitting its pulsations. Aneurism of the cœliac axis gives rise to a movement near the umbilicus and to the right or left of the median line. *Pulsation of the liver*, which is of vascular origin and therefore synchronous with cardiac pulsation, is seen in the hepatic area in dilatation of the right heart.

Gastric and Intestinal Movements. *Peristaltic movement*, whether of the stomach, or of the large or small intestine, may be visible through the abdominal walls. In gastric dilatation and gastroptosis the waves may be seen in rhythmical succession passing from left to right in the centre of the abdomen. Their general course is usually from the left upper to the right lower quadrant. When due to movements of the large intestine, the waves follow the course of the canal; while those which emanate from the small intestine are confined to the region around the umbilicus. Visible peristalsis when of gastric origin indicates obstruction at the pylorus; intestinal peristalsis is seen when the lumen of the bowel is obstructed. The movements may be excited by filliping the abdomen with a towel wrung out of cold water.

FIG. 183.



Varicose veins from pressure of aneurism of abdominal aorta.

Shape. In general enlargement of the abdomen the shape is uniform. In very fat subjects and in women with relaxed abdominal walls the abdomen may be *pendulous*. (See Fig. 182). In ascites the tissue over the umbilicus may protrude and form a localized prominence in women whose abdominal walls have previously been relaxed. Abdominal enlargements due to ascites sometimes assume a peculiar cone-shape (see Abdominal Walls), the base corresponding to the plane of the abdomen, the apex protruding below the umbilicus; this is particularly the case when the patient has had to maintain the semi-erect position for some time. Local enlargements, such as morbid growths or changes in the size of viscera, produce irregularities in the surface corresponding in position to the internal lesion. The shape varies momentarily in hysterical dis-

tention. In wasting disease of the viscera, as cancer of the retroperitoneal glands, the enlargement is replaced by retraction during the later stages, causing undue prominence of the affected viscera.

Palpation and Percussion.

Palpation and percussion in diseases of the abdomen may be discussed together.

Position of Patient. Generally the best position is the recumbent one, because it admits of examination without too great exposure, and because in that position the abdominal muscles are partly relaxed; when the latter need to be still further relaxed, the head and shoulders should be partly elevated and the knees drawn up. In certain obscure tumors much can be learned by having the patient rest on the hands and knees or assume the knee-chest position: the pulsation of aneurism can thus be distinguished from pulsation transmitted by a tumor, as the latter falls away from the vessels in the knee-chest position so that the pulsation is lessened. A tumor surrounded by coils of intestine may become more distinctly palpable. A good plan to secure muscular relaxation when palpating the liver and spleen is to have the patient sit on a chair with the body leaning forward and the thighs flexed, while the feet rest on a stool or the rung of another chair.

Method. The examining hand should be warm, as the sudden application of cold throws the abdominal muscles into involuntary contraction. By grasping the abdominal walls between the thumb and fingers their thickness and the relative proportion of *fat* can be estimated, as well as the presence or absence of *œdema* of the skin, which, although general, is especially marked in the lateral and posterior portions of the abdomen. Relaxation of the abdominal walls is observed after dropsy and pregnancy. Redundant skin remains in folds when pinched up, particularly in those suffering from abdominal cancer.

When it is desired to explore deeply, the patient should be instructed to breathe with the mouth open, and the examining hand pressed firmly in during expiration, and held there, if need be, during several long respirations. The palm of the hand should be laid upon the surface; after the muscles are relaxed, the flexed fingers may be used to palpate. The same procedure is adoped when it is desirable to get the percussion-note of a body lying deep in the abdomen—that is, the finger is pressed firmly and deeply in, and then percussed; in this way any superficial resonance due to overlying intestine is largely eliminated.

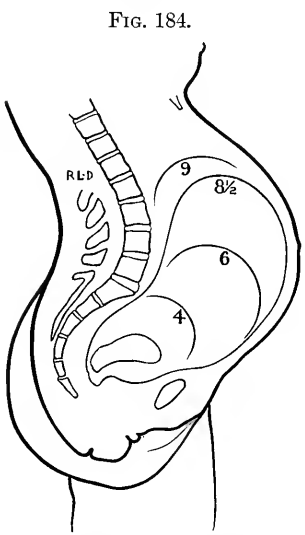
When palpating to determine the lower edge of the *liver* or *spleen*, the palmar surfaces of the fingers are pressed into the abdomen at different levels from below upward until the edge of the organ is felt. When the liver is in its normal position, the edge of the right lobe extends to the margin of the ribs, and may be found by pressing the fingers in as described and having the patient take a long breath.

By *palpation* the information obtained by inspection is confirmed: the character of the abdominal walls and of any enlargement that may be pres-

ent is determined; the precise location of pain is ascertained; the condition at the hernial rings and the movability of tumors are investigated. By passing the hand gently over the surface of the abdomen, the condition of the integument—whether smooth and elastic, or harsh and dry—is readily determined. Any marked *unevenness*, such as is produced by umbilical or inguinal herniæ, by striæ, by large tumors of the pylorus, by cancerous nodules, or by hydatid cyst of the liver, is at once detected. The degree of *tension* of the abdominal walls is easily appreciated. It is increased, although not uniformly, whenever the abdomen is very much enlarged; but some persons are so sensitive to touch that any attempt at palpation throws the abdominal muscles into such rigid contraction that examination is impossible. *Rigidity* of the abdominal walls may be the only sign of acute peritonitis and is quite common when the disease is local. Hurried palpation throws the recti into sudden spasm. Local contractions point to inflammation underneath. Tuberculous peritonitis is characterized by distention with board-like rigidity or preternatural hardness; a condition to which the French apply the term *carreau*. *Peritoneal friction* is most frequently felt over the liver, and occurs chiefly in chronic peritonitis.

General Enlargement of the Abdomen. The abdomen differs very much in size in different persons, depending not only upon the thickness of the fat in the abdominal walls and omentum, but also upon the calibre of the intestines themselves, which are apt to be much distended in those accustomed to eating large meals. In general, the belly is more protuberant in infants and children than in adults. Enlargement occurs in *obesity*, and it is often difficult to tell whether the excessive deposit of fat in the abdominal walls and omentum accounts for the whole enlargement or only serves to mask the presence of a tumor. Enlargement of the belly is only one of the features, though frequently the most pronounced evidence of obesity; whereas in enlargements of the abdomen from *tumors* and *ascites* (Plates XXXVIII. and XLIII.) there is usually a marked contrast between the size of the abdomen and that of the rest of the body.

FIG. 184.



Size of uterus at various periods of pregnancy. (JEWETT.)

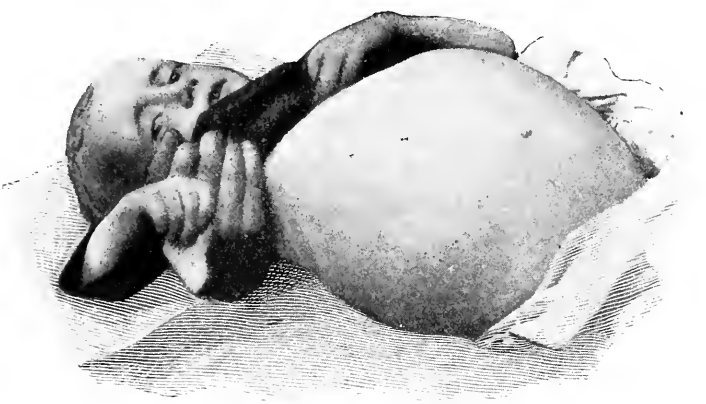
During the last month or two of *pregnancy* the abdominal enlargement is general, especially when the woman has previously borne children.

Enlargement due to *accumulation of gas* within the bowels is general and may attain a very high degree, lending the abdomen a uniformly arched appearance like that of a barrel. The diaphragm may be forced upward so as to interfere seriously with respiration and heart action. In debilitated children the enlargement due to flatulence is associated with flaccidity of the abdominal walls, causing lateral and central enlargement.

A moderate degree of distention may be the result of eating certain articles of food, such as turnips or beans. Excessive accumulations of gas are observed in typhoid fever; peritonitis, operative and non-operative; and in stenosis of the colon or rectum from any cause. Excessive distention is also common in hysteria.

Dilatation of the Colon. In children the enlargement of the abdomen may be general. The dilatation may be temporary, as in constipation with obstruction; or in rare instances permanent, when the distention of the abdomen gradually becomes enormous. The dilatation often begins in childhood and continues through adult life, unless it becomes so severe as to cause death from malnutrition, copræmia, and pressure. Congenital obstruction, the eating of oatmeal and similar food are some of the causes. The constipation may continue uninterruptedly for several weeks, during which period the dulness in the track of the colon increases,

FIG. 185.



Case of dilatation of colon. (GRIFFITH.)

and fecal tumors may be discovered on palpation. This condition is temporarily relieved by diarrhœa lasting two or three days, which may be preceded by vomiting of a fecal character and during which enormous quantities of feces are passed. After the bowels have been opened the distention continues, dulness being replaced by tympany. In the cases reported by Hughes and by Osler the abdomen was uniformly enlarged and coils of intestine were visible through the thin abdominal walls. Formad's patient was an adult. It must be remembered that intestinal peristalsis is also observed in constriction of the bowels. (See page 508.) The movement of the intestine above the seat of stricture is undulating or worm-like, and the bowel itself is dilated. From a consideration of the recorded cases of so-called *idiopathic dilatation of the colon*, Treves believes that although in adults enormous dilatation of the large intestine may undoubtedly occur independently of mechanical obstruction, in children it is probably due to congenital defects in the terminal portion of the bowel. In some cases it is certainly due to elongation and kinking of the sigmoid flexure, causing more or less constant obstruction—a condition that has been well studied by Göppert.

Enlargement of the abdomen simulating ascites may be due to retroperitoneal and peritoneal *lipomata*. Fluctuation even may be detected, but repeated puncture fails to reveal fluid. A negative result should always suggest lipoma; and if the tumor causing the enlargement is of slow growth, more conspicuous on one side than on the other, and causes little if any general disturbance except progressive emaciation, dyspnœa, and sometimes œdema of the legs, the probability is all the more in favor of lipoma. The tumor is usually crossed by a portion of the intestine.

Other causes of general abdominal enlargement are *hydatid cyst, cancer of the bowel or peritoneum, fecal accumulation,* and *diseases of the liver and gall-bladder.* (See Plate XLIV.) Enlargement of the latter organs may give rise to only a local swelling in the right upper quadrant; but when they attain very large dimensions, as happens not infrequently in cancer, amyloid disease, and hydatid cyst of the liver, inspection may show only general enlargement, with small prominences corresponding to cancerous nodules or small cysts.

Splenic enlargement attains its greatest development in leukæmia, so-called "splenic anæmia," and in chronic malarial poisoning. It is often visible as a general enlargement of the belly, but may also produce a more marked prominence over the lower left ribs and the corresponding lumbar region. (See Plate XLVI.)

In *diseases of the kidneys* associated with great enlargement a prominence is usually visible in the flanks and lumbar region corresponding to the kidney involved unless there is considerable emaciation; anteriorly the enlargement, if any be visible, usually appears to be general.

Enlargements of the abdomen beginning in the lower quadrants and becoming general secondarily are usually of pelvic origin. The most common are those due to *pregnancy, retroperitoneal sarcoma, ovarian* or *parovarian cysts, uterine fibroids* and *fibrocysts,* and *abscess* or *peritoneal effusion* as in chronic peritonitis. As a greatly distended bladder may give rise to confusion, it is a good rule to pass a catheter and make sure that the viscus is empty before proceeding further with the examination.

Local Enlargements or Tumors of the Abdomen. An enlargement in the *region below the sternum* or epigastrium usually indicates distention or *dilatation of the stomach* (see Diseases of the Stomach), or a *tumor at the pylorus* which is almost always cancerous. Epigastric prominence is also seen in large eaters. It is not uncommon, however, to find here a *cancerous nodule* projecting from the surface of the *liver* or a *hydatid cyst* of the same organ. The diagnosis is made by determining, with the aid of palpation and percussion, whether the tumor is continuous with the liver and moves with respiration; its apparent depth from the surface; the presence or absence of tenderness and fluctuation; and whether the subjective symptoms point to disease of the stomach or of the liver. *Aneurism, cancer of the large intestine,* and a morbid growth in the *left lobe of the liver* are among the possible causes of enlargement in this region, in which, or to the left of the median line and near the level of the umbilicus, *effusions into the lesser peritoneal cavity* are also found. Much more rarely a swelling in the epigastrium is caused by a tumor of the pancreas, such as cyst, abscess, or cancer, or by sclerosis of that organ.

According to the studies of Fitz, *pancreatic cyst* is marked by deep-seated colicky pain occurring in paroxysms; the evacuation from the bowel of matter resembling saliva, the presence of much undigested fat and sugar in the urine, salivation, and the occurrence of jaundice. *Cancer of the pancreas* is recognized by the presence of a painful tumor in the epigastrium, which in most cases will be found absolutely immovable. The pain is not aggravated by the taking of food, but is said to be more severe in the erect posture. The bowels are constipated and the stools may or may not be fatty. The emaciation, as in all cancerous affections, is progressive, and in the last stages there may be occasional vomiting and persistent jaundice. (See Plate XLVI.)

A *rigid rectus muscle* is capable of simulating a tumor. In hysterical subjects rigidity of the abdominal muscles with tympanites sometimes gives rise to a swelling known as "phantom tumor." Such swellings are less constant in shape and character than genuine tumors, and, although dull on percussion, appear more superficial; they sometimes disappear under friction with the hand, and invariably under full anæsthesia; when, in addition, the stigmata of hysteria are present, all doubt is at once removed. These phantom tumors are, as Fitz has pointed out, often really cases of dilatation of the colon.

Enlargements in the *right upper quadrant* or right hypochondrium are most frequently due to disease of the liver and gall-bladder, to which the reader is referred for a fuller exposition of the subject. Less frequently a much *enlarged* or *cystic kidney* causes swelling in this region; the diagnosis rests on the history, the direction in which the tumor has grown, the findings in the urine, and the relation of the ascending colon to the tumor, for renal tumors push the bowel before them as they grow, so that their dulness is obscured by the tympany of the superimposed colon. (See Plate XLIV.)

Primary *malignant disease of the suprarenal bodies*—a rare affection—is often accompanied by swelling of the upper abdomen.[1] The clinical picture, even when both organs are involved, is not that of Addison's disease, although some of the symptoms of that disorder, such as pigmentation, vomiting, asthenia, and pain in the back, may be present in a minor degree. The growth extends forward and in many respects resembles renal tumor; although it may also simulate a tumor of the liver, an enlarged gall-bladder, or a pancreatic cyst.

Enlargement in the *right lower quadrant* or right iliac region occurs with affections of the *cæcum* and *appendix*, as appendicitis, typhlitis and perityphlitis with or without pus-formation, fecal accumulation and fecal abscess, carcinoma, and stricture of the ileocæcal valve; and with *ovarian and tubal disease*, such as ovarian tumor, cyst of the broad ligament, pelvic abscess—usually of tubal origin—and extra-uterine pregnancy. Other conditions to be thought of in this region are acute and chronic *tuberculous peritonitis*, and an *enlarged* or *movable kidney*. In these conditions the most exact information in regard to physical characteristics is obtained by means of palpation and percussion, which, with the clinical history, enable the physician to distinguish one from the other.

[1] Rolleston and Marks, American Journal of the Medical Sciences, 1898.

Appendicitis. The subject of appendicitis will be treated formally in a separate section (see Diseases of the Intestines); there are, however, a few general considerations in connection with the local examination of the abdomen that may be emphasized at this point, even at the risk of some repetition.

The information supplied by palpation and percussion in perforation of the appendix will depend upon the rapidity with which perforation has supervened and upon the stage at which the examination is made. Generally speaking, after the sudden onset of pain in the right iliac fossa, in a person previously in good health, there is tenderness on palpation in that region, which at first is localized, but may spread with great rapidity over the whole abdomen. On the other hand, the tenderness may at first be general and subsequently become localized over the appendix. After a time the tension in the part is increased, the overlying abdominal muscles become firm and rigid from spasm, and the percussion-resonance impaired. Examination with the finger in the rectum may discover a tense, swollen appendix or a tumor in the pelvis.

But the disease may be *fulminating* in character, perforation being followed by the rapid development of peritonitis with collapse, so that when the patient is seen there is no more tenderness over one part of the abdomen than over another.

Again, the appendix may suffer *repeated attacks* of inflammation without perforation, but with the development of *local peritonitis*, manifested by increased thickness in the region of the cæcum, tenderness, diminished resonance, and increased resistance to the percussed finger. Sometimes an enlarged and hardened appendix can be made out by palpation, both during an attack and in the intervals.

In still other cases, of slower development, a distinct *perityphlitic abscess* develops. In addition to local pain and tenderness a swelling appears above Poupart's ligament, over the skin, which becomes brawny and pits on pressure with the finger-tips. The tumor is dull on percussion, and obscure, deep-seated fluctuation may be made out by rectal examination.

In *fecal impaction of the cæcum* a tumor forms in the course of the cæcum, extending upward from Poupart's ligament. It is usually oblong and rounded, of a doughy consistency, and may appear uneven or lumpy on closer palpation; there is no tenderness unless the cæcum itself becomes inflamed. A valuable diagnostic point in the differentiation of fecal tumors which give rise to some distention of the abdomen was discovered by Gersuny, who first called attention to the "adhesive symptom." If strong pressure is slowly made with the finger-tips, and then gradually withdrawn, a peculiar sensation due to the separation of the intestinal mucous membrane from the fecal matter is transmitted to the hand as it is removed from the abdomen. If the feces are dry and hard, the sensation may not be perceptible until an oil enema has been given. When the feces are soft, either naturally or artificially, the tissues remain depressed for some little time before they slowly separate from the mass and return to their normal position. Delayed separation of the abdominal walls from the tumor is also characteristic of fecal accumula-

tion. The diagnosis is made by the situation and character of the tumor; its disappearance under the influence of purgatives; and the absence of pain, tenderness, and constitutional symptoms.

If the impaction causes a localized colitis, or so-called *typhlitis*, the tumor is tense, tender, and painful; the dulness being sharply limited by the boundaries of the cæcum.

In *stricture of the ileocæcal valve* due to cancer there is frequently a tumor in the right lower quadrant, between the umbilicus and the anterior superior spine of the ilium, or between the latter and the ribs. The diagnosis is based on the gradual development of the tumor; the history of colicky pain, vomiting, and constipation, possibly preceded by diarrhœa; and the presence of visible peristaltic movements. The abdomen is somewhat distended at the site of the irregular tumor, which is tender and dull on percussion. The disease is very rare, and is said by Fenwick to be more common in women between twenty and forty years of age.

Intussusception manifests its presence by a swelling in the right lower quadrant or to the right of the navel; the tumor is generally distinct, of the shape of the bowel, not very tender, and harder than the tumor of appendicular inflammation. The diagnosis from the latter is made by the difference in the character of the tumor, by the pain being colicky and recurring in paroxysms, by vomiting and constipation being more marked, and by the tenesmus and passage of blood and mucus from the bowel. The association of the last-named group of symptoms with a tumor and a constant desire to defecate is most characteristic of intussusception, If the intussusception is low down, a tumor may be detected within the rectum by digital exploration. There may be distinct hemorrhage or the passage of the invaginated portion of the bowel per rectum. Intussusception is the most frequent cause of intestinal obstruction in infants and young children, and occurs nearly twice as often in males as in females. Stercoraceous vomiting is not so common as in other forms of acute obstruction of the bowel.

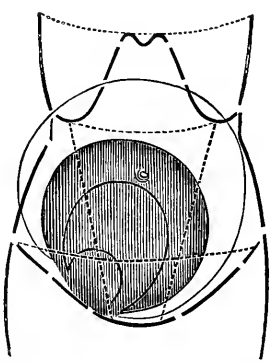
In *pelvic abscess* a swelling sometimes makes its appearance on the right side, above Poupart's ligament. The swelling is somewhat more median than in perityphlitic abscess, and it is not so sharply defined as the tumor of typhlitis; but the diagnosis from these affections must be made by the history, which is usually that of an antecedent salpingitis or of previous abortion or miscarriage. Vaginal examination discovers that palpation of the uterus causes pain; that the uterus is fixed, instead of being freely movable; and that the pelvis is blocked by an exudate on the affected side.

In *pelvic hæmatocele* a tumor may form in the lower half of one of the lower quadrants. The condition is distinguished from appendicitis, perityphlitic abscess, and pelvic abscess by the absence of fever and constitutional signs of suppuration; from perityphlitic and pelvic abscess by its sudden onset, usually at a menstrual period, by the fact that tenderness is less marked, and by the anæmia and collapse which follow its appearance. It is almost invariably the result of rupture during *extra-uterine pregnancy*, and may therefore be preceded by the passage of decidua and by the objective signs of pregnancy. Pelvic hæmatocele is

distinguished from abscess by its occurrence in a woman without antecedent tubal or uterine disease, and by relative absence of tenderness and fixation of the uterus.

Tumors of the right ovary at first produce a gradual enlargement in the right groin, unaccompanied by pain, fever, or impairment of health until the tumor has attained considerable size. As the tumors are usually cystic, fluctuation is present and the percussion-note is dull; by bimanual examination with the fingers of one hand in the vagina, the tumor can be traced into the broad ligament, and the uterine displacement which it occasions made out at the same time. Cystic ovarian tumors grow from the starting-point diagonally toward the median line. There are dulness

FIG. 186.



Position of an *ovarian tumor* of the right side in various stages of enlargement. The shading indicates the *percussion-dulness in ovarian dropsy of moderate extent;* the umbilical region is dull from the presence of fluid, and the flanks remain clear. The outer circle shows a farther extent which the dulness may reach in ovarian dropsy. (BRIGHT.)

in front of the abdomen and a clear percussion-note or tympany in the flanks. (Fig. 186.)

When the *left upper quadrant* or left hypochondrium is the seat of enlargement, one of the following conditions should be suspected: dilatation or carcinoma of the stomach; enlargement of the spleen (*q. v.*); movable kidney or a renal tumor; and effusion in the lesser peritoneal cavity.

The enlargement may also be due to *fecal accumulation* in the left transverse and descending colon, a condition that is recognized by the painlessness and doughy consistence of the tumor, and a history of constipation; although both patient and physician may be misled by apparent diarrhœa or even dysentery, with fluid or semifluid dejections mingled with scybala, and sometimes mucus and blood.

An interesting cause of swelling in this region and in the lumbar region is *perigastric* or *subdiaphragmatic abscess* (*q. v.*).

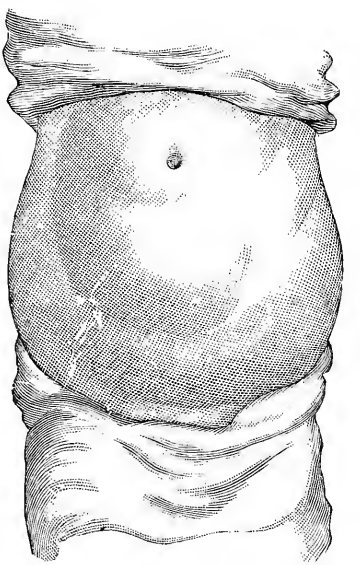
Enlargements in the *left lower quadrant* or left iliac region in women are usually due to ovarian tumors, pelvic abscess, pelvic hæmatocele, or uterine fibroid, the diagnostic points of which have been referred to under enlargements in the right iliac region. When these conditions can be excluded, an enlargement in this region may indicate fæcal accumulation or tumor—usually cancerous—in the sigmoid flexure or descend-

ing colon, volvulus, tuberculous peritonitis, or enlargement or displacement of the kidney or spleen (*q. v.*). Fecal abscess also may occur here, and the tumor of intussusception may be found on the left side.

Enlargement *about the centre of the abdomen* or umbilical region may be due to umbilical hernia; to gastroptosis, or a floating kidney, spleen, or liver; to cancer of the stomach, liver, or intestine, or sarcoma of the retroperitoneal glands; to hydatid disease of the liver or a tumor of the gall-bladder; or finally to tuberculous disease of the omentum or mesenteric glands (*tabes mesenterica*).

In *gastroptosis* (see Fig. 187) there is often an appearance that is quite characteristic—an elastic swelling suggesting the stomach by its shape, and presenting at or just below the umbilicus while the epigastrium is flattened. A similar appearance is seen in some cases of gastric dilatation and in retroperitoneal sarcoma. Enlargement of the

FIG. 187.



Stomach inflated, showing gastroptosis.

umbilical region associated with a more prominent swelling about the lower end of the sternum is observed in cancer of the stomach; in cancer or other disease—such as hydatid cyst—of the liver or gall-bladder the enlargement extends to the ribs on the right side; while effusions into the lesser peritoneal cavity and disease of the spleen and pancreas cause enlargement extending from the umbilicus to the left costal border. Undue projection of the vertebræ must not be mistaken for a tumor. *Tabes mesenterica* occurs nearly always in children and presents the physical signs and symptoms of tuberculous peritonitis, with the addition that enlarged mesenteric glands may sometimes be felt. The children grow pale and anæmic and waste away; they have apparently causeless attacks of diarrhœa productive of foul-smelling stools containing much

undigested food. The abdomen is large, and appears even larger than it is by contrast with the emaciated body; it is tender and its walls are thicker and less elastic than in health. Signs of tuberculous disease may be found in other organs of the body.

These different conditions must be distinguished one from the other by means of the various methods of physical diagnosis. The general principle upon which to proceed is to endeavor, by palpation and percussion, to discover the organ to which the tumor belongs. The length of time that the tumor has been known to exist must be ascertained by careful inquiry of the patient, as well as its effect upon the general health and upon the functions of the suspected organ or organs, particularly the occurrence of vomiting, constipation, diarrhœa, or jaundice.

Facts gathered in this way, carefully analyzed and then studied with reference to the physical properties of the tumor—whether it is hard or soft, fluctuating, doughy, or firm—will generally suffice for a provisional diagnosis. Often a positive diagnosis can not be made at the first examination, and sometimes it is possible only after watching the progress of the case for a considerable time.

Enlargement *above the pubis* or in the hypogastric region is due most frequently to pregnancy, fibroid tumor or fibrocyst of the uterus, or to distention of the bladder. Fibroid tumors occur most frequently in sterile women and are usually accompanied by hemorrhage. Bimanual examination of the uterus reveals an irregularity of the surface of the womb when the tumor is external, and the passage of a sound suffices to detect the presence of a growth projecting into the uterine cavity. The enlargement may be due to *a distended bladder*, and it is a good rule always to be sure that this viscus is empty before beginning an examination.

In *acute tuberculous peritonitis* a diffuse swelling of gradual development sometimes appears in this region; there is no tenderness, although on inquiry a history of antecedent pain and fever is obtained; the tension is increased, and the percussion-note is dull, but there is no palpable tumor. The general health is greatly impaired, loss of flesh is rapid, and diarrhœa and sweats are common. A tuberculous focus may be discovered in the lungs.

Enlargement above the pubis, associated with flattening of the upper half of the abdomen and abnormal distinctness of the lesser curvature, also occurs in *dilatation of the stomach* and *gastroptosis.*

Enlargement in the *flanks and lumbar regions* may accompany malignant tumors of the kidneys, hydronephrosis and pyonephrosis, perinephric abscess, and large renal cysts; although, as a rule, renal enlargements do not produce any visible change in the back. On the *left side* an enlargement may also be due to perigastric or subdiaphragmatic abscess, or to an enlarged or dislocated spleen. On the *right side* the cause may be an enlargement, as from hydatid cyst of the liver, or a retroperitoneal sarcoma.

Diminution in Size of the Abdomen—Retraction. The size of the abdomen is diminished in wasting diseases, and in diseases that either interfere with the ingestion or assimilation of food or destroy the appetite.

This class comprises cancer of the œsophagus and stomach, chronic lead-poisoning, anorexia nervosa, chronic diarrhœa, and tuberculosis of childhood. Retraction of the abdomen is characteristic of the second stage of tuberculous meningitis in children. Wasting of the subcutaneous and omental fat and atrophy of the abdominal organs cause the abdomen to become concave or scaphoid.

THE STOMACH.

Inspection.

Direct inspection of the epigastric region often affords much positive information. When there is much loss of abdominal fat and the stomach is well distended, its outlines can sometimes be traced with the eye, and the position and size of the viscus may be outlined by observing the shadow corresponding to the lower curvature which moves with respiration. A shadow corresponding to the lesser curvature is also seen in gastroptosis. (See Fig. 187.) The best position for the examiner is behind and above the patient's head while the latter is lying down. If the lower curvature can be traced considerably below the navel, the stomach is almost certainly dilated, and if at the same time there is a prominent swelling in the pyloric region, and the patient gives a history of progressive loss of weight and cachexia, the dilatation is probably due to cancer of the pylorus. A marked groove extending from the umbilicus to the ribs, about or to the left of the nipple-line, is seen in cases of dilatation with gastroptosis. It marks the position of the lesser curvature. The lower border is also marked by a groove extending in a curve from the pubis toward the first groove.

Peristaltic waves may be seen to move spontaneously from left to right, or after tapping the region, or applying an ether spray or faradism. When the pylorus is obstructed, antiperistaltic waves may also be seen. The waves of the muscular contraction begin at the cardiac end or fundus, and extend to the pylorus; hence they begin under the ribs of the left side and extend downward toward the right lower quadrant. They vary in extent with the degree of dilatation. (See page 508.)

Distention of the stomach with carbon dioxide (see Percussion), or better, with air by means of a hand-bulb syringe, frequently brings the outlines of tumors of the pylorus plainly into view, while at the same time any tumor lying behind the stomach becomes less distinct, and false tumors due to spasm of the gastric muscular coat vanish. Distention also helps to map out the entire organ, to separate it from surrounding viscera, and to estimate its size and position. It therefore affords a means of distinguishing gastroptosis from dilatation.

Gastrodiaphany or Transillumination of the Stomach. Einhorn has succeeded in transilluminating the stomach with an Edison lamp fastened to a soft-rubber tube. The wires to the battery are carried through the tube. After the stomach contents have been removed, the patient is to take one or two glassfuls of water, and the apparatus after lubrication is then inserted. The examination must be made in a dark room. By

means of gastrodiaphany the approximate position and size of the stomach and the presence of tumors of the anterior wall are determined. The results are not strictly accurate, however, as the intestines when empty are also illuminated. The form and size of the stomach are not so readily brought out as the topographic relation of tumors of the stomach and those in the vicinity of that organ. Gastrodiaphany is of service in some cases to distinguish dilatation from gastroptosis.

Rontgen Light. The outline of the stomach may be observed by the use of the *x*-ray, provided the patient has been given 10 or 20 grains of bismuth subnitrate.

Palpation.

Palpation of the stomach is closely associated with auscultation, inasmuch as the former also elicits sounds (succussion, gurgling) which are helpful in diagnosis. The hand must be placed flat upon the abdomen and pressure made by flexing the last phalanges. To make deep palpation, gradually increasing pressure with a rotary movement must be employed. It may be of advantage to palpate in the knee-elbow position, so that deeply seated tumors, if movable, may fall forward against the abdominal wall. (See Auscultation.) Palpation, however, elicits information independently of auscultation, chiefly in conditions of disease.

Epigastric pulsation is common in anæmia; in nervous dyspepsia; in valvular disease of the heart, particularly tricuspid regurgitation, which produces a liver-pulse; and in the rare case of aneurism of the abdominal aorta.

Increased resistance may be due to the hypertrophy of the muscular coat which coexists with distention of the stomach. A shrunken condition of the stomach with increased resistance may be due to a diffused carcinoma of the walls of the stomach; or, rarely, to the so-called "fibroid stomach,"—atrophy and thickening of the walls from chronic gastritis.

Increased resistance limited to the pylorus is found in *carcinoma*. The same effect produced by a tense right rectus muscle must be excluded.

Position of Gastric Tumors. Cancers of the pylorus are situated usually between the xiphoid cartilage and the umbilicus, frequently a little to the right of the median line; but they may be found below the umbilicus and, exceptionally, still lower down. Adhesions to neighboring organs commonly prevent the tumor from being moved. When it has formed adhesions to the liver or diaphragm, it moves freely with respiration; and even when there are no adhesions, pyloric tumors may move a little with respiration, and may often be displaced several inches laterally by manipulation or change of posture. Tumors of the lesser curvature show decided respiratory movement.

As a rule, tumors due to gastric cancer are small, hard, and irregular, and gradually increase in size.

Non-malignant tumors are found in rare instances, as well as tumors due to adhesions around old ulcers and to puckered scars. The latter are distinguished from cancerous tumors not by the physical examination, but by their duration and clinical history. Sometimes a fibroid pylorus may

be felt as a firm, cylindrical mass about the size of the terminal phalanx of the thumb.

Another method of determining the position and size of the stomach is by *internal exploration* combined with external palpation. The introduction of a bougie and determination of its position by palpation is dangerous and no longer used. A similar method, originated by Boas and free from danger, is the passage of a stomach-tube along the greater curvature and palpation of it in this position.

Tenderness. Tenderness is elicited by palpation in gastritis; in dyspepsia, especially the catarrhal form; in ulcer; and in cancer. In gastritis and dyspepsia the tenderness is usually diffuse and is not constant; in cancer the tenderness is usually limited to the seat of the tumor, but is not so marked or so sharply localized as in ulcer. In ulcer tenderness is rarely absent; even when there is no pain it is very decided, and sometimes is so sharply localized that the tender spot can be covered with the tip of the finger. Pain in the stomach from ulcer is chronic, circumscribed, and variously described as burning or wound-like. It is aggravated by palpation and by food or drink, especially hot stimulating drinks, and relieved by cold, soothing drinks. It is accompanied frequently by pain in the corresponding vertebræ.

Diffuse pain is met with in acute and chronic gastritis and in cancer of the stomach-walls.

Percussion.

Position of the Stomach. The stomach is a distensible organ, and does not occupy a fixed position. It is depressed by downward pressure of the diaphragm in deep inspiration, by emphysema, left pleural effusions, enlargements of the liver and spleen, and tight lacing; it is elevated by any causes that greatly distend the bowels or peritoneal cavity—tympanites, peritoneal effusions, tumors, and the like. Moreover, after food is taken the stomach is distended and its position changed, the organ being rotated anteriorly from below, the greater curvature rising and looking more forward, while the anterior surface has a more upward presentation. (Plate XL., Fig. 1.)

The *cardiac end* of the stomach is fixed to the diaphragm by peritoneal attachments; the cardiac orifice lies behind the sternal insertion of the left seventh rib. The *pylorus*, on the contrary, is freely movable when the stomach is empty; it is nearly in the median line, but when the stomach is full, it is pushed several inches to the right; it lies between the right sternal and parasternal lines, on a level with the tip of the xiphoid cartilage.

Obrastzow[1] divides the space between the navel and the xiphoid cartilage into three equal parts, and says that the *lower border* of the stomach, both in men and in women, is in the lower or supra-umbilical third.

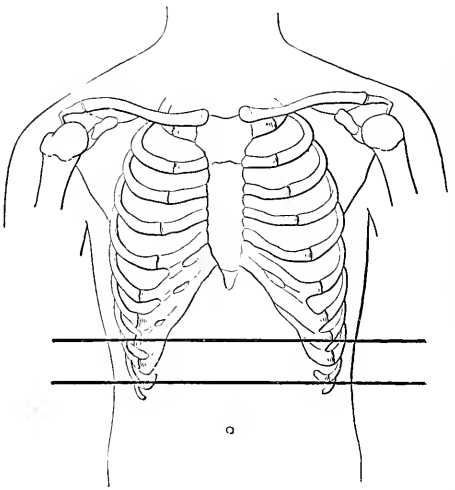
In children under fifteen years the lower border rarely extends to the umbilical line; after fifty years, on the contrary, it often extends below the navel. In conditions of bad nutrition it falls nearly to the navel.

According to Pacanowski and Wagner, the *upper border* of the stomach,

[1] Deutsch. Arch. für klin. Medicin., Bd. xliii. 5, 417–456.

in the left parasternal line, lies at the lower border of the fifth rib or in the fifth intercostal space, rarely at the fourth rib or in the sixth intercostal space. In the left nipple-line it lies between the fifth interspace

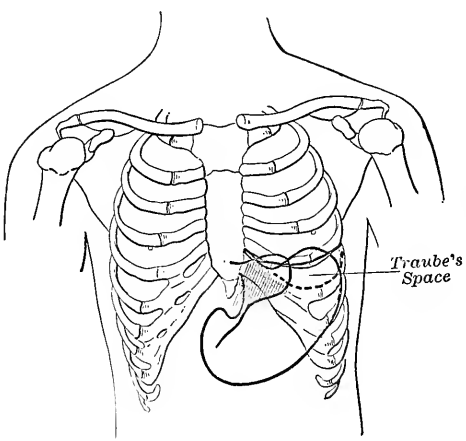
FIG. 188.



Obrastzow's lines: the greater curvature is in the lowest supra-umbilical third.

and the sixth rib, occasionally in the fourth interspace or at the seventh rib. In the anterior axillary line it lies at the lower border of the seventh or eighth rib, rarely above the sixth rib, never below the eighth rib.

FIG. 189.



Traube's semilunar space.

Traube has called special attention to the left lower portion of the thorax which projects over the stomach, the "semilunar space." The upper limit is a crescentic line starting from the sternum in the sixth interspace

and extending in a curved line, corresponding approximately to the curve of the rib, to the axillary line. It is known as "Traube's line." In health this space gives a tympanitic note, unless the stomach or transverse colon is full, or the omentum very fatty. In left pleural effusion it is dull. (See Diseases of Lungs.)

Area of Stomach Tympany. A part of the anterior portion of the stomach and its lower border can be determined by percussion. Ordinarily, the most suitable position for examining the stomach is the recumbent one, with the knees drawn up to relax the abdominal muscles. The stomach contains air at all times, but the amount varies greatly. The percussion-note is tympanitic, high in pitch, frequently with a metallic ring; its quality is peculiar—"stomach tympany."

In order to separate stomach, from colon tympany which resembles it, the stomach may be distended with gas while the colon contains solid or liquid matter; or, if the colon be filled with gas, the patient may be allowed to stand and drink a glass or two of water. In either case the contrast between a dull and a clear note marks the boundary between stomach and colon.

Ziemssen recommends carbon dioxide (developed by mixing sodium bicarbonate and tartaric acid) to distend the stomach; the quantity employed for adult men is 7 grammes ($1\frac{3}{4}$ drachms) of sodium bicarbonate and 6 grammes ($1\frac{1}{2}$ drachms) of tartaric acid. Adult women should receive 1 gramme less of each. As carbon dioxide sometimes causes an uncomfortable oppression, ordinary air is preferred by some. It can be forced in with a hand-bulb syringe attached to an ordinary stomach-tube. The percussion-note over tumors of the pylorus is imperfectly tympanitic. Welch describes it as "tympanitic dulness." Less frequently it is dull, and rarely it is flat.

The percussion-area of the stomach is *increased* (1) by causes external to the stomach: contraction of the liver, old pleurisy with retraction of lung, emphysema, former pregnancies, bad nutrition, and tumors pulling down the stomach; (2) by intrinsic causes as distention of the stomach. Conversely, the percussion-area is *diminished* by causes external to the stomach: enlargement of the liver and spleen, left-sided pleural effusion, pneumothorax, and hypertrophy of the heart.

Actual *diminution in size* of the stomach itself is difficult to demonstrate clinically with certainty. If upon inflation the greater curvature remains at a higher level than 3 to 5 centimetres above the umbilicus, diminution in size is highly probable; but even then the lower border may be prevented from descending by adhesions to surrounding viscera.

Enlargement of the stomach is generally due to *dilatation*, and is marked clinically by a low position of the greater curvature. Dilatation of the stomach, according to Boas, can be differentiated from descent of the organ only when the greater curvature is more or less below the level of the navel, and when the greatest height of the stomach exceeds 10 to 14 centimetres (4 to $5\frac{1}{2}$ inches). But descent and dilatation are frequently present together. (Plate XL., Fig. 1.) It must not be forgotten that when there is descent, the normal tympany is lowered and the tympanitic area above the ribs is replaced by dulness.

Sometimes when the stomach is distended with air, the right margin will be seen to extend far beyond the ordinary limits. Michaelis points out that this may be due to defective motor power, especially if the right margin is more than 9 centimetres from the median line. The distention to the right is due to actual enlargement and not to dislocation. The author believes that dilatation of the antrum of the pylorus causes this enlargement. Enlargement of the stomach downward is usually associated with good motor power, whereas enlargement to the right is an indication of feeble motor power.

Auscultatory percussion is a most satisfactory method of determining the borders of the stomach and its size. The gastric area can readily be defined from that of the liver, spleen, and colon: first, with the stomach normal; second, inflated with gas; third, filled with fluid. It is well to determine the results in the recumbent posture, and then in the upright, so as to determine if the stomach falls from its normal position. Liquid may be injected through the stomach-tube, or the patient may drink successive portions, percussion being employed after each tumblerful (eight ounces) taken. After the site of the dulness has been determined, the patient should lie down, when the fluid falls backward and the air in the stomach comes to occupy an anterior position, so that the dull note is replaced by a tympany. The change is a sign that the fluid is in the stomach, and serves to distinguish stomach from colon tympany. The force used in percussion should be very light; indeed, a fillip with the nail is sometimes sufficient. It may even be well to allow the blow to glance from the surface, as the perpendicular stroke brings out the general abdominal resonance. The use of coins is sometimes of advantage. In dilatation of the stomach the percussion-note sometimes varies in tone over the viscus from dull to tympanitic, or *vice versa*, because the organ contracts under the influence of the blows. Some have described a clinking percussion-sound, not unlike that of the "cracked pot" over the thorax.

Auscultatory friction is also employed in the same manner as auscultatory percussion, the finger-tips being rubbed lightly over the surface. As long as the rubbing is made over the hollow organ over which the stethoscope is placed, and not moved more than 2 inches from it, the friction is heard distinctly. This method is very unreliable.

Auscultation.

By means of auscultation it can be determined whether or not there is obstruction at the cardiac orifice. On listening over the œsophagus with the stethoscope, while the patient is swallowing a liquid, a spurting sound is heard, followed in from five to ten or twelve seconds by a second sound, which marks the escape of the fluid from the cardiac orifice of the œsophagus into the stomach, so-called "deglutition-murmur." When there is obstruction at the cardiac orifice, the second sound may be delayed as long as a minute.

When the stomach is partly filled with fluid, a *succussion* or *splashing* sound can be produced by moving the patient quickly from side to side, or by quickly compressing the stomach and allowing it to rebound

immediately. Such compression may be made alternately, first in the neighborhood of the fundus of the stomach and then in the region of the umbilicus. Both hands should be employed. The splashing sounds are also developed by rapidly tapping, with the finger-tips held perpendicularly, the region between the ribs and the transverse umbilical line on the left side. The ear need not be applied to the body while the movements are made, as the sound is audible at a distance of several inches. Such sounds are abnormal if they are heard long after digestion should be completed and the stomach empty. If they are heard more than three hours after a light, or six hours after a full meal, they indicate slow digestion or deficient motility, and afford a means of determining the approximate position of the lower boundary of the stomach. Normally, after the drinking of fluids, a splashing sound is not developed lower than the umbilical line. If it is heard below this line, it is an indication of dilatation or of deep position of the whole stomach. Dilatation is very probable if the splashing sound is heard below the navel in a fasting stomach. A good idea of the extent and location of the splashing, and hence of the lower boundary, can be obtained by using the stethoscope after the stomach has been inflated. This sound is also a sign of atony. After the patient has swallowed from 50 to 100 cubic centimetres of water, no splashing sound is heard unless there is atony of the stomach-walls; but, if the atony is pronounced, a smaller quantity will be sufficient to develop the sound. It is to be remembered that the splashing sound of itself does not indicate disease. It is significant only when taken with other signs, and when it is found after more than one examination. It is unreliable at best, as fluid in the colon readily causes error.

THE INTESTINES.

Inspection.

Local and general enlargements of the abdomen have been discussed in the preceding pages. Movements due to increased peristalsis of the intestines are seen in obstruction. The intestine above the point of obstruction may swell into a well-defined tumor which becomes hard and dull tympanitic on percussion.

Palpation.

Tenderness, peristalsis, peritoneal friction, the bubbling of gas through a constriction of the bowel, and tumors are recognized by palpation. It is often necessary to place the patient on all-fours or in the knee-chest position.

Percussion.

The normal note is tympanitic. Local areas of dulness may be due to intestinal tumor. Light percussion should be employed. A dull tympany indicates a solid mass surrounded by the distended intestines. The outline of the large intestine can be ascertained by filling it with water.

THE LIVER.

Topographical Anatomy. (See Plates V., VI., and VII.) The right lobe of the liver is applied to the concavity formed by the lower lobe of the right lung, being separated from it by the diaphragm. The thin lower edge of the right lung overlaps the liver at its upper part, but the greater portion of the anterior surface of the right lobe of the liver is in contact with the ribs. The under surface of the liver is in relation with the stomach, transverse colon, duodenum, right kidney, and right suprarenal capsule. "The highest part of its convexity on the right side is about one inch below the nipple, or nearly on a level with the external and inferior angle of the pectoralis major. Posteriorly the liver comes to the surface below the base of the right lung, about the level of the tenth dorsal spine." (Holden.)

A needle thrust into the right side, in the axillary region, between the sixth and seventh ribs, would traverse the lung, and then go through the diaphragm at its central attachment, into the liver. The lower border of the liver extends in the median line one-third of the distance from the tip of the xiphoid cartilage to the umbilicus. In the right mammary line it extends to the lower border of the ribs; and in the midaxillary line to the tenth rib. The upper border is opposite the upper border of the sixth rib in the mammary line, and extends horizontally to the axilla to the ninth rib behind.

The attachments of the liver permit of a certain amount of movement. Hence, the liver can be depressed by deep inspiration, emphysema of the lungs, or right pleural effusion. When the patient lies upon his left side, the left lobe of the liver rises higher and the right extends lower; and *vice versa*, when the patient lies upon the right side, the liver turning upon the suspensory ligament as an axis. (Gerhardt.)

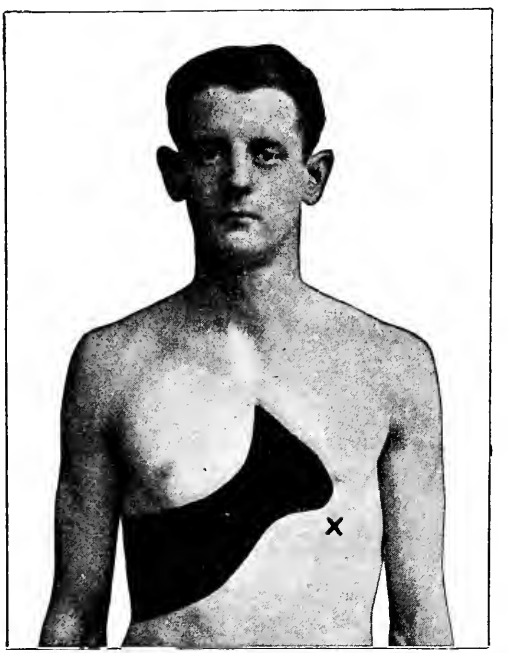
Inspection.

Inspection is not of very great assistance in the diagnosis of diseases of the liver. Frequently there is a *swelling* or *tumor* in the right upper quadrant, which may or may not be produced by an enlargement of the liver, but which should direct attention to that organ. The lower right zone of the thorax may also be distinctly prominent. Such a swelling may be observed in amyloid disease, hydatid tumor, cancer, abscess, and less frequently in fatty liver. In amyloid and fatty livers the projection in the right upper quadrant, which may extend to the left beyond the median line, presents a smooth surface, whereas in hydatid disease there is frequently a rounded projection at some part of the prominent area, and, in cancer, several nodules may be sufficiently large to cause slight rounded projections which the eye is more apt to detect after the sense of touch has first directed attention to their presence. Enlargement and occasionally pulsation of the superficial abdominal veins are accompaniments of cirrhosis.

Palpation.

By palpation the lower border of the liver can be determined in thin subjects, or in those in whom the liver is greatly enlarged. It may be difficult to determine the border when the abdomen is distended with gas. Careful palpation must be made with the tips of the fingers, pressing them firmly inward along the margin of the ribs, at the same time procuring relaxation of the abdominal muscles by having the patient take a full breath, and having the legs drawn up and the shoulders elevated. The pressure should be made in the interval following the acts of inspiration. By care and patience the fingers can be pushed deeply inward and be made to feel the border of the liver, even in health. Care must be taken not to cause contraction of the right rectus muscle, for if this takes place, the

FIG. 190.



Showing at *x* mark the so-called area called Traube's semilunar space, where in health percussion gives a tympanitic note, which becomes flat in left-sided pleural effusion. The solid block represents hepatic and cardiac dulness.

indurated mass may simulate tumor or enlargement of the liver. The left lobe of the liver, below the ensiform cartilage, extends half-way to the umbilicus. Here it is most accessible to palpation. By palpation we also determine the size of the gall-bladder and the degree of movement of the liver during respiration. On full inspiration the liver descends, and during the act of expiration rises again. This movability is of service in distinguishing the liver from other organs that are fixed within the abdomen.

In *amyloid disease* the lower edge is smooth and rounded, the tissue

dense and unyielding to pressure, and the anterior surface, as a rule, is perfectly smooth, but when the liver is also cirrhotic or syphilitic, the surface may be irregular and fissured.[1]

The *fatty liver* also has a rounded smooth border; but its tissue is not so dense and resistant, except when cirrhosis coexists. The surface of the organ is smooth.

In *single abscess* the liver is enlarged, but not uniformly and not invariably. When the abscess is located in the right lobe and nearer the anterior than the posterior surface, palpation may be able to detect not only enlargement, but also an area of deep-seated obscure fluctuation, surrounded by a zone of hard tissue. The tumor is round, smooth, tense, tender, and painful. (See Plate XLII.)

In *multiple abscess* the liver is enlarged uniformly, and usually none of the abscesses is large enough to be felt as a distinct prominence. The liver is tender and painful.

In *hydatid disease* the degree of enlargement depends very much upon the situation of the cyst, upon its stage of development, and upon the activity of the echinococci. Sometimes the cyst is so small that its existence remains unsuspected; at other times the enlargement is so great as to fill the abdominal cavity. As in the case of abscess, the possibility of detecting the tense, globular, fluctuating, painless tumor characteristic of the disease depends upon its situation. When it is on the anterior surface or lower border, it is easily detected, especially if the tumor is at all large; but when it projects from the posterior surface or from the upper or lateral borders, detection is difficult and may be impossible.

In *congestion of the liver* the enlargement is not so great as in abscess, nor are pain and tenderness so pronounced. Moreover, the enlargement is usually not permanent. The lower border, which may project below the edge of the ribs, is smooth.

In *hypertrophic cirrhosis* the enlargement is moderate, the surface smooth or but slightly roughened, denser than normal, and somewhat tender. (See Plate XLII.)

In *cancer* the enlargement resembles that of single abscess and hydatid tumor in that it is irregular. But, unlike hydatid tumor, the irregularities are due to knobs or bosses which project from the surface of the liver, are usually entirely free from fluctuation, and are tender on palpation. There may be a single large mass, or a number of knobs or nodules. The part projecting below the ribs may be free from nodules.

Palpation of the liver may discover a *friction* from perihepatitis, and *pain* or *tenderness* from that cause or from cancer or abscess. *Pulsation* of the liver may be a transmitted impulse from the abdominal aorta or a venous pulse, such as occurs also in the jugulars from tricuspid regurgitation.

Floating liver is diagnosticated by feeling in the lower, most frequently the right portion of the belly, a large tumor which may however easily be confounded with tumors of other organs. It can be distinguished as liver: (1) by recognizing the notch; (2) by the presence of a tympanitic note in the normal hepatic region, as loops of intestine lie between the

[1] Musser. "Amyloid Disease of Liver," Penna. State Medical Journal, 1899.

diaphragm and liver; (3) by the excessive movability of the tumor; and (4) by the fact that it is possible to replace the liver; (5) by its size and consistency. It occurs almost exclusively in women, possibly as the result of a congenital lengthening of the suspensory ligament, although more likely from relaxed abdominal walls. It may be confounded with ovarian cyst, appendicitis with tumor, and movable right kidney with hydronephrosis.

Glénard's method, or *procédé du pouce*, sometimes enables the examiner to feel the edge of the liver when ordinary methods of palpation fail to reveal it. The method consists essentially in thrusting the liver forward with the fingers of the left hand, while with the thumb in abduction the

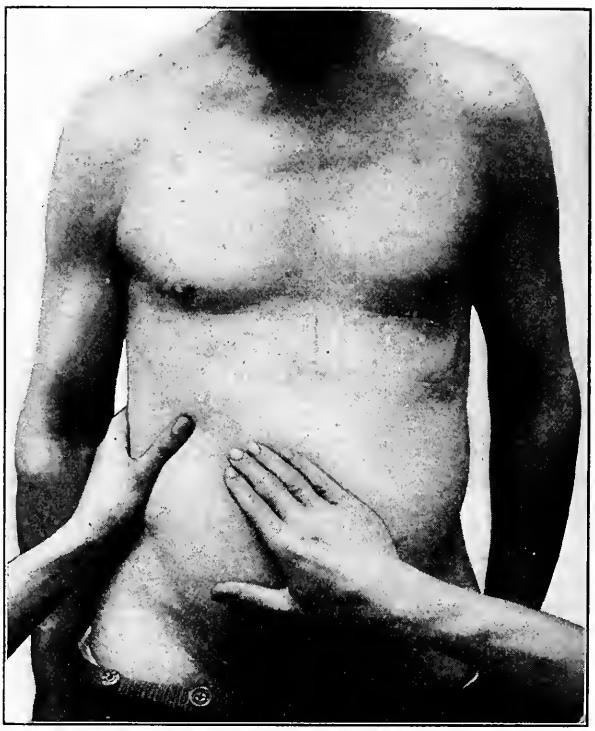
FIG. 191.

Glénard's procédé du pouce.

edge of the liver is felt for, the right hand meanwhile forcing the intestines out of the way. The examiner sits on the right side of the bed facing the patient, who lies on his back with the legs extended and the shoulders slightly raised on a bolster. The liver is pressed forward, with the fingers of the left hand applied to the lumbar region, while the thumb of the same hand in abduction remains free to palpate the anterior surface of the body. The right hand in extreme adduction is then applied to the abdomen and, while deep pressure is made for the purpose of forcing the coils of intestine up under the liver, the hand is rotated to a transverse

position on the abdomen. (See Figs. 191 and 192.) The patient now takes a deep breath, and at the same time the ball of the left thumb glides upward and outward and from behind forward. The last step is repeated with the thumb at successively higher levels until the edge of the liver is felt.

FIG. 192.



Glénard's procédé du pouce.

Constriction of the liver from tight lacing (Schnürleber) occurs chiefly in women. Tight corsets, and, still more, tight waist-bands, force the liver downward, especially the right lobe, so that it can be palpated. In more pronounced cases a furrow, often palpable, is produced, and below this a constricted lobe which may extend as far down as the anterior superior spine of the ilium and carry the gall-bladder with it. In other instances the right lobe is elongated, extending even to the crest of the ilium.[1]

Lobes so depressed are usually thin and easily movable, and can be grasped with the hand and moved to and fro. If the lobe does not reach so far downward, it is more rounded and blunt in shape. It is not always easy to demonstrate its connection with the liver, because coils of intestine overlying the liver in the furrow make palpation difficult, and cause a tympanitic note between the liver-dulness and the dulness of the constricted lobe.

[1] Musser. Transactions Philadelphia Pathological Society, vol. x.

Confusion with tumors of other kinds can usually be avoided by deep palpation or percussion.

Gall-bladder. When the gall-bladder has a certain degree of fulness, it may, according to Gerhardt, be not only felt in healthy persons, if the stomach and bowel are empty, as a smooth, round, fluctuating tumor at the lower border of the liver, but be even visible and be outlined by percussion. If a line is drawn from the right acromion process to the umbilicus, it will bisect the gall-bladder at the point where it passes over the margin of the ribs. The fundus is situated below the edge of the liver, at about the ninth costal cartilage, just outside the edge of the right rectus muscle. Palpation is easy when, owing to closure of the cystic duct, the gall-bladder is distended with bile or inflammatory exudate, or enlarged by thickening of its walls or an accumulation of gallstones. A pear-shaped tumor is then felt which, if not adherent to the border of the liver, is movable with it. In simple stasis—*hydrops vesicæ fellеæ*—and in purulent inflammation the tumor is tense and elastic; in inflammatory or carcinomatous thickening of the wall, dense and irregular. Calculi can often be recognized by their form or hardness, or by the sound made by rubbing them together.

Percussion.

Limits of Liver-dulness in Health. The upper border of the liver-dulness is found by percussing from above downward toward the liver, beginning anteriorly at the third interspace, on the lateral surface of the chest at the fourth, and posteriorly at the angle of the scapula. In health the upper border is found anteriorly at the fifth interspace, in the axilla at the sixth, and in the back at the ninth interspace; and the liver-dulness should extend downward to the margin of the ribs. In the aged the lower border falls short of this position by at least an inch, and in deep-chested persons the entire liver-dulness may be not more than two inches wide in front. The width of the liver-dulness is about 4 inches in the right midclavicular line, 6 inches in the midaxillary line, and 3 inches in the midscapular line.

In children the lower border of the liver is normally lower than in adults on account of the relatively greater size of the viscus. For the same reason the upper border is at a higher level.

Diminution in size can be determined by percussion only, and is usually best recognized in the anterior and lateral regions. Diminution in size is due to simple or acute yellow *atrophy* of the liver or to *cirrhosis*. It must not be confounded with the apparent diminution that takes place in emphysema, or that which occurs from distention of the bowels with flatus, as in peritonitis. Absence of hepatic dulness may occur when there is gas in the peritoneal cavity. When there is much distention of the intestines by gas, the anterior and lateral hepatic areas may be tympanitic.

Enlargement of the Liver. In order to detect enlargement of the liver, inspection, palpation, and percussion—both superficial and deep—

are employed; palpatory percussion is sometimes of great advantage. Any marked increase in the hepatic dulness beyond the normal limits (see page 526) usually means a corresponding increase in the size of the organ. The enlargement may be uniform and the shape of the liver-dulness normal, or the outline may be irregular, or again the enlargement may be limited to one lobe. If the enlargement is irregular, the liver-dulness may begin at a higher point in the anterior than in the lateral region, or may extend beyond the margin of the ribs in a limited area.

When the enlargement is limited to the left lobe, it is revealed by increase in the dulness from the xiphoid cartilage downward as far as the umbilicus. The entire middle region to the navel may be filled by the enlarged liver.

Uniform enlargement of the liver is due to congestion, hypertrophic cirrhosis, fatty degeneration, amyloid disease, leukæmia, cancer, and sometimes to hydatid disease and abscess.

Enlargement of one lobe of the liver is due to hydatid disease, to abscess, or to cancer, in nearly all cases. Either the right or the left lobe may be the seat of such enlargement.

Enlargement in one direction is due also to the three conditions just indicated. Although in abscess or hydatid disease enlargement *downward* is more common, it may be directly upward, the lower border of the liver occupying the normal position. When enlargement of the liver extends *upward*, it is due to a cyst or an abscess in the convex surface of the right lobe.

Irregularity in the shape of the liver-dulness occurs in cancer, in abscess, in corset liver, and hydatid disease. Notwithstanding the apparent irregularity, enlargements of the liver, usually with the exception of corset liver, conform to its usual outline, with but moderate variations, and always occupy the *normal site* of the organ.

Diagnosis. Enlargement of the liver must be distinguished from enlargement of organs in contiguity with the liver, and from structures usually containing air which have become solid or non-resonant. The enlargement must therefore be distinguished from pleural effusion, from disease of the lungs causing dulness on percussion, or from disease of the abdominal organs causing increased dulness near the hepatic region. Hence, in renal tumors, in tumors of the large intestine or stomach, in ovarian tumors, in tumors due to accumulation of feces, the physical signs on percussion may simulate enlargement of the liver. It is well to bear in mind the conditions which *simulate enlargement of the liver*. Of these we have:

Congenital Malformation. The liver may be of abnormal shape, and the area of dulness increased in a particular direction; it may be quadrangular or rounded. The liver may be found in the right pleural sac in congenital diaphragmatic hernia, and in that case the increase of dulness upward will simulate enlargement of the liver. Congenital malformations may be suspected in the absence of any symptoms of hepatic disease or of conditions that may cause other forms of spurious enlargement. Moreover, the increased dulness will have existed from early life.

Rhachitis. Owing to the malformation of the chest the position of the liver may be such that the hepatic dulness will appear to be increased. For the same reason the liver may be felt below the margin of the ribs.

Disease of the spinal column causes dislocation, on account of which the liver may apparently be increased in size.

Pleural Effusion. The distinction is sometimes difficult. The symptoms of the pulmonary affection must be considered. General conditions that cause hydrothorax must be borne in mind. The difficulty in distinguishing enlargement of the liver from a pleural effusion arises because the hepatic dulness is continuous with that caused by the presence of fluid in the pleural sac. In pleural effusion, however, there is uniform bulging of the affected side; the liver is not movable, and chest-expansion is lessened. The upper border of dulness may be movable if the effusion is not large, and the line of dulness is S-shaped—that is, high behind and high in front. If the effusion is large, the upper limit of dulness is horizontal. In enlargement of the liver the lower ribs are often everted; while in pleural effusion a depression may be seen between the lower margin of the ribs and the upper surface of the liver, if the latter is dislocated by pressure of the fluid. Sometimes an enlargement of the liver may give rise to secondary pleural effusion, so that too often, after finding pleural effusion, the size of the liver is not estimated.

Pericardial effusion and dilatation of the heart are said to simulate enlargement of the liver. The history of the case, the origin and mode of development of the symptoms, and the physical signs of cardiac disease, point to the true nature of the lesion.

Subdiaphragmatic Abscess. The history of the case is generally essential to a diagnosis. The accumulation between the liver and diaphragm causes the latter to be pushed downward. It is very difficult to distinguish the true from the false enlargement in these instances. *Aspiration* may help in the diagnosis.

Abnormal Condition of the Abdominal Parietes. Increased tension or spasm of the recti muscles, giving rise to phantom tumors of the abdomen, simulate enlargement of the liver. They occur in young girls, and are associated with gastro-intestinal catarrh and symptoms of hysteria. Anæsthesia must often be employed to disperse the swelling.

Tight Lacing. This may displace the liver upward or downward, according to the direction of the pressure. It may also, by exerting lateral compression, bring more of the liver into contact with the anterior abdominal wall; and finally, if the constriction is due to pressure of a strap or tight cord, a portion of the liver may be more or less detached and appear as a movable tumor.

Enlargement of the abdominal contents may cause spurious enlargement of the liver. In the same way increased abdominal pressure from ascites or tympanites causes the liver to rise higher than normal.

a. The *accumulation of feces* in the colon. This causes extension of the liver-dulness downward, on account of which it may be thought that the patient has liver disease. A purgative must be given.

b. An *ovarian cyst.*

c. The presence of *ascites*. Exclusion of the latter is sometimes difficult, because the ascites may be loculated and situated in the hepatic region. It may give rise to symptoms of hepatic enlargement. Probably aspiration alone can establish the diagnosis. Ordinary ascites should be easily distinguished by the physical signs and the result of exploratory puncture.

d. Tumors of the omentum, chiefly tuberculous, may occupy such relation to the liver as to increase the dulness downward. The history, the occurrence of the omental tumor, with symptoms of tuberculosis, may aid in determining the condition.

e. In *tumors of the kidney*, which simulate enlarged liver, it is found that the edge of the liver can not well be felt; but Murchison thinks the fingers can usually be inserted between the ribs and the upper part of the renal tumor. The renal tumor, however, is not fixed. It is rounded on every side; and has the well-known shape of a kidney. Renal tumor may be associated with changes in the urine.

f. Enlargements of the liver must be distinguished from *pancreatic cyst*, or *effusion into the lesser peritoneal cavity*. This can usually be accomplished with ease, except in hydatid disease of the left lobe near the suspensory ligament. In effusion into the lesser peritoneal cavity the tumor occupies the left upper quadrant, and may extend downward as far as the transverse umbilical line. It causes dislocation of the heart, so that the apex is as high as the third interspace and beyond the mid-clavicular line. It is accompanied by an increase in the dulness posteriorly, so that the upper limit may extend to the angle of the left scapula. Puncture may furnish the necessary information.

Pain. The presence or absence of pain may sometimes furnish a clue to the nature of the enlargement of the liver. Murchison considers this a reliable distinction. *Painless enlargements* of the liver are due to passive congestion, to hydatid disease, to leukæmia, to fatty and amyloid disease of the liver. *Painful enlargements* of the liver are seen in abscess, cancer, and syphilitic disease with perihepatitis, and sometimes in great passive congestion.

Aspiration.

We are warranted in determining the nature of an obscure enlargement of the liver or of the gall-bladder by aspiration. In abscess, pus is found; in hydatid disease, the characteristic fluid may be withdrawn.

In a case of local enlargement the apex of the swelling should be aspirated. If puncture is performed near the upper border, the needle should be thrust downward; if near the lower border, upward. The left lobe should be aspirated with care, in order to avoid entering the stomach. (See Aspiration in Diagnosis.)

Auscultation.

By auscultation we may detect a *friction-sound* in perihepatitis; a *grating* or *rubbing* during palpation when the gall-bladder contains calculi; a continuous murmur in tricuspid regurgitation.

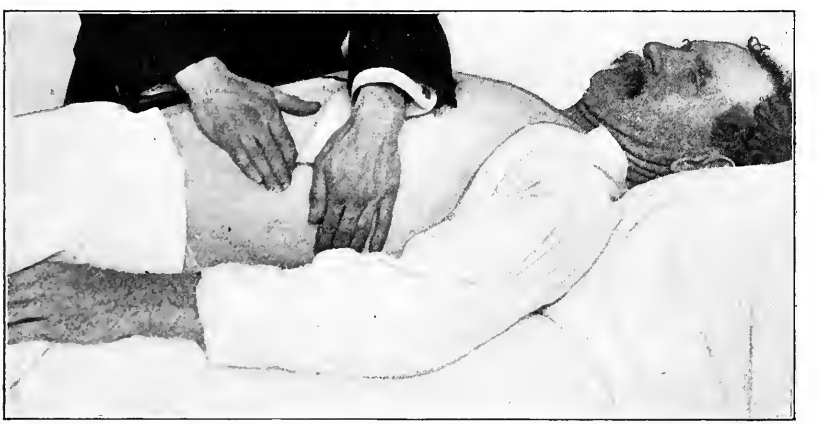
THE SPLEEN.

Topography of the Spleen. (Plate VII.) The spleen lies in the left upper quadrant, beneath and in contact with the diaphragm above, and with the tail of the pancreas, the cardiac end of the stomach, and the suprarenal capsule below. It extends from the upper border of the ninth to the lower border of the eleventh rib, and from the mid-axillary line backward toward the spine.

Palpation.

As the spleen lies entirely behind the ribs, it does not, under normal conditions, admit of palpation; but when it is *enlarged*, it becomes accessible to palpation in proportion to the degree of enlargement and of the relaxation of the abdominal walls. It *moves with respiration* and can not be said to be enlarged unless the edge of the organ is palpable at the end of a deep inspiration, even if there is increased dulness in the lower axillary region. An enlarged spleen usually retains its normal *shape*, and the direction of the enlargement is *downward* and *inward*. When the enlargement is moderate, the smooth, blunt, rounded anterior surface and

FIG. 193.

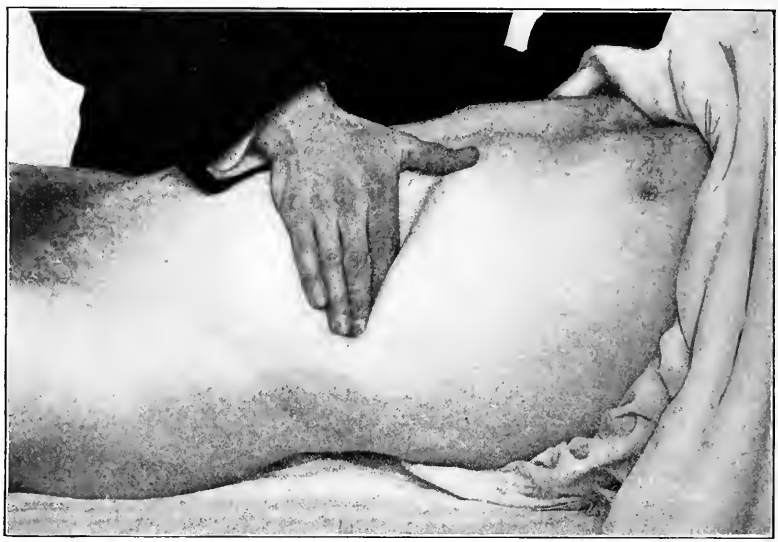


Feeling for the edge of the spleen.

sharp edge of the spleen can be felt at the margin of the ribs during deep inspiration; when the enlargement is great, as in *leukæmia*, the organ can be grasped with both hands and its hilus clearly mapped out. The same thing can be done in the rare instances of *floating spleen*, but here a knee-chest position will favor successful palpation. The posterior border of an enlarged spleen can usually be made out by passing the hand backward over the resisting organ; in children this border is always readily found. In fact, the spleen is always more readily palpable in children than in adults; it is also more movable, and therefore can be brought forward more easily to the median line by bimanual palpation. Between the posterior border of an enlarged spleen and the mass of lumbar

muscle there is a non-resistant space which, as well as the direction of enlargement of the spleen, is due to the costocolic fold of peritoneum. (Jenner.) No such space exists in renal enlargement. In

FIG. 194.



Feeling for the edge of the spleen.

splenic leukæmia the spleen may be larger after a meal, yield a creaking fremitus on palpation and a murmur on auscultation, and may even pulsate.

The spleen may also *diminish* in size after diarrhœa or free hemorrhage.

Percussion.

Being a solid body, the spleen yields a *dull sound* on percussion, contrasting with pulmonary resonance above, intestinal tympany below, and stomach tympany anteriorly. Posteriorly and below, the splenic dulness merges into that of the lumbar region and kidney. The upper posterior portion is hidden behind the diaphragm and overlapping lung, and hence is not accessible to percussion. Practically, therefore, the *normal splenic dulness* extends between the ninth and eleventh ribs, in the middle and posterior axillary lines, the spleen being there in contact with the ribs. (Plate IV., Fig. 2.)

In percussion of the spleen the patient should lie on his right side. Beginning from above downward, the ribs are percussed gently until pulmonary resonance is replaced by dulness; then proceeding from the gastric area toward the axilla until tympany yields to dulness. In the same way, percussing from below upward, the line is reached where intestinal tympany gives way to dulness.

Splenic dulness may be encroached upon by the tympany due to the stomach or colon distended with gas; or its dulness may appear increased

through distention of the stomach and colon with solid matter, a left pleural effusion, or basal pneumonia. The spleen may also be pressed upward by ascites or by a large abdominal tumor, so that its normal dulness is much lessened.

If the ligament which holds it in place becomes relaxed, the spleen may become floating. According to Stintzing, a *floating spleen* is increased in density, is generally enlarged, and is recognized by its form (notch, etc.), by being movable to and fro, and by the absence of splenic dulness in the normal position, and its reappearance when the spleen is replaced.

THE KIDNEYS.

The data obtainable by observation are secured: 1. By physical examination of the kidney. 2. By an examination of the urine. 3. By catheterization of the ureters. 4. By a skiagraphic examination.

Topography of the Kidneys. (Plate V., Fig. 2.) The kidneys are situated in the right and left lumbar regions respectively, the left being a little higher than the right. They extend from the eleventh rib, or twelfth dorsal vertebra, to the third lumbar vertebra. The left kidney is in contact above the spleen, and the right with the liver.

The kidneys are enveloped in fat; their distance from the anterior surface of the abdomen renders them inaccessible to percussion from that direction, and the thick dorsal and lumbar tissues, coupled with the relation of the kidneys with the spleen and liver, which give a dull note on percussion, make it difficult to outline the kidneys from behind.

Palpation.

Palpation of the normal kidney is difficult. It can only be bimanual. Place the fingers of one hand below the last rib outside of the lumbar muscles—erector spinæ—and apply the other below the ribs in front. Firm, persistent pressure with the abdominal muscles relaxed, especially in thin subjects, will often enable the normal kidney to be felt.

Palpation of the kidney becomes easy when it is either enlarged or displaced. In the case of an enlarged kidney the patient should lie upon his back or be slightly turned to the opposite side; one hand is placed beneath the kidney and pressed upward, while the other is pressed firmly and steadily from above, or laterally toward the kidney. In this manner the kidney can be grasped between the two hands, its size estimated, and its physical characteristics as regards hardness, softness, fluctuation, and mobility determined. Enlargements are also detected by palpation of the abdomen. (See Palpation of the Abdomen.) The fact that the tumor moves a little with respiration aids in its detection; and if it is unusually movable the edge of the hand can be slipped above its upper end, by turning edgewise that border of the hand which is adjacent to the ribs. A renal tumor is usually 2 or 3 inches to either side of the median line, a little above the transverse umbilical line.

A very favorable position for palpating movable kidneys is that assumed by standing and leaning forward over a chair, with the trunk

supported by the hands resting on the seat of the chair. In this position the abdominal muscles are relaxed and the kidneys fall forward.

In the diagnosis of renal tumors, in general, it should be borne in mind that they are slightly *movable* with *respiration* unless adherent, as in malignant disease, abscess, and cysts. Unless too large they preserve their *reniform shape*, and press in front of them the ascending or descending *colon*. The position of the colon should therefore always be ascertained, and to this end it may be necessary to inflate it.

Percussion.

The best results are obtained by having the patient lie face downward, and placing a cushion under the belly so as to make the lumbar regions a little more prominent. Strong percussion is required, and an artificial plessor and pleximeter are to be preferred. Percussion should be conducted with a view to marking the angle which the liver-dulness and splenic dulness make with that of the kidney on the right and left side respectively. The kidneys extend below the lower lines of liver and splenic dulness, and laterally for a width not greater than 4 inches. The difficulties in the way of outlining the kidneys by percussion are greatly increased in persons with much flesh, or when the abdominal walls are waterlogged, as they are in ascites, and it is practically impossible under such circumstances to be sure of the boundaries of the kidneys. The colon must be emptied to yield trustworthy results.

Catheterization and Exploration of the Ureters.

Examination of the bladder, the ureters, and the pelvis of the kidney has been wonderfully advanced by the genius of Howard Kelly. The following instruments are required for the examination of the bladder: female catheter; urethral calibrator; a series of urethral dilators; a series of specula with obturators; common head-mirror, and a lamp, Argand burner, or electric light; long, delicate mouse-tooth forceps; suction-apparatus for completely emptying the bladder; ureteral searcher; ureteral catheter with a handle; small bran-bags for elevating the pelvis.

The procedure is as follows: empty the bladder; measure the external urinary meatus; dilate the urethra to 12 or 15 mm.; insert speculum of diameter of last dilator and remove obturator; elevate the hips of the patient about a foot above the level of the table; inspect with light; remove residual urine by suction or with cotton and mouse-tooth forceps.

For anæsthesia, a pledget of cotton saturated with a 5 per cent. solution of cocaine may be introduced seven minutes before dilatation. On removal of the obturator the bladder becomes distended with air. The bladder is viewed by turning the speculum; each ureteral orifice is brought into view by turning the speculum 30 degrees to one side or the other. Kelly says: "The orifice appears as a dimple or little pit, or in inflammatory cases as a round hole in a cushioned eminence; at other times as a V with the point directed outward; again, it may be scarcely

visible even to a trained eye, appearing as a fine crack in the mucosa, and occasionally is so obscure as to be recognized only by the jet of urine as it escapes, or by a slight difference in the color of the mucous membrane at that point. In rare cases it has the form of a truncated cone with gently sloping sides; this appearance is most apt to be developed in the knee-breast position. The bladder mucosa is usually of a slightly deeper rose color around the ureter, and in the presence of an inflammatory process it even appears deeply injected."

Catheterization of the Ureters. The catheters are sterilized; they are stiffened with a wire stylet. The orifice is exposed, and then the outer end of the catheter being held above the shoulder by an assistant, the conical end is introduced and pushed up the ureter, the stylet being removed at the same time. The speculum is removed and again introduced beside the first catheter. The other ureter is then catheterized; both catheters are properly labelled and allowed to drain into test-tubes plugged with sterilized cotton and fixed in a block of wood. By catheterization, aspiration, and exploration of the ureters with a bougie, the source of pyæmia anywhere from the urethral orifice to the renal pelvis can be found, renal calculi diagnosticated, strictures of the ureter located, hydronephrosis distinguished from soft malignant growths, and the functional value of each kidney determined.

Kelly suggests the following guide to the ureteral orifice: "A point is marked on the cystoscope $5\frac{1}{2}$ centimetres from the vesical end, and from this point two diverging lines are drawn toward the handle with an angle of 60 degrees between them. The speculum is introduced up to the point of the V, and turned to the right or left until one side of the V is in line with the axis of the body; then by elevating the endoscope until it touches the floor of the bladder the ureteral orifice will usually be found within the area covered by the orifice of the speculum."

By means of a searcher, or sound, the suspected orifice is further examined.

CHAPTER XXXVII.

THE RÖNTGEN RAY IN MEDICAL DIAGNOSIS.

THOSE who concern themselves with internal medicine do not as yet seem to appreciate the advantages of the Röntgen ray so fully as does the surgeon. There are many reasons for this difference of opinion, the most obvious one being that the conditions in which the medical man is obliged to depend upon its use for diagnosis are much more obscure, the skiagraphic or fluoroscopic image is correspondingly more difficult of interpretation, and there is more room for error than in surgical cases. Experience has not yet fully proved the utility of the *x*-ray in those cases which the clinician classes as obscure, for the images are equally as obscure and often misleading; confidence in one's results and the power of properly interpreting them comes only after considerable experience.

A child can operate an *x*-ray machine, and even take a picture, so simple is the mechanism; but the best diagnostician in the world will fail to read many a plate properly without a wide experience. Too much warning cannot be given against an entire dependence upon what some one else sees in a picture, nor should the physician allow himself to be influenced by a preconceived opinion to see what his conscience tells him he does not see; he should have sufficient experience with *x*-ray work, if he wishes to make any use of it at all, to observe the conditions as depicted upon the radiograph or in the fluoroscope. The skiagrapher should have some general knowledge of the patient's condition before setting to work with the *x*-ray. It is a mistake and likewise unfair to inform a skiagrapher that the patient has "something the matter with his chest," and expect him to find out the trouble. Some knowledge is required of what is to be looked for, and then every effort can be made to prove or disprove the existence of the lesion.

Armamentarium and Technic.

When the Röntgen rays are used for diagnostic purposes, the ray must first be produced and then made visible to the eye either directly through the fluoroscope or indirectly through the skiagraph. The armamentarium consists of the following parts: A source of electricity. An apparatus for transforming the available current into one suitable for use. The *x*-ray tube. The fluoroscope. Sensitive photographic plates.

The **source of electricity** may be a set of storage cells, the street current as supplied in towns for lighting by incandescent lamps with a voltage of 100 or slightly more, or the static machine. A *storage battery* as now made is bulky, heavy, and expensive; it may leak, has a limited period of usefulness, needs frequent recharging, and requires great care

and watchfulness. It is useless for continuous work in places where there is no available current for recharging. On the other hand, it is the best and most reliable source of electricity when the *x*-ray apparatus has to be transported to rural districts for temporary use. The *static machine* is not so efficient as the "coil," but is the only one that can be employed in localities not supplied with a street current.

The **coil** is a machine used to transform the electric light supply from a storage battery or street wires into a current of sufficient electromotive force to excite the *x*-ray tube. If the static machine is used, no such transforming apparatus is required, the connections being made directly with the tube. The coil consists of the following three essential parts :

1. An **interrupter** to make and break the current. The "break" being the most potent factor in creating a powerful induced current, a properly constructed interrupter should be so made as to cause the most rapid and frequent breaks possible, and in mechanical interrupters there is always a device with which to regulate these two factors.

2. The **condenser** is designed to eliminate as far as possible *self-induction*. This is one of the most important parts of the machine, and the one that is usually not efficient in the cheaper coils.

3. The **coil** itself, which consists of two parts, the *primary* through which the interrupted current passes to produce the induced current in the *secondary* coil, which is the current used to excite the tube.

The **x-ray tube** is a glass bulb in which exists a high vacuum. In the centre is placed the platinum anode or positive pole with connections on the outside, and in one end facing this is the kathode or negative pole, made of aluminium and concave in shape. The glass should be exceedingly thin and free from lead, otherwise the tube will not yield rays sufficient or powerful enough to accomplish the work. The poles are connected outside with the terminals of the generating apparatus, and the current passes through the vacuum space from the anode to the kathode. In a tube with a properly regulated vacuum there will be produced a beautiful and delicate yellowish-green luminosity, the shade of color being one of the indices in determining the quantity and quality of the rays. It is best to have the apparatus working in a moderately darkened room in order to observe the coloration properly, and for fluoroscopic work there should be some means of shutting out all light. The focal point of the aluminium kathode is on some part of the platinum anode, and from this as a central point the *x*-ray emanates in a hemispherical field everywhere in front of the platinum. There is always a tendency while at work or at rest for the vacuum of the tube to become too high for the transmission of the current; hence modern tubes are made with some vacuum regulating attachment, and all but automatic self-regulating tubes are now out of date. At present, the best tubes have an accessory glass bulb attached, so that when the vacuum becomes too high the current passes by a spark gap through the accessory regulating tube and acts there upon some chemical substance, liberating gases that pass into the main bulb and reduce the vacuum. The hardness of the tube can be fairly well controlled by varying the width of the spark gap.

X-ray tubes are divided into two classes,—the *hard* or *high vacuum* and the *soft* or *low vacuum tube*. The former requires a current of higher electromotive force to excite it, but suffers less harm thereby than would a soft tube, and at the same time, by using such a current, more penetrating rays are produced for the work, and consequently shorter exposures suffice. In the use of the fluoroscope it must be borne in mind that the rays may be so penetrating as to pass through the object to be observed, without causing much shadow effect. Therefore in such work the state of the vacuum requires careful attention.

The **skiagraph** is the impression upon a sensitized photographic plate of the varying degrees of shadow cast by parts of the body of different density. The plates should be very sensitive and carefully prepared, and before being used are usually placed in two paper envelopes, the inner one black and the outer one yellow, thereby shielding them from the action of light.

The *length of exposure* required depends mainly upon the penetrative power of the ray, the part of the body and density of the tissues, and the distance of the plate from the tube. Experience alone determines all these points, and the operator must know his machine, tube, and plate as he knows his intimate friends.

The most important part of skiagraphy is the *development* of the plate, and here again it may be said that while almost any one can take a picture, the experienced skiagrapher alone can bring out by development what is required in it. An exposure of from one to ten minutes will "take" a picture, but the careful work of from thirty minutes to an hour may sometimes be required in developing the plate.

The Fluoroscope. This is a device by means of which the shadows cast by the object examined may be immediately seen and studied. The *x*-ray in itself makes no impression upon the human retina; but when it is brought in contact with certain substances it renders them luminous. The substances employed are calcium tungstate and barium platinocyanide, the latter giving the best results. The crystals are carefully spread over a piece of card-board, and when the room is darkened, the luminosity caused by the rays striking the screen can be plainly seen. Usually a hood is placed around the board so as to shut off the light from the observer's eyes. In this way the screen is brought into contact with the body to be examined, the latter being between the fluoroscope and the tube.

The fluoroscope has its *uses*, its *disadvantages*, and its *dangers*. It is most valuable to the surgeon for such purposes as determining the presence of a fracture, the degree of deformity, and the effect of the dressing applied during the course of treatment. To the medical man, the thorax is the region of its greatest usefulness, as will be mentioned under the uses of the *x*-ray in that part. The fluoroscope gives but a momentary picture of the condition under examination, while in the skiagraph we have a permanent record of the appearance at that particular time. Fluoroscopic examinations can be frequently made and take but little time; but no records remain save in the observer's mind or possibly on a chart, while the plate can always be used for reference.

Using the fluoroscope is to the operator the *most dangerous part of x-ray work.* His necessarily close proximity to the tube is certain, without great precaution, to cause serious, painful, and troublesome burns upon the hands and face; and conjunctivitis or more serious eye trouble may result. These dangers can however with care be almost entirely eliminated. The hood of the fluoroscope may be covered with some substance opaque to *x*-rays, such as white lead paint, or sheet lead, thus screening the face. The handle should have in front of it a sheet lead shield to protect the hand, and a piece of thick lead glass may be inserted in the hood to shield the eyes. For additional protection to the body, a sheet lead screen may be placed between the patient and the examiner. The person who constantly uses an *x*-ray apparatus should bear in mind that the effects of the ray are cumulative, so there should not be the slightest neglect in observing all these precautions, otherwise constant exposure will sooner or later make the operator a victim of such results as serious burns of the backs of the hands and fingers, cracking and brittleness of the nails, redness and eruptions on the face, injury to the eyes, and falling out of the hair.

The *protection of the patient,* likewise, must not be neglected, for by his close proximity to the tube during frequent and prolonged fluoroscopic examinations burns of the skin may be easily produced, especially if the case is used for purposes of demonstration.

A burn is very painful and persistent. Weeks or months are required to heal one of considerable severity, and usually an ugly scar is the final result. The physician should not only consider his patient's welfare, but should also realize the medico-legal aspect of the question; for the courts are not inclined to be lenient with a person who through carelessness allows a patient to be burned. Since rigid rules should therefore be observed in this kind of work, it may be safe to say that from *five to ten minutes* should be the *limit of exposure,* nor should a second examination be made for at least three or four days.

Uses of the Röntgen Ray in Internal Medicine.

The Röntgen ray will now be considered according to its usefulness as applied to the diagnosis of conditions in the various parts of the body.

The Head. The head is the most difficult region that the skiagrapher has to deal with, particularly as regards the discovery and localization of such lesions as tumors, cysts, or hemorrhages within the cranial cavity. This cavity is enclosed by a bony envelope with numerous irregularities, particularly in the base; sutures separate the various bones; there are grooves for the lodgement of vessels; density and thickness of the bony encasement change at every point; large venous sinuses are present; the external ear throws a distinct shadow; and there are many large air-cavities with thin walls. All these irregularities and anatomical structures tend to make the shadow of the head very complex. Then, too, it must be remembered that blood does not transmit the *x*-ray readily, and as the brain is a very vascular organ, the normal

shadows cast by a dry skull or by an abnormal structure must be very faint and indistinct. Many of the shadows of normal heads differ greatly in outline and density, yet there are a few that have a marked similarity in all individuals. A careful study should be made of skiagraphs of a large number of normal heads, the anatomy of the skull should be familiar, and the usual positions of abnormal growths known before the operator can expect to have any value placed upon his work in this line. If he has recourse to the cadaver, it will be well to make experimental pictures of heads after tumors or blood-clots have been placed inside the cranial cavity, so that the density and other appearances of such specimens may become known. Likewise, skiagraphs of heads in which the larger bloodvessels have been carefully injected would be valuable for references. A good picture of the normal head will show with a fair degree of clearness the external auditory meatus, auricle, petrous portion of the temporal bone, the three tables of the skull-cap, the frontal, ethmoidal, sphenoidal, and mastoid sinuses, the antrum of Highmore, outlines of the orbit, the coronal suture at least, often the grooves for the middle meningeal arteries, or perhaps, rather, the vessels themselves, the superior longitudinal sinus, the base of the skull, particularly in the region of the pituitary body, the scalp, and in the female the hair.

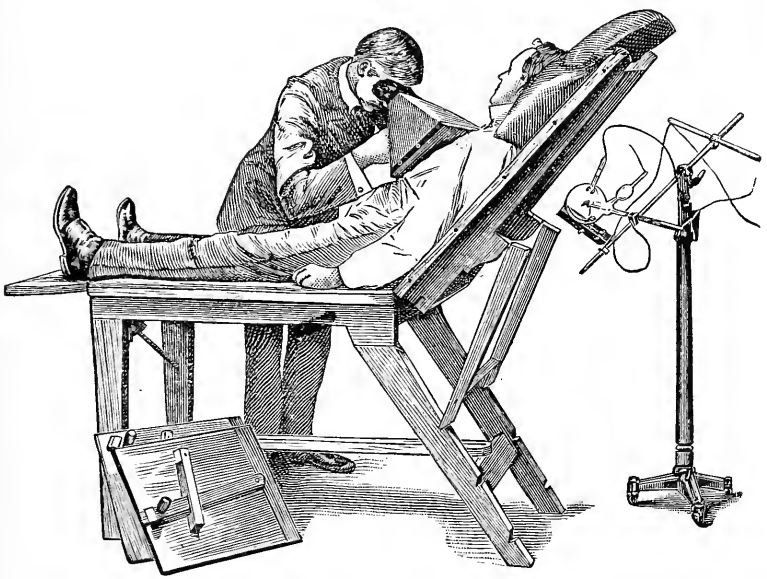
The results of skiagraphy of brain tumors, cysts, abscess, or blood-clot are, on the whole, of doubtful value for diagnostic purposes, but improved technic and increased experience may in the future make the *x*-ray of much more importance. A few men have been able to obtain shadows of suspected tumors and other lesions, and to confirm their findings by operation or autopsy. Such tumors as sarcomata, tuberculomata, gummata, and fibromata have been demonstrated in the skiagraph by skilful operators. Blood-clots being rather opaque when they are near the surface, as an extradural hemorrhage, are shown with greater ease and yield fairly clear shadows. A thick-walled abscess may be demonstrated. Cystic conditions are represented by a local diminution in the normal density of the shadow of the area, and the skiagraph may be of value in determining the diagnosis between the cyst, a clot, and a tumor. The nearer the lesion is to the side of the head and to the plate, the denser will be the shadow and the clearer its outline. The fluoroscope is of little or no value in this part of the body.

The Neck. The region of the neck, from a skiagraphic point of view, belongs almost entirely to the domain of surgery, except in the case of aneurisms of the large vessels, and in conditions in which it is difficult to draw a sharp line of distinction between the domains of two great branches of the profession, as in tumors.

The Thorax. In this part of the body the *x*-ray has perhaps the greatest field of usefulness as applied to medical diagnosis, and here some remarkable observations may be made by means of the fluoroscope, which is of more real value here than skiagraphy. The rays have little difficulty in passing through the lung tissue, and the heart, vessels, sternum, spine, and mediastinal structures are practically the only impediments. These same conditions make possible the study of cardiac

expansion and of the movements of the diaphragm—observations impossible on the skiagraph. Moreover, these examinations can be made practically as often as desired in the study of thoracic lesions, and take little time or trouble, whereas the two latter factors require the number of skiagraphs to be limited. By the fluoroscopic method the organs or lesions of the chest may be viewed in several different positions, and the degree of penetration of the rays can easily be regulated to suit the case. In short, the use of the fluoroscope gives us more general information in a much shorter space of time than skiagraphy, and shows conditions impossible to demonstrate by the latter method, which, however, preserves a permanent representation instead of a mental impression or diagram on a chart.

FIG. 195.



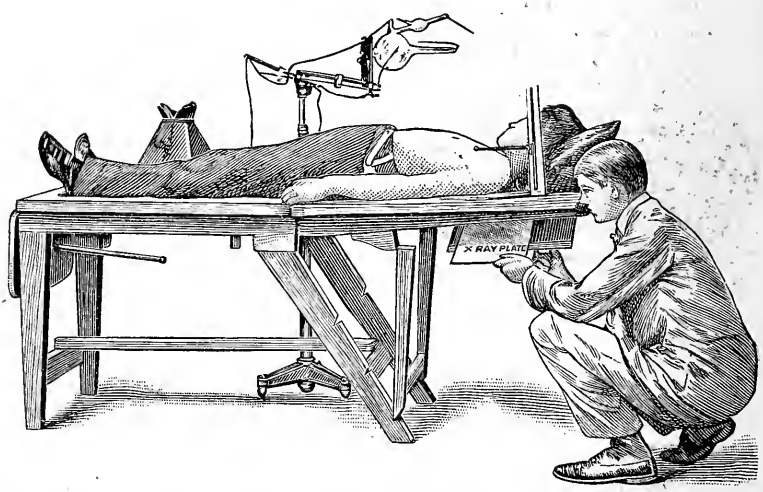
Fluoroscopic examination: Crooks tube and standard Queen radiographic table, with plate-holders removed and tube underneath.

Not only should the *x*-ray be used for such aid as it can give in the diagnosis of all thoracic lesions and to add to our experience in learning its value, but it should also be applied in all cases that may be called "obscure." In such cases the physical examination should be made first, so as to aid the fluoroscopic examination later. The latter in turn may bring to view some condition different from the suspected one, and thus alter the diagnosis, which should be as well founded as possible beforehand. The *x*-ray frequently shows some lesion within the thorax that can not be detected by the older methods of diagnosis. For example, phthisis, central pneumonia, aneurisms without physical signs, and the position of the heart.

Diaphragm. The diaphragm makes a clear shadow upon the skiagraphic plate because of the very slight range of motion during normal

respiration. In regard to the fluoroscopic examination we quote the following: "Normally, this musculotendinous structure stands dome-like with its highest point opposite the fourth rib on the right and the fourth space on the left, its shadow being very dark, and especially so over the right side, owing to hepatic density immediately beneath. The difference in height is slight, 1 to 2 centimetres. On ordinary inspiration the curve of the summit and the phrenicocostal angle alter but slightly, but on deep or forced inspiration there is a little flattening of the curve and a slight increase of angle may be noticed." [1]

FIG. 196.



Radiography: showing position of tube, Queen radiographic table, Queen localizing apparatus, and method of interchanging plates.

Therefore, the diaphragm does not have its curve flattened to any extent during respiration, but moves straight up and down somewhat like a piston. The excursion is slightly greater on one side than on the other.

"Abnormally, the diaphragm may stand low as in emphysema, or high as in pulmonary tuberculosis. Again, it may be higher on one side than on the other, and it has been seen that this occurs in tuberculous processes on the side of the affected lung. The movements of the diaphragm may be limited, as in:

"1. Loss of lung capacity.

"2. Obliteration of the pleural sinuses through inflammation.

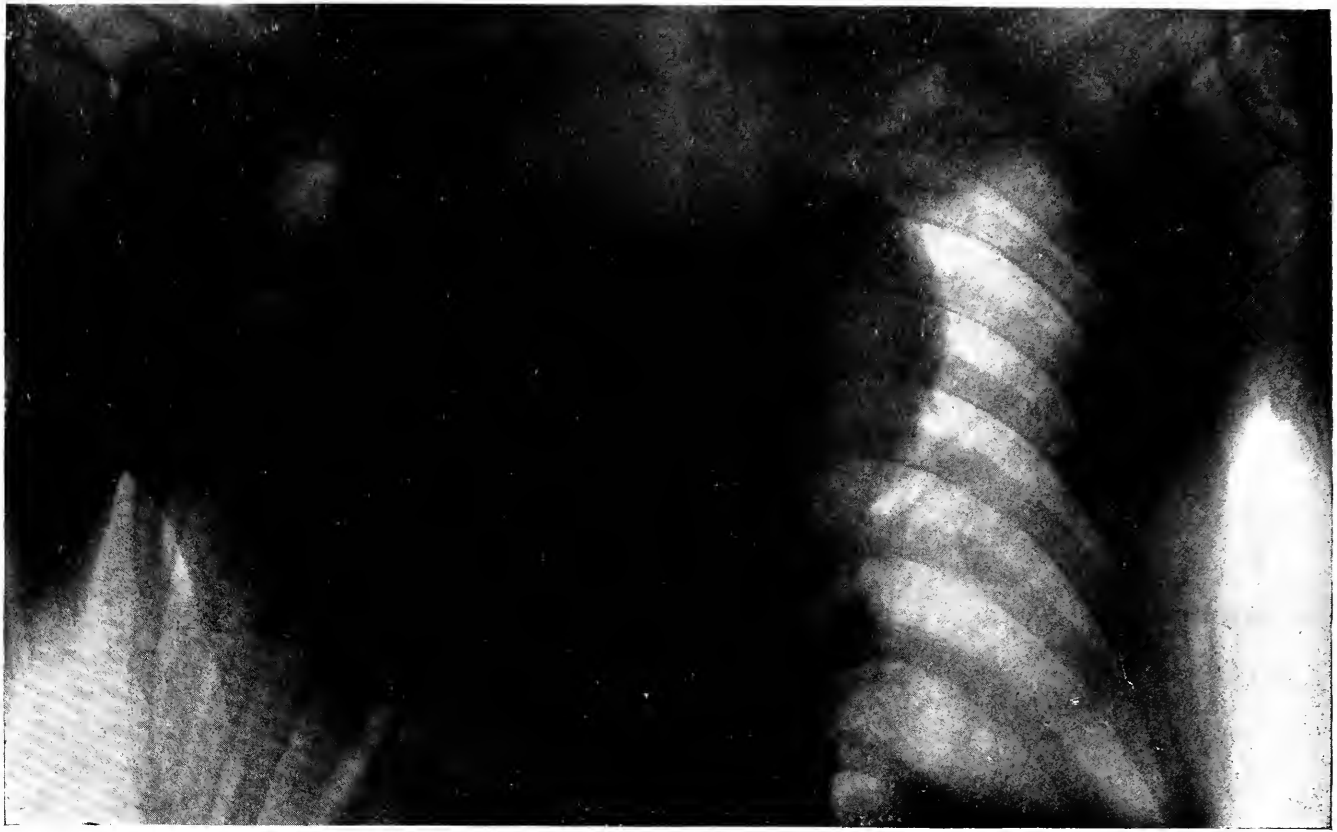
"3. Adhesions and fibrosis about the diaphragmatic pleura, anchoring it either to lung or mediastinum, as in pleurisy or peribronchitis with fibrosis.

"4. One side may be greatly depressed as in pneumothorax.

"5. The diaphragm has been described as in a state of immobility or fluttering movement as in asthmatic cases.

"Thus, the fluoroscope has cast another ray of light upon the possible

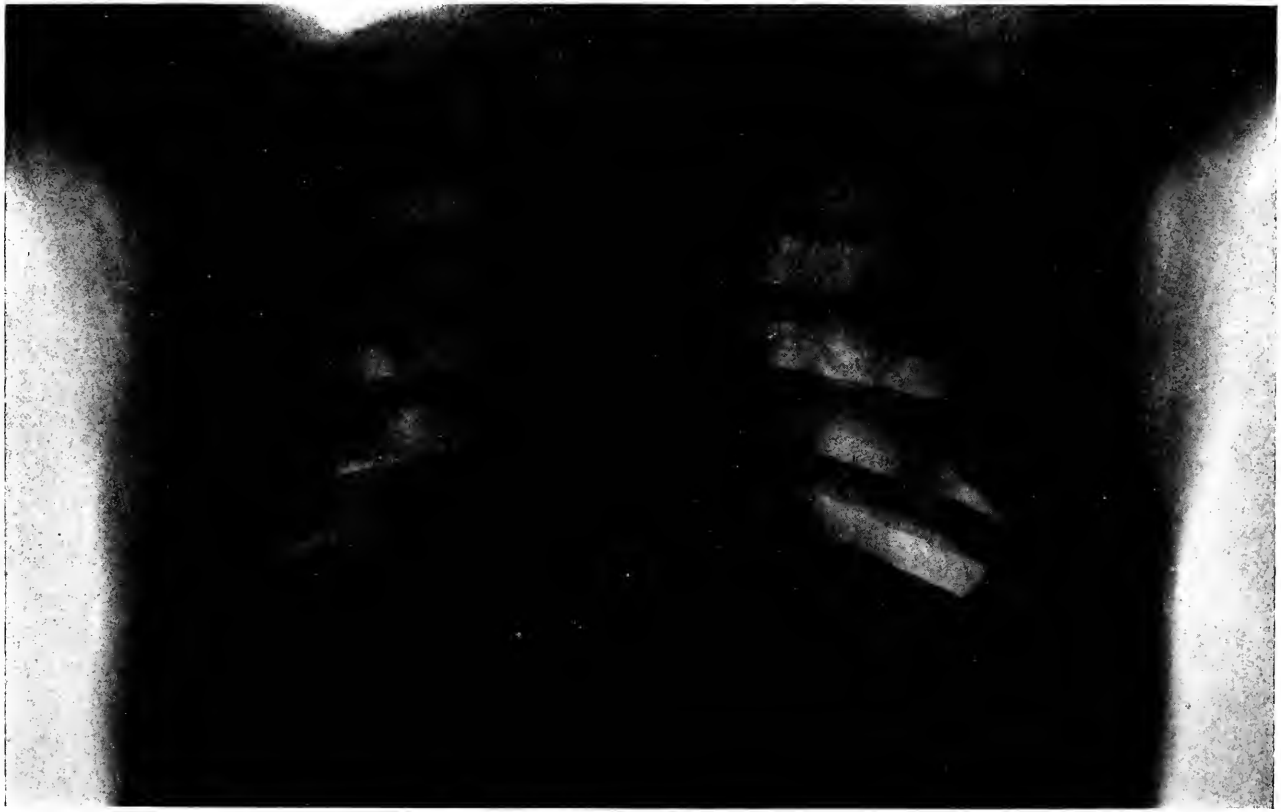
[1] Hamilton, read before the Canada Med. Assoc., Sept., 1902.



Aneurism of the Entire Arch of the Aorta.

It is about nine inches in diameter and occupies almost the entire upper portion of the chest. A part of the shadow of the heart, which is comparatively small, is shown downward and to the left of the shadow of the aneurism. A portion of the shoulder-joints is shown at the upper corners of the skiagraph. The examination and skiagraph were made by Dr. G. E. Pfahler, at the Philadelphia Hospital.

PLATE VIII-a.



Cardiac Hypertrophy and Dilatation of the Aorta.

The shadow of the aorta is shown in the upper central and the shadow of the heart in the lower left portions of the chest. They are about one and one-half times the normal size. The examination and skiagraph were made by Dr. G. E. Pfahler, at the Philadelphia Hospital.

causes of asthma, a spasmodic action of the diaphragm. In making fluoroscopic examinations it is quite easy to trace the outlines of these movements either upon thin paper or upon the chest-wall itself, and then comparison afterward becomes easy."[1]

Heart. The heart has been given its share of study under the *x*-ray, and most extraordinary claims have been made for this method of examination. But, in point of fact, very little is to be said of its value from a practical point of view beyond the observation of the size, position, and degree of expansion. Some observers have claimed that the deep and superficial areas of dulness can be mapped out, but the technic is a little too complicated and requires too much experience for the average observer. There are numerous results still to be obtained, however, and the realm is practically limitless at present. Among the results of examination so far recorded have been: a difference between strong and weak muscular action; a suggestion that the apex-beat is presystolic by comparison between movement and sound; a difference between hypertrophy and dilatation; pericarditis with effusion has been distinguished from hypertrophy or dilatation. It is claimed that the organ moves during respiration, following the central tendon of the diaphragm downward during inspiration. The cardiac apex has been seen at a different point than where the impulse is felt, showing the latter to be due to the impact of the ventricle against the chest-wall.[2] The heart being in constant motion, the skiagraph gives a dim or blurred outline to the organ's shadow. Often the extremes of expansion and contraction can be made out.

Aorta. The aorta can be observed just above the heart shadow, behind the sternum and to the left of the spine; but most of its shadow is blended with that of the spine. It is first noticed above the sternal portion of the clavicle, to the left side of the usual central dark shadow of the chest, where the descending arch stands out. The remaining portion is not to be observed, as it runs behind the heart.

Aneurism of the thoracic aorta can be recognized with the fluoroscope by the situation of its shadow and its pulsatile character. The pulsation is diminished or may be absent if the sac contains a clot. Aneurisms have been discovered by the *x*-ray when they failed to yield the usual signs. On the other hand, cases have been reported in which although the skiagraph showed almost unmistakable evidence of aneurism, none was found at the autopsy. Fluoroscopic views should be taken in as many directions as possible. The diagnosis of aneurism of the innominate artery is possible, but the shadow is demonstrated with greater difficulty.

Lungs and Pleura. Healthy lung tissue being easily penetrated by the Röntgen ray, a fluoroscopic study of the normal thorax will show a bright area to each side of the central shadow cast by the heart and spine, and terminated below by the moving diaphragm. The normal position, shape, and range of motion of the diaphragm being known, some idea may be formed of lung capacity, and alterations from any cause may be studied with the fluoroscope. The lung-areas allow of even greater penetration during full inspiration than during expiration. Examination in disease

[1] Hamilton, *ante*.
[2] Abrams, American Medicine, January 3, 1903.

should note *abnormal dark shadows, areas of abnormal brightness,* and *alterations* in the *height, shape,* and *movements* of the *diaphragm.* Shadows found in place of a normal penetration may be indicative of such conditions as consolidations of phthisis, pneumonia, malignant growths of the lung, pleura, or mediastinum, gangrene, infaret, compressed lung, echinococcus cyst, empyema, thickened pleura, etc. Emphysema and pneumothorax may allow of greater transmission of the rays and give greater brightness than normal.

In *phthisis* we note that an area of consolidation in any stage is indicated by a distinct shadow even before physical signs can be obtained. The lessened expansion of the lung may be discovered by noticing a lessened downward excursion of the diaphragm on full inspiration, perhaps before lessened expansion of the chest is noted by inspection. The lowest point reached may be higher than the normal height in health, the diaphragm being drawn upward. The apices should always be compared anteroposteriorly in both directions. There may be found a distinct difference in density, or the clavicle or upper ribs, or both, may show a blurred shadow on the suspected side. The outlines of shadows of all ribs should be carefully examined, otherwise some small area of consolidation may escape detection. Not only is the *x*-ray important in early diagnosis, but likewise the extension of the diseased area can be easily studied, and progress of the disease and even cavity formation watched.

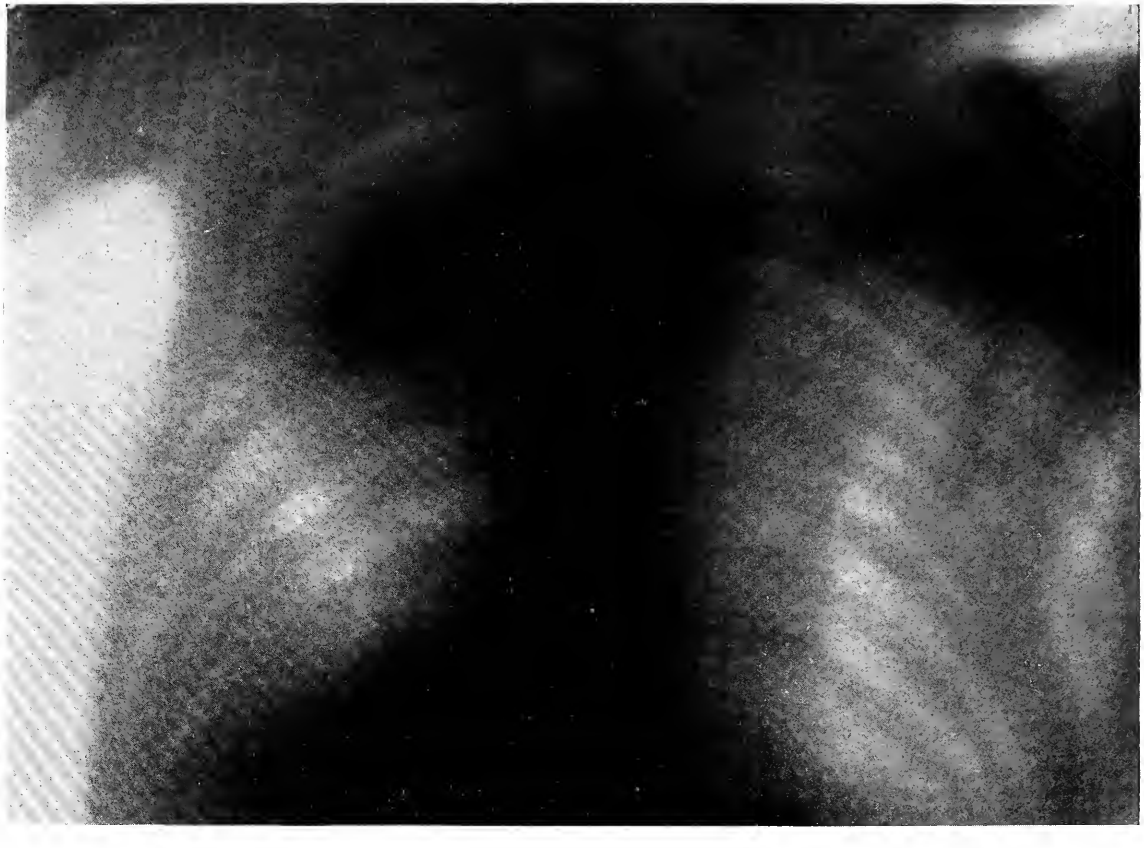
Pneumonia. The areas of consolidation are easily seen with the fluoroscope, and in the *x*-ray we have a valuable means of locating the lesion in obscure central pneumonia even when auscultation and percussion fail. The excursions of the diaphragm are restricted, but the shadow must be distinguished from that of a consolidation at the base, or in case of a pleural effusion or empyema accompanying the same condition. Any displacement of the heart is easily observed. Cases have been observed in which the changes in density were readily recognized from day to day after the crisis, during the progress of resolution. It is possible to study the formation of a secondary pleurisy with effusion, or empyema, and in the case of a basal pneumonia the line of the diaphragm has been seen distinctly.

Pleural Effusion. In large pleural effusions the whole or a great part of the chest is dark and the heart shadow is displaced. Sometimes it is possible to outline a circumscribed effusion with some distinctness. In hydro- or pyo-pneumothorax the level of the line of the fluid may be observed, and also the accompanying cardiac displacement. The density of the shadow has been said to be of use in determining the character of the fluid. In pneumothorax, no fluid being present, the diaphragm is found very low and its movements abolished on the affected side.

Emphysema. In emphysema it is to be noted that the distended lung is brighter than normal; the diaphragm is lower and does not show its normal excursion. It is said that in the late stages the heart occupies a more vertical position, which is not changed much on deep inspiration, and that enlarged ventricles and dilated right auricle may be observed.

Œsophagus. Attempts have been made to demonstrate tumors and

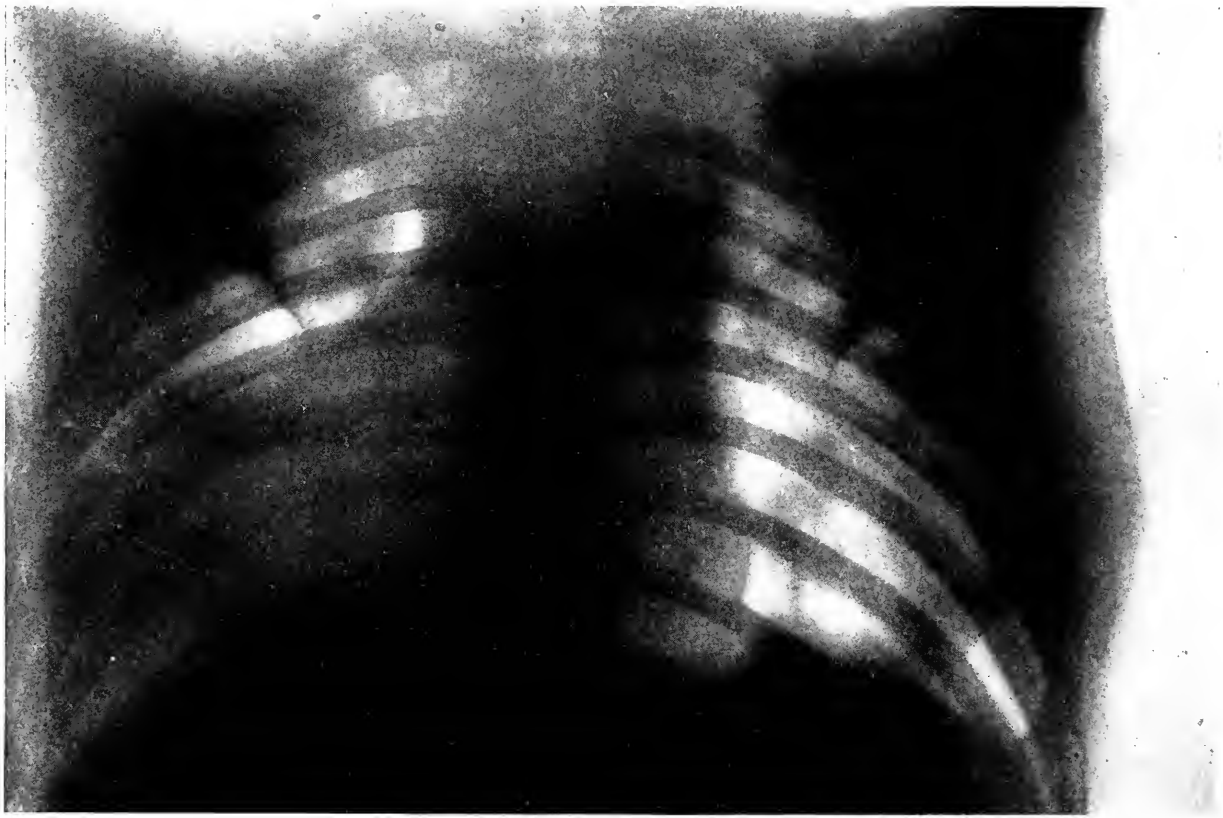
PLATE IX.



Aneurism of the Entire Arch of the Aorta.

The aneurism is about eight inches in diameter and is indicated by the shadow which occupies the greater part of the upper portion of the chest. Continuous with this shadow is the shadow of the heart, which extends downward and to the left of the skiagraph. The examination and skiagraph were made by Dr. G. E. Pfahler, at the Philadelphia Hospital.

PLATE IX-a.



Cardiac Hypertrophy and Dilatation of the Aorta.

The shadow of the aorta is shown in the upper central and the shadow of the heart in the lower left portions of the skiagraph. These shadows are more than twice the size of the shadows of a normal heart and aorta. The examination and skiagraph were made by Dr. G. E. Pfahler, at the Philadelphia Hospital.

diverticula of the œsophagus, but the central shadow of the chest usually interferes. In the case of diverticula a strong emulsion of bismuth should be swallowed by the patient previously.

The Abdomen. The abdomen is a field of great importance for the Röntgen ray in diagnosis, not because of its usefulness in any large variety of diseases, but because of the very great dependence now placed upon it in a certain limited number of conditions. Of these, the first to be mentioned is *renal calculus.* The work accomplished in this line has been remarkable in its success and has added much to precision in diagnosis. Mistakes may prove fatal, and hence the importance of great accuracy where so much depends on the skiagraph. The commonest mistake is that a negative result is obtained; but on the other hand, a calculus shadow has been clearly shown by a plate on a number of occasions where the stone could not be found at operation or autopsy. Each year sees the lowering of the percentage of errors, however. Very minute stones have been found with the *x*-ray. One plate alone should never be accepted as proof, but one or two additional ones should always be taken to confirm the first. The fluoroscope is of no value whatever in this work.

Liver. Very little information of value can be obtained by examination of the liver, except perhaps as to its size, position, and outline. A distended gall-bladder may be shown with careful technic, and also gall-stones in the ducts or in a *distended* gall-bladder. In comparison with the value of the *x*-ray in the detection of renal calculi, its importance in the diagnosis of gallstones is slight. Inspissated bile is not nearly so dense as concentrations of uric acid or oxalate crystals, and the biliary stone is situated farther from the plate whether the latter be placed under the patient's back or under his abdomen.

Stomach. As to the stomach, carcinoma when well advanced may be demonstrated. By special technic, such as inflation with bismuth powder, the size or outline may be studied. Of course the organ should be emptied and previously cleansed, and it may be well to state that in all abdominal work the gastro-intestinal tract should be as nearly free from contents as possible. Lately some observations have been made in regard to the peristaltic waves after bismuth had been freely taken.

Tumors. Tumors may be observed in some cases almost anywhere in the abdomen, and abscess of the kidney, perinephric abscess, and enlargement of the spleen are some of the other possibilities.

The Pelvis. In the pelvis the ray is applied to tumors, ureteral and vesical calculi, phleboliths, pregnancy, etc., but surgery here derives more assistance than does medicine.

SECTION VI.

LABORATORY DIAGNOSIS.

CHAPTER XXXVIII.

BACTERIOLOGICAL DIAGNOSIS.

Bacteriological examination includes:

1. The finding of the specific micro-organism in the blood or tissues, or in the pathological secretions or excretions.

2. The isolation and cultivation of the micro-organism.

3. The inoculation and the reproduction thereby of the disease in animals.

The morphology of the causal micro-organism in many infections is so characteristic that it can be readily identified when found in the blood or in the secretions. Thus an examination of the blood, with or without staining, will disclose the presence of the micro-organisms of relapsing fever and of anthrax, and the protozoa of malaria. The examination of inflammatory products of an infection, as the sputa in pneumonia or tuberculosis, is sufficient to determine the nature of the pulmonary infection. On the other hand, in some infections, the absence, or rather failure of detection, of the micro-organism in the fluids or discharges is not proof that the disease is not present in the suspected individual. The infection tuberculosis well illustrates the propositions in the last two sentences. If the bacillus is found in the sputum of a suspected case, the diagnosis is established, and no further procedures for diagnostic purposes are necessary. In other clinical forms, as tubercular pleurisy, or empyema, and glandular and joint-tuberculosis, the micro-organisms are few and difficult to find. Cultures or, more conclusive still, inoculations must frequently be resorted to before a final conclusion can be arrived at. It is possible that spores alone exist, as, for example, in tetanus—morphological elements difficult to detect by staining and microscopical methods, which may, however, rapidly multiply in favorable culture-media and under favorable inoculation conditions. Again, micro-organisms have been found in certain infections; and although thus far their causal relationship to the disease has not been fully proved, nevertheless their constant occurrence in the special affection, and in it alone, renders their presence of high diagnostic value. Thus the amœba of dysentery and *Plasmodium malariæ* of Laveran are diagnostic of their respective affections.

Method of Procedure. To determine the presence of most infections the following procedures are necessary:

A. Examination of the blood.

B. Examination of the pathological secretions and excretions.

C. Examination of products of infectious inflammation obtained by exploratory puncture or evacuation of abscesses. (See Chapter XLV., Part I.)

D. Inoculations of animals with pure cultures of the organisms or with the products of inflammation, as cheesy matter from a tuberculous abscess.

E. The use of products of bacterial growth to secure reaction, as tuberculin in tuberculosis, and mallein in glanders. (See Tuberculosis.)

When there is no distinctive pathological fluid, all the fluids of the body must be examined. In other cases the pathological discharge (pus), or perhaps the diseased tissue must be studied. We get a clue to the direction which the examination is to take from the nature of the symptoms. In cases of pulmonary disease, the sputum; of faucial disease, the membrane, pus, or other secretions from the fauces; in intestinal disease, the discharge from the bowels; and in genito-urinary disease, the urine should be examined. It must not be forgotten that in many, even highly fatal diseases, the blood is not invaded by micro-organisms. Death is due to the development of toxic substances. Hence, as in cholera and diphtheria, the presence of the micro-organism is not sought for in the blood, but in the specific excretion or exudation. (See Tuberculosis.)

Collection of Material. A definite, careful method must be observed when the pathological product is removed from the patient or collected for investigation. (See Chapter XLV., Part I., Exploratory Puncture.) All instruments to be used in removing the fluid must be previously sterilized, and the pathological product obtained must be placed in sterilized glass bottles or tubes. Exposure to air should be as brief as possible. The fluid obtained must not be contaminated with blood or antiseptic fluids used for flushing or other surgical procedure. If, for instance, an abscess is opened or purulent peritonitis cut down upon, tube inoculations can be made at the operating-table. The previously sterilized platinum point should be kept before use in a test-tube closed with sterilized cotton. It is dipped into the pus before it flows over the skin, and the pus should be free from the blood of the incision. It is at once transferred to the medium in the test-tube. Sputum should be collected in a previously sterilized bottle, or one thoroughly cleansed by boiling. The bottle should have a wide mouth. Care must be taken to secure sputum, and not the secretion from the mouth and fauces. Purulent rather than mucoid portions are to be sent for examination. Intestinal discharges may be collected in sterilized glass jars and examined as soon as practicable. It may be necessary to keep the discharge at the temperature of the body. (See Feces—amœba dysenterica.)

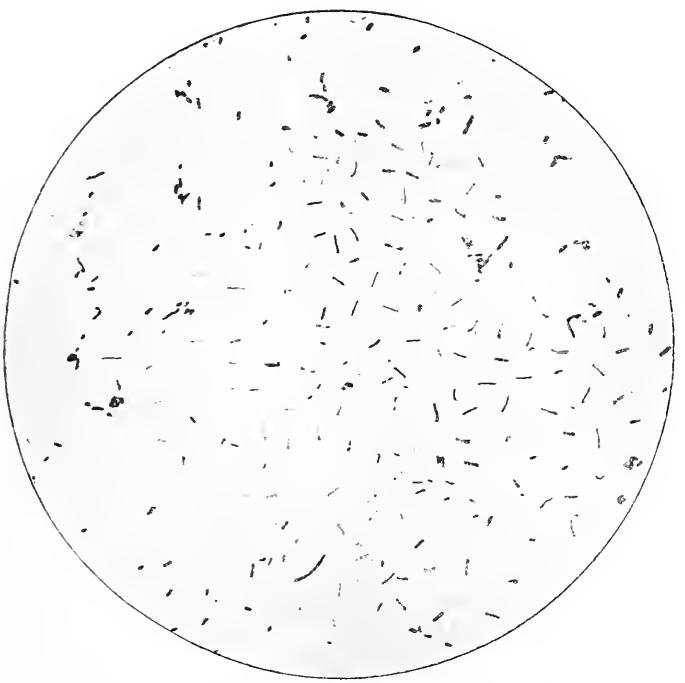
A. Examination of the Blood.

Serum Diagnosis. The phenomena of agglutination consist in the gradual approximation, clumping, and loss of motility in the micro-organisms of some infectious disease when the blood of a patient suffering from that disease is brought in contact with them. This is known as the

serum or Widal reaction, and by means of it a number of infectious diseases can now be recognized. If a drop of bouillon culture of a motile micro-organism is examined with a high-power lens, the organisms are seen darting about and across the field with great rapidity in various directions. If to 10 parts of a pure culture of certain varieties of infectious micro-organisms, 1 part of the serum of a patient suffering from that infection is added, the motility of the organisms is checked and clumps appear in the field. The clumps enlarge rapidly and are easily visible under a magnifying power of 500 diameters.

Serum from patients suffering from other diseases or from healthy patients does not produce agglutination if the proportion of serum to culture in the mixture is 1 to 10 or less. The reaction is specific. Thus typhoid bacilli are not clumped by any serum other than that of a typhoid patient, or a patient immunized against typhoid fever by a more or less recent attack of the disease. Typhoid serum when used with a certain degree of dilution and examined within a certain period of time clumps no organism except the typhoid bacillus.

FIG. 197.



Bouillon culture of typhoid bacilli before the addition of diluted typhoid serum (×500). After CABOT—serum diagnosis.

Serum diagnosis has become a valuable mode of recognition of typhoid fever, paratyphoid fever, Malta fever, yellow fever, tropical dysentery, and glanders. It may be of use in other infections, as cholera and the pneumococcus infections, but these diseases are more accurately diagnosticated by other bacteriological methods, and need not be considered here.

Three methods of securing the serum reaction are employed: the microscopic, or quick test of the fluid serum or blood; the microscopic, or quick test of the dried blood; and the macroscopic, or slow test, not in vogue at present in this country, although advocated by Wright in England. Each of these methods is of value. The observer should select one and make it his object to become thoroughly familiar with that selected.

Microscopic or Quick Test with Fluid Serum. The steps are: (1) to collect the blood; (2) to add it in certain proportion to the fluid culture; and (3) to examine the slide and cover-slip.

1. COLLECTING THE BLOOD. The blood is secured by puncture. If the ear is selected, it can be bled freely or blood squeezed out by the milking process until about fifteen drops have been collected in a small tube or pipette; although it is not necessary to observe strict antiseptic precautions, the instruments and tube should be thoroughly cleansed. The blood thus collected is allowed to coagulate, which may require several hours. It is to be remembered that the clot collects on the sides of the tube and over the surface of the blood. To secure the serum, this clot must be removed with a bit of wire.

FIG. 198.

The same, five minutes after the addition of typhoid serum (dilution 1 to 10), showing typical clump reaction (×400). (CABOT.)

Instead of the serum from the blood the serum of a blister may be used, or the serum from blood which has been drawn directly from a vein with antiseptic precautions.

2. DILUTION. One drop of the serum is added to forty drops of a bouillon culture. The same dropper must be used for each fluid, so that

the size of the drops will be equal. The fluids are to be mixed intimately in a small test-tube. A drop of this mixture of culture and serum is placed upon a cover-glass, which is then inverted over a hollow ground slide and examined under the microscope with the immersion lens. Within twenty minutes clumping should take place. If the reaction does not take place, a new mixture should be made in the proportion of 1 to 20 or 1 to 10. If there is no reaction with this dilution, the test is negative. Instead of making successive mixtures, three tubes can be prepared at once, containing 10, 20, and 40 drops each of the culture. A drop of serum can be added and the test conducted as above. In most laboratories at present the dilution of the serum is the method employed. To 1 drop of serum 5 drops of bouillon or salt solution are added: both being measured by a pipette. 1 drop or 1 loop of the mixture added to an equal sized drop or loop of a bouillon culture gives a dilution of 1 to 10. Other higher dilutions are made accordingly.

3. Examination of Slide. A No. 7 Leitz dry lens or oil-immersion lens can be used with a No. 3 or No. 4 eye-piece. Artificial light is preferable to daylight; if the latter is used, a small aperture diaphragm is the best. It is very necessary that the slide and cover-slip be thoroughly cleansed.

The Reaction. In a complete or typical reaction the field shows the presence of large clumps of bacilli isolated and motionless. (See Fig. 198.) No motile bacilli can be seen. The clumping may occur instantaneously or gradually. If the reaction is very marked, Greene states, a mottling can be seen with the naked eye. Clumping and cessation of motion are the essentials of the reaction, providing they take place within a certain time and notwithstanding a certain degree of dilution of the serum. When the reaction is feeble, small clumps appear, or, as Widal calls them, agglutination-centres. As the field is studied, bacilli are seen moving toward the centres and gradually ranging themselves in loose masses, sometimes like the spokes of a wheel. Durham has called attention to a peculiar spinning motion of the bacillus around one of its own ends, which is seen in some of the fields in which a few isolated bacilli remain. Such movements occur at the margin of the clump.

It is very necessary to examine a drop of the pure culture before the addition of any serum, to make sure that clumping has not already taken place, particularly if the culture is old or has undergone sedimentation. It is desirable that the bacilli should be isolated and actively motile.

Time-limit and Dilution. As Cabot forcibly states, only when clumping occurs within a certain time and in a certain degree of dilution is it of diagnostic importance. The test is quantitative and not qualitative. A dilution of 1 to 10 is quite sufficient if the time-limit for the reaction is at least fifteen minutes. Clumping of typhoid bacilli taking place fifteen minutes after 1 part of serum has been added to 10 of the culture constitutes a probable typhoid reaction. Various observers select different dilutions. Thus, Wilson and Westbrooke make a dilution of 1 to 50 with a two-hour time-limit. Durham uses a dilution of 1 to 17 or 1 to 20.

The whole blood can also be used in a fluid state. A drop of the blood can be drawn directly into 10 drops of the culture previously

measured. This method is of great advantage for rapid work. The same dropper should be used for measuring the culture and subsequently the blood. With the microscope at the bedside the test can be made rapidly with but little risk of failure.

A still more convenient method consists in the employment of the pipette, used for diluting the blood in counting leucocytes. The blood from the finger is drawn up to the 0.5 or 1.0 mark on the stem, and the bulb then filled with distilled water. The mixture is then blown into a small test-tube. As the dilution has already been made, a drop of bouillon culture may be added to it directly and examined as above.

The Reaction with Dried Blood. To Wyatt Johnston, of Montreal, is due the credit of working out this simple but accurate method of testing for the reaction. It is of special value for sanitary work where blood has to be sent by mail for examination. The blood is collected on glass or *glazed paper*. In this manner it can be preserved for an indefinite time and transported easily. If the drop of blood is dried on a glass slide, it can be dissolved by the addition of a little water, and the culture can then be added in the way previously described. If the drop is dried on paper, it can be cut out with a pair of scissors and rubbed up in a watch-glass with one drop of water. When the blood is dissolved, ten drops of culture are added, and the examination is carried on as in the previous method.

Some operators collect the blood in the eye of a wire loop of a given size, and after placing it on a glass, dilute with water in the proportions desired, ten loopfuls of water being the amount usually selected to mix with the drop in the wire loop. Wilson and Westbrooke have modified Johnston's technic as follows: They use a bit of platinum wire, No. 19 gauge, one end of which is bent into a loop, the inside diameter of which is 2 mm. The loop is used to collect the blood, several drops of which are deposited on a bit of aluminum foil, No. 40 gauge, 5 cm. square. After the blood is dried the foil is rolled up. At the laboratory the bit of foil is then cleared of blood, which flakes off easily. 1 mgm. of dried blood and 200 mgm. of distilled water are weighed out and mixed. This gives an exact dilution of 1 to 200 by weight; 1 to 50 dilution by volume. A hanging drop of the dilution is inoculated with the bouillon culture and examined. The *time-limit* is two hours.

It is essential for the success of the reaction that a pure culture of the typhoid bacillus should be employed. The most suitable culture for diagnostic work is that which is the most actively motile. It is true, however, that many observers recommend the attenuated cultures. They hold that an actively motile culture is too sensitive, and may cause clumping even with normal serum. If a fresh culture is kept at room temperature and transplanted every two or three days, it will maintain its motility and sensitiveness for a long period. The incubator bouillon cultures of twelve hours' growth are probably the most satisfactory. Johnston, whose experience is worth following, thinks the motility must not be excessive. He reduces the motility of the bacilli by transplanting his agar cultures once a month, growing them at room temperature. The

bacilli from this culture, grown for twenty-four hours on bouillon, show a slight gliding motion, which differs from the darting motion seen in an active culture. The bouillon, Johnston holds, should be slightly acid, contrary to the general rule, which states that it should be neutral. It is quite necessary that the bouillon culture should be young—that is, of twelve to twenty-four hours' duration in the incubator or two days at room temperature. When a culture is made under these circumstances, it should be free from sediment and only slightly turbid before it is used. It should also be free from spontaneous clumping and from non-motile or sluggish forms.

Value. The question may well be asked, What is the value of the serum reaction? Let us answer by referring to typhoid fever chiefly. When it is recalled that this reaction takes place in about 98 per cent. of all cases of typhoid fever, it can readily be seen what a constant phenomenon it is in the course of continued fever. As a symptom therefore it is one of the most constant. Its presence, however, cannot be determined in a large number of cases before the eighth or tenth day. It has been found as early as the third day, and, on the other hand, may be absent until after convalescence has set in. In a large majority of cases the reaction appears, however, before the fourteenth day. In a few instances, as Widal pointed out, the reaction disappears as soon as the temperature remains normal. In other instances it may continue several months, and in rare cases has been found as long as ten years after the disease.

It is thus seen that the presence of the serum reaction is a valuable diagnostic symptom of some disease, notably of typhoid fever. Its absence, however, does not disprove the presence of the disease. Sometimes the blood of a patient ill with some other disease, who has previously had typhoid fever, may give a positive reaction, and thus lead to a false diagnosis. Absence of reaction in a supposed case of typhoid fever implies, in 98 per cent. of all cases, that this infection is not present, providing, of course, that the technic is correct and that repeated examinations have been made. Serum diagnosis in the following diseases is employed: (1) glanders; (2) Malta fever; (3) yellow fever; (4) cholera; (5) relapsing fever; (6) typhoid fever; (7) tropical and other dysenteries; (8) paracolon and paratyphoid infections.

Leucocytosis. The presence of leucocytosis is characteristic of many infections, and, on the other hand, argues against not a few of the most common of the infectious disorders. Accurate study of the number of white cells has led to fairly definite conclusions as to the diagnostic value of their increase or their diminution. The method of determining the number is described in the next chapter, which may be referred to in order that the student may also learn the conditions under which leucocytosis occurs physiologically. Pathologically we find inflammatory leucocytosis or the leucocytosis of infectious disease occurring with such frequency as to be diagnostic. A classification of the degree can be roughly made only:

1. In Asiatic cholera, relapsing fever, scarlet fever, diphtheria, syphilis, and erysipelas, leucocytosis occurs to a *moderate degree.*

2. In smallpox in the stage of suppuration, septicæmia, trichinosis, glanders, beri-beri, acute rheumatism, cerebrospinal meningitis, and gonorrhœa it is also found, but more *constant* and more *marked*.

3. In most pyogenic infections, especially abscesses, commonly in pneumonia, in inflammations of serous membranes, and in gangrenous inflammation, leucocytosis is *great*. Infections due to the streptococcus, not infrequently show little or no increase in the leucocytes.

The *significance* of leucocytosis depends not alone upon the number of the white cells, but also upon their rise and fall in the course of the disease. The intensity of the local inflammation attending the infection is not a measure of the degree of leucocytosis. Moreover, the degree of fever does not affect the leucocytosis. Fever may occur without increase in the white cells, and the opposite condition may also obtain. When leucocytosis and fever are due to the same infection, they may rise and fall together, as is often seen in cases of pneumonia.

Absence of Leucocytosis. While the presence of leucocytosis is significant of various infections, its absence is likewise of great significance. Hence, if there is no leucocytosis, it is possible that either typhoid fever, malaria, influenza, measles, rötheln, or tuberculosis is present. The blood-count can in this manner be employed to distinguish typhoid fever, in which there is an absence of leucocytosis, from a pyogenic infection, as appendicitis, in which the other signs and symptoms may be quite similar. Pneumonia, on the other hand—an infection characterized by great leucocytosis—may in this manner be distinguished from tuberculosis, in which there is an absence of leucocytosis.

When leucocytosis occurs in the course of any disease in which it is normally absent, it indicates a complication. In typhoid fever it is an indication of intestinal perforation and peritonitis, because of a mixed infection. On the other hand, a fall of leucocytes in a disease in which they are increased is suggestive of localization of the infection, as the "walling off" of the abscess in appendicitis. Such fall in pneumonia unattended with indications of a crisis may be of grave prognostic omen.

Blood Cultures. For direct bacteriological examination of the blood, culture-methods are resorted to. After the skin has been cleansed and made aseptic either a considerable portion of blood is withdrawn from a vein with a sterilized hypodermatic needle or blood is directly withdrawn with the instrument described by Ewing. The blood is then transferred to the various media, and its further treatment is carried on in accordance with bacteriological methods. (See Cultivation of Micro-organisms.)

B. Examination of Pathological Secretions and Excretions.

Microscopical examination, with and without staining, and *culture-methods* are employed, as detailed in the sections to follow:

In *nasal discharges* the bacillus of diphtheria, of glanders, of tuberculosis, the pneumococci, as well as pyogenic micro-organisms, may be found.

In the *mouth* the micro-organisms peculiar to that cavity and the micro-organisms of actinomycosis may be found.

In the *fauces* and *pharynx* the bacillus of diphtheria and pyogenic micro-organisms are discovered.

The *sputa* (see Diseases of Lungs) yield the tubercle bacilli, the pneumococcus, the bacillus of influenza and of actinomycosis.

The *feces* (see Diseases of Intestines) are examined for *Bacillus coli communis*, the spirillum of cholera Asiatica, *Bacillus typhosus*, tubercle bacillus, *Amœbæ coli*, and other protozoa.

The *urine*. Pyogenic micro-organisms, tubercle bacillus, typhoid bacillus, the pneumococcus, and gonococcus are found in the urine. Cover-slip preparations of the pus and cultures as described in the section devoted to Diseases of the Kidneys are examined.

C. Examination of the Products of Infectious Inflammation—Material Secured by Exploratory Puncture.

Material removed by exploratory operation or puncture may be serous, bloody, or purulent. (See Chapter XLV., Part I.) It must be examined bacteriologically, microscopically, by culture-methods, and by inoculation. Bacteria are not usually found in serous fluids unless treated by sedimentation, and even then it is often necessary to inoculate.

The most important pathological product is *pus*. Fresh and stained preparations are examined; most of the ordinary aniline dyes can be used; for certain micro-organisms Gram's method is satisfactory, and is used as a differential stain. Cultures should also be made. We may find only one, sometimes two at the same time, of the following micro-organisms: 1. *Staphylococcus pyogenes aureus*. 2. *Staphylococcus pyogenes albus*. 3. *Staphylococcus epidermidis albus* (Welch). 4. *Streptococcus pyogenes*. 5. The tubercle bacillus. 6. *Bacillus typhosus*. 7. Actinomyces. 8. The bacillus of glanders. 9. The bacillus of anthrax. 10. The bacillus of leprosy. 11. The bacillus of tetanus. 12. The bacillus of influenza. (See Sputum.) 13. *Micrococcus lanceolatus*. 14. *Bacillus coli communis*. 15. The gonococcus (Gram's method).

Methods of Staining Blood, Pus, and Discharge. It is well to consider these collectively. Many have been devised, but those of clinical value are the following:

1. Aqueous solutions of basic anilines.
2. Löffler's alkaline methyl-blue.
3. Koch-Ehrlich's aniline water solutions.
4. Ziehl's carbol-fuchsin.
5. Löffler's method of staining flagella.
6. Gram's method.
7. Friedländer's method.
8. Günther's method.

1. *Basic anilines*. Aqueous solutions of the basic aniline colors—fuchsin, gentian-violet, and methyl-blue—are used of such strength that they can be seen clearly through an ordinary test-tube. They may be kept on hand in bottles with pipettes, or made from concentrated alcoholic solutions as needed. They are used by simply dropping a few drops on the cover-glass preparation, which is held with the forceps, allowing it to

remain about thirty seconds, and carefully washing off in water. The specimen is placed on a slide, *bacteria down*, and the excess of water removed with blotting-paper.

2. *Löffler's alkaline methyl-blue solution.* Certain bacteria take a stain more readily when an alkali has been added. The formula is as follows:

Concentrated alcoholic solution of methyl-blue	30 c.c.
Caustic potash, 1 : 10,000	100 "

It is used in the same way as the simple solution.

3. *Koch-Ehrlich's aniline water solutions.* Add to 100 c.c. of distilled water, aniline oil, drop by drop, thoroughly shaking after each drop until the water becomes opaque. Then filter. Add 10 c.c. of absolute alcohol and 11 c.c. of a concentrated alcoholic solution of either fuchsin, methyl-blue, or gentian-violet.

4. *Ziehl's carbol-fuchsin solution.*

Distilled water	100 c.c.
Carbolic acid	5 gm.
Alcohol	10 c.c.
Fuchsin	1 gm.

The use of these various stains will be described in the description of the different bacteria.

5. *Löffler's solution for flagella.*

Tannic acid, 20 per cent.	10 c.c.
Cold saturated solution of ferric phosphate	5 "
Saturated solution of fuchsin	1 "

A few drops of this solution are placed on the cover-glass containing the bacteria and very gently heated until they begin to steam, and then the cover-glass is washed off in water. The preparation is then stained with aniline water fuchsin. Different bacteria require different reactions, and so a few drops of an acid or alkaline solution are recommended to be added as the case requires. As a rule, however, the results obtained when neither acids nor alkalies are added are just as satisfactory as those following such additions.

6. *Gram's method.* The cover-glass preparation is stained with a Koch-Ehrlich solution of gentian-violet for twenty to thirty minutes, and then decolorized in

Iodine	1 gm.
Potassium iodide	2 "
Distilled water	300 c.c.

After remaining in this for five minutes the preparations are rinsed in alcohol, and the process repeated until the violet color has disappeared.

For *Friedländer's* and *Günther's methods*, see Sputum.

To detect *spores* of bacilli double staining may be employed. The preparation is first stained in a hot Ziehl-Neelsen fuchsin solution, then decolorized with alcohol containing from 0.2 to 0.3 per cent. of hydro-

chloric acid. When stained again with methylene-blue, the spores appear red and the bacilli blue.

The "Hanging Drop." By the examination of colonies in the *hanging drop* we learn of the movement of the micro-organism. Place a drop of physiological salt solution on a cover-slip, and add a tiny portion of the colony on a platinum wire; place the slip, drop down, on a glass slide, in the centre of which is a depression. Fix the slip by applying a thin layer of vaseline around the margin of the depression. Care must be taken in focusing that the lens does not break the glass, which may be readily done because of its transparency. The bacteria are seen in motion; on account of which their position is constantly altered. This motion must not be mistaken for the Brownian movement of suspended particles, which is vibratory from molecular tremor.

Cultivation of Micro-organisms. The object is to isolate the pathogenic organism from all other organisms, and to exclude organisms that may be introduced from without by unclean instruments or other means. *Pure* cultures are thus obtained.

Cultures. Pus removed at an operation or by exploratory puncture, pathological secretions from the various cavities, orifices, or passage-ways, are transferred to media and placed in a reservoir of proper temperature, whereby any suspected micro-organism may grow.

Microscopical Examination of Colonies. Just here may be stated the methods employed for the study of the morphology of the colonies secured by plate and other means of cultivation.

Cover-glass preparations are made as follows: Place on the cover-glass a small drop of distilled water. With a platinum needle take up the smallest possible quantity of the colony to be examined, mix it with the drop and spread over the surface of the glass. Dry under cover or by holding *with fingers* over a flame, the layer of bacteria being away from the flame. When dry, pass the specimen with forceps three times through the gas or alcohol flame to "fix" the albumin. It is then ready for staining.

D. Inoculation of Animals.

Another method of determining the pathogenic character of morbid material, as sputum, pus, or exudation, is by inoculating animals with a pure culture. This is done either by feeding the animals or by injecting subcutaneously, intravenously, or into the peritoneal or pleural cavity, and in rare instances into the anterior chamber of the eye or into the cranial cavity.

As animals are subject to only a few of the microbic diseases of man, many experiments must often be made before a susceptible animal is found, and no conclusions can be reached as to the pathological power of a micro-organism until this point has been determined. The clinical course of the artificial disease must be observed to fulfil the diagnosis, and the difficulty of reproducing faithfully in animals the clinical manifestation seen in man is often one of the gravest obstacles to this method of diagnosis.

Examination of the animal is made as soon as possible after death. The autopsy is made with antiseptic precautions. After the skin is removed only sterilized instruments are to be used. The macroscopical appearances and the mode and progress of infection are noted for the purpose of aiding in the diagnosis. When the organs are exposed, material for culture is first obtained by inserting a platinum needle through a small puncture in the capsule. Afterward cover-glasses may be prepared for immediate examination. Blood is taken from one of the cavities of the heart. After the autopsy all remains are to be burned and all instruments carefully sterilized.

NOTE. For further information concerning technic the student must refer to the work of Abbott on the "Principles of Bacteriology" and to Sternberg's "Manual of Bacteriology" for an exhaustive account of the technic, and the morphological and bacteriological characteristics of all bacteria, pathogenic and non-pathogenic.

CHAPTER XXXIX.

THE BLOOD.

Normal Blood.

THE blood consists of corpuscles and serum. The formed elements are: (1) red blood-cells, erythrocytes; (2) leucocytes; (3) platelets. The hæmokonium granules (see page 564) are often given among the normal elements of the blood.

Physical Appearance. For the purpose of examination of the blood a drop or two is sufficient. In olden times much stress was laid upon the physical character of the blood drawn in bulk. The significance of the "buffy coat" was dwelt upon by all clinicians, not alone because of its value from a therapeutic standpoint, but also because it was held to indicate the type of the disease that was present. At present, however, we rely very little upon naked-eye examination. By this examination we may be able to distinguish bright-red arterial blood from darker venous blood, and also when arterial blood has become deficient in oxygen from any of the causes of venous engorgement and cyanosis. In chlorosis and hydræmias, the blood is pale as though mixed with water, while in severe leukæmias it has a slight milky tinge. On the other hand, in carbon monoxide poisoning the blood becomes of a brighter red, while in poisoning with potassium chlorate and aniline, and in grave cases of poisoning with nitrobenzol and hydrocyanic acid, it is brownish-red or chocolate-colored.

For accuracy in diagnosis reliance must be placed upon instruments of precision. These are the microscope, the hæmoglobinometer, the hæmocytometer. By this examination we determine (1) the size and shape of the red cells; (2) the morphological characteristics of the white cells; (3) the number of red cells; (4) the number of white cells; (5) the presence of new elements, as nucleated red cells and myelocytes; (6) the presence of parasites; (7) and the amount of hæmoglobin.

Technic. A drop of blood for this examination may be taken from the lobe of the ear or the finger-tip. The surface should be thoroughly cleansed with alcohol, and dried carefully. If the finger is used, it should not be unduly constricted. The puncture should be made forcibly and quickly, in order that the blood may ooze freely. If it is difficult to secure the blood, it is well to allow the first or second drop to escape before any is collected. When the flow is started and the finger cleansed, the succeeding drops are gathered on cover-slips. If the lobe of the ear is selected, it should be steadied with the fingers of the left hand, which at the same time stretch the skin. It may be necessary to puncture to the depth of $\frac{1}{8}$ inch, or even deeper if the skin is bloodless. The puncture should be made on the lower surface or edge of the lobe. A surgical

needle, a small lancet, or the bayonet-pointed instrument devised for the purpose, should be used. The nib of a new steel pen, one-half of which has been broken off, answers fully as well.

It is well to remember the precaution insisted upon by all who examine blood frequently, to beware of "bleeders." It sometimes becomes a very serious matter when hemorrhage is started in a hæmophilic.

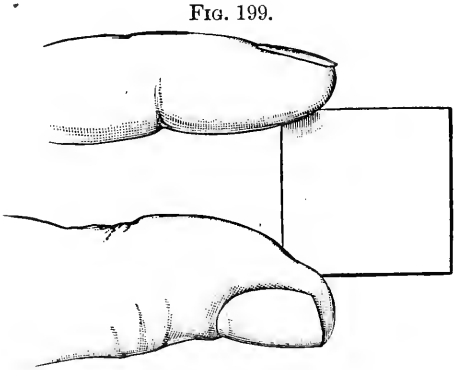
As soon as the blood flows freely without pressure, the apex of a drop may be touched by the cover-glass, which has been previously prepared. The cover-glass should not touch the skin, and as soon as it is covered by the blood it should be placed face downward upon the slide, or if cover-slip preparations are to be made, upon a corresponding cover-glass. The precaution must be taken to have the slide and cover thoroughly cleansed. It is well to keep them in alcohol or in a weak acid solution after they have been previously cleansed with soap and water, and when removed from the alcoholic solution they should be thoroughly polished with a clean handkerchief. The blood will then spread evenly over the surface with the slightest pressure upon the cover-glass. If the slide and cover are warmed slightly before using, it will not be necessary to use pressure.

Blood collected in this way may be examined fresh or be put aside for staining and future examination.

Examination of Fresh Blood. By the examination of fresh blood we learn of the presence of parasites, the number of red and white cells approximately, the degree of coloring of the red cells, and their shape and size, and the presence of blood-platelets. An unusual increase in leucocytes may be detected, and the diagnosis of leukæmia made without further investigation. In a well-prepared specimen the rouleaux formations are avoided except as the blood masses toward the edges of the cover-slip.

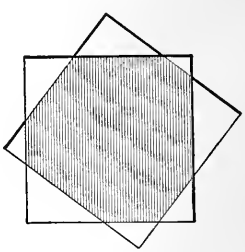
Here may be mentioned the hæmokonium granules, or blood dust (Müller), the small dancing coccus-like refractive bodies seen in greater or less abundance in all blood; their significance is unknown, and they are not readily recognized in stained specimens.

FIG. 199.



Proper method of holding a cover-glass. (CABOT.)

FIG. 200.



Illustrating the position of cover-glass during the spreading of blood films. (CABOT.)

COVER-SLIP PREPARATIONS. For the purpose of future study, and particularly in order to determine the differential count of the white corpus-

cles, cover-slip preparations are made. The covers are cleansed and the blood secured in the manner previously described. The cover-glass, which has been touched to the summit of the drop, is let fall upon another somewhat diagonally. (See Fig. 200.) The drop spreads over the adjoining surfaces of the cover-glass. As soon as spreading ceases, slide the glasses off, but do not lift them apart. Manson introduced the use of tissue-paper drawn over a slide, with the object of getting a more uniform thickness of film. Pakes uses this method applied to cover-glasses, which should be not less than $1\frac{1}{2}$ inches by $\frac{3}{4}$ inch. The cover-glasses are held in a clip and smeared by means of cigarette-paper cut into strips across the direction of the rib. The cover-slip should be dried in a gas or alcohol flame at once, which fixes the preparation. Good preparations can be made readily by simply taking the drop (a large one) at the edge of a well-cleaned slide and spreading it by drawing the slide over another as if scraping its surface; with care a smooth, regular layer of blood is obtained.

"Fixation" may also be done by alcohol and ether, or by corrosive sublimate solution. The cover-glass should be immersed for one-half hour in equal parts of alcohol and ether. After such fixation malarial organisms and nucleated red corpuscles are more readily found.

Fixation with formol is quickly secured. Dilute 1 part of formol with 9 times its volume of water; dilute 1 part of this mixture with 9 times its volume of alcohol. The resulting fluid will fix immersed specimens in one minute. Formalin vapor will fix specimens in fifteen to thirty minutes.

Fixation by heat is best when the white cells are to be studied. According to this method, the cover-slips are put in a dry-heat sterilizer at a temperature of 110° to 115° F. If this cannot be done, place the cover-slips on one end of a copper plate at least a foot long, the other end of which is heated by a Bunsen burner or a gas flame. The cover-slips should be placed on the plate at that point on which water boils when dropped upon the surface of the copper. The boiling-point should be first well fixed at a constant distance from the flame. The cover-slips should be placed face downward, and kept there from one-half to one and one-quarter hour. When cool they are ready for staining.

STAINING. The greatest care should be taken to have a perfectly clean dry cover-glass, which should be handled with forceps to avoid moisture and soiling. (1) The prepared cover-glass should then be immersed for a few minutes in a solution of eosin:

Eosin	0.5
Alcohol (70 per cent.)	100.0

This solution should be diluted one-half before using. (2) The cover-glass should then be dried and stained for three or four minutes in a saturated aqueous solution of methylene-blue, also diluted one-half before using (Chinzinsky-Plehn mixture). Or, instead of the latter, stain for half an hour to several hours in Delafield's hæmatoxylin. This hæmatoxylin stain is made in the following manner: To 400 c.c. of a saturated solution of ammonia alum, add 4 grammes of hæmatoxylin

crystals dissolved in 25 c.c. of strong alcohol. Leave this solution exposed to the light and air in an unstoppered bottle for three or four days. Filter and add 100 c.c. of glycerin and 100 c.c. of methyl alcohol. Allow the mixture to stand until the color is sufficiently dark. Then filter and keep in a tightly stoppered bottle. The stain should ripen for at least two months before using. For blood-work the solution is used in its full strength. By this double stain, a modification of *Ehrlich's hæmatoxylin-eosin mixture*, the red corpuscles are stained red, the nuclei blue, the bodies of the leucocytes light lilac and their nuclei darker, the eosinophile granules a brilliant red.

Ehrlich's Tri-acid Stain. The Ehrlich tri-staining mixture is the best that can be selected for staining. Thayer says the following is a satisfactory modification of Ehrlich's formula:

Saturated aqueous solution of acid fuchsin	2.00
Water	3.00
Saturated aqueous solution of orange-G.	6.25
Saturated aqueous solution of methyl-green	6.00

To be added, drop by drop, while shaking the solution:

Water	15.00
Alcohol	10.00
Glycerin	5.00

Ehrlich's latest formula is as follows:

Saturated aqueous solution of orange-G.	13–14	c.c.
Saturated aqueous solution of acid fuchsin	6–7	"
Distilled water	15	"
Alcohol, 95 per cent. or absolute	15	"
Saturated aqueous solution of methyl-green	12.5	"
Glycerin	10	"
Alcohol, 95 per cent. or absolute	10	"

Mix in foregoing order, using same graduate and rod. Methyl-green is added drop by drop, the mixture being thoroughly stirred.

The stain is spread over the cover-glass specimen with a glass rod, and in from one to five minutes washed off with water. If the cover-glass has not been heated very long, it will not be necessary to keep the stain long in contact with the blood, but specimens which are heated an hour require at least five minutes for the stain to take. After the specimen is stained and washed in water it should be dried between layers of filter-paper and mounted in balsam. It can then be examined at leisure with a $\frac{1}{12}$ oil-immersion lens with diaphragm open.

Specimens heated for one or two hours stain better than those which have been exposed to heat only a short time. The red cells appear orange or buff, the nuclei of the colorless corpuscles green or greenish blue, the neutrophilic granules a violet or lilac color, the eosinophilic granules a deep red. The nuclei of nucleated red corpuscles, when present, are stained an intense deep green, almost black.[1]

Jenner's stain has given satisfaction. It is made as follows: 1.

[1] Thayer, loc. cit.

Grubler's yellow water-soluble eosin, 1.2–1.25 per cent. watery solution. 2. Grubler's medicinal methylene-blue, 1 per cent. watery solution—equal parts. Mix in open basin and let stand for twenty-four hours. Filter and dry precipitate in air or oven at 55° C. two or three times. Use 0.5 gramme in 100 c.c. of pure methyl alcohol. No fixing is required; stain in a small moist chamber one to five minutes. Wash in distilled water till of a pale-pink color. Acid and basic granules are stained; platelets and malarial parasites also show well.

Another method much used and urged by Hewes is as follows: The blood, after fixation, is subjected for four minutes to the modified Ehrlich stain, which is made as follows:

Ehrlich-Biondi-Heidenhain three-color mixture	1.7 gm.
Acid fuchsin	0.05 "
Absolute alcohol	2 c.c.
Distilled water	18 "

After immersion wash the specimen in water and then subject it from one-half to ten seconds to Löffler's solution of methylene-blue. Again wash the specimen, dry, and mount in balsam.

Löffler's solution is saturated alcoholic solution of methylene-blue, 30 c.c.; potassium hydroxide (1 : 10,000 solution), 100 c.c.

The Red Corpuscles or Erythrocytes.

The ordinary red blood-cells measure $\frac{1}{3200}$ inch (7–8 μ); the leucocytes, $\frac{1}{2500}$ inch. In an adult man the red cells number from 5,000,000 to 5,500,000 to the cubic millimetre; in an adult woman the number is usually less, being from 4,500,000 to 5,000,000. There are 5000 to 10,000 leucocytes in a cubic millimetre of blood, or 1 to 350–600 red blood-cells. In thickly spread blood the cells are arranged in the form of rouleaux. If the latter are absent in such a preparation, it is an indication of great reduction in the number of red cells.

In thinly spread films the red cells are recognized by their color and shape. They vary from 6 μ to 9 μ in diameter. The lighter colored centre, due to the biconcavity of the corpuscle, sometimes causes confusion. It must be remembered, too, that the corpuscles readily become crenated, an appearance which may be confounded with pigmentation or other abnormal change. A slight molecular movement is sometimes also seen, which must not be confounded with the amœboid movements in dying cells or with the rapid motion of malarial pigment.

Poikilocytosis. The variations in size and shape are indications of disease. In forms of anæmia the red cells may be larger than normal; they may be irregular in shape, or they may be smaller than normal. Large cells are known as *macrocytes*, small cells as *microcytes*. Cells that are irregular in shape are known as *poikilocytes*. They may be oval, pointed, angular, or reniform.

Achromia. When the red cells are stained, the hæmoglobin takes the orange-G of the tri-colored mixture of Ehrlich, modified by Thayer, causing the red cells to be brilliant yellow or pale orange in tint. An

idea of the amount of hæmoglobin can thus be obtained. When the hæmoglobin is diminished, the centre is pallid, although in extreme poverty of hæmoglobin the colored rim may be only a faint outline (achromic forms).

Nucleated Red Corpuscles or Blasts. They contain one or more nuclei. The stroma takes the golden acid stain and the nucleus the pure basic stain. They are divided in accordance with their size and the depth of the color of the nuclei, into three varieties:

1. **The Normoblast.** It is the size of a normal red blood-corpuscle. The stroma is golden in color; the one or more nuclei are deeply bluish-black and homogeneous. The nucleus occupies one-fourth to three-fourths of the whole corpuscle, and it is deeper in color than the nuclei of the white blood-corpuscle. The normoblast is the parent cell of the red blood-corpuscle.

2. **The Megaloblast.** This cell is larger than a normal red blood-corpuscle. The color of the stroma is less intense than that of the normoblast, and the nucleus is bluish green rather than black, and not compact and homogeneous, showing a well-marked nuclear network. Its nucleus is more compact and more clearly defined than the nucleus of the white blood-corpuscle. The megaloblast is found in the marrow of the embryo and in severe anæmias.

3. **The Microblast.** This is a smaller cell than the normal red blood-corpuscle. There is but little stroma and the nucleus is deep black.

Blasts are found in anæmia. An excess of normoblasts indicates very active regeneration of blood.

Polychromatophiles. These are red blood-corpuscles in which the stroma takes not only the normal acid-staining elements, but also the basic or neutral stains. They are degenerate forms of red blood-corpuscles.

Degenerate Forms. The coloring-matter is irregularly distributed and the stroma appears disintegrated.

When stained in the above-described manner, we can readily find nucleated red cells, but the fibrin line and blood-plates are destroyed as a rule.

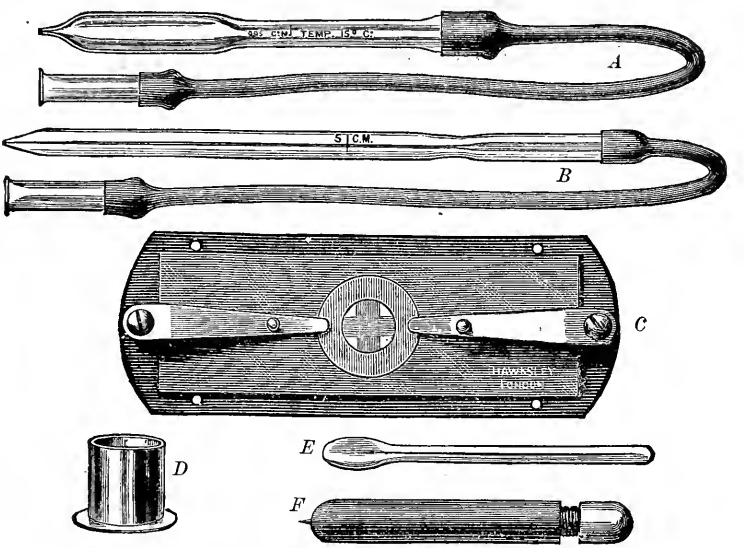
Blood-counting. It is of the greatest clinical importance to be able to estimate the number of red cells in a given quantity of blood, in order that approximately at least we may know of its globular richness. For this purpose hæmocytometers are used.

The hæmocytometers, or blood-counters, most frequently used in this country are those of Gowers and Thoma-Zeiss.

Hæmocytometers. *Gowers' instrument* (Fig. 201) consists (1) of a small pipette, *A*, which when filled holds exactly 995 c.mm. (it is for measuring the diluting fluid); (2) a capillary tube, *B*, graduated for 5 c.mm.; (3) a small glass jar, *D*, in which the dilution is made; (4) a small glass stirrer, *E*, for mixing the blood and diluting fluid in the jar; (5) a small lancet, *F*; (6) a brass stage-plate, *C*, carrying a glass slip on which is a cell $\frac{1}{5}$ mm. deep. The bottom of the cell is divided into $\frac{1}{10}$ mm. squares. On the top of the cell rests the cover-glass, which is kept in place by the pressure of two springs proceeding from the ends of the

stage-plate. 995 c.mm. of the diluting fluid are measured and blown into the mixing-jar; then 5 c.mm. of blood are added and the two thoroughly mixed. A small drop of the mixture is then placed upon the cell, the cover-glass gently adjusted and held in place by the springs. From five to ten minutes should be allowed to elapse, so that the corpuscles will have time to settle to the bottom of the cell. The stage-plate is then placed under a microscope, and the number of red blood-cells in 10 squares counted. This number multiplied by 10,000 gives the number in a cubic

FIG. 201.



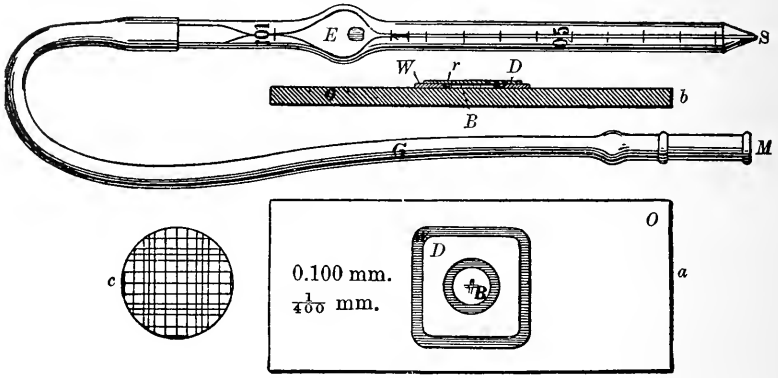
Hæmocytometer of Gowers.

centimetre of pure blood. It is better to count a large number of squares, take the average, and multiply by 100,000. This number is the product of the dilution (200) by the square surface of the cells, 100 (10 × 10), and again by 5, the depth of the cell: 200 × 100 × 5 = 100,000. To facilitate seeing the fine lines marking the squares, a soft black lead-pencil should be gently rubbed over them before the drop of diluted blood is placed on the cell. Counting of the white cells is made much easier if the diluting fluid is colored a pale violet with a very small quantity of gentian-violet. The white cells then appear a distinct blue, while the red cells are unaltered. As diluting fluids, a 1 per cent. solution of common salt, or a 2.5 per cent. solution of potassium dichromate, as recommended by Daland, may be employed; or *Toisson's fluid* can be used. It is made as follows: distilled water, 160 c.c.; glycerin, 30 c.c.; sodium sulphate, 8 grammes; sodium chloride, 1 gramme; methyl-violet, 0.025 gramme.

The *Thoma-Zeiss hæmocytometer* (Fig. 202) is preferred by most clinicians. It consists of a heavy glass slip (*a*), in the middle of which is a cell (*B*) exactly $\frac{1}{10}$ mm. in depth. The cell is limited at the periphery by a circular gutter to prevent fluid placed upon the cell from flowing beyond it between the slip and cover-glass. The floor

of the cell is ruled into squares whose sides are $\frac{1}{20}$ mm. Double lines mark out large squares, each containing 16 small squares. Thick, carefully ground cover-glasses (*D*) are provided in the case. The ordinary Potain *mélangeur* (*S*) is used to measure and mix the blood. It consists of a capillary tube, the upper portion of which is blown into a chamber (*E*) holding 100 c.mm. The stem of the tube is graduated at 0.5 and at 1 c.mm.

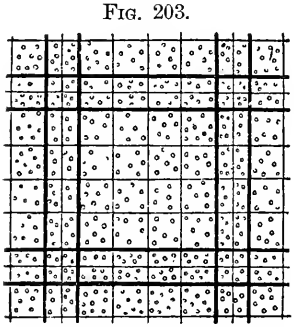
Fig. 202.



Thoma-Zeiss blood-counting apparatus.

To use the instrument, a drop of blood is obtained from the finger or lobe of the ear, the point of the capillary tube is inserted into the drop, and blood sucked up to the mark 1 c.mm. The point of the tube is then quickly wiped free from excess of blood and inserted into the diluting fluid, which is drawn up to the level of the mark 101. The proportion of blood and diluting fluid is then 1 to 100 c.mm. The blood and diluting fluid are now thoroughly mixed. The diluting fluid in the stem of the *mélangeur* is now blown out and a drop of the blood-mixture placed on the cell. The cover-glass is adjusted carefully to avoid bubbles and to prevent escape of the fluid between it and the slip. The cover-glass is now pressed firmly down until Newton's color-rings appear, and then the slip is allowed to stand for five or ten minutes, until the corpuscles have settled to the bottom of the cell.

Fig. 203.



Appearance of blood in the Thoma-Zeiss cells.

The cell is ruled into 400 small squares, groups of 16 squares being separated by double lines. The surface of a square is $\frac{1}{400}$ square millimetre, and the depth of the cell being $\frac{1}{10}$ millimetre, the space overlying each square is $\frac{1}{4000}$ cubic millimetre. In estimating the number of corpuscles in a cubic millimetre of blood, multiply the number of corpuscles counted in all the squares by 4000 and the product

by the dilution, which is 1 to 100 or 1 to 200, according as 1 or 0.5 c.mm. of blood has been used. The last product is now to be divided by the number of squares which have been included in the count, the quotient being the number of corpuscles in a cubic millimetre of blood. The results are accurate in proportion to the care exercised in the measurement of the blood and diluting fluid, and especially in proportion to the number of squares counted.

In the estimation of *white blood-cells* the pipette made by Zeiss is employed. In this instrument the blood is diluted 10 times by a solution of 1 part of a 0.3 per cent. acetic acid solution to 10 parts of distilled water. By means of this solution red cells are dissolved and the nuclei of the white cells are rendered distinct and easy of recognition. Toisson's fluid, mentioned above, may also be used. The ordinary Thoma-Zeiss slide is employed, and the average number of white cells in each small square is multiplied by 40,000. To obtain accurate results, 4 entire fields should be counted.

Hæmatocrit. The hæmatocrit is an instrument devised for the estimation of the percentage-volume of red corpuscles by means of centrifugal force. In Daland's article will be found a full description of the instrument, and from the same article the following method of using it is abstracted: "The finger or ear and apparatus are prepared as above. An incision is made deep enough to produce a good-sized drop of blood. This is drawn into a hæmatocrit tube by means of suction through an attached rubber tube, one finger being placed over the free end when the rubber tube is removed to prevent the loss of blood. The filled tube is then placed in the frame of the hæmatocrit and a second prepared exactly as the first. The larger wheel is then rotated for two minutes at 77 turns of the handle-crank per minute (giving altogether 20,000 rotations of the frame), and the result read from the scale multiplied by 2 gives the percentage-volume. It has been found by experimenting that each division upon the scale of the hæmatocrit tube represents 100,000 corpuscles." This procedure is not available for the determination of the volume of leucocytes unless the number exceeds 20,000, at and above which number an approximate estimate may be readily determined. A distinct white band appearing between the red cells and the clear fluid, having the width of one line, may be considered as representing from 15,000 to 20,000 leucocytes.

Number of Red Blood-corpuscles in Health. The normal number of red cells—as already stated—is approximately 5,000,000 per cubic millimetre. They may be reduced to 500,000. A reduction below 3,000,000 indicates grave anæmia. When the reduction is below 1,500,000, the anæmia is said to be pernicious or malignant. It must be remembered that temporarily the red cells are reduced during menstruation and lactation. At puberty there is also a reduction. On the other hand, when the blood is concentrated by profuse sweating or exhaustive diarrhœa, the number of red cells is increased, while they are lowered when the blood is diluted by large draughts of fluid or by subcutaneous injections of fluid. A cold bath may temporarily concentrate the peripheral blood, and thereby increase the number of cells. Red cells

are always lessened in the aged, and are reduced in number after great exertion. They are increased in number after fasting, and diminished after a meal, particularly if much fluid is taken.

Oligocythæmia. Oligocythæmia is the name applied to a diminution in the number of red blood-cells, from whatever cause. It is usually associated with *oligochromæmia* (deficiency of hæmoglobin), which, however, in idiopathic anæmia is absolute, not relative. Marked oligocythæmia can be detected with the microscope alone, and can be estimated accurately with the hæmocytometer or hæmatocrit. (See Fig. 202.)

The White Corpuscles.

The white or colorless corpuscles are recognized by their absence of color, by their irregular shape, and their size, which is larger than that of the red, and by the amœboid movements which they undergo, particularly if placed on a warm stage. They number from 4000 to 10,000 per cubic millimetre. They are readily recognized by the peculiar affinity which they have for various aniline dyes. They appear as granular nucleated cells in stained specimens. The method of staining has been described, and the varieties of leucocytes found in normal blood, indicated on page 573. In addition to determining the number by counting, as described in the paragraph which gives the method of counting the red cells, a so-called differential count is made. This count enables us to determine the proportion of the many varieties of leucocytes.

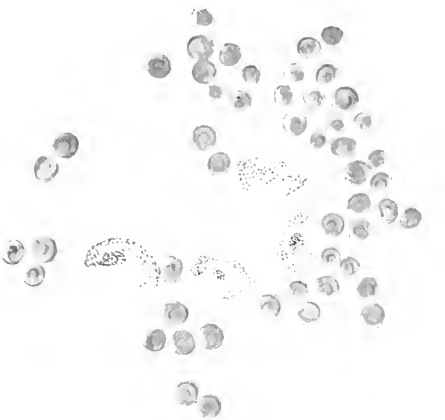
In counting the white blood-corpuscles, Phear advises the use of the camera lucida (see below). The most convenient form is the Zeiss-Abbé drawing camera, used with the stage of the microscope in a horizontal position. The image of the field is projected on a piece of paper or cardboard lying horizontally on the table immediately to the right of the microscope-stand. The ruled squares on the floor of the hæmocytometer cell are accurately marked out on the cardboard. The image of the corpuscles which lie on the unruled part of the cell floor is thrown by means of the camera on the cardboard, and the corpuscles which appear to lie over each square are included in the count. It is convenient to use a mechanical stage. It is essential that the eye-piece, objective, and tube-length used during the count should be the same as on the occasion of marking out the squares on the cardboard. For the dilution of the blood, that recommended by Sherrington[1]—consisting of distilled water, 300 cubic centimetres; sodium chloride, 1.2 grammes; neutral potassium oxalate, 1.2 grammes; and methylene-blue, 0.1 gramme—is excellent. The blood-corpuscles are not stained, but their shape and color are preserved. The nuclei of the white corpuscles are in every instance stained, facilitating the distinction of the white from the red cells. For the differential count of the white corpuscles it is desirable to work with an immersion lens.

Varieties of Leucocytes. In the normal blood there are found the following varieties of leucocytes: 1. *Small mononuclear* forms, which are

[1] Proceedings of the Royal Society, vol. lv.

PLATE X.

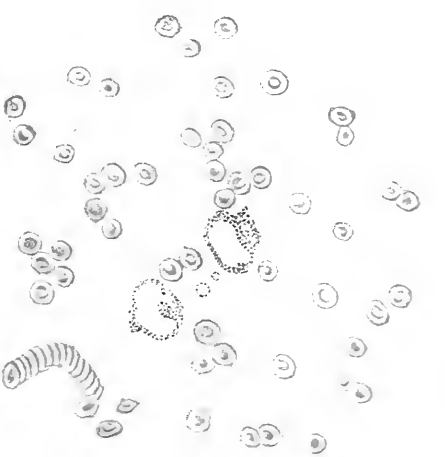
Fig. 1.



Blood from Case of Pneumonia, showing Leucocytes.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

Fig. 2.



Normal Blood, showing Rouleaux and Leucocytes.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

cells about the size of a red blood-corpuscle, and have a round, large, deeply staining nucleus, surrounded by a narrow rim of non-granular protoplasm. These are known as *lymphocytes*. 2. *Large mononuclear* leucocytes several times as large as the small mononuclear form. They have a round or oval nucleus, with a relatively larger amount of non-granular protoplasm. 3. *Transitional* forms, which resemble the last named, except that the nuclei are indented or S-shaped. The occurrence of a few "neutrophile" granules in the protoplasm is generally described. 4. *Polymorphonuclear* leucocytes. These are usually about the size of the transitional variety, but may be somewhat smaller. The nuclei are twisted and irregular and stain deeply. The protoplasm contains granules that stain by a combination of basic and acid dyes, but by neither alone. The cells are therefore called "*neutrophiles.*" Some deny the existence of "neutrophile" granules, claiming that they are really acidophilic. 5. Leucocytes similar to the last form, except that their protoplasm contains highly refractive granules that are stained by acid dyes alone. For this reason they are usually called *eosinophiles*. 6. *Mast-cell*, usually described as an occasional element of blood; a tissue-cell, seen in about 0.5 per cent. With Ehrlich's triacid stain it appears as a polymorphonuclear cell, with distinct vacuoles in the protoplasm, representing large unstained granules. The granules stain with basic stains, such as methylene-blue and dahlia. The cell is large and somewhat irregular, and presents one of Ehrlich's basophilic granulations.

The proportion of each variety in the normal blood is fairly constant: lymphocytes, 15 to 25 per cent.; polymorphonuclear, 65 to 80 per cent.; large mononuclear and transitional forms, 6 per cent.; and eosinophiles, 2 per cent. or less. (See Plate X.)

Differential Counting. After the specimen is carefully stained with the triple solution it is ready for differential counting of the white cells, as well as determining the presence of nucleated red cells. To make the differential count, a large number of leucocytes should be studied. The best plan is to begin at the upper left-hand corner of the blood-film and count across the film to the right-hand corner. Then move the slide so that an adjacent field comes into view, when the process is to be repeated. In this manner the entire field is covered. In ordinary leucocytosis a thousand leucocytes can be seen in a $\frac{7}{8}$ inch cover-glass specimen. We may find an abnormal variety of leucocytes; an abnormal proportion of some one of the normal leucocytes; an abnormal number of all the leucocytes.

Fluid Preparations. A. G. Phear lays stress on the advantages of fluid preparations over the cover-slip method. In the cover-slip method leucocytes are inevitably flattened and distorted in the process of making and fixing the film; some are washed away during the staining; others obscured by the red corpuscles. In the fluid preparation the white cells are fixed and preserved as approximately spherical bodies; camera lucida drawings and measurements of them may be relied upon as accurate. A solution of methylene-blue (0.2 per cent.) in 40 per cent. alcohol is used for diluting the blood. The red corpuscles are laked so that the white cells alone remain conspicuous. "A small quantity of the diluting solu-

tion is added to a drop of blood on a glass slide and the two are thoroughly mixed by directing a current of air through a pipette on to the surface of the fluid. The fluid is allowed to spread as a thin film under a cover-glass and the edges then sealed with vaseline." The contour of the normal polymorphonuclear cells is rounded. Their diameters vary from 9 μ to 12 μ. The complex nucleus can be made out by changing the focus, the nucleus being, in fact, "an undivided elongated body, in places deeply constricted, elsewhere bulged into rounded lobes." The lymphocytes and the large hyaline cells represent the extremes of cells, differing in the amount of protoplasm around the nucleus; all grades are readily found. The nuclear diameter is fairly constant in these cells, varying only between 4.5 μ and 5.5 μ. Large oval cells, as much as 14 μ in length, with the nucleus large and irregular, usually reniform, are seen. The protoplasm becomes rapidly and uniformly stained an opaque blue color with methylene-blue. The coarsely granular or eosinophile cells (diameter from 9.5 μ to 10.5 μ) are at once recognized in the film prepared with methylene-blue solution, notwithstanding the absence of an acid dye; the large refractile granules are stained a greenish color. The cells containing basophile granules (diameter about 8 μ) have a characteristic appearance. The protoplasm contains granules of medium size, many of which are aggregated in one or more deeply stained clumps near the surface of the cell. The non-granular part of the protoplasm is stained a peculiar mauve or purple color. The nucleus is usually massed at the centre of the cell, and stains a slate or grayish-blue color.

Separate counts over different areas of one preparation give uniform results, showing that the blood was evenly mingled with the diluting fluid. Not less than 500 cells should be enumerated at a time; the more the better. It is desirable to use a mechanical stage and to work with an immersion lens. The blood should always be procured, if possible, before the first meal of the day is taken, since this is the time at which the influence of meals is least likely to be evident.

Ross' Method of Examining for Parasites in a Large Drop of Blood. Spread a large drop of blood with a loop or slide, dry in air or gentle heat, add aqueous solution of eosin (Romanowsky) for ten to fifteen minutes (the eosin takes out hæmoglobin, as specimen is not fixed, and stains stromata of corpuscles, parasites, and leucocytes); wash very gently, stain for a few seconds with methylene-blue (Romanowsky), wash gently and examine. One has practically twenty times the amount of blood by this method as by the ordinary method.

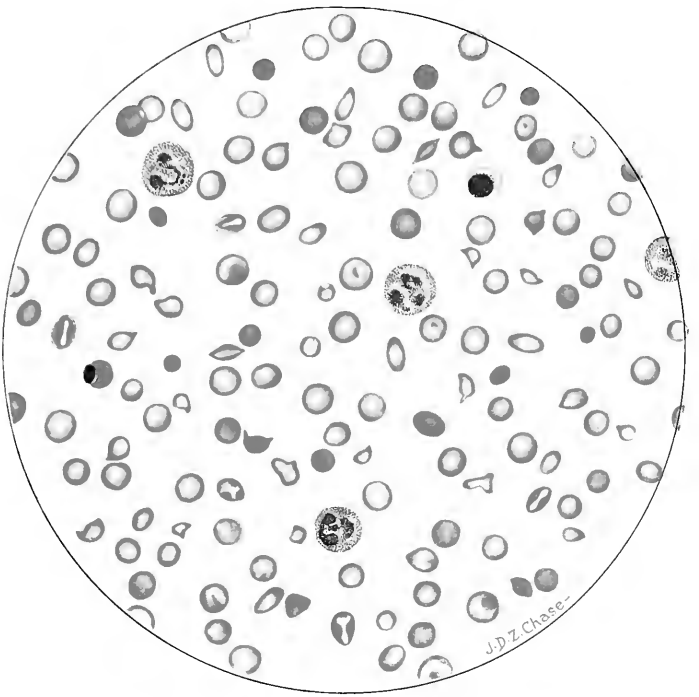
Leucocytosis.

Leucocytosis is a temporary increase in the number of white blood-cells of the same morphological varieties as in health, with an *excess* of the *polynuclear forms* (neutrophile leucocytosis). Such increase may be physiological or pathological, as indicated in the following:

Physiological Leucocytosis. (1) Pregnancy (14,000 and upward); (2) during digestion (from 1000 to 7000 above normal; more in children); (3) new-born (12,000).

PLATE XI.

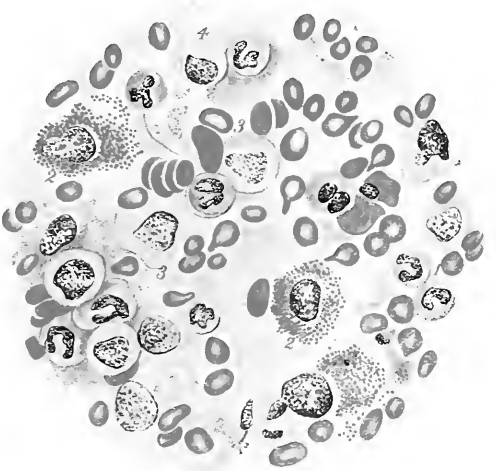
FIG. 1.



Secondary Anæmia.

Pallor of red cells. Moderate poikilocytosis. Presence of normoblasts. Slight increase in polymorphonuclear leucocytes.

FIG. 2.



Leukæmic Blood.

1. Polynuclear leucocytes.
2. Eosinophile cell (mononuclear).
3. Large mononuclear leucocyte.
4. Small lymphocyte.

(Oc. 4, ob. 1-12 immersion.) Drawn by J. D. Z. Chase.

Pathological Leucocytosis. An excess of leucocytes occurs in the following diseases: (1) leukæmia; (2) diseases of lymphatic glands; (3) disease accompanied by exudations, as pleurisy, pericarditis, meningitis, polyarthritis, and especially croupous pneumonia; (4) inflammatory conditions associated with exudation, as appendicitis, pyonephrosis, perinephritic abscess, tonsillar and retropharyngeal abscess, acute pancreatitis, cholangitis; (5) many acute infectious diseases, as varicella, pertussis, variola, vaccinia, epidemic cerebrospinal meningitis, cholera, typhus fever, trichinosis, glanders, diphtheria, scarlet fever, erysipelas, pyæmia and septicæmia, rheumatism, abscess, and gangrenous inflammation; (6) after hemorrhage; and (7) just before death,—leucocytosis of agony. On the other hand, *leucocytosis* is not found in uncomplicated cases of (1) *influenza;*[1] (2) uncomplicated cases of *typhoid fever;* (3) *tuberculosis* when not associated with cavity formation or hyperplasia of lymphatic glands;[2] (4) many forms of carcinoma and sarcoma, gastric ulcer, and benign pyloric stenosis,[3] although it may be present in gastric carcinoma; (5) *measles.*

Leucopenia. Diminution of the number of leucocytes is seen (1) in starvation, as in cancer of the œsophagus; (2) the latter weeks of typhoid fever; (3) leukæmia complicated by infection.

Diagnostic Value of Leucocytosis. The value in diagnosis of determining the presence of leukocytosis is great. Its absence excludes the first series of conditions; its presence the last. If leucocytosis is present in the course of or convalescence from typhoid fever, it points to a complication, as thrombosis. A post-febrile rise due to a complication may be distinguished from a true relapse by an increase of the white cells.

It must be remembered that few symptoms or signs are pathognomonic. We deal largely with averages in diagnosis. The facts about leucocytosis are on a par with other data for diagnosis. In the majority of cases, indeed, the presence or absence of leucocytosis represents more than ordinary data. As an isolated fact, however, as with other symptoms, it is of no value whatsoever; but when considered in relation to other data, as the temperature, the digestion, the circulation, the excretions, etc., the number of leucocytes is important.

Leucocytosis is best determined with a hæmocytometer. Dry preparations, according to Ehrlich's method, are necessary for a study of the various forms of leucocytes. (See under Leucocythæmia, page 839, and Plate XI.)

Increase of Special Leucocytes. Lymphocytosis. Both forms of lymphocytes are seen. A *relative* increase, with or without a total increase of leucocytes, is seen in infants, and is common in rickets and hereditary syphilis. It is found in some forms of scurvy. In adults lymphocytosis occurs in chlorosis and pernicious anæmia, and in the secondary anæmia of syphilis and typhoid fever. It occurs in hæmophilia, in adenitis, and splenic tumors. It is also found at the end of scarlet fever and measles, typhoid fever, gout, in pneumonia with delayed resolution,

[1] Boston Medical and Surgical Journal, March 22, 1894.
[2] Stein and Erbman, Deutsch. Arch. f. klin. Med., Bd. 56.
[3] Schreuger, Zeitschr. f. klin. Med, 1895, 27, 475.

and in some forms of phthisis (Cabot). *Absolute* lymphocytosis occurs in lymphatic leukæmia.

Eosinophilia. An increase in the percentage of eosinophiles, with or without leucocytosis, is seen in many affections of the bones, of the skin, of the female genital apparatus, and in asthma. The bone diseases are osteomalacia, sarcoma, carcinoma, and those affections of the bone and marrow in which pernicious anæmia and splenic myelogenous leukæmia are seen. The skin diseases are urticaria, pellagra, herpetiform dermatitis, pemphigus, herpes, eczema, prurigo, psoriasis, lupus, and myxœdema. In the eruption of scarlet fever and syphilis eosinophiles are increased, but not in measles or smallpox. In various affections of the uterus and ovary, in functional disorders connected with the same and with pregnancy, as vasomotor affections, the eosinophiles are increased. They are also increased in gonorrhœa and prostatitis. They are increased in bronchial asthma at the time of the paroxysm to as much as 10 to 20 per cent. of the number of leucocytes. Such increase is not seen in renal and cardiac asthma. Brown found marked increase in the eosinophiles in trichinosis —in fact, established the differential count as a method of diagnosis. They are also increased in other forms of the helminthiases, but not constantly as in trichinosis. *Diminution* in the eosinophiles takes place during digestion and in most of the infectious disorders accompanied by leucocytosis, and in typhoid fever and diphtheria. Malignant disease with hemorrhage which causes leucocytosis is, however, associated with diminution of the eosinophiles. Neusser has indicated the following diagnostic points of value in eosinophilia. They are given by Cabot as follows:

1. In the diagnosis between puerperal mania and puerperal sepsis eosinophilia points to the former.
2. Between a tumor connected with the genital system and one not so connected, eosinophilia points to the former.
3. In determining whether a given case of hysteria, neurosis, or psychosis is likely to be benefited by castration, the presence of eosinophilia favors the operation.
4. In malignant disease an eosinophilia points to a metastasis in the osseous system (tumors of the spleen are not included in this rule).
5. In cases of doubtful syphilis, eosinophilia associated with lymphocytosis (see above) speaks in favor of syphilis.
6. The diagnosis of any obscure form of "uric acid diathesis" is helped by finding an increase of eosinophiles.
7. In distinguishing malignant liver disease from other liver disease, eosinophilia points to the latter.

Pathological Leucocytes. MYELOCYTES. The occurrence of myelocytes in the blood is pathological. Their well-known occurrence in myelogenous leukæmia and pernicious anæmia need not be referred to. They have been found, however, in a number of infections, particularly in diphtheria, but usually only when there is present a grave form of anæmia. Their occurrence is not of great diagnostic value. They are *non-amœboid*. They are large mononuclear neutrophiles or eosinophiles (eosinophilic myelocyte), with large, well-defined, lateral, spherical

nuclei. Occasionally they are small, when they are recognized by the granules and the very pale large nucleus. The *mastzellen* are coarsely granular basophiles. The nucleus is fragmented or three-lobed.

NEUSSER'S GRANULES. When making a differential count we also study certain other granulations in the leucocytes. Neusser has described perinuclear basophilic granulations in the leucocytes, which are demonstrated by staining the blood with the following modification of Ehrlich's triple stain :

Saturated aqueous solution of acid fuchsin	50 c.c.
Saturated aqueous solution of orange-G	70 "
Saturated aqueous solution of methyl-green	80 "
Distilled water	150 "
Absolute alcohol	80 "
Glycerin	20 "

The granules in question occur as separate bodies or as groups, lying in the protoplasm immediately around the nucleus. They are met with in the mononuclear forms in particular, and, according to Neusser, are composed of some derivative of the nucleo-albumin and indicative of increased uric acid formation. The granules are claimed to occur in gout, and also in certain cases of myelogenous leukæmia, tuberculosis, and diabetes, and to be significant of the uric acid diathesis " in the clinical sense." In discussing Neusser's paper, Löwit called attention to the fact that similar granules occur in the leucocytes of the bone-marrow of rabbits.

Other observers have found these granules in a variety of conditions, and incline to regard them of less significance than Neusser is disposed to admit. Futcher has shown that the granules may be found in any blood by modifying the stains.

Platelets.

The blood-platelets have generally been considered structureless bodies, and to be the degeneration-products of red cells or corpuscular elements of uncertain nature; recent workers have demonstrated that the platelets are amœboid nucleated cells; and have confirmed the theory of their thrombus-forming property (" thrombocytes ").

Argutinsky shows the nucleus by fixing the blood in mercuric chloride and alcohol and staining by the Nocht-Romanowsky method.

The " cell " is an amœboid finely granular spindle cell with an oval nucleus.

Jenner's stain will usually reveal the platelets in large numbers.

In the fresh specimen blood-platelets appear as irregular bodies, one-eighth to one-fourth the size of a red cell, of a pale bluish-white color; they tend to clump in irregular masses and rapidly disappear; they are frequently increased in tuberculosis; a marked increase is noted in acute conditions of hæmatolysis; diminution has been noted in hæmophilia and purpura.

Fibrin.

The fibrin of the blood can be seen under the microscope in a thick specimen of blood after standing for some time, as a filamentous network

between the cells; platelets or their remains are usually seen forming a nucleus from which the fibrin threads spread.

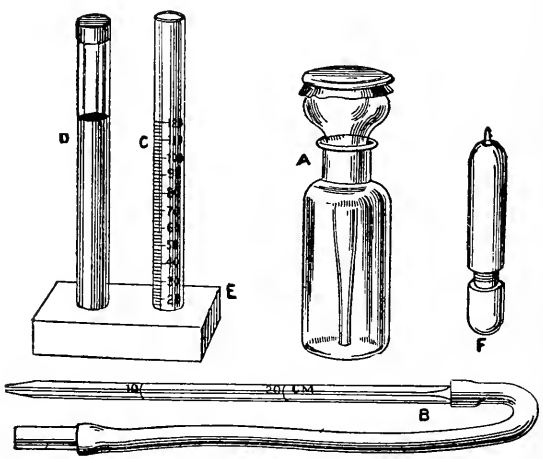
Fibrin is increased in infectious and inflammatory diseases; diminished in amount in pernicious anæmia and purpura.

The Hæmoglobin.

An estimation of hæmoglobin is made in order to determine the richness of red cells in this substance. For this purpose a hæmoglobinometer is used.

Hæmoglobinometers. **Gowers' hæmoglobinometer** (Fig. 204) consists of (1) a closed tube, *D*, containing coloring-matter representing the color human blood should have normally if diluted 100 times; (2) a corresponding empty tube, *C*, graduated in an ascending scale from 10 to 120; (3) a capillary glass tube, *B*, marked at 20 cubic millimetres; a small guarded lancet, *F*, and a small bottle with a pipette stopper, *A*, for distilled water. A few drops of distilled water are first placed in the empty tube, *C*, to prevent the coagulation of the blood, which would

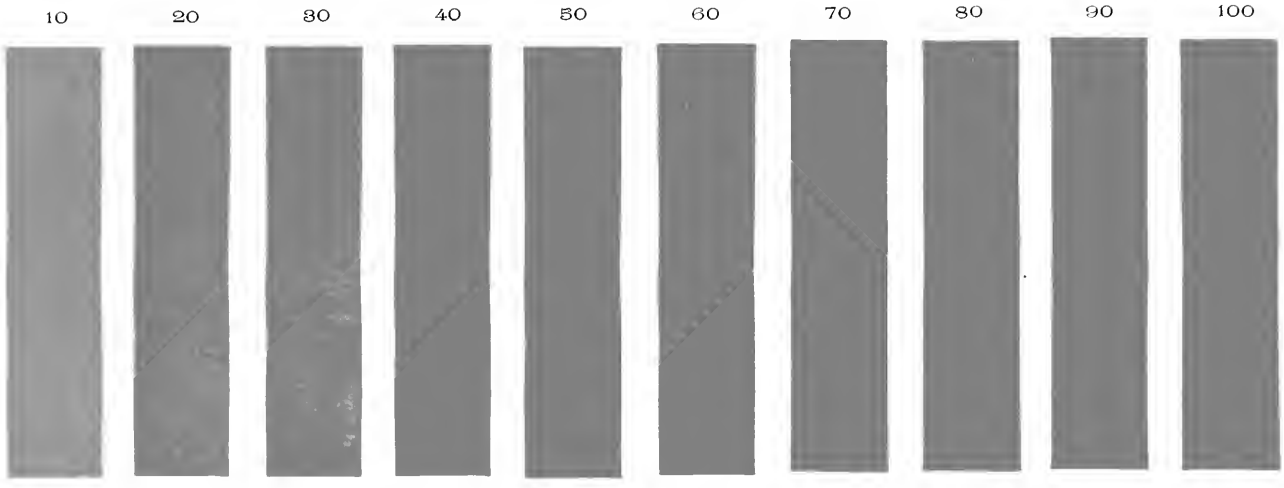
FIG. 204.



Gowers' hæmoglobinometer.

occur if the blood were first put in the tube. The finger or lobe of the ear, previously cleansed with water and ether, is then deeply stabbed with the lancet, so that the blood will flow freely, care being taken to avoid squeezing the punctured part; 20 cubic millimetres of blood are then quickly drawn up in the capillary tube and at once blown into the graduated tube, which is shaken to allow the blood to become diffused in the water. The tubes containing the standard coloring-matter and the diluted blood are now held up, side by side, against a sheet of paper, and distilled water added, drop by drop, with repeated shakings, until the colors in the two tubes match. The height to which the column of diluted blood and water has risen in the graduated tube represents the percentage of hæmoglobin contained in the blood tested.

PLATE XII.



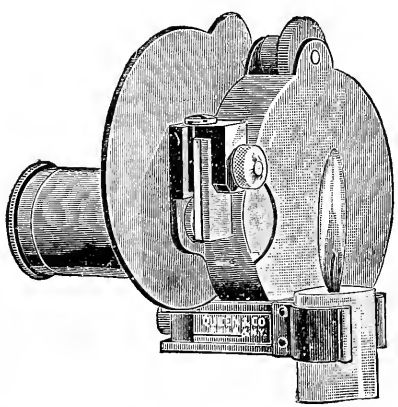
Tallquist's color scale for estimating Hemoglobin.

Fleischl's hæmometer consists of a small metal table with an aperture in the middle, under which is a reflector made of plaster-of-Paris. The opening is occupied by a small well having a glass bottom and divided into two equal compartments. The standard color of the blood at different dilutions is represented by a wedge of glass, colored with Cassius purple, which is, of course, pale in color at the extreme edge and deepens in intensity with its thickness. This wedge of glass is moved under the table by a rack and pinion, and is accompanied by a graduated scale. One-half of the well receives simply the light from the plaster-of-Paris reflector, while the other rests upon the ruby glass and obtains light through it. The light from a candle, gas-jet, or oil lamp must be used. A small pipette and several capillary tubes about $\frac{3}{8}$ inch in length, and mounted on slender metal handles, are employed to obtain the necessary amount of blood; each tube will hold sufficient normal blood to produce, when properly diluted, a color corresponding to that of the ruby glass at the 100 mark. For use, one end of a capillary tube is carefully lowered upon a drop of blood, which immediately fills it; the tube is then at once washed in one of the compartments of the well, which contains some water. The compartments are now equally filled with water, and the well so placed that the side containing blood receives yellow light, as from a candle, while the other receives light through the wedge of glass. The glass is now moved by the rack and pinion until the intensity of the color in the two compartments is the same, and the percentage is then read off through the small opening behind the well.

Both Gowers' and Fleischl's instruments are about equally accurate, and both are graduated for a higher percentage of hæmoglobin than is the average among Americans, which may be as low as 96 per cent.

Tallquist color-scale test for hæmoglobin, in which the drop of blood is taken on white filter-paper and compared with a scale of colors by daylight, is simple, but unless it is done immediately the test seems very uncertain. (Plate XII.)

Fig. 205.

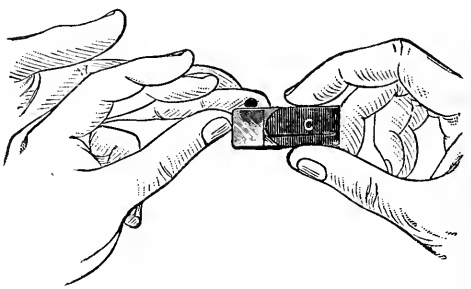


Dare's hæmoglobinometer.

Dare's hæmoglobinometer is compact and reliable, and has the advantage of using undiluted blood. It consists of a large flat pipette which

fills by capillary attraction. (Fig. 206.) This pipette and a color-scale are compared directly against a candle-flame, being observed through a camera

FIG. 206.



Dare's hæmoglobinometer.

tube; the color of the scale is made to agree with that of the blood, and the percentage of hæmoglobin reckoned accordingly. (Fig. 205.)

Color-index. The hæmoglobin usually increases or diminishes with increase or diminution of the red cells. If there is any variation from this percentage, the determination of this variation is known as the color-index. In a healthy individual with 5,000,000 red cells per cubic millimetre the normal percentage of hæmoglobin should be 100. We then say the color-index = 1. If the hæmoglobin is diminished, the color-index is less than 1. The color-index is estimated, first, by reducing the count of the cells to a percentage; second, by dividing this percentage into the hæmoglobin percentage. Thus if the normal percentage of red cells is present—that is, 100—and the hæmoglobin is reduced to 50 per cent., the color-index is $\frac{50}{100}$, or 0.5. Reduction of the red cells to 2,500,000 = 50 per cent. of the normal; now, if the hæmoglobin is 40 per cent., the color-index will be $\frac{40}{50}$, or 0.8. This is true of secondary anæmias. In pernicious anæmia, on the other hand, the color-index is plus, and serves perhaps as the most important sign in distinguishing primary and secondary anæmia. A blood-count of 2,000,000 with hæmoglobin of 60 to 70 per cent. is often seen, or of 1,000,000 with hæmoglobin of 40 to 50 per cent.

Oligochromæmia. Diminution in the amount of hæmoglobin is seen in anæmia, and usually the reduction is lower than the reduction of the red cells. In chlorosis the reduction in hæmoglobin is very great, and in consequence the color-index is lower than in secondary anæmias. The average hæmoglobin percentage in a large number of chlorotic cases studied by Cabot and by Thayer was about 42 per cent. At the same time in most of these cases the number of red corpuscles was over 4,000,000.

It has been observed by Justus that after beginning mercurial inunctions in syphilis the hæmoglobin may drop from 10 to 20 per cent.; this has been considered a valuable diagnostic aid and has been repeatedly confirmed.

Melanæmia. Melanæmia is a rare condition, in which black, brown, or yellow granules are seen floating, either free among the blood-cells, or

more commonly enclosed in cells resembling leucocytes. Melanæmia occurs in malarial fevers, particularly the chronic forms, and in melano-sarcoma and relapsing fever.

Lipæmia is the presence in the blood of fats, usually in the form of small droplets, easily detected by the microscope. The diagnosis can be confirmed by treating the fresh preparation with a 1 per cent. solution of osmic acid, followed by a weak aqueous solution of eosin. The fat-drops will appear black among the faintly stained acid corpuscles. A saturated solution of Sudan-3 in 96 per cent. alcohol will stain fat-drops bright red or orange. Lipæmia occurs in chronic alcoholism, chronic nephritis, and diabetes, and after injuries to the bone-marrow.

The Glycogen Reaction and Iodophilia. This reaction consists in the appearance of mahogany-brown granules in the leucocytes and plasma after staining with iodine 1 part, potassium iodide 3 parts, water 100 parts, and pulverized acacia in excess. It is more evident in conditions of purulent or serous exudates and inflammatory processes especially where leucocytosis coexists. It is considered due to glycogen or an amyloid-like substance

Bremer's Specific Reaction of Diabetic Blood. Saturated aqueous solutions of eosin (watery) and methylene-blue are mixed so that a neutral reaction is obtained. The resulting precipitate is gathered on filters, dried and powdered; to 24 parts of this, 6 parts of powdered methylene-blue and 1 of eosin are added: of this mixture, 0.025–0.05 gramme is dissolved in 10 c.c. of 33 per cent. alcohol. Stain specimen for four minutes after fixing by boiling in alcohol and ether (equal parts) for four minutes. Diabetic blood has a greenish tint. Normal blood is reddish violet. Microscopically, the same difference is seen.

Williamson finds that diabetic blood and urine decolorize methylene-blue. For this test, 2 c.mm. of diabetic blood are dissolved in 40 c.mm. of water, 1 c.c. of methylene-blue (1 : 6000) and 40 c.mm. of liquor potassæ are added; place vessel in boiling water for four minutes. Diabetic blood decolorizes this solution; normal blood leaves it a deep blue.

Alkalinity of the Blood. The total alkalinity of the blood is best determined by Landois' titration method, as follows: Prepare a decinormal solution of tartaric acid by dissolving 7.5 grammes of the chemically pure salt in 1 litre of distilled water. By diluting, centinormal and millinormal solutions are obtained. Prepare a series of solutions as follows:

I. contains 0.9 c.c. of centinormal tartaric acid solution + 0.1 c.c. of saturated potassium sulphate solution.

II. contains 0.8 c.c. of centinormal tartaric acid solution + 0.2 c.c. of sulphate solution.

IX. contains 0.1 c.c. of centinormal acid + 0.9 c.c. of sulphate solution.

X. contains 0.9 c.c. of millinormal acid + 0.1 c.c. of sulphate solution.

XVIII. contains 0.1 c.c. of millinormal acid + 0.9 c.c. of sulphate solution.

In each of a series of watch-glasses mix 1 c.c. of fluid (each watch-glass containing a different strength, as in the series above given) with 0.1 c.c. of blood. This can be done by a graduated pipette. The pipette of a Thoma-Zeiss hæmocytometer answers very well.

Test the contents of each watch-glass with a strip of delicate litmus-paper, and note in which solution the reaction is neutral. This operation must be done quickly, the whole process not taking more than one and a half minutes. (V. Jaksch.)

Suppose 0.4 c.c. of tartaric acid neutralizes 1 c.c. of blood, then 0.4 c.c. of tartaric acid will neutralize 0.0016 gramme of caustic soda; therefore 0.1 c.c. of blood = 0.0016 sodic hydrate, and 1 c.c. = 0.16. The normal alkalinity is 1 part of NaOH to 26 to 30 parts of blood, or 1 c.c. of blood = 0.33 to 0.38 gramme of NaOH.

Behrend's Method of Testing the Alkalinity of Blood. 1 c.c. of blood is centrifugalized with 5 c.c. of a 1 per cent. NaCl solution; first the blood-serum, then the corpuscles are titrated with a 1 : 50 acid solution, litmus being used as indicator.

The alkalinity of the blood is diminished in: 1. Fevers and cachexias. 2. Toxic conditions, as uræmia, diabetes, and jaundice; or certain poisons, as carbon dioxide and phosphorus. 3. Pernicious anæmia, simple anæmia, and leukæmia. 4. Chronic articular rheumatism and gout (*not* in acute articular rheumatism)—this may perhaps be due to the accompanying anæmia. It is increased, perhaps, in chlorosis, though this is doubted by some authorities.

Uric Acid. **Garrod's Test.** By this test we can determine the presence or absence of *large* amounts of uric acid in the blood. A few cubic centimetres of blood-serum or of serous fluid are placed in a watch-crystal and treated with 6 to 10 drops of a 30 per cent. solution of acetic acid. Immerse a linen thread in the fluid, and keep it at a low temperature for from twelve to twenty-four hours. If uric acid is present in large amounts, at the end of twenty-four hours crystals collect upon the thread. Their nature is determined by the microscope (see Urine) and the murexide test. The serum may be secured by a blister.

Specific Gravity. The specific gravity of the blood is best determined by the following method: Prepare a series of solutions of water and glycerin in such proportions that they form a series gradually ascending in specific gravity from 1040 to 1080. Place from 80 to 100 c.c. of each solution in a series of small glass jars and bring a drop of blood exactly in the middle of each, as follows: A hypodermatic syringe is connected by a small rubber tube with a right-angled glass capillary tube. A drop of blood is obtained from the finger in the usual manner, and is drawn by means of the syringe into the capillary tube. By a gentle motion of the syringe a small drop is expelled into the fluid from the point of the tube. The drop will remain stationary if the specific gravity of the fluid equals that of the blood; it will sink if the fluid be of less specific gravity than that of the blood, or will rise if the fluid has a greater specific gravity than the blood. By repeated examination the specific gravity of any specimen can be easily determined. The glycerin mixture can be preserved by the addition of a small amount of thymol, and may be used a second time; but in that case it is necessary to determine its specific gravity anew before it is used.

By the specific gravity one can estimate the amount of hæmoglobin because the former runs parallel to the percentage of the latter. Two

methods are employed—the water-and-glycerin method and the method of Hammerschlag.

Hammerschlag's method is as follows: Mix in a urinometer glass such quantities of chloroform and benzol that the specific gravity is about 1059. Take a drop of blood from the punctured ear with a medicine-dropper or a capillary tube, and blow it into the chloroform-benzol mixture. The blood does not mix, but floats like a red bead. Add chloroform, drop by drop, if the blood sinks to the bottom; add benzol if it rises to the top. After each addition stir the mixture with a glass rod. When the drop remains stationary, its specific gravity is the same as that of the fluid. Take the specific gravity and you have the specific gravity of the blood. Air should not be blown into the fluid with the blood drop. The following table gives the relations of the specific gravity to the hæmoglobin, from which an estimate of the hæmoglobin can be made:

Specific gravity.			*Hæmoglobin.*			
1033	to	1035	25	to	30	per cent.
1035	"	1038	30	"	35	"
1038	"	1040	35	"	40	"
1040	"	1045	40	"	45	"
1045	"	1048	45	"	55	"
1048	"	1050	55	"	65	"
1050	"	1053	65	"	70	"
1053	"	1055	70	"	75	"
1055	"	1057	75	"	85	"
1057	"	1060	85	"	95	"

The specific gravity of the blood is normally less in women, and is diminished in severe symptomatic anæmias, pernicious anæmia, chlorosis, leukæmia, and, according to Monti,[1] in nephritis. It is increased during infancy and in acute febrile diseases, as pneumonia, pleurisy, etc.,[2] and also in diphtheria.[3]

Coagulation-time is an estimate of the time required for the blood to clot. It is valuable, particularly in some surgical relations and various hemorrhagic conditions. The method devised by Wright is the best at our command.

The normal coagulation-time is from three to five minutes. In hæmophilia and purpura the coagulation-time is much delayed. In severe jaundice, particularly the obstructive form, the coagulation-time of the blood is often much delayed, and serious hemorrhage has occurred during operations for the relief of such conditions.

Parasites in the Blood.

The principal vegetable parasites are those associated with the infections and described in Chapter I., Part II. They are: (1) spirilla of relapsing fever; (2) tubercle bacilli, very rarely; (3) anthrax bacilli; (4) bacilli of glanders; (5) typhoid bacilli; (6) streptococci and staphylococci; (7) the bacilli of yellow fever; and probably

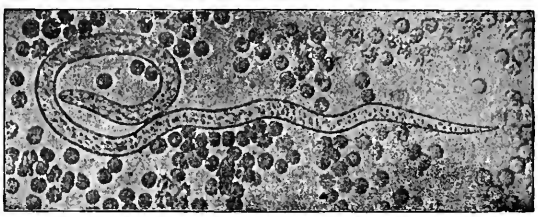
[1] Archiv f. Kinderheilk., Bd. xviii., S. 261.
[2] Monti, *ibid.*
[3] Fibrenthal and Bernhard, *ibid.*, Bd. xvii., H. 5 u. 6.

if carefully sought, almost every organism capable of causing a general infection—*e. g., B. aërogenes capsulatus,* pneumococcus, meningococcus, *B. coli communis, B. paracoli, B. pyocyaneus,* etc.

The animal parasites are : (1) *Filaria sanguinis hominis ;* (2) plasmodium of malaria.

Filaria Sanguinis Hominis. Filariæ are found in the blood and lymph of persons who live in the tropics, and in a few instances have been found in native Americans. (John Guitéras.) They have a blunt, rounded head with a tongue-like process and a long, pointed tail. They produce lymphatic swellings (particularly of the scrotum), chyluria, and hæmaturia.

FIG. 207.



Filaria alive in the blood. Instantaneous photomicrograph. Four hundred diameters magnification. Four millimetres Zeiss apochromatic. (F. P. HENRY.)

Patrick Manson [1] says the following are the commonest mistakes in the search for filariæ : (1) the use of too high a magnifying power ; (2) employing too strong illumination ; (3) searching unmethodically and in too small a quantity of blood ; (4) looking for filariæ in blood drawn from the body at a time when the particular species sought for is normally absent from the circulation. He describes three forms : *Filaria sanguinis hominis nocturna* (the ordinary form) ; *Filaria sanguinis diurna ;* and *Filaria sanguinis perstans.* The last appears to be the one associated with the disease known on the west coast of Africa as "*sleeping sickness.*" He prefers dry preparations of the blood, stained with a 0.5 per cent. eosin solution or a weak solution of fuchsin (one drop of the saturated alcoholic solution to an ounce of water). If a thin film of blood, before it has fully dried, be held over acetic acid so as to imbibe the fumes, and be then stained in a 0.5 per cent. solution of eosin, the blood is stained, but any filariæ remain pearly white.

The filariæ may have been discovered accidentally, or are sought for because of *hæmatochyluria,* or *lymph-scrotum, elephantiasis,* or varicose groin *glands* ("Demerara groin"). In the former the chyluria is intermittent. Microscopically, the urine contains molecular fat-globules or granules and a few red corpuscles.

[1] Trans. Seventh International Congress of Hygiene and Dermography, vol. i. p. 93.

CHAPTER XL.

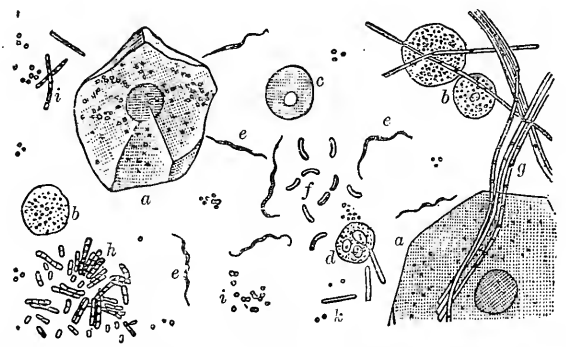
THE SALIVA AND NASAL SECRETIONS.

THE SALIVA.

THE saliva is derived from the parotid, submaxillary, and sublingual glands, and from the mucous glands within the mouth. The mouth should be washed with a warm alkaline solution and afterward with cold water, in order that the saliva obtained may be perfectly pure for examination. After the washing the glands may be stimulated by the application of dilute acid on a glass rod. The normal amount secreted in twenty-four hours varies from 1 to 2 pints. It is colorless or of a light-bluish color, and somewhat stringy. On standing in a conical glass two layers form, the upper clear, the lower cloudy. The reaction of saliva is alkaline.

Microscopical Examination. The following formed elements are observed: 1. Salivary corpuscles of the appearance of, but larger and more granular than, a white corpuscle. 2. Epithelium. The squamous

FIG. 208.



Buccal secretion. (Eye-piece III.; obj., Reichert $\frac{1}{15}$; homogeneous immersion; Abbé illumination, open condenser.) Friedländer's and Günther's method. (VON JAKSCH.)

a, epithelial cells; *b*, salivary corpuscles; *c*, fat-drops; *d*, leucocytes; *e*, *Spirochæte buccalis*; *f*, common bacilli of mouth; *g*, *Leptothrix buccalis*; *h*, *i*, *k*, different fungi.

variety derived from the mouth is seen. The cells are large in size and of polygonal shape. 3. Fungi. In health the mould and yeast fungi are seldom found. In disease they are present in large numbers; fission-fungi are met with in great numbers, both in health and in disease. In health small and large colonies of micrococci are found along with abundant bacilli. Miller has studied the micro-organisms of the mouth carefully and exhaustively[1] both by microscopical examination and culture-methods. The following are found to be pathogenic: (1) *Leptothrix*

[1] See Dental Cosmos.

buccalis; (2) *Vibrio buccalis;* (3) *Spirochæte dentium;* (4) *Micrococcus tetragenus;* (5) *Micrococcus de la rage;* (6) the micrococcus of sputum septicæmia; (7) the bacillus of decaying teeth, three varieties of the staphylococcus; (8) *Bacillus crassus sputigenus;* (9) *Bacillus salivarius septicus* and *Bacillus septicus sputigenus.*

The thrush-fungus, actinomyces, the tubercle bacillus, and the bacillus of diphtheria are also found in the saliva. It must not be forgotten that *Diplococcus pneumoniæ* or *Micrococcus lanceolatus,* which is the specific cause of pneumonia, is found in the saliva of some persons in health. It is also called *Bacillus sputi septicæmici.*

The characteristic parasite of thrush is ribbon-shaped, varying in length, and composed of long segments which often contain highly refractive nuclei at either end. The segments are homogeneous; they vary in length, those nearest the extremities being somewhat shorter. When mounted in glycerin, they are readily seen. Spores are also seen.

Chemical Examination. The chemical characters of the secretion depend upon the activity of the different glands. The saliva contains a trace of albumin, found by heating; a ferment which changes starch into sugar; mucin; and occasionally potassium sulphocyanide. In disease, as the quantity is diminished rather than increased, examinations have rarely been made. In *ptyalism* the saliva should be collected after rinsing the mouth frequently, especially after eating. The reaction is found to be alkaline, and the specific gravity low, 1002 to 1006.

Albumin is tested for by the usual methods. The sulphocyanides are detected by a solution of ferric chloride. When this is added to the fluid, a bright-red color appears which does not disappear with heat; a similar color, due to the precipitation of meconic acid, may be obtained by the same test from the saliva in opium-poisoning.

Sugar is tested for by the methods used in the examination of the blood. The diastatic ferment is detected by adding 5 c.c. of saliva to 50 c.c. of starch solution and placing the mixture in a warm chamber or on a water-bath heated to 40° C. After an hour's time the fluid will show the presence of grape-sugar.

Nitrites are detected by adding a little saliva to a mixture of starch paste, potassium iodide, and dilute sulphuric acid: if nitrites are present, a blue color results.

The Saliva in Disease.

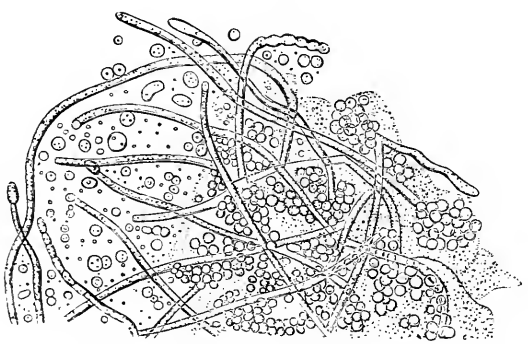
Amount. In catarrhal stomatitis the secretion is *increased.* It is acid and contains epithelium in excess. In ulcerative stomatitis it is also increased, is of a dark-brown color, fetid, and alkaline. It contains degenerated epithelium, leucocytes, blood-corpuscles, and many forms of fungi. It is increased and acid in thrush. It is increased in pregnancy, in rabies, and in glosso-labio-laryngeal palsy. I have seen it in excess in the convalescence of typhoid fever. It is increased by the internal use of jaborandi, sometimes to such a degree that large pathological exudates may be entirely removed by a free flow of saliva. Von Leube has recently recorded such occurrences, and also reports a remarkable case in

which spontaneous increase of the secretion of saliva repeatedly overcame an ascites due to hepatic cirrhosis, the daily amount of saliva at times being more than 3 quarts. Von Leube has successfully treated cases of dropsy by exciting an increase in the flow of saliva.

The *reaction* becomes acid in diabetes, gout, rheumatism, and mercurial poisoning. *Urea* may be found in cases of nephritis, particularly in uræmia, and the salivary glands undoubtedly have a decided excretory action in some cases of nephritis, acting vicariously in the place of the damaged kidney. There is no *sugar* in the saliva in diabetes.

Fenwick has investigated the changes in *potassium sulphocyanide* in disease. By a scale of colors he was enabled to compare the saliva in which potassium sulphocyanide had been detected in health with the saliva in various diseases. He believes that the amount of this ingredient is indicative of the degree of functional activity of the organs of nutrition. It is increased in acute inflammation and in the earlier stages of cancer and phthisis; in acute congestion of the liver from stimulants or food excess; and in rheumatism, gout, and in the convalescence of typhoid fever. When the power of the nutritive organs is diminished, the potassium sulphocyanide is decreased, as in late phthisis and cancer, the later stages of chronic diarrhœa and dysentery, in chronic catarrhal jaundice, in ascites, and in passive congestion of the abdominal viscera. Fenwick believes that tedious recovery and frequent relapses will occur if this element is found in excess in acute rheumatism.

FIG. 209.



Oïdium albicans, the vegetable parasite of muguet or thrush. (Reduced from CH. ROBIN.)

Leptothrix Buccalis. This genus of bacteria is seen in ribbon-like bundles composed of numerous segments; it is stained a bluish red by potassic iodide solution. It is most frequently seen in the tartar of the teeth.

THE NASAL SECRETIONS.

Physical Characters. The character of the secretions is of diagnostic significance. They may be liquid, semisolid, or solid. The liquid secretions may be serous, mucous, or purulent. *Serous* secretions occur in acute rhinitis, hay fever, and idiopathic rhinorrhœa, and follow bursting of cysts. The secretion of *mucus* occurs in the later stages of inflamma-

tion of the mucous membrane and in the chronic forms of inflammation. A *mucopurulent* secretion is seen in chronic rhinitis, and pure *pus* in abscesses of the septum or cavity. In hereditary syphilis it is at first mucopurulent, then purulent, and then sanious. A sanious acrid discharge, with false membrane discharged or evident on inspection, is due to *diphtheria*. A fetid, sanious, or ichorous discharge, with frequent attacks of epistaxis, attends malignant nasal growths. A discharge of blood is known as epistaxis. The *semisolid* secretion may be due to mucus alone, or to blood-clots mingled with serum or with pus. The latter occurs in atrophic and hypertrophic catarrhs. The *solid* secretions may be mucous crusts, as in acute and chronic catarrhs, blood-crusts after epistaxis and traumatism, membrane in diphtheritic rhinitis, slough from ulcers, and rhinoliths. The latter are gray or greenish brown in color, hard and rough, either fixed or movable.

Microscopical Character. The normal secretion from the nose contains squamous and ciliated epithelium, isolated leucocytes, and various fungi. The fluid is thick, alkaline in reaction, and has a slight odor. It contains mucin. In disease of the nasal cavities the fluid changes. In acute nasal catarrh it is more copious and thinner. It remains alkaline, and contains epithelium and fungi. When the stage of suppuration is reached, the secretion may consist entirely of pus. Cerebrospinal fluid may also be discharged through the nose in certain brain tumors. In such fluid, albumin is absent. Detection of this fluid is of diagnostic value, as it points to the central lesion.

Charcot-Leyden crystals are found in the nasal secretion in asthmatic patients, and sometimes in acute coryza.

Bacteriological Character. In diphtheria the characteristic microorganism is seen. Recognition of glanders may be based upon finding the bacillus in the nasal secretion. (See page 795.) Cultivations may be made. The nature of ulcers may be determined by microscopical examination. The tubercle bacillus can sometimes be detected. A pneumococcus or bodies that resemble it have been found in the secretion in ozæna. Thrush fungi have also been found, as well as mould-fungi.

Meningitis. Of very great importance in diagnosis must be mentioned the examination of the secretion removed from the nasopharynx and the upper portion of the nose by swabs. In cases of meningitis the infection atrium is often the nares. "Smears" of the "swabs" show the presence of tubercle bacilli, of *Diplococcus intracellularis* and other infective microorganisms.

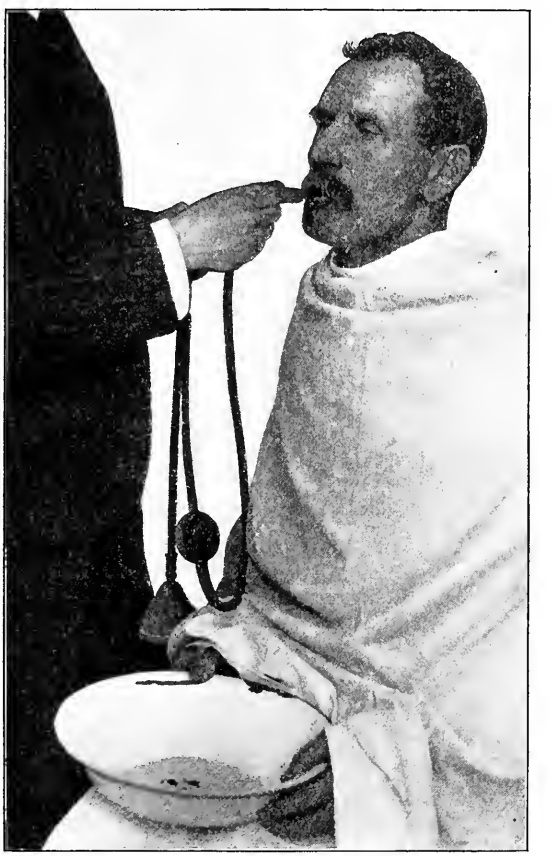
CHAPTER XLI.

THE STOMACH CONTENTS.

EITHER the contents are withdrawn with a stomach-tube or the vomitus is examined.

Mode of Procedure. 1. A test-breakfast (Ewald) or a test-dinner (Leube) is administered, or the *fasting* stomach contents are removed. *Ewald's test-breakfast* consists of 1 or 2 ounces (35 grammes) of

FIG. 210.



Illustrating expression and lavage.

bread without butter and a cup of weak tea ($\frac{1}{3}$ litre) without sugar, or the same amount of water. The *Leube-Riegel test-dinner* includes a large plate of soup (400 c.c.), a large portion of beefsteak or other meat, some

potatoes, and a roll. (See Boas' Meal. Lactic Acid.) 2. Remove the contents of the stomach in from forty minutes to one hour after the breakfast, and three or four hours after the dinner, by aspiration or by expression.

Aspiration consists in the withdrawal of the stomach contents by suction; either with the ordinary stomach-pump, by means of a bottle exhausted of air, as employed for paracentesis, and connected with the stomach-sound, or by connecting the sound with a hand-ball aspirator or Politzer bulb. *Expression* consists in compressing the abdominal muscles, imitating the act of straining in defecation. The patient takes a deep inspiration and then contracts the muscles as above. If the tube is sufficiently long, it can be bent, so as to assist expression with siphonage. Aspiration is less disagreeable to the patient, and is necessary when the stomach contents are not sufficiently fluid to flow easily. Expression is not to be employed when there are old ulcers, ulcerating carcinoma, phthisis with antecedent hæmopytsis, or a disposition to menorrhagia.

A soft-rubber tube with two good-sized openings near its distal extremity should be selected. Stockton suggests a tracing of rings around the tube 1 inch apart, beginning 20 inches from and ending 30 inches from the lower extremity, for the purpose of measuring the length of tube inserted. In healthy adults the distance from the incisor teeth to the lower border of the stomach is about 22 inches; in dilatation it may be from 24 to 30. The distance is partly determined by success in the siphonage. If the return-flow of fluid does not take place, it is well either to withdraw the tube or push it further on; for if too long it may curve above the level of the fluid, or if too short it may not reach the fluid.

It is sufficient simply to moisten the tube, since the saliva acts as a lubricant. It may, if desired, be oiled, or coated with the white of an egg. The patient should be seated, and the tube at once passed to the back of the pharynx, and pushed toward the œsophagus with or without the guidance of the finger. It is at once grasped by the œsophagus or lower pharynx, and if the patient is instructed to swallow and to breathe slowly, it is rapidly carried downward by deglutition. Mucus that accumulates in the mouth after the tube has been passed should be allowed to dribble outward and not be swallowed. It is often of advantage to reassure the patient by having him pronounce the letter "a" or some short syllable. It is not necessary to extend the head backward. After the tube has descended below the level of the fluid in the stomach the contents are removed by aspiration or compression into a convenient vessel; or the tube may be attached to the apparatus used for paracentesis, or to a tube entering a bottle in which a vacuum is created by an ordinary rubber bulb apparatus; or to the aspirator of Boas, which is a modification of the ball syringe. A valve is placed between the stomach-sound and the syringe. If a hard tube is used, it must be guided by the operator, who should stand back of the patient supporting the head, which should not be thrown too far backward. The tube can be passed by the operator seated in front of the patient. This kind of tube is used with the stomach-pump.

These methods furnish the most reliable information of the condition

of the stomach and its secretions: because both the quantity and character of the secretion can be accurately ascertained; and because being able to choose the time of examination, we can decide whether or not the findings are normal, and if not, in which particular they indicate disease. They make a diagnosis possible before sufficient data can be obtained by other methods.

One of the objective expressions of the *morbid process* or of altered function is seen in changes in the character of the contents of the stomach. The contents are obtained for examination when discharged from the stomach (vomit) or when removed artificially (lavage). Both fluids are studied by inspection, including microscopical examination, and by chemical and bacteriological analysis. The sense of smell enables one to differentiate many varieties of fluids. Alteration of function is also seen in alteration of digestion, and is estimated by chemical and physiological methods. The activity of the digestion must be determined by ascertaining the duration of digestion and its degree of completeness, which depend upon three factors: (1) the motor power; (2) the absorptive power; (3) the digestive power of the gastric secretions. To secure objective data, therefore, the following procedures are necessary:

Examination of the stomach contents.
Determination of the digestive power of the stomach.
Determination of the motor power of the stomach.
Determination of the absorptive power of the stomach.

Physical and Chemical Examination of the Stomach Contents.

A. Physical examination:
   1. The reaction.
   2. The odor.
   3. The character and quantity. Inspection.

B. Chemical examination.

A. Physical Examination. It is to be observed that familiarity with the products of digestion and the length of time required for the completion of the act is essential.

Quantity. If a person has taken no food or drink between the evening meal and the following morning, the quantity should not be more than 3½ fluidounces. The quantity of fluid, after digestion of a test-breakfast has continued for one hour, should be from 30 to 40 c.c. The filtrate is clear and yellow or yellowish brown in color. If digestion is normal, the fluid should contain free hydrochloric acid, pepsin, and rennin (the milk-curdling ferment), but no lactic acid. Albuminoids should be converted into proteoses and peptone, and starches into achroodextrin, dextrose, or maltose, though small amounts of erythrodextrin are usually present.

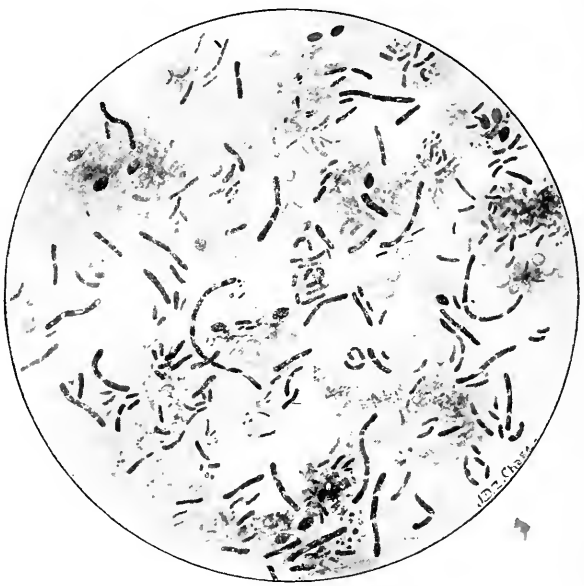
Reaction. The normal reaction of the contents of the stomach is acid from the presence of hydrochloric acid in the gastric juice; it may be alkaline in cases of hemorrhage or in the vomiting known as water-brash.

Odor. The odor is sour normally; it may be aromatic from the presence of the fatty acids, fecal in obstruction of the bowels with fecal

vomiting, and, finally, may indicate the nature of poisonous ingesta—ammonia, phosphorus, carbolic acid. The dark, frothy material from a dilated stomach is of a foul, yeasty, or putrid odor.

Inspection of the Stomach Contents. By ordinary inspection the *quantity* and the *character* of the vomitus or stomach contents are noted. The most important evidence of secretory change is achylia gastrica (absence of secretion), in which the bread is returned in the form in which it was taken, except that it appears water-soaked. The changes due to retention are mentioned below. The microscope may show unchanged starch-granules, undigested muscle-fibres in large amounts, sarcinæ, yeast-cells, or Oppler-Boas bacilli (see page 595.) In this

FIG. 211.



Oppler-Boas bacillus in vomitus.

manner valuable information as to the digestive, motor, and absorptive power is ascertained. Not only do we learn whether digestion has taken place or not, but also the variety of food that is undigested—whether proteids or hydrocarbons.

DIGESTIVE POWER. If undigested food is found after digestion should normally be completed, the digestive energy is deficient. Undigested food should not be found later than six or seven hours after an ordinary meal of mixed food.

MOTOR POWER. An abnormally great quantity of solid matter and a small amount of chyme indicate abnormal retention, which is usually due to motor weakness (atony, dilatation of the stomach), or dilatation in conjunction with insufficient motor power. Sometimes, when there is a large residue in the stomach, the contents separate into three layers, the uppermost consisting of mucus or undigested food; the second,

generally the thickest, of fluid; and the lowest, of chyme. Such a formation points to abnormally long retention as the result of stenosis and consecutive dilatation, or to motor weakness.

The stomach should be empty much sooner if only starches are taken, as in Ewald's test-breakfast. One hour after the administration of a test-breakfast of 35 grammes of white bread and 300 grammes of water there should remain about 40 c.c. Hence, if after such a breakfast there is found a much greater quantity, then motor absorptive insufficiency may be considered to exist. A filtrate of 100 to 300 c.c. is due either to hypersecretion or more probably to organic obstruction to the outflow, such as stenosis of the pylorus, adhesions or dislocation of the pylorus. To make sure that the stomach contains nothing at the time of giving the breakfast, it must first be emptied. The character of the food taken is observed, as undigested particles may be seen in the contents.

We can often discover by inspection whether food is brought up by *vomiting* or by *regurgitation*. When digestion is normal and the individual has eaten meat, regurgitation of food from the œsophagus can be differentiated from vomiting by the appearance of muscle-fibres; if the food is vomited, the fibres are in a state of disintegration: if not, they are intact.

Mucus. Mucus is found in small quantity normally, but is increased in catarrhal affections of the mouth, throat, or stomach. When its source is the mouth, *saliva* also is generally present. Mucus is recognized by its stringy, tenacious character. The chemical test is performed by pouring the mucus into cold water and, after gently agitating the vessel, pouring off the supernatant water; if a little liquor potassæ is then added, the mucus is dissolved by the alkali. The addition of acetic acid forms a precipitate which is insoluble in an excess of acetic acid, thus distinguishing mucus from the precipitate of syntonin, as the latter is soluble in an excess. Pigmented mucus in vomitus is usually derived from the bronchial tubes.

Bile and *intestinal juice* may be regurgitated into the stomach as the result of violent emesis, or when the pylorus is much relaxed, or in stenosis of the duodenum below the common duct; bile is then present in large quantities if the stomach is dilated.[1] Bile is recognized by the usual tests, the most satisfactory being iodine (see under Examination of Urine); intestinal juice is recognized by its peculiar properties and the presence of leucin and tyrosin. Persistent absence of bile in the vomitus is an indication of pyloric stenosis.

Blood. Blood is found in ulcer; cancer; acute, especially toxic, gastritis; injuries to the mucous membrane from the use of the tube for expression; and after violent retching. It is also common in cirrhosis of the liver, and may occur in purpura, peliosis rheumatica, the hemorrhagic diathesis, and in yellow fever. Blood mixed with gastric mucus may come from the lung, the act of coughing having excited vomiting.

If the blood is unaltered, it can be distinguished from all other substances by microscopical examination. Occasionally the blood has the appearance of coffee-grounds. The hemorrhage has taken place slowly

[1] Hochhaus, Berlin. klin. Wochensch., 1891, No. 17.

under these circumstances. In fact, the more rapid the bleeding the brighter the color of the blood. The *hæmin test* usually serves to distinguish it. The suspected material is filtered and a little of the filtrate evaporated in a watch-glass; when dry, a small portion is mixed with finely pulverized salt upon a glass slide; it is then covered with a cover-slip underneath which one or two drops of glacial acetic acid are allowed to flow. The acetic acid is evaporated by slowly heating the slip over a small flame, and when dry, a few drops of water are allowed to flow under the cover-slip to dissolve the salt. If the vomit contains blood, brown rhombic crystals of hæmin (hæmin hydrochlorate) will appear under the microscope. As they are very small, a magnification of about 300 diameters will be necessary to bring them readily into view. This test is not always reliable; it is sometimes negative with stomach contents even when considerable quantities of blood are present. The *guaiacum* test may be fallacious, as the same color-reaction takes place when the test-liquid contains bile or saliva or a starchy substance like potato. It is performed as follows: add 2 or 3 drops of the tincture of guaiacum to a small portion of the gastric contents in a test-tube and pour ozonic ether on the surface; when the liquids meet, a blue color develops. Bile may be distinguished from blood by tests for the former—color-reaction with iodine or nitric acid. If blood is present in the stomach contents, it may be detected by the test for iron. To the gastric contents, "coffee-grounds," in a porcelain capsule, add a small quantity of potassium chlorate and a few drops of a strong acid, HCl. Heat over a flame and add a few drops of a 5 per cent. solution of potassium ferrocyanide. If iron is present, Prussian blue will be formed.

Pus. Pus is rarely present in sufficient quantity to be detected by the naked eye, but it sometimes occurs in phlegmonous gastritis and when an abscess has ruptured into the stomach. Strauss states that pus-cells are never present in notable number except in cases of abscess communicating with the stomach and in ulcerating cancer. He considers their presence a valuable sign of cancer. Pus may be in the vomitus and yet come from the lungs. It is usually a muco-pus, and is recognized by the pigmented pellets or strings of mucopurulent material.

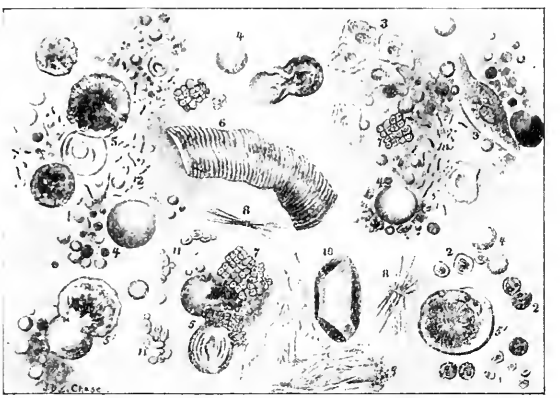
Fecal matter is vomited in complete obstruction of the bowels and, according to Vierordt, in severe diffuse peritonitis. It is recognized partly by its appearance and partly by its odor.

Worms are sometimes vomited; the round worms not so very infrequently; oxyurides and ankylostomata rarely.

Microscopical Examination. The illustration (Fig. 212) shows the various matters that may be found in vomited matter. Briefly, they are columnar and squamous epithelium; white blood-corpuscles acted on by gastric juice; and red blood-corpuscles. The corpuscles are usually isolated. The red are rarely perfect, and in the white little more than the nucleus remains. From the food we may also find muscle-fibres, fatty globules, and fat-needles, elastic and connective-tissue fibres, starch-granules, and vegetable cells. Muscle-fibres are recognized by their transverse striation. Fat-globules are soluble in ether, and are recognized by their refracting power. Starch-granules stain blue with iodo-potassic iodide solution.

In addition, *fungi* of many forms are found, as the mould-fungi, the yeasts (torulæ), and fission-fungi. The latter are recognized after staining by the iodo-potassic iodide solution, which colors them blue. The most important fission-fungi are *Sarcinæ ventriculi*. They are of a dark-gray tint, stain mahogany-brown to reddish brown with the above-mentioned solution, and resemble in shape corded bales of goods. (See Bacteriological Diagnosis.) *Torulæ* and *sarcinæ* are present when fermentation is in progress, and hence indicate delayed digestion from motor insufficiency or deficient digestive energy. Sarcinæ are also usually an indication of dilatation of the stomach from a *benign* cause, since they flourish only in stagnating contents which are decidedly acid from HCl, and these conditions are afforded by benign pyloric stenosis, but not by carcinoma.

FIG. 212.



Microscopical appearance of stomach contents: 1, red blood-corpuscles; 2, leucocytes; 3, squamous epithelium; 4, fat-globules; 5, starch-granules; 5′, starch changed by action of the gastric juice; 6, muscular fibre; 7, *Sarcinæ ventriculi;* 8, fat-crystals; 9, pieces of orange; 10, phosphatic crystal; 11, yeast fungi; 12, bacilli and micrococci.

The "*Oppler-Boas bacilli*" have a contrary significance. These are long, thick, club-like organisms, often pointed at one end, and usually showing a decided Brownian movement. They are usually found in cancer cases, and often in enormous numbers, and are of about the same value in diagnosis as the presence of large amounts of lactic acid.

In cases of carcinoma careful examination of the precipitate will often show cells in process of division, symmetrically or otherwise. While undoubtedly more frequently seen in this condition, it must be remembered that non-malignant structures, such as ulcer, may give the same cellular figures. Fragments of tumors, giving conclusive evidence of malignant disease, are at times found in vomitus and gastric contents.

B. Chemical Examination. A chemical examination is made to determine (1) the presence of free acids; (2) the degree of total acidity of the stomach contents; (3) the presence of free HCl; (4) the presence of lactic acid; (5) the presence of volatile acids; (6) the presence of products of digestion and the digestive power; (7) the presence of pepsin; (8) the presence of rennin; and (9) the character of the carbohydrates.

Hydrochloric acid is the normal acid of the gastric juice. Normally lactic acid is found during the first half-hour of digestion, when starches have been taken; but when only meats have been taken, lactic acid is not found so early. The secretion of hydrochloric acid is not delayed; but as the acid is at first combined, it can not be detected as free acid until half or three-quarters of an hour later.

1. **Free Acids.** The most sensitive test for free acids is *Congo-red.* Filter-paper soaked in a saturated solution of the dye and allowed to dry is turned a deep blue if free acid is present. Prepared with a weak solution, the filter-paper is turned to a light blue by hydrochloric acid, and violet by organic acids. The reaction may be obtained with solutions as weak as 1 : 150,000. When no reaction is obtained, entire absence of free acidity may be assumed.

The presence of free acids, as indicated by the Congo-red test, shows that:

a. HCl—inorganic acid—may be present alone.

b. Lactic, butyric, or acetic acid—organic acids—one or all, may be present without HCl.

c. HCl and one or more of the organic acids may be present together.

Free acidity may be due (1) to fixed acids—hydrochloric or lactic acid—*fixed acidity;* (2) to volatile acids—butyric or acetic acid—*volatile acidity.*

2. **The Total Acidity.** This is determined by titration. The stomach contents must be well filtered. Fill a Mohr burette with a decinormal solution of caustic soda.[1] To 10 c.c. of the filtered gastric fluid add 2 drops of a saturated alcoholic solution of *phenolphthalein.* Allow the caustic soda solution to drop slowly from the burette into the fluid until a faint rose-red color is produced that does not disappear on shaking. The color is produced by the action of the alkali on the phenolphthalein. 4 to 6 c.c. of the caustic soda solution are required to neutralize the acid in *normal digestion.* The degree of acidity is expressed in percentage. Thus if 4 c.c. neutralize 10 c.c., the total acidity will amount to 40 per cent.; or if 6 c.c. are required, to 60 per cent. If more or less than the amount just indicated of the alkaline solution is required to neutralize the acid, the total acidity is increased or diminished and hence is abnormal.

3. **Free Hydrochloric Acid.** Filtered gastric contents are necessary. *Tropæolin* 00 is declared by Boas to be an absolutely certain test for HCl. A saturated alcoholic solution is of an orange-yellow color. Three or four drops of it are placed in a white porcelain dish and spread upon the sides by rotating it. The same amount of the fluid to be tested is then allowed to trickle down the sides of the dish and intimately mixed with the tropæolin. (Or evaporate the dye to dryness and then add the suspected liquid.) Upon heating the dish over a small flame, splendid lilac-blue to blue streaks characteristic of HCl will appear if that acid is present. No organic acid gives a like color.

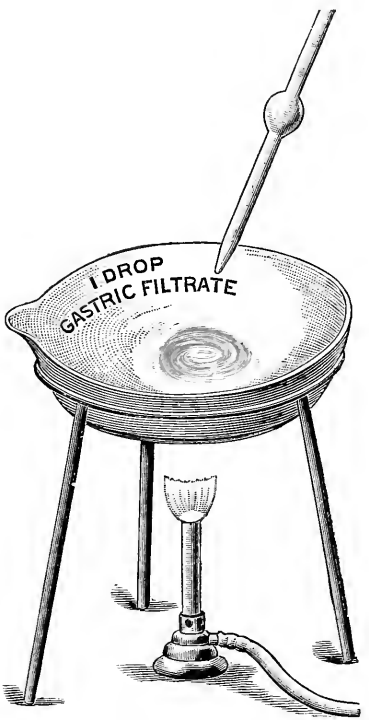
[1] Decinormal solution of sodium hydrate is of the strength of 4 grammes of pure sodium hydrate to the litre of distilled water. The sodium hydrate must be pure. This weight of sodium hydrate (4 grammes) will exactly neutralize 3.65 grammes of hydrochloric acid. Since sodium hydrate readily absorbs water the solution should always be made of approximate strength and then corrected by titrating with a decinormal oxalic acid solution.

Tropæolin-paper is turned brown by gastric juice containing HCl, the brown changing to blue upon the paper being heated. Organic acids give a brown color also, but it disappears upon heating.

Töpfer's test for the detection of free HCl is performed as follows: Dimethylamidoazobenzol is employed in a 0.5 per cent. solution of alcohol. To a few cubic centimetres of filtered stomach contents 1 to 4 drops of the reagent are added in a test-tube or beaker. If hydrochloric acid is free, a rose-red color is produced when the filtrate is added to the reagent. The drug reacts to HCl only when the latter is in a free state. Its reaction is not interfered with by salts, peptone, glucose, sodium chloride, or starch. If organic acids are present in a concentration of from 0.5 to 0.8 per cent., a distinct reaction may be brought about, and smaller amounts give a decided orange color.

Phloroglucin-vanillin, introduced by Günzburg, is a very sensitive test for HCl. It does not react to organic acids, and is now generally relied

Fig. 213.



Phloroglucin-vanillin test for free HCl.

upon. As the Günzburg reagent does not keep well, it is best put up as follows: phloroglucin, 2 gm.; alcohol, 50 c.c.; vanillin, 2 gm.; alcohol, 50 c.c. These are mixed in equal quantities as needed. One drop of the reagent is put into a porcelain dish with two or three drops of the stomach filtrate. Upon *cautious* heating over a *small* flame a beautiful carmine

colored surface is formed, especially at the edges. (Fig. 213.) Filter-paper soaked in this solution and moistened with a few drops of stomach filtrate containing HCl, changes to a beautiful carmine on heating, which is unaltered by the addition of ether. *Günzburg's* original test is employed with the same solution, except that 30 parts of alcohol are used. 1 drop of the solution and 1 drop of the fluid to be examined are evaporated to dryness on a water-bath; the appearance of a rose-red color indicates the presence of hydrochloric acid.

Congo-red Test. The solution must be kept in a dark bottle. If it turns reddish, it should be rejected, as it will not react. Some of the gastric fluid is shaken with 100 c.c. of ether until organic acids are removed. The Congo-red test is then employed.

Boas' Resorcin Test. Dissolve 5 grammes of resorcin and 3 grammes of cane-sugar in 100 c.c. of weak spirit. Apply the test in exactly the same way as Günzburg's. A similar rose-red coloration is produced in the presence of free hydrochloric acid. It is the cheapest solution that can be employed.

In testing for the presence of HCl it is better to give the patient a meal which is known to be digestible within a certain time by normal stomachs, otherwise HCl may appear to be absent because it is still combined with albuminoids. Ewald's test-breakfast is the simplest. In one hour the contents of the stomach may be aspirated and tested for HCl.

Quantitative Test for Free HCl. To a measured quantity of the gastric fluid add drop by drop from a burette a decinormal alkaline solution until the free acid is neutralized. This can be determined by checking the titration from time to time, and examining with Günzburg's reagent; 1 c.c. of the alkaline solution is equivalent to 0.00346 HCl. Multiply the number of cubic centimetres required to neutralize 10 c.c. of the gastric solution by 0.003646, and again by 10: the result will be the percentage of acidity.

The following method is easy of employment: To 2 or 3 drops of Töpfer's solution of dimethylamidoazobenzol are added 10 c.c. of gastric contents, and a decinormal soda solution allowed to flow in drop by drop until a pure yellow color takes the place of the red. The number of cubic centimetres of solution of soda that will neutralize the free HCl in 100 c.c. of stomach contents is multiplied by 0.00365. The result is the percentage of HCl. If 4 c.c. of soda solution are required to remove the red color, multiply 0.00365 by 40, the number equals 0.14 per cent. free hydrochloric acid. One may also use the Ewald method of expressing the result by number, as in the determination of the total acidity.

Quantitative Determination of Combined Hydrochloric Acid.—The method elaborated by Cohnheim and Krieger modified by Fisher[1] is accurate and convenient. The method is as follows: Determine the total acidity by titrating 5 c.c. of the gastric filtrate with 0.1 normal NaOH solution, using a 1 per cent. alcoholic solution of phenolphthalein as an indicator. Take another 5 c.c. of the filtrate, add sufficient calcium phosphotungstate solution to make 20 c.c., and call this Ta 1. Let it stand three or four minutes, add animal charcoal and filter.

[1] American Journal of the Medical Sciences, October, 1903, p. 666.

Determine the total acidity of this filtrate, using a 1 per cent. solution of rosolic acid as an indicator; call this Ta 2. Then Ta 1 minus Ta 2 equals the combined HCl in the 5 c.c. of the filtrate of the gastric contents used.

If the gastric contents show an absence of free HCl, proceed as follows:

1. Determine the hydrochloric acid deficiency by adding 0.1 normal hydrochloric acid, using a 1 per cent. alcoholic solution of dimethylamidoazobenzol as an indicator. Determine the total acidity of this mixture, using phenolphthalein as an indicator. This total acidity minus the quantity of hydrochloric acid added will give the original total acidity of the 5 c.c. of the filtrate of gastric contents employed.

2. Take 5 c.c. of the gastric filtrate, add 0.1 normal hydrochloric acid sufficient to make the new total acidity at least 60 per cent.; add sufficient calcium phosphotungstate solution to make 30 c.c. and titrate with rosolic acid as an indicator, calling the resulting acidity Ta 2. The difference between Ta 2 and Ta 3 will represent the quantity of hydrochloric acid combined with all the albumin in the 5 c.c. of gastric filtrate employed. Call this Total CHCl. Then this Total CHCl minus the hydrochloric acid deficiency equals the combined HCl in 5 c.c. of the gastric filtrate.

	Employed subacid gastric contents	5	c.c.	
	HCl deficiency (dimethylamidoazobenzol)	1	"	= 20
	Total acidity (phenolphthalein)	3	"	= 60
	Original total acidity	(60–20)	"	= 40
Ta 2	Added HCl in excess	3	"	= 60
	Added calcium phosphotungstate[1]	18	"	=
Ta 3	Total acidity (rosolic acid) of	30	"	= 30
Total CHCl	Total possible combined HCl	(60–30)	"	= 30
	Actual combined HCl in gastric contents .	(30–20)	"	= 10

Ta 2 — Ta 3 = Total CHCl

Total CHCl — HCl deficiency = combined HCl.

4. Lactic Acid. If the stomach contents are colorless, apply the following tests; if they are yellowish, make an ethereal extract, as described below, and then apply the tests. The presence of lactic acid may be determined by Uffelmann's reagent: mix 1 drop of pure carbolic acid with 5 drops of a dilute solution of neutral ferric chloride; add sufficient water to render the whole of an amethyst-blue color; to this add a few drops of the gastric fluid; a mere trace of lactic acid will change the blue to a light yellow or greenish yellow. The test for lactic acid is obscured by phosphates and simulated by excess of HCl or when glucose or alcohol is present in the gastric juice. The lactic acid should be removed by extracting with ether, as follows: 50 c.c. of gastric contents are reduced to 10 c.c. by heat in an evaporating-dish over a water-bath. After the concentrated solution cools add 50 c.c. of ether. The volatile acids are driven off by heat, the lactic acid is dissolved by ether, and hydrochloric acid remains in the residue. Apply the test for lactic acid to the ethereal extract if it is acid. The following is more delicate: add 1 drop of a solution of ferric chloride to 50 c.c. of water and

[1] The calcium phosphotungstate is made as follows: dissolve 4 grammes of phosphotungstic acid in 100 c.c. of distilled water by boiling, and neutralize the boiling solution with an excess of calcium carbonate. The boiling should be continued for a few minutes to drive off the CO_2. The solution should then be filtered.

add suspected solution; the presence of lactic acid causes a green-yellow coloration.

5. The Volatile Acids. These acids are best detected by their odor, their volatility, and their reaction.

Butyric acid is recognized by the pungent odor of rancid butter given off when the stomach contents are evaporated. It is recognized by the following reaction: to a small quantity of the liquid add a small quantity of alcohol and 2 drops of strong sulphuric acid; heat for a short time: a characteristic smell of butyric ether, like that of "pineapple rum," is given off.

Butyric acid is also detected by Uffelmann's reagent. A few cubic centimetres of the filtered gastric fluid are shaken with 3 or 4 times the amount of ether; the ether is poured off when it rises to the top, fresh ether is added, and the washing repeated; after the third washing the ether that cannot be poured off is evaporated by means of a water-bath; add a few drops of water to the residue and then an equal amount of the reagent: a tawny-yellow color with a reddish tinge results and the characteristic odor is produced. As much as 1 part of the reagent in 2000 is required.

In addition to Uffelmann's test the volatile acids may be detected by boiling a few cubic centimetres in a test-tube, over the mouth of which blue litmus-paper is placed. If acid is present, its vapor will change the blue to red. *Acetic acid* is recognized by its odor, particularly after heating the solution. It may be detected as follows: an ethereal extract of the gastric contents is evaporated on a water-bath, and the residue dissolved in water; after the watery solution has been neutralized with sodium carbonate, neutral ferric chloride solution is added, when a blood-red color results if acetic acid is present.

Alcohol is detected by its odor and by Lieben's iodoform test: distil the stomach contents, add to a portion a small quantity of liquor potassæ, and then a few drops of iodio-potassic iodide solution; a precipitate of iodoform takes place slowly if alcohol is present. If acetone is present, the precipitate forms rapidly.

6. The Products of Digestion. The ultimate products of digestion are the proteoses and peptones. If they are present in the stomach contents, it shows that hydrochloric acid and pepsin must have been secreted in the stomach. If vomiting occurs soon after food is taken, or if there is obstruction at the lower end of the œsophagus, these products are not present. *Syntonin* is a product of digestion which precedes the two above given. To ascertain if digestion has taken place, it is necessary to test for syntonin only and then employ the biuret test. Syntonin is detected by neutralizing the gastric contents with a solution of sodium hydrate. The precipitate is syntonin, which is soluble in an excess of alkali, and may be again precipitated by an alkali. After filtration and removal of the syntonin, proteoses and peptone are detected by the biuret test.

7. Pepsin. If HCl is present, add 5 c.c. of a gastric filtrate to a small piece of egg-albumin. Allow digestion to take place for several hours at 37° to 40° C. Non-digestion indicates absence of pepsin.

If HCl is absent, pepsinogen is found alone. Add 2 drops of a 25 per cent. HCl solution to 10 c.c. of the gastric contents. Add to this

solution a small portion of egg-albumin. If it is dissolved, pepsinogen was converted into pepsin by HCl.

8. **Rennin** (the milk-curdling ferment). This may be detected as follows: 5 to 10 c.c. of cows' milk of neutral reaction are boiled and added to neutralized and filtered gastric juice; the mixture is placed on a water-bath and heated to 30° or 40° C.: if the ferment is present, the casein of the milk is precipitated in flakes or in a curdy mass in from twenty to thirty minutes.

9. **The Carbohydrates.** Add a few drops of Lugol's solution to the gastric contents: if starch is present, it turns blue; if erythrodextrin, purple. If the digestion has proceeded so far as to change starch into achroödextrin, maltose, or dextrose, the iodine hue remains unchanged. The digestion of starches varies inversely with the amount of HCl present: in anacidity they are completely digested an hour after a test-breakfast; in hyperacidity there may be little digestion. In normal states there is usually some erythrodextrin, as indicated by a violet color after adding iodine.

When digestion is normal, the stomach contents removed from five to seven hours after a test-dinner are neutral and contain only a few flakes of mucus. At the end of five hours the stomach contents are acid and contain peptone, some undigested muscle-fibres, and starch-granules. If the stomach contains undigested food at the end of seven hours, and the contents are acid and contain peptones, a delay in digestion is indicated.

THE MOTOR POWER. Ewald and Sievers have suggested the use of salol; 15 grains are given, and normally salicylic acid should be detected in the urine in from forty to sixty minutes, or in seventy-five minutes at the latest. If its appearance is deferred still longer, motor insufficiency is indicated. The sign is of value only when the excretion is delayed. Urine containing salicylic acid gives a dark brownish-red color upon the addition of a drop of ferric chloride. A lack of motor power is also indicated by: (1) the presence of food in the fasting stomach before breakfast; (2) the presence of food in the stomach two hours after a test-breakfast has been taken; (3) the presence of food in the stomach five to seven hours after a test-meal.

Klemperer's *oil-test* is somewhat more accurate although disagreeable: 100 grammes of oil are introduced into the stomach with the stomach-tube; in two hours the stomach contents are removed by aspirating, the patient having previously taken a little water; the oil present is dissolved by ether, the solution evaporated, and the residuum of oil weighed: 75 to 80 per cent. of the oil should be discharged in two hours.

THE ABSORPTIVE POWER. Penzoldt and Faber recommend the administration of 3 grains of chemically pure potassium iodide—*i. e.*, free from iodic acid—a short time before dinner. Any fragments of free iodine adhering to the iodide are first carefully washed away. The saliva is tested for iodine with starch-paper and fuming nitric acid. If absorption is active, a violet color is obtained in from six and one-half to eleven minutes and a blue color in from seven and one-half to fifteen minutes. Zweifel directs that 3 grains (0.2 gramme) of potassium iodide be administered in a gelatin capsule, and $3\frac{1}{2}$ ounces of water (100 c.c.) taken: iodine is

detected in the saliva after about eight minutes. The character of the food taken is said to have considerable influence in retarding the appearance of the reaction, so that the blue reaction may not appear for forty-five minutes. Boas states that in dilatation of the stomach the reaction may be delayed two hours, and in cancer as long as eighty-two minutes. Both *motor* and *absorptive* power are recognized most satisfactorily by discovering evidences of retention of the contents upon the use of the tube.

Clinical Value of a Chemical Examination of the Stomach Contents. While it cannot be gainsaid that the chemical analysis of the stomach contents is of the utmost clinical value, it is just as certain that the information obtained in this way does not quite satisfy the expectations of the clinician and cannot be relied upon exclusively in the formation of a diagnosis. Still less can it be utilized as the sole basis for scientific therapeusis; such an attempt would inevitably lead to failure in the diagnosis, and hence any rational mode of treatment must rest not only on the chemical findings, but also on the physical examination of the stomach and its contents, and above all on a careful consideration of the history and progress of the case and of the subjective symptoms. In short, the practitioner must take a broader view of the case and exhaust every means of investigation if he would arrive at a correct estimate of the disease, even more so than in the case of almost any other organ in the body. Nor must he be discouraged if he does not possess the skill of the laboratory expert; the methods of examination here described are quite simple in their application and within the reach of any educated physician. For practical purposes all that is absolutely necessary is the determination of the total acidity and of the presence of free hydrochloric or of free organic acids. Even if the patient's condition is such as to contraindicate the removal of the stomach contents, an approximate diagnosis—perhaps not so precise as might be desirable—can usually be made after a thorough physical examination and a painstaking review of the subjective symptoms.

The chief points of value relate to the degree of acidity of the stomach contents and may be summed up briefly as follows:

Subacidity, which represents a diminution in the amount of total acidity, and **anacidity**, called achylia gastrica, are conditions, which consist, respectively, in diminution and in total lack of hydrochloric acid, and occur in a variety of organic and functional diseases of the stomach and in certain systemic disorders associated with general wasting and with atrophy of the gastric mucous membrane. The secretion of HCl is diminished in chlorosis and pernicious anæmia, in the acute infectious diseases, and in chronic wasting diseases, including tuberculosis, diabetes, and Addison's disease; the deficiency being due to functional disturbances of hæmic or nervous origin. Subacidity is the rule in the advanced stages of chronic catarrh of the stomach and chronic gastric dyspepsia from irregularities in the diet, and may also be present when the mucous membrane is congested, or affected with acute catarrh with atrophy. Hydrochloric acid is often absent in cancer; but unless the anacidity is found to be constant after repeated examination and two or more additional diagnostic factors of value are present, the diagnosis of cancer can not be made with certainty. In the gastric neuroses the

total acidity may be increased or diminished, or may vary at different periods in the same case, as will be more fully explained in the sections devoted to Secretory Neuroses. (See page 1022.)

Hyperacidity, or *excessive total acidity*, may be due to an excess of hydrochloric acid, when the term *hyperchlorhydria* is used, or to the presence of organic acids which normally exist only in negligible quantities. Hyperchlorhydria, when it is not a secretory neurosis, is characteristic of the early stages of gastric irritation; it is therefore found in cases of acute gastritis and in the early stages of gastric dyspepsia, and is practically always present in gastric ulcer. Excess of organic acids—lactic, butyric, and acetic—points to fermentation, as these acids are the result of bacterial activity which is favored by diminution or absence of the normal antiseptic, hydrochloric acid, and by loss of motor power. *Bacillus acidi lactici* and *Bacillus aërogenes* in the presence of carbohydrates produce lactic acid; while acetic acid is a product of alcoholic fermentation, which in turn is often due to the presence of sarcinæ. The organic acids are accordingly found in conditions associated with weakness of the muscular coat such as dilatation, organic obstruction at the pylorus, and cancer.

Lactic acid, as Boas has pointed out, is usually present in carcinoma; but its presence is not, as he first asserted, pathognomonic of the disease. Except, however, when it is derived directly from the food, as after a meal of meat in the form of sarcolactic acid, it is found in appreciable quantities only when there is loss of motor power with lack of free hydrochloric acid, and these conditions are found chiefly in cancer. The presence of lactic acid is the most common objective sign in that disease, and is therefore a valuable diagnostic point, the more so as it can be detected before a tumor is palpable; but its absence does not by any means exclude carcinoma. If, therefore, in a given case lactic acid is found and hydrochloric acid is absent, particularly if there is stagnation of the stomach contents, cancer can be safely diagnosticated. In order to eliminate sarcolactic acid, Boas recommends a test-meal consisting of 1 to 2 litres of oatmeal gruel with a little salt to make it palatable, which is to be removed by expression one hour after it has been taken. It is well to wash out the stomach six hours before the test-meal is given. Since, however, under normal conditions only a minute quantity of lactic acid is found in the stomach contents after an Ewald breakfast, the use of the Boas meal is unnecessary, as considerable quantities only are distinctive.

Diminution of free hydrochloric acid means deficiency of functional activity, and goes hand in hand with diminished motor and absorptive power. As hydrochloric acid directly and indirectly is responsible for the degree of total acidity, the variations in the quantity of free HCl in the stomach-contents practically coincide with variations in the total acidity.

The clinical value of the remaining chemical tests and investigations need not be explained. They indicate inability of the gastric function to accomplish digestion, but do not point to any special gastric affection. They are of value in distinguishing gastric neuroses from organic disease. In both there are pronounced gastric symptoms; if the examination shows normal digestive powers, a neurosis is indicated.

Gastric Hemorrhage. Hemorrhage from the stomach, *hæmatemesis*, or vomiting of blood, is due to an organic lesion or to the effects of acute irritant poisoning. Care must be taken to see that the blood is not from the upper air-passages or has been swallowed. If hemorrhage is profuse, the blood may cause irritation of the larynx and provoke paroxysms of coughing. It is often difficult, therefore, to distinguish between hemorrhage from the lungs and hemorrhage from the stomach.

Hæmatemesis.	Hæmoptysis.
1. Previous history points to gastric, hepatic, or splenic disease.	1. Cough or signs of some pulmonary or cardiac disease precede the hemorrhage in many cases.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.	2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.	3. The blood is frothy, bright red in color, alkaline in reaction. If clotted, it is rarely in such large coagula, and mucopus may be mixed with it.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.	4. The cough persists, physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days. (Osler.)

The hemorrhage may continue within the stomach without exciting vomiting. The general symptoms of hemorrhage may appear first as pallor, dimness of vision, giddiness, or faintness. The blood is usually acted upon by the gastric juice, and is dark, clotted, and partly digested or mixed with food. Its reaction is acid. In large hemorrhages the blood may be fluid and of a scarlet color; but if retained for any length of time, it is coagulated. When there is only a small amount of blood, the vomited matter has the appearance of coffee-grounds; when the quantity is large and the blood digested, the material appears like tar.

Vomiting is usually followed by a characteristic black or tarry stool, which may be distinguished from hemorrhage of the intestinal canal below the duodenum by the color of the contained blood. In intestinal hemorrhage the stools are dark red and not necessarily tarry. The dark stools must not be confounded with the same character of stools seen after the administration of iron or bismuth. In rare instances a hemorrhage into the stomach may take place from disease of the lower part of the œsophagus.

Causes. 1. General diseases from changes in the blood such as scurvy, purpura, hemorrhagic smallpox, yellow fever, acute yellow atrophy of the liver, severe anæmia, leukæmia, Hodgkin's disease, and pernicious anæmia. 2. Ulcer of the stomach. 3. Cancer of the stomach. 4. Ulcer of the duodenum. 5. Portal congestion, as in cirrhosis of the liver and other forms of chronic hepatic disease. 6. Disease of the spleen. 7. Congestion due to disease of the heart. 8. Chronic Bright's disease with atheroma. 9. Rupture of an aneurism. 10. Vicarious menstruation. 11. Cohen asserts that it occurs in vasomotor ataxia.

Profuse and sudden hemorrhage, in the absence of well-marked symptoms of disease, is in nearly all cases due either to latent ulcer or to congestion of the stomach from early cirrhosis of the liver.

CHAPTER XLII.

THE SPUTUM.

Sputum is the term applied to all the products of secretion of the respiratory mucous membrane and to other substances that may be brought up through the respiratory tract. It may consist of materials from the mouth, nose, throat, or lungs. Its character in disease may vary with the part affected, as well as with the disease itself.

General Characteristics of Sputa.

In describing sputum we note its quantity in twenty-four hours, its color and transparency, odor, its specific gravity, its reaction, its composition and consistency, its form or varieties, and whether it is homogeneous or heterogeneous.

Quantity. The amount expectorated in twenty-four hours varies from a few cubic centimetres, as in incipient phthisis and in slight bronchial catarrh, to 1000 c.c., as in a discharging empyema and in bronchiectasis.

Color and Transparency. The color varies with the composition of the sputum and with the nature of the disease. In œdema of the lung it is almost transparent and colorless; in acute bronchitis it is white; in pneumonia it is "rusty"; in abscess of the liver with amœbic characteristics it is brownish red or like "anchovy sauce." Red-streaked and prune-juice sputum results from the presence of blood. Red-currant-jelly sputum has occurred in malignant disease of the lung and has been found in hysteria. Black sputum is commonly seen in coal-miners. Purulent sputa, no matter what the cause, is yellow or greenish yellow.

Odor. Most sputa have no odor. It is characteristic only in a few cases. That of bronchiectasis, gangrene, and putrid bronchitis is particularly *heavy* and *fetid*—a characteristic which renders its origin almost unmistakable.

Reaction. This is always alkaline. Contamination of the sputum with the contents of the stomach, however, may cause it to give an acid reaction.

Specific Gravity. The specific gravity may vary from 1.0043 (mucous sputum) to 1.0375 (serous sputum). (Von Jaksch.)

Consistence. This often varies inversely with the amount of expectoration. The sputum may be liquid or it may be very tenacious.

Varieties or Forms of Sputum. Sputa may appear in homogeneous or heterogeneous forms.

Homogeneous Forms. Various forms of sputa present a homogeneous appearance.

Mucous Sputum. On account of the mucin it contains, this form is usually glairy, clear, and tough. It is seen in the early stage of acute

bronchitis and in œdema of the lung. A small amount of mucus is expectorated in health, and in cities and smoky towns it is apt to contain black pigment-particles, due to inhaled soot. Mucous sputum is seen in cancer of the lung.

Purulent Sputum. This is composed almost entirely of pus. It is seen most typically in a case of empyema discharging through a bronchus. It may also occur in bronchiectasis, in chronic bronchitis, in abscess of the lung, of the liver, or more rarely of the mediastinum when the contents are discharged through a bronchus. It may constitute the discharge of a tubercular vomica.

Watery or Serous Sputum. This results from œdema of the lung. It is often discharged after paracentesis of the chest, when it is also called albuminous expectoration. It may begin during the operation, or as late as two hours after it, and in a few hours from one to three pints may be discharged.

Sanguinous or Bloody Sputum—Hæmoptysis. Blood may be seen in greatly varying quantities; it may have many different sources, and it may be of slight or grave significance. It may come from the gums, the nose, the pharynx, or the larynx; in all cases such possible sources should be examined. There may be cases in which bleeding from the stomach (hæmatemesis) or œsophagus may simulate hemorrhage from the lungs. More often, however, people speak of vomiting blood that really has come from the lungs as having been vomited by them. Usually that from the lung is bright red and much more frothy, while that from the stomach is darker and acid, and may contain particles of food. Diagnosis is most difficult when some blood from the lungs is first swallowed and then vomited.

Hemorrhage from the Lungs. Hemorrhage from the lungs may occur in a number of conditions. It is not in itself a symptom of lung disease. The hemorrhage may be small in amount and may continue over a considerable period of time, or it may be sudden and profuse, at once terminating the life of the patient.

Causes of Pulmonary Hemorrhage. Hæmoptysis may be due to: (*a*) affections of the lungs; (*b*) affections outside of the lungs; and (*c*) menstrual disturbance.

Affections of the Lungs. Hemorrhage may be a symptom of several pulmonary conditions.

1. *Congestion of the Lungs.* This may lead to hemorrhage. The amount of blood is small; it may be limited to streaking of the expectoration, or it may be discharged in a few mouthfuls. This form of hemorrhage is seen in (*a*) *organic heart disease*; it is also a characteristic feature of the first stage of (*b*) *croupous pneumonia*, the rusty-colored sputum being due to rupture of the capillaries; in (*c*) *hemorrhagic infarcts* hemorrhage occurs, and is diagnostic if attended by the sudden formation of a consolidated area in the lung; in (*d*) *phthisis* it also occurs (see below).

2. *Tuberculosis.* Hemorrhage may occur in tuberculosis either (*a*) as the first symptom of the disease, on account of collateral congestion around infiltrated areas, or (*b*) later, on account of ulceration through

an artery after excavation of the lung has taken place. In the early stages the hemorrhage is usually profuse, but not fatal. It may occur repeatedly during a series of weeks, excited, no doubt, by the violent non-productive cough which attends the earlier stages of this disease. In the later stages, when the vessels are ulcerated, the patient may have repeated hemorrhages, varying from a few ounces to half a pint or a pint. These may occur daily, or may be repeated at intervals of a week or more over a long period of time. The patient usually experiences much relief after the hemorrhages that occur at long intervals Indeed, the dyspnœa, the cough, and the oppression of the chest subside in a remarkable degree, and by their gradual recurrence the occurrence of another hemorrhage often may be predicted. Death rarely ensues on a large hemorrhage from a phthisical ulceration ; yet it may possibly occur. The writer has seen four instances of hemorrhage into a large cavity, three with external hemorrhage, all of which caused death instantly. Hemorrhage with the expectoration of calcareous masses may recur frequently (*c*) in patients with healed or quiescent tubercle.

3. *Cancer.* In the absence of other causes, hemorrhage recurring frequently may be due to cancer of the lungs, especially if associated with currant-jelly sputum.

4. *Plastic Bronchitis.* Hemorrhage is of common occurrence in plastic bronchitis when large bronchial casts are expelled.

5. *Gangrene.* In pulmonary gangrene and in abscess of the lung hemorrhage occurs frequently, often causing death. The odor and the character of the sputum indicate the nature of the lesion.

Affections outside of the Respiratory Tract.

1. *Disease of the Bloodvessels.* An *aneurism* situated close to the trachea and bronchi may rupture into those tubes, causing hemorrhage. The hemorrhage is usually profuse and often induces sudden death. Sometimes for days the profuse hemorrhage is preceded by small hemorrhages. The physical signs of aneurism are sufficient to explain the cause. The bleeding can sometimes be seen in the trachea, when an aneurism of the innominate artery or of the aorta presses upon that tube.

Gouty endarteritis affecting the branches of the pulmonary artery is usually responsible for the hemorrhages that take place in the aged of both sexes independently of disease of the heart or of the parenchyma of the lungs.

2. *Cardiac Disease.* In diseases of the heart hemorrhage does not usually take place until the later stages of the disease, when secondary congestion of the lungs sets in. It may, however, be an early symptom in mitral stenosis. The hemorrhages may amount only to a staining of the sputum or to the expectoration several times during the day of an ounce or more of blood.

3. *Affections of the Blood.* In these conditions hæmoptysis is usually associated with hemorrhages in other portions of the body. Thus it may occur in hæmophilia, in purpura, in scurvy, and in anæmia. It also may occur in jaundice in connection with hemorrhages in other situations.

Hemorrhage from the lungs may be part of the blood dyscrasias of the

severe infections, such as typhoid fever, hemorrhagic smallpox, typhus fever, and the like.

4. *Without Known Cause. Disturbance of the Menstrual Function.* Pulmonary hemorrhages occur in which it is difficult to find any cause for the discharge. They are quite common in females sometimes at the menopause, in other cases during menstruation, in others at the menstrual period as a vicarious menstruation. A number of cases that have come under the writer's observation have had this tendency for years without the development of pulmonary disease, and apparently without much influence on the general health. Indeed, it may be said that in the absence of organic disease recurrent hemorrhage from the lungs in women is not of grave significance.

Symptoms. The only symptom of a *small hemorrhage* may be the presence of blood in the expectoration, or the spitting up of a small amount of blood accompanied by a slight cough. In either instance, unless his mental condition is rendered obtuse by disease, the patient is alarmed by the hemorrhage. Consequently he may experience palpitation of the heart, beside other nervous phenomena. Apart from the nervousness excited by the sight of blood, no other symptoms are caused by small hemorrhages, or even by hemorrhages of moderate amount.

The symptoms of a *large hemorrhage* depend upon the amount of blood that is lost. Only faintness and giddiness may be present with or without pallor. When the symptoms are more pronounced, extreme pallor develops; the pulse becomes rapid, small, and feeble; the extremities become cold, the face becomes bathed in perspiration; and syncope may occur. If the patient recovers from the syncope, he is extremely restless, breathing hurriedly and sighing. There may be some nausea. Moderate delirium and mild febrile symptoms often follow the restlessness. If the hemorrhages do not recur and if the patient's fears are calmed, the color will return gradually and the heart's action will become stronger and slower. These symptoms usually occur whether the hemorrhage is due to disease of the lungs or to an aneurism rupturing into a bronchus. If the hemorrhage is large, it differs somewhat in the two conditions. In the *rupture* of a *large aneurism* the blood rapidly wells up into the throat and pours out through the nostrils and mouth. With such hemorrhage the end may come in a few minutes. The blood from an aneurism is bright red, but is not frothy unless the discharge is very slow and becomes mingled with air in the vessels. In *pulmonary hemorrhages* the discharge is not so profuse, and is attended by coughing. With each cough, blood is raised, a full mouthful at a time. The blood discharged from the lungs is bright in color and is very frothy, being mixed with air. There are no clots in the discharged fluid. In rare cases of pulmonary hemorrhage an abundant stream pours out, which is dark in color, free from clots, and not mixed with air (large cavity).

Diagnosis. Hemorrhage from the lungs must be distinguished from hemorrhage from the upper air-passages, the mouth, the stomach, and the œsophagus. Thus a discharge of blood from the mouth may occur from cracks or from varicose veins in the *pharynx*. It is not abundant, however, in these conditions, and the blood is mingled with mucus. Hem-

PLATE XII-a.

FIG. 1.

Viscid, Rusty Sputum of Pneumonia.

FIG. 2.

Nummular Sputum.

PLATE XII -b.

Sputum of Bronchiectasis in Three Layers.

orrhage from the *gums* may be taken for pulmonary hemorrhage, unless there is stomatitis, or inflammation of the gums from scorbutus or ptyalism. In *stomatitis* the blood is thin, fluid, often offensive, and of a cherry-juice color. Hemorrhage from the *lungs* is distinguished from hemorrhage from the stomach by the difference in the way in which it is discharged and the difference in the character of the blood. If from the *stomach*, the blood is vomited and is mixed with particles of food or other gastric contents. It is dark in color, often of the appearance of coffee-grounds, and it is not mixed with air, and hence is not frothy. The rapid hemorrhage from the ulceration of an *aneurism* into the *œsophagus*, or rupture of varicose veins at the lower end of the œsophagus, can not be distinguished by its appearance from the hemorrhage following rupture of an aneurism into a bronchus. The recognition is dependent upon the physical signs of the case and the history of the patient's illness.

Heterogeneous Forms. These are mixtures of any two or three of the homogeneous forms.

Mucopurulent Sputum. The most common form of sputum consists of mucus and pus mixed in varying proportions, and is termed mucopurulent. Such sputa are found in the same conditions that give rise to purulent sputa. *Nummular sputum* is the name given when flat, coin-shaped masses are formed, that sink to the bottom of a vessel containing water, as in phthisis and in chronic bronchitis. When the masses are more spherical, the term *globular sputum* is employed. At times the sputa may be seen to separate into three distinct layers, the upper one frothy, mucopurulent, greenish yellow or dirty green, sometimes lumpy, sometimes composed of shreds; the middle layer thin and watery, and containing shreds from the upper layer; the bottom layer, apparently made up of pus and débris, opaque, and without air-bubbles. This condition usually points to gangrene of the lung, but it may occur also in bronchiectasis.

Cheesy particles, in size from that of a millet-seed to that of a pea, are observed in the second and third stages of phthisis and usually contain large numbers of tubercle bacilli.

Mucoserous Sputum. A mixture of serum and mucus, as the name implies.

Mucosanguinous, Serosanguinous, and Sanguino-mucopurulent Sputa. Mucopurulent sputum streaked with blood is frequently seen in phthisis. In pneumonia the rusty sputum is the result of an admixture of mucus and blood, and usually contains small air-bubbles. With change in the blood-coloring matter a yellowish or greenish tinge may be imparted. In certain cases of chronic pneumonia, in which the blood remains longer in the lung tissue, the expectoration has a darker or prune-juice color. The same color may be observed when there is a slight leakage from an aneurism. When pneumonia is accompanied by expectoration of large amounts of blood, it is often of tuberculous origin. Blood may be mixed with the greenish expectoration of gangrene. According to Finlayson, this is especially true in children. In chronic valvular disease of the heart, and in oozing from aneurism, frothy mucus containing more or less blood is commonly seen. "Currant-jelly" sputa

are more or less characteristic of malignant growths of the lungs, while the expectoration from a liver abscess with amœbæ is reddish brown in color, from the mixture of blood, pus, and bile-elements, and is not unlike "anchovy sauce."

Casts in the Sputum. The unaided eye may distinguish other foreign substances, such as fibrinous and spiral casts of the bronchi or trachea.

Laryngeal Sputum. The sputum from the larynx is generally scanty; it is not frothy, and is colorless and transparent; it is often discharged in small globules; it may be streaked with blood. Sometimes pseudo-membranes are coughed up. It is doubtful if purulent sputum ever comes from the larynx, excepting in cases of perichondritis in which the abscess bursts into the larynx. Laryngeal sputum is found in catarrh and malignant tumors. It is blood-streaked when the catarrh is very intense, or after injuries.

Sputum from the Œsophagus. The *expectoration* in diseases of the œsophagus is characteristic. It is usually a glairy mucus, often frothy or viscid. It is not coughed up, but after welling into the pharynx is hawked up. It is abundant in acute and chronic inflammation and in cancer.

Method of Collection. Sputum that is to be examined should be collected in perfectly clean vessels which contain no fluid, preferably in glass or white earthenware spittoons. Care should be exercised to prevent the entrance of extraneous substances such as tobacco or particles of food whether from the mouth, from outside sources, or from the stomach through vomiting. Tobacco, prunes, and bread crusts are at times mistaken for blood. It is also necessary to see that the matter sent for examination is derived from the lungs, and is not simply the oral and faucial accumulation. If practicable, the mouth and pharynx should first be rinsed with a warm alkaline solution. The true sputum is always coughed up.

Sputa which upon examination has been found to contain tubercle bacilli should not be allowed to dry in the air, but should be thoroughly mixed with a 1 : 20 carbolic acid solution, or a 6 per cent. formalin solution should be added to the sputa after the examination is completed.

Microscopical Examination of the Sputum.

The Preparation of the Sputum. We usually require in the examination one or two glass dishes or plates, a large and a small piece of window-glass, mounted needles, forceps, slides, cover-glasses, watch-crystals, a good microscope and accessories, and certain staining fluids.

The sputum may be examined (1) when fresh and unstained or (2) after having been hardened or dried and then stained.

Preparation of the Fresh and Unstained Specimen. The fresh and unstained specimen may be prepared for examination (*a*) with a hand-lens or (*b*) with a microscope.

Examination with a Hand-lens. A portion of the sputum is placed upon a piece of window-glass, about 15 cm. square, which has been

painted black on its under surface or is laid upon a black ground. A smaller piece of window-glass, about 10 cm. square, is placed over the large glass so as to press out the sputum in a thin layer. Or the sputum may be spread out on a piece of black paper by means of an ordinary glass slide. The thin layer is then examined with a hand-lens or with the unaided eye.

Examination with the Microscope. A particle or a drop of the sputum is placed upon a glass slide and covered with a cover-glass, by means of which it is pressed until flattened into a thin layer. The specimen is then examined first with the low power of the microscope and afterward with the high power.

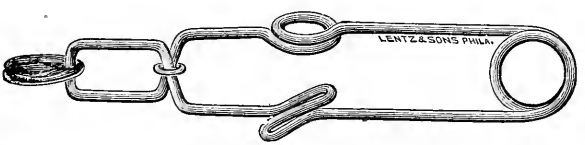
The Preparation of Sections for Microscopical Examination. Schmidt[1] *fixes* sputum in 0.5 per cent. salt solution saturated with mercuric chloride, *hardens* in alcohol, and *sections* in the usual manner. For *hardening* sputum Zenker's fluid has been found most satisfactory. After hardening, the sputum is *embedded* in paraffin and cut. In many cases it is advisable to roll up the sputum in a little ball before fixation. For the study of spirals thick pieces should be embedded in celloidin; for the study of the cellular elements, thin sections are embedded in paraffin.

Sections of sputum with mucin swell when treated with watery solutions of the dyes; hence the *celloidin should be first removed* to prevent folding of the sections. All specimens of sputum, except the very thin ones, can be prepared in the manner described.

Preparation of the Dried Specimen. A small amount of the purulent portion of the sputum is spread in a thin and uniform layer on a perfectly clean cover-glass by means of forceps, needles, a platinum loop, or other instrument, which previously must be sterilized by being held a moment in the flame of a Bunsen burner or spirit lamp. Or a small amount of sputum may be pressed between two cover-glasses, which are then slid apart. The sputum is then dried in the air, or more quickly by holding the cover-glass some distance above the flame of a burner or lamp. Finally, it is passed three or four times through the flame and so "fixed."

A second rapid method is as follows: select with the sterilized platinum loop a suspicious yellowish particle from the sputum; smear it thinly over one end of a slide which has previously been passed several

Fig. 214.



Ordinary cover-slip forceps.

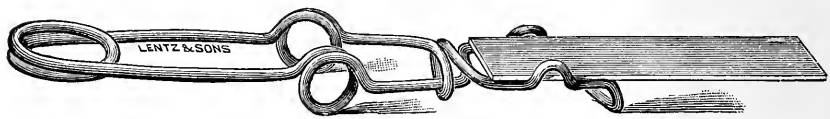
times through the flame of an alcohol lamp or Bunsen burner. Dry by holding over flame and fix by passing several times through the flame.

Staining of the Sputum. The methods employed in staining the dried specimens and the sections are the same. The specimen should be examined as soon after it has been raised as possible.

[1] Zeitschr. f. klin. Med., 1892, p. 476.

Any of the freshly prepared aniline stains may be used. Methylene-blue is the stain most generally employed. The method of staining varies with the object in view. When the leucocytes are to be studied, a stain of eosin and hæmatoxylin is best. For the detection of tubercle bacilli the specimen must first be stained with carbol-fuchsin and then destained with sulphuric acid. The methods for staining the various elements will be described under the appropriate head.

Fig. 215.



Boston's slide-holder.

The solutions needed are: (1) aniline-oil gentian-violet solution; (2) iodo-potassic solution (iodine, 1 part; potassium iodide, 2 parts; water, 300 parts); (3) saturated aqueous solution of eosin; (4) Löffler's alkaline methylene-blue; (5) a mixture of 95 per cent. alcohol 4 parts, and ether 6 parts; (6) 95 per cent. alcohol; (7) absolute alcohol; (8) xylol.

For staining sputum, more particularly in searching for influenza bacilli, Smith's directions are as follows: fix in flame, cover with aniline-oil gentian-violet, and steam gently; wash off the excess of aniline-oil gentian-violet with iodo-potassic iodide solution; cover with iodo-potassic iodide solution, and steam as before; decolorize as much as possible with 95 per cent. alcohol. Wash a few seconds in alcohol-ether solution; wash in water; stain a few seconds in saturated aqueous solution of eosin; wash off excess of eosin with Löffler's blue, color with Löffler's blue, steaming as before; decolorize slightly with 95 per cent. alcohol; wash in absolute alcohol; follow with xylol; mount in Canada balsam, and examine with an oil-immersion lens.

Appearance of Stained Specimen. The appearance of a specimen stained by this method is as follows: the protoplasm of the leucocytes, lymphocytes, and other cells takes the eosin stain, as do the red blood-corpuscles; the nuclei of the cells stain with the Löffler blue. Eosinophiles appear as in stained blood preparations. Gram-staining organisms appear black or deep violet, while Gram-decolorizing bacteria take the Löffler blue stain. Capsules about organisms, when present, are stained by the eosin, as are cilia.

Microscopical Constituents of the Sputum. (See Fig. 216.) **White Blood-corpuscles.** These are present in all sputa, but in varying number and size. They are usually of the polymorphonuclear variety, and are most abundant in purulent sputa. Often they contain fat-drops and pigment-particles. In stained preparations of sputa in cases of acute croupous pneumonia, influenza, pneumonia, or phthisis, frequently many of the leucocytes contain large numbers of organisms—*i. e.*, pneumococci, influenza bacilli, or tubercle bacilli.

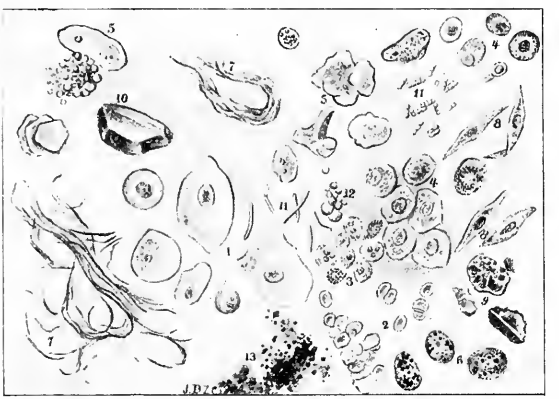
Eosinophile cells are frequently found in large numbers in the sputum in cases of asthma. They are also present in the sputum in acute and

chronic bronchitis and in phthisis. Their presence in the sputum in cases of phthisis is considered by Teichmüller to be of favorable import.

Red Blood-corpuscles. These are to be found in most sputa, but they may be so few as not to give a red color. The source is often high up in the respiratory tract. When they are present in large numbers, the sputum is more or less tinged, and in hæmoptysis it is almost wholly made up of red cells. Usually each cell is well preserved, but they may appear as pale bodies or as rings, the pigment remaining in the sputum as pigment-particles or as crystals of hæmatoidin, as in pneumonia.

Epithelium. Two general varieties of epithelium are found in the sputum—squamous and cylindrical. The former comes from the mucous membrane of the mouth, the tongue, the tonsils, the true vocal cords, and perhaps from the salivary and the small bronchial glands. Squamous epithelium is of no clinical importance. (See Fig. 216.)

FIG. 216.



Various objects from sputum: 1, squamous epithelium; 2, red blood-corpuscles; 3, polynuclear leucocytes; 4, alveolar cells; 5, myelin-cells; 6, pigment-cells; 7, elastic tissue fibres; 8, squamous cells; 9, hæmatoidin crystals; 10, phosphate crystals; 11, fungi; 12, fat-globules; 13, free pigment. (Original observation.)

CYLINDRICAL CELLS in sputum are rarely perfect. It is uncommon to find the cilia intact, and still more so to see them in motion, while the body of the cells is likely to be changed. They are found in inflammations of the trachea and bronchi, or of the posterior nasal fossa, a locality where, it must be remembered, ciliated epithelium exists.

"ALVEOLAR" EPITHELIUM, so called, when found in the sputum, is more important than the above, as different observers consider its presence to have more or less clinical significance. The cells are elliptical or round, somewhat larger than white corpuscles, with a single nucleus, which is indistinct without the addition of acetic acid. The protoplasm is granular and contains particles of iron-dust, carbon, or blood-coloring matter, and often fat-drops. The cells may also have undergone complete fatty degeneration, and they have been considered the source of myelin-drops in the sputum.

Bizzozero has shown that alveolar epithelium not only occurs in almost all pulmonary affections, but also at times in normal sputum.

Detection. A small bit of sputum is placed on a microscope-slide and a cover-slip applied. Examine with varying powers, and again, after acetic acid is added, and after the cells are stained with an aqueous solution of methylene-blue.

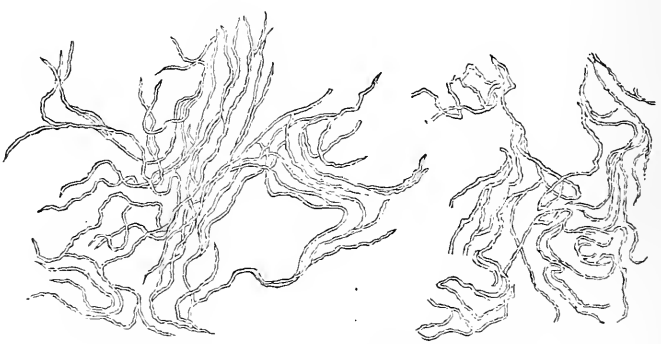
Frequently in cases of heart disease with failing compensation, especially where the mitral valve is affected, the alveolar cells may contain large amounts of blood-pigment.

Giant cells have been found in the sputum of phthisis cases.

Elastic Fibres. As the presence of elastic fibres in sputa is of much import, denoting destruction of the lung tissue of the bronchi, or the larynx or bloodvessels, the presence of elastic fibres from food remaining in the mouth must be especially guarded against. Elastic fibres may be mistaken for fat-crystals. They are found as single threads in bundles, or they may show an alveolar arrangement. They are to be recognized by the double contour and the curling ends, and at times by their alveolar arrangement. They may be due to tuberculosis, abscess of the lung, bronchiectasis, gangrene of the lung, pneumonia (von Jaksch), and rarely to destructive diseases of the larynx. In a very great majority of cases they are due to tuberculosis. It is uncommon to find them in gangrene, probably because, as Traube first suggested, they are destroyed by a ferment. (See Fig. 216.)

Elastic tissue from the alveoli often shows the diagnostic alveolar arrangement: the fibres that form a bronchus are branched; those from an eroded artery appear in the form of a network; or the fibres may be bound together. (See Fig. 217.)

Fig. 217.



Elastic fibres of lung tissue obtained from sputa after digestion in caustic soda. (Drawn by Dr. John Wilson.)

Detection of Elastic Fibres. The method employed by Osler, modified from Sir Andrew Clark's, is the best. A small amount of thick, purulent portions of sputum is pressed out in a thin layer between two pieces of plain window-glass, 15×15 cm. and 10×10 cm. The particles of elastic tissue appear on a black background as grayish-yellow spots, and can be examined *in situ* under a low power. Or the upper piece of glass may

be slid off till the piece of tissue is uncovered, when it is picked out and examined on a microscope-slide, first with a low power, such as the 1- or ½-inch objective, and then with a higher power. At first there will be some difficulty in distinguishing with the naked eye between elastic fibres and particles of bread or milk-globules, or collections of epithelium and débris, but with practice such mistakes can be avoided; the microscope always reveals the difference. This method is much easier of accomplishment and is quite as satisfactory in results as the one generally employed—boiling an equal quantity of sputum and solution of caustic potash (8 to 10 per cent.) for a short time, and then allowing the mixture to stand for twenty-four hours in a conical glass. The elastic tissue remains intact and is found in the sediment.

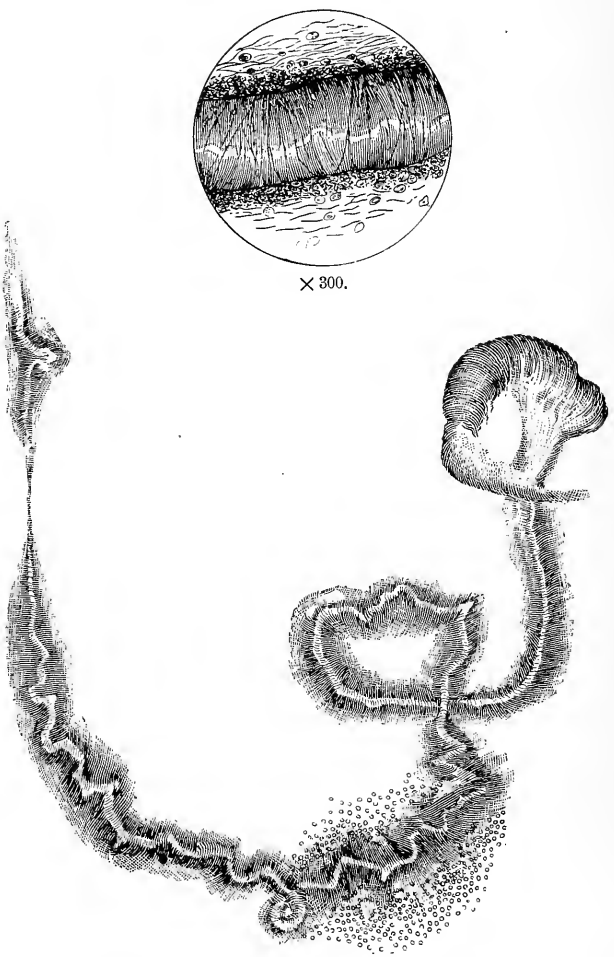
Connective Tissue and Cartilage. In fragmentary bits these are rare constituents of sputum. The former may occur with abscess or gangrene of the lung, and the latter when there is ulceration of the larynx.

Fibrinous Coagula. These striking, tree-like bodies are found in the sputa of plastic bronchitis, and at times in those of pneumonia, phthisis, and of diphtheria and croup when these have extended into the bronchi. They are usually mixed with mucus and are rolled into a mass. Their peculiar form is best seen when they are unravelled in water. They are then seen to be a complete mould of a small bronchus with its ramifications. The size varies greatly. They may be many centimetres long. In fibrinous bronchitis the size and shape of the moulds in different attacks may be exactly similar, as if they came from the same bronchus. They are grayish-white in color, hollow, and on transverse section are seen to be made up of cast upon cast. With the microscope leucocytes, blood-cells, and alveolar epithelium, and at times Charcot-Leyden crystals and Curschmann's spirals are found in the meshes. They are almost pathognomonic of fibrinous bronchitis. When they occur in any number in pneumonia, they make the prognosis unfavorable. Blood-casts of the smaller bronchi have been found in cases of hæmoptysis. They are rare, and have no apparent connection with the fibrinous coagula.

Spirals. Under this name are included spiral bodies that are found in the sputa of bronchial asthma, and occasionally in those of pneumonia and of capillary bronchitis (von Jaksch), and of chronic pulmonary tuberculosis. (Vierordt.) At the beginning of an asthmatic attack tough rounded balls are expectorated—"perles" of Laennec—which, if freed from the mucus surrounding them and spread on a glass with a dark background, may be seen by the naked eye to have a twisted, spiral form. With the aid of the microscope they are found to be made up of spirally arranged mucin in a more or less tight twist, and containing many cells from the alveoli and bronchi. In some of these spirals a shining central thread runs through the entire length like a core, remarkable for its clearness and its high refractive index. The fine fibres composing the spiral may or may not be closely arranged. Epithelium and Charcot-Leyden crystals may be found lying among the coils. The main constituent of the spirals is mucin, and Osler has suggested that the central thread is made up of transformed mucin. On the other hand, von Jaksch believes it is chemically distinct from the mucin spiral and more like

fibrin. Vierordt considers it either made of tightly twisted central fibres or to be an optical image of a core-cavity. This spiral is probably the result of an acute bronchiolitis. Why it should assume this remarkable form is an open question. It has been suggested (Osler) that the ciliated epithelium of the bronchi may have a rotary action, and their action, combined with the spasm of the bronchioles, causes the spiral formation.

FIG. 218.



Spirals from bronchial tubes. × 80. (After LEYDEN.)

The spirals are best *stained* with Weigert's fibrin-method, which stains them blue; yet these central threads are not fibrin: because (1) they are perfectly homogeneous; (2) they assume a violet color after prolonged staining (fibrin is always blue); (3) they occur as unformed blue masses which could only be compact mucin masses; (4) they give a specific

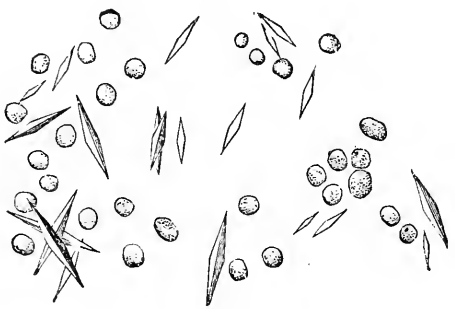
mucin reaction with thionin; (5) they assume a greenish color when Ehrlich's triacid stain, as modified by Babes, is used. (See Fig. 218.)

That there is a connection between the spirals and the Charcot-Leyden crystals seems very probable. The latter are absent from the sputum at the beginning of an attack of bronchial asthma; but if a portion of such sputum is allowed to stand for twenty-four to forty-eight hours, care being taken that evaporation does not take place, crystals will be found. As has been said, the crystals are often found among the spirals, even when they are seen nowhere else. Later on, the spirals disappear, but crystals derived from them (?) continue to be expectorated. (See Fig. 218.)

The method of examining for spirals is as given above under Preparation of Sections.

Crystals. Charcot-Leyden, cholesterin, hæmatoidin, fatty, tyrosin, oxalate of lime, and triple phosphate crystals are to be found in sputa under various conditions.

FIG. 219.



Charcot crystals. (SCHEUBE.)

CHARCOT-LEYDEN CRYSTALS. These are octahedral, sharply pointed, colorless or slightly bluish crystals, soluble in warm water, alkalies, acetic acid, and mineral acids. The practised, unaided eye may recognize these as small yellowish bodies, not unlike grains of sand; under the microscope they are unmistakable. Their size varies greatly. They occur most abundantly during and after an attack of bronchial asthma; but may also be seen in sputa of acute and chronic bronchitis and tuberculosis. They are identical with crystals found in semen, feces, leukæmic blood, and bone-marrow. Their connection with spirals has been mentioned above. Schreiner considers them to be the phosphate of an unknown base, which Ladenburg and Abel think may be identical with æthyleninim or di-æthyleninim. This identity, however, is disputed by Th. Kohn.

Detection. Examine the sputum of an asthmatic patient a day or two after the beginning of an attack for round, hard, yellowish bodies, and place these under the microscope with different powers. The Charcot-Leyden crystals are readily recognized. (See Fig. 219.)

CHOLESTERIN CRYSTALS. These crystals are similar to those of cholesterin found elsewhere, being thin rhombic plates, often with irregular corners and with high refractive index. They are soluble in ether, and

when treated with dilute sulphuric acid and tincture of iodine become violet, blue, or green, and then red. They may be present in the sputum of tuberculosis, simple and hydatid abscess of the lung, and in pus from an abscess of another organ, such as the liver. They have little clinical significance.

Hæmatoidin Crystals. Hæmatoidin crystals are at times recognizable by the naked eye as distinct spots of yellowish or brownish-red color. Under the microscope they have a brownish-yellow or ruby-red color, and are either in the form of small rhomboid prisms or of fine needles, arranged singly or in bunches of various shapes, or they may occur as free pigment-particles without crystalline form; smaller particles may be contained within a leucocyte. Their presence indicates that blood has remained in the respiratory tract for some time before being expectorated, or that an abscess has discharged into a bronchus. They occur in phthisis, following hemorrhage; in thoracic aneurism when blood is oozing into the lung; in gangrene; and in abscesses discharging through a bronchus. Von Jaksch states that when the crystals are contained in cells there has been a preceding hemorrhage, but that when there is considerable free hæmatoidin one infers that an abscess of a neighboring organ has discharged into the lung.

Fatty Crystals. Crystals of margaric acid appear as long, thin needles, greatly curved or bent at one end like a fish-book, and occur either singly or in bundles. They are found in unhealthy pus, as in gangrene, putrid bronchitis, bronchiectasis, and tuberculosis; in the plugs formed in inflamed tonsils; and in purulent sputum which is allowed to stand in a warm place. They dissolve in ether and in boiling alcohol; this characteristic, together with the regularity of their curve, should distinguish them from elastic fibres, with which they are sometimes confused by beginners.

Tyrosin Crystals. These have been found in the sputum of putrid bronchitis and of an empyema discharging into the lung, and usually occur in conjunction with leucin. They are most abundant in sputum that has been allowed to stand for some time. Under the microscope they appear as fine needles, and can be mistaken for fatty crystals. They are without diagnostic import.

Oxalate of Lime and Triple Phosphates. These have been noted occasionally in sputa; the former in a case of diabetes, and also in an asthmatic; the latter occur only in alkaline sputa, as they are soluble in acids.

Uric acid crystals have been observed by Moore in the sputum of a gouty patient.

Concretions of lime are rarely present in the sputum. They arise usually from the bronchial glands or lungs, from foci of tuberculosis which have become healed with the deposition of lime salts. They may be single or multiple. Hievoilés reports finding tubercle bacilli in the centre of one of these concretions.

Corpora Amylacea. Starch-like bodies, known as corpora amylacea, have been found in the sputum after pulmonary hemorrhage and in that of pulmonary gangrene. They have the shape of starch-corpuscles, and

sometimes give the amyloid reaction with iodine or potassium iodide. Their presence is without clinical significance.

Animal Parasites. Echinococcus cysts, generally broken into fragments, and only very rarely in a perfect whole, are to be found in the sputum when there is rupture of a cyst of the liver or of the lung into a bronchus. Scolices and free hooklets may be recognized, and pieces of the cyst-wall will be known by their remarkable formation. Their presence is of great clinical value.

Infusoria have been found in the expectoration from gangrene of the lungs. They belong to the monad and cercomonad varieties.

Distoma hæmatobium eggs may occur in sputa when the lung tissue is broken down by its presence, the eggs being thrown off in the sputum.

Distoma Westermanii or pulmonale is found in the sputum in Japan in certain cases resembling phthisis. Both the worm and the ova may be present in the sputum.

Amœba Dysenteriæ (*Amœba Coli*). Of far more interest and importance is the presence of this parasite in the expectoration. A full description of the amœba will be given in the chapter on Dysentery. The amœbæ are the same in every respect when found in the sputum, except that they are often slightly larger. The sputum containing the amœbæ is partly diffluent, tenacious, frothy, and bright red in color at first, due to the presence of blood, but later is brick or brownish-red in color and sometimes is bile-stained. Small yellowish-white cheese-like particles are seen. Upon exposure to the air the sputum becomes thin, syrupy, and oily, looking much like anchovy sauce. The sputa are alkaline and of a faintly sweetish odor, never putrid. Later on, they become more purulent, somewhat nummular, reddish yellow, and contain less blood. If there is a favorable termination, they become more fluid and frothy, with less blood and pus, and on standing show the three layers. The quantity varies from 25 c.c. to 500 c.c. in twenty-four hours. Under the microscope there will be found, beside the amœba, red blood-corpuscles, leucocytes, alveolar and oval epithelium, and bodies looking like degenerated liver-cells without a nucleus; occasionally, elastic fibres, hæmatoidin, leucin, tyrosin, and Charcot-Leyden crystals and bacteria are seen. The cheesy particles are made up of amorphous granular matter and oil-globules. Amœbæ are constantly present in varying numbers, usually not so many as in the stool, but they are somewhat larger. The number varies from day to day, and diminishes with the disappearance of the cough and expectoration. The sputa should be examined as soon after their discharge as possible, and in the interim should be kept at a temperature of 30° to 35° C. If examined in a warm stage, active movements of the amœbæ will be kept up much longer.

The amœbæ should be examined under various powers: $\frac{1}{2}$, $\frac{1}{5}$ or $\frac{1}{7}$, and $\frac{1}{12}$ inch objectives. Of these, the $\frac{1}{5}$ or $\frac{1}{7}$ inch will be found most suitable for following the movements. The amœbæ measure from 10 μ to 20 μ. They are readily recognized by their size, formation, and movements. That they have important clinical value is true, as cases have been reported in which the observer diagnosticated hepatic or hepatopulmonary

abscess secondary to amœbic dysentery, by the peculiar anchovy-sauce expectoration, and by the subsequent detection of the amœbæ.

Vegetable Parasites. The specimen is prepared in accordance with previous directions.

Moulds. *Oidium albicans* may be a constituent of the sputum when the bronchi are invaded by it, but usually it comes from the saliva. Certain other moulds have lately been considered to cause disease of the lungs by multiplication, but nothing very definite has resulted from the experiments thus far made.

Yeast-fungi. Von Jaksch reports having seen scattered yeast-cells in the pus from a phthisical cavity. Otherwise we have no knowledge of yeast being found in sputa.

Fission-fungi. *Leptothrix.* Leptothrix occurs alone in putrid bronchitis, in the sputum or in the bronchial plugs, along with fatty acid and hæmatoidin crystals. It is probably derived from the mouth, having thence entered the air-passages; or it may be taken up from the mouth by the expectoration. It is recognized by its staining blue with iodine and potassium iodide.

Sarcinæ Pulmonales. Sarcinæ pulmonales may be seen in sputa. They are larger than *Sarcinæ ventriculi*, with which they have no connection. They have no pathological significance.

Non-pathogenic bacilli and cocci may occur in all sputa, but are without significance. They are more numerous in fetid sputa. They stain with methylene-blue and other simple dyes.

Tubercle Bacillus. The organism which causes tuberculosis is a rod-shaped fungus, straight or slightly curved, without motion, varying in length from 2 μ to 5 μ (about one-fourth to one-half the diameter of a red blood-corpuscle). It usually has a beaded appearance when stained, due to the spores, which do not take up the stain that affects the rod as a whole, and which often bulge slightly beyond the edge. It is probable that this beaded appearance is caused by the contraction and breaking up of the stainable portion, permitting one to see the empty spaces between the fragments and the outer membrane. Bacilli presenting this appearance are supposed to be undergoing degeneration. Attention has recently been called to the presence in the sputum of branching forms of the tubercle bacillus. The bacillus of tuberculosis cannot be recognized in the sputum unless stained. It is recognized by the fact that when once stained, it is not decolorized by acids. This staining peculiarity belongs to but few organisms, including the smegma bacillus, the bacillus of leprosy, and the bacillus of syphilis. As under ordinary conditions these bacilli are not met with, this peculiarity in staining in a vast majority of cases is diagnostic of tubercle bacilli.

Recently Pappenheim found in the sputum from a case of gangrene of the lung stained by Gabbet's method numerous bacilli which were considered to be tubercle bacilli. At the autopsy no evidence of tuberculosis could be found. Further examination led Pappenheim to believe that these bacilli were smegma bacilli. A similar case has been recently seen in which large numbers of bacilli were present in the sputum in a case of gangrene of the lung secondary to a subdiaphragmatic abscess, which,

stained by Gabbet's method, were considered to be tubercle bacilli. The autopsy showed no evidence of tuberculosis macroscopically or microscopically. Inoculation from the lung into a guinea-pig was also negative. Fränkel has observed similar bacilli in the sputum when stained by Gabbet's method from patients with bronchiectasis.

Other members of this acid-resisting group of bacteria were found in butter by Rabinowitsch and others ("butter bacillus"), and upon certain sorts of fodder and in stable manure by Moeller ("grass bacillus II." and "timothy hay bacillus"), etc. Abbott and Gildersleeve have shown that the majority of the acid-resisting bacteria may be distinguished from true tubercle bacilli by their inability to resist decolorization by a 30 per cent. solution of nitric acid in water.

Significance of the Presence of the Tubercle Bacillus. The greatest importance attaches to the presence or continuance of tubercle bacilli in sputa. It indicates tuberculosis of the lung or of the larynx; in the vast majority of cases, of the former.

Tubercle bacilli are often to be found in the sputum when physical signs are not yet present or are indefinite. The number varies so greatly in different cases, and in the same case at different times, that in a recent attack it is impossible to judge of the extent of the disease by the number present in a given preparation.[1]

The absence of bacilli from sputa has no true value unless negative results are obtained after many trials and careful examinations by an experienced observer with the use of good stains. Hence too great care can not be taken in each and every step.

Staining the Tubercle Bacillus. In staining a cover-glass preparation the specimen is first dried and fixed. The edge of the cover-glass, with sputum side up, is then grasped with forceps and covered with the staining solution (care being taken to prevent the fluid extending to the under surface), and is held in or just above the flame until the solution boils for a second or two or until a bubble rises. The excess of the solution is washed off in water, and the slip is treated with the decolorizing agent until the color is almost or wholly removed. It is again washed in water to remove the excess of the decolorizer, and mounted for examination or given a contrast-stain; the latter is preferable.

Rapid Method. The rapid method with the specimen smeared on a slide is as follows: Cover the dried sputum with the desired stain, and steam gently for two minutes over a low flame (the slide may be held with the fingers, or after heating may be laid aside for a moment); wash off the excess of stain with water, then cover the stained sputum with the decolorizing agent and the counterstain, which should not remain more than thirty seconds. Wash away excess with water, dry the slide by blowing upon it through a pipette, and cover with a clean cover-glass, using distilled water as a mount. This method is extremely satisfactory for ordinary clinical work, especially with Ziehl's and Gabbet's solution.

[1] "A Method for the Examination of the Actual Number of Tubercle Bacilli in Tuberculous Sputum." By George H. F. Nuttall, M.D. Ph.D., Johns Hopkins Hospital Bulletin, May, 1891. This method is of pathological but not of diagnostic interest.

If fuchsin is used to stain the tubercle bacilli, methylene-blue is a good contrast-stain; while if gentian-violet is selected, Bismarck-brown is better in contrast. These contrast-stains are made as needed by dissolving sufficient dye in a few cubic centimetres of water to make the solution just transparent as seen through a test-tube of 14 mm. diameter and then filtering; or, a concentrated watery solution may be made for stock just as the concentrated alcoholic solutions of fuchsin and gentian-violet were made, diluting a small quantity of this, when needed, with enough distilled water to make it just transparent in a similar test-tube. To apply the contrast-stain, place a few drops on the cover-glass that has been prepared as above—stained, decolorized, and washed—allow it to remain thirty or forty seconds, wash off in water, and mount for examination on a glass slip, in water, oil of cloves, or Canada balsam. A drop of water will serve perfectly well for examining when the preparation is not to be preserved. In the microscopical examinations a $\frac{1}{12}$ inch oil-immersion lens and Abbe condenser, or, at the least, a $\frac{1}{7}$ or $\frac{1}{8}$ inch objective should be used. If gentian-violet has been used, the tubercle bacilli appear as dark-blue rods, with all other bodies brown, if Bismarck-brown is used for contrast-stain; while in the fuchsin staining for tubercle bacilli, and methylene-blue as a contrast, the former will be found as red rods in a blue field (background). (See Plate XII.–C, Fig. 2.)

The above rapid method of staining takes much less time than the method usually described, and gives most satisfactory results. The steps in the old method are the same as given above, except that instead of placing the staining solution on the smeared and dried cover-glass, and holding it in or above the flame until the solution boils, the cover-glass is floated in a cold solution, in a watch-glass, sputum side down, for twenty-four hours, or in a hot solution for six to eight minutes, or until moisture appears on the upper surface of the cover-glass. The remaining steps are similar.

Tubercle bacilli do not stain with the simpler dyes, but when stained by solutions of dyes made more penetrating by the addition of aniline oil, carbolic acid, or like substances, *they retain the color when subjected to decolorizing agents.* In this they differ from all other organisms, excepting, as stated, the smegma bacillus, the bacillus of leprosy, and the bacillus of syphilis.

A number of methods have been devised for the detection of the tubercle bacillus by means of its peculiar action toward stains. The most satisfactory are those known as the Koch-Ehrlich, Ziehl-Neelsen, Gabbet, and Gibbes. These methods differ chiefly in the solutions used. Slightly modified from the original, they are as follows:

I. *Koch-Ehrlich method:*

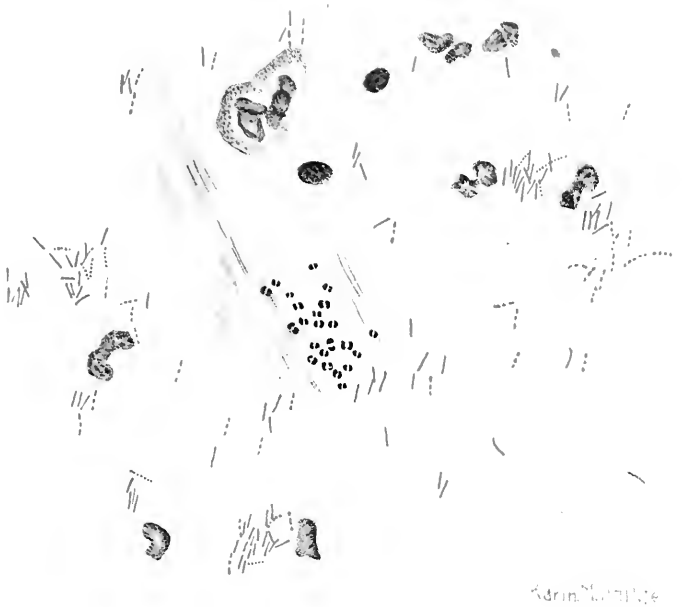
Solutions Used.

A. Concentrated alcoholic solution of fuchsin or gentian-violet.
B. Saturated solution of aniline oil in water.
C. 30 per cent. solution of nitric acid in water (decolorizing solution).

Solution A. Place in a clear bottle fuchsin or gentian-violet in substance to one-fourth its capacity, and fill with alcohol (95 per cent.);

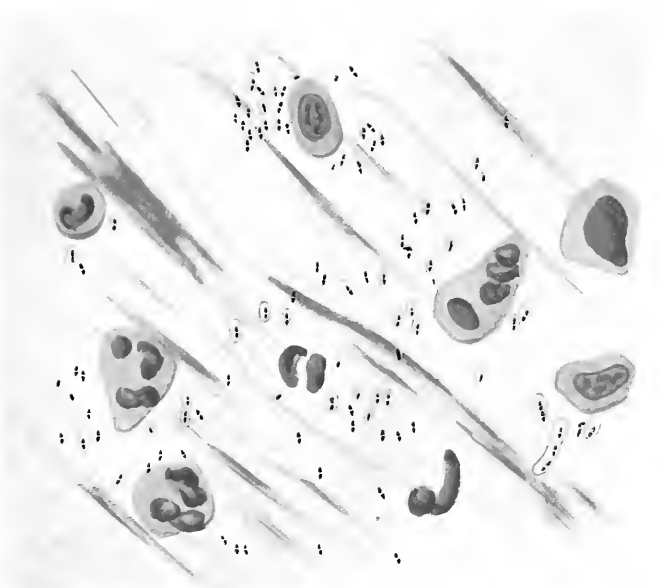
PLATE XII-c

FIG. 1



Tubercle Bacillus (Red). Diplococci (Unclassified) Blue.

FIG. 2



Diplococcus Pneumoniæ (Pneumococcus).

shake well; cork and allow it to stand for twenty-four hours. If all the dye has been dissolved, add more and shake; then let the solution stand for another twenty-four hours, and so on until some of the dye remains permanently undissolved at the bottom of the bottle. This solution remains good until used.

Solution B. To about 100 c.c. of distilled water, in a flask or other suitable vessel, add aniline oil, drop by drop, shaking the flask continuously, until the solution is opaque or until drops of the oil float on the surface. Then filter through moist filter-paper until the filtrate is perfectly clear. This solution must be made fresh as needed.

Solution C. Mix a few cubic centimetres of nitric acid and water in about the above proportion, never stronger, each time bacilli are to be stained.

The Koch-Ehrlich solution is made by adding 11 c.c. of the fuchsin or gentian solution (A) and 10 c.c. of absolute alcohol to 100 c.c. of the clear aniline filtrate (B). It should not be used after it is a week old.

II. *Ziehl-Neelsen method:*

Solutions Used.

A. Carbol-fuchsin solution:

Distilled water	100 c.c.
Carbolic acid (crystalline)	5 gm.
Alcohol	10 c.c.
Fuchsin in substance	1 gm.

This solution can also be prepared by adding a saturated alcoholic solution of fuchsin (see above) to a 5 per cent. watery solution of carbolic acid until a metallic lustre is seen on the surface of the fluid. This solution does not decompose so easily as those made with aniline oil.

B. Decolorizing solution of nitric acid, and
C. Contrast-stain of methylene-blue, as above.

The preparation and staining are exactly the same as in method I. The tubercle bacilli are stained red, the other bodies blue.

III. *Gabbet's method:*

Solutions Used.

A. Carbol-fuchsin solution (as in II.).

B. Methylene-blue solution:

Methylene-blue[1]	2 gm.
Sulphuric acid	25 "
Distilled water	75 c.c.

This solution is apt to decompose if old.

The cover-glass is prepared and stained with the carbol-fuchsin solution and washed in water as in I. Then (instead of decolorizing with nitric acid or adding a contrast-stain) the slip is washed for twenty to thirty seconds in the methylene-blue solution until a faint blue replaces the red tinge in the (slip) sputum; the excess of the solution is washed off in water, and the slip is mounted and examined as above. The

[1] An alcoholic solution of methylene-blue should first be made, and then added drop by drop, with constant stirring, to the sulphuric acid and water.

tubercle bacilli are stained red and the other bodies blue. In sputum from gangrene of the lung and bronchiectasis, decolorization with alcohol, in addition, must be employed to eliminate the presence of the smegma bacillus.

The writer has found that this method can be rapidly applied, and that it gives good results; he recommends it highly.

IV. *Gibbes' method:*

Solutions Used.

A. Fuchsin	3 gm.
Methylene-blue	1 "
Mix thoroughly in a mortar.	
B. Aniline oil	5 c.c.
Alcohol	20 "

Dissolve and add B to A slowly, stirring vigorously until A is evidently dissolved, then add 20 c.c. of distilled water, and keep in a stoppered bottle, ready for use.

Prepare slip and stain with this solution, as with the others, up to the point of decolorizing. Then wash with alcohol until the dye ceases to come away. Mount and examine as above. Tubercle bacilli will be stained dark red, the other objects dark blue.

When the bacilli are few in number, Biedert proposes that the following preliminary steps be taken: About 4 c.c. of sputum are mixed with 8 c.c. of water and 1 c.c. of solution of caustic soda, and boiled a few minutes, when about 15 c.c. of water are added and the whole again boiled until a homogeneous fluid is formed. This is allowed to stand in a conical glass for twenty-four to forty-eight hours, when the sediment is stained by the Ziehl-Neelsen or Gabbet method. Or, the homogeneous fluid can be put at once in a centrifugal machine, and the resulting sediment stained.

Sputa hardened in Zenker's fluid, embedded in paraffin, and cut, have proved most satisfactory in the study of the branching forms of the tubercle bacillus, the study of giant cells in the sputum in phthisis, and in the study of bacteria in the sputum in cases of pneumonia.

It is well to remember that in the absence of a proper decolorizing agent hot water applied for some minutes has been shown to decolorize very satisfactorily.

Streptothrix. Mycelial tufts or granules in the discharges from the lesions show the organisms, which are very resistant to acids and stain well by the stains used for detection of tubercle bacilli. Others are less resistant and are better revealed by Gram's stain. It is possible and probable that many instances of streptothrical pulmonary infection have been passed unrecognized, the organisms either having been considered tubercle or not having been revealed at all.

Smegma Bacillus. Pappenheim distinguishes smegma bacilli from tubercle bacilli by staining with a solution of corallin in absolute alcohol saturated with methylene-blue, when decolorization takes place without acid. If fat acids and myelin are present in the sputa, the bacilli are in

all probability not tuberculous. They are not found in mucopurulent but in putrid sputum.

The smegma bacilli stain with most of the reagents used in demonstrating tubercle bacilli, but are unable to resist decolorization by a 30 per cent. solution of nitric acid in water. The tubercle bacilli are, however, considered more resistant to decolorizing agents. After staining with carbol-fuchsin, if the specimen is treated with saturated alcoholic solution of methylene-blue for three to five minutes, the smegma bacilli will usually give up their stain. Or if the specimen stained with carbol-fuchsin is treated with 5 per cent. HCl for three minutes, then 70 per cent. alcohol for ten minutes, and finally with Gabbet's methylene-blue, the tubercle bacilli withstand the decolorizing and remain stained. Smegma bacilli are smaller, are not grouped like tubercle bacilli, and do not show beading.

PNEUMOCOCCUS—DIPLOCOCCUS PNEUMONIÆ; MICROCOCCUS LANCEOLATUS. The causative factor in most cases of acute croupous pneumonia in its typical form is a paired lancet-shaped coccus, often irregular in size, with a tendency to chain formation. Frequently oval or conical forms are present, and there is apt to be variation in the size of the cocci forming the pair. The organism has a distinct capsule. In the sputum of croupous pneumonia these pneumococci are usually present in large numbers. Their presence within leucocytes and their tendency to chain formation have been especially noted in such cases.

Staining the Pneumococcus. Pneumococci are stained in cover-glass preparations with the ordinary aniline dyes, as given above. The capsule may be stained and differentiated in the same way, but it more often requires a special method. Welch recommends the following: Dried cover-glass preparations are treated first with glacial acetic acid, which is allowed to drain off, and then (without washing in water) with aniline oil gentian-violet solution. (See under Tubercle Bacilli.) The staining solution is repeatedly added to the surface of the cover-glass until all of the acid is displaced. The specimen is now washed in a weak salt solution (about 2 per cent.), and examined in the same, not in balsam. The capsule and coccus can then be differentiated. Sputum stained by Gram's method, thoroughly decolorized by alcohol, and counterstained with a watery solution of eosin, or a 1 per cent. aqueous solution of aurantia, has been found satisfactory for photomicrographic work. Degenerative and involution-forms of the pneumococcus are constantly met with. There will be variations in size and shape, and the capsule may contain only the remains of a coccus, or it may be entirely empty.

Significance of the Pneumococcus. This micro-organism is found in nearly all cases of acute croupous pneumonia, and in many cases of bronchopneumonia. Its presence has also been observed in health in the saliva. It is found also in acute pleuritis, endocarditis, pericarditis, peritonitis, acute purulent meningitis, and otitis media. Its presence in empyema is considered of favorable import. It has also been found in cases of synovitis, osteomyelitis, and abscess formation in various situations. It may cause a general septicæmia—*i. e.*, pneumococcus septicæmia.

Bacillus Capsulatus Mucosus or Friedlander's Bacillus Capsulatus. This organism is found in the sputum in health in a certain number of cases. In association with the pneumococcus it can cause pneumonia. It can also produce pneumonia by itself in rare instances.

In three fatal cases of pneumonia due to the capsule bacillus alone there have been found in the sputa large numbers of capsule bacilli. These were frequently inside of leucocytes, and many alveolar cells were filled with these bacilli.

Bacillus of Influenza. This organism is found in the sputum in cases of influenza or influenza pneumonia. It was first isolated from the sputum by Pfeiffer. The organism appears as a small bacillus with rounded ends. Its length varies somewhat, and thread-like involution-forms may appear. It *stains* more deeply at the ends than at the middle, and the long forms may show irregularity of staining.

Whooping-cough Bacilli. Minute bacilli have been discovered in the sputum in cases of whooping-cough by Czplewski, Koplik, Zusch, and others. At present the results are not sufficiently uniform to prove that these bacilli are of etiological import in the disease.

Actinomyces. When the lung or pleura is infected by this fungus, actinomyces may be found in the sputum. The disease in these organs is rare. Macroscopically they appear as small kernels, yellowish white or greenish yellow, and having the shape of a millet-seed. Under the microscope they are recognized by the rounded, club-like bodies projecting from all sides of an unformed central mass. They are seen better when not stained. (See page 810.)

CHEMISTRY OF SPUTUM.

As the chemical examination of the sputum does not aid us in diagnosis, it has little or no value. *Mucin*, *nuclein*, and *serum-albumin* are constituents of sputum in health. *Peptone* is present whenever there is pus, and is especially marked in pneumonia. *Volatile fatty acids*, such as butyric and acetic, occur at times, markedly so in pulmonary gangrene. *Glycogen* has been obtained by Solomon, and a *ferment* resembling one of the pancreatic ferments has been detected, especially in pulmonary gangrene and putrid bronchitis. Of inorganic substances, *chlorides of sodium and magnesium; phosphates of sodium, lime, and magnesium; sulphates of sodium and lime; carbonate of sodium, lime, and magnesium;* and in a few cases *phosphate of iron* and *silicates* have been obtained. (Von Jaksch.)

CHAPTER XLIII.

THE URINE.

Inspection of Urine.

THE urine is not simply an index of the condition of the kidneys. It varies, within the bounds of health, in color, quantity, and quality. Food, exercise, and other conditions modify the secretion. It can readily be seen, therefore, that any general disease and many local diseases cause alterations in the character of the urine. Any abnormality of the urine may be symptomatic of renal disease or of disease beyond the point at which the urine passes out of the body. Usually abnormal changes in the urine, due to the general condition, do not give rise to local renal symptoms or to abnormal renal function. The exception is seen when an excess of uric acid, of urates, or of oxalates is passed. They may give rise to local pain and may set up sufficient irritation to cause albuminuria.

The urine in health is a clear yellow or amber-colored fluid, having a specific gravity of about 1020, and generally acid in reaction. It contains normally about 45 parts in the 1000 of solid matter, the principal part of which is urea—21½ parts. The other solids are uric acid and its salts; certain extractives—creatin, creatinin, ammonia, hippuric acid, xanthin, hypoxanthin, sarcin, pigment, etc.; and chlorides, phosphates, sulphates, with their bases, soda, potash, lime, and magnesia.

The *volume* of urine passed in twenty-four hours is usually from 40 to 50 ounces, but it may fall to 30 ounces or rise to 70 without the existence of disease. Women are believed to pass from 5 to 10 ounces less than men. The volume is diminished when the skin is acting freely, as in warm weather, and when the bowels are loose; and, on the other hand, cold, constipation, and nervous excitement, especially if it induce anxiety and fear, all tend to increase the quantity secreted.

Color. The *color* of the urine is due largely but not wholly to urobilin, which is formed from the hæmatin of the blood. The color deepens when the urine is concentrated, which occurs after a hearty meal, or exercise, especially in warm weather; and it becomes paler when a large quantity is passed. The color is frequently changed in disease. In fevers the urine soon after being passed is apt to become *turbid* from the precipitation of urates, and the color varies from white, especially in children, to yellow, brown, or pink. When the precipitate settles, the supernatant urine may be high-colored and clear, or slightly opaque from some suspended matter.

The admixture of pus and chyle gives the urine a *milky* color. The urine may also be *yellowish white* and turbid from phosphates, semen, sarcinæ, and bacteria.

The urine is *red*, reddish brown, or "smoky" in acute nephritis, the color being due to blood. It is bloody in hæmaturia, cancer of the kidneys and bladder, and in injuries of the genito-urinary apparatus. The urine is very red and clear when concentrated and containing a large amount of urates. The red color of the urine may be due to hæmoglobin, constituting *hæmoglobinuria*, or to excess of urobilin, as in scurvy and pernicious anæmia. Hæmoglobinuria occurs as the result of the action of certain poisons, such as potassium chlorate; in infectious diseases, such as scarlet fever; and in malarial fevers; also in a peculiar disease known as paroxysmal hæmoglobinuria.

Again, a *golden-red* discoloration of the urine is common in jaundice; frequently the upper layers have a greenish tinge by reflected light.

Finally, a *red color* is produced by the internal administration of logwood and fuchsin.

A *yellow* color, when opaque, may be due to suspended phosphates and urates. Urine is sometimes of a golden-yellow or saffron color in jaundice, and from the effects of santonin, picric acid, and rhubarb taken internally. A yellow or yellowish-white turbidity may be due also to a mixture of pus and phosphates, and sometimes to semen, sarcinæ, and bacteria. The urine usually becomes more or less opaque and yellow when it has undergone alkaline fermentation. Such a change occurs normally within a longer or shorter time after the urine has been passed. It is promoted by heat and exposure to air, and retarded by cold and exclusion from air. If possible, the urine should be examined before this fermentation has occurred. Pathologically, in cases of cystitis, the urine when passed is already in a state of alkaline fermentation.

The urine is sometimes *chocolate-brown* when it contains blood and the blood has been acted upon by the urine, producing methæmoglobin.

Brown, greenish-brown, or *black* urine may result from contained bile-salts; from indican; from carbolic acid, creosote, and tar used internally or externally; from the internal use of senna, and in cases where there are melanotic tumors. Senator injected melanin into human beings and obtained in four cases only a large indicanuria.

Urine is *pale* usually in proportion as it is copious in quantity. It is paler in those who are using milk or vegetable diet than in those who eat meats. Under the influence of nervous excitement, especially anxiety and the dread of an approaching ordeal, such as an examination, an abnormal quantity of very pale urine is secreted.

Pathologically, *pale* urine is characteristic of diabetes, chronic Bright's disease, and polyuria. Such urine is also secreted in hysterical attacks, at the crises of febrile diseases, and in anæmic conditions.

Quantity. The volume may be increased, diminished, or unchanged in disease. It is *increased* principally in three diseases—diabetes mellitus, diabetes insipidus, and in the middle period of chronic Bright's disease, especially in the interstitial form. In diabetes mellitus it sometimes exceeds 32 pints. It may be increased also in hypertrophy of the left ventricle, which induces greater pressure in the renal arteries as well as in the whole arterial system; and also in cystic degeneration, and in double hydronephrosis.

The urine is *diminished* in acute nephritis and in the final stages of chronic nephritis; sometimes also it is diminished in the middle period of chronic nephritis, but usually it is here increased. All diseases which directly or indirectly impair the force of the circulation lessen the secretion of the urine. Hence, the quantity is diminished in diseases of the heart muscle and in valvular diseases not fully compensated; in emphysema and in chronic bronchitis. It is lessened also in cirrhosis of the liver. In febrile diseases the urine is scanty and high-colored, and sometimes it is almost suppressed (anuria).

The urine is sometimes *suppressed* in acute nephritis, such as follows scarlet fever, and in the final stages of all the organic affections of the kidneys—chronic nephritis, hydronephrosis and pyonephrosis, etc. It may result (1) from the destruction of the secreting tissue of the kidney or interference with its nervous or vascular supply, or (2) from mechanical obstruction to the outflow of urine. To the first class belong the cases of suppression occurring in acute and chronic nephritis, and those due to shock and collapse, whether occurring in the collapse stage of yellow fever, cholera, and other grave febrile diseases, or from serious internal injuries.

Such suppression sometimes follows slight operations on the urethra (urethral fever); or results from the internal administration of drugs, the excretion of which occasions violent irritation of the kidney—such as cantharides, turpentine, and even the inhalation of ether. Clinically, suppression not due to obstruction is distinguished from the obstructive form by the character of the urine, which is usually not entirely suppressed, and by the more rapid course of the disease. The urine, according to Roberts, is either concentrated or it contains albumin, blood, and casts. Death or recovery results within a day or two. In the obstructive form, on the other hand, the urine which escapes past the obstacle is pale, watery, and devoid of albumin and casts.

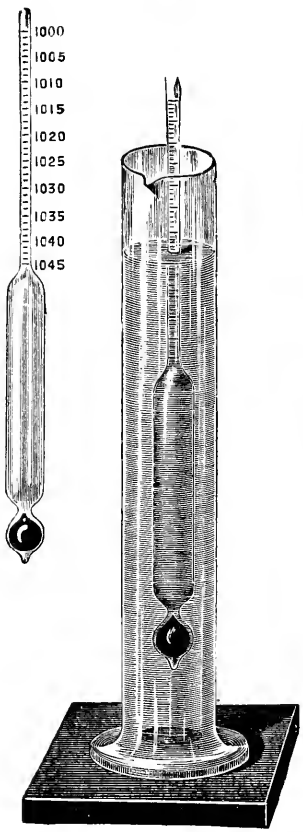
Obstructive suppression is the result of the plugging of the ureter by a calculus when the opposite kidney is either absent or incapable of secreting. It also results from the occlusion of the ureters by morbid growths especially at the vesical orifices, from lateral pressure upon the ureters, or from some interference with or malformation of the ureters or renal arteries.

Acute transient obstructive suppression occurs sometimes in persons with enlarged prostates or old strictures who have drunk too freely of alcoholic beverages, and perhaps have wound up a debauch by sexual intercourse.

Density. The average density of normal urine is about 1020. It may fall to 1015 or rise to 1025, depending upon the quantity of fluid and food taken, the condition of the atmosphere, especially as regards temperature, and upon mental influences usually of an emotional character. The specific gravity of the urine is tested by a urinometer graduated for degrees of density between 1000 and 1040. Only a reliable instrument should be used. As the density of the urine passed at different times during the day varies greatly, the urine for the whole twenty-four hours should be saved and a specimen of this tested.

The method of taking the specific gravity is very simple. A test-tube or graduate, having a diameter of about $1\frac{1}{4}$ inches and a length of 6 or 7 inches, is filled with urine to such a point that the lowest part of the urinometer, when inserted, floats clear of the bottom of the tube. The instrument must also float free of the sides of the tube. The specific gravity should then be read off from below—that is to say, by holding the tube up so that the level of the fluid is a little above that of the eye. Most *urinometers* are graduated for 60° F., but in ordinary examinations it is not necessary to have the urine exactly at this temperature; it should, however, be allowed to cool after it has been passed, otherwise the specific gravity will appear to be too low.

Fig. 220.



Urinometer. (W. Simon.)

In *disease* the specific gravity varies more widely than in health; it may fall to 1005 or 1000 in diabetes insipidus and chronic Bright's disease, and rise to 1060 or even higher in diabetes mellitus. As a rule, to which the urine in diabetes mellitus is the principal exception, the color is an index of the density; pale urine being of low density and high-colored urine of high density.

The density is increased when the urine is scanty in amount, whether as the result of fever, acute nephritis, large consumption of solid food, exercise, or free sweating. In all such cases the specific gravity rarely rises above 1035, and usually not above 1028 or 1030. When the specific gravity rises above 1035, and the urine is pale in color, the presence of sugar is to be suspected; and when it rises above 1040, sugar is almost certainly present.

The specific gravity is lowered by drinking copiously, by the effect of external cold, by a diet of vegetables and milk, and in general by the same causes that make the urine copious. Usually, but not always, a urine containing a large amount of albumin is of low density.

Pathologically, a low specific gravity is encountered in diabetes insipidus, in which it may fall nearly or quite to 1000; generally in the middle or quiescent period of chronic Bright's disease; in the crisis of fevers; in obstructive suppression; in hysterical attacks, and in hydronephrosis.

Specific Gravity as an Index of the Amount of Solids. If the last two figures of the specific gravity be doubled, the sum will represent the amount of solid matter in 1000 grains of urine. This is Trapp's method; the estimate is only approximate, but it is useful. Of course, the urine for twenty-four hours must be used.

Reaction. The reaction of *healthy urine* is usually *acid*, but it may be neutral or slightly alkaline about two hours after a meal of mixed food. The acidity is tested with litmus-paper: the blue paper is turned purple or red by an acid, and the red paper is turned blue by an alkali. Violet paper is to be preferred, as it is suitable for showing both reactions, an alkali turning it blue and an acid red.

The acidity of the urine is *increased* in gout, lithiasis, acute rheumatism, diabetes, chronic Bright's disease, and as the result of the administration of vegetable or mineral acids.

The urine is *alkaline* because of alkaline fermentation in the bladder in cystitis; from the presence of much blood or pus; from prolonged immersion of the body in a cold bath; in debilitating diseases and in some cases of nervous dyspepsia, and as the result of the internal administration of alkalies.

Urinary Sediments. A white flocculent sediment, composed of epithelium and mucus, occurs normally in most urines after they have stood for some hours.

A dense sediment, varying in color from that of brown sugar to pink or red, consists of amorphous urates. It dissolves upon the application of heat. A sediment usually resembling red pepper, but sometimes of a brown color, consists of uric acid, the presence of which may be proved by the murexide test. The suspected material is placed in a crucible or evaporating-dish with a few drops of nitric acid. As heat is applied the uric acid or amorphous urate dissolves with effervescence. Heat is now kept up until the material is evaporated to dryness; it is then allowed to cool. If it is now touched with a glass rod that has been dipped in strong ammonia, a characteristic blue or violet color is produced. Uric acid is not usually so abundant as the sediment of amorphous urates; it sinks more rapidly, and is deposited from acid, high-colored urines.

A yellowish or whitish sediment may consist of sodium urate.

A white sediment usually consists of phosphates, associated with which we sometimes find a white sediment consisting of ammonium urate, with or without pus. Such urines are alkaline. A white sediment may be due to uric acid, especially in children.

A yellowish-white sediment may consist of pus, with or without mucus. If the urine is acid, the sediment is loose and free to move; but when the urine is alkaline, the sediment consists of a viscid, coherent mass, which can be drawn out into tough, stringy filaments.

A chocolate-brown sediment, occurring in a reddish, smoky urine, consists of blood from the kidneys. Clots of blood come from the ureters, bladder, or urethra.

Odor. The odor of normal urine is sometimes spoken of as aromatic, but generally it is sufficiently characteristic to be described best as urinous. When the urine is concentrated, the odor is intensified, and may become unpleasantly strong, like the urine of the horse.

Certain articles of food, such as garlic and asparagus, give the urine characteristic odors. Turpentine, both when taken internally and inhaled, gives to it the odor of violets. The odors of copaiba and cubebs can be detected in the urine of patients who are taking these drugs.

In marked cystitis the natural urinous odor becomes more pungent, and is blended with a strong ammoniacal odor. When much pus is present, and the urine has stood a while, a putrid odor is developed.

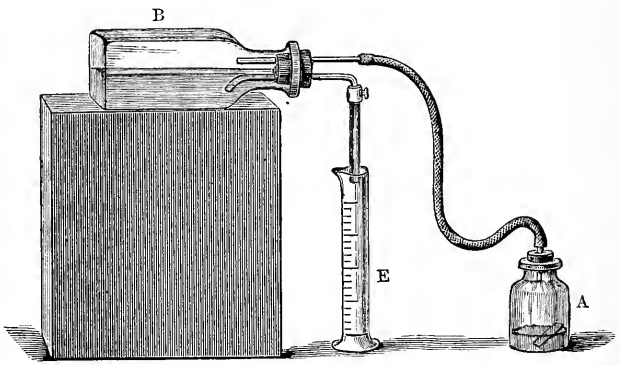
In diabetes mellitus the urine has a sweetish, hay-like odor. In diabetic coma the odor is sometimes like that of chloroform, due to the presence of acetone and diacetic acid in the urine. This odor, however, is more likely to be detected in the breath.

Chemical Examination of the Urine.

Examination of the urine by the unaided senses, which has been dwelt upon thus far, is simply preliminary to an examination by chemical methods and by instruments of precision, particularly the microscope.

Urea. Urea is freely soluble in water, and hence never appears as a sediment. It is the most important final product of nitrogenous disintegration in the body, and is an index of the eliminative power of the kidneys. Usually the density of the urine increases in proportion to the amount of urea contained in it. The average daily amount of urea excreted by an adult man between the ages of twenty and forty years is

FIG. 221.



Squibb's ureometer.

about 500 grains. The urea, like the total volume of the urine, is subject to variations within the limits of health. It is increased after a meal, especially if the latter be rich in nitrogenous food; after copious ingestion of liquids, and by a close atmosphere. On the other hand, fasting, free perspiration, a loose condition of the bowels, and a vegetable or milk diet diminish the quantity of urea. Again, the quantity varies with the age of the person. According to Ralfe, at five years the daily amount is 180 grains; at twelve, 320; at twenty-one, 535, and at forty years, 555 grains.

A large man will excrete absolutely more than a small man, and a large muscular man will excrete relatively more than a fat man of the same height.

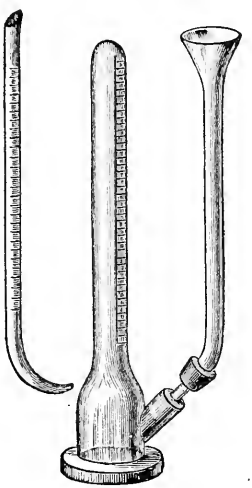
The excretion of urea is increased in fever and inflammatory diseases;

in diabetes mellitus and insipidus; in malaria, pernicious anæmia, and after a crisis in pneumonia. It is increased also by certain beverages, as coffee, and by many drugs, especially those which act as hepatic stimulants.

It is diminished in all forms of nephritis, especially when uræmia results; in acute gout and chronic rheumatism; in disease accompanied by emaciation and cachexia; in leprosy, pemphigus, melancholia, imbecility, catalepsy, hysteria, and cholera. (Saundby.)

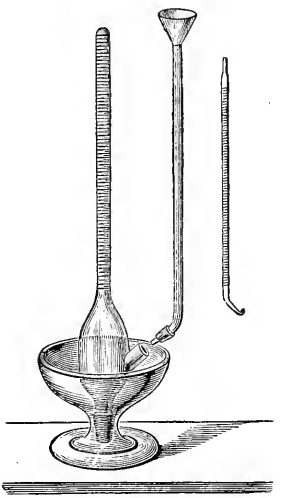
Estimation of Urea. For the methods employed in the exact quantitative estimation of urea the student is referred to special works on the urine.

Fig. 222.



Green's ureometer.

Fig. 223.



Marshall's ureometer.

For ordinary clinical purposes the apparatus devised by Professor Charles Doremus, and known by his name, gives sufficiently accurate results. The principle upon which it is based is that urea when brought in contact with sodium hypobromite is decomposed, and free nitrogen is eliminated. The nitrogen evolved is the measure of the urea contained in the urine. The instruments are graduated so that each division of the scale represents 1 grain of urea per fluidounce of urine.

Specimens should be taken from the mixed amount of twenty-four hours on account of well-known variation at different times.

The hypobromite solution is prepared by dissolving 100 grammes of sodium hydroxide in 250 c.c. of water, cooling the solution, and then adding 25 c.c. of bromine.

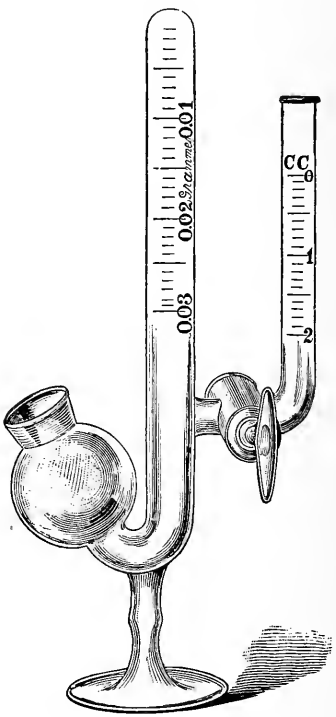
It is better, however, to prepare the hypobromite solution freshly for each examination. This can readily be done by having a solution of sodium hydroxide containing 6 ounces to a pint of water. It should be kept tightly corked with a rubber or paraffined stopper. The sodium hydroxide solution is poured into the long tube of the ureometer to the mark =, then $\frac{1}{10}$ its volume of bromine is introduced by means of a

pipette, and sufficient water added to fill the long arm and the bend of the tube. The hypobromite solution should fill the tube completely, and any bubbles rising to the top of the tube should be removed before the introduction of the urine. The pipette is then filled with the urine up to the 1 c.c. mark, any urine adhering to its surface being carefully wiped off. The pipette is introduced carefully, so as not to compress the bulb

Fig. 224.

Hüffner's ureometer.

Fig. 225.



Doremus' ureometer.

until the point extends as high up as possible beyond the bend. The bulb is now compressed slowly until 1 c.c. of urine has been introduced. Decomposition of the urea occurs and bubbles of nitrogen rise to the surface of the long arm of the tube; when bubbles of gas cease to be evolved, the volume of nitrogen gas is read off, and according to the graduations on the tube considered as so many grains of urea per fluid-ounce of urine, or as so many milligrammes of urea in 1 c.c. of urine, according to whether it is graduated in the English or in the metric system.

Chlorides. The presence or absence of chlorides is sometimes of diagnostic value. They are increased when absorption of exudates or transudates is going on, and in malarial fevers, diabetes insipidus, and Bright's disease. They are diminished or absent in pneumonia during its progressive stage, and in fevers. The chlorine of the chlorides can be detected and roughly estimated by an 8 or 10 per cent. solution

of argentic nitrate. A few drops of nitric acid are first added to the urine, to prevent the silver from precipitating phosphoric acid. A single drop of the silver solution mentioned will precipitate the chlorine of the chlorides in a thick white lump, which falls to the bottom of the test-tube, provided the amount present is normal. If, on the other hand, the quantity is diminished to 0.1 per cent. or less, it will not be precipitated in a lump or lumps, but a white cloudiness is produced which renders the whole solution opaque. If no precipitation or cloudiness occurs, the chlorides are absent.

Sulphates. The daily excretion of sulphuric acid is about $2\frac{1}{2}$ grammes, chiefly in the form of sodium and potassium salts—inorganic sulphates. The organic sulphates are combinations with phenol, cresol, indol, skatol, etc.

Test for Inorganic Sulphates. To 10 c.c. of urine acidulated with a few drops of hydrochloric acid, 3 c.c. of a 10 per cent. barium chloride solution are added. If the normal amount of sulphates is present, an opaque milkiness develops; if the precipitate is thick and creamy, the sulphates are in excess; if the specimen merely becomes opalescent, they are diminished.

An increase in the percentage of sulphates when not due to the ingestion of phenol and allied substances for medicinal purposes, indicates the presence of a putrefactive process in the body, as when there is a putrid abscess or retention of the intestinal contents.

Qualitative Tests for Albumin. *Serum-albumin* is of common occurrence, but can not ever be looked upon as a normal constituent of the urine, though its presence by no means indicates disease of the kidney. While it is the ordinary form of albumin present in the urine, other proteids, as globulin, mucin, peptone, albumose, fibrin, and also hæmoglobin and methæmoglobin, are found at times. The most trustworthy tests for ordinary albumin (serum-albumin) are: boiling with the addition of nitric or acetic acid; overlaying cold nitric acid with urine (Heller's test); the picric acid, the potassium ferrocyanide, and the potassio-mercuric iodide (Tanret's) tests. The author believes that many of the recent tests, such as sodium tungstate, acidulated brine, magnesium nitrate, phenic-acetic acid, and trichloracetic acid, are too sensitive and precipitate other substances in the urine, and therefore are not reliable for clinical work. *Serum-globulin* responds to all the following tests for serum-albumin. Its differentiation is not difficult, but usually unnecessary. (See note on page 650.)

Boiling and Nitric Acid Test. A narrow, long test-tube is filled two-thirds full of urine and the upper third boiled thoroughly, and then a few drops of nitric acid are added. Any albumin present will be coagulated and appear as a white cloud, contrasting strongly with the clear unboiled urine beneath it. When the albumin is moderate or even small in amount, it can be detected without difficulty by simply holding the test-tube up to the light. When there is only a faint trace present, it will be overlooked unless the tube be examined against a dark surface in such a way that the light falls upon it from above, in front, and preferably a little to one side. A cloud may escape detection when

looked for by artificial light, but may be distinct by daylight. Serum-globulin is also precipitated by this test. But serum-globulin is not often present by itself, and its significance is not yet understood. It may be detected in any urine, as Roberts points out, by diluting the urine with pure water, the urine then becoming more or less milky. It may be removed from urine by saturating the latter with magnesium sulphate and filtering off the precipitated globulin. The presence of serum-globulin in no way interferes with the test for serum-albumin.

If the urine is opaque from amorphous urates, it is unnecessary to filter them out; heat much below boiling will dissolve them, the precipitation of albumin occurring later at a higher temperature.

If the urine is alkaline or faintly acid, phosphates will produce a cloud upon heating the urine; but they are instantly dissolved upon the addition of a few drops of nitric or acetic acid.

Mucin produces an opalescence upon heating with an organic acid, but Saundby declares that it coagulates not in flocculi, as is the case with albumin, but in the form of tiny filaments.

Boiling and Acetic Acid Test. This is preferred by many to the preceding test. It is performed in a similar manner. Acetic acid is, however, not reliable for acidulation; it precipitates the mucin which is often found in healthy urine, forming a white cloud which is apt to be mistaken for albumin; this is especially true in urines of high specific gravity containing uric acid, urates, or oxalates.

The Nitric Acid (Heller's) Test. This test, while not so delicate as the acetic acid test, is very simple and accurate in its results. Cold nitric acid is poured into a test-tube to the depth of about an inch. The tube is then inclined to an angle of about 45 degrees, and urine allowed to flow gently down upon the acid by trickling along the side of the tube from a pipette or glass tube. At the point of contact of the acid and urine a zone of white, coagulated albumin forms. The test can also be made as follows: Into a short, broad test-tube several cubic centimetres of urine are poured; nitric acid is introduced with a pipette provided with a rubber bulb by passing the pipette through the urine to the bottom of the tube and gently pressing the rubber bulb; care must be taken to withdraw the pipette as soon as the last portion of acid is expelled, so that no air-bubbles will break up the point of contact of the urine and acid. The thickness of the white zone is generally an index of the amount of albumin present. If there is barely a trace of albumin, half an hour may be required to develop any opalescence.

A cloud of urates is sometimes produced and obscures the test. This cloud does not, however, begin at the point of contact and extend upward, but at the upper level of the urine and extends downward, and is dissipated by heat.

Patients who are taking copabia or cubebs pass a urine which gives a white zone at the point of contact with cold nitric acid, but heat diminishes the opacity, and the precipitate is soluble in alcohol; the odor of the drugs in the urine assists in the detection of their presence.

The Picric Acid Test. This is an extremely delicate test for albumin. A saturated solution of picric acid is allowed to flow down upon and

slightly mix with the upper layers of the urine, which half fills a good-sized test-tube. At the point of contact an opaque white zone of coagulated albumin is formed. If no white zone appears, albumin is almost certainly absent. Hence, the picric acid test is a valuable negative test. But, unfortunately, a white zone is formed by peptone, mucin, and various alkaloids, particularly quinine. The white zone produced by the presence of the substances just named disappears upon the application of heat, whereas an opalescence due to albumin becomes diffused throughout the whole urine.

The Potassium Ferrocyanide Test. This test is highly recommended as simple, rapid, and accurate by Purdy, who performs it as follows: Into a test-tube are poured 15 to 30 drops of acetic acid, and then two or three times that amount of potassium ferrocyanide solution (1 to 20) is added, and the two thoroughly mixed by shaking the tube. The urine is now added to the depth of two-thirds of the test-tube. If any albumin is present, it will be precipitated throughout the whole volume of urine in the form of a milky-white flocculent cloud, the intensity depending on the amount of albumin present. By this method all modifications of albumin, acid or alkaline, are precipitated and the precipitation of mucin is avoided. It gives no reaction with phosphates, urates, peptones, vegetable alkaloids, or the acids found in the urine after the ingestion of copaiba, etc. This test may also be performed as follows: An ordinary test-tube is half filled with urine and a drachm or two of the potassium ferrocyanide solution (1 to 20) are added. After thoroughly mingling the reagent and the urine, a few drops of acetic acid are added. If albumin is present, it will plainly come into view. This test, therefore, depends upon the production of a cloudiness or milkiness throughout the entire mixture in the tube. To some eyes the albumin is not so readily perceived as in those tests which depend upon the formation of a distinct line at the point of contact.

The Potassio-mercuric Iodide (Tanret's) Test. The solution is made as follows: potassium iodide, 3.32 grammes; mercuric chloride, 1.35 grammes; acetic acid, 20 c.c.; distilled water, about 30 c.c. (the potassium iodide and the mercuric chloride should be dissolved separately in the water and the solution mixed, to which the acetic acid is added and the whole made up to 60 c.c. with distilled water). As thus prepared, the test is applied by the contact-method by overlaying the reagent with urine. This test responds to all modifications of albumin, also to peptones and proteoses, as well as to the vegetable alkaloids and acids found in the urine after the ingestion of copaiba, etc. All reactions except those occurring with albumin, mucin, and the acids found in the urine after the ingestion of copaiba, etc., disappear with heat. It is a very good and delicate control-test for albumin. The solution, however, is of a yellowish hue, quite similar to the color of urines of low specific gravity; this sometimes renders the line of contact difficult to perceive.

It is well to follow a routine method in testing for albumin: first, by boiling and the addition of nitric acid, and then the contact-test (Heller's); if there is a doubt, either the potassium ferrocyanide or picric acid test; finally, Tanret's solution will reveal minute quantities of albumin, and may be used as a confirmatory test.

In all the tests for albumin mentioned a clear urine is necessary, especially when the amount of albumin is very small. This can be obtained by filtration when the opacity is due to pus, blood, mucus, or uric acid; and more effectively by the addition of a small quantity of sodium hydroxide, warming slightly, and filtering. If the filtrate is not clear, a few drops of magnesium fluid (magnesium sulphate, pure ammonium chloride, and pure liquor ammoniæ, of each, 2 drachms; distilled water, 2 ounces), as recommended by Hoffmann and Ultzmann, may be added, and the urine again warmed and filtered.

Résumé of Tests for Albumin.

I. The *heat* test.

A. Method: Albumin is precipitated on boiling.

B. Exceptions: 1. In alkaline urines albumin may be overlooked from the formation of soluble potassium and magnesium compounds. When patients are taking alkaline salts, the test may be fallacious.

2. An excess of acid may also interfere with the test.

3. Feebly alkaline or neutral urines produce a precipitate of earthy phosphates, but it is instantly soluble in a small quantity of acid.

4. Patients on a vegetable diet pass urine containing carbonates which precipitate with heat. The addition of an acid causes great evolution of gas.

II. The *heat* and *acetic acid* test.

Method: Determine the reaction of the urine. If alkaline, make faintly acid with acetic acid; then boil and add a little more acetic acid. If there is no precipitate, boil again. The acetic acid precipitates nucleoproteids, which are excluded by the methods above described.

III. The *heat* and *nitric acid* test.

A. Method: Bring the urine to the boiling-point and add nitric acid drop by drop, shaking the mixture between each addition. A small precipitate is thrown down even if a very small amount of albumin is present. The nitric acid should not exceed more than one-tenth of the volume of urine examined. The urine must not be heated after the addition of the acid.

B. Exceptions: 1. In concentrated urines, uric acid or its salts may precipitate. Distinguish from albumin by filtering off the precipitate and testing it by the biuret reaction, or dilute the urine with an equal volume of water when uric acid will not precipitate.

2. Resin acids in turpentine, benzoin, cubebs, and other balsams, if present in the urine, are precipitated by nitric acid. Distinguish from albumin by adding 1 or 2 volumes of alcohol when the solution is cool. The precipitate of resin acids is dissolved.

3. In urines containing biliverdin a precipitate is formed. Distinguish from albumin by adding alcohol, which dissolves biliverdin.

IV. *Cold nitric acid* test.

A. Method: Pour the urine gently on the nitric acid. The albumin coagulates in the presence of an excess of strong nitric acid. A ring appears at the surface of contact if albumin is present. A second ring may be seen $\frac{1}{10}$ to 1 cm. above the junction, due to nucleoproteids. Distinguish from albumin by repeating the test with urine diluted with 2

or 3 volumes of water. The albumin ring diminishes and the nucleoproteid ring is unchanged or increased. A haze due to nucleoproteid may form, and also continue after dilution.

B. Exceptions: 1. In concentrated urines a secondary ring due to uric acid may form above the junction. It is soluble on gently heating, and does not form when the urine has been diluted.

2. In highly concentrated urine a precipitate of urea nitrate may fall. Distinguish by its crystalline nature.

3. Resin acids cause a precipitate of uniform cloudiness. Distinguish by solubility in alcohol.

4. In highly colored urines the urinary pigments form a colored ring at the plane of contact, and in bilious urines the play of colors, as in Gmelin-Malin-Heintz's test for bile, is seen.

5. The urine of patients taking alkaline iodides gives a dense brown ring of iodine. Distinguish by adding a few cubic centimetres of chloroform and mixing them. A violet tinge is imparted to the liquid.

6. Albumoses are precipitated, as well as all forms of albumin. Distinguish by the previously mentioned tests. Peptone and vegetable alkaloids are not precipitated.

V. The *potassium ferrocyanide* and *acetic acid* test.

A. Method: It is best performed as a ring test. The urine should be carefully run into a mixture of 20 or 30 drops of acetic acid and 60 or 90 drops of saturated solution of potassium ferrocyanide. A white ring forms at the junction if albumin is present. With small amounts of albumin the ring takes some minutes to form.

B. Exceptions: 1. Albumoses are precipitated. They are soluble in excess of acetic acid. They disappear on heating and reappear on cooling.

2. Resin acids give a precipitate which is soluble in alcohol.

3. Phosphates, urates, alkaloids, and peptones are not precipitated.

VI. Roberts' *brine* test.

Saturated sodium hydrate solution with 5 per cent. hydrochloric acid. It does not darken the urine nor precipitate uric acid.

A. Method: Use the ring test, which shows albumin and albumoses.

B. Exceptions: Resin acids precipitate. Distinguish by dissolving in alcohol.

VII. The *salt* and *acetic acid* test.

The acetic acid is substituted for HCl, and a large excess of salt solution used.

A. Method: The salt solution is first added to the urine and thoroughly mixed. Acetic acid is then poured in. Nucleoproteids are not precipitated. (All other forms of albumin are precipitated.) Salt and vinegar may be used, and the mixture heated in a metal spoon.

B. Exceptions: 1. Albumoses form and disappear on heating, to reappear on cooling.

2. If albumoses and albumin appear together, boil for a short time and filter the hot fluid through a warm filter. The clear filtrate becomes turbid from albumoses as it cools.

3. Resin acids and uric acid are precipitated, the latter only in concentrated urines and after standing. Distinguish by the usual tests.

V. and VI. do not generally precipitate nucleoproteids. With VII., if equal parts of urine and salt solution are used with a few drops of acetic acid, nucleoproteids are not precipitated. The solution must be boiled when test VII. is employed.

VIII. *Salicylsulphonic acid* test.

All forms of albumin are precipitated. The precipitate becomes flocculent on heating. If the urine is alkaline more of the reagent is needed than if acid. Phosphates, urates, bile, alkaloids, and drugs do not give a reaction.

A. Method: After adding the solution to the urine, heat and allow to stand.

B. Exceptions: Albumoses are precipitated, but disappear on heating and reappear on cooling.

IX. *Trichloracetic acid* test.

Exceptions: 1. Precipitates uric acid when in excess. Distinguish by heating, which dissolves the acid, or by diluting the urine before applying the test.

2. Nucleoproteids give an opalescence. Albumoses are not precipitated.

X. *Picric acid* test.

A. Method: A saturated solution of picric acid must be used alone, or in combination with hydrochloric or acetic acid. Value doubtful.

B. Exceptions: Uric acid, creatinin, nucleoproteids, alkaloids, potassium salts, and albumoses are precipitated.

XI. *Millard's reagent.*

Value doubtful. Precipitates albumoses, nucleoproteids, alkaloids, and resin acids. Distinguish by usual tests.

XII. *Tanret's reagent.*

Very delicate. Precipitates all forms of albumin, albumoses, nucleoproteids, peptones, alkaloids, and resin acids. Distinguish by usual tests.

XIII. *Spiegler's reagent.*

Delicate. Precipitates albumin, albumoses, and nucleoproteids, but not peptones.

XIV. *Acetic acid* test.

Method: Filter the urine and add acetic acid to a portion, pouring the two in the tube held against a black background. Albumin and nucleoproteids are precipitated. Distinguish by diluting the filtered urine with 2 or 3 volumes of distilled water, then add acetic acid, and compare the precipitate with that in an undiluted specimen. A nucleoproteid precipitate will increase in intensity. An albumin precipitate will diminish or remain unchanged.

Salicylsulphonic acid is the most delicate test. An objection to it is the fact that it precipitates nucleoproteids. Control the test by Heller's cold nitric acid test, from which the nucleoproteids are removed as above described.

Quantitative Estimation of Albumin. The most direct method is by coagulating the albumin by boiling, collecting it upon a weighed filter, washing with water and finally with alcohol, drying and weighing it. Such a process, however, consumes too much time for clinical purposes,

and it is not faultless. An approximate estimation may be made by boiling the urine in a test-tube, adding several drops of nitric acid, allowing the albumin to settle, and then comparing the depth of albumin with the height of the column of urine. In this way we may speak of urine furnishing one-tenth or one-quarter of its bulk of coagulated albumin.

Esbach has invented an *albuminometer* (Fig. 226) which gives good results. The solution used to precipitate the albumin consists of 10 grammes of picric acid and 20 grammes of citric acid, chemically pure and dry, dissolved in 900 c.c. of hot water; and after cooling, diluting the solution to 1000 c.c. The urine is diluted with a definite amount of water if it contains too much albumin. The albuminometer is filled to the mark U with urine, and from that mark to R with the reagent. The tube is then corked with a rubber stopper, turned upside down 10 times, so as to mix the urine intimately with the reagent, and then allowed to stand undisturbed for twenty-four hours. At the end of that time the depth of the sediment of coagulated albumin is ascertained by observing where the top of the sediment comes in contact with a mark on the scale on the tube. Each mark corresponds to 0.1 per cent. of albumin.

Fig. 226.

Esbach albuminometer.

This estimation, as already stated, is not absolutely accurate. Nevertheless, if used systematically, and always in the same way, relative values will be obtained, and these are the most important in watching the progress of a case, as they give positive information regarding an increase or diminution of the amount of albumin in the urine. It scarcely need be said that the urine tested must be a portion of the whole twenty-four hours' urine.

The estimation of the amount of albumin is also readily made with the centrifugal machine: to 10 c.c. of the albuminous urine are added 3.5 c.c. of potassium ferrocyanide solution (1 to 10) and 1.5 c.c. of acetic acid; the mixture is then revolved in the machine about three minutes, and the amount of precipitate read off.

Albuminuria. Albuminuria is not indicative of disease of any one organ, nor does it point to any general pathological condition. It occurs:

1. In diseases of the kidney: acute and chronic Bright's disease, amyloid disease, tuberculosis, cancer, abscess, and calculus.

2. In disturbances of the circulation: diseases of the heart and chronic pulmonary diseases, as emphysema; obstruction of the renal arteries or veins, cirrhosis of the liver, peritonitis, pregnancy, abdominal tumors; in passive congestion due to great weakness; in anæmia and Graves' disease.

3. In febrile and inflammatory diseases: in the eruptive and infectious fevers, in rheumatism, diphtheria, pneumonia, and gout.

4. In blood diseases: purpura, leucocythæmia, and scurvy.

5. From the poisonous action of drugs: lead, turpentine, and others.

6. In nervous disorders: concussion of the brain and cerebral hemorrhage, epilepsy, tetanus, and delirium tremens; as Pye-Smith remarks, it is doubtful whether albuminuria is caused by the nervous diseases.

7. In local extrarenal affections: pyelitis, cystitis, gonorrhœa, and leucorrhœa.

8. As a functional disturbance: in young persons, particularly of the male sex, after exercise, a special diet, or a cold bath. Albumin may be found after rising in the morning, or early after dinner, or toward evening. On account of its occurring only at certain times it has been called "cyclical" or "intermittent," and because there is no evident disease present, it is occasionally spoken of as "physiological" albuminuria.

Goodhart examined the urine of 1500 individuals and noted albumin in 272, or in 20 per cent. In 39 cases the albuminuria could not positively be said to be due to disease of the kidney. Of these 39, 26 were males and 13 females. In 32 of the 39 cases it was temporary, and in most of them it disappeared within forty-eight hours or sooner. In 2 cases there were oxalates in the urine; in 1 hæmoglobinuria; in 8 leucorrhœal discharges and discharges from other parts of the genital passages (see division 7); and in 17 a markedly neurotic temperament. These last he thinks the most typical cases of intermittent albuminuria; on the whole, he regards the condition as less common than has been supposed.

One variety of functional albuminuria is apparently due to irritation of the kidney produced by the excretion of oxalates and uric acid. The urine is of increased density, 1028, 1030, or higher, and contains uric acid or oxalate of lime, or both, and cylindroids. Tube-casts are uncommon. The albuminuria usually disappears under proper diet. This condition is sometimes called "morbus Da Costæ."

It is conceded that there may be albuminuria of renal origin without renal disease, but the diagnosis must be by exclusion, and can be reached safely only after extended observation. The most important elements in the diagnosis are: the age of the patient, unimpaired general health, a specific gravity of the urine normal or above normal; the fact that the albuminuria is influenced by diet and exercise, and that it tends to disappear under suitable regimen. The prognosis is favorable.

Mucin. Nucleo-albumin, or nucleoproteid, is nucleic acid and chrondrosulphonic acid combined with a proteid. Sometimes, pathologically, taurocholic acid enters into the combination. This is not true mucus, but urinary mucus. It is present in the urine in health, being especially abundant in women from the admixture of the vaginal secretion, and in excess in inflammatory conditions of the urinary tract. It is distinguished from albumin by the fact that it gives a precipitate upon the addition of a vegetable acid, as acetic or citric. The precipitate is increased by removing the salts of the urine by dialysis, or by dilution of the urine with two or three volumes of distilled water, diminishing thereby the relative proportion of salts to mucus. It is precipitated by dilute mineral acids, but is soluble in concentrated mineral acids or dilute alkalies.

According to Roberts, the best method for the detection of mucin is by means of a saturated solution of citric acid, employed in the same manner as the contact-method of applying the nitric acid test for albumin. A small quantity of the urine is first put in a test-tube, and citric acid allowed to trickle down the sides of the tube until it forms a distinct layer below the column of urine. If mucin is present, there will gradu-

ally appear an opalescent zone immediately above the layer of acid. Acetic acid, mixed with one-third of its volume of glycerin, answers admirably as a test for mucin. Sometimes, when mucin is very abundant, the addition of an excess of acetic acid produces a marked milkiness in the urine, which is not discharged by boiling.

Blood. Urine containing blood is usually red in color or reddish brown and opaque, but it may be chocolate-brown if the blood is present in large quantity and has been acted upon by the urine. Such urine necessarily contains albumin.

Blood occurs in the urine from (1) *diseases of the kidney* and *urinary passages*, among which are Bright's disease, acute congestion of the kidney, renal calculus, cancer, tuberculosis; from ureteritis, cystitis, urethritis, and injuries; (2) from *general diseases*, such as the eruptive and intermittent fevers, scurvy, purpura, peliosis rheumatica, leucocythæmia, cholera; (3) from *adjacent organs*, as in menstruation and hemorrhage from the uterus; (4) from the *toxic action of drugs*—cantharides, turpentine, and other violent irritants of the kidney; (5) *vicariously*—occasionally menstruation fails to occur and hæmaturia replaces it. The same is true of bleeding from piles. Latour has reported a case of asthma which subsided suddenly upon the appearance of hæmaturia.

The chemical tests for blood are the same as those for its coloring-matter, and will be referred to under Hæmoglobin.

Hæmoglobin. Hæmoglobin is, of course, present whenever blood is, but sometimes it occurs independently of hæmaturia. Thus it is found in grave infectious diseases, as the result of the toxic action of drugs, such as carbolic acid, and in an independent disease known as paroxysmal hæmoglobinuria. A suitable test consists in adding 1 or 2 drops of freshly prepared tincture of guaiac to about 1 drachm of urine, then shaking the mixture and adding several drops of a solution of hydrogen peroxide. If blood-coloring matter be present, a beautiful blue coloration will be produced.

The same test answers for methæmoglobin and hæmatin.

Paroxysmal Hæmoglobinuria. The urine contains blood, or only the coloring-matter of the blood. Hæmoglobinuria is more frequent in adults; it may be excited by a cold bath, or exposure to cold, or by exertion. It is sometimes associated with Raynaud's disease. The attacks come on suddenly, often preceded by chills. Sometimes fever accompanies the disease. Vomiting and diarrhœa occur with hæmoglobinuria. Pain in the loins is sometimes complained of. The paroxysm may last a day or two, or two or three paroxysms may occur in the course of twenty-four hours.

Albumose (proteoses, propeptone, or Meissner's peptone). Formerly the reactions which determine the presence of the albumoses were thought to indicate the presence of peptone. The latter substance is extremely rare. Recent chemical investigations show that that which was called peptonuria is truly albumosuria. Albumose has been found in the urine in osteomalacia and diseases of the medulla of bone and in myxœdema. When persistent, it is in all probability due to multiple tumors of the bones or to myxœdema. The albumosuria may be considered as primary. Transi-

tory albumosuria is found in pneumonia, deep-seated suppuration, meningitis, and in dermatitis, intestinal ulcer, measles, scarlatina, and mental diseases. Its frequent occurrence renders its presence of not much diagnostic value. According to von Jaksch, its presence may indicate that a suppurative process exists. In the diagnosis of epidemic cerebrospinal from the tubercular meningitis, transitory albumosuria points to the former if no ulcerative tuberculous process exists elsewhere. Urine containing it does not respond, at first, to the heat and nitric acid test, but on cooling a precipitate forms which responds to the *biuret test.* (In this test the urine is first treated with about one-half its volume of sodium hydroxide solution, and then a 1 per cent. solution of cupric sulphate is added drop by drop. If albumose is present, the resulting cupric hydroxide is dissolved, and the fluid becomes of a violet-red color.) The probability of the presence of albumose is strengthened if a turbidity occurs with the acetic acid and potassium ferrocyanide test (acetic acid, specific gravity 1064, to which a few drops of a 10 per cent. solution of potassium ferrocyanide have been added), and also with the biuret test applied directly to the urine itself. Albumin also responds to this test.

The best test for albumoses is that of Hofmeister, modified by Salkowski. 20 to 50 c.c. of urine are acidified with acetic acid and then added to an equal quantity of a saturated solution of common salt, boiled and filtered. In this manner the urine is freed from albumin ; the albumin remaining as a filtrate while the albumose is redissolved. The filtered fluid containing the albumose is placed in a beaker and a few drops of HCl added. A solution of phosphotungstic acid is added and the precipitate consolidated by heat into a coherent mass. Then pour off the supernatant fluid ; wash the precipitate with water and dissolve in a solution of soda (specific gravity 1.16), which is added drop by drop until dissolved. If the solution is blue, it is to be gently heated, to decolorize. A few drops of a 1 per cent. solution of copper sulphate are added to the soda solution. If a red or violet color, the *biuret reaction,* results, albumose is present.

The late N. A. Randolph suggested the following test, which is given by Tyson : To 5 c.c. of urine, which must be cold and faintly acid, add 2 drops of a saturated solution of potassium iodide and then 3 or 4 drops of Millon's reagent. If albumoses or bile-acids are present, a yellow precipitate falls. If the yellow precipitate does not respond to the test for bile-acids, it is due to albumose.

Sugar (glucose). Next to albumin, sugar is the most important abnormal constituent of the urine. It is not present in normal urines in quantities that can be detected by ordinary clinical methods. The best methods for its detection are Fehling's test and the fermentation test.

Fehling's Test. Fehling's solution is prepared by dissolving 34.652 grammes of pure crystallized cupric sulphate in about 200 c.c. of water. About 173 grammes of sodic potassium tartrate (Rochelle salt) are dissolved in about 480 c.c. of sodium hydroxide solution of 1.14 specific gravity. The cupric sulphate solution is added slowly to the sodic potassium tartrate solution, stirring constantly until all of the cupric sulphate solution has been added. The bluish-white precipitate of cupric

hydroxide which first forms will, on stirring the liquid, be completely dissolved. The blue liquid is then diluted with water to exactly 1000 c.c. 1 c.c. of this solution will be reduced by 0.005 gramme of glucose. Fehling's solution is prone to undergo decomposition, and to avoid this, it is best to keep the cupric sulphate and sodio-potassium tartrate solutions in separate bottles closed with rubber stoppers. To accomplish this, the 34.652 grammes of cupric sulphate are dissolved in water and diluted to 500 c.c., and the sodio-potassium tartrate is dissolved in water and diluted to 500 c.c., and the two solutions preserved in separate bottles The solutions, prepared in this manner, are used by mixing one volume of the cupric sulphate solution with an equal volume of the sodio-potassium tartrate solution. The resulting liquid will be Fehling's solution, and 1 c.c. of it will be equal to 0.005 gramme of glucose.

Certain precautions are necessary in the application of this test: 1. Any albumin present must be removed by boiling and filtration. 2. The Fehling solution, diluted with 4 to 5 volumes of water, must be boiled first and the urine added to it; the urine must not be boiled first and the Fehling solution added to it. Boiling the reagent first is a test of its stability; if a precipitate occurs, the solution is unfit for use. As Wormley correctly says, a precipitate is more likely to occur when the Fehling solution has been diluted with 4 or 5 times its volume of water than on boiling the undiluted solution. 3. Prolonged boiling is to be avoided. The solution is to be heated to the boiling-point and the urine then added: if no precipitate indicating sugar occurs until urine is added almost equal in volume to that of the reagent, the mixture should be again heated to the boiling-point and set aside. 4. When the earthy phosphates are abundant, it is well to get rid of them by adding a small quantity of sodium hydroxide and filtering before applying the sugar test. 5. Changes in color may occur from the presence of urea, uric acid, and extractives. These changes can be obviated, when necessary, by the method proposed by Seegen, who recommends repeated filtering through animal charcoal until the urine is rendered colorless. Fehling's test is then applied to the filtered urine.

The method of applying Fehling's test is as follows: Fehling's solution is poured to the depth of about $\frac{1}{4}$ inch into a test-tube, and diluted with 4 or 5 times its volume of water, and heated until it begins to boil; then 1 or 2 drops of the suspected urine are added. If it be ordinary diabetic urine, the mixture after an interval of a few seconds will suddenly turn to an intense opaque-yellow or reddish-brown color, and in a short time an abundant yellow or reddish-brown precipitate falls to the bottom. If, however, the quantity of sugar present be small, the suspected urine is added more freely, but not beyond a volume equal to that of the diluted Fehling's solution employed. In this latter case it is necessary to raise the mixture once more to the boiling-point. It is then allowed to cool slowly. If no cuprous oxide is thrown down when the liquid becomes cold, then the urine may be pronounced sugar-free.

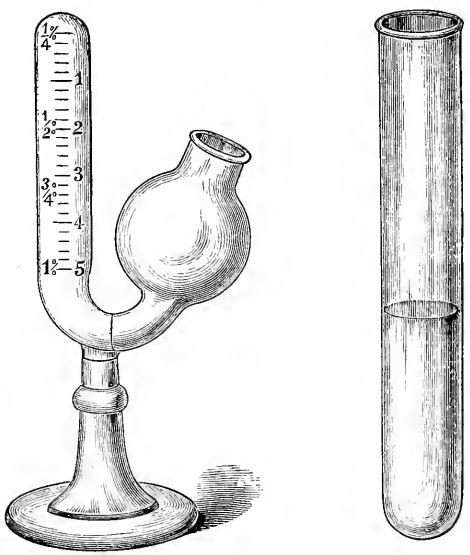
Sir William Roberts has recently pointed out the value of repeated filtration through animal charcoal of urine which reacts doubtfully to the test for sugar; by this filtration the urates, uric acid, and other normal

constituents of the urine, which have more or less power of reducing Fehling's solution, are removed, while the sugar passes through and is found in undiminished quantity in the filtrate.

The test is made as follows: A test-tube is charged with Fehling's solution to the depth of about $\frac{1}{4}$ inch, diluted with 4 or 5 times its volume of water, and brought to the boiling-point; the urine, filtered through charcoal, is added to the depth of about 2 inches, and the two fluids mixed. The flame of a lamp is then applied to the upper half of the column of liquid, and this is boiled for a couple of seconds. If sugar is present, the upper half loses its blue color and assumes a yellowish tinge, and the earthy phosphates which are thrown down in light flakes by the alkali of the test are tinted more or less of a golden color by the precipitation on them of the euprous oxide.

The Fermentation Test. This is based upon the fact that sugar by fermentation with yeast breaks up into alcohol and carbon dioxide. It is a reliable but not a very delicate test for sugar. A piece of yeast-cake the size of a pea is added to a test-tube full of urine. The open end of the tube is inverted under water in a saucer or beaker. If sugar is present in amounts larger than $2\frac{1}{2}$ grains to the ounce, bubbles of carbon dioxide collect at the upper part of the tube after standing twelve hours in a temperature of about 90° F.

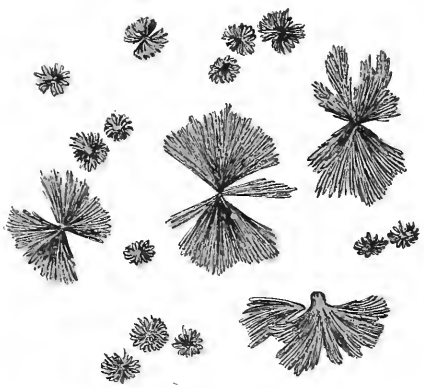
FIG. 227.



Einhorn's saccharimeter.

The Phenylhydrazin Test. Von Jaksch believes this test to be a very accurate one. About 2 grains of phenylhydrazin hydrochloride and about 3 grains of sodium acetate are put into a test-tube half-full of water. The contents of the tube are heated and the tube filled with the suspected urine. The tube is kept for fifteen or twenty minutes in boiling water,

PLATE XIII.



Crystals of Phenyl-glucosazone.

(Oc. 4, Obj. D.) Drawn by J. D. Z. Chase.

and then put in a vessel of cold water. When a large amount of sugar is present, a deposit of yellow needle-like crystals is visible to the naked eye; but when only a small amount is present, the sediment must be examined under the microscope. The crystals appear singly, or in sheaves and fine radii. Yellow plates and brown balls do not indicate sugar. (Plate XIII.)

Quantitative Estimation of Sugar. Fehling's solution may be used with a burette and measured quantities of urine and reagent. Wormley recommends a method which answers very well for office-use: 1 c.c. of Fehling's solution is diluted in a large test-tube with 4 c.c. of distilled water, and boiled. 0.1 c.c. of the suspected urine is then added from a graduated pipette. Heat is then applied, the precipitate watched, and then another 0.1 c.c. added, and heat again applied. The addition of 0.1 c.c., followed by heat, is continued until it is found after subsidence that all color is removed from the diluted Fehling's solution. If in doing this 1 c.c. of urine has been added, it will have contained just 0.5 per cent. of sugar. If more than 1 c.c., it will have contained less than 0.5 per cent. If exactly 2 c.c. are used, it will have contained exactly 0.25 per cent. If 0.1 c.c. has been used, the urine will have contained 5 per cent. of sugar. If the quantity of sugar in the urine is large, the urine should first be diluted with a measured volume of water, allowance being made for this in the estimation.

When the quantity of sugar is relatively large, fermentation is the simplest and most trustworthy method. Roberts has shown that saccharine urine loses by fermentation 1 degree in density for every grain of sugar contained in an ounce of urine. For example, if the urine before fermentation had a specific gravity of 1040, and after fermentation a specific gravity of 1010, then the urine contained 30 grains of sugar to the ounce. In the application of this method, about 4 ounces of diabetic urine are put in a 12-ounce bottle, and a piece of Vienna yeast about the size of a pea is broken up and then added to it. This bottle is closed with a perforated cork to allow the CO_2 to escape, and stood aside in a warm place to ferment. Beside it is placed a tightly corked 4-ounce bottle filled with a specimen of the same urine, but without the yeast. In about twenty-four hours the fermentation will have ceased. The specific gravity of the fermented urine is then taken and also that of the unchanged urine. Every degree of loss in density represents 1 grain of sugar per ounce of urine.

Bremer's Diabetic Urine Test. Take 2 parts of eosin and 3 parts of gentian-violet; dry, powder, and mix. Add to a test-tube of urine. In normal urine only the eosin dissolves, giving an orange-red tint to the urine; the gentian-violet remains on the surface of the urine or falls to the bottom in solid masses, giving no coloration to the eosin-stained urine. In diabetic urine eosin dissolves in a few seconds; the gentian-violet begins to fall from the surface, leaving a heavy trailing stain in the urine, which eventually becomes entirely violet. The majority of diabetic urines, especially the pale greenish shimmering ones, give the test. Occasionally the urine of chronic Bright's or of other diseases may react.

Williamson's Test. Diabetic urine to which has been added methylene-

blue in alkaline solution, will on gentle heating on a water-bath completely lose the blue color given by the stain.

Glycuronic acid occurs in urine after administration of chloral, butyl-chloral, nitrobenzole, camphor, curare, morphine, chloroform, fusel oil; reduces Fehling's solution, is dextrorotatory, and does not ferment. Many temporary glycosurias are probably due to this substance.

β-Hydroxybutyric, oxybutyric acid, in severe diabetic cases, usually with diacetic acid and acetone, has strong lævorotatory action, and may interfere with the polariscope test. If urine is strongly lævorotatory after fermentation, this substance is probably present.

Indican. An excess of indican in the urine is known as indicanuria. The substance is detected by several methods. Jaffe's test: Equal volumes of hydrochloric acid and urine are mixed. By means of a glass pipette a solution of sodium hypochlorite is dropped into the fluid. An indigo-blue color is produced if indican be present. The hypochlorite must not be added in excess. A quantitative determination is made by the colorimetric process of Salkowski. A rough analysis is first made to determine the quantity of calcium hypochlorite which causes the greatest amount of indigo to unite with it. If the urine contains much indican, a small portion, as 2.5 to 5 c.c., is diluted with water to 10 c.c.; if there is but little indican, 10 c.c. of the urine are used without dilution. An equal quantity of hydrochloric acid is added. To this the amount of hypochlorite solution with which, in the first test, indigo combined in the greatest amount is added. Then the liquid is neutralized with sodium hydroxide; and sufficient sodium carbonate is added to make it alkaline. The indigo-blue is thus precipitated and collected on a filter. The precipitate is repeatedly washed with water until the alkaline reaction disappears. The filtrate is dried and extracted by heating with chloroform until the latter no longer assumes a blue color. The chloroform extract is increased to a round number of cubic centimetres by the addition of chloroform, and placed in a vessel with parallel sides. The intensity of its color is compared with that of a freshly prepared chloroform solution of indigo-blue of known strength. To one or other of these, chloroform is added until the tint of both is the same. The quantity of indigo-blue derived from the urine is determined, and its percentage calculated from the intensity of color and strength of the solution of indigo of known strength. In health 5 to 20 mgm. of indigo-blue are passed in twenty-four hours.

Indican is increased by animal diet—an increase which, under other circumstances, is pathological. Its presence is a sign of intestinal putrefaction. It may accompany decomposition of albumin in cavities. It is present in empyema and in puerperal peritonitis. By detection of its presence in these diseases cavities due to pus may be distinguished from those due to other causes. Indican is increased in acute diarrhœa and in intestinal tuberculosis. Von Jaksch states that large quantities of indican in the urine imply that abundant albuminous putrefaction or putrid suppuration is in progress in the system. It must not be forgotten that indicanuria will often arise in simple constipation.

Bile-pigments and Bile-acids. Bile-pigment, or bilirubin, occurs in

the urine in cases of hepatogenic and hæmatogenic jaundice, and in portal thrombosis.

Gmelin's test and its modifications are the ones usually employed: a small quantity of nitric acid, to which some nitrous acid has been added, is put in a test-tube and then gently overlaid with urine: if bile-pigment is present, a series of colors appear at the junction of the two fluids—green, blue, violet, and yellow. A green color (biliverdin) must be present to prove the existence of bile-pigment.

The same test may be applied by placing a few drops of the acid upon one side of a plate and the urine on the other, and then allowing the two to run together: the play of colors takes place, as before, at the line of junction of the acids and urine.

Rosenbach's modification is an improvement: about 200 c.c. of urine are allowed to flow through pure white filter-paper, and then a drop of nitric acid is placed upon the paper saturated with the urine: the colors appear as before described.

A very simple test consists in allowing a few drops of nitric acid to fall into a test-tube full of urine: if bile-pigment is present, a green color appears at the line of junction of the two fluids. This test may fail, however, if only small quantities of bile-pigment are present.

The tests for bile-acids are either too elaborate or too unsatisfactory for clinical use.

Pus. Pus is found in the urine whenever there is suppuration or a catarrhal condition of the genito-urinary tract. Hence it occurs in abscess of the kidney, pyonephrosis, pyelitis, tuberculosis, cystitis, gonorrhœa, leucorrhœa, etc. It is relatively common in women, from a catarrhal condition of the vulva and vaginal mucous membrane, and is therefore of less significance than in men. Urine containing much pus is slightly albuminous; but frequently pus-cells are found in urine which gives no reaction for albumin.

The chemical test for pus is its conversion into a tenacious (gelatinous), glairy mass by boiling with caustic potash.

Acetonuria. An excess of acetone occurs in the following diseases: (1) in diabetes; (2) in cancer independent of starvation; (3) in starvation; (4) in certain psychoses; (5) in auto-intoxications; (6) in derangement of digestion; (7) in fevers. In diabetes acetone indicates an advanced stage of the disease. Lieben's test for acetone is as follows: to several cubic centimetres of distilled urine a few drops of iodo-potassic iodide solution and sodium hydroxide are added: if acetone is in excess, precipitation of iodoform takes place, which may be recognized by its odor.

Diaceturia. Diacetic acid is found in the urine in diabetes, in fevers, and in auto-intoxications. It is common with children in fever. It is of grave significance in the urine of adults. Coma usually follows its occurrence in the urine in fevers and in diabetes. Test: a concentrated solution of ferric chloride is cautiously added to the urine: if a precipitate be formed, it should be removed by filtration and more chloride added to the filtrate. If diacetic acid be present, the liquid will become claret-red in color.

Hæmatoporphyrinuria. This is a rare constituent of the urine derived from the blood. It is said to be a form of hæmatin freed from iron. Nakarai thinks that the occurrence of hæmatoporphyrinuria is constant in lead-poisoning, and occurs with some degree of frequency in intestinal hemorrhage.

Alkaptonuria. The substance in the urine which has been identified as alkapton is also known as pyrocatechin,[1] protocatechuic acid,[2] urrhodinic acid,[3] glycosuric acid,[4] uroleucinic and unoxanthinic acids,[5] and homogentisinic acid.[6] It reduces copper, as does glucose, and its occurrence is of interest because the presence of the substance has led to the diagnosis of glycosuria in many instances, in consequence of which persons have been refused life insurance. The urine containing this substance deepens in color on exposure to air. It is of a peculiar aromatic odor, and reduces cupric salts rapidly. There is, however, no reaction to the fermentation test, to Böttger's bismuth test, or to phenylhydrazin, and no deviation of polarized light. The urine does not contain bile-pigment. It is of normal specific gravity, and becomes very dark on the addition of an alkali, or of a temporarily bluish-green color with ferric chloride. Ammonio-nitrite of silver is instantaneously reduced when added to the urine, with a deposit of metallic silver.

Alkaptonuria is usually congenital. Several members of the same family may have it. No symptoms attend the condition.

NOTE.—*Serum-globulin* is converted into a coagulated proteid when heat is applied or concentrated nitric acid added to a solution. Globulin is soluble in dilute salt solutions. If urine rich in globulin is added drop by drop to a large volume of distilled water, the globulin is precipitated as the percentage of salt is reduced by dilution. Globulin is also precipitated by dialysis. If a portion of the urine containing globulin is saturated with magnesium sulphate or half-saturated with ammonium sulphate, globulin is precipitated.

Hills[7] describes the method as follows: "25 to 50 c.c. of the urine are made neutral or slightly alkaline with ammonium hydroxide, and the precipitated phosphates removed by filtration. An equal volume of a saturated solution of ammonium sulphate is then added, the mixture shaken and allowed to stand for some time, and finally filtered. The precipitate is washed with a half-saturated solution of ammonium sulphate for the removal of the last traces of albumin, and the filtrate and precipitate tested for albumin and globulin respectively, as previously described. The formation of a precipitate upon the addition of either magnesium or ammoninm sulphate is not in itself evidence of the presence of globulin.

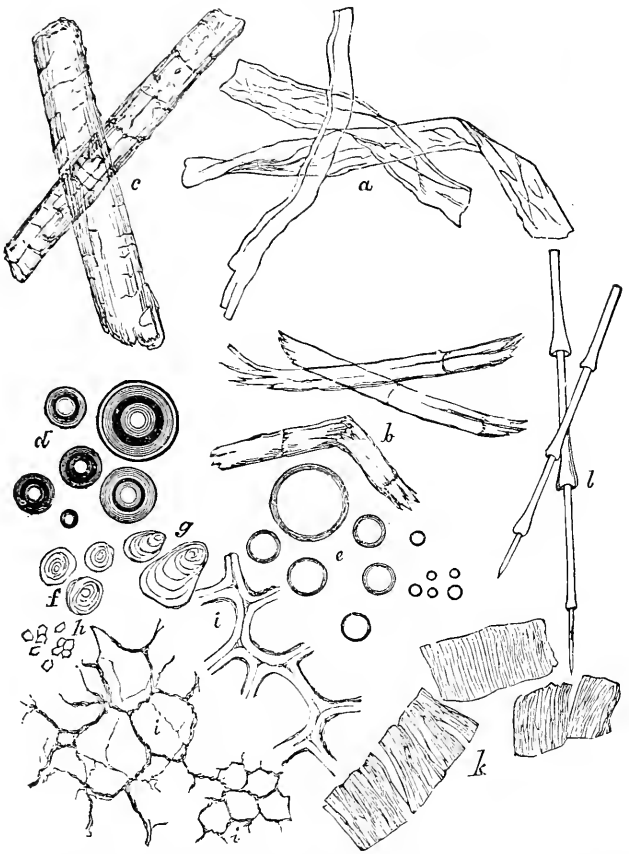
[1] Ebstein and Müller, Virchow's Archiv, Bd. lxv. S. 394.
[2] Smith, Dublin Journal of Medical Science, 1882, vol. i. p. 465.
[3] Kirk, British Medical Journal, London, 1886, vol. ii. p. 1017.
[4] Marshall, Medical News, Philadelphia, 1887, p. 35.
[5] Kirk, British Medical Journal, London, 1888, vol. ii. p. 232.
[6] Baumann and Wolkow, Zeitschr. f. physiol. Chem., Strassburg, Bd. xv. S. 228.
[7] Boston Medical and Surgical Journal, 1899, vol. cxli. No. 6.

Microscopical Examination of the Urine.

Microscopical examination of the urine is chiefly concerned with the sediments, and these are conveniently divided into the organized and unorganized.

The *organized deposits* in the urine are blood, pus, mucus, epithelium, casts, spermatozoa, micro-organisms, cancerous and tuberculous matter, and entozoa.

FIG. 228.



Extraneous matters found in urine: *a*, cotton-fibres; *b*, flax-fibres; *c*, hairs; *d*, air-bubbles; *e*, oil-globules; *f*, wheat-starch; *g*, potato-starch; *h*, rice-starch granules; *i*, *i*, *i*, vegetable tissue; *k*, muscular tissue; *l*, feathers.

The *unorganized deposits* are uric acid and its compounds, oxalate and carbonate of lime, phosphates, leucin and tyrosin, cystin, and cholesterin.

Normal urine forms a slight sediment, consisting of epithelium from different parts of the genito-urinary tract, principally from the bladder in males and from the vagina and bladder in females. There are also crystals of the different urinary salts, sometimes mucus, and a few white blood-cells; and if the urine has stood a while, especially if it is alkaline,

more or less bacteria. It may accidentally contain extraneous matters derived from the vessel which contains it or from the air. (Fig. 228.)

The centrifugal machine has now become an important adjunct to the rapid and accurate microscopical examination of the urine. There are numerous varieties to be secured at the instrument-stores, some of which are devised solely for urinary examination, while others have additional apparatus for examination of the blood and sputum. The majority of them are revolved by hand. Electricity can be readily applied to any of them and labor be saved by such a device. The advantages of centrifugal force over the older gravity method employed in microscopical examination are marked. Some few of them can be briefly outlined:

1. Centrifugalization secures complete, rapid, and concentrated sedimentation. It is therefore best suited to microscopical diagnosis.

2. Casts or other organic material, if present, can be studied carefully before they are macerated or partially destroyed by bacteria or changed by the deposition of amorphous or crystalline material. This is a most important aid to correct diagnosis.

3. Crystals, if present at the time of urination, can be discovered and differentiated from those that normally crystallize out after some hours.

Certain bodies—hyaline casts, for instance—because of their rather light specific gravity, do not settle on simple standing of the urine, and thus escape detection. These with all other substances are thrown down with the centrifugal machine.

5. Bacteria are discovered with greater ease, especially the tubercle bacillus.

The method commonly used for the examination of the urinary sediment is as follows: The urine for examination (the chemical analysis having previously been made) is decanted until there remains but a small amount in the bottle, which amount contains any sediment already formed and heavier organic materials. This is then poured into one of the tubes of the centrifugal machine to within one-half inch of the top; if but one specimen of urine is to be examined, fill both tubes with specimens from the same urine. If there is not sufficient urine to do this, fill the remaining tube or tubes with water. It is well to mark the external metal shields of the tubes with a figure, say 1 and 2, or *a* and *b*, so that the urines, if different specimens, may not become confused. The tubes are then rapidly revolved for three minutes, removed from the machine and a few drops of the sediment withdrawn with a pipette and placed upon the slide for examination under the microscope. It is necessary to remember that care must be exercised in removing this sediment from the tube. The straight glass pipette without a pointed end seems to give the best results in securing the sediment. The finger is placed upon one end, the pipette inserted to the bottom of the tube, and the finger elevated just enough to secure a few drops of the sediment that has been cast down by centrifugalization. The same object is attained if the finger is entirely removed as soon as the point of the tube reaches the bottom of the conical glass; but in that case more than the lowest layers of the sediment are sucked up, and hence all but a few drops should be allowed

to flow out when the tube is removed from the urine. In this way the drops reserved for the microscopical examination will contain the sediment from the very bottom of the glass. In this sediment, in pale urines free from much urates, phosphates, and pus, the casts will be found, if any are present in the urine. It is most important to examine the bottom layers of the sediment when the latter is scanty, or when phosphates or urates have begun to precipitate after the urine has been standing some time. If the urine is already cloudy with phosphates, urates, or pus, when it is put aside to settle, any casts that may be present will be carried down with the heavier sediment, and will be found intimately mixed with it, or even on top of the other sediment. The few drops reserved for microscopical examination are now deposited on several slides without a cover-glass, and examined carefully for organic and inorganic constituents.

If the urine contains but the normal amount of mucous cloud, a very small whitish sediment or cloud is found at the bottom of the tube. If calcium oxalate is present, a small filmy, whitish sediment is seen. The sediment of amorphous urates is pinkish, fawn, or salmon color. Uric acid appears as a "brick-dust" sediment. Pus produces a heavy yellowish sediment; phosphates a heavy white sediment, which is sometimes yellowish white from admixture with leucocytes. Blood in small quantities produces a rather characteristic brownish deposit. Large amounts of blood appear as reddish coagula at the bottom of the tube.

With some of the centrifugal machines the various urinary salts and the amount of albumin present can readily be estimated. Such instruments are provided with graduated tubes, into which the urine and the necessary reagents are put and the resulting precipitate rapidly cast down.

In this manner Purdy estimates the chlorides, sulphates, and phosphates, and also the amount of albumin most satisfactorily. It is questionable, however, whether the estimation of the salts is accurate.

If the centrifugal machine can not be employed, proceed as follows: 6 or 8 ounces of the urine to be examined should be allowed to settle in a bottle as soon after being passed as possible. The bottle should be tightly corked, because urine exposed to the air decomposes very quickly; it should be sent to the laboratory as soon as possible after being passed, in order that an examination may be made before fermentative changes spoil it for trustworthy analysis. After standing twelve, or preferably twenty-four hours, nearly all of the solid matter will have collected at the bottom of the bottle. The supernatant clear fluid can now be poured off, and the lower portion of the urine and the sediment poured into a conical subsiding-glass. If the urine is febrile, there may be by this time a large deposit of amorphous urates, which will obscure the search for casts; they may be dissolved by gentle heating without destroying the casts, and the clear urine again allowed to settle for a few hours. So, too, if phosphates are abundant, they should be gotten rid of by gentle heating, and by acidulation with 2 or 3 drops of dilute acetic acid. After the urine in the conical subsiding-glass, which will now amount to an ounce or two, has stood for a few hours, any casts that may be present will have fallen to the bottom. If the urine is highly concentrated (1030

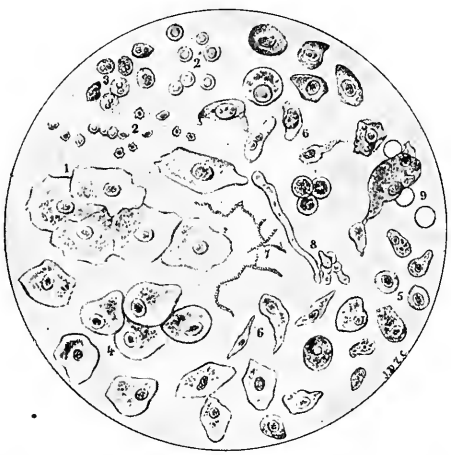
or more), epithelium, blood, and casts will be suspended longer; hence it may be well to dilute the urine before allowing it to settle. A specimen of the sediment is then transferred to a slide for microscopical examination, as already described.

All the pipettes used in examining urine must be kept clean. They should be allowed to stand in water which is frequently changed, and carefully rinsed in running water before being used.

Organized Sediments.

Blood. If the blood comes from the kidney, it is usually intimately mixed with the urine, which remains of a red or reddish-brown color, and contains possibly tube-casts and renal epithelium. The blood-cells appear singly, have frequently lost their hæmoglobin, and hence look like pale-yellow disks. (See Fig. 229.)

Fig. 229.



Cellular elements from the urine: 1, squamous epithelium; 2, red blood-corpuscles; 3, polynuclear leucocytes; 4, transitional cells; 5, epithelium from the kidneys; 6, epithelium from the bladder; 7, *Micrococcus aureæ;* 8, yeast-fungi.

Sometimes blood coagulates in the ureters, and long, cylindrical plugs are passed, causing symptoms resembling those of renal colic. When blood comes from the bladder or neck of the bladder (fissure), there are frequent micturition, acute pain and tenesmus, and the blood is not intimately mixed with the urine. When the hemorrhage is from the neck of the bladder, it often occurs as a few drops of blood at the end of micturition, accompanied with great pain and a sense of faintness. Intermittent hæmaturia, according to von Jaksch, points directly to calculus or tumor of the bladder.

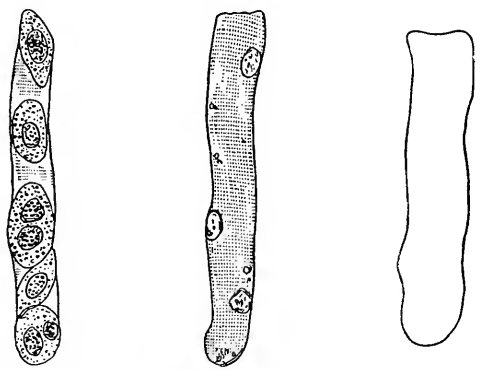
Blood-cells when unaltered are unmistakable on account of their well-known biconcave appearance. When they have lost their coloring-matter they appear as circular, very pale disks, with extremely faint outline and feeble refractive power. Absence of a nucleus serves to distinguish them

from yeast-spores, and the latter, moreover, are often oval in shape. They are less likely to be confounded with the ovoid and circular shapes of calcium oxalate crystals, because the latter are not common, and can be seen usually in their more common forms as octahedra and dumb-bells in the same urine.

Pus. The sources of pus in the urine have been mentioned. The pus-corpuscle is an opaque, spherical, granular cell, usually somewhat larger than blood-cells. In concentrated urines the pus-cell is small; in dilute urine, or urine to which water has been added, it swells sometimes to twice its original size. At the same time it becomes less granular, and two, three, or four nuclei may appear. The addition of acetic acid also causes it to swell, and brings out the nuclei more distinctly and rapidly. Sometimes the pus-cells are discrete, sometimes in dense clumps, and sometimes nothing but a dense mass of pus-cells appears in the field of the microscope.

The source of the leucocytes must be a matter of inference from the general characters of the urine. If red blood-cells are also present, the probability of finding white blood-cells is increased, but pus-cells are not necessarily excluded. So, too, if much mucus be present in the urine, the doubtful cell may be a mucus-corpuscle. Some clue to the source of the pus can be obtained from the urine itself. Urine containing pus from the kidney is usually acid, whereas in cystitis it is alkaline, and almost always contains phosphates, mucus, and abundant bacteria. An exception to this is found in the cystitis of certain general infections. Thus typhoid cystitis and cystitis due to *Bacillus coli communis* are regularly associated with acid urine. Again, pus from the kidney or kidney pelvis is apt to vary greatly in amounts or be discharged intermittently; and the urine when filtered free from pus-cells is usually still albuminous. Renal epithelium and casts may also be found.

FIG. 230.



Epithelial and hyaline casts.

Casts. Casts are the most important of the urinary deposits. They vary greatly in number and size.

Casts may be numerous, so that nearly every field contains a dozen or more; or they may be very few, not more than one or two being found

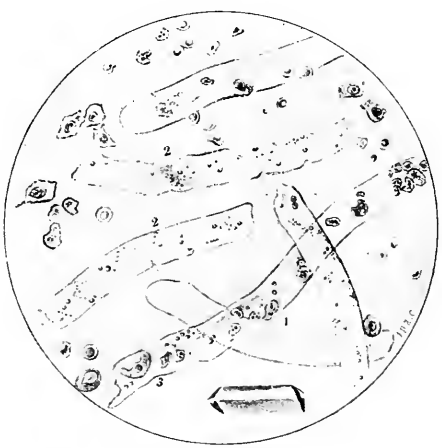
on a slide. The best routine method for microscopical examination is as follows: place a few drops of the urinary sediment upon the slide; spread the drops in a thin layer; use no cover-glass; *examine with the low power* —a diameter of 50—with a small amount of light; the whole slide can

FIG. 231.

Hyaline casts and cylindroids in hypostatic congestion of kidney. Low power.

be carefully searched in three minutes, and the casts discovered can be minutely studied with the higher power. When but few casts are present, several slides can be rapidly examined with the low power, and an accurate estimation of the number made.

FIG. 232.



Hyaline casts from a case of acute nephritis: 1, plain hyaline cast; 2, granular deposit on hyaline cast; 3, cellular deposit (blood and epithelium).

Tube-casts usually indicate acute or chronic nephritis; but they are *sometimes* found in cases of renal calculi; in icterus, usually without

PLATE XIII-a.

FIG. 1.

1. Hyaline Casts with Granular Matter and Epithelial Cells deposited upon them. 2. Amyloid (waxy) Cast.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

FIG. 2.

Blood-Casts from Case of Acute Nephritis.

(Oc. 4, Ob. D.) Drawn by J. D. Z. Chase.

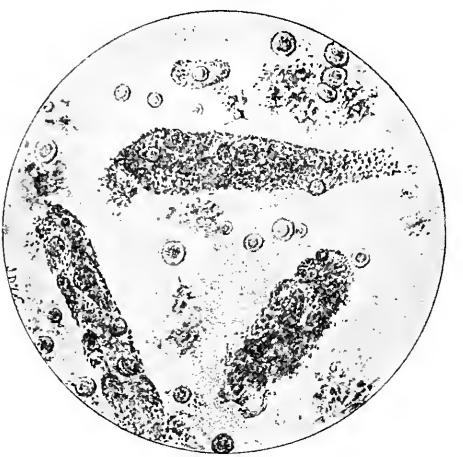
albuminuria; in diabetes, and sometimes in secondary congestion of the kidney.

Several varieties of casts are found.

1. **Hyaline casts,** as their name implies, are clear, translucent bodies, which refract light so slightly that they are easily overlooked. They have well-defined margins, the ends being frequently rounded; they are rarely very long, and are straight or but slightly bent. They are rarely equally translucent throughout; at some part more or less granulation will generally be found. They vary in diameter from that of a white blood-cell to 6 or 8 times as large. They can be stained, and so rendered more distinct, by allowing a drop of gentian-violet solution to flow in under the edge of the cover-glass. (Figs. 231 and 232.)

2. **Granular casts** are hyaline casts which appear granular either from some deposit on their surface or from a granular change of the cast itself.

FIG. 233.



Granular casts.

When the granulation does not interfere with the translucency, the casts are described as "pale" or "slightly" granular; and when they become very dark, so as to resemble closely a blood-cast, they are called "dark" or "opaque" granular casts. (Plate XIII.–A, Figs. 1, 1; and Fig. 233.)

3. **Waxy casts** appear to the eye to be more solid in structure than the hyaline casts; they also appear more cylindrical in form, are more or less yellow in color, and are apt to be larger than hyaline casts. (Plate XIII.–A, Figs. 1, 2.)

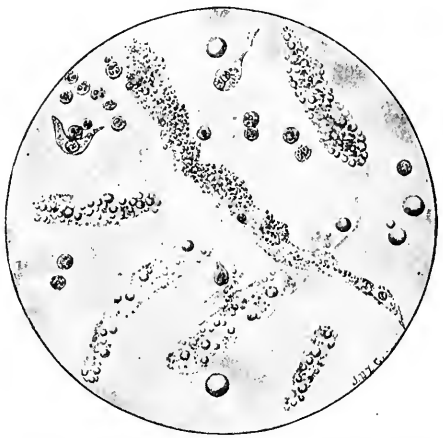
4. **Fatty casts** are hyaline or faintly granular casts on which are deposited, in spots, minute oil-drops. If the oil-drops are very abundant, they are sometimes called "oil-casts." (Fig. 234.)

5. **Blood-casts** are either made up of a mass of blood-cells pressed into a cylindrical shape, or more frequently they are hyaline casts studded with blood-cells. (Plate XIII.–A, Fig. 2.)

6. **Epithelial casts** sometimes seem to be composed entirely of epithelial

cells closely packed. Such casts are relatively rare, and very beautiful. Ordinarily, just as in the case of blood-casts, an epithelial cast consists of a hyaline cast more or less covered with renal epithelium. (Plate XIII.–A, Figs. 1, 1 ; and Fig. 230.)

Fig. 234.



Fatty casts from a case of chronic parenchymatous nephritis.

7. **Pus-casts** are usually described as more or less regularly occurring in purulent kidney affections.

8. **Cylindroids** are very common. In general appearance they resemble hyaline casts ; but they are apt to be much longer, bent, twisted, or split, and to have a striated or finely ribbed appearance on close examination.

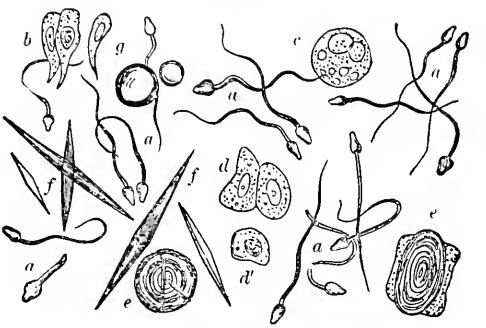
Fig. 235.

Cylindroids.

Moreover, the diameter of the cast frequently varies greatly at different points ; sometimes it appears constricted in several places, and in other cases one end tapers into a thread. Often cylindroids consist of fine, narrow, ribbon-like threads. (Figs. 231 and 235.)

Spermatozoa. Spermatozoa are easily recognized by their tadpole shape and by the vibratile motion of their long, delicate tails. They are found in the urine of both sexes after sexual intercourse. (Figs. 236 and 237.)

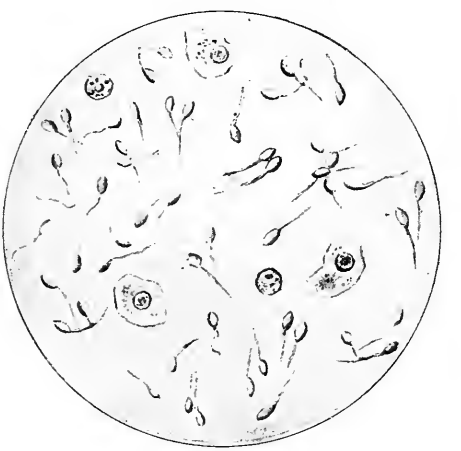
Fig. 236.



Human semen: *a*, spermatozoa; *b*, cylindrical epithelium; *c*, bodies enclosing lecithin granules; *d*, squamous epithelium from the urethra; *d'*, testicle-cells; *e*, amyloid corpuscles; *f*, spermatic crystals; *g*, hyaline globules. (Von Jaksch.)

Many continent men have occasionally nocturnal emissions, accompanied by erections and erotic sensations. These can not be looked upon as abnormal, and they are compatible with robust health. There are other persons, neurotic, anæmic, and generally constipated in habit, who have emissions at night two or three times a week, of which they are

Fig. 237.



Spermatozoa from urine. (Original.)

unconscious until they awake and find themselves wet. Semen may also be lost during micturition and defecation, especially when much straining is required. Such a condition (spermatorrhœa) is abnormal. It is due to general nervous and muscular relaxation, associated with nervous dyspepsia and anæmia, and aggravated by sedentary life, constipation, and the reading of salacious literature or the cultivation of erotic thoughts.

In young men, it sometimes follows habits of masturbation, which have been broken up but have left behind a hyperæsthetic condition of the prostatic portion of the urethra, with or without dilatation of the orifices of the ejaculatory ducts; or a stricture of gonorrhœal origin may be its cause. Students and over-worked and over-strained business and professional men are the ones most frequently affected.

However caused, the condition is apt to beget a most distressing state of despondency, in which the patient imagines all possible ills, and is liable to drift into a hysterical, melancholic, even suicidal frame of mind, and so falls a victim to quacks.

Epithelium. Epithelium from the kidney, bladder, and genito-urinary passages occurs in the urine. Epithelial deposits in male urine are very scanty unless there is some disease of the kidney or bladder, or a catarrhal condition of the prostatic urethra, such as is left from an old gonorrhœa. On the other hand, considerable epithelium may be normally present in the urine of women, being derived principally from the vagina and bladder.

Vaginal epithelium consists of large flat pavement-cells, and is readily distinguished.

The type of epithelium of the kidney, kidney pelvis, ureter, and bladder is the same, and it is not possible to distinguish with certainty the cells which come from each. If the cells are scanty, von Jaksch thinks they come from the ureter. He has found them in moderate quantities and superimposed upon one another.

Renal cells closely resemble the oval polygonal cells from the deeper layers of the bladder, but they have a relatively larger nucleus. (See Fig. 229.)

Fat. Oil is found in the urine in fatty degeneration of the kidney and its epithelium, and occasionally in the urine of those who are taking cod-liver oil, and in calculous diseases of the pancreas. Tyson suggests that it may come from cystic cheesy degeneration of the kidney. It is also found in chronic nephritis, in phosphorus-poisoning, and in diabetes mellitus, as well as in chyluria. The urine is turbid, but clears when agitated with ether. The fat may be separated by a sedimentator, and can be recognized by its refractive properties.

Staining for Fat. Reeder recommends Sudan-3 for staining human secretions and excretions to determine the presence of fat. Large fat-droplets take a bright-red, and small droplets a yellow or orange color. Fat can thus be demonstrated in the blood in lipæmia, lipuria, and chyluria. By this method fat can be demonstrated in the stomach contents and in the feces of adults with jaundice. A saturated solution of Sudan-3 in 96 per cent. alcohol is employed. Equal parts of this solution and 96 per cent. alcohol are added to the urine. In urinary sediments the fat-droplets in casts stain a scarlet red.

Chyle. Chyluria is a more or less milky condition of the urine, due to the presence of fat, which probably gains entrance to some part of the urinary tract by rupture of the lymphatic vessels. A case has been reported by Saundby in which a young unmarried girl being pregnant, compressed her abdomen so much, in order to conceal her condition, that

œdema of the legs, thighs, vulva, and lower parts of the abdomen resulted. After confinement the urine became milky, and remained so far many days. It contained fatty matters and cholesterin, but no albumin or sugar.

Fat and albumin appear at the same time in *parasitic chyluria*, which is due to *Filaria sanguinis hominis*, whose embryos obstruct the lymphatics. They recur at long intervals. Red and white blood-corpuscles are also found in small amounts. The urine coagulates on standing or gelatinizes.

Entozoa. The most common is the *echinococcus* or *hydatid*. When this infects the kidney or urinary vessels, hooklets and even cysts have been passed in the urine. The disease is extremely rare in this country.

Filaria sanguinis hominis, which causes parasitic chyluria, is occasionally found in the urine. (See Filaria.)

Bilharzia hæmatobia sometimes lodges in the urinary tract and causes hæmaturia. It is peculiar to Egypt.

Distoma hæmatobium. Common in Egypt and Abyssinia. Eggs collect in great masses in the urinary passages, and lead to inflammation, ulcers, stenosis, etc. Eggs found in the urine alone make the diagnosis possible.

Strongylus Gigas. Very rare. Symptoms of pyelitis. (The parasite is of the size of an earth-worm.)

Intestinal worms may creep into the bladder through fistulous or other openings, and be discharged through the urethra.

Micro-organisms. Normal urine contains no micro-organisms at the time it is voided. As the result of exposure to air, however, they may develop in great abundance. The non-pathogenic organisms found are classed as mould-fungi (hyphomycetes), yeast-fungi (blastomycetes), and fission-fungi (schizomycetes).

Mould-fungi, according to von Jaksch, are found rarely in foul normal urine. Yeast-fungi are also rare in normal urine. Fission-fungi are found in urine which is undergoing ammoniacal decomposition.

Sarcinæ, usually smaller than those of the stomach, are occasionally met with, especially, according to Roberts, when there is some disorder of the urinary organs, renal pains, painful micturition, cystitis, etc.

Fig. 238.

Vibriones in urine. (Roberts.)

Under the name *bacteriuria*, Roberts and others have described cases in which the urine contained bacteria at the time of being voided. He makes four groups: (1) cases in which the presence of bacteria is associated with incipient putrefactive changes in the urine; (2) cases associated with ammoniacal fermentation of the urine; (3) cases in which common forms of bacteria are present without decomposition of the urine; and (4) cases in which micrococcus-chains are voided in the urine.

The pathogenic organisms which are more or less closely associated with infectious diseases, as typhoid fever, septic processes, and tuberculosis, are found at times in the urine, and can be demonstrated by the proper staining-methods or cultural properties.

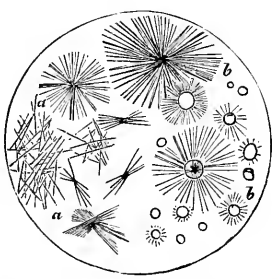
Dock has given an admirable account of the occurrence of the trichomonas in the genito-urinary passages. This parasite belongs to the flagellate infusoria. The prominent symptoms caused in Dock's case were painful, difficult, and frequent urination, followed by hæmaturia. The urine contained pus, epithelium of all kinds, and a number of bodies slightly larger than pus-corpuscles of a peculiar amyloid appearance—the trichomonades.

Morbid Growths. The urine very rarely contains the elements of morbid growths. Von Jaksch says he never has found the urinary findings in any way reliable in the case of tumors of the kidney. The detection of cancer cells or pigmented cells, such as occur in melanotic cancers, may confirm the diagnosis if the clinical symptoms point to cancer. Tumor-elements are most likely to be found in ulcerating tumor of the bladder.

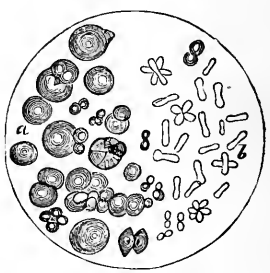
Unorganized Sediments.

Uric Acid. Uric acid is present in small quantities (8 to 10 grains a day) in normal urine. It is *increased* in febrile and wasting diseases, such as phthisis; in diseases of the liver and spleen (leukæmia), and in malarial fever, diabetes, scurvy, rhachitis, and following an attack of gout. Excessive use of milk is said to increase it. Its excretion is also increased by certain drugs—colchicum, corrosive sublimate, salicylic acid, and euonymin. It is *diminished* in anæmia, chlorosis, during a paroxysm of gout; in chronic nephritis; and by the use of certain drugs—large doses of quinine (Ranke), caffein, sodium chloride, sodium carbonate, lithia, and potassium iodide. (Plate XIII.–B, Figs. 1 and 2.)

Fig. 239.



Sodium urate: *a*, *a*, from a gouty concretion; *b*, *b*, artificially prepared by adding liquor sodæ to the amorphous urate deposit. (Roberts.)



Ammonium urate spontaneously deposited: *a*, spheres and globular masses; *b*, dumb-bells, crosses, rosettes. (Roberts.)

According to Roberts, a deposit of uric acid occurring twelve to twenty-four hours after the urine has been passed has no pathological significance. If the deposit occurs within three or four hours after the urine has been passed, it is certainly not natural. It is frequently

PLATE XIII-b.

FIG. 1.

Uric Acid.

A. Common forms. *B*. Amorphous urates.

(Ob. D. and A., Oc. 4.) Drawn by J. D Z. Chase.

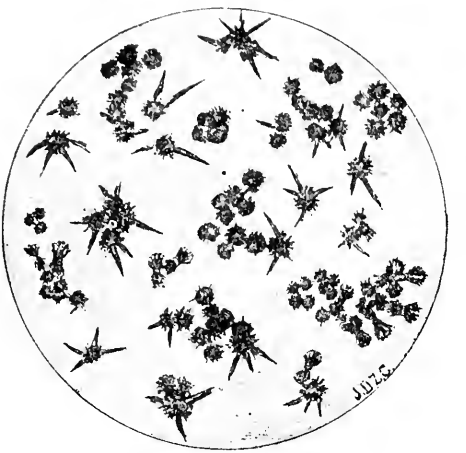
FIG. 2.

Combination of Uric Acid and Calcium Oxalate.

(Oc. 4, Ob. D) Drawn by J. D. Z. Chase.

observed in convalescence from febrile complaints, especially articular rheumatism; also in the middle periods of chronic Bright's disease, in chorea, in certain types of diabetes, and in enlargement of the spleen.

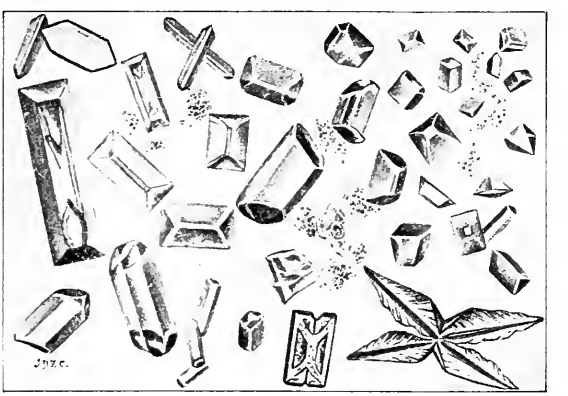
FIG. 240.



Ammonium urate. (Original.)

If, however, the uric acid is precipitated before the urine cools, or immediately afterward, it is possible that the same precipitation may occur within some part of the urinary channels, and so form a calculus.

FIG. 241.



Triple phosphates. (Original.)

Urates. Amorphous urates appear under the microscope as opaque granular particles, which dissolve upon heating, and respond to the murexide test. The deposit is more or less dense, and is sometimes arranged so as to resemble granular casts.

Sodium urate appears as spherules or globules, from which project short spines either straight or curved. It occurs most frequently in concen-

trated acid urines, such as are passed by children with acute febrile diseases. (Fig. 239.)

Ammonium urate resembles sodium urate. It is frequently associated with phosphatic deposits, and is precipitated from alkaline urines. Sometimes it appears in the shape of dumb-bells. (Figs. 239 and 240.)

Phosphates. Phosphates appear in the urine as ammonio-magnesium phosphate and as the crystalline and amorphous calcium phosphate. They are precipitated in alkaline or faintly acid urines, which produce a cloud upon being heated; the cloud is distinguished from albumin, as already pointed out, by the fact that it disappears when the urine is acidulated with acetic or nitric acid. Ammonio-magnesium phosphate is easily recognized by its rhombic prisms—"coffin-lid" shape. Other shapes are produced by modification of the primary one, chiefly by bevelling of the edges and hollowing out of the sides. These crystals are usually large, and are frequently found, together with amorphous phosphates, bladder epithelium, and pus, in cases of cystitis.

FIG. 242.

Calcium phosphate crystals. (Original.)

Amorphous phosphate of lime consists of fine granular particles much resembling amorphous urates, but distinguished from them by not disappearing upon the application of heat, but instantly dissolving when the urine is acidulated.

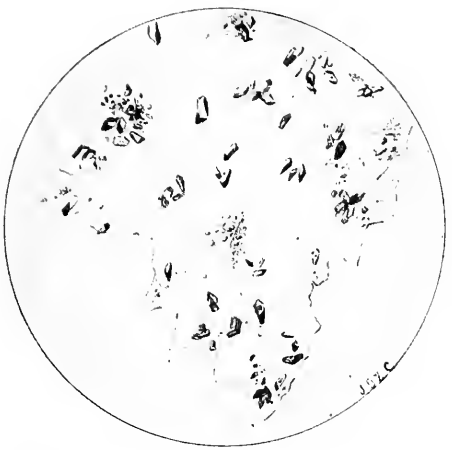
Crystalline phosphate of lime is a not infrequent deposit. It is found as narrow-wedged crystals, occasionally grouped in the form of stars, sheaves, or bundles, with their apices at a common centre.

According to Roberts, this deposit in quantity is an accompaniment of some grave disorder. He has found the stellar phosphates in cancer of the pylorus, once in phthisis, and more than once in patients exhausted by obstinate rheumatism. It may, however, occur in health when the urine is rich in lime and its acidity greatly reduced.

In one or two cases of renal colic the writer has observed numerous shining particles, which upon microscopical examination have been

shown to be an opalescent film covered with small, sharp phosphatic (probably calcium) crystals. (Fig. 243.)

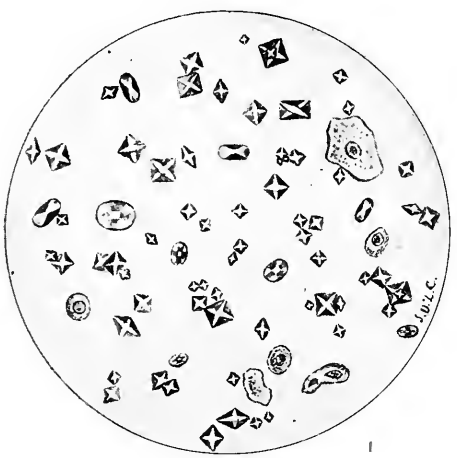
Fig. 243.



Opalescent film in a case of renal colic. (Original.)

Oxalate of Lime. Oxalate of lime occurs in the form of small octahedral crystals, or more rarely as dumb-bells, and in the form of ovals or disks. It is precipitated almost always from acid urines. (Plate XIII.–B, Fig. 2; and Fig. 244.)

Fig. 244.



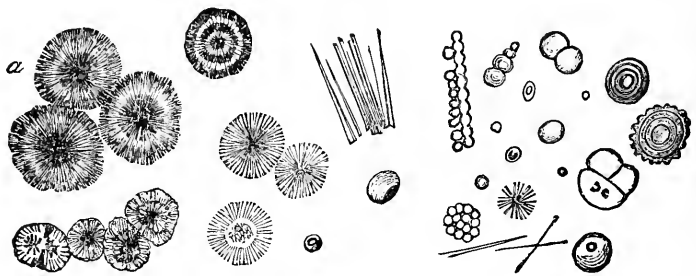
Calcium oxalate.

Oxaluria. According to Beneke, oxaluria has its proximate cause in an impeded metamorphosis, an insufficient activity of that stage which changes oxalic acid into carbonic acid.

When oxalates are constantly found in the urine, a condition of profound hypochondriasis is found to exist, but it has no necessary relation

to the oxaluria. An increase of oxalates in the urine is found in diabetes, especially when there is diminution in the amount of sugar. They are in excess in certain forms of indigestion. Their constant passage may be attended by pains in the back and loins. Flatulent and nervous dyspepsia usually accompany the increase, and neurasthenia also may be present.

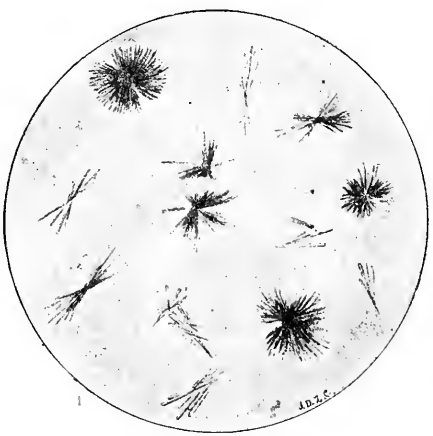
FIG. 245.



Crystals of leucin (different forms). (Crystals of creatinin chloride of zinc resemble the leucin crystals depicted at *a*.) The crystals figured toward the right consist of comparatively impure leucin. (From CHARLES: *Chemistry*.)

Cystin. Cystin occurs in the form of hexagonal prisms, either as irregular masses or superimposed upon one another so as to form truncated pyramids. It is a very rare sediment, but appears to be most common in children and young male adults. Several members of the same family have been known to pass it. Its chief clinical significance arises from the fact that rarely it is the basis of calculi.

FIG. 246.



Tyrosin crystals. (Original.)

Leucin and Tyrosin. Leucin and tyrosin are generally described together, though the former is not spontaneously deposited from urine. It appears in the form of spheres, which refract light strongly and have a radiating arrangement. (Fig. 245.)

Tyrosin has been found as a sediment of a light greenish-yellow color in typhoid fever and acute yellow atrophy of the liver. It appears in the form of tolerably long, needle-like crystals, or as bundles and sheaves. Frerichs attaches great importance to leucin and tyrosin in the diagnosis of acute yellow atrophy of the liver. (Fig. 246.)

Cholesterin. This occurs at times in fatty degeneration of the kidneys, jaundice, chyluria, diabetes, and according to Pohl, in the urine of epileptics treated with potassium bromide. (Fig. 247.)

FIG. 247.

Crystals of cholesterin. (Original.)

Melanin. Melanin is held in solution or suspended in small granules. The urine is dark in color, and blackens intensely when sulphuric acid or ferric chloride is added to it. A concentrated solution of the chloride serves to detect the presence of the substance. A few drops added to the urine turn it gray. If a few drops more are added, the phosphates are precipitated along with the coloring-matter. Both are dissolved by an excess of the iron solution. Melanin is usually found in cases of melanotic carcinoma.

CHAPTER XLIV.

THE FECES.

General Considerations and Macroscopical Appearance.

THE number of stools in health varies chiefly with the individual and the character of the food taken. After infancy one passage in twenty-four hours is the rule, but it is natural for some persons to have two or three; and for others to have but one passage in two, three, or four days. Such a condition is termed constipation, while pathological constipation is properly called *obstipation*. The opposite condition is known as diarrhœa. The amount and character of food and drink ingested influence the number of stools. Exercise also plays a role; increased or diminished peristalsis, from whatever cause, will induce diarrhœa or constipation respectively. In disease both extremes are observed—absolute non-passage of feces for days, as in obstruction, or an almost continuous discharge, as in some forms of intestinal inflammation. It is well to remember that diarrhœa may be the symptom of obstipation, as when impacted feces in typhoid causes looseness of the bowels.

Quantity. The quantity of feces varies with the quantity and nature of the food. If most of the food is digested, there will be but little left to form feces. In any disease that prevents the absorption of digested food or causes an increase in the fluid contents of the intestine, as cholera, the amount of feces will be increased. In health about 140 to 200 grammes are voided in twenty-four hours.

Form. The form and consistence of healthy stools vary somewhat. They are commonly cylindrical and firm or mushy. When they remain long in the intestinal canal and the water is extracted, they become hard and may form balls, or flattened masses known as scybala. These are frequently seen in convalescing typhoid patients. On the other hand, the feces may be without form, and are then liquid, either watery as in cholera, or purulent or bloody. Many diseases cause such a condition.

Odor. The odor of feces is sometimes more or less characteristic of certain conditions. Thus the stools of nursing infants have a sour smell, while in infantile diarrhœa and when fermentation takes place, they have an odor of sebacic acid. When urine is mixed with the stools, the odor is ammoniacal; when blood is present, the odor is often stale.

Reaction. The reaction is not constant. In intestinal catarrh with acid fermentation it is acid, and in alkaline fermentation it is alkaline.

Color. The color of the stool varies too much to be of diagnostic value. When much meat is eaten, the color is very dark, chiefly owing to the presence of hæmatin and sulphide of iron. A lighter color is seen when a vegetable diet is taken, the color according to Fleischer, being

then due chiefly to urobilin. With absolute milk-diet the color almost disappears. Unaltered bile-pigments are never found in the feces normally. The ingestion of some varieties of fruit, notably huckleberries, and of certain drugs as iron and bismuth, renders the stools black. Calomel causes green stools, on account of the biliverdin discharged. Green stools may also receive their color from the presence of a bacillus which produces a green pigment. Santonin, rhubarb, and senna cause yellow, and hæmatoxylin red stools. The last fact is important, as parents or nurses should always be warned to expect red passages when hæmatoxylin is given.

The feces may be red or reddish from the presence of unaltered blood; or black when the blood has undergone changes—the so-called "tarry stools" are of this character. With a decrease in the amount of bile the stools become less highly colored; and if the bile is cut off, they become clayey. This color may in some cases be due to the presence of fat left undigested because of the lack of bile. On the other hand, if from disorders of the stomach and intestine the contents pass through too rapidly, the feces may contain unaltered bile and unchanged bile-pigment, thus giving a green or yellow color and showing the bile-reaction.

Constituents. The constituents of feces that can be recognized by the naked eyes are numerous. Seeds, stones, skins of fruit and berries, and the fibres of vegetables are often seen in normal stools. In the passages of children and weak-minded individuals foreign substances of all descriptions may be present. Foreign bodies and partially digested portions of food may be mistaken for parasites. Portions of tumors from the digestive tract may appear in the feces.

In certain diseases of the stomach and small intestine, and in those who eat very fast and do not properly masticate their food, undigested and unchanged particles of food may be seen in the stools.

Shreds of mucous membrane of varying size are passed with the feces. Von Jaksch saw such a shred 5 cm. long and 3 cm. broad in a case of cholelithiasis. Various sized pieces of membrane, consisting of transformed mucus, are passed in membranous enteritis. Particles resembling sago-grains, perhaps the result of over-indulgence in farinaceous food, have been found.

Gallstones in the feces have great clinical value. They may escape detection if not properly sought for. When suspected, each stool should be passed through a linen sieve, the fecal masses being softened with water. The gallstones may be found as small crumbling masses, composed chiefly of cholesterin (intrahepatic calculi), or as hard, irregular, smoothly worn, shining, and many-sided stones, sometimes as large as an egg, usually the size of a pea. Enteroliths are occasionally seen. They are said to originate in the appendix.

Blood may be present in the feces in varying proportions and conditions. When found unaltered on the surface of scybalous masses, it is from the rectum or large intestine, and probably the result of traumatism. Bleeding piles may cause such an appearance, and may even cause very free hemorrhage. Severe hemorrhage may come from ulceration of the rectum or colon due to malignant disease or severe inflammation. The blood

may be intimately mixed with the feces, and have its origin in the large intestine; but much more commonly its source is in the stomach or small intestine. Under such circumstances it is nearly always more or less changed by the intestinal juices, and is brownish red or black (the tarry stool mentioned above), or has the appearance of coffee-grounds. The brighter the color of the blood, the nearer is the source of hemorrhage to the anus. The more retarded the passage the greater the change; while, if quickly expelled, blood from the small intestine may be passed unchanged, as in the hemorrhage of typhoid fever. The microscope may detect blood when the naked eye fails, but the blood-corpuscles are usually destroyed unless the hemorrhage is large and quickly discharged. The hæmin and guaiac tests or the spectroscope may be used. It must be remembered that the hæmin test sometimes fails even when blood is present, and that blood may be found in small amounts as a result of eating raw meats, sausages, and the like. It is also to be remembered that certain drugs, as already stated, may color the feces red, and simulate blood.

Mucus may be present in the stools in health, but when in any marked quantity there is a catarrh of the mucous membrane of the intestines. When hard scybala are covered with mucus or the mucus is seen in shreds, the large intestine is the seat of a catarrh; although mucus may be mixed with thin stools, as in dysentery. Usually, however, when the mucus is finely divided and mixed with the feces, it comes from the small intestine. Mucous shreds have already been mentioned. In cholera the particles of mucus look like boiled rice, hence the term "rice-water stool."

Fatty stools, to the naked eye, appear greasy, or even clayey when there is much fat, even though bile-pigment may be present.

Pus may be present in large quantities from rupture of an abscess into the intestinal tract, or when there are ulcerations from various conditions, producing pus in considerable quantities.

Microscopical Examination of the Feces.

Many animal parasites are not microscopic, but it is convenient to consider them in the following paragraphs. A small portion of the solid feces to be examined is placed on a slide, moistened with a 0.5 per cent. salt solution, and a cover-slip applied; or if liquid, various drops are to be examined. The constituents found will vary with the food taken as well as with disease.

Constituents Derived from Food.

There may be portions of digested or undigested food. In general it may be said that the presence of large pieces of unchanged food, or many small particles of undigested or only partially digested food, indicate defective digestion in the stomach or small intestine. If unchanged bile is present, some particles will be colored yellow—another indication of disordered function.

We may see muscle and elastic fibres in more or less abundance according to the quantity of meat eaten by the patient. The former are

recognized by their transverse striation; the latter, by their double contour and curling ends. Fat may be present as fatty globules or in the form of needles—fatty crystals. Much fatty food increases their number, and they are seen plentifully in alcohol-poisoning, in jaundice, in pancreatic diseases, tuberculosis of intestines, diseases of the mesenteric glands, and enteritis. The crystals may be transformed into fat-drops by the addition of acid and heat. When meat is eaten freely, areolar tissue may be present, but its presence otherwise points to defective digestion. Schmidt considers that if remnants of areolar tissue are present after a test-meal of 100 grammes of chopped meat, it points to defective gastric digestion, while the presence of undigested muscular fibre indicates disturbance of intestinal digestion. Various forms of vegetable cells are commonly seen, in which granules of starch may be contained or the starch particle may be free. The presence of starch-granules or of many vegetable remnants containing starch, or a general blue color on the addition of iodine, always indicates imperfect starch digestion. Normally, starches are practically completely digested. Undigested milk occurs in the stools of children and when diarrhœa prevails; a substance, probably casein, has been described by Nothnagel as occurring in the feces of persons who have intestinal disturbances.

In persons living on vegetables most of the above constituents will be absent, and in infants who partake only of milk, the derivatives of meat are absent, while there will be an excess of fatty crystals, fat-globules, and coagulated products.

Constituents from the Alimentary Tract.

Epithelium. In every normal stool will be found *epithelium* of the squamous variety. Occasionally the columnar form is seen, and modified epithelial cells are very common. In intestinal catarrh their number is greatly increased.

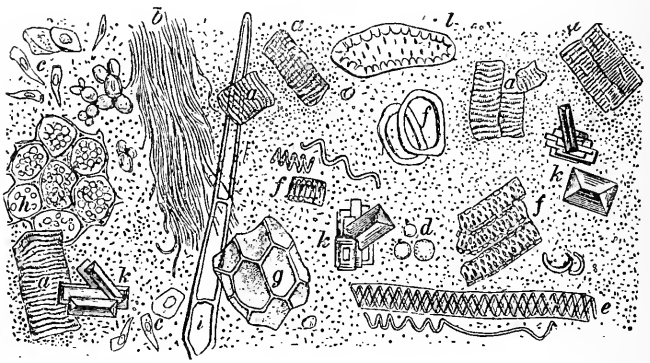
Red Blood-corpuscles. In the majority of blood-stained stools red blood-cells are not found; in their stead will be seen masses of free blood-coloring matter and rhombic crystals of hæmatoidin. Red cells are seen in dysentery, in bloody stools in which the blood comes from near the anus, as in hemorrhoids, and when blood is discharged with the feces soon after the occurrence of the bleeding. If there is any doubt as to the presence of blood, when the corpuscle can not be found, a true decision can sometimes be reached by examining for hæmin crystals by Teichmann's method. A portion of feces is dried and powdered, placed on a slide with a grain of common salt, and covered by a cover-slip. A few drops of glacial acetic acid are directed beneath the slip, the slide is heated just to boiling, and if blood has been present, reddish-brown rhombic crystals of hæmin will usually soon be found; but, as stated, the test is not wholly reliable.

Leucocytes. Leucocytes are frequently seen in normal stools; when pus is present or discharged into the intestinal canal, they are found in great numbers, as in ulceration of the intestine and in abscess.

Molecular débris, or detritus, occurs in all feces as part of the waste-products.

Crystals. *Fat crystals* are the most important. They have been quite fully considered above. There seems to be little doubt that the crystalline needles found in the feces are salts and fatty acids, and not tyrosin.

FIG. 248.



Collective view of the feces. (Eye-piece III., objective 8 A, Reichert.) *a.* Muscle-fibres. *b.* Connective tissue. *c.* Epithelium. *d.* White blood-corpuscles. *e.* Spiral cells. *f,i.* Various vegetable cells. *k.* Triple phosphate crystals in a mass of various micro-organisms. *l.* Diatoms. (VON JAKSCH.)

Charcot-Leyden crystals, similar to those already described (see Sputum), have occasionally been met with in the stools of typhoid fever patients, in dysentery, intestinal tuberculosis, and ankylostomiasis.

Hæmatoidin crystals occur as reddish-brown, hard, needle-shaped bodies, usually in clusters, and free or enclosed in masses of mucin or a substance resembling it. They have been found in the feces of breast-fed infants, in cases of chronic intestinal catarrh, and, by von Jaksch, in the stools of a case of nephritis.

Crystals of various *salts of calcium*, of *triple phosphate* and *cholesterin* will often be recognized, but they have no diagnostic value. When bismuth is being administered, black rhombic crystals of the sulphide of that metal will be recognized.

Parasites.

Animal and vegetable parasites flourish in the intestinal tract, and the presence of some of these in the feces is of the greatest clinical importance.

A. Animal Parasites. Following Leuckart's classification, we will consider these parasites under the secondary heads:

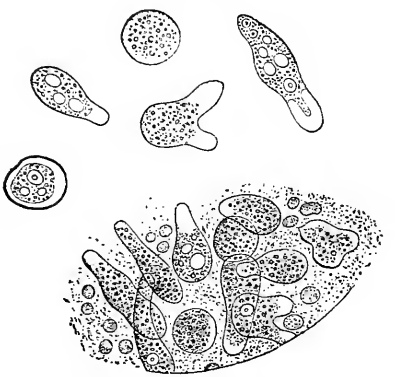
I. Protozoa. 1. RHIZOPODA. This variety is made important because *Amœba dysenteriæ* or *Amœba coli* belongs to it.

a. Amœba Dysenteriæ—Amœba Coli. This protozoön has been found so many times by various observers in different parts of the world that it is now considered by many writers to be the causative factor of so-called tropical dysentery. The subject has received special study in this

country by Osler,[1] Stengel,[2] Dock,[3] and Councilman and Lafleur.[4] The work of Councilman and Lafleur is at the present time the best that has been published in any country; and to it the reader is particularly referred. The following notes are based on this book:

Amœbæ dysenteriæ vary in size from 0.012 to 0.035 mm. They are found most plentifully in the small gelatinous masses often to be seen in the feces. They vary in number in different cases, and in the same case at different times. The severer the lesions the more numerous are the amœbæ. When not active, they are round or oblong, and highly refractive. They contain one or more vacuoles of varying size. Occasionally the division into an ectosarc and endosarc is easily made out. When thus inactive, they may be confounded with swollen connective-tissue cells and compound granular bodies found in feces. The active amœbæ have, however, a characteristic movement. This consists of progression and of thrusting out and retraction of pseudopodia. Their activity varies greatly. It is best seen when the body heat is maintained. The stools should be passed into a clean and warm pan, and examined immediately or kept

FIG. 249.



Amœba coli. (HALLOPEAU.)

warm until examined, and a warm stage should be used with the microscope. The division into ectosarc and endosarc is usually clear during activity. The ectosarc is composed of a hyaline homogeneous mass, as are the pseudopodia, while the endosarc is made up, not of granular matter, but of a dense homogeneous mass enclosing vacuoles and a nucleus. The vacuoles may vary in size as well as in number. There may be one or two large ones, or the entire endosarc may appear as made up entirely of small vacuoles. The nucleus is sometimes plainly seen as a small rounded body, but is more often difficult to distinguish from the vacuoles. Dried cover-slip preparations may be stained with the various aniline dyes, but the results are not satisfactory.

The amœbæ will often be found to enclose bodies such as red blood-corpuscles, pus-cells, blood-coloring matter, bacilli, and micrococci.

[1] Johns Hopkins Hospital Bulletin, May, 1890, vol. i., No. 5.
[2] Philadelphia Medical News, 1890.
[3] Texas Medical Journal, April, 1891.
[4] Johns Hopkins Hospital Reports, vol. ii., Nos. 7, 8, 9.

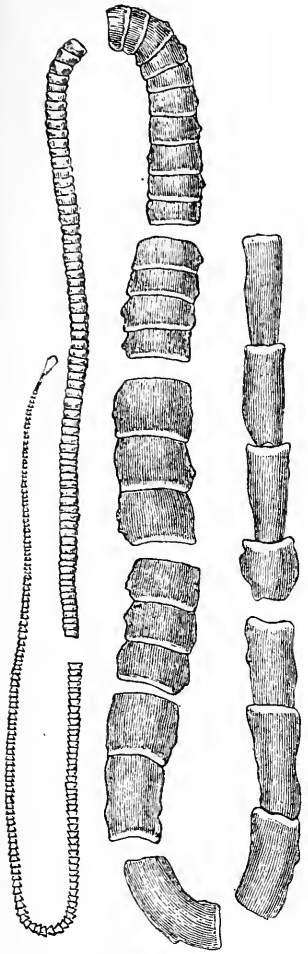
In examining feces for *Amœbæ dysenteriæ* the suggestion given above concerning the warm bed-pan and warm stage of the microscope, and, above all, the immediate examination of the stool, should be adhered to. The small gelatinous masses should be selected when present. In obtaining the material it is often of advantage to pass well into the lower intestine an ordinary rectal tube, in the eye of which more or less of the mucous exudates from the surface of the gut is likely to adhere, suitable particles from which may be employed for the microscopical examination. In some cases of chronic amœbic dysentery, especially when much thickening of the wall of the colon exists, one may fail repeatedly to find the parasites in the ordinary dejection; but may succeed excellently if a saline aperient be given and the watery dejecta following this be used for examination. In searching for the amœbæ it is usually sufficient to make use of the ordinary laboratory powers of the microscope; but for the details of structure it is advisable to have recourse to the $\frac{1}{12}$ inch oil immersion lens. It is desirable to encounter the amœbæ in active movement, in order to appreciate the number and character of the pseudopods, and the degree of mobility of the parasite, these features being utilized in the positive recognition and classification of these organisms. It should be advised therefore that at regular intervals, of half minute intermission, hasty sketches be made of any amœboid body found, in order that the best and a fixed appreciation of the above characters may be had.

2. Sporozoa: *Coccidium perforans* (Leuckart) (*C. hominis*) has been met in the stools of human beings occasionally. These sporozoa, which in their earlier stages have existed in the lining epithelial cells of the intestine, at the stage of encystment escape into the intestinal lumen; and may be found in large numbers in the dejecta. They are short elliptical bodies, very like the familiar *Coccidium oviforme* of the liver of rabbits, but smaller in size.

3. Infusoria. *a. Cercomonas intestinalis* (*vel Lamblia intestinalis*, etc.) is a pyriform protozoön, about 10 or 15 μ in length, nucleated, with a marked mouth-like depression on one side anteriorly, and provided with four pairs of flagella, three pairs symmetrically placed about the border of the depression mentioned and the fourth pair containing the posterior pointed extremity. While more frequent in the lower animals, it has often been encountered in man. Its most frequent habitat is in the duodenum and jejunum, but occurs also in the large intestine. In the stools it is often in the encysted stage, but may be encountered in an active state. It commonly occurs in the stools of chronic diarrhœa, but is of uncertain pathogenic importance.

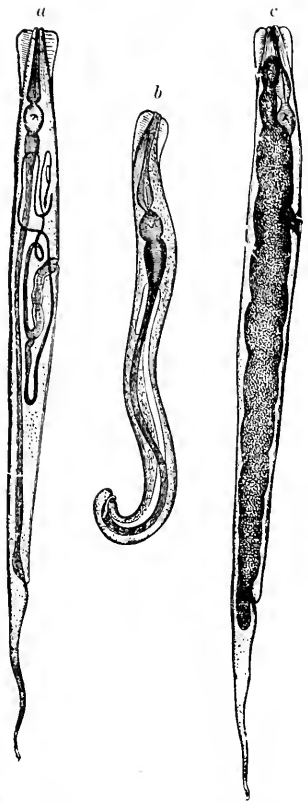
b. Trichomonas intestinalis (*vel Tr. hominis, Cercomonas intestinalis, C. hominis*, etc.) is somewhat smaller than the preceding, being about 10 or 12 μ in length, of a pyriform or subovoid shape, tapering at each extremity, and provided with five flagella (four attached to the anterior extremity and the fifth passing from the anterior part backward as the border of an undulating membrane and extending some distance beyond the posterior extremity of the cell). The appearance of cilia is given along the side by the undulating membrane. Close to the base of the flagella is a mouth-like depression. Within the cell a definite nucleus

FIG. 250.



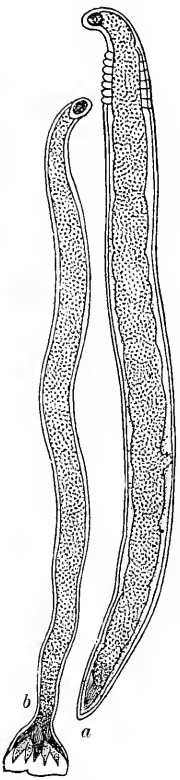
Tænia saginata: natural size. (After LEUCKART.

FIG. 252.



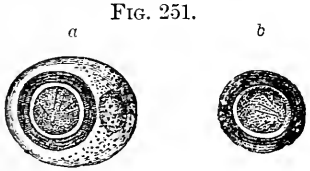
Oxyuris vermicularis, magnified: *a*, young female; *b*, male; *c*, mature female, full of eggs. (PAYNE.)

FIG. 253.

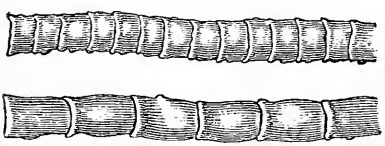


Uncinaria duodenalis, magnified: *a*, female; *b*, male. (BRISTOWE.)

FIG. 251.

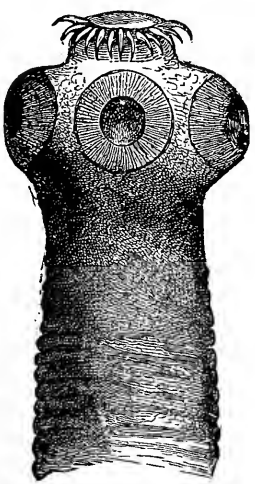


Ova of *T. solium:* *a*, with yolk; *b*, without yolk, as in mature segments. The hard brown shell is indicated. (LEUCKART.)



Half-grown and mature proglottides of *Tænia solium:* natural size. (After LEUCKART.)

FIG. 254.

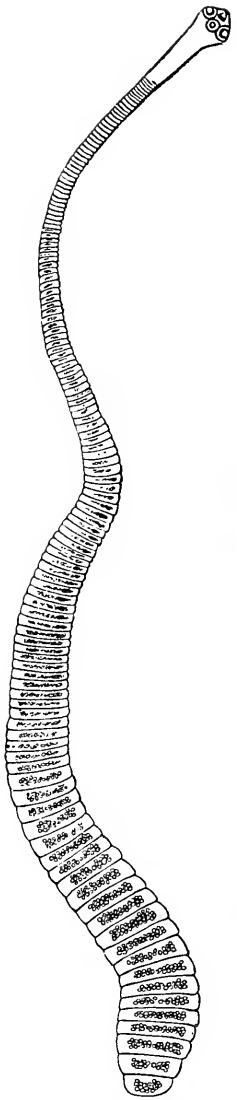


Head of *T. solium:* × 45. (LEUCKART.)

FIG. 255.

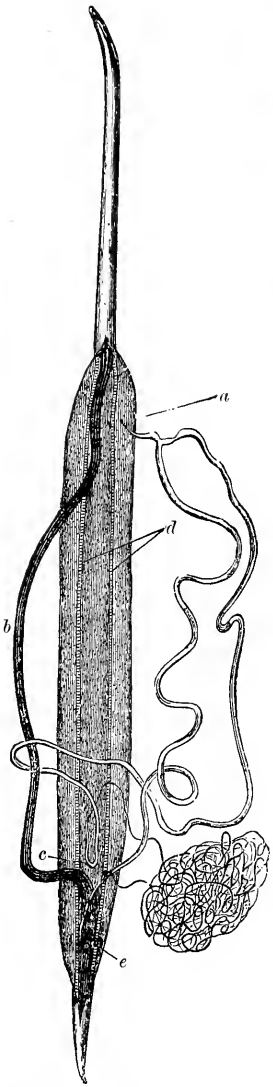
Tænia cucumerina: natural size. (After LEUCKART.)

FIG. 256.



Hymenolepsis nana: × 12. (After LEUCKART.)

FIG. 257.

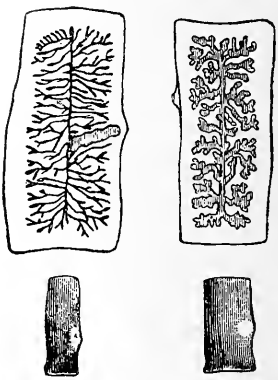


Ascaris lumbricoides, dissected and walls thrown back: *a*, genital orifice; *b*, intestine; *c*, oviducts; *d*, longitudinal band; *e*, ovaries. (HELLER.)

FIG. 258.

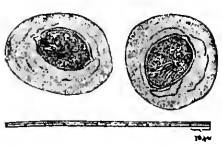
Head of *Tænia saginata*. (EICHHORST.)

FIG. 259.



Proglottides of (*a*) *Tænia saginata* and (*b*) *T. solium;* natural size, and enlarged three times to show arrangement of uterus. (After LEUCKART.)

FIG. 256*a*.

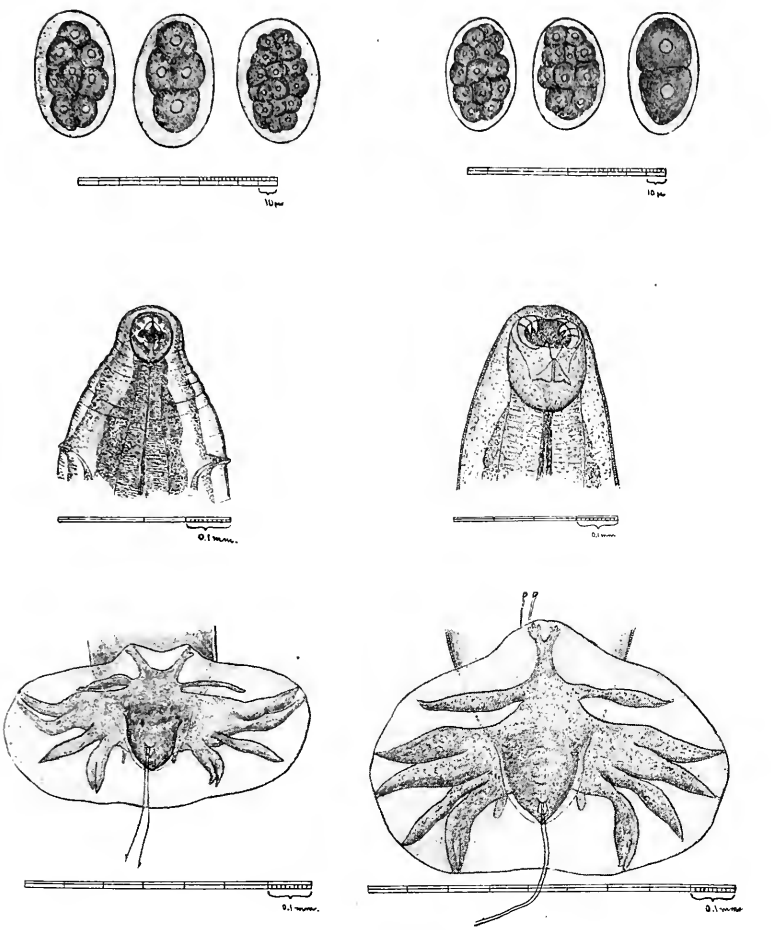


Ova of *Hymenolepsis nana*. (Smith.)

FIG. 260.

Proglottis of *Bothriocephalus latus:* natural size, and enlarged three times. (After EICHHORST.)

Fig. 261.

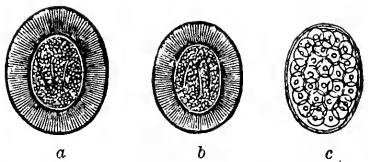


Above: to the left, three ova of *Uncinaria Americana* in different stages of segmentation, from 66 μ to 68 μ in long diameter: to the right, three ova of *Uncinaria duodenalis* in varying stages of segmentation, long diameter from 58 μ to 62 μ.

Middle: to the left, the head of *Uncinaria Americana* (dorsal view), exhibiting the smaller size of the head and more tapering anterior extremity of the worm in comparison with *U. duodenalis*, and its buccal armature consisting of lips; to the right, the head of *Uncinaria duodenalis* (dorsal view), drawn to same scale, showing its greater size and the hook armature of the buccal border.

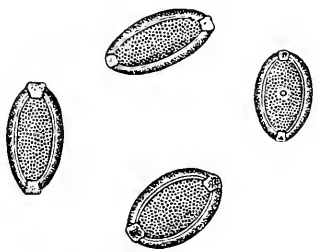
Below: to the left the caudal border of male *Uncinaria Americana* (dorsal view, dorsal lobe of bursa thrown forward), showing comparative size of this worm in relation to that of *U. duodenalis*, partly divided dorsal lobe and suggestion of a ventral lobe, the dorsal ray divided nearly to base, each of its divisions having a bipartite tip, showing also the tip of tail within bursa, the long barbed sexual spicules, and the arrangement of the rays of the bursa; to the right, caudal bursa of male *U. duodenalis*, showing larger size of bursa, entire dorsal lobe, absence of ventral lobe, division of dorsal ray limited to distal third and tripartite tips of each division, with other features in general similar to *U. Americana*. (Smith.)

FIG. 262.



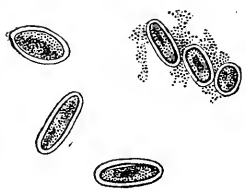
Eggs of (*a*) *T. saginata*; (*b*) *T. solium*; (*c*) *Bothriocephalus latus*: × 300. (After EICHHORST.)

FIG. 263.



Eggs of *Trichuris trichiura*: × 275. (EICHHORST.)

FIG. 264.

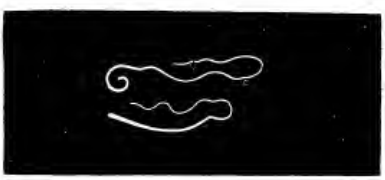


Eggs of *Oxyuris vermicularis*: × 275. (After EICHHORST.)

FIG. 265.

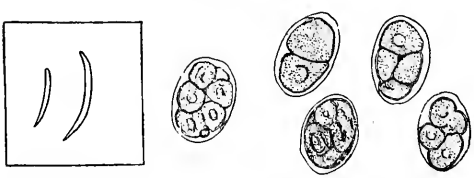
Oxyuris vermicularis: natural size. (EICHHORST.)

FIG. 266.



Trichuris trichiura, male and female: natural size. (EICHHORST.)

FIG. 267.



Uncinaria duodenalis, with eggs. (After BLICKHAHN.)

with nucleolus may be distinguished. It is usually encountered in cases of chronic diarrhœa, but its pathogenic influence is doubtful.

Balantidium coli (*vel Paramœcium coli*) is a large ciliate infusorian, of a somewhat flattened oval shape, measuring from 70 to 100 μ in its long diameter. It is colorless and uniformly covered with cilia, having a special border of cilia at the anterior (blunt) extremity. Its interior is finely granular, contains two contractile vacuoles, a bean-shaped nucleus, and a variable amount of extraneous substances which have been ingested from the intestinal contents. In general it closely resembles the ordinary "slipper animalcule" of the gutter. It is found, like the preceding infusoria, in the stools of patients with chronic diarrhœa, but its influence in the production of the condition is doubtful.

II. Vermes. Verminous intestinal parasites are more generally known than the above protozoa, and their clinical importance is less uncertain. Both local and general symptoms of importance may be predicated of individual examples. They are most conveniently outlined following the usual classification of flat and round worms:

1. PLATODA. 1. *Tapeworms—Cestoda.* These parasites infest only the small intestine, to the walls of which they cling by the head. The head and neck are small; the joints are flat and form long ribbons. The distal joints continually drop off and can easily be recognized in the stools by the naked eye, and the eggs by the use of the microscope. The feces are best washed in water and broken up to obtain the eggs. As the lower joints are lost, new ones take their place from above. The more important are the following:

a. Tænia solium reaches a length of 2 to 3 metres. The head is the size of a pin-head. The neck is 2.5 cm. long, as thick as a thread, and without joints. The segments forming the body are short and broad near the neck, but as they increase in size there is more growth in length than in width. The average dimensions are 9 to 10 mm. by 6 to 7 mm. The head appears dark, the body white. The joints are easily detected in the feces by the naked eye. Under the microscope the head is seen to be spheroidal, with 4 pigmented sucking-disks surrounding at the base a rostellum, which is a "crown of hooks"—chitin hooks—about 24 in number. In the ripe segments, or proglottides, is seen the longitudinal uterus with about 12 horizontal ramifications to a segment. The eggs are round or oval, 0.035 mm. long, with a thick striated shell when ripe, and contain hooklets.

b. Tænia mediocanellata or *saginata.* This worm is 4 or 5 metres long. The head is slightly larger than that of *T. solium*, and more pigmented, and the segments are longer, thicker, and darker. The head is supplied with 4 powerful sucking-cups, but has no rostellum or hooklets. The uterus in the ripe segment is much more finely branched than in the solium, and these segments have independent movement. The eggs are very similar to those of *T. solium*, but may be somewhat larger.

c. Tænia Echinococcus. This tapeworm in its adult stage is a common parasite of the intestine of dogs. In its embryonic stage the bladder worms are ordinarily found in the viscera or flesh of man, and a number of the mammalia. The cysticercus is peculiar in that it is capable of

multiplication to the formation of large collections of the bladder worms, thus constituting the so-called "hydatid cyst." Instances of hydatid formation in the gall-bladder, gall-ducts, appendix vermiformis, or in immediate proximity to the intestine, have been known to present in the feces from time to time the characteristic bladder worms. The cysticercus is a small round or ovoid body, of a somewhat nacreous tint, varying in size from that of a buckshot to that of an ordinary Malaga grape (and even much larger), filled with a clear fluid (or in case of death of the embryo with a somewhat turbid fluid in which the hooklets of the rostellum are to be found) and containing the developed pyriform head of the future tapeworm, with its 4 suckers and rostellum of about 40 hooklets.

d. Hymenolepis nana (*vel Tænia nana*). In length, *T. nana* is only 10 to 15 mm., and 0.5 mm. in breadth. The round head is but 0.3 mm. in diameter. The segments are all short, and at the lower end of the body are 4 times as wide as they are long. The head is found to have 4 round suckers at the base of a rostellum that can be inverted. At the base of the rostellum are about 22 hooklets. The uterus is oblong and filled with eggs. The eggs have a double membrane. This tapeworm has recently been found in a number of instances in this country. It is especially likely to be found in children. Its ova are nearly spherical, about 40 μ in diameter, colorless, with double wall. The embryo within measures about 22 or 23 μ, is colorless and granular, and is provided with six embryonic hooklets easily discernible. At each pole of the embryo is to be seen a slight protuberance, and between the inner and outer shell membranes, several filaments may be detected. (Fig. 256.)

e. Hymenolepis flavopunctata (*vel Tænia flavopunctata*). This small tapeworm has been encountered several times in the United States. It attains a length of about one foot. The head is small, clubbed, and unarmed. The neck is short, the developed links 1 mm. in length and 2 to 2.6 mm. wide. The last links are slightly longer and from shrivelling become nearly as narrow as long. Each fully developed link is marked by a small yellow spot in the posterior portion. The ova are similar in appearance to those of *H. nana*, but larger, measuring 60 μ in diameter.

f. Bothriocephalus latus. This is the largest of the tapeworms likely to be met in the human intestine, measuring 7 or 8 metres or more in length. The head is of an elongated club shape, provided with two long, narrow slits, one on each side, serving as suckers. There are neither hooklets nor rostellum. The proglottides are short near the head and very wide, but toward the lower end of the worm become nearly square. Each ripe link shows a reddish or brownish rosette-like mass near the middle, caused by the coiled uterine tube filled with the yellowish or faintly brownish eggs. These eggs may be encountered in the dejecta, as oval, thin-shelled, brownish objects measuring about 70 μ in length and 40 to 45 μ in breadth, showing more or less advanced segmentation and provided with an operculum or lid at one end for emergence of the ciliated embryo. The embryo makes its habitat in the tissues of certain fish.

It will not be necessary to describe certain other varieties that are rarely met with.

Flukeworms, Trematoda. *a. Distoma hepaticum* measures 28 mm. by 10 mm., and is shaped like a leaf. A short head is situated at the broad end and has one sucker; on the under surface is another sucker, and between the two is the opening of the uterus, a highly convoluted arrangement. This worm is not often seen in man. Its habitat is in the hapatic ducts, where it gives rise to the condition known as liver rot. The worm is hermaphroditic; and its eggs after discharge from the genital pore, find their way along the bile-ducts, and into the intestine. They are thin-walled, of a brownish-yellow hue, ovoid in shape, usually seen in advanced segmentation, and measure from 120 to 140 μ in length and 70 to 90 μ in width. A lid may be demonstrated on one end for the emergence of the embryo. The intermediate stages of the parasite are passed in the tissues of snails, and subsequently attached to a blade of grass.

b. Distoma lanceolatum. This fluke is much smaller than the preceding, being more elongated in shape, but preserving the general leaf-shape of the Distoma hepaticum. It is about 8 mm. long and 1.5 to 2.5 mm. broad. The extremities, especially the anterior, are sharper than those of the preceding. It is semitransparent, and flecked with brown from the eggs within the body. It is also more common in the lower animals than in man. Its habitat is in the bile passages, its eggs being carried thence into the intestine. They are ovoid, with thin shell, of a light-brown color, measuring about 40 to 45 μ in length and 20 to 30 μ in width. The tiny embryo is ordinarily visible within the granular contents. An operculum may be shown on one end.

c. Distoma sinense (*vel D. japonicum*). A fluke of about the same size as the preceding, but more pointed anteriorly and more rounded posteriorly than the latter, reddish in color, and when living, nearly transparent. This parasite is found with some frequence—China and Japan—in the human biliary passages. It has also been met in the cat, in the same regions. The eggs may be found in the dejecta, dark (almost black) ovoid bodies, about 30 μ long and 16 or 17 μ broad; they are apt to show in the interior the ciliated embryo. The intermediate life history is not known.

d. Distoma buski (*D. crassum*). This is a rare human parasite found in the human intestine in China and the neighboring continental and insular districts. A case in an East Indian Lascar sailor dead from typhoid fever, has recently been encountered by Tenille, of Galveston, Texas (personal communication). The parasite is quite large, 3 to 7 cm. in length and 10 to 20 mm. broad. The eggs are 125 μ long and 75 μ broad.

II. Nematoda. Round worms with complete digestive canal. *a. Ascaris lumbricoides.* This is the parasite usually referred to by the term round worm. It has a light cream color and resembles the common earthworm in shape. The male worm is about 250 mm. long, and the female 400 mm. The head is made up of 3 prominent lips, and is supplied with microscopical teeth. The vulva is in the posterior third of the body. The eggs are rounded, brownish, 0.06 mm. in diameter, and covered, when fresh, by a rough albuminous coat over a hard shell. This worm has the small intestine for its habitat. It may pass with the stools or work its

way into the stomach and be vomited (the writer has seen them thus vomited during the etherization of a child of ten years). They have been the cause of jaundice by crawling into the ductus choledochus, and may infest the larger hepatic ducts. An enormous number may be present in the intestine at one time.

b. Oxyuris vermicularis. The thread-worm, or seat-worm, inhabits the large intestine, and is often present in the stool as a white, thread-like body; the male 5 mm. and the female 10 mm. long. They often wander out of the anus and into the vagina. The head has a number of small lips, and is covered with a thick skin. The female has 1 vagina and 2 uteri. The eggs are colorless and unsymmetrical, have a laminated shell and a diameter of about 4 mm. They are not often seen in the stools because they hatch out in the intestine.

c. Uncinaria duodenalis (*vel Anchylostoma duodenale*). This parasite, the European form of hook-worm, is in its parasite life met in the upper part of the human small intestine, where it attaches itself by its special mouth armature to the mucous membrane, wounding the latter and sucking blood from the tissues. Such tiny wounds continue to bleed when the worm changes its places of attachment; and, should the openings be very numerous, through serious and long-continued blood loss, there is apt to be serious, and even fatal anæmia produced. The worm is bisexual, the female measuring from 10 to 18 mm. in length, the male 8 to 10 mm.; both are of a cream-like color, ranging to a faint red (if there be much blood débris in the intestine, individual worms may be blotched with brownish dots and patches). In each sex the head is turned back dorsally, forming the tiny hook from which the common name, "hook-worm," is derived. The head has a cup-shaped mouth, with oval, and at the depth, with pharyngeal armature. The oval armature is of importance in recognition of the species, consisting of a pair of hooklets curving into the mouth from the buccal border on either side of the ventro-median line, and a dorsal pair of prominent tooth-like structures at the dorso-median line—the appearance being given of six hooklets at the border of the buccal capsule. The female ends posteriorly in a finely tapering manner, the male in a blunt point surrounded by a lobed bursa supported by characteristic rays. The vaginal opening is at about the posterior third of the length of the worm. The ova are colorless, oval in shape, thin walled, with more or less segmented contents, and measure 50 μ in length and 32 μ in width. These ova develop after discharge upon some moist spot into minute rhabditiform embryos, capable of independent existence in dirty water or moist soil; in their free life they undergo several moults. They are known to be capable of gaining entrance to the intestine of the future host by penetrating through the skin (Loos), and in unknown manner making their way to the proper intestinal habitat. In so doing it is believed they are responsible for certain itch-like lesions, known as ground-itch or water-itch. It is possible that they also gain entrance to the intestine of the host by the direct alimentary route as in swallowing the dirty water in which these embryos live.

d. Uncinaria Americana. Recently in the West Indies and our

Southern States a parasite almost identical with the above, has been found, and has by Stiles been classified as species distinct from the European hook-worm. Its effects are similar to those of the latter. Its habitat is similarly in the upper portion of the human small intestine. It is slightly shorter than the above measurements given for the European hook-worm and is distinguished from the latter by the fact that instead of possessing the hooked buccal armature of the head, the border of the capsule on each side of the ventro-median line projects into the mouth as a large uniform semilunar lip, a smaller lip of similar appearance existing on either side of the dorso-median line. Other points of difference may be noted in the rays of the caudal bursa of the wall, and in the fact that the vaginal opening of the female is nearer the middle of the worm than in the European species. The ova are somewhat larger characteristically, but otherwise have the same appearance. They measure 60 to 68 μ in length by about 40 μ in width. The intermediate stages and mode of infestment of the host have not been completely discovered, but probably are similar to those of the European form.

e. Trichinis trichinora (*Trichocephalus dispar*). The common whip-worm, so-called from its shape is 4 to 5 cm. in length, the female being longer than the male. It is recognized by the marked contrast between the anterior and the posterior portions. The former is thin and thread-like, the latter expanded and broad, and in the male curled up. The eggs are brownish, about 0.05 mm. long and half as broad, and have a button-like projection at either end; they are to be recognized in the stools, where large ones may be present. There may be only a few or thousands of the forms present in the body. They live chiefly in the cæcum and large intestine. They have been thought to cause beri-beri by some writers.

f. Trichina spiralis. It is the adult trichinæ which exist in the intestine and are found very frequently in the feces. These produce the embryos, which become muscle trichinæ. The adult male is 1.5 mm. long and the female twice that length. The former has 2 projections from the posterior extremity, between which are 4 papillæ. The female has a tubular uterus and a tubular ovary in the posterior half of the body.

g. Strongyloides intestinalis (*vel Rhabdonema intestinale*). The parasitic and the free generations of this worm in the older texts have been regarded as separate species, the parasitic form being known as the *Anguillula intestinalis*, the free form (found in feces after development outside the body, or in mud) as *Anguillula stercoralis*. The parasitic form is a parthenogenetic female, a little over 2 mm. in length, cylindrical, slender and finely tapering posteriorly. The vulvar opening is at the posterior third of the body. A large number of these may infest the intestine. Ova of a greenish-yellow color, oval, and of advanced segmentation, are deposited, which quickly (several hours at most) hatch into embryos of a rhabdite type. These at time of birth are from 200 to 240 μ long, and about 12 μ broad; when found in fresh stools they are further grown, 500 to 600 μ long and 16 to 20 μ broad. Under favorable conditions these develop into sexually mature rhabditoid males and females (Anguillula stercoralis), and the females deposit ova or living

young, very like those produced in the intestine but a little more slender, which if circumstances permit gain access to the digestive tract of another host where they pass their parasitic life as Anguillula intestinalis. Their pathogenic influence is uncertain.

D. *Rhabdonema. Strongylides.* Under *Rhabdonema intestinale* we now include two small nematodes, which were termed *Anguillula intestinalis* and *A. stercoralis*, and which are probably one and the same. They are found in the stools of cases of endemic diarrhœa of hot countries. Usually the young embryos, which have developed in the intestinal canal, are dejected with the stools. These sexually mature embryos are 0.8 and 1.2 mm. long, male and female respectively. They are round and have a cone-shaped head. There are 2 jaws and 2 teeth in each. The adult worm is about 2.2 mm. long and 0.04 mm. thick. The mouth has 3 lips. The vulva is at the beginning of the posterior third. The eggs might be easily confounded with those of *Ankylostomum duodenale*, but are somewhat more pointed, and larger. The rhabdonema infests the small intestine, and is frequently found in connection with ankylostoma. Echinococcus hooklets and portions of the striated cyst-wall have been found in the feces. The rupture of a hydatid cyst into the intestine may be discovered when the above structures are found, pointing to a cyst in the abdominal cavity.

B. **Vegetable Parasites.** We find both *pathogenic* and *non-pathogenic* vegetable parasites in the feces. The latter we have classed as (1) moulds, (2) yeasts, and (3) fission-fungi.

1. Moulds. The only mould found in the stools is the thrush fungus when children are the subjects of thrush in the mouth. It is of rare occurrence in the feces, and has no special clinical import.

2. Yeasts. In all feces, in health or disease, yeast-fungi exist. They are most numerous in acid stools. They are round or ovoid, and usually occur in groups. They stain dark brown with a solution of iodine and potassium iodide, while apparently similar cells become violet or blue with the same dye.

3. Fission-fungi. Bacteria are found in greatest numbers in the feces, chiefly as bacilli, micrococci, and spirilla. They may be grouped as torulæ or sarcinæ. They present active movement, and may be separate or in colonies. *Bacillus coli communis* (*B. termo*) is the most frequent form met with both in health and disease. It is not yet determined what relations it holds to normal and abnormal conditions, or what is the true relationship between it and certain other bacteria. *B. subtilis* is another bacterium found both in health and disease. As above stated, there are various organisms which stain brown with iodo-potassic iodide solution, and others which become blue with the same dye. Von Jaksch has studied these latter closely. They assume various forms, as long or short rods, and stain different shades of blue or violet. One of them is *Clostridium butyricum* of Nothnagel. It occurs as large round cells, like yeast-fungi, and stains like the tubercle bacilli with the Ziehl-Neelsen fluid. Von Jaksch finds these fungi in greater abundance in intestinal catarrh. They are present in both acid and alkaline stools.

Bacillus coli communis has been found in the blood, in various organs,

in feces of cholera patients, in healthy feces, in the air, and in putrefying infusions; it can also be found in the peritoneal exudate in most cases of peritonitis.

Morphology. A bacillus, 4 μ to 6 μ by 2 μ to 3 μ, with rounded ends, in cultures sometimes a short oval. Five or more flagella have been observed attached to the organism.

The remaining fungi on the list are pathogenic.

Spirillum Choleræ Asiaticæ. (See page 797.)

Spirillum of Cholera Nostras. *Morphology.* Longer and thicker than the spirillum of Asiatic cholera; central part thicker than ends. Stains like the true cholera spirillum.

Biological Properties. Culture. A thick, stocking-like funnel of liquefaction instead of a fine, straight funnel.

Typhoid Fever Bacillus. This bacillus is present in the stools of typhoid fever patients, but can not be directly differentiated by microscopical examination alone, either when stained or unstained. It is necessary for its detection to make pure cultures according to bacteriological methods. The bacillus is about as long as the tubercle bacillus, but much thicker, being one-third as thick as it is long. The ends are rounded. It is best stained by concentrated aqueous solutions of methylene-blue, the dried preparations on the cover-slip being prepared as above. (See Typhoid Fever.)

Tubercle Bacillus. The bacillus of tuberculosis is frequently found in the feces of persons suffering from intestinal tuberculosis and occasionally in the feces of cases of pulmonary tuberculosis, when sputum has been swallowed. When tubercle bacilli are constantly found in the feces, and in large quantities, it points to the former condition almost to a certainty. They are detected by the methods employed in the examination of sputum.

Bacillus of Shiga in Tropical Dysentery and Summer Diarrhœa. The organisms have been found by Shiga and Flexner in children. Some of them resemble very closely *Bacillus coli communis.* Bacillus A is a bacillus with rounded ends, 3 μ to 4 μ by 0.7 μ.

Chemical Examination of the Feces.

The chemical examination of the feces has but slight clinical value, with the exception of the rather elaborate procedures necessary in quantitative fat estimations. Mucin and albumin are normally present; peptones in different diseases. (Von Jaksch.) Among the acids to be found are bile-acids, volatile and fatty acids, formic, acetic, butyric, and propionic acids; while phenol, indol, skatol, cholesterin, and fats, according to the same author, are always present. The demonstration of these substances will not aid in the diagnosis.

The normal *coloring-matter* of the feces is urobilin and pigments derived from the food. The presence of urobilin may be shown by the proper tests. As before stated, bile-pigment never occurs in the feces in health; it is present when there is catarrh of the small intestine. Blood-pigment is usually in the form of hæmatin. As might be expected, ptomaïns have been obtained from the feces of certain diseases caused by fungi.

CHAPTER XLV.

EXPLORATORY PUNCTURE.

The presence or absence of fluids in the natural cavities of the body, as the pericardium, the pleura, the abdomen, or the gall-bladder, must frequently be ascertained by means of puncture or aspiration. The fluid is secured at the same time by the puncture for examination. The fluid of tumors or cysts is likewise withdrawn to complete a diagnosis by determining its chemical, microscopical, or bacteriological character. Certain rules of procedure are necessary, and, as they are common to the method in whatsoever situation employed, may be considered in this section.

The Instruments. If it is the desire of the observer to determine the presence of fluid, an ordinary grooved needle may be used. If, however, fluid is to be obtained for examination, a syringe or aspirator must be used. An ordinary hypodermatic syringe, or the syringe of Pravaz may be used if the needles are sufficiently long. A special aspirator made

FIG. 268.

Exploring trocar.

for diagnosis by instrument-makers is the best. The needles are sufficiently long; the barrel large enough to hold sufficient fluid for any method of examination. If the diagnosis is to be followed by aspiration, the apparatus of Dieulafoy, or any equally perfect apparatus, may be used at once. An exploring trocar is used for the removal of pieces of solid tissue as from muscle or from tumors of the lung.

Preparation of Instruments. The instruments should be sterilized in a steam sterilizer or boiled. This does not apply to the needles alone, but every portion of the instrument should be cleansed, because, for instance, the contents of the barrel of the syringe pass through the needle. After sterilization they should be carried to the patient in sterilized test-tubes plugged with cotton-wool. When not in use, the needles should be kept in absolute alcohol and the syringe in carbolic acid solution, 1 : 20. Before using, the carbolic acid should be washed from the syringe and needle with boiling water; they are then to be sterilized as described. Unless the carbolic acid is removed from the syringe, its presence may serve as an antiseptic or disinfectant, and thus interfere with the culture-tests to which the material drawn is to be subjected.

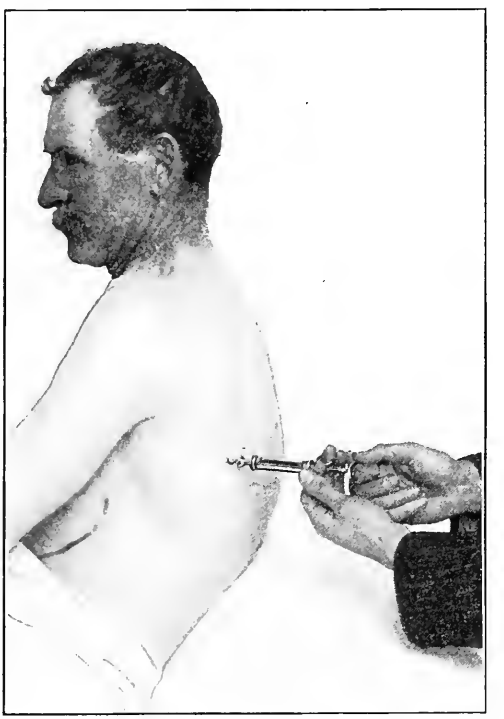
Preparation of Skin. The skin should first be cleansed with green-soap and water, then with alcohol and ether, then with a solution of car-

bolic acid (1 : 30) or of bichloride of mercury (1 : 1000). After thorough cleansing, the parts should be kept covered with a towel soaked in bichloride solution until the time of operation. At the time of puncture the surface should be anæsthetized by ethyl chloride, the rhigolene spray, by ice and salt, or in adults by the Schleich method of subcutaneous anæsthesia. Care must be taken, if the patient is aged or poorly nourished, or the skin œdematous, not to carry the freezing process too far on account of the danger of local gangrene.

The Point of Puncture. The points selected for aspiration depend upon the cavity to be explored or the situation of the cyst.

The Pleura. To withdraw fluid within the pleura, it is best to select a point for aspiration in one of the lower interspaces of the chest, because the fluid is more likely to accumulate in this position and because complete aspiration can there be performed if necessary. The sixth or

FIG. 269.



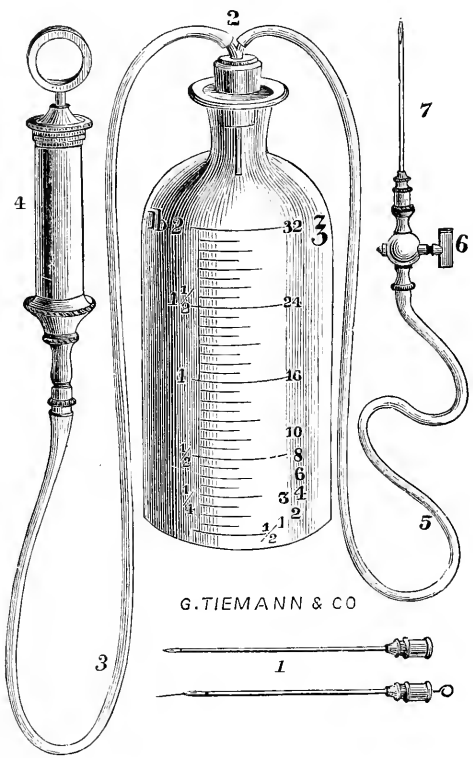
Aspiration of a pleural effusion.

seventh interspace in the anterior axillary line, or the eighth or ninth interspace in the posterior axillary or scapular line, may be selected. On the right side the upper interspace of the two should be chosen on account of the position of the liver. If the contents tend to point or break out at any particular spot on the surface of the chest, the puncture may be made in this area. In suspected loculated empyema or effusions,

the point of puncture should be at the site of greatest dulness and least fremitus.

The Pericardium. For aspiration of the pericardium three points of election have been recommended: first, the usual position of the apex-beat, in the fifth interspace, inside of the midclavicular line; second, the space between the ensiform cartilage and the left seventh cartilage, the point advised by Roberts; third, Rotch has tapped the fifth right interspace a number of times on the cadaver, and thinks that this situation is a proper one in the living subject. The writer has aspirated the pericardium in several instances inside of the normal position of the apex. Care must be taken to insert the needle slowly and with the point directed downward and toward the left axilla when this position is selected.

FIG. 270.

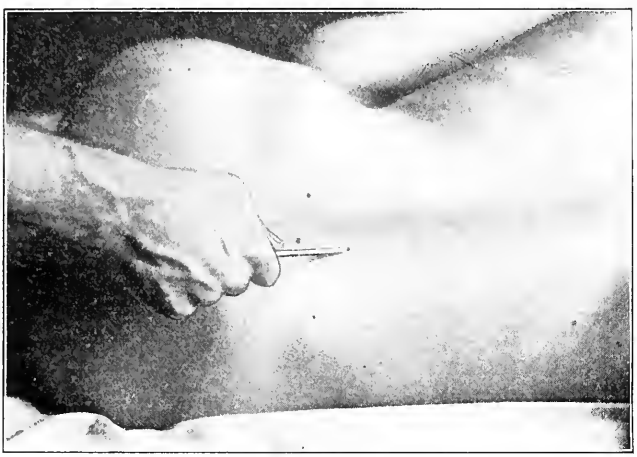


Aspirator.

The Abdomen. It should be remembered that no attempts at puncturing the abdomen should be made if pus is suspected, unless the surgeon is ready to perform laparotomy at once. Indeed, an exploratory operation is performed with so little detriment to the patient by modern surgeons that, on the whole, it should be advocated instead of puncture. There are times, however, when the latter must be resorted to. The writer has performed it in a number of instances without any

danger having ever arisen—always refusing to do so in cases in which pus was probably present in the peritoneal cavity, in the case of tumors or of organs the seat of suppuration. Explorations of this character are probably more feasible in connection with diseases of the *liver*. It does not appear to be harmful to insert needles into that organ, and valuable information is often gained thereby.

FIG. 271.



Lumbar puncture.

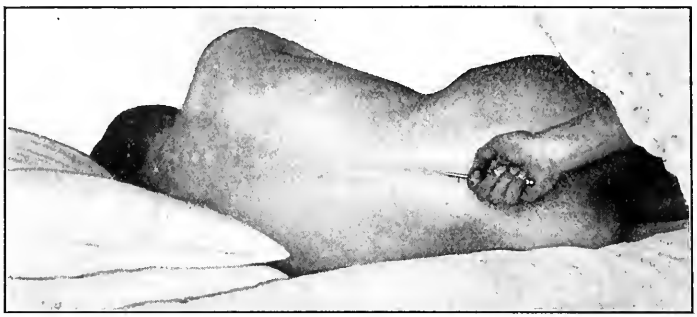
The median line should be selected for the puncture in aspiration of the *abdomen*, to determine the character of the peritoneal contents. The bladder must be emptied and a point midway between the umbilicus and pubes selected.

The Vertebral Canal. Spinal or Lumbar Puncture. Proposed by Quincke, the procedure has been carried out by many clinicians and has proved to be a means of establishing a diagnosis. Cerebral lesions are diagnosticated and intracranial pressure relieved because of the continuity of the spaces in the brain and the spinal canal. (See Cerebrospinal Meningitis.)

METHOD. The patient should lie on the right side, with the knees drawn up and the left shoulder turned forward. The puncture is made with an antitoxin needle or the needle of a large hypodermatic syringe, which may then be used to withdraw the fluid. The syringe itself may be removed and the fluid allowed to ooze through the needle drop by drop. A needle 4 cm. in length and 1 mm. in diameter is suitable for infants; a longer needle for children over ten years and for adults. Ordinarily the puncture is made between the third and fourth lumbar vertebræ to one side or the other of the median line. The point at which the needle enters the skin should be at the level of the lower end of the spinous process of the fourth lumbar vertebra, one-half inch to one side. The position of the fourth lumbar vertebra may be determined in one of three ways: (1) By counting carefully from the *vertebra prominens*—this

is difficult unless the patient is emaciated, and even then there may be trouble in distinguishing the individual spines of the dorsal vertebræ. The attachment of the twelfth rib can usually be made out, and the spine below it corresponds to the twelfth dorsal vertebra, and is almost immediately

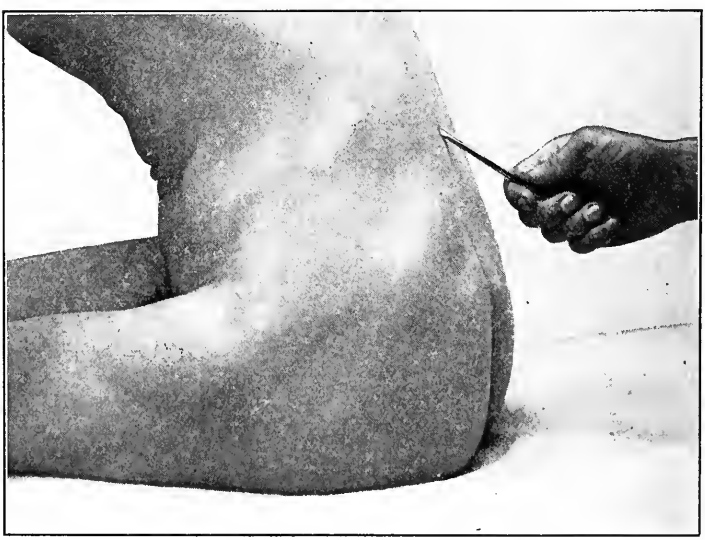
FIG. 272.



Lumbar puncture—introducing the trocar.

above the body of the first lumbar vertebra. (2) The method of Jacoby, which depends upon the fact that a line drawn through the crests of the ilia across the back intersects the spinous process of the fourth lumbar vertebra. (3) The spinous process of the fifth lumbar vertebra is con-

FIG. 273.



Lumbar puncture—introducing the trocar.

siderably more prominent than the spinous processes of the sacrum, and this peculiarity may be utilized as a check upon the other two methods. Practically in all cases all three methods should be employed. A mark on the skin may be made with an indelible pencil or with a soft lead

pencil. As the former is soluble in the alcohol and ether used in preparing the skin, the latter is preferable.

Technic. The technic is as follows: The patient may be either sitting up or lying upon the side. The former position can only be used in patients who are conscious and not suffering from severe disease, as, for example, in cases of brain tumor and chronic hydrocephalus. The patient sits preferably cross-wise upon a narrow operating-table; the body is bent well forward, separating the spines of the vertebra as much as possible. The latter position should be used if the patient is seriously ill. The patient should lie upon the side with the thighs flexed upon the abdomen and the thorax bent over the knees. The site of the puncture should be surrounded by sterile towels. The point of the needle is placed directly over the mark upon the skin; the needle is held at an angle of 45 degrees to the surface of the back, and inclined slightly toward the median line. It is best held—if unprovided with a handle—with the blunt end resting against the palm of the hand, and the shaft grasped between the index and the middle finger and thumb. If it is provided with a handle, the shaft should lie over the index finger, against which it is firmly pressed with the thumb, and the handle is steadied against the palm by the remaining three fingers. It should be thrust forward steadily and slowly, and not turned or twisted. In the great majority of instances, if the patient's back is well arched and the needle is thrust in the direction described, it will enter the spinal canal without difficulty. When it has been inserted between $2\frac{1}{2}$ and $3\frac{1}{2}$ inches, if it has not met with obstruction, it can be assumed that it is in the lumen of the canal and the mandril may be withdrawn. It should not be thrust in farther than this for fear of injuring the point of the needle against the anterior surface of the spinal canal. As soon as the mandril has been withdrawn, ordinarily two or three drops of clear or bloody fluid will flow from the tube. The metal plug should then be inserted into the end of the needle, the zero point on the glass brought as nearly as possible on a level with the spinal puncture, and the intraspinal pressure determined. The fluid can then be permitted to flow into prepared receptacles until it begins to drop very slowly; the needle should then be withdrawn and the opening sealed with a little cotton and collodion.

Certain *modifications* may be employed. A preliminary incision may be made through the skin at the site of the puncture. This should be sufficiently large to admit the needle without touching the skin on either side. It diminishes the liability to infection of the spinal canal by germs carried with the needle from the surface of the skin, but heals more slowly than the simple puncture wound. Infection practically never follows a simple puncture. The puncture may be made in any of the other interlumbar spaces where the difficulties are not much greater.

Of the *accidents* that may happen during the operation, the most important is the faulty direction of the needle, so that instead of finding the intervertebral space it strikes upon the arch of the lumbar vertebra. If this occurs, it is only necessary to withdraw the needle a short distance, alter its direction slightly, and thrust again. Usually the direction of the needle is too nearly perpendicular to the plane of the body, in which

case the butt should be slightly lowered. There is considerable variation in the distance between the interspaces in different individuals; in some they are so close together that it is impossible to insert a needle of ordinary size, and the operation becomes futile. If the cerebrospinal pressure is low, and the needle is very dull or has become dull by striking against a bone, it may be difficult to pierce the spinal membranes. When this occurs, of course no fluid is obtained. Sometimes if the needle is thrust in too forcibly and strikes against either the arch of the vertebra or the anterior wall of the spinal canal, it may be bent or broken. If the former occurs, the withdrawal of the needle may be very difficult; if the latter, the piece will probably remain within the spinal canal, where it may give rise to symptoms of irritation. If the needle, in piercing the canal, wounds one of the large veins, the spinal fluid will be bloody, and this may render its subsequent examination unsatisfactory. If it presses against one of the roots of the cauda equina, it may cause pain or twitching of the muscles of the extremities. Sometimes pseudomembrane with pus or gelatinous fluid fills the spinal canal, and hence no liquid is withdrawn. Adhesions may cause the fluid to be loculated.

Sometimes when the needle has been inserted and the spinal fluid has flowed freely, it may suddenly stop. This is due to some obstruction, usually a nerve-root, pressing against the end of the needle. It can usually be dislodged by reinserting the mandril. The cerebrospinal fluid should flow out rather slowly, preferably drop by drop. If it comes in a forcible stream, its flow should be checked either by pinching the rubber tube, or, if the patient is sitting up, having him lie down. If during the operation the patient should complain of headache, dizziness, or nausea, or if he groans, the operation should be interrupted at once and the patient placed either horizontal or with the lower portion of the body slightly elevated.

Data Obtained from Spinal Puncture. The normal pressure of the cerebrospinal fluid when the puncture is made below the conus terminalis and the patient is upright, is between 40 and 100 mm. If the patient is lying down, the pressure ranges between zero and 40 mm. If the pressure is above 150 mm. with the patient in the upright position, it is distinctly pathological. In some cases it may even reach 800 mm. Increase in the cerebrospinal pressure indicates irritation of the central nervous system, due to the presence of a tumor or to some toxic substance in the blood, as for example in uræmia and lead-poisoning, or to some inflammatory process in the cerebrospinal system, particularly meningitis.

Chemical Examination of the Cerebrospinal Fluid. Ordinarily the cerebrospinal fluid contains a mere trace of albumin. Under pathological conditions it may contain as much as 5 to 10 per cent., particularly in meningitis and in brain tumors. If the fluid is bloody, the percentage of albumin may be considerably increased without indicating a pathological change. It contains a substance—pyrocatechin—that reduces Fehling's solution. This ordinarily disappears a few hours after the fluid has been withdrawn. Sugar is said to be present in cases of brain tumor, but not in meningitis.

Histological Examination. The histological examination comprises a careful enumeration of the cells that may be present, and staining for various forms of bacteria. The fluid should be centrifugated, and the sediment spread upon cover-glasses and stained by the usual methods for blood and bacteria. If there is a considerable proportion of large endothelial cells, the rise in pressure is probably non-inflammatory. If the polymorphonuclear forms are in excess, acute cerebrospinal meningitis is probably present. If the mononuclear forms of leucocytes predominate, the infection is presumably tuberculous. A diagnosis based merely upon the cytological examination can not be considered certain. The discovery of bacteria is of more importance. Tubercle bacilli are sometimes found. The meningococcus can be recognized by its form, by the fact that it is intracellular, that several micro-organisms are found in a single cell, and that they stain with unequal intensity. The other forms of micro-organisms are usually found in the fluid. If there is reason to suspect an inflammatory process, cultures and inoculations should be made.

Other methods of obtaining the cerebrospinal fluid, such as puncture of the lateral ventricles of the brain through an opening in the skull, or tapping the fourth ventricle through the intervertebral space in the upper cervical region, are really surgical operations.

Chemical Examination. *Character of fluid.* The fluid is usually clear and limpid in tuberculous meningitis. In other forms of meningitis it is cloudy and turbid. It is purulent in leptomeningitis. Blood may be found in hemorrhage into the lateral ventricles.

Bacteriological Examinations. Cover-glass preparations are made of the fluid, and cultivations taken at once. Streptococci, staphylococci, the pneumococcus, and the meningococcus (*Diplococcus intracellularis*) may be detected in purulent meningitis. Tubercle bacilli have been found in tuberculous meningitis, especially after sedimentation. After the fluid has been twenty-four hours in a conical glass the fine clot which forms should be examined for bacilli. The absence of bacilli does not exclude tuberculosis. The positive result, however, is diagnostic.

Inoculation, as in a case by Lafleur, will cause tuberculosis in a guinea-pig, and is diagnostic. A clear fluid does not exclude purulent meningitis; usually, however, the fluid is purulent, turbid, or rich in leucocytes.

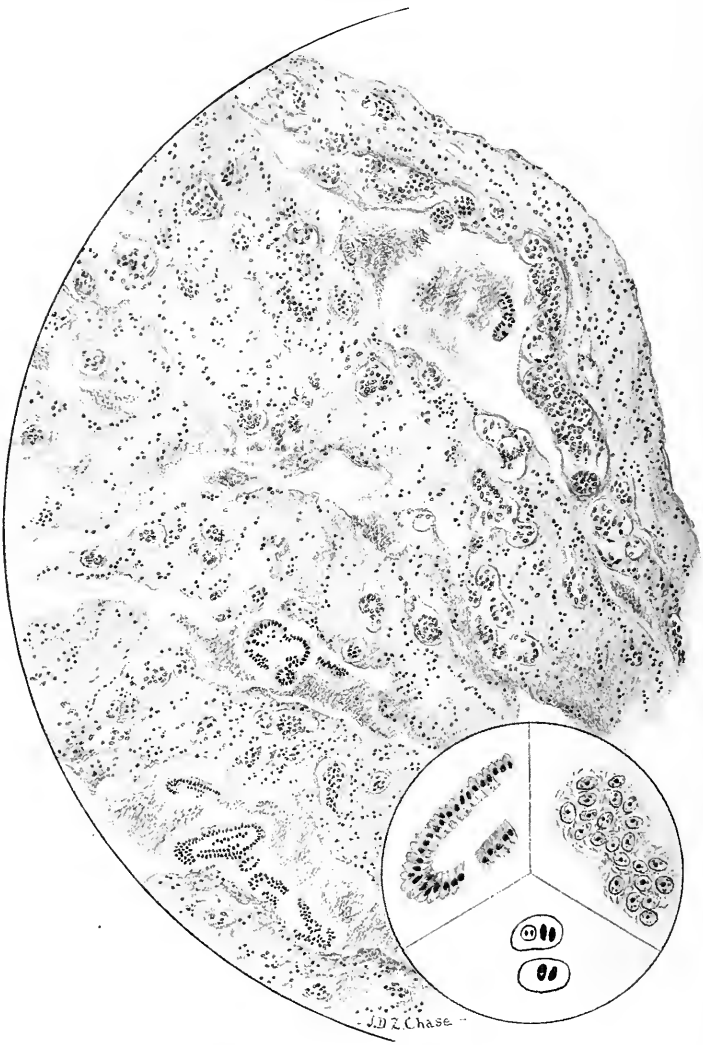
Cysts and tumors, with fluid contents, should be punctured over the point which presents externally, and where they are evidently in closest proximity to the external wall.

The Spleen. The spleen has been punctured for therapeutic and diagnostic purposes. If the organ is hard, as in chronic malaria, it may be done without danger; but if it is enlarged and soft, as in infectious diseases, such as typhoid fever, it is hardly justifiable to puncture it, because of the danger of subsequent rupture. Risk attends the puncture of other organs, as the kidney. The writer has seen a serious hemorrhage follow such puncture, and, of course, septic inflammation may arise. Exploratory operation is more suitable for determining its condition.

THE EXAMINATION OF FLUIDS AND DISCHARGES.

While the fluids to be examined can be obtained by exploratory puncture, it sometimes happens that they are discharged spontaneously, as in the case of an empyema.

FIG. 274.



Degenerated carcinomatous tissue removed during "paracentesis abdominis": 1 and 2, specimens of cells found in exudate from same case; 3, multinucleated cells and "cell inclusion" from case of rectal carcinoma.

The following general methods apply to the examination, in whatever way material is obtained. When derived from the natural cavities, the

fluids are known as exudates or transudates. Fluids are also obtained from cysts, but do not require different methods of examination.

The naked-eye appearances are first noted; then microscopical examination with and without staining is resorted to. Chemical examination is also required. Often culture-preparations and inoculations must be resorted to, as in the case of pus or of serous exudation.

The Exudates.

They may be composed of pus, seropus, gangrenous débris, blood, or pure serum or chyle. When pus, seropus, or putrid fluid is withdrawn, it implies absolutely an inflammatory origin. Blood and serum may be associated with inflammation, simple or infectious; but may also point to impediments in the general or lymphatic circulation. Blood or bloody serum is usually of tuberculous or cancerous origin. Its absence does not imply the absence of either disease. A chylous exudate is usually due to obstruction of the lymph-channels.

Purulent Exudates. Pus ranges in color from gray to greenish yellow. It is turbid, of high specific gravity, and alkaline. It varies in consistence. When standing after removal, it separates into two layers; the upper layer is light yellow and transparent, and the lower opaque. Pus may be mixed with blood, and is then reddish brown. (See Abscess of the Liver.) When it has undergone decomposition, it is thin, green, or brownish red, and of a penetrating odor.

Microscopical Examination. WHITE CORPUSCLES. If the specimen is fresh, the cells exhibit the movements that are common in leucocytes. If a solution of iodine and potassium iodide is added to them, they change to mahogany color. If the pus is old and the cells are dead, they are shrunken and granular. Enormous giant cells and cells loaded with fat are seen in pus.

RED CORPUSCLES. In fresh pus, red corpuscles are also seen along with blood-pigment or hæmatoidin crystals.

FREE FAT-GLOBULES and FAT-PARTICLES are present.

EPITHELIUM is rarely seen. In the pus from the pleural cavity, if cancer is present, the vacuolated *epithelial* and *endothelial* cells sometimes seen in cancer may be observed. (See case of Steele and Girvin.)

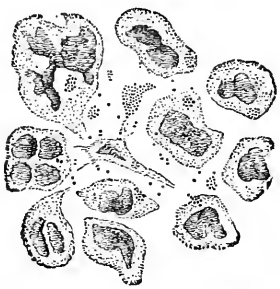
Bacteria. Micro-organisms are always detected with the aid of staining methods. (See Chapter XXXVIII., Bacteriological Diagnosis.) The micro-organisms are usually the determining cause of the suppuration. Suppuration, however, may be caused by chemical substances, although this is of rare clinical occurrence. Of the various bacteria found, the micrococci and bacilli are the most numerous. The commonest of these are *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes; Amœba dysenterica*, in abscess of the liver and secondary abscess of the pleura and lung. It was found in an abscess of the jaw by Flexner.

THE PYOGENIC BACTERIA. 1. *Staphylococcus Pyogenes Aureus.* This micro-organism is found in acute abscesses and boils, sometimes also in

infectious osteomyelitis and ulcerative endocarditis. In addition to other portals, it may enter the tissue through abrasions or the hair-follicles.

Morphology. In cover-glass preparations they appear as small, round bodies scattered among the pus-cells, rarely within them, single, in pairs or in clusters. They stain readily with the basic aniline dyes. (See Fig. 275.)

FIG. 275.



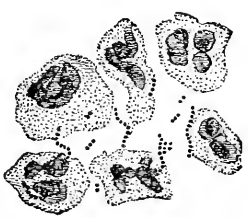
Pus with staphylococcus. × 800. (FLÜGGE.)

2. *Staphylococcus Pyogenes Albus.* It is also found in acute abscesses, but less often than the "aureus," and is less virulent. It is morphologically identical with the "aureus," but develops no pigment. The surface cultures are milky white, and the mass at the bottom of the liquefying gelatin is white.

3. *Staphylococcus epidermidis albus* (Welch) closely simulates *Staphylococcus pyogenes albus.* It is the most common micro-organism on the surface of the body, and is often present in parts of the epidermis too deep for disinfection, save by heat. It is supposed to be the usual cause of "stitch-abscess."

4. *Streptococcus Pyogenes.* It is found in acute abscesses, *erysipelas,* otitis media, puerperal metritis, *infectious endocarditis,* pseudodiphtheria, scarlatinal angina, and most purulent inflammations of a phlegmonous character. It is the organism most commonly found in inflammations having a spreading tendency.

FIG. 276.



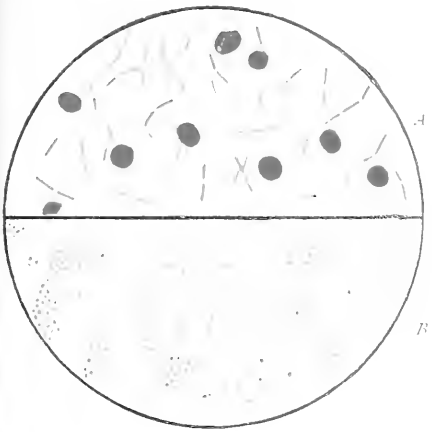
Streptococcus pyogenes in pus. × 800. (FLUGGE.)

Morphology. Cover-glass preparations show spherical cocci of varying sizes, which form chains of 4 to 20 elements, the chains often forming tangled masses. This organism is stained by the basic anilines or by Gram's method. (See Fig. 276.)

Inoculated, it causes erysipelatous or phlegmonous inflammation.

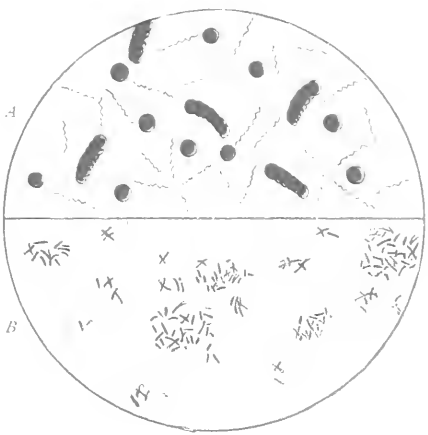
PLATE XIII-c.

FIG. 1.



A. Anthrax. *B.* Streptococcus and Staphylococcus.

FIG. 2.

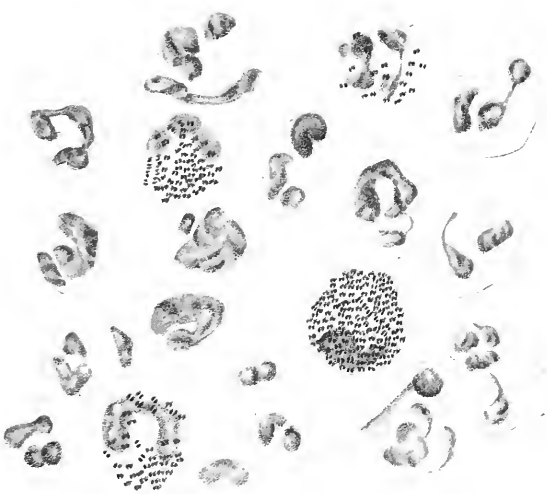


A. Recurrent Spirilla *B.* Leprosy.

FIG. 3.

Diphtheria Bacillus.

FIG. 4.



Gonococcus.

5. *The Tubercle Bacillus.* This is seen at times in pus removed from phthisical cavities, and the pus of abscesses, particularly about glands. It may be detected by methods of staining adopted in the examination of the sputum. Pus may be of tubercular origin, and the micro-organisms may not be detected by the usual microscopical methods. Its absence therefore does not imply the absence of tuberculosis. Culture-methods and inoculation should be resorted to, particularly the latter.

6. *The Bacillus of Syphilis.* The pus under these circumstances is usually derived from ulcers or inflammations, or from secretions about the vulva or prepuce. The actual relationship to syphilis has not been demonstrated.

Lustgarten's method is as follows: After immersion for twenty-four hours at the ordinary temperature in the gentian-violet fluid of Koch-Ehrlich, the cover-glass preparation is removed and washed for a few moments with absolute alcohol. It is then placed for ten seconds in a 1 per cent. or 2 per cent. solution of potassium permanganate; a watery solution of pure sulphurous acid is then poured over it, after which it is washed in water. If the preparation still shows its color, it must be reimmersed for a few seconds in the potash solution and then in the sulphurous acid, and again washed with water.

7. *Actinomyces.*

8. *The Bacillus of Glanders.*

9. *The Bacillus of Anthrax.*

10. *The Bacillus of Leprosy.*

11. *The Bacillus of Tetanus.*

12. *The Bacillus of Influenza.* (See Sputum.)

13. *Micrococcus Lanceolatus* or *Pneumococcus.* The pneumococcus is often found in the pus of empyema and pericarditis, whether from the pleural cavity or after it has burrowed from this situation. It occurs in cerebrospinal meningitis. It is easily detected by the usual staining methods (for which see Sputum).

14. *Bacillus Coli Communis.* This is found more commonly in infections within the abdominal cavity. (See Feces.)

15. *The Gonococcus.* It is constantly present in virulent gonorrhœal pus, usually within the pus-cell or attached to the surface of epithelial cells. *Morphology:* Micrococci, usually joined in pairs or groups of four flattened and separated, when stained, by an unstained intracellular space. Stains easily with aniline—not by Gram's method. No other coccus is of the same shape, nor present at the same time within the cells, except the meningococcus, which is of the same shape, likewise intracellular, and is decolorized by Gram's method. (See Plate XIII., C, Fig. 4.) It grows more abundantly and readily, however.

PROTOZOA IN THE PUS. Cercomonads have been observed in the pus obtained from an empyema, and are probably derived from the lungs. Flexner has found *Amœba dysenteriæ* in the pus from an abscess of the jaw. It is found in abscess of the liver and secondary abscess of the lung. (See Sputum and Feces.)

VERMES. Filaria have been found in abscess of the liver. The pus from a hydatid cyst contains membrane and hooklets.

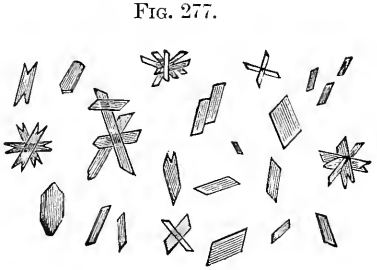
CRYSTALS. Crystals of *cholesterin* are found in the pus from cold abscesses, suppurating ovarian cysts, and fetid discharges. They are similar to the crystals described under sputum.

Hæmatoidin crystals indicate a previous hemorrhage; they are most frequent in suppurating hydatid cysts. (See Fig. 277.) *Fatty needles* are found in old pus and gangrenous exudates. (See Fig. 278.) *Triple phosphates* are frequently seen in pus, and are of the same appearance as the phosphates in the urine. The carbonates and phosphates are seen in fetid pus.

Chemical Examination of Pus. This does not yield any information of diagnostic value.

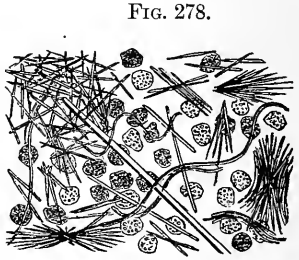
Serum-albumin, globulin, and peptone are detected by methods employed in the examination of urine. Fresh pus contains sugar. After being boiled with an equal weight of sodium sulphate and filtered, the filtrate is examined by the reagents used in the examination of urine for sugar. Pus also contains bile-pigments and biliary acids, cholesterin, and salts of sodium, and the fatty acids in jaundice. Von Jaksch has found acetone in pleural exudates.

FIG. 277.



Rhombic crystals of hæmin. (CHARLES.)

FIG. 278.



Pus from putrid empyema. (Eye-piece III., obj. 8, A. Reichert.) Shrunken leucocytes. Fat-crystals. (VON JAKSCH.)

Seropurulent Exudates. They resemble purulent discharges, chemically and morphologically. They point to antecedent inflammation.

Putrid Exudates. The exudates are brown or brownish green in color. The odor is penetrating and offensive. They are usually alkaline in reaction. On *microscopical examination* old leucocytes and crystals of fat, cholesterin, and hæmatoidin are seen; fission-fungi of various forms are also seen. (See Figs. 277 and 278.)

Hemorrhagic Exudates. Hemorrhagic exudates contain *red blood-corpuscles* and *hæmoglobin* in large amount. *Fatty endothelial cells* are found. Quincke states that when the glycogen-reaction is shown, if the fluid is from the pleura, carcinoma is probably present. A positive diagnosis depends upon the discovery of the epithelial cells (see page 691) which are seen in cases of cancer. Hemorrhagic exudates in the pleura are due most frequently to cancer, to tubercle, or to scurvy. To determine its exact nature as tubercle, inoculation and cultures are sometimes necessary.

Serous Exudates. The fluid is clear and light yellow or straw-

colored. On standing a white fibrinous clot is deposited. On *microscopical examination*, red blood-corpuscles, leucocytes, fatty globules, and endothelial cells are found. They may be bunched in groups or scattered about. The micro-organisms, if present, are detected with difficulty. If ulcerating tuberculosis of the pleura is present, the bacillus may be found, but tuberculous pleurisy may exist without ulceration, and hence the fluid does not contain the bacillus. Cholesterin crystals are found in old serum. On *chemical examination* the fluid contains more than 3 per cent. of serum-albumin and globulin; peptone is absent in pleural exudations; sugar in small amount and acetone are found. The specific gravity of the fluid is above 1018.

Ascites. Evidence of the nature of an ascitic fluid—that is, whether it is an exudate or a transudate—may be obtained from the specific gravity and the percentage of albumin that it contains. The transudate that occurs in cachectic conditions, such as grave anæmias, chronic nephritis, amyloid disease, carcinomatosis, etc., has a specific gravity of 1010 or less, and contains 1 per cent. or less of albumin. The transudate that occurs in obstructive conditions in the liver and the portal circulation, such as cirrhosis of the liver, pylephlebitis, etc., has a specific gravity of from 1010 to 1014, and contains from 1 to 3 per cent. of albumin. The exudate that occurs in local inflammatory conditions of the peritoneum, tuberculous and cancerous peritonitis, etc., has a specific gravity of from 1017 to 1020 or higher, and contains from 4 to 6 per cent. of albumin. The fluid contained within an ovarian cyst has a specific gravity, as a rule, of 1025 or higher.

The percentage of albumin in the ascitic fluid may be calculated according to the formula of Reuss, which is said to be subject to an error of less than 0.25 per cent. According to this formula, the percentage of albumin equals three-eighths of the last two figures of the specific gravity (the specific gravity minus 1000), minus 2.8. Thus, should the specific gravity of the ascitic fluid be 1016, the percentage of albumin may be calculated, as follows: $\frac{3}{8}$ of 16 equals 6; 6 minus 2.8 equals 3.2, which is the percentage of albumin.

Chylous Exudates. True chyle is found in fluids of low specific gravity. Such an effusion is rich in fat and is due to leakage of lymphatics into the peritoneal cavity. It is known as a chylous effusion. Chyliform effusion is a term applied to the second variety of effusions mentioned in this section. The fluid has the property of chyle. Sometimes in peritoneal exudation, particularly if the patient has been upon a milk diet, the fluid contains fatty matter, which gives it a milky appearance. The same character of fluid is seen in obstruction of the thoracic duct.

Special Effusions. **Effusions into the Pleura.** The effusion may be serous, serosanguinolent, bloody, or purulent. It is of the greatest importance to distinguish the various forms of infection. Bacteriological examination is necessary. In purulent exudation, if micro-organisms are absent (staphylococcus and streptococcus), it is probably tuberculous; serofibrinous exudations are usually free from fungi. When *Micrococcus lanceolatus* is found, it is of favorable prognostic omen.

To distinguish the *effusion of inflammation* from that of *transudation* (obstruction) the specific gravity is suggestive. In the inflammatory effusions the specific gravity is high; they also contain a large amount of fibrin and more than 3 per cent. of albumin.

Transudates.

This class of fluids is serous, bloody, or chylous. The specific gravity is lower than in inflammatory effusion. The color is light and the reaction usually alkaline. On microscopical examination but little is found. In pleuritic effusions there may be considerable endothelium, which, if mixed with blood, may be due to carcinoma. Serum contains albumin and sugar, the former in great excess. Peptone is always absent. The fluid coagulates with difficulty on boiling.

Runeberg[1] lays stress upon the diagnostic importance of the amount of albumin in pathological transudates and exudates. His experience warrants the following statements:

1. Inflammatory processes, 4 to 6 per cent. of albumin.
2. Venous stasis, 1 to 3 per cent. of albumin.
3. Marked hydræmic conditions, as in amyloid degeneration or nephritis, 0.1 to 0.5 per cent.
4. Combination of two or three of the above causes, 0.2 to 6 per cent.

In old transudates due to venous stasis, even without inflammatory complications, the percentage of albumin may be high.

Contents of Cysts.

In aspiration of the abdomen and of the pleura, cysts are sometimes evacuated, the nature of which is often determined by an examination of the fluid. It is within the province of this work to discuss hydatid cysts, pancreatic cysts, and the cystic kidney. As tumors of the ovary so frequently resemble tumors in other situations, it is well also to discuss in this section the nature of the fluid withdrawn from them.

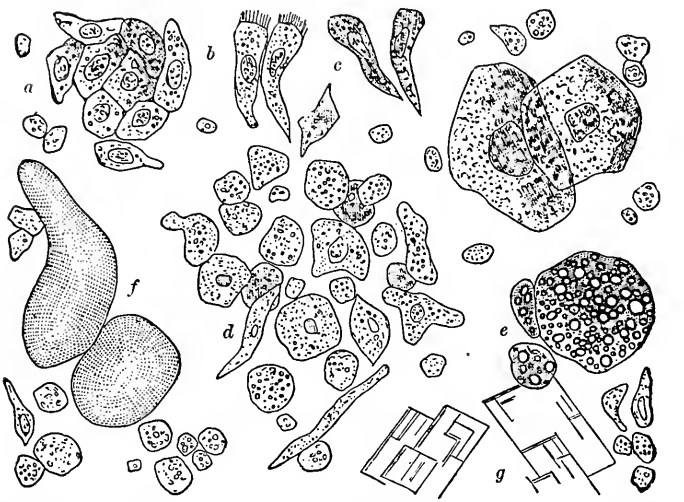
Hydatid Cysts. The fluid of hydatid cysts is clear, alkaline, and of a specific gravity of 1010. It contains sodium chloride in excess, grape-sugar in small amount, and very little if any albumin. On microscopical examination hooklets are found, as in the sputum from hydatid cyst of the lung, as well as portions of membrane. The membrane is recognized by its peculiar transverse striation and the granular appearance of its inner surface. The heads or scolices are sometimes found. Two circles of hooklets and four disks on the anterior aspect cross the head, which is separated from the posterior part by an annular constriction. (See Sputum and Feces.) If suppuration has taken place, the original nature of the cyst can not be made out unless hooklets are found. After the fluid has been standing in a conical glass vessel the bodies may be found in the sediment.

Ovarian Cysts. The fluid from an ovarian cyst is of high specific gravity, 1026, of alkaline reaction, contains but a small amount of

[1] Runeberg, J. W., "On the Diagnostic Importance of the Amount of Albumin in Pathological Transudates and Exudates." Berliner klin. Wochenschrift, 1897, No. 33.

albumin, and does not coagulate. On microscopical examination various forms of epithelial cells are seen, colloid bodies, and cholesterin crystals. If hemorrhage has taken place in the cyst, the color of the fluid is correspondingly changed, and besides the squamous, columnar, and ciliated varieties, some epithelium in the stage of fatty degeneration and red and white blood-corpuscles are seen. In colloid cysts the usual concretions are found. (See Fig. 279.)

FIG. 279.



Contents of an ovarian cyst. (Eye-piece III., obj. 8, A. Reichert.) *a*, squamous epithelial cells; *b*, ciliated epithelial cells; *c*, columnar epithelial cells; *d*, various forms of epithelial cells; *e*, fatty squamous epithelial cells; *f*, colloid bodies; *g*, cholesterin crystals. (VON JAKSCH.)

In *dermoid* cysts, in addition to the above, squamous epithelium, hairs, and fatty, hæmatoidin, and cholesterin crystals are detected. *Ovarian fluid* contains albumin and methæmoglobin, or paralbumin. The latter is detected by mixing a portion of the fluid with three times its bulk of alcohol. It is then allowed to stand for twenty-four hours, when it is filtered. The precipitate is removed and suspended in water. After filtering, the filtrate is seen to be opalescent, and is tested as follows:

1. On boiling no precipitate is formed, but the fluid becomes turbid.
2. There is no change with acetic acid alone.
3. The fluid becomes thick and of a yellowish tint when treated with acetic acid and potassium ferrocyanide.
4. There is a change to a violet color when treated with concentrated sulphuric and acetic acids.

Some observers differ from the above statement in their description of the fluid of an ovarian cyst; all agree as to the large number of cell-elements. At one time it was thought that the fluid contained a special cell, but this view has been abandoned. In rare cases the specific gravity may be lower than that of the fluid of ordinary ascites. A fluid of low specific gravity, with a small amount of albumin, is said to be characteristic of a cyst of the broad ligament.

Cystic Kidney. The fluid from a cystic kidney can be recognized by

the properties it derives from the renal secretion. Urea and uric acid in large amounts point to its true source. Renal epithelium is of the greatest diagnostic value. (See Urine.) If epithelium from the urinary tubules can be detected after the fluid has settled, the diagnosis is absolute. (See Hydronephrosis.) It must not be forgotten that both urea and uric acid may be found in other cysts, as in those of the ovary, if they communicate with the urinary tract.

Pancreatic Cysts. The fluid from cysts of the pancreas is of a specific gravity of 1012, but may be as high as 1028. It contains cholesterin crystals in abundance, and blood or pigment. Serum-albumin is present, but metalbumin is not found. Three diastatic ferments are present:

1. If on examination for sugar the latter is found to be a maltose, its presence is of diagnostic significance.

2. The most pronounced property of pancreatic fluid, and that by which we are enabled to distinguish it from other fluids, is the power of digesting albumin without the presence of an acid.

Boas[1] developed the method of examination. The fluid is to be added to milk. After the casein is precipitated the biuret test is applied, as follows: Heat the substance with caustic potash and add, drop by drop, a 10 per cent. solution of sulphate of copper. If digested albumin is present, the fluid assumes a reddish-violet color. No other cystic fluid can dissolve albumin in alkaline solution.

It is not necessary that albumin or fibrin should be employed in performing this test, as it is sufficient to add milk to the secretion; when in such cases the casein of the milk is precipitated, and the biuret test is applied to the resulting filtrate, and the test compared with a control-milk from which the casein has been removed (this can be done by adding very dilute acetic acid with constant stirring), the digestive property of the liquid under examination may be with certainty determined. The peptone would not be precipitated with the albumin, and as all albumins give the same reaction as peptone with the biuret test, the albumin should be removed before applying the test. It is removed from the filtrate by a saturated solution of ammonium sulphate. Then test the resulting filtrate with the biuret test, and compare with the control test as above.

3. The pancreatic fluid also emulsifies fats. In large cysts, however, particularly if of long standing, the physiological properties of the pancreatic juice are sometimes wanting.[2] In the case referred to by Boas and reported by Karewski, the protracted duration of the cyst modified the character of the fluid, and hence rendered its nature doubtful. Moreover, in the exploratory puncture the stomach was penetrated. For two reasons the author advises against exploratory puncture. First, the age of the cyst is not known, hence an analysis would be misleading. Second,

[1] Deutsche med. Wochenschr., 1890, Bd. xvi., S. 1095.

[2] In a case operated on by Penrose the analysis of the fluid was as follows: Sp. gr. 1025; reaction slightly alkaline; serum-albumin; no metalbumin; diastatic ferment absent; maltose absent. By Boas' method, power to digest albumin appeared to be great; but when the albumin remaining in the filtrate was removed from the pancreatic fluid, it failed to show that peptone was formed. The method, therefore, appears to be fallacious in this class of cases. The cyst was old, and the fluid no doubt had lost its physiological properties. Cholesterin was present in enormous amount; tyrosin crystals were scarce.

the danger of puncturing other organs is too great. Exploratory laparotomy is preferable.

Cyto-diagnosis. An endeavor has been made to associate certain cells of the blood and tissues found in exudates with certain clinical conditions. Wedal and Ravant have given the most systematic information. The result of their investigations showed that :

I. Tuberculous Affections. Pleurisies and meningitis were associated regularly with an increase of the mononuclear elements of the blood in the exudate—lymphocytosis up to 65 and 100 per cent.

II. Acute Infections. Streptococcus and pneumococcus pleurisies, etc., are accompanied by an increase of the polymorphonuclear blood elements in the exudates.

III. Mechanical Disturbances. The exudate most frequently contains the cells of the serous membrane, endothelial cells either simple in form or polynuclear and vacuolated.

There has been abundant confirmation of the earlier findings, and one may say that a lymphocytic exudate is always suggestive of a primary tuberculous affection. The multinuclear elements may be slightly increased in the first few days, or if secondary infections ensue, or if a tuberculous process is of the nature of an extension from adjacent parts, as pyopneumothorax, but in the end the lymphocytes will predominate.

Quincke, Eichhorst, and others have endeavored to diagnosticate malignant growths from the character of the cells in exudates or secretions. This has been readily done when tumor particles have been present, but can not be relied upon in all cases. The presence of fatty granular cells giving the glycogen reaction has been wrongly considered suggestive. The presence of numerous cells showing mixtures, typical and atypical, is frequently seen in exudates due to malignant irritation and is by some considered diagnostic. If the character of the cells is such that they can be differentiated from endothelial and other cells, a conclusion may be reached, and it is to be noted that the endothelial cells present as a result of simple irritation, only rarely show mitotic figures; they are also, as a rule, larger than other cellular elements. Such cells with mitotic figures may, however, be scant or absent in malignant exudates, and cells showing amitotic division of nucleus may be the only feature.

Infectious Exudates. In one of my cases of ascites studied by Gwyn, small round cells, single or in bunches (suggestive of carcinoma), led to a careful examination of the fluid. No mitoses were seen, but the further presence of regular cylindrical cells in a glandular arrangement, resembling a Lieberkühn crypt, and pieces of tissue enabled me to diagnosticate an intestinal carcinoma. Steele was so fortunate as to make a diagnosis of pleural carcinoma by finding cells in the exudate.

Multinuclear cells, cells with mitotic figures, and with inclusions (Russel's bodies, cancer parasites, etc.), were observed in the stools of a patient who on proctoscopic examination was shown to have a rectal cancer. The sputum of a case with obscure pulmonary symptoms was found to consist so largely of small round mononucleated cells that a suspicion of a new growth was entertained. Autopsy revealed a sarcoma of the lung.

PART II.

SPECIAL DIAGNOSIS.

CHAPTER I.

THE INFECTIONS.

INFECTIONS RECOGNIZED BY THEIR CLINICAL COURSE.

This group includes most of the contagious and epidemic eruptive fevers. Their recognition is largely based on the duration of the period of incubation, and upon the mode of onset and clinical course of the respective infection. They are: *typhus fever*, *smallpox*, *varicella*, *scarlet fever*, *measles*, and *rubella*. They are also known as the eruptive fevers, and upon the character of the respective eruption, which bears a definite symptomatic relation to the infection, the diagnosis is, in great part, based.

Typhus Fever.

Typhus fever is an acute contagious disease, occasionally occurring sporadically, and often becoming epidemic in the presence of destitution, filth, over-crowding, and poor ventilation. It is characterized by abrupt onset with chill or with chilliness, a *rapid rise* of temperature, lassitude, headache, and pains in the back and limbs. By the end of the first day the temperature is 104° to 105° F. On the fourth or fifth day a peculiar spotted eruption appears, which at first is macular and subsequently petechial. It is further characterized by adynamia or ataxia, low muttering delirium, a suffused, heavy, drunken expression of countenance, by the absence of local lesions, and by a crisis which occurs on or about the fourteenth day.

Typhus fever is variously known as *ship fever*, *jail fever*, and *camp fever*.

Incubation. The period of incubation is usually about twelve days; it may be five or eight days, or even a shorter time, depending upon the virulence of the poison and the susceptibility of the patient. Malaise may precede by a day or two the onset of the disease.

Invasion. Invasion is characterized by headache, faintness, vertigo, chilliness, or a distinct rigor, pains in the back and thighs, loss of appetite, nausea, constipation, and extreme weakness. The prostration is sometimes so great as to compel the patient to go to bed at once. The

pulse is frequent, 100 or 140, and in grave cases shows a marked tendency to become small, soft, and feeble. The patient is restless and sleepless, and is annoyed by tinnitus. The *expression* of the flushed face is listless and dull.

The Eruption. About the fourth or fifth day the typhus eruption begins to appear. It consists at first of dull-red spots of irregular size and shape. They are most numerous on the covered parts. Moore[1] says they are detected first near the axillæ and on the wrists, then on the sides of the abdomen, afterward on the chest, back, shoulders, thighs, and arms. The skin is also mottled by another crop of maculæ under the skin ("mulberry rash").

When the disease is fully developed, the face is flushed, the conjunctivæ red, the pupils contracted, so as to resemble pin-holes ("ferrety eye"), the tongue dry and brown, the teeth covered with sordes, the skin dry, hot, and stinging to the touch. The patient lies upon his back oblivious to his surroundings. Headache has given place to delirium, which may be wild and fierce, but is more commonly low and muttering. There are marked ataxic symptoms—subsultus tendinum, tremors, and picking at the bedclothes. Incontinence of urine and feces sometimes occurs. The breathing is frequent, shallow, and noisy, and the pulse more rapid, soft, and feeble. The macular rash now becomes petechial. The patient is in a typical "typhoid state." The stupor may gradually clear up, or, on the other hand, deepen into coma; or the patient may die from progressive weakening of the heart, with or without pulmonary complications.

In the majority of favorable cases, on or about the fourteenth day, the first sign of recovery is a sound sleep, from which the patient awakes refreshed and rational. The temperature falls with great rapidity and the pulse and respiration improve—a typical crisis has occurred.

Certain *objective phenomena* of the disease require special mention. The eruption is more copious in severe than in mild cases. A dull and livid color is a grave sign. Purpura and hemorrhages are sometimes met with in severe cases. The eruption does not occur in successive crops.

The patient seems to be surrounded by a vapor of a pungent, musty *odor* which is peculiar.

The *heart* early shows the effect of the poison. The impulse is diminished, and the first sound is less distinct. In grave cases, with threatening heart-failure, the sounds are feeble and distant, the impulse imperceptible.

The *pulse* is usually very much more frequent than normal, but may be abnormally slow (50 and even 30 per minute); this is sometimes a bad sign.

The *weak heart* and prostrate position of the patient favor congestion and œdema of the lungs. This condition is common.

Digestive symptoms have already been referred to. Vomiting, tympanites, and diarrhœa are rare, and still more so is intestinal hemorrhage.

The *urine* is scanty and high-colored. Slight albuminuria is common,

[1] Eruptive and Continued Fevers, by J. W. Moore, Dublin, 1892.

and a few casts are found, but distinct nephritis is unusual. Convulsions, when they occur after the first week, are almost always uræmic and almost invariably fatal. They may be due to retention of the urine, as recorded by Stokes and Corrigan.

Duration. The duration of the disease is from six to fifteen days; the average period is twelve to fourteen days. An abortive form is met with in some epidemics, the disease being of a mild type and subsiding at the end of a week. In some cases so large a dose of the poison is absorbed by the patient that he is stricken down in a few hours or a few days. To this form the name "blasting typhus" has been appropriately given. The most important complications are hyperpyrexia, laryngitis, bronchitis, and congestion of the lungs, extreme ataxia or profound adynamia, nephritis, heart-failure, and parotitis or other inflammatory glandular swellings.

Laryngitis with œdema is a very rare but very dangerous complication.

Diagnosis. *H. D.* Hygienic conditions; epidemic exposure.

S. D. Clinical course.

O. D. Eye; eruption; range of temperature.

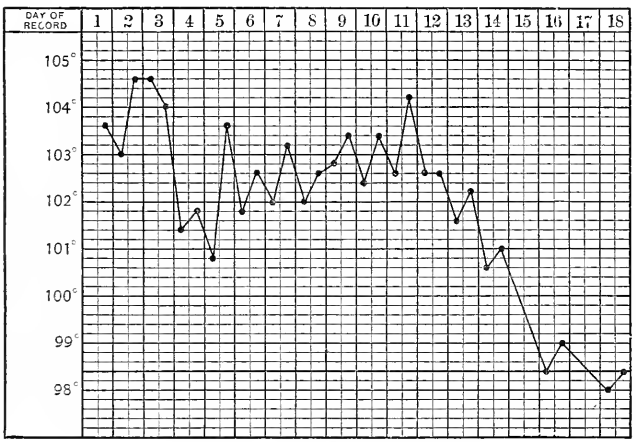
P. D. Negative.

L. D. Negative.

D. D. From cerebrospinal meningitis, uræmia, pneumonia, typhoid fever.

Cerebrospinal fever is distinguished from typhus fever by greater intensity of the headache, by retraction of the head and hyperæsthesia, by greater liability to vomiting, by the presence of leucocytosis, by the result of lumbar puncture, and by the absence of the macular petechial

FIG. 280.



Typhus fever—typical. (DOTY.)

eruption and the drunken, besotted aspect of typhus fever. In cerebrospinal fever the patient suffers with photophobia, and is liable to local palsies of the eye-muscles (strabismus) and to general convulsions. Convulsions do not occur in typhus except from a complicating nephritis or retention of urine.

Uræmia is distinguisned from typhus by the preceding history, by the absence of high temperature, and by the presence of œdema of the face or extremities, a history of vomiting or diarrhœa preceding the stupor. The condition of the urine and the absence of eruption are the final tests.

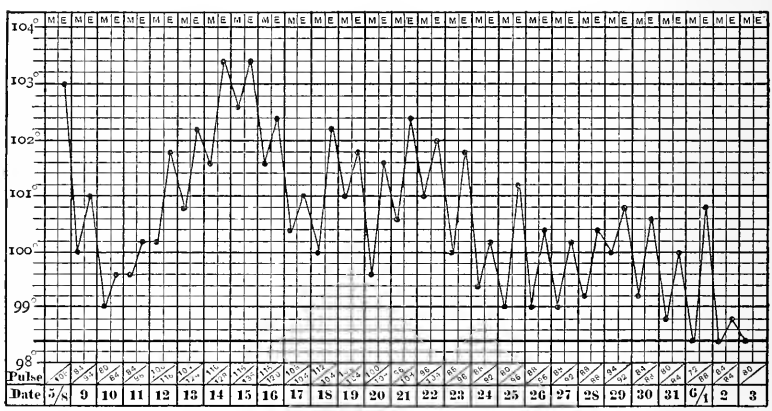
Pneumonia is distinguished by the frequent respiration and relatively slower pulse, and by the local physical signs and absence of eruption.

Typhoid fever is distinguished by its slow onset and marked abdominal symptoms. The eruption of typhus is petechial and comes out on the fourth or fifth day; that of typhoid fever consists of rose spots and appears on the seventh or eighth day. In typhus fever the severe initial chill, the sudden onset, the greater prostration, and the earlier appearance of cerebral symptoms are helpful in distinguishing it from typhoid fever. The serum reaction must be employed.

Variola.

The temperature in variola, or smallpox, pursues a definite course, which renders it of value in the diagnosis. Its sudden rise to an unusual height without local inflammation but with severe backache is significant. Its fall with the appearance of the eruption, followed in two or three days by a secondary rise, is very characteristic.

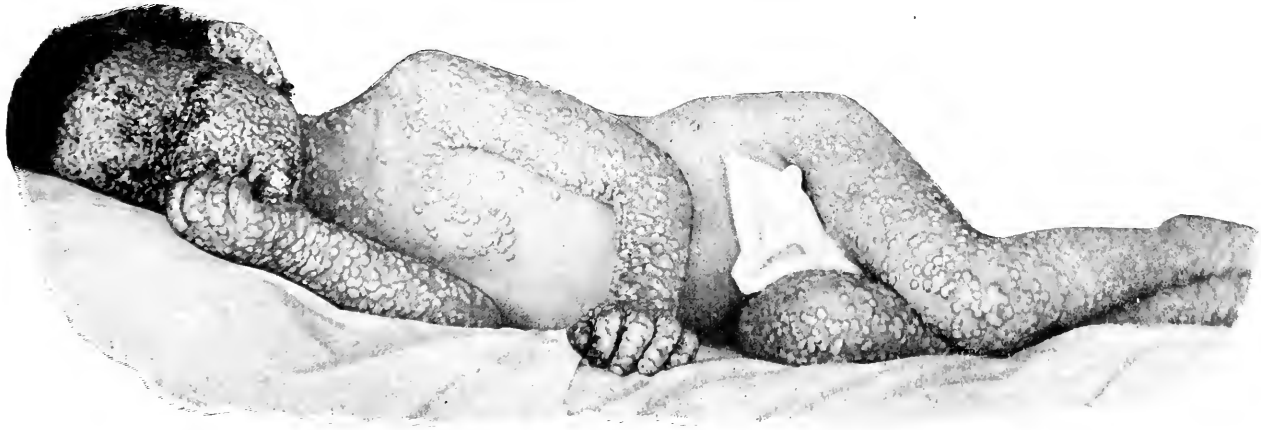
Fig. 281.



Temperature in smallpox. Adult: mild case. (Original.)

Variola, or smallpox, is a specific, infectious, and contagious fever, beginning abruptly with chill, high temperature, headache, vomiting, sweating, and intense pain in the back. On the second or third day of the disease a characteristic shot-like, papular eruption appears, the papules rapidly developing first into vesicles and then into pustules; with the appearance of the rash the temperature falls, but rises again toward the end of the week in the pustular stage (fever of maturation or suppuration). The contents of the pustules are discharged, crusts form and are cast off about the eighteenth day. The disease may be accompanied by a

PLATE XIV.



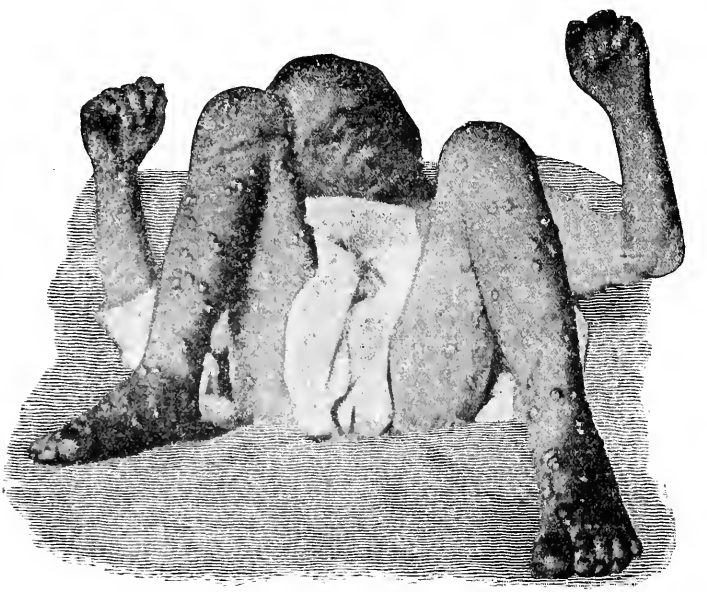
Variola on the Seventh Day of Eruption. Confluent on Face and Semi-confluent on Extremities.

number of complications, particularly hemorrhages into the skin (purpuric smallpox) and from the mucous membranes (hemorrhagic smallpox), both forms being popularly called "black smallpox." For convenience of description the disease may be divided into four stages: (1) incubation, (2) invasion, (3) eruption, (4) desquamation.

Incubation. This stage lasts from ten to fourteen days, and is usually unaccompanied by any symptoms except, toward its close, by malaise.

Invasion. The invasion is abrupt, and is marked by chilliness or a distinct rigor, headache, severe pain in the lumbar regions, and sometimes delirium or convulsions, especially in children. The most prominent symptoms are the excruciating headache and backache. The temperature usually rises rapidly to 104° F. or higher in the first twenty-four or forty-eight hours. (See Fig. 281.) Headache and backache continue; there are pain in the epigastrium, a coated tongue, loss of appetite, nausea or vomiting, constipation, and copious perspiration. Prostration is extreme. Erythematous eruptions are not uncommon, especially on the inner surfaces of the legs and thighs. Petechiæ are found in Simon's triangle, the base of which is at the umbilicus and apex at the knees.

FIG. 282.



Discrete variola on the sixth day of eruption. (WELCH.)

The stage of *invasion* lasts generally three days; but it may be shortened to two in very severe cases or lengthened to four in very mild ones, and in complicated and hemorrhagic cases it merges into the stage of eruption. (See Plate XIV.)

Eruption. The characteristic eruption of smallpox appears first as minute specks resembling flea-bites. These in two or three days develop into small papules which feel like shot under the skin. In a day or two

more the papules become vesicles, at first containing a clear fluid, which, however, rapidly becomes turbid; they are umbilicated. In the course of another day or two the vesicles have become pustules and are globular in shape. The period of ripening or maturation, when pustulation is at its height, lasts about three days; it is characterized by a marked secondary fever, the temperature rising as high as, or higher than, in the onset of the disease. The pustules now begin to dry up (desiccation) and form dry scales or scabs, which are cast off toward the end of the third week of the disease (eighteenth day); when the pustules have been sufficiently deep to involve the true skin, characteristic scars, called pits, are left.

The *eruption* appears on the forehead, along the margin of the hair, and in the scalp, then over the rest of the face, especially about the nose and lips, subsequently progressing over the rest of the body from above downward. The eruption is most abundant upon the face and hands, often being confluent here when discrete elsewhere. The face may appear horribly swollen, bloated, and disfigured, and both face and hands are extremely painful from the great distention and the pustules, which are really small dermal abscesses.

Varieties. Three varieties of variola, depending upon the number and disposition of the pocks and upon the presence of complications, are recognized: (1) discrete; (2) confluent; (3) malignant.

In **discrete** variola the pocks are not numerous, and are separated from each other by intervening healthy skin.

In **confluent** smallpox the pustules are close-set, occupy almost the whole body, and coalesce, so that the face looks as though covered with a black, rough mask; the mucous membranes are also covered. The symptoms of the invasion are intensified, and the eruption may appear before the third day. Patients are liable to suffer with profuse salivation, uncontrollable vomiting or diarrhœa (especially children), and with delirium, which is often violent and destructive. The face is dreadfully swollen and the eyelids may slough; the feet and limbs also may be swollen and painful. There may also be severe bronchitis and pneumonia, abscesses, extensive sloughing, and a pyæmic condition.

Malignant, or "black smallpox," is a form in which the blood is so altered that hemorrhages into the skin or from the mucous membranes occur. In the former case there are petechiæ and ecchymoses upon the skin; in the latter more or less profuse hemorrhages occur from the womb, kidney, bowels, lungs, and stomach. The mind of the patient remains clear and he is conscious of his peril. The eruption is delayed or does not occur at all.

Varioloid is a mild form of smallpox occurring in a person partially protected by vaccination, or in a person who, from other causes, does not possess the average susceptibility. It is characterized, apart from its mildness, by great irregularity in the development of the symptoms. The initial symptoms, as a rule, are as severe as in ordinary smallpox. Prodromal eruptions, especially the erythematous, are very common. The eruption may appear first on the face, or on the chest and trunk first, and later upon the face. The fever subsides with its appearance. The

eruption passes from the papular to the vesicular stage, as in ordinary smallpox; but here the process, as a rule, ceases, the vesicle drying up on the fifth or sixth day of the eruption. If pustules form, they do not reach their full development. The eruption is always discrete. There is usually no secondary fever.

Diagnosis. *H. D.* Exposure; epidemic; vaccinia; or previous attack.
S. D. Backache; headache; vomiting.
O. D. Temperature course; eruption.
P. D. Negative.
L. D. Negative.
D. D. Pneumonia; typhus fever; measles; varicella; syphilis.

When fully developed, smallpox will not be mistaken for any other disorder. In the initial stage, however, there may be doubt whether the disease will prove to be pneumonia, cerebrospinal meningitis, or typhus. If the patient has been exposed to smallpox and is unprotected by vaccination, and he is suddenly seized with a chill, high temperature, and excruciating pain in the lumbar region, there is great probability in favor of smallpox. If the patient has complained of headache, pains in the ankles and other joints, and is seized with a severe rigor, explosive vomiting, and great weakness of the limbs, the chances favor meningitis in the absence of known exposure to smallpox.

In *pneumonia*, vomiting, chill, and high temperature succeed each other, but excruciating backache is wanting, and, on the other hand, the respiration is increased out of proportion to the pulse, and even in this early stage there may be cough and roughening of the respiratory murmur on one side.

Typhus fever begins abruptly with chill and high temperature; but the eruption which comes out on the fourth or fifth day is first macular and later petechial, the temperature does not fall with the appearance of the eruption, the aspect of the patient is drunken and stuporous, the conjunctivæ are injected, the eye ferrety, the skin dry, hot, and biting to the touch (*calor mordax*).

Measles. In the papular stage of the eruption smallpox may be mistaken for measles; but the red, swollen, blear-eyed, photophobic little patient with measles, with the characteristic coryza and obstinate cough, presents a very different appearance from that seen in variola. Moreover, the eruption of measles is relatively flat, smooth, and velvety; that of smallpox is acuminate, hard, and shot-like. The temperature in smallpox falls as the eruption appears; that of measles remains high and even increases. The papules of measles do not develop into vesicles.

Chickenpox. In the vesicular stage varioloid may be mistaken for chickenpox. In the latter the eruption is practically vesicular from the start, occurs without prodromata, appears first upon the chest and neck, later upon the face and scalp, is usually very scanty, and rarely becomes umbilicated or pustular. There are, however, severe forms of varicella, in which fever, restlessness, and cough precede the appearance of the rash, which is copious, some of the vesicles being inflamed at the base, some umbilicated, and some with purulent contents. These cases are most common in scrofulous children whose hygienic surroundings are

bad. In such cases the diagnosis can not be made from the eruption. A consideration of the following points must decide: (1) History of exposure to varicella on the one hand or smallpox on the other. (2) The presence or absence of effective vaccination or of scars of antecedent varicella. (3) The age of the patient; smallpox occurs at all ages, varicella only in childhood. (4) The discovery among neighboring children of varicella or varioloid. (5) The rapid evolution of a varicella pock.

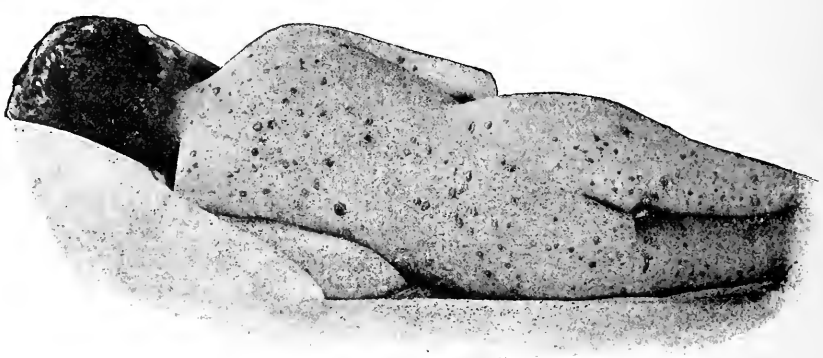
Varicella.

Varicella is one of the infections of childhood in which the fever is very mild and may not attract attention. It is an acute, specific, infectious fever, occurring almost exclusively in children, and characterized by the appearance, in successive crops, of colorless or pearly vesicles which dry up and are shed in from two to five days. It is attended with very little constitutional disturbance. A second attack is extremely rare.

Incubation. The incubation is generally about two weeks, but may be one or three weeks. In ordinary cases the first evidence of the invasion of the disease is the appearance of the eruption. In other cases, the severer ones, the child may be noticed for some hours or several days to be indisposed, and complain of loss of appetite, nausea, headache, and vague muscular pains. The *fever* is almost always moderate—100° to 101° F.

Eruption. The eruption consists first of hyperæmic macules, compared by Trousseau to the rose-rash of typhoid fever. These macules

FIG. 283.



Varicella on the fifth day of eruption. (WELCH.)

rapidly become first papules and then vesicles. The papules are not hard as in variola. They appear at first upon the chest, neck, face, and scalp, then upon the trunk and limbs. The development of the vesicles is so rapid that the eruption appears vesicular from the start. The vesicles vary in size from that of a pinhead to that of a small pea. They are very superficial, and usually rest upon a base that is slightly or not at

all hyperæmic. The contents are at first watery, and subsequently become pearly. The reaction of the fluid is alkaline. Distinct umbilication is rare, and pustulation still more rare, but both occur. The vesicles almost always dry up and form yellowish or brownish scabs, which drop off, leaving a slightly reddened, sometimes depressed spot. Sometimes the vesicles are to be seen upon the buccal mucous membrane and upon the throat. While most of the eruption appears on the first or second day, fresh vesicles continue to appear for several days.

Desiccation. Desiccation usually occurs by the fourth or fifth day, and may be present in the first day or two. As the eruption appears in successive crops, all stages, from the initial macule to the dried scales, can often be seen in one case.

Usually the vesicles are widely scattered, a dozen or two over the entire body. They are most numerous upon the back, and may be as close together as in discrete variola.

In scrofulous and badly nourished children the lesions are more inflammatory and pustules are more common. If they are scratched, ulceration ensues. A gangrenous form has been described by Eustace Smith and others; the cases are apt to be fatal.

In ordinary cases, during the eruption the child is rarely more than indisposed; complications are rare, and the prognosis most excellent. The physician is not often consulted except to have his opinion as to the diagnosis. (For the differential diagnosis from smallpox, see Variola.)

Diagnosis. *H. D.* Exposure; epidemic.

S. D. Negative.

O. D. Eruption; "crops."

P. D. Negative.

D. D. Variola (*q. v.*); vesicular and pustular eczema; impetigo.

Varicella is distinguished from *vesicular* and *pustular eczema* by the fever, the symmetrical grouping and discrete character of the lesions, the comparative absence of itching and burning, and its shorter course.

Impetigo is distinguished by the absence of fever, the more local character of the eruption, and the fact that it is generally pustular. The eruption is more common upon the face and hands than in varicella.

Scarlet Fever.

In this eruptive fever the course of the temperature varies somewhat with the severity of the infection. In many instances *fever* would not be detected without the use of the thermometer. In others it may rise to a greater height, and even be hyperpyretic. Its onset is sudden; it reaches its greatest height when the eruption is complete.

The *temperature* in *scarlet fever* usually conforms to a clearly defined type, increasing gradually until the third or fourth day, when the acme is reached. It declines by lysis in a period of four days. A seven days' chart would be pyramidal in shape. In septic forms (*scarlatina anginosa*), with ulceration of the fauces, the fever continues and becomes remittent. In malignant scarlatina, hyperpyrexia is likely to ensue rapidly.

Scarlet fever is an acute, specific, contagious, and infectious disease,

characterized by a sudden onset, with vomiting, sore throat, and high fever, followed in twelve or twenty-four hours by a bright-red, punctiform eruption, by a very frequent pulse, by a desquamation which is often in large flakes, by a very variable degree of severity, and by a large number of complications and sequelæ, especially nephritis and inflammation of serous membranes.

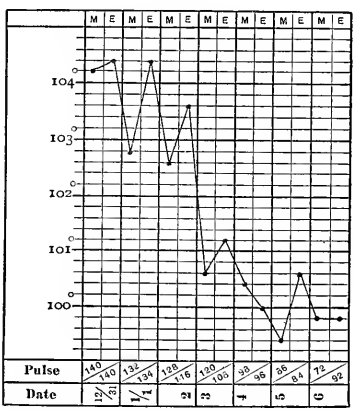
Scarlet fever preferably affects children from one to five years of age. The liability to it diminishes after the tenth year; but it is very rare under the age of six months. Puerperal women are very susceptible to the poison, and the existence of open wounds favors infection. The disease occurs in epidemics at longer intervals than is true of measles. Cases are most numerous in the autumn and winter months. The peculiar poison is doubtless a living organism, but it has not been isolated as yet. It is very tenacious of life, being capable of infecting, through clothing in which it has been retained, months after the poison has been absorbed by the clothing.

Few diseases vary so greatly in severity in different cases and in different epidemics. It may either be the mildest or the most malignant of diseases.

Incubation. The period of incubation is remarkably short, generally from three to five days; but it may be only a few hours, and in exceptional cases six days.

Invasion. The invasion is abrupt. It is very common to be told that a child was apparently well on going to bed, but awoke in the middle of the night vomiting profusely and complaining of sore throat. The child is found in the morning with a temperature of 103° or 104° F., a pulse of 120 to 140, and a scarlatinal eruption beginning to show upon the neck and upper part of the chest. Close observation in such cases might have

FIG. 284.



Scarlet fever. Mild attack; intense eruption. (Original.)

discovered that the child was feverish on going to bed, and somewhat chilly before that. Onset with decided chill, vomiting, and nervous symptoms indicates a severe case.

Symptoms. The *subjective symptoms* of scarlatina are few ; they consist usually of pain in swallowing, with stiffness of the neck muscles, some headache, thirst, malaise, and a moderate amount of weakness. In the eruptive stage the skin itches, burns, and is frequently hyperæsthetic.

The *objective symptoms* and their order of succession are very characteristic. Vomiting is the rule, except in mild cases, and hence is of importance in diagnosis, especially in otherwise doubtful cases. The temperature is high at the onset, frequently 103° or 104° F. It falls a degree or so in the morning ; but the following evening, when the eruption is usually at its height, it rises to 104° or 105° F., and then gradually falls to normal in the course of a week in ordinary cases. (Figs. 77 and 284.)

The *pulse-rate* is characteristically frequent, being 120 to 160 oftener than slower. This frequency is not an indication of danger.

The *blood* shows a leucocytosis, beginning on the first day and continuing through convalescence. A close relationship exists between the degree of leucocytosis and the rash. Suppurative complications tend to increase the number of white cells. The finely granular eosinophiles are greatly increased during the first few days. The mononuclear cells and lymphocytes are diminished at first, but after a short time their percentage increases.

The *throat* exhibits a uniform flush extending over pharynx, tonsils, soft palate, and sometimes foward on the hard palate, nearly to the teeth. Sometimes dark-red points can be distinguished on the soft palate. The tonsils are inflamed and projected toward the median line from each side. Frequently the mouths of the follicles are blocked by a creamy-white exudate. It is not uncommon to find a severe follicular tonsillitis at the first visit.

The *tongue* is at first covered with a thick, creamy fur, through which enlarged red papillæ show. The enlarged papillæ look like small grains of red pepper sprinkled on the tongue. Sometimes the papillæ are elevated and have a button-like appearance. The symptoms appear very early in the disease, and may continue for three or four weeks. The coating soon disappears from the tip, leaving it bright red—the "strawberry tongue."

The *skin* is hot and dry. The characteristic eruption usually appears within twenty-four hours, often within six to eighteen hours, of the chilliness or vomiting which marks the onset. Sometimes it comes out very slowly, seeming to be just ready to appear, but not appearing in its full development for four or five days.

The intensity of the *eruption* varies from a scarcely perceptible erythema to the color of a boiled lobster, and is proportionate to the severity of the disease. In ordinary cases the patient appears to be covered with a uniform red efflorescence ; but a closer inspection shows that there are darker red spots, between which the skin is more or less erythematous. It is first seen about the ears and neck, and spreads with great rapidity, covering the entire body in a day. It is most intense upon the trunk and flexor surfaces. Upon the extensor surfaces the punctate character is better seen. Pressure causes the redness to disappear, but it immediately reappears. Papular and vesicular forms of eruption are also seen. The

physiognomy in scarlet fever is peculiar. The circle about the eyes, nose, and lips remains pale, and in marked contrast with the rest of the fiery red face. Itching and burning are annoying symptoms at times. The eruption fades gradually, in ordinary cases, when there is no pressure or irritation, disappearing toward the end of the week.

The eruption is succeeded by *desquamation*, which is extensive in proportion to the intensity of the eruption. The flakes are larger than in measles, and in severe cases the epidermis may come off in long strips. This shedding from the hands and feet is sometimes so great that the cutaneous cast is compared to a glove. This stage may be protracted for several weeks, danger of infection lasting as long as desquamation continues.

The *urine* is at first scanty, high-colored, and febrile. Later, when desquamation is in progress, there is great liability to albuminuria as a complication.

Varieties. In addition to the ordinary form already described scarlatina exhibits many irregular forms. There may be only a sore throat or follicular tonsillitis. If a rash is present, it is very faint, and hence easily overlooked. The diagnosis in such cases must be made from the fact of exposure to infection and from the appearance of the throat. The occurrence of vomiting is very important in the diagnosis, as it is rare in ordinary pharyngitis and tonsillitis. Often such cases escape detection altogether, until possibly a dropsy from scarlatinal nephritis indicates their nature.

Severe diarrhœa may prevent the eruption from developing upon the skin. It appears upon the fauces, and the diagnosis is based upon this, the pulse and temperature, and the fact of exposure.

In **scarlatina anginosa** the strength of the poison is spent upon the throat. Pain is great and deglutition difficult. The tonsils are greatly swollen so as almost to occlude the fauces, and their surfaces are covered with creamy exudate. The cervical glands are swollen, and there is a tense and brawny cellulitis. Sometimes the tonsils become gangrenous, and the cervical or submaxillary glands suppurate or become gangrenous, with resulting pyæmia and death. Suppuration may extend to the ears and maxillary sinuses. In this form, also, a false membrane is sometimes found upon the fauces—post-scarlatinal diphtheria. It is probably not due to the Klebs-Löffler bacillus, but to a streptococcus.

In **malignant** forms the attack is ushered in with a chill, followed by hyperpyrexia, convulsions, marked ataxic symptoms, or stupor. The profound blood-disturbance is shown by the dusky hue of the eruption. Some patients lie in coma-vigil, others are very restless and delirious. Vomiting and diarrhœa are sometimes superadded. Patients may emerge from this condition and succumb later to a nephritis or to grave anginose symptoms; but death in a few days is the rule. In rare cases the dose of poison is so enormous that death takes place in a few hours, without the appearance of any eruption.

Complications and Sequelæ. The severe local symptoms mentioned under the anginose variety, together with convulsions, hyperpyrexia, and ataxic symptoms, may properly be regarded as complications. Apart from these the most frequent are nephritis and endocarditis or pericarditis.

Nephritis generally appears with the beginning of desquamation. It is nearly as frequent in mild as in severe cases, probably because the danger of exposure to cold is greater in the former, although the scarlatinal poison unquestionably has a selective affinity for the epithelium of the kidney. The symptoms do not differ from those of acute parenchymatous nephritis occurring under other circumstances. In some cases we have weakness, languor, slight fever, and prolonged convalescence; in others, œdema, anuria, convulsions or coma from uræmia.

Endocarditis is often preceded by tenderness and soreness of the muscles and joints—scarlatinal rheumatism. Endocarditis and pericarditis develop in the course of the fever, giving rise to an increase or continuance of the fever, to local pain or dyspnœa, and to the usual physical signs.

Pleuritis and *meningitis* also may occur. Much more common complications are *otitis*, peripheral *neuritis*, and affections of the joints, grouped as *scarlatinal rheumatism*. Paralyses, peripheral and central in origin, are occasional sequels of the disease. Scarlatina is found also in association with other diseases.

Diagnosis. *H. D.* Exposure to cause; epidemic.

S. D. Sore throat; vomiting; headache; clinical course.

O. D. Eruption; fauces; tongue.

P. D. Negative.

L. D. Leucocytosis; albumin in urine.

D. D. Muscles; rubella; acute local faucial infections; acute gastritis; acute meningitis; pneumonia; diphtheria.

Sudden onset, rapid rise of temperature, persistent and causeless vomiting, and sore throat lead one to suspect this affection. The characteristic eruption and its mode of evolution, the rapid pulse, the peculiar tongue, the circle of pallor on the face, are characteristic of the eruptive stage. "The appearance of a punctate eruption in the axilla and in the groins, together with the congestion of the tonsils and a punctate eruption in the roof of the mouth, no matter whether there is any eruption anywhere else or not, are positive proofs of scarlet fever." (McCollom.)

Unfortunately, all cases do not develop to the same degree, so that frequently we must wait for the period of desquamation; more infrequently, for the occurrence of sequelæ, as acute nephritis, otitis, or adenitis.

Measles. Scarlet fever is distinguished from measles by the mode of onset, which is sudden, with chilliness, high temperature, vomiting, and sore throat, and great rapidity of the pulse; whereas the onset in measles is gradual, with coryza, cough, moderate fever, perhaps looseness of the bowels, but no sore throat. The eruption of scarlatina occurs on the first day, that of measles on the fourth; the former consists of dark-red spots with intervening erythematous skin, the whole looking at a distance like a uniform bright-red flush; the latter consists of raised, rounded, or flattened spots or blotches, velvety to the touch, and, upon the body and extremities, grouped in patches with crescentic outlines. The temperature in scarlatina subsides gradually after the rash has reached its height; that of measles increases until the eruption is complete, then subsides by crisis.

The rash of scarlet fever persists for six or eight days; that of measles fades as soon as it is complete, on the fourth day. In the former, desquamation is in flakes or large strips; in the latter it is branny and nearly invisible. Scarlatina involves by preference the serous membranes and kidneys; measles, the mucous membranes and lungs.

Scarlatina has to be differentiated from *pharyngitis, tonsillitis,* and digestive disturbances, attended with vomiting, high temperature, and, occasionally, erythematous eruptions. In ordinary pharyngitis and tonsillitis the redness is more apt to be confined to the pharynx, tonsils, and arches of the soft palate; in scarlatina it extends as a flush over the soft and hard palate and buccal surfaces. In the former, high temperature, a very frequent pulse, and vomiting are unusual; in the latter they are the rule. The glands of the neck also are more apt to be involved in the latter.

In *acute gastritis* there is usually a history pointing to indiscretion in eating, with constipation. The pulse is not so frequent as to suggest scarlatina, sore throat is absent, and any erythema present lacks the characteristic dark-red points, and is not followed by desquamation.

Rubella. The diagnosis from rubella is difficult at times. It differs from scarlatina in presenting mild catarrhal symptoms, sneezing, suffusion of the eyes, and cough, with a relatively fleeting eruption. The latter perhaps appears most frequently upon the back and chest. Often the eruption is the first thing noticed amiss with the child. It more commonly resembles the rash of measles than that of scarlatina, but when it resembles the latter most it is apt to be discrete and of a darker red. There may be a very intense rash without much constitutional disturbance, the temperature being lower and the pulse much slower than would be expected in a scarlatina presenting the same appearance. Nausea may be present, but vomiting is very rare. The post-cervical and post-auricular glands are more commonly enlarged in rubella than in mild scarlatina, though this symptom is not invariable.

Diphtheria is distinguished by its gradual onset, patches of false membrane developing upon the fauces early. In anginose scarlet fever, with severe follicular tonsillitis, the differential diagnosis is essentially the same as between simple follicular tonsillitis and diphtheria (*q. v.*). In addition, the pulse and temperature have a much higher range in scarlatina. The erythema of diphtheria is distinguished from the eruption of scarlatina by its fleeting character and by the absence of desquamation.

Meningitis. Grave cases which begin with repeated vomiting, convulsions, delirium, and insomnia simulate meningitis; but a satisfactory cause for the latter is lacking, while the excessive heat of the skin, sore throat, very frequent pulse, and early eruption clear up the diagnosis.

Pneumonia. So, also, the onset with vomiting, convulsions, and high temperature resembles pneumonia; but in the latter the respiration is proportionately more frequent than the pulse, with altered breath-sounds and percussion-sounds, while sore throat and eruption are wanting.

Measles.

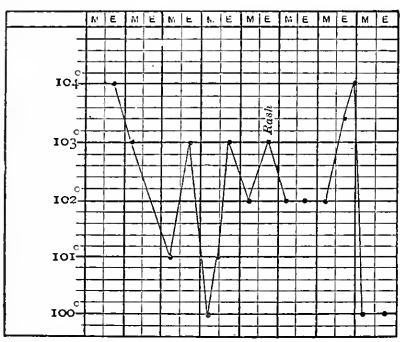
The course of the *fever* in this affection resembles that of smallpox in that after the initial rise of the first twenty-four hours the temperature remains normal until the appearance of the eruption on the third day. It is an acute, specific, infectious, and highly contagious fever, characterized by coryza and bronchitis, a red, papular eruption coming out on the fourth day, and followed by a branny desquamation about the ninth or tenth day. The mucous membranes are especially liable to complications.

Measles occurs in epidemics, especially in cold weather, but individual cases are met with in large cities at all seasons of the year. It is so contagious that when one case develops in a household or institution almost every person exposed to it and not protected by a previous attack acquires it. Children from one to five years of age are most susceptible to the poison, but it may occur *in utero* and in old age; moreover, the same person may have several attacks, showing that one attack does not afford the same protection as an attack of scarlatina or variola.

Measles is sometimes found in association with scarlatina and varicella, but it is especially liable to occur after pertussis.

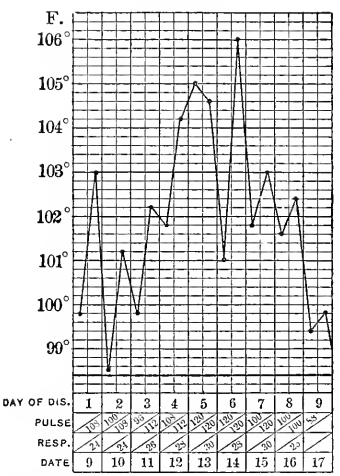
The specific cause of the disease has not yet been isolated.

FIG. 285.



Measles. Temperature taken on the first day, made higher as the result of attendance at school and exertion. (Original.)

FIG. 286.



Measles. Lower temperature second and third days. Hyperpyrexia sixth day. Abundant eruption. Bronchitis severe. (Original.)

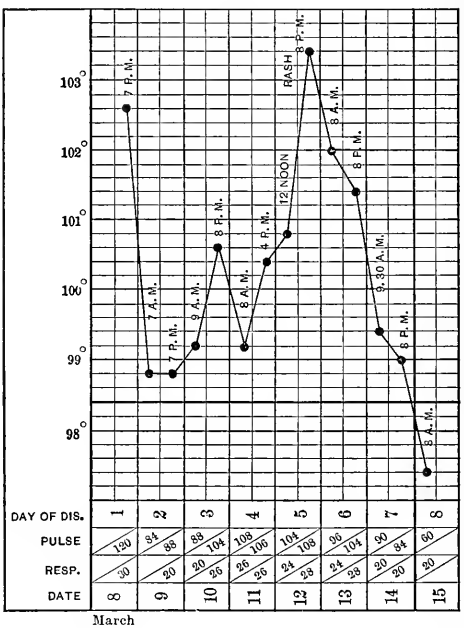
Incubation. The period of incubation lasts from eleven to fourteen days. During this time the patient may exhibit no symptoms, or may be irritable and restless, with disturbed sleep and occasional cough, and looseness of the bowels.

Invasion. The invasion is marked by cough and fever, and by redness of the eyes and lacrymation, sometimes with photophobia, sneezing, and an irritating, watery discharge from the nose, which subsequently becomes mucopurulent, and by cough and fever. In short, the early

symptoms are those of a severe coryza. These symptoms last from three to five days (generally four) before the *eruption* appears.

But an eruption is commonly visible upon the base of the uvula and soft palate, as raised, discrete dark-red papules, several days before it appears upon the body. Another mucous membrane eruption peculiar to this infection has been accurately described by Koplik (1897). His observations have been corroborated, so that "Koplik's sign" is a well-established fact. Its importance can be understood when the necessity for early diagnosis for quarantine purposes is realized. This sign appears twenty-four hours, forty-eight hours, and even three to five days before the skin eruption. It precedes the conjunctivitis and begins at the first rise of temperature. The eruption appears on the mucous membrane of

FIG. 287.



Measles. Characteristic chart. Female, aged twenty-seven. (Original.)

the cheeks and lips. It is not seen on the palate or the fauces. It is at first discrete and then becomes confluent. It is at its height when the skin eruption appears and is spreading. In strong daylight this pathognomonic eruption is seen to consist of small, irregular spots of a bright-red color, in the centre of which is seen a bluish-white speck. The bluish-white speck is very small and delicately colored, requiring direct and strong daylight to see it. A combination of the speck on the rose-red background is a positive sign of the invasion of measles. The spots must not be mistaken for *sprue*, which is opaque, white, coarse, and plaque-like. When the rose-red spots coalesce, Koplik describes the appearance of the mucous membrane to be made up of large areas of rose-

PLATE XV.

Fig. I.

Fig. II.

Fig. III.

Fig. IV.

The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

red, studded all over with minute raised bluish-white specks, relieved here and there by the normal hue of the uninvaded mucous membrane. The accompanying figures from Koplik's latest paper illustrates this important sign. (Plate XV.) By this sign measles can be differentiated from rubella, scarlet fever, aphthous stomatitis, forms of erythema and urticaria, drug eruptions, the antitoxin eruption, and syphilides.

The *temperature* rises during the first day to 100° or 102° F., or higher if the case is to be a severe one. The bowels are frequently inclined to be loose and the passages somewhat greenish. The temperature falls on the second day to normal or nearly normal, and then steadily rises until it reaches its acme with the full development of the eruption, when, in uncomplicated cases, it falls rapidly to normal. With the coming out of the eruption the coryza increases in severity, and cough is a prominent and annoying symptom. It consists of a series of five or six explosive efforts without expectoration. In severe cases the cough is almost incessant, so that rest is much interfered with. It depends upon a catarrhal inflammation of the entire respiratory tract, from the nose to the bronchioles.

The Eruption. The eruption on the skin appears first about the neck, face, and wrists, and spreads in two or three days over the entire body. It is usually most copious upon the face, which is swollen, dark red in color, and closely set with papules, which are elevated, rounded at the summits, and feel velvet like to the touch. When to this picture is added that of a severe coryza with mucoserous exudate, which often glues the eyelids together and oozes out upon the face, and a corresponding condition of the nasal orifices, the physiognomy is at once seen to be unusual. At this stage, moreover, photophobia is often considerable, the child burrowing its head in the pillow to escape the light.

The *eruption* is not apt to be confluent upon the body; here the dark-red, elevated, smooth papules are very distinct. Sometimes they are grouped so as to form crescentic outlines. The eruption fades in the order in which it appeared, and is followed by a fine, branny desquamation. With the completion of the eruption the fever falls rapidly to or below normal, the coryza and bronchitis improve correspondingly, and in forty-eight hours convalescence is fully established.

Complications. The complications of measles affect for the most part the mucous membranes of the respiratory and digestive tracts. The bronchitis, which is always present, may become capillary, or be associated with œdema or with areas of catarrhal pneumonia. These are the most frequent and the most dangerous complications. Pneumonia may develop while the eruption is coming out, in which case the eruption is delayed or the spots have a dusky or bluish hue (black measles). More commonly, perhaps, pneumonia is discovered when, the eruption being complete, a crisis should occur. Epistaxis is not usually dangerous. Profuse diarrhœa is very exhausting and delays the evolution of the eruption. Severe conjunctivitis, sometimes with ulceration of the cornea, is not uncommon. Otitis media occurs oftener as a sequel than as a complication. Noma, or cancrum oris, is a rare complication of measles occurring in ill-fed, badly nourished children. It is frequently fatal.

Convulsions may occur as a complication, especially when pneumonia is developing.

Sequelæ. In cases in which there has been diarrhœa, measles is sometimes followed by considerable weakening of the digestive power. The catarrh of the respiratory tract, which almost invariably accompanies it, predisposes to the development of whooping-cough and tuberculosis. Paralysis may follow measles. It may be central or peripheral in origin, but generally is of the hemiplegic type; cases of acute poliomyelitis, acute ascending paralysis, and disseminated myelitis have also been reported.

Varieties. Measles without catarrh is rare. It can not be distinguished from a measles-like rash, seen in rötheln, except by the occurrence in the neighborhood of other cases of undoubted measles.

Measles without eruption is to be recognized by the coryza, possibly with eruption on the soft palate, the course of the temperature, and the exposure to specific infection.

Black measles is the name given to malignant forms in which, owing to complications, particularly pneumonia, the skin is dusky and the eruption comes out poorly and has a bluish color. In rare instances the eruption shows a hemorrhagic tendency, the spots being livid or ecchymotic. Actual hemorrhages from mucous surfaces may occur, the patient dying in coma or convulsions.

Diagnosis. *H. D.* Epidemic eruption; age.
S. D. Coryza; cough; clinical course.
O. D. Fever range; eruption; Koplik's spots.
P. D. Bronchitis.
L. D. Bronchitis.
D. D. Eruptive fevers.

Rubella.

In a few instances this affection may run its course without *fever*. In the large majority of cases, however, a moderate degree of fever prevails, and in some it may reach a considerable height.

Rubella is an acute, specific, contagious, and infectious fever, characterized by a gradual onset, with moderate fever, sore throat, and slight coryza. The eruption which appears without prodromata, usually resembles measles more than scarlatina. The duration, however, is shorter than measles, the disease milder, and complications are rare.

The disease is amply proved not to be a hybrid of measles and scarlet fever. The incubation-period varies from one to three weeks, but is generally about two. As a rule, this period is passed without symptoms.

The invasion is without prodromata, or none more definite than languor and indisposition, the first thing noticed being the eruption. This in some cases consists of pale-red, smooth, slightly raised blotches, closely resembling measles, but more pronounced on the trunk, and discrete. This is probably a very rare form. More commonly it consists of rose-red macules or papules, occasionally confluent, but usually discrete, and most marked upon the trunk. In still other cases the eruption closely resembles that of scarlatina, differing chiefly in being a paler

red and accompanied by less heat of skin. Sometimes the eruption is circumscribed, as upon the face or limbs. It is usually the seat of considerable itching, and this may be the first symptom that attracts the patient's attention. It will be seen that the eruption is multiform in character. Concurrently with the eruption there is usually slight rise in temperature (100° to 101° F.), suffusion of the eyes, with slight lacrymation and photophobia, and slight pharyngitis; nausea is not uncommon, but vomiting is very rare. Higher temperatures have been recorded in a few cases, and so have nervous symptoms, such as delirium and convulsions, but they are chiefly interesting as very exceptional possibilities.

The eruption extends over the body in twenty-four to thirty-six hours, less rapidly than in scarlatina, and pales much more quickly, fading on the portions of the body first attacked before reaching its height on the last, and being completed in three or four days. Sometimes a branny desquamation succeeds.

In addition to the mild coryza and eruption, the most important objective symptom is swelling of the cervical glands, all of them being sometimes swollen, especially those behind the sternomastoid, the auricle, and along the margin of the hair. This adenopathy, however, can not be relied upon exclusively in the differentiation from scarlatina and measles.

Rubella has few complications: bronchitis, pneumonia, and otitis occur rarely, and still more rarely false membrane on the throat, and albuminuria. The prognosis is excellent. It ends almost invariably in recovery, except in very feeble children.

INFECTIONS RECOGNIZED BY LOCAL SYMPTOMS.

The following infections are characterized by *local* manifestations which are of greater diagnostic significance than the *fever*. These local manifestations must therefore be carefully considered in the diagnosis, as they must be relied upon for recognition of the particular infection. The infections belong to Class I. and Class II. of the classification in Chapter XXXII., Part I.

Mumps.

The swelling is characteristic. It usually begins on one side. The swelling of the parotid gland is observed in front of the ear, then it extends below and around it and behind the ramus of the jaw. Unless there is much collateral œdema the outline of the gland is preserved. The gland is tender and boggy, not indurated. Viewing the face from the front, the midlateral aspects are seen to bulge. The ears stand out from the head. The jaws are fixed. The submaxillary glands are usually enlarged.

The diagnostic features of mumps include the symptoms of the invasion, the general symptoms, and the local signs.

Invasion is sudden, with chilliness, a rise in temperature which is generally moderate (101° to 103° F.), and pain at the angle of the jaw. The corresponding parotid gland as well as the adjacent cellular tissue

begin to swell rapidly. Along with pain on movement of the jaws, any acid liquid, as vinegar, which stimulates salivary secretion, increases the pain. At times the submaxillary glands are involved instead of the parotids, or they may be enlarged and painful several days before the parotid is affected. The disease may be limited to one side or involve the opposite side, as the process in the one first attacked subsides. Rarely it is bilateral from the start. When the swelling has lasted from three to five days, the fever subsides and the swelling begins to disappear rapidly. At this time, however, the opposite side may be attacked or the testicles become inflamed. Usually it is the right testicle. Rarely in girls and women the ovary or mamma is inflamed. Resolution is extremely rapid, and usually the disease is not followed by sequelæ. Sometimes, however, deafness is left. In fact, sudden deafness sometimes announces the commencement of an attack.

If to these attacks we add the data obtained in the social history, the age of the patient (usually under fifteen), and the history of exposure to or the presence of an epidemic, the diagnosis is easily made.

Glandular Fever.

It must not be forgotten that many persons, children especially, have irritable lymphatics (lymphatism) which become enlarged and tender whenever any toxic substance sweeps through the lymph-current. Hence, in milder fever from many causes, as from "cold," catarrh, trauma, or gastro-intestinal disorders, the glands are enlarged. These conditions must not be considered glandular fever, a mistake, I suspect, often made. May not glandular fever be this intoxication?

Glandular fever is an infectious disorder, the cause of which has not been accurately determined. It is characterized by *fever*, usually occurring abruptly, with headache, pains in the limbs and in the lymph-glands of the neck. On examination of the fauces a slight pharyngitis is observed and the tonsils are enlarged. The rise of temperature is accompanied by frequent nausea and vomiting. The temperature rises abruptly to about 102° F. In the second twenty-four hours the glands of the neck, particularly those behind the sternocleidomastoid muscles, enlarge. They are tender. Although there may be some slight œdema, there is no redness or swelling of the skin. The fever continues for three or four days; the glandular enlargement, however, may persist for several weeks, and may end in suppuration.

The infection usually occurs in children between the age of five and eight years, or earlier in life. It may be epidemic. The other lymphatic glands about the neck and in the axilla and groin may be enlarged. In not a few instances there is enlargement of the spleen, and cases of enlarged liver and mesenteric glands have been reported. The absence of an eruption serves to determine the infection from the eruptive fevers associated with adenitis, particularly measles and rubella.

Whooping-cough.

The attention of the physician is called to this infection by the peculiar character of the *respiratory symptoms*. *Fever* is more notable as an expression of one of the complications—bronchopneumonia—than of the general infection. It may, however, be a serious symptom of the infection.

Whooping-cough is a specific catarrhal inflammation of the respiratory passages, involving especially the trachea and bronchi, and characterized by paroxysms of cough, which are succeeded by spasmodic closure of the glottis and a peculiar inspiratory whoop. The disease occurs especially in childhood, is contagious and infectious, and is sometimes epidemic. Whooping-cough may be conveniently divided into three periods:

1. The catarrhal stage.
2. The spasmodic stage.
3. The stage of gradual subsidence of the disease.

First Stage. The patient appears to have an ordinary cold. The amount of redness of the mucous membrane of the eyes, nose, and throat varies considerably, but there is not much discharge from the mucous surfaces. The cough is dry, and sometimes a ringing quality can be detected. The patient is irritable, has slight fever, diminished or capricious appetite, and restless sleep. A mild bronchitis of the larger tubes can be detected by physical exploration.

The cough gradually becomes more frequent and paroxysmal, the eyes are red and suffused, and there is a mucopurulent discharge from the nose. The face often looks slightly swollen, especially about the upper part and under the eyes. Lymphocytic leucocytosis is common.

Second Stage. Transition from the first to the second stage is marked by the appearance of the characteristic whoop. The paroxysmal cough is made up of a series of rapid expiratory efforts, diminishing in force and duration; when these cease, there succeeds a prolonged crowing inspiration—the whoop. There may be only one paroxysm of coughing at a time, but more commonly, and always in severe cases, one paroxysm is succeeded by another. During the coughing the child's eyes become suffused, the tears overflow, and there is a discharge of serum or mucopus from the nose, and of saliva and bronchial secretion from the mouth. The face becomes swollen and dusky. If the child is walking about, it grasps some object for support during the paroxysm; or, if old enough, rushes for the water-closet or a basin, because the seizure usually terminates in vomiting. The matters vomited consist of tenacious mucus and the contents of the stomach. The mucus may be streaked with blood, and occasionally there is pure blood. During severe paroxysms, hemorrhages are apt to occur; these are generally small and most frequently submucous. In well-marked cases, when the disease has lasted some time, the face has a characteristic appearance: it is swollen, sodden, and dusky, with dull, heavy, red, and watery eyes. There is often ulceration of the lingual frænum.

The number of paroxysms varies from two or three to twenty or thirty or more in twenty-four hours, and they are worse at night.

The whoop, while characteristic, is not present in every case, being

absent especially in babies and very young children. Sometimes children have "choking spells" without much coughing and without the whoop. Again, when pneumonia or measles occurs as a complication, the whoop usually ceases for the time, but may reappear later.

Third Stage. The third stage is less well defined than the first two. It may be said to begin when the nocturnal exacerbations become less frequent and severe. The number of paroxysms during the day diminishes, and vomiting is a less frequent accompaniment. Appetite improves, and the child gains in flesh and passes more restful nights.

The duration of the disease is variable. Ordinarily it lasts from six to eight weeks, but it may be prolonged for several months. The patient is liable, whenever he catches a fresh cold, to a temporary return of the spasmodic cough, sometimes with the whoop.

The great majority of the cases occur before the sixth year, and most of these between the second and fourth years.

Rheumatic Fever.

Rheumatic fever is an infection associated with local symptoms of *joint*, *endocardial*, and *pericardial* inflammation. The local symptoms are so intense as to call attention at once to the nature of the infection apart from the course of the *fever*, as it is largely upon these symptoms that the diagnosis is made.

An acute, general, febrile, non-contagious disease, characterized by specific inflammation of the *joints* and contiguous structures, hence called *acute articular rheumatism*. It is further characterized by a tendency of the inflammation to involve the larger joints successively, to skip from one joint to another, and to be associated with endocarditis or pericarditis.

The predisposing causes of rheumatic fever are *heredity*, which is operative in 25 or 30 per cent. of the cases; *age*—81 per cent, of first attacks occur between the eleventh and thirtieth years (Pye-Smith); *sex*—in childhood girls are more frequently affected than boys, but after that period sex appears to have no influence. Multiarticular inflammations, sometimes rheumatic in nature, are met during convalescence from scarlatina and dysentery. They also occur in association with the puerperal state and gonorrhœa, when they are probably pyæmic. The nature of the polyarthritis which occurs in connection with dengue and hæmophilia is obscure.

Symptoms. The *onset* of the disease is not characterized by constant symptoms. Sometimes the fever and joint-inflammations are preceded a day or two by debility, wandering pains in the joints or muscles, and loss of appetite. Other cases are marked by a chill or repeated attacks of chilliness, followed in a day or two by fever and inflammation of the joints. In rare cases the onset may be followed not by inflammation of the joints, but by inflammation of the serous membranes, particularly those of the heart and its sac.

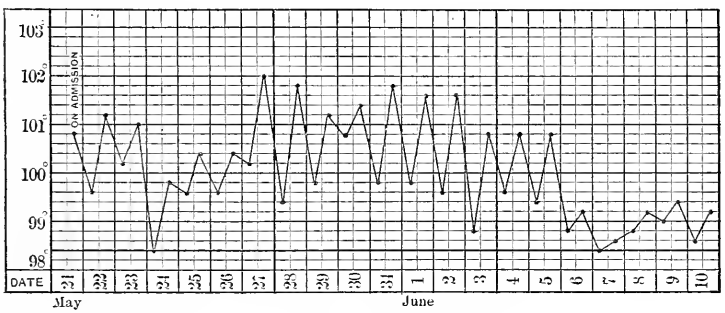
The *temperature* may rise a day or two before there are any joint-symptoms, or fever and arthritis may begin almost simultaneously. The

temperature rises rapidly to 102°, 103°, or 104° F., and one or more of the larger joints, generally the knee and ankle, become painful, tender, swollen, and hot.

The Joint. There may be great pain on motion before there is evident swelling or much local tenderness. The pain varies from mere discomfort to the most excruciating suffering. It is always aggravated by motion or pressure, and is at times so exquisite that the lightest touch, the weight of the bedclothing, or the jar of the bed from a heavy step in the room makes the patient cry out. It may extend beyond the joint to neighboring tendons and nerves. The swelling likewise varies greatly; sometimes there is only slight puffiness with increased distinctness of the cutaneous veins, increased heat in the part, but no general redness; in other cases there is considerable swelling about the joint, so that the bony prominences are obliterated, the surface being tense, red, and very hot to the touch. There is not often effusion into the joint. Swelling is most marked in the wrist and ankle, and less so in the shoulders, hips, elbows, and knees. It is very common on the dorsal surface of the hand.

Multiplicity of Joints Affected. A characteristic peculiarity of rheumatism is its tendency to involve one joint after another. One or several joints may be affected at first; it is very common for the right ankle to be affected, and then in a short time the opposite ankle, followed by the left knee and right knee, and so on with the other joints. The inflammation usually lasts from two to four days in each joint. The

Fig. 288.



Rheumatic fever. Admitted fourth day of disease. (Original.)

process may subside in one articulation and begin in another with startling rapidity. At one visit the physician may find the patient's right ankle swollen, hot, and unbearably painful, and on the next day the right ankle may be quite well and the patient be found suffering acute pain in the right knee or left ankle.

The *pulse* in the early stages of rheumatism is moderately accelerated (99 to 110); it is regular, of good volume, often bounding, and sometimes hard. The *urine* is scanty, high-colored, abnormally acid, and deposits on cooling a copious precipitate of urates, resembling red sand. The *skin* does not feel so hot as one would expect from the temperature. It is continuously covered with a copious, acid, and somewhat pungent

perspiration. *Nervous symptoms* are not marked. There may, however, be slight nocturnal delirium. Sleeplessness from pain is common.

The *temperature* in rheumatic fever is not usually very high; it is much oftener under than over 103° F. In rare cases, however, especially when the fever is complicated with pericarditis, pneumonia, or some disturbance of the heat-regulating apparatus, the temperature may attain the extraordinary range of 106°–112° F. Such high temperatures may occur suddenly or gradually, and are sometimes attended with marked brain-symptoms (so-called cerebral rheumatism).

Endocarditis and *pericarditis* may occur at any period of rheumatic fever; they may even precede the joint-inflammations. They are most common, however, in the first two weeks of the disease. The younger the patient and the more severe the attack, the greater the liability to heart-complications. They occur in about one-fourth of all cases. Endocarditis is most common; often it is the only lesion, but sometimes it is associated with pericarditis and more rarely with myocarditis. These complications usually give rise to no symptoms at first. Hence, the heart should be examined daily. A sense of constriction in the præcordia or the pit of the stomach, an anxious expression of the face, with pallor, a change in the frequency, but especially in the rhythm of the pulse, and the occurrence of cough or dyspnœa should attract attention to the heart. The physical signs of the respective lesions are fully described under Diseases of the Heart.

The setting in of convalescence from rheumatic fever is marked by an improvement in the tongue, which loses its coating and becomes less red, and by an increase in the secretion of urine, which remains of high specific gravity. The fever subsides gradually, the joints cease to be red, swollen, and tender, the acid sweats lessen, and the appetite improves. In proportion to the duration of the case and its severity the patient is left with debility and marked anæmia, both red cells and hæmoglobin being diminished. In anæmic cases a hæmic murmur may be heard over the base of the heart. In some cases acute dilatation has been observed, with a tricuspid murmur.

Complications and Sequelæ. Apart from heart complications which have been mentioned, *pleuritis*, *pneumonia*, and *bronchitis* occur in from 10 to 15 per cent. of the cases. They are frequently bilateral, and are very much more common in rheumatic fever with pericarditis or endocarditis than in simple rheumatic fever. Moreover, the pulmonary complications are frequently latent, and would be overlooked but for the daily physical examination of the chest. On the other hand, they may develop with great suddenness, and what appeared to be a full-blown pneumonia may subside suddenly as a fresh joint is affected. They behave more like sudden active congestions than true pneumonias. Rheumatic pleurisies are characterized by the rapidity with which effusion takes place, the persistence of pain in the side during effusion, the tendency to involve both sides in succession, the readiness with which the effusion is absorbed, and an acute course.

Nervous System. The most common complication of the nervous system is *delirium*, which is generally associated with insomnia and hyper-

pyrexia, but the latter is not constant. These brain symptoms generally appear in the second week of illness, and about the time of convalescence, or while the joints are still inflamed. The delirium may be low and muttering, accompanied by ataxic symptoms or even by tremors and spasms of muscles; or it may be furious. In favorable cases a deep sleep ushers in recovery; in unfavorable cases the delirium persists with adynamia, the patient dying in collapse or coma, possibly preceded by convulsions.

Chorea sometimes occurs as a complication, but it is more common as a sequel of mild cases in children. *Cerebral meningitis* occurs occasionally, especially when there is ulcerative endocarditis. *Cerebral embolism* is another rare complication.

Various *spinal symptoms* occur in some cases, at times with, and at times without demonstrable lesion of the cord or its membranes. Tetanus, myelitis, and spinal meningitis may all be simulated. Perhaps these symptoms are due to high temperature; but hyperpyrexia is possible without the occurrence of cerebral or spinal symptoms.

Nephritis is rare, but sometimes hemorrhage into the kidney occurs with its usual symptoms. Peritonitis is extremely rare.

Various *erythematous eruptions* are seen from time to time, and occasionally *purpura*. Subcutaneous nodosities have been described by several writers. They are attached to the tendons, fasciæ, and periosteum, and are most frequent on the back of the elbow, the ankles, and patella. They are painless, and may occur in any form of rheumatism.

Diagnosis. Rheumatic fever is distinguished from *gout* by the profuse acid and acrid sweating, the tendency to involve a number of joints, and particularly the larger ones, the greater intensity of constitutional symptoms, the great liability to heart-complications, and the absence of uric acid from the blood.

It is distinguished from *pyæmia* by the wandering character of the inflammation; the acid sweats; the absence of any antecedent condition which would develop purulent foci—such as injuries, abscesses, or specific eruptive fever; the absence of chills, and the fact that in rheumatic fever the sweats are constant, whereas in pyæmia they follow a fall in the temperature. Cutaneous abscesses do not occur in rheumatism, and after its subsidence the joint's usefulness is not impaired.

Acute synovitis resembles rheumatic fever, because in both occur symptoms of pain, tenderness, and swelling in connection with a joint. Usually, however, but one joint is involved in synovitis, and there is a history of exposure to cold or injury. The effusion is limited to the synovial sac, is frequently abundant, and fluctuation can easily be detected. The constitutional symptoms are much less marked than in rheumatism. The *acute arthritis of infants*, which is pyæmic in character and often of gonococcus origin, must be distinguished from rheumatism.

Milk-leg, or *phlegmasia alba dolens*, differs from rheumatism in that it usually occurs in women after confinement, or as a complication or sequel of fever, as typhoid fever. Usually one leg is affected, or part of the leg, especially the calf. This becomes tense, tender, uniformly swollen, and the seat of great pain. The leg is moved with much difficulty. The

femoral vein may be found to be knotted and tender. There is almost always evidence of antecedent disease.

Acute periostitis when close to a joint simulates rheumatism; but the tenderness and heat are not in the joint itself; they are superficial, and are associated with less swelling. Pitting on pressure is common; and circumscribed fluctuation usually discloses the presence of suppuration. Pyæmic symptoms are added to the local symptoms, particularly if *ostitis* or *osteomyelitis* is present.

The articular symptoms of *glanders* are to be distinguished by the patient's occupation, the mode of onset, the associated symptoms, especially the presence of one or more pustules, and the fact that the painful joints are not so apt to be swollen and red as in rheumatic fever.

In *syphilis* joint-pains frequently occur, but their character is made out by the fact that the joints are not inflamed, and that the pain is much worse, or occurs only at night, by the history, and the therapeutic test.

In some diseases of the brain and spinal cord joint-inflammations of *trophic* origin occur. They are distinguished by the coexistence of some lesion of brain or cord, with hemiplegia or other palsy, and of other trophic changes, such as bed-sores, atrophied muscles, loss of hair, shiny skin, and defective growth of nails.

Subacute Articular Rheumatism. In some instances the joint-inflammation is less severe, and is accompanied by only slight fever. One or more joints may be affected. Subacute rheumatism differs from the ordinary form in being milder in degree and more persistent, lasting sometimes for months. It is generally subacute from the beginning, but may represent a secondary modification of the type in those who have had several attacks of rheumatic fever and have been left in a very sensitive condition.

The type of rheumatic fever is usually subacute in children, and often only one joint is involved. Cardiac complications are more frequent than in adults, and chorea may occur as a sequel. Erythema nodosum and subcutaneous nodosities are more common in children.

Dengue.

The peculiarity of the *fever* in this infection is that it is attended by severe pains in the muscles and joints. It is an acute contagious disease, occurring in epidemics, and characterized by severe pains in the head, back, and joints, by the presence of various skin eruptions, a prolonged convalescence, and a very low rate of mortality.

The disease occurs in epidemics in tropical and subtropical countries, and rarely in cooler climates. It derives its name, dengue (dandy), from the stiff and unnatural gait assumed by convalescent patients. In the southern parts of the United States an expressive name given to the disease is "breakbone fever."

The specific cause of the disease is believed by McLoughlin to be a micrococcus which he isolated. The period of incubation is short, varying from a few minutes to several days, or even a week. Invasion is

sudden and is rarely preceded by prodromata. It is marked by chilliness or a chill, and very severe pains in the head, back, and limbs. In children the onset may be marked by convulsions, which are sometimes followed by stupor and vomiting. The pains are sometimes excruciating, and are accompanied by tenderness of the muscles; there is extreme debility. The temperature rises to 102° or 103° F., but rarely is much higher. The pulse is frequent—110, 120, or more. In from one to three or five days the temperature falls to or below normal (the remission), accompanied by sweating or diarrhœa, and fluctuates about this level for several days, when a second and moderate rise in temperature, which is of short duration, occurs. During the first rise in temperature there is a transient, generally scarlatiniform rash, which is not followed by desquamation. The urine is febrile but not albuminous. During the remission eruptions —scarlatiniform, herpetic, urticarial, or miliaria like—begin to appear, accompanied by the secondary rise in temperature. The eruptions may come out in successive crops, and are followed by desquamation. Convalescence is now established, but may be interrupted by relapses. Strength is regained slowly. The most frequent complications are disorders of the nervous system, but bronchitis and diarrhœa occasionally occur.

Beri-beri.

Beri-beri is an infectious disorder with fever which prevails in epidemic form in tropical and subtropical countries. It is characterized by multiple neuritis associated with anasarca. By most observers it is believed to be an acute infection, although not a few think it is an intoxication due to certain kinds of food; this view prevails in Japan. Conditions predisposing to infections, such as over-crowding, hot and moist weather, and exposure to the elements, are usually present. It is far more common in men, and usually attacks subjects between the ages of sixteen and twenty-five.

Several clinical forms are seen. In the most complete form there is rapid loss of power in the legs and arms, with atrophy of the muscles. The patients complain of pain, and later œdematous symptoms may appear. With the loss of power in the legs there is paræsthesia, with frequent palpitation of the heart and dyspnœa. The pain in the muscles is associated with weakness and tenderness. In milder degrees of this form, pain, weakness in the legs, diminution of the sensibility, and paræsthesia are the most common symptoms. Their onset is gradual and accompanied by catarrhal symptoms. The symptoms may recur from time to time, and are much aggravated during the warm season. The disease may recur in incomplete form for ten or fifteen years.

Following the pain and weakness of the muscles, in some cases œdema becomes very pronounced, associated with effusions into the serous cavities. General anasarca is attended by palpitation and rapid action of the heart and dyspnœa. In this so-called wet or dropsical form atrophy of the muscles is not observed until the œdema disappears. In some instances the infection is very intense, and is characterized by more marked cardiac

symptoms. In these instances acute dilatation may be followed by cardiac paralysis and death in twenty-four or forty-eight hours.

The diagnosis is based upon the occurrence epidemically or endemically in tropical regions of peripheral neuritis with œdema. Thus far no bacteriological diagnosis has been made.

Constitutional Syphilis.

Intermittent, remittent, or continuous *fever* is attendant upon this infection some time during its course. (See Afebrile Infections, Chapter XXXII., Part I.) Failure to recognize the cause of this febrile phenomenon has led to many mistakes in diagnosis.

Constitutional syphilis may be acquired or congenital.

Acquired syphilis is characterized, (1) by the initial lesion, or chancre, which appears usually in about three weeks after contagion; (2) by a period of incubation generally lasting six weeks, but varying from one to three months; (3) by so-called secondary symptoms, comprising febrile symptoms, symmetrical polymorphous skin eruptions, ulcers upon the tonsils, adenitis, less frequently mucous patches in the mouth, or condylomata about the anus, iritis and retinitis, and loss of hair; (4) after an interval varying from several months to twenty years, by so-called tertiary phenomena, which manifest themselves in some cases. These are due to chronic inflammatory indurations of the skin and subcutaneous tissue, resulting in suppuration and ulceration; or of the bones, producing periostitis and necrosis; or of organs, producing gummata and cirrhosis; or of the nervous system, resulting in gummata or chronic degenerative changes. The lesions of this period are unsymmetrical.[1]

Course. The course of syphilis in different persons varies as widely as that of any of the eruptive fevers. In some the chancre is a mere papule which heals almost unnoticed; no secondary symptoms appear, and tertiary symptoms also are altogether wanting; or a chronic degeneration of the nervous system develops after the lapse of many years, the patient in the meantime remaining in apparent health. All this may occur, too, without the aid of specific treatment. In other cases the disease is malignant; tertiary symptoms appear very early or appear to take the place of secondary symptoms; ulceration may rapidly melt down and destroy the alæ of the nose or the soft palate; or rebellious periostitis with necrosis may attack the tibiæ, the nasal bones, or the cranium.

Secondary Symptoms. In an ordinary case of acquired syphilis, in about six weeks after the appearance of the chancre the patient complains of languor, weariness, slight fever, pains in the bones, and impaired digestion, and shows a tendency to anæmia. An *eruption* now appears. It is most marked on the trunk and upper extremities, especially the chest and forehead (*corona Veneris*). The eruption may be roseolous, squamous, vesicopapular, papular, pustular, bullous, or tubercular. The color has been aptly compared to that of raw ham. The enlargement of the inguinal, epitrochlear, and postcervical glands, which precedes the eruption, persists. Shallow *ulcers* with a sharply defined grayish outline appear

[1] *Fever* is a constant accompaniment of all forms of syphilis. (See Fever.)

on both tonsils. They are painless and do not spread. Ulcers are also liable to appear upon the pharynx, buccal surfaces, tongue, angles of the mouth, penis, vulva, vagina, and around the anus. In the mouth these are apt to be very painful, and may persist for weeks or months in spite of treatment. Relapses are not uncommon. Sometimes there are raised, white patches upon the pharynx. Sometimes the hair becomes very thin and falls out, leaving the patient without eyebrows and more or less bald. *Iritis* and *retinitis* are usually later symptoms. Other symptoms occasionally occurring at this stage are *periostitis*, usually slight, and *onychia*. The most common of the symptoms enumerated are the eruption and the tonsillar ulceration. The eruption comes out gradually during two or three weeks, and persists for about two months. Rarely, however, it is fleeting, or, on the other hand, is unduly prolonged. The secondary symptoms last from six to eighteen months. After their disappearance the patient may remain entirely well for life. In other cases after apparent health, lasting for months or years, the tertiary phenomena already mentioned appear. In the interval the patient may have suffered from various local skin eruptions or ulcers upon the buccal mucous membrane.

The *tertiary lesions* of syphilis are the late *syphilides* (see Skin) and *gummata* of the skin, subcutaneous connective tissue, muscles, or internal organs. Visceral syphilis is seen at this stage. In the brain and spinal cord gummatous tumors, gummatous meningitis, gummatous arteritis, and localized scleroses are found. The symptoms are those of brain tumor when the cerebrum is affected, and of tumor, meningitis, or sclerosis when the cord is affected. In syphilis of the lung we may find gummata scattered through the lung or a fibrous interstitial pneumonia beginning at the root of the lung. Diffuse syphilitic hepatitis or gummata may be found when the liver is affected. The rectum is the most common seat of syphilis of the digestive tract. Myocarditis and localized gummata and endarteritis occur in cardiac syphilis, while in vascular syphilis obliterating endarteritis and gummatous periarteritis are found. Syphilitic orchitis often occurs. Its presence may aid in the diagnosis of obscure visceral syphilis.

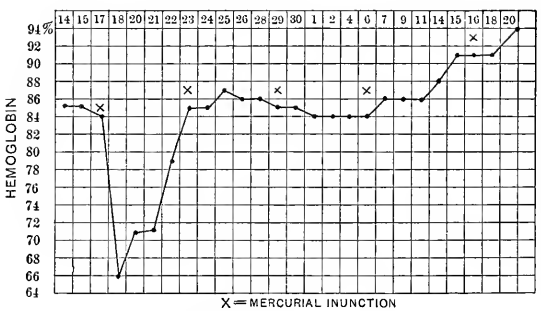
Hereditary syphilis differs in some respects from the acquired form. At birth the syphilitic infant usually exhibits no evidence of its inherited taint. In the course of from one to twelve weeks it develops a catarrhal inflammation of the nasal mucous membrane, which causes snuffling in breathing, and hence is called "snuffles." An *eruption* soon appears, symmetrical in distribution. It is most frequently erythematous or papular, but it may be squamous, vesicular, pustular, or bullous. In hereditary syphilis the eruption is more apt to be moist and to affect the genitalia and flexures of the thighs than in acquired syphilis; it has, however, the same ham color as is seen in the acquired form. Coincident with the "snuffles" and the eruption appear *stomatitis* and *ulcers* at the angles of the mouth, and sometimes condylomata around the anus. Meantime the child has begun to waste, to be peevish, to be anæmic, and gradually to assume the appearance of a wizened, dried-up old man. As in acquired syphilis, *iritis*, though uncommon, and inflammation of the other structures of the eye may occur, but nodes and disease of the liver are rare.

The infant very frequently dies during this period from exhaustion and inanition.

If the child survives for a year, the secondary symptoms usually disappear and the disease becomes latent. Relapses may occur, and in them, according to Hutchinson, condylomata are likely to appear. The same observer states that the tertiary period may begin at any time after the fifth year, but it is commonly delayed till about the period of puberty. In the meantime the patient may appear fairly well, but usually his development is retarded, there is a tendency to anæmia, and he has often nasopharyngeal catarrh, flattening of the bridge of the nose, premature decay of the upper incisor teeth, and a protuberant forehead. The *teeth* may be perfectly normal in cases which are characteristically syphilitic. The malformation, which was first described by Hutchinson, affects especially the upper central incisors of the permanent set, and "consists in a dwarfing of the tooth, which is usually both narrow and short, and atrophy of its middle lobe. This atrophy leaves a single broad (vertical) notch in the edge of the tooth, and sometimes from this notch a shallow furrow passes upward in both anterior and posterior surfaces nearly to the gum. The notching is usually symmetrical. It may vary much in degree in different cases; sometimes the teeth diverge, and at others they slant toward each other." (See Chapter VIII., Part II.)

Further, the patient may have had or may now be attacked with *keratitis* of both eyes, producing cloudy opacities and accompanied by considerable photophobia. Again, there may be nodes upon the long bones, with nocturnal exacerbations of pain. Cerebral deafness, according to Hutchinson, may occur, but cerebral blindness is rare. There may be ulceration upon the legs, and periostitis and necrosis. The patient usually recovers completely, but he is more liable to die from intercurrent disease than a healthy person, and in general has less resisting power, especially to tuberculosis.

FIG. 289.



Reduction of hæmoglobin after mercurial inunction in syphilis.

Diagnosis. The diagnosis of *hereditary syphilis* is based upon the occurrence of snuffles and skin eruptions, and the existence of keratitis or of cicatrices, especially about the angles of the mouth. A history of repeated miscarriages is suggestive of maternal syphilis. The

diagnosis of *acquired syphilis* is based upon the history of chancre, when that history is obtainable; upon the existence of polymorphous eruptions, or of non-traumatic ulcers upon the legs of young adults, or of scars in the groins or over the tibia, or of nodes, or of alopecia associated with sore throat or mucous patches. The presence of obscure disease of the bones, glands, or spinal cord should lead to the search for a possible syphilitic infection.

Examination of the *blood* during mercurial treatment may, in accordance with Justus' observations, show the presence of syphilis. If this disease is present, the percentage of hæmoglobin falls suddenly and rapidly during the hours immediately following the first administration of the drug. These observations have been confirmed by Cabot. The accompanying chart shows the effect of mercury upon the blood. (See Fig. 289.)

Weil's Disease.

The occurrence of *jaundice* without local hepatic symptoms during the course of *fever* suggests an infectious process. It is a well-known symptom of pyæmia and septicæmia. In the following infection, fever and jaundice are co-ordinate symptoms. *Acute febrile jaundice*, which rapidly becomes malignant, occurring in butchers, laborers, and brewers, has been described by Weil. After exposure to cold generally, as in a beer-vault, the patient is seized with a chill, followed by fever, with headache, vomiting, and epigastric pain. Jaundice sets in rapidly. The temperature remains high or may be intermitting. Stupor, delirium, and coma, albuminuria with suppression of urine, subcutaneous hemorrhages, and hemorrhages from mucous membranes, rapidly ensue. Black vomit occurs early. In one of my cases there was enlargement of the liver, with subcutaneous œdema over the hepatic area. The microscopical appearances were those of acute diffused parenchymatous inflammation. In another, a brewer, the liver was enlarged, but without unusual change, save congestion.

The delirium is sometimes violent. The appearance and symptoms suggest acute yellow atrophy of the liver, but the etiological distinctions are noteworthy: the liver is not small; leucin and tyrosin are not found in the urine; the jaundice is more intense. Yellow fever (*q. v.*) is excluded by the absence of the external conditions attendant upon epidemic and contagious diseases.

Miliary Fever.

The occurrence of *fever* in association with profuse *sweating* is rarely seen without attendant signs of pyogenic infection. When several cases with these symptoms occur at the same time, suggesting an epidemic, the following disease must be considered.

Miliary fever, or *sweating-sickness*, is an infectious disease, occurring in epidemics, and characterized by moderate fever, profuse sweating, tenderness and a sense of oppression in the epigastrium, and a vesicular eruption. The disease has occurred epidemically in England, but is

not met with now outside of France and Italy. After mild prodromal symptoms the disease sets in suddenly with moderate fever, profuse sweating, and epigastric distress, sometimes amounting to anguish. The characteristic eruption appears on the third or fourth day. It consists first of small reddish macules, in the centre of which a vesicle develops. The latter varies in size from that of a pinhead to that of a pea. The contents are at first clear, but subsequently become purulent. Desiccation and desquamation follow. The eruption is generally most profuse upon the neck and trunk. Sometimes there are marked nervous symptoms, and even convulsions and fatal collapse. Miliary fever is distinguished from rheumatism by the moderate fever and absence of joint-swellings, and from malarial fever by the absence of chills, of periodicity in the febrile movement, and absence of malarial organisms from the blood. The duration of the disease is from one to four weeks. The mortality in some epidemics has been very high, in others very low.

INFECTIONS RECOGNIZED BY THEIR RELATION TO ANIMALS.

Milk-sickness.

This is an acute disease affecting cattle, and transmitted from them to human beings in milk or meat. The disease is limited to a few sparsely settled localities west of the Allegheny Mountains. It is characterized by great *debility*, with muscular *tremor* upon motion (hence the name "trembles"), *vomiting* (hence called "puking fever"), a peculiar fetor of the breath, obstinate *constipation*, and moderate *fever* or subnormal temperature. The vomited matters are said to be of a peculiar soapy material, of yellowish or greenish color. The duration is usually less than a week. The patient may sink into a typhoid condition and die in coma, or he may die in a few hours. Convalescence is protracted.

Foot-and-mouth Disease.

A specific, infectious disease, communicated to man through cattle, sheep, or pigs, and characterized by a *stomatitis*. It is communicable by milk; the period of incubation is from three to five days. Invasion is characterized by slight *fever*, heat and soreness of the mouth, and the development of vesicles which burst and leave shallow ulcers. Saliva is freely poured out. The tongue swells greatly, and eating is painful. Vesicles sometimes appear about the fingers, but not upon the feet. The disease lasts from one to two weeks, and ends almost invariably in recovery.

Hydrophobia.

An acute specific disease communicated to human beings by the bites of animals similarly affected. The animals most frequently affected are the dog, fox, wolf, cat, and skunk; 90 per cent. of the cases in human beings are due to dog-bites.

The period of *incubation* is uncommonly long and very variable—from two weeks to two months usually. It is said in some cases to be a year or more. The disease has been divided into three stages—the melancholic, the spasmodic, and the paralytic.

In the *melancholic stage* there is pain, hyperæsthesia, or even reopening of the healed wound. The patient is extremely depressed in spirits and may be irritable. He seems to be laboring under a constant tension of fear, and is keenly sensitive to light, sounds, or draughts. He is affected with thirst, but his attempts to swallow water cause intensely painful spasm of the larynx.

The *second stage* is reached usually on the second day. The laryngeal spasms are increased and lead to intense dyspnœa and to pitiable struggling and gasping on the part of the patient. In addition to the convulsive seizures, the patient foams and froths at the mouth, and his face expresses the extreme terror and mental anguish he feels. The second stage lasts from one to three days, and is followed by the *third stage*, exhaustion intermitting with paroxysms of less severity. The patient may now be able to swallow easily, but there is great weakness of the heart, and death may occur from failure of the heart, from asphyxia, or in a convulsion. The duration, as indicated, is only a few days. The result is practically always fatal, but recovery may be possible. Bites of the face are the most likely to be fatal.

Glanders, actinomycosis, and anthrax are infections, the nature of which is suggested by the relation of the patient to infected animals.

INFECTIONS RECOGNIZED BY EXAMINATION OF THE BLOOD.

Microscopical Examination. *Fresh Blood and Smears.* (See page 564.) The following infections are recognized by the examination of fresh blood: *relapsing fever*, *malaria*, *yellow fever*, and *anthrax*. *Typhoid fever* may also be recognized by this means, but is more frequently diagnosticated by means of serum diagnosis and by culture-methods. By *staining* coverslip preparations of the blood the diagnosis by the direct method is confirmed.

Serum diagnosis enables us to determine the presence of typhoid fever, yellow fever, tropical dysentery, Malta fever, and possibly tuberculosis. (See page 552.)

Bacteriological examination of the blood corroborates the diagnosis of *typhoid fever* made by the above methods. By it we are also enabled to determine the presence of *gonorrhœal infection*, of *cerebrospinal meningitis*, of the *pneumococcus infection*, and, in many instances, of infection due to the *staphylococcus*, *streptococcus*, and *Bacillus coli communis*. The gonococcus infection alone will be considered.

It must be remembered that the micro-organisms can not be found in the blood until late in the course of the disease, and even then the infection must have a certain degree of intensity. Unfortunately, they can not be demonstrated in the majority of cases. Positive cultures for the

above reasons are very valuable. Negative cultures do not exclude septic infections.

Relapsing Fever.

Relapsing fever is the first infection to be considered, because historically it is the most important. It is the first infection in which a micro-organism was found to be causal, and is one to which Koch's laws can be applied. It is an acute infectious and contagious disease, occurring in epidemics, and characterized by the sudden onset of a febrile period lasting five or seven days, which is followed by an intermission lasting usually a week, and this in turn by a relapse lasting three days. Its development is favored by filth and famine, but the specific cause is believed to be the spirillum of Obermeier, which is constantly present in the blood during the febrile stage.

The stage of *incubation* lasts from five to eight days (Pepper), during which the patient may complain of malaise, lassitude, and flying pains. The *invasion* is sudden. It manifests itself by a chill or chills, frontal headache, pains in the back and limbs, vertigo, and great physical weakness. The temperature rises very rapidly, reaching 105° F., 106° F., or even higher, in the first day or two. The face is flushed, epistaxis sometimes occurs, the headache and other pains persist, but delirium is not common. The appetite is usually lost, thirst intense, the tongue coated white but moist, the bowels constipated. A mild catarrhal jaundice is not infrequent. Pepper states that nausea and vomiting are prominent symptoms, the matter vomited at times containing blood. Tenderness with pain in the epigastrium is frequently complained of.

The *urine* is scanty, high-colored, and frequently contains albumin and casts; when jaundice exists, the urine contains bile-pigment and sometimes blood.

There is no peculiar *eruption* in relapsing fever; but in this, as in other fevers, erythemata, petechiæ, and sudamina may be present.

The *pulse* is often very frequent and soft, and hæmic murmurs may be audible.

The *objective symptoms* are few. They consist of a flushed face, sometimes with slight jaundice and epistaxis, tenderness in the epigastrium, with moderate enlargement of the spleen and liver, and considerable cutaneous hyperæsthesia, with tenderness along the nerve-trunks. Bronchitis and sometimes hypostatic congestion of the lungs, with their usual physical signs, may be present.

These symptoms continue without much change until the fifth or seventh day, when a decided *crisis* occurs; the latter, however, is sometimes deferred until the tenth day. The *temperature* within twelve hours falls from 106° or 108° F. to or below normal; the pulse diminishes in frequency from between 120 and 130 to 60 or 70; vertigo, headache, and other pains disappear as by magic. The crisis is marked most frequently by a profuse sweat, sometimes by diarrhœa, epistaxis, metrorrhagia, or intestinal hemorrhage. The patient now enters upon convalescence without fever, and apparently makes rapid strides toward complete recovery. On the seventh day from the crisis, however, a sudden relapse occurs,

with a repetition of the symptoms of the first attack. The temperature may be higher and the febrile symptoms more severe, but the duration is shorter—only three or four days. The spirilla, which disappeared in the apyretic interval, are again found in abundance. A second crisis, with its associated symptoms, now occurs. The spirilla again disappear, and in the majority of the cases there is no further bar to complete recovery. A second, third, and even a seventh relapse may occur, as in a case reported by Pepper. Organic lesions do not follow this disease, unless they have occurred as complications; but even in ordinary cases the patient is left weak, anæmic, and with poor circulation. Relapsing fever occurs at all stages, but is most common in adults.

The duration varies according to the number of paroxysms. If there is only one, it is about eighteen days.

Microscopical Examination of Blood. In the blood at the height of the disease the spirillum of Obermeier is found. It is a slender, wavy, thread-like organism of spiral shape, seven or eight times the length of a red blood-cell, with a very lively forward movement in the direction of the long axis. The length varies from 16 μ to 40 μ, the width is 0.1 μ. Under a low power the blood may appear to be in motion as the result of their movement. They have so far been found only at the height of the febrile attacks; but von Jaksch states that so long as a relapse is threatened the blood contains peculiar, highly refracting bodies resembling diplococci, which are especially numerous before the attack; in some cases it has seemed to him that these diplococci at the very beginning of an attack develop into short, thick rods, from which the spirilla develop; they may, therefore, prove to be spores. Staining is unnecessary for the detection of the spirilla, but cover-glass preparations of the blood can, if desired, be stained with fuchsin or gentian-violet or Löffler's methylene-blue.

Serum Diagnosis. It sometimes happens that a diagnosis must be made during the afebrile period, when the organisms have disappeared entirely from the peripheral circulation. Löwenthal's method is as follows: A drop of the suspected blood is mixed with one containing the living micro-organisms. The mixture is sealed with wax between slide and cover-glass and left in the thermostat at 37° C. for half an hour. Blood from a patient who has just had a paroxysm will destroy the spirilla, so that they lose their motility and spiral curl, and accumulate in bunches. The reaction is like that of Pfeiffer's phenomenon rather than that of agglutination. It is to be remembered that the bactericidal power of the blood dies out before the next paroxysm.

Typical relapsing fever can also be produced by injecting infected blood into monkeys.

The most frequent *complications* are seen in the lungs, kidneys, and heart. Lobar pneumonia is the most frequent. The heart becomes weakened by the very high fever and thrombosis, or sudden failure results. Embolism is very frequent. Suppurative parotitis, abscess of the spleen, profuse epistaxis, abortion in pregnant women, and neuritis deserve mention.

Under the name "bilious typhoid" a malignant form of relapsing fever has been described. It is characterized by intensity of the symp-

toms of the ordinary form, and by bilious or bloody vomiting, jaundice, and delirium; or by collapse, with a purple nose, a small, frequent, and weak pulse, rigidity of the abdominal muscles, tenderness in the epigastrium, and a cold, clammy skin. In some of the cases described by Graves, intussusception of the intestines was found after death. In other cases uræmia is an active factor.

Diagnosis. Unless the blood be examined, the earlier cases in an epidemic may not be recognized until the occurrence of the characteristic relapse. The diagnosis is based upon the occurrence of an epidemic, the presence of the predisposing factors, the clinical course, and the examination of the blood. Relapsing fever is most likely to be mistaken for *typhus fever*, which occurs under similar conditions. The aspect of the two diseases is very different. In typhus there is a heavy, stupid, sometimes besotted expression, with slight redness of the eyes and a contracted pupil. The patient lies oblivious of his surroundings, with low muttering delirium and ataxic symptoms. In relapsing fever, on the other hand, the sensorium is rarely much disturbed, the spleen and liver are enlarged, and there is hyperæsthesia. Moreover, in typhus there is a spotted eruption, later becoming petechial. In relapsing fever this is absent.

Anthrax.

The next infectious disease, the cause of which can be determined by an examination of the blood, is anthrax. This affection is also of historical importance, and is probably the best worked out of any of the infections common to man and the lower animals. It is also called malignant pustule, charbon, wool-sorters' disease, or splenic fever. It is derived principally from herbivorous animals, and characterized by the development of a pustule or boil, with extensive brawny œdema and subsequent toxæmia; or toxæmia may appear first and metastatic abscesses subsequently. The disease also attacks the gastro-intestinal mucous membrane and the lungs.

Anthrax is caused by the anthrax bacillus and its toxins. Outside of the body it forms endogenous spores, which are extremely tenacious of life, and to which infection is invariably due. They infect not only the carcasses of animals, but also the soil, and utensils used in the care of the animals; and they persist with infective power in the hides, hair, hoofs, and wool. It is possible that it may be transmitted to man by stings of insects, particularly flies and mosquitoes.

The period of incubation varies from a few hours to several days. In the form known as *malignant pustule* the patient has a prickling or burning feeling, which may lead him to think he has been stung by an insect on some exposed part of the body, particularly the hand, face, or neck. At the seat of irritation, first a papule, then a vesicle develops. The vesicle may attain considerable size. The contained fluid quickly passes from clear to bloody, and then escapes leaving a dark-brown or black scab (anthrax).

The original vesicle may be surrounded by a series of smaller ones. Instead of disappearing, the base of the vesicle becomes inflamed and indurated, the induration extending to surrounding tissue and causing a

condition of brawny œdema. A whole arm or one side of the face and neck may be swollen. There may or may not be an associated lymphangitis and adenitis.

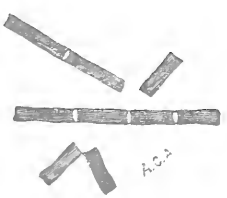
The general health does not suffer at first, but in a day or two fever sets in, accompanied by delirium, sweating, great weakness, enlargement of the spleen, severe pains in the limbs, and diarrhœa. Death, preceded by collapse, may occur in from five to eight days (Fagge), or the tissue occupied by the pustule may slough out.

Bollinger and others have called attention to *anthrax œdema*, in which there is no pustule, but only a yellowish or greenish swelling of the tissues. Gangrene may ensue. It is seen most frequently in the eyelids, but may be on the head, hand, or arm.

Intestinal Form. Anthrax of the gastro-intestinal mucous membrane, as described by Bollinger, presents the following symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid; the spleen is enlarged. Dyspnœa and lividity appear, with restlessness and excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms, there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

Wool-sorters' Disease. Still another form of anthrax occurs among the wool-sorters of Bradford, England; it is characterized by intense dyspnœa and a feeling of oppression or constriction. Breathing is labored, but not much accelerated. Only a few coarse râles are to be heard on auscultation. The expectoration may be abundant and bloody, or absent. There is a tendency to collapse, with cold, bluish skin, and a subnormal axillary temperature. The rectal temperature, however, is raised two or three degrees. Death may occur in coma and convulsions, or the patient may die suddenly, the mind being clear. The duration of the disease is from one to five days. Bell says that those who survive for a week generally recover.

FIG. 290.



Bacillus anthracis, highly magnified, to show swellings and concavities at extremities of the single cells. (ABBOTT.)

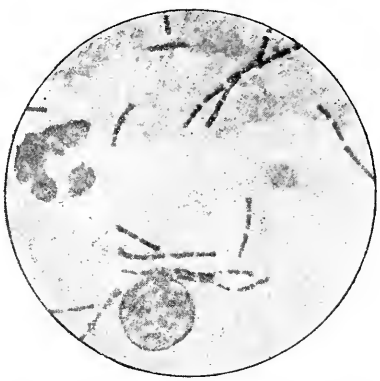
Examination of Blood. *Bacillus anthracis* is found in the blood of the patient or the pus of the lesions of anthrax or malignant pustule.

Morphology. A bacillus, 2 μ to 3 μ up to 20 μ to 25 μ in length and 1 μ to $1\frac{1}{4}$ μ in breadth. The bacilli are often joined end to end in long threads, which are massed in bundles. As found in animals they are short rods with square ends. They stain best with Löffler's blue, but also with the basic anilines and by Gram's method. When in the stage of spore-formation, the threads look like strings of beads.

CULTURES. They support the diagnosis.

Inoculation. When *inoculated*, the organism produces the pustule of anthrax. If inoculated into the abdominal wall of a guinea-pig or rabbit, death follows in forty-eight hours. No reaction is seen at the point of inoculation, but beyond this the tissues are œdematous. Ecchymoses

FIG. 291.



Bacillus anthracis in the blood of a guinea-pig. × 1040. (GIBBS.)

are seen, and the underlying muscles are pale. The spleen is enlarged, dark in color, and soft. Cover-slip preparations confirm the diagnosis.

Anthrax bacilli are not so numerous in human blood as in that of the lower animals. They are most likely to be found in the spleen, which is apt to be much swollen.

Diagnosis. In doubtful cases a mouse or guinea-pig should be inoculated with the blood. *Carbuncle* is distinguished by its tendency to develop upon the back or shoulders and other covered portions; anthrax is more apt to occur on uncovered portions of the body. In carbuncle there is a series of openings resembling a sieve, filled with pus and plugs of necrotic tissue. In anthrax there is at first a central black crust. The boggy feeling of carbuncle is different from that of the brawny œdema of anthrax. Finally, in carbuncle, anthrax bacilli are not found in the blood.

The intestinal and thoracic forms are distinguished by the occupation of the patients, the absence of other adequate cause, and the result of the blood-examination, cultures, and inoculation experiments.

Malarial Fevers.

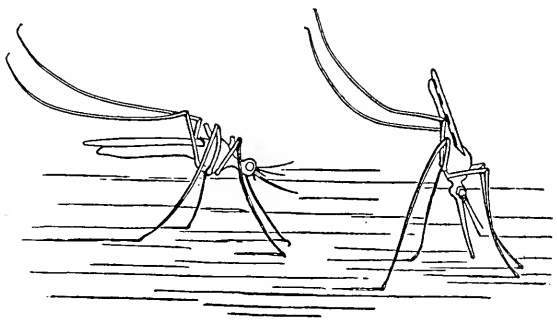
The next infection which will be considered is one of the most common the world over. In its various forms it is recognized by direct

examination of the blood. Its clinical features are such that often but little difficulty surrounds its recognition, but no case should be unqualifiedly pronounced malaria without an examination of the blood. It comprises a group of fevers associated with the protozoan organism of Laveran, and is characterized by periodic paroxysms of chill, fever, and sweat. Malaria is not contagious, but can be transmitted by inoculation.

Malarial fevers, while most prevalent in tropical and subtropical regions, are found also throughout the temperate zone, especially in autumn and spring. In Europe their favorite habitat is Italy, and in the United States the Southern and Southwestern States. Conditions that especially favor their development are marshes and swamps fed partly by sea-water, low ground along streams of slow current, and freshly upturned soil.

The protozoan organism described by Laveran exhibits several different forms, which he regards as stages in the development of one organism, but which may be different species. Golgi maintains that there are several distinct varieties of parasites whose periodicity in development

FIG. 292.



Culex and *Anopheles*. *Anopheles* is recognized by its spotted wings and tilted attitude.

and sporulation corresponds with the different types of fevers. This *Plasmodium malariæ* passes through one cycle of its development in the body of a variety of the mosquito known as *Anopheles claviger*. The disease is contracted by the inoculation of the human subject by the infected mosquito.

Intermittent Fever. This is a type of malarial fever in which the temperature remains normal between the paroxysms.

A malarial paroxysm is characterized by (1) chill, (2) fever, and (3) sweating, occurring in the order named and in immediate succession. The time between the beginning of one paroxysm and the beginning of the next is called the "interval," that between the conclusion of a paroxysm and the beginning of the next the "intermission." The interval varies in different forms of intermittent fever: in the quotidian there is a paroxysm every day, with an interval of twenty-four hours; in the tertian there is a paroxysm on alternate days, with an interval of forty-eight hours; in the quartan there is a paroxysm every third day, with an interval of seventy-two hours. In double quotidian there are two paroxysms in the twenty-four hours, but not of the same intensity.

In the double tertian there is a paroxysm every day, the first and third and second and fourth corresponding as to hour and intensity. That is to say, if there be a paroxysm at 10 A. M. Monday there will be another severe paroxysm at 10 A. M. Wednesday, while on Tuesday and Thursday there will be milder paroxysms, but at another hour than 10 A. M.

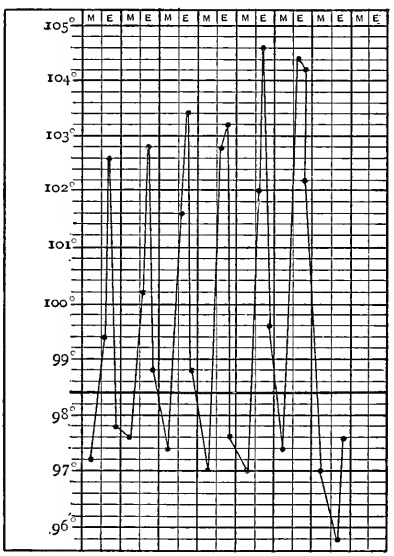
In the double quartan severe and mild paroxysms succeed each other every other day, but each third day is free from paroxysm.

While the rule is for malarial fevers to occur periodically at the same hour, if the disease is growing worse, the second paroxysm may occur an hour or two earlier (anticipation), or, if it is growing better, an hour or two later (postponement). (See Figs. 74, 75, 76.)

Quotidian intermittents are slightly more common than tertian, while the quartan variety is rare.

The *incubation*-period probably varies widely, depending upon the intensity of the poison. As a rule, repeated exposure is necessary to develop the disease in temperate climates. During the period of incubation the patient may suffer from headache, drowsiness, pains and aching in the limbs and back, constipation, a coated tongue, and thirst.

FIG. 293.



Intermittent fever. Temperature every six hours. Morning and evening temperature; highest at chill.

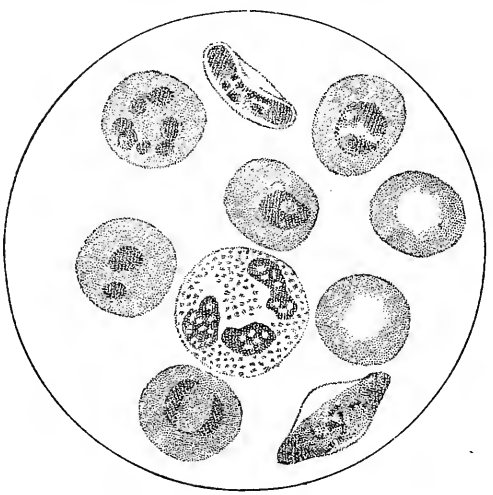
The *onset* of a typical malarial paroxysm is marked by chilly sensations, especially along the spine, accompanied by yawning and the development of "goose-flesh." Then a decided chill sets in, the patient shaking violently. The face is pale and pinched, the lips blue, the nose pointed; as the chill becomes worse the teeth chatter, the whole body feels cold, the skin feeling rough, dry, cold, and harsh. The finger-nails and toe-nails

are blue, the skin being wrinkled upon the palmar and plantar surfaces. The superficial bloodvessels are so contracted that a drop of blood is obtained with difficulty. The voice is thin and weak, almost inaudible.

The volume of blood driven from the surface leads to congestion of the viscera, particularly the spleen, liver, and stomach. Nausea and vomiting are not uncommon. The spleen is perceptibly enlarged, and frequently, also the liver.

Although the surface temperature is depressed, the internal *temperature* is rising, and may be two or three degrees above normal. By degrees the severity of the chill abates and the patient asks to have the extra bed-clothing removed. Reaction sets in; the surface bloodvessels dilate and the skin becomes flushed. The temperature continues to rise, often reaching 103° to 106°, pulse and respiration increasing correspondingly in frequency. The patient complains of a throbbing, dizzy headache, and vomiting may recur. The bowels remain constipated. The temperature now begins to fall, and the sweating stage succeeds. Perspiration appears first upon the forehead, face, and neck, and gradually extends over the rest of the body. The perspiration becomes more and more profuse, until

FIG. 294.

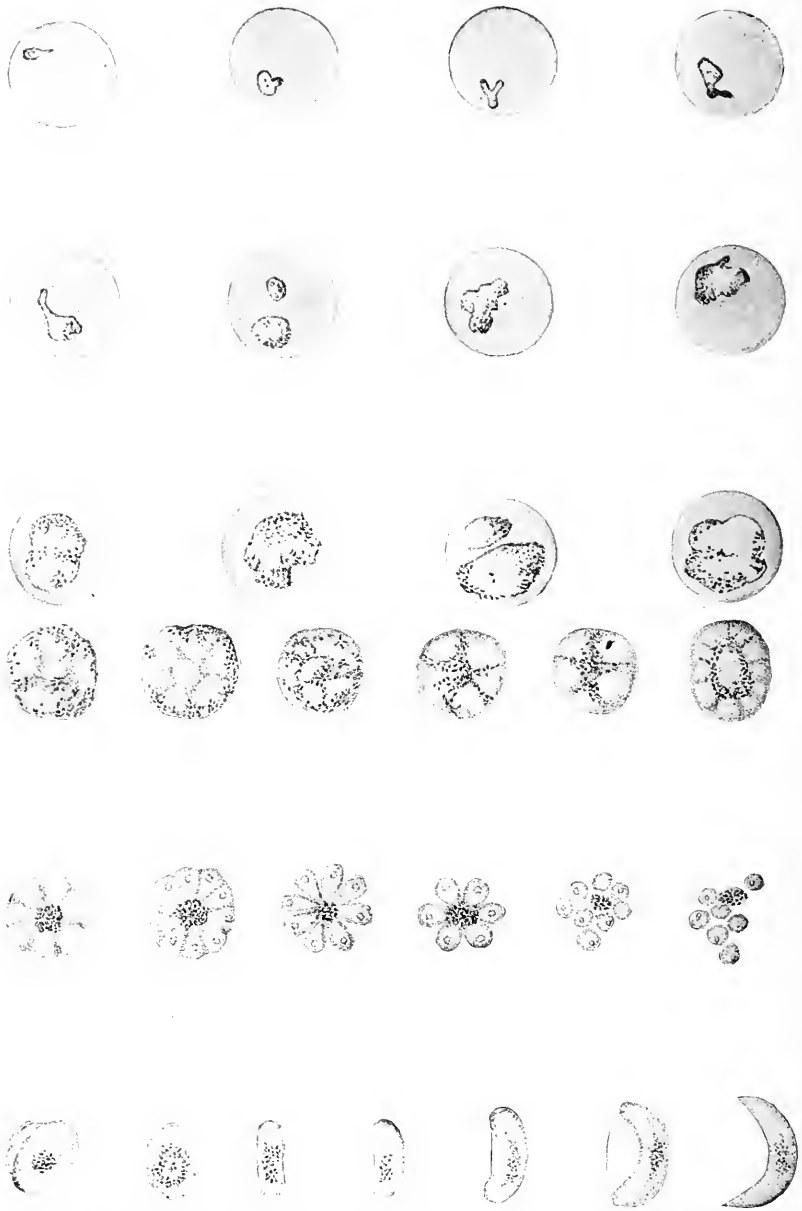


Malarial plasmodia. (Reproduced from colored plate.) To the right, two normal red blood-cells with central depression. In addition, several others with bluish contained bodies and pigment-sprinkled cells, which show the endogenous development of the plasmodia. Besides, two of Laveran's bodies, one exhibiting a delicate little basket appearance. Near the centre a polynuclear white cell with bluish nuclei and red granulation. (H. RIEDER.)

the whole body is drenched with it. All the subjective symptoms vanish with wonderful rapidity, and the patient, with the exception of exhaustion, seems to be restored to complete health. The hot stage lasts from one to two hours, the cold stage from three to eight hours, and the sweating stage from two to six hours.

In the interval between paroxysms the patient is free from fever, but is anæmic, weak, and has impaired appetite and constipation. During the entire paroxysm the mind remains clear.

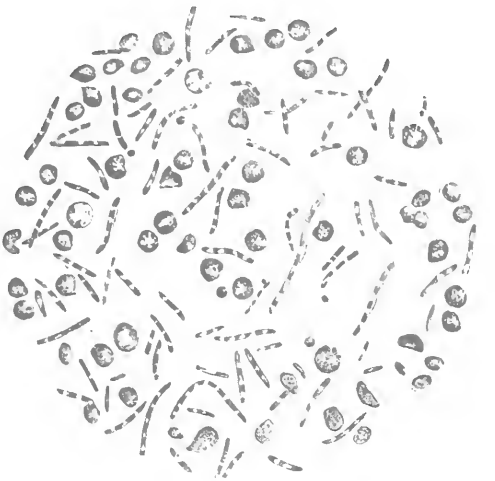
FIG. 295.



The first twelve figures show the malarial plasmodium. It is a pale amœboid body inside the red corpuscle. It increases in size at the expense of the corpuscles. In the last four of the twelve it is enlarged and contains pigment-granules derived from the hæmoglobin. The figures of the fourth row show progressive stages in the process of cleavage of the plasmodium and shifting of the pigment-granules. In the fifth row the process of cleavage is seen to be completed, and final isolation of the spores has taken place. The dark granules are pigment-granules. The last row shows oval parasites—Laveran's corpuscles observed in atypical cases of malaria. (From GOLGI, "Studien über Malaria," *Fortschritte der Medicin*, Bd. iv., Tafel III.)

PLATE XVI.

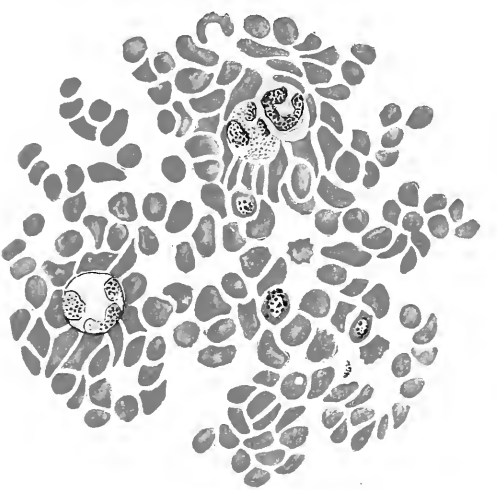
FIG. 1.



Anthrax-bacilli from Rabbit's Spleen.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

FIG. 2.



Protozoa of Malaria, Intracellular and Crescentic Forms.

(Oc. 4, ob. $\frac{1}{12}$ immersion.) Drawn by J. D. Z. Chase.

The chief *objective symptom*, apart from the phenomena of chill, fever, and sweat already described, is the occurrence of plasmodia in the blood. (See Plate XVI., Fig. 2; and Fig. 294.)

Examination of the Blood. The plasmodia of malaria were first pointed out by Laveran. They have been studied in Italy, especially by Marchiafava and Golgi, and in this country by Councilman, Osler, and Dock. Minute amœboid bodies are found in the red corpuscles. These become pigmented with altered hæmoglobin, and grow until they fill nearly the whole of the cell, the pigment being arranged chiefly in a peripheral ring. Later, the amœboid bodies become spherical and transparent, the pigment collecting in the centre. Sporulation now begins, and a fresh crop of small rounded parasites appears, to begin the same cycle over again in fresh corpuscles. (Plate XVI., Fig. 2.)

Three forms of parasites are described: 1. The *tertian*, which sporulate at the end of forty-eight hours, begin as small amœboid intracorpuscular bodies, gradually enlarge, produce fine brownish pigment-granules, and finally completely fill the corpuscle. In sporulation the segments number fifteen to twenty.

2. The *quartan*, which sporulate at intervals of seventy-two hours, are smaller; amœboid movement is not so marked; when full grown, the parasites are smaller, and the corpuscles tend to shrink about them and to become a deeper greenish color. They sporulate with five to ten segments, in a very beautiful characteristic rosette appearance.

3. The *æstivo-autumnal* are smaller, and contain less pigment. The period of sporulation is still in dispute. They usually form ovoid, crescentic or round bodies with coarse pigment-granules in the centre.

Golgi maintains that in tertian malarial fever the period between invasion of the corpuscles and the sporulation is two days; in quartan, three days, the difference in cycle being due to a difference in the parasites.

The onset of the fever corresponds in time to the division of the parasites.

The crescentic form described by Laveran is said to be more common in the irregular forms of malarial fever. Canalis says that it only makes its appearance several days after the beginning of the fever. It is somewhat longer than a red blood-cell, and the pigment tends to collect in a focus about the middle of the parasite. Subsequently it becomes oval and divides into eight or more daughter-cells.

Another form with flagella is occasionally found. Councilman says it is most common in blood drawn directly from the spleen.

The plasmodium of malaria may be *stained* as follows: Cover-glass preparations of the blood spread very thinly are dried in the air and fixed by immersion for twenty minutes or half an hour in a mixture of equal parts of alcohol and ether. They are then stained for twenty to thirty minutes in concentrated aqueous solution of methylene-blue, 60 parts; 0.5 per cent. solution of eosin in 75 per cent. alcohol, 20 parts; distilled water, 40 parts; 20 per cent. solution of potassium hydroxide, 12 drops. The cover-glasses are then washed in water, dried, and are then ready for mounting. The red blood-cells are stained rose, the nuclei of leucocytes a deep dark-blue, and any plasmodia a delicate sky-blue.

Aronson and Phillips' staining method is as follows: Make concentrated aqueous solutions of orange-G, acid rubin, and crystallized methyl-green, leave them to settle, then mix in these proportions: orange-G. 55; acid rubin, 50; distilled water, 100; and alcohol, 50. To this add methyl-green, 65; distilled water, 50; and alcohol, 12. Leave the mixture standing for a week. A well-diluted solution should be used for staining purposes; one drop of the mixture should be added to 25 c.c. of water; the stain should be left on for twenty-four hours and the fixing of the preparations, done before staining, carried out at a temperature of 120° C. As a result the red corpuscles are stained orange, nuclei greenish blue, neutrophile corpuscles violet, and eosinophile red.

One of the best stains is that of Romanovsky: 1 per cent. watery solution of eosin, 2 parts; saturated aqueous solution of methylene-blue, 1 part, in watch-glass. Put the specimens, prepared by heat or alcohol, face downward, floating on top of the solution; cover with another glass. Keep in a moist chamber. Stain one-half to three hours; better two hours. The plasmodia are stained a clear blue; the corpuscles a pale red.

Another good stain is made as follows: 0.5 per cent. aqueous solution of eosin, 1 part; saturated solution (aqueous) of methylene-blue diluted one-half with distilled water, 1 part. Stain as is Romanovsky's method for twenty-four hours. The results are the same.

Futcher has called attention to an extremely valuable method. Upon a dried cover-glass specimen a little of a 1 per cent. solution of formalin in 90 per cent. alcohol is poured and allowed to remain for one-half to one minute. The specimen is dried between leaves of filter-paper; absolute alcohol is then poured over the glass, which is once more dried, and then stained for twenty to thirty seconds in Marchand's solution of phenol-thionin, which is prepared as follows: saturated solution of thionin in 50 per cent. alcohol, 20 parts; 2 per cent. solution of carbolic acid, 100 parts. The solution must stand several days.

The specimen is then washed in water, dried between filter-paper, and mounted in balsam. If the specimen has not been stained too long, the corpuscles take a very slight greenish hue, while the parasites are of a deep-violet color. The preparation should not be stained too long, as in pigmented parasites granules of pigment may be obscured by the depth of the color. This method is particularly valuable inasmuch as it brings out with great clearness the small ring-shaped hyaline bodies of æstivo-autumnal fever with their chromatin dot.

Examination of the blood discloses the presence of a high degree of anæmia. The hæmoglobin is usually diminished in greater proportion than the corpuscles. There is a marked reduction in the leucocytes. This leucopenia is most marked after a paroxysm. There are a relative diminution of the polynuclear forms and a relative increase in the mononuclear forms. In severe post-malarial anæmias, as Thayer points out, the blood is characteristic of pernicious anæmia.

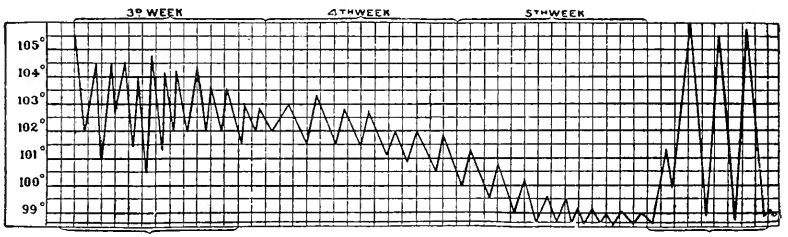
Irregular Form. Irregular forms of intermittent fever are more common in Philadelphia than the typical form just described.

In the *mild form* the patient complains of great lassitude, irritability of temper, and drowsiness during the day, but at night tosses upon his

bed and gets up in the morning more tired than when he went to bed. The back and limbs ache, and the latter feel as though they would give way under him. There is severe throbbing headache, with some dizziness and faintness. The bowels are constipated; the tongue heavily coated with yellow fur. The temperature is moderately elevated and the patient has great thirst. Nausea and vomiting are absent, though there is little desire for food. There may be a burning feeling referred to the splenic region. The patient is worse on alternate days, and the attacks may be preceded by slight creeping chills. On inquiry the patient will be found to live in a low-lying district near one of the rivers, or in a damp house over an unclean, moist cellar, or adjoining a place where soil has been freshly upturned.

In the form known as "*dumb ague*," there is a periodically great depression, with aching in the head and limbs, a sensation of coldness rather than chilliness, but no marked fever and sweating. Nausea and vomiting may, however, be present. Da Costa says he has seen it manifest itself by excruciating pain over the kidney and almost entire suppression of urine. There may also be severe paroxysms of gastralgia. It is more common in old residents of malarial districts.

FIG. 296.



Cold tub baths. Abundant malarial organisms.

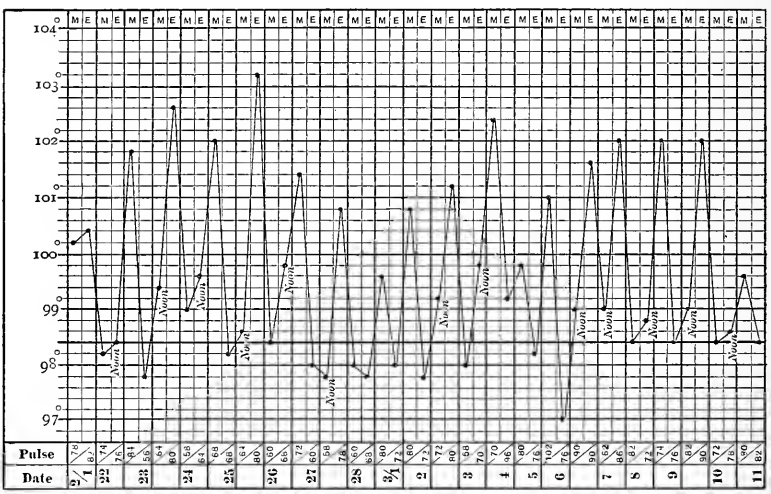
Malarial fever associated with enteric fever. (THOMPSON.)

In *masked* malarial fever the poison manifests itself in an attack of neuralgia, especially of the supraorbital nerve and gastric nerves. Malaria may also be latent until some impairment of the resisting power brings it to light. Hence, it appears as a complication of pneumonia and dysentery and typhoid fever (Fig. 296), especially in the southern and southwestern portions of the United States. Moreover, women who have previously had intermittent fever may suffer a recurrence after confinement; it must not be confounded with so-called *puerperal malarial fever*, which is unfortunately more common and of infectious origin.

Diagnosis. The essential points in the diagnosis of intermittent fever are the periodical recurrence of paroxysms of chill, fever, and sweating, or of attacks of dumb ague, or of paroxysms of neuralgia, without organic lesion, associated with the presence in the blood of pigment and plasmodia, and with enlargement of the spleen and possibly of the liver. The so-called therapeutic diagnosis may be made—an intermittent fever which does not yield to proper doses of quinine in three days is not malarial. A typical malarial intermittent fever is not likely to be mistaken for anything else. (See Fever, pages 263, 264.) It needs, how-

ever, to be distinguished from *septicæmic fever*, due to absorption into the blood of pus and the toxins produced by bacteriological growth. Such fever occurs in *tuberculosis*, especially in the stage when cavities form and pus collects; in the puerperal state, in empyema, subphrenic abscess, abscess of the liver, or, indeed, in any form of suppuration. Here, also, there are recurring chills, with fever and sweating, but the attacks are not regularly periodical and intermittent; sometimes the fever is intermittent and sometimes remittent, the chills recur at irregular intervals, and are not so violent as in the malarial attack. The essential difference, however, lies in the fact that a local cause can be found to explain them, such as tuberculosis either of the lung or of some other viscus, or a collection of pus in an organ or cavity, or a fetid discharge from the womb, with local tenderness or peritonitis; moreover, the patient loses flesh more or less rapidly, his blood is free from malarial germs and pigment, and quinine does not control the fever. (Plate XVI., Fig. 2.)

Fig. 297.



A form of intermittent fever from syphilis. J. D., aged twenty-six years. Secondary period. Mercury and potassium iodide relieved it. Observe that the pulse-frequency is not increased.

It is more difficult to make a diagnosis between malaria and the intermittent fever of *hepatic* origin (described elsewhere by the author) because physical signs of any local trouble may be wanting. But the fever of hepatic origin is not regularly intermittent, is not controlled by quinine, but may be by measures directed to the origin of the trouble, and jaundice may be present.

Urethral fever, occurring as the result of operations upon the urethra, or simply from the passage of a catheter or bougie, may be mistaken for malarial fever; but the paroxysm is usually single, and the history of the operation and the absence of plasmodia from the blood clear up the diagnosis.

Syphilitic fever is distinguished by a tendency for the chill, fever, and

sweating to be nocturnal in recurrence, and by evidence of a syphilitic infection coupled with absence of malarial organisms from the blood.

Remittent Malarial Fever—Æstivo-autumnal Type. A type of malarial fever characterized by a remission instead of an intermission in the febrile paroxysms. It is due either to a great intensity of the malarial poison or to a different species of organism. It is much more rare in temperate climates than either quotidian or tertian intermittent, and is attended with more gastric disturbance and a much larger mortality (twelve times greater, according to the statistics of the War of the Rebellion).

The *onset* is more abrupt than in intermittent fever. Prodromata are not so common, but when they occur they are of the same character. The chill is not usually so violent, nor the cold stage so long as in intermittent fever; on the other hand, nausea and vomiting are common, and in some cases there are bilious vomiting and diarrhœa, tenderness over the stomach and spleen, and sometimes jaundice. The temperature rises rapidly from 103° to 106° F., and remains high for a longer time than in intermittent fever, the hot stage lasting in severe cases from six to eighteen or twenty hours. During this stage the patient suffers from headache, pains in the back and limbs, great thirst, and gastric irritability. A remission now succeeds. The temperature falls two or three degrees, but not to normal; free sweating occurs, the nausea and vomiting cease, and the patient becomes much more comfortable. He may fall asleep from exhaustion, but if awake is conscious of weakness, aching in the limbs, and perhaps nausea. In the course of some hours the temperature again rises, often to a higher point than before, but frequently without antecedent chill. The same subjective symptoms are repeated, and another remission follows. Daily paroxysms usually occur, those on alternate days being severe. The temperature often reaches its highest point at the third paroxysm. The disease generally runs its course in from nine to twelve days, but it may last much longer. The type of fever may change to intermittent, which is a favorable sign, or become continued and again remittent, or remain remittent throughout; finally, the fever may subside gradually, or, less commonly, by crisis. The urine is febrile but not albuminous. (See Examination of Blood, page 743.)

Pernicious Malarial Fever. This, as the name implies, is a form of malarial fever with destructive tendency. It is also called malignant and congestive fever. It may be intermittent or remittent. Nearly 24 per cent. of the cases occurring in the U. S. Army from May 1, 1860, to June 20, 1866, proved fatal.

Bemiss[1] divides this variety of malaria into three classes: the algid, or congestive, form; (2) the comatose form; (3) the hemorrhagic form. To this another class, (4) the gastro-enteric form, may be added. It is important to remark that the first paroxysm does not usually, in any of these forms, indicate that the type of the disease is pernicious. The first seizure may, however, prove fatal.

1. The *algid form*, according to Bemiss, occurs more frequently than any other, its perniciousness being due to an aggravation of the cold stage of an intermittent attack. The patient is extremely weak, with cold

[1] Pepper's System of Medicine, 1885, vol. i., p. 666.

extremities, pinched features, blue lips, and faint voice. Respiration is shallow, the pulse rather slow, feeble, and irregular; he is further exhausted by vomiting and liquid, offensive diarrhœa, the passages sometimes being involuntary. There may be copious perspiration, but the internal temperature is very high. The mind may be clear, or there may be deep stupor. Unless speedy relief can be afforded the attack ends fatally.

2. In the *comatose form* the patient is completely unconscious, the skin hot "and of a muddy, semi-jaundiced hue." (Bemiss.) Both pulse and temperature are increased. In other cases coma is preceded by wild delirium, resembling acute meningitis. The comatose form is most apt to occur in those who continue to reside in a malarious region without proper safeguards against its poisonous influences.

3. In the *hemorrhagic form* there has been, as a rule, previous alteration of the blood, the bloodvessels, and other tissues, from long-continued malarial poisoning or cachexia. Then, when intense congestion of these parts occurs as the result of the surface-chill, hemorrhage follows. In some districts, however, and at certain seasons, there has been a special predilection of the poison for the kidney, with resulting hæmaturia. The prominent symptoms are a prolonged chill with high temperature; nausea and vomiting, sometimes with the expulsion of a greenish-black fluid; œdema of the lower extremities; general anasarca, occasionally œdema of the lungs, and hydrothorax; bloody and albuminous urine, with tube-casts; and intense jaundice. Pain in the right hypochondrium or over the kidneys is common. Bemiss asserts that uncomplicated malarial fever has not a hemorrhagic tendency.

4. The *gastro-enteric form* has for its prominent symptoms nausea, vomiting, diarrhœa, intense thirst, extreme restlessness, a frequent feeble pulse, and urgent dyspnœa. "The breathing is deep-drawn; each expiration is followed by two short inspirations." (Da Costa.) The patient is cold and partly collapsed. Reaction may or may not occur. The patient may have several paroxysms of pernicious malarial fever and succumb in any one of them. Convalescence is slow. The most frequent sequelæ of malarial fevers are anæmia, neuritis, paralyses, and malarial cachexia.

Typhoid fever is distinguished from pernicious malarial fever by its gradual onset, the absence of chills and vomiting, as a rule; and, on the other hand, the presence of epistaxis, delirium, and ataxic symptoms, tympanites and diarrhœa, with pale-yellow, watery stools, and rose-colored spots. The temperature in typhoid is more continuously high, the daily oscillations being of shorter range. A history of exposure to malarial infection and of previous attacks can often be obtained. The urine of typhoid exhibits the diazo reaction; that of malarial fever does not. The results of the blood examination (Widal's test) and the bacteriological studies would settle the diagnosis in most doubtful cases. It must be remembered that a mixed infection occurs sometimes, in consequence of which the plasmodium of malaria may be found either at the beginning or in the decline of the typhoid infection.

Malarial cachexia occurs especially in those who have lived for a long time in malarious regions, and who may or may not have had typical

malarial attacks. The patient suffers from dyspepsia and constipation, with occasional bilious attacks; the face is of a pale lemon-yellow color, and may be slightly jaundiced; there is marked anæmia, with pigment and crescentic and flagellate forms of plasmodia in the blood, together with great enlargement of the spleen (ague-cake) and some enlargement of the liver. The patient is weak and languid, and sometimes has considerable mental depression.

SERUM DIAGNOSIS.

The infections just described are recognized by an examination of fresh blood or cover-slip preparations. The next group of infections may be recognized by serum diagnosis. Too much stress must not be placed upon this method of diagnosis, yet its value is so great that one is fully justified in giving it a high place among the precise methods of diagnosis of infections.

Typhoid Fever.

The first of the infections to which such diagnosis has been applied *in extenso* is typhoid infection or typhoid septicæmia. This infection is caused by *Bacillus typhosus*. Its most common expression is a symptom-complex which attends a septic process and local intestinal ulceration combined. This symptom-complex is known as *typhoid fever*. In rare instances the infection is stated by some to be unattended by fever. More frequently a febrile course, following a definite continued type and lasting from twenty-one to twenty-eight days, prevails. In mild or abortive forms the fever rarely reaches 103° F., and declines from the seventh to the fourteenth day. In the grave forms the fever is often very high and attended by cerebrospinal, renal, pulmonic, or severe gastro-intestinal symptoms.

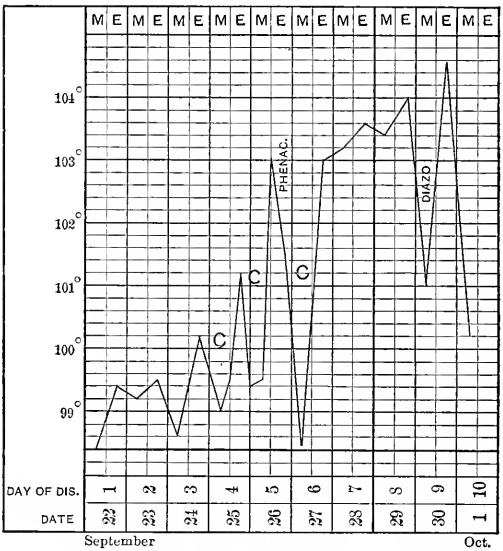
The most important infection prevailing in the temperate zone is the one we are now about to consider. It is an acute, specific, infectious, and mildly contagious disease, characterized by a gradual onset, a continued fever, an eruption of rose-colored spots, marked nervous and abdominal symptoms, and an average duration of three or four weeks.

It occurs sporadically and epidemically, and in large cities is apt to be endemic. Its special habitat is in temperate climates, but it may occur anywhere. It is relatively rare in the southern and southwestern portions of the United States. It is more frequent in the latter part of the summer and in the autumn and winter, and following hot and dry summer weather. Young adults are especially prone to it, but cases have occurred at all ages. Change of residence from the country to the city is a predisposing factor. Those living in cities often acquire immunity, but they may lose it upon moving elsewhere. The state of previous health does not seem to have any influence.

Incubation. The period of incubation in typhoid fever varies from four or five days to three weeks; more commonly it is from one to two weeks. During this time the patient usually is languid, becomes tired easily upon exertion, has severe headache, sleeps poorly, and his rest is

disturbed by dreams. There is often, even thus early, a dull and listless expression of the face. Toward the close of this period, and in severe cases, there may be colicky pain in the abdomen, a tendency to looseness of the bowels. cough, epistaxis, mental sluggishness, and chilliness.

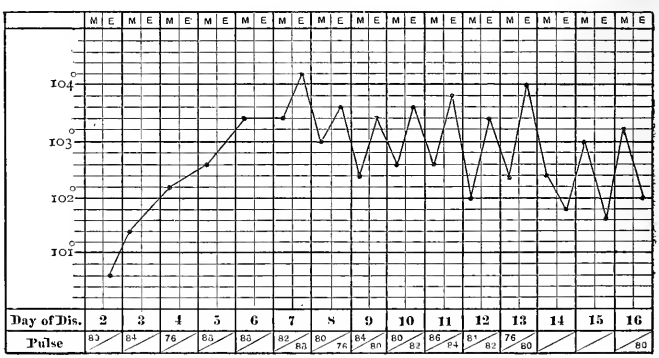
FIG. 298.



Temperature ranges; first week of typhoid fever. (DOCK.)

Pepper was led repeatedly to anticipate the approach of typhoid fever by the unusual dulness of hearing and by the persistent occipital headache coming on after a few days of general malaise.

FIG. 299.



Mild typhoid fever. Gradual ascent. (Original.)

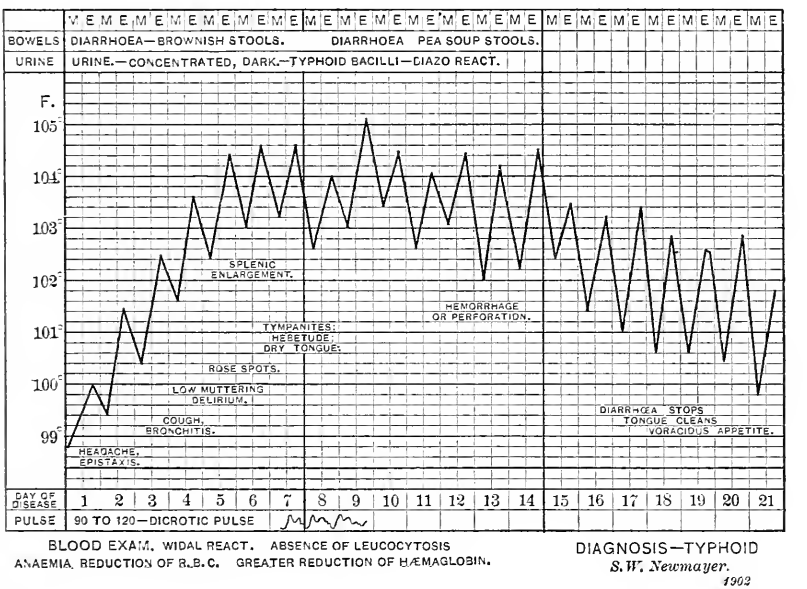
While the disease may begin abruptly, a gradual onset is so much the rule that it becomes an important factor in the diagnosis from other disease-conditions.

Invasion. Invasion is not sharply marked. There may be chilliness, but a decided chill is unusual except when pneumonia is part of the initial process. Muscular weakness, headache, and mental sluggishness are more pronounced, and the physician is consulted because these symptoms persist, or because fever is discovered. The beginning of fever is the most constant indication of the onset of the disease, and two very important early symptoms are cough from bronchitis, and enlargement of the spleen.

Symptoms. The most prominent and constant subjective symptom during the first week is *headache.* Other very common symptoms are tenderness, rarely pain in the iliac region, more or less prostration, and impaired appetite or complete anorexia.

The *objective symptoms* are therefore the most important. The face is pale rather than flushed, and has a dull, listless, apathetic expression. The tongue is heavily coated with a white fur which later becomes yellow. The abdomen is somewhat distended and tympanitic on percussion.

FIG. 300.



Typhoid fever. Course of fever and relation to symptoms.

There is usually tenderness in the right iliac region, and gurgling upon palpation is pretty constant. Constipation may be present at first, and sometimes persists throughout the disease. A tendency to diarrhœa is, however, characteristic of the disease. Even if constipation exists at first, a laxative is apt to produce an excessive effect. The number of stools varies from two or three to a dozen or more in twenty-four hours. They are light yellow in color (resembling pea-soup), thin, watery, and offensive. The movements are not usually attended with pain, and in severe cases may occur involuntarily.

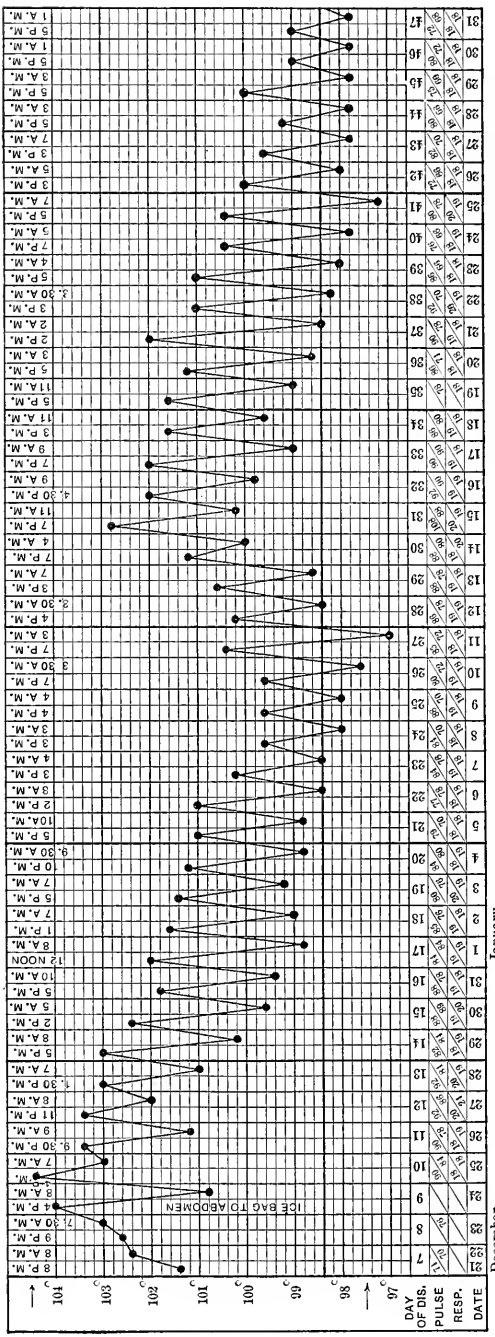
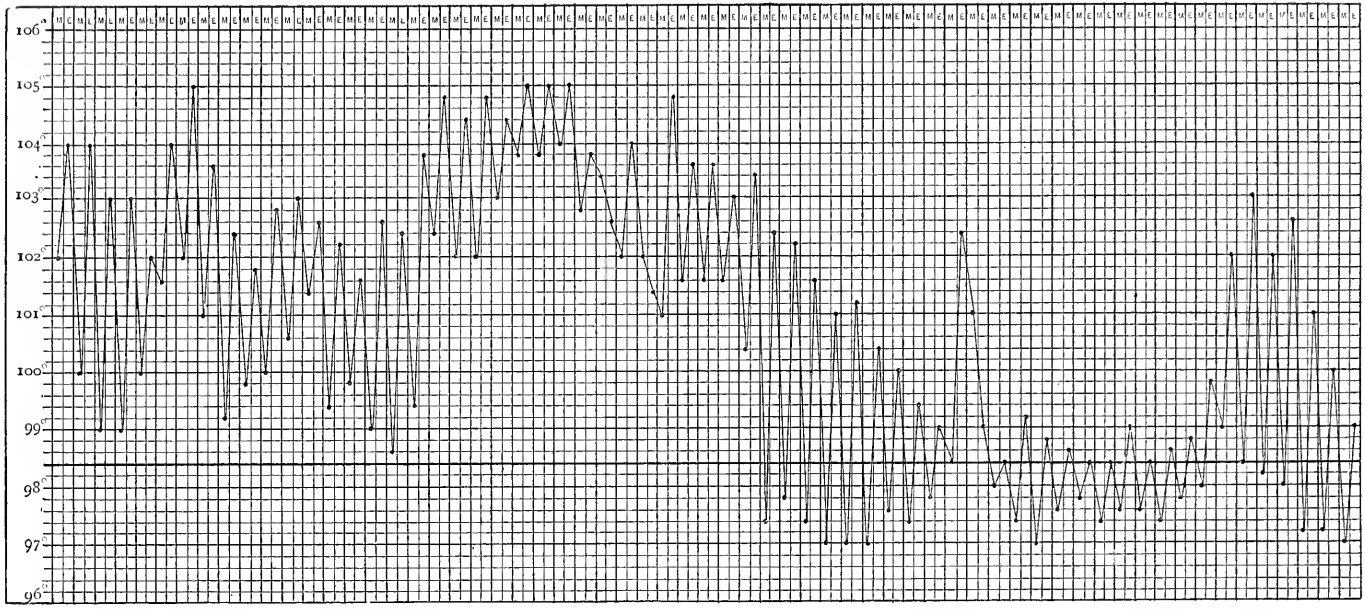


Fig. 301.

Typical chart, mild typhoid fever with an intercurrent relapse. (Original.)

FIG. 302.



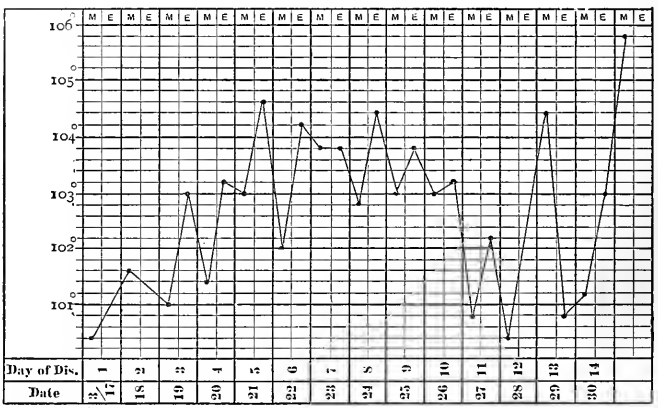
Protracted typhoid fever, not modified by antipyretics. Aberrant temperature during the first twenty days. Duration, sixty-three days. Recrudescence on the fifty-fifth day. (Original.)

Enlargement of the spleen is a very constant symptom. It may be detected at the onset, increases up to the height of the fever, subsides during convalescence, but recurs during a relapse. It covers a percussion-area in the left hypochondrium of four to eight finger-breadths.

The *temperature-curve*, when not modified by treatment, shows a gradual ascent during the first four or five days of the disease, with morning remissions. The temperature rises a degree or two in the evening and falls half a degree or a degree in the morning. This "step-ladder" ascent is characteristic. By the end of a week a temperature of 103°, 104°, or 105° F. is reached, and it remains continuously high, with slight morning remissions, during the second and less frequently during the third week. In the third or fourth week the morning fall of temperature gradually becomes greater, and by the end of the week the temperature sinks below the normal in the morning.

The temperature in mild cases may never rise above 103° F. at any time, and most of the time varies between 100° and 102°; or it may be 104° from the start; more frequently during the second and third weeks there are marked oscillations of the temperature—a sudden fall from 104° to 101°, or a rise from 103° to 105° or 106°. Hyperpyrexia is a temperature above 105°.

FIG. 303.



Grave typhoid fever. Death. M., aged twenty-two years. Ataxic symptoms. (Original.)

The *pulse* is full, and in favorable cases slower than the pyrexia would lead one to expect. It is more frequently under 110 than over 120. In the second week it is markedly dicrotic.

The *heart-sounds* are unchanged apart from complications, but in the second and third weeks the first sounds often are feeble, indicating heart weakness. A pulse of 120 or more is a graver sign in typhoid fever than in other diseases. Therefore, when it becomes very frequent and feeble, the extremities cool, and the lips bluish, the outlook is gloomy.

The *urine* is at first scanty and high-colored. A slight degree of febrile albuminuria is not uncommon, and in rare cases the whole force of the poison seems to be spent upon the kidneys, the urine containing,

besides the usual blood and casts, biliary coloring-matter. In conditions bordering on coma the patient may have retention of urine, or, on the other hand, he may pass it involuntarily. To obtain the *diazo reaction* of Ehrlich two solutions are necessary. The first (*a*) consists of 2 grammes of sulphanilic acid, 50 c.c. of hydrochloric acid, and distilled water 1000 c.c. The second (*b*) consists of a 0.5 per cent. solution of sodium nitrite. These solutions are kept in separate bottles. 50 parts of solution *a* and 1 part of solution *b* are poured into a test-tube and an equal volume of urine added. The test-solutions and urine are now thoroughly shaken and then carefully overlaid with 1 c.c. of ammonia. At the junction of the two a pink or ruby ring develops. Upon agitation the foam on the top of the mixture is also colored red. Normal urine gives a light-brown ring. This reaction is helpful in diagnosis, but may occur in acute phthisis, tuberculous meningitis, and other diseases. According to Pepper, it is rarely absent in measles. The reaction is fairly constant in typhoid fever after the first week.

The *respiration* in uncomplicated cases increases in frequency with the rise of temperature. It usually ranges between 24 and 36. The slight bronchitis present in the beginning in most cases causes no trouble; sometimes it lasts throughout and contributes to the tendency to hypostatic congestion, which is always present. The physical signs are those described elsewhere in these conditions.

The *nervous symptoms* are often very prominent. In mild cases they consist of hebetude and nocturnal delirium, or they may be absent altogether. Usually, however, by the beginning of the second week, there is some mental confusion, with nocturnal delirium. In more severe cases, and later in the disease, the delirium is of a low muttering character, with more or less continuous hallucinations of sight and sound. The patient can be roused by a question and makes an intelligent answer, but speedily lapses into semi-consciousness. Picking at the bedclothes or efforts to catch imaginary objects are very common. Sometimes the delirium is wild and noisy, and the constant presence of someone is needed to keep the patient from getting out of bed. Patients have jumped out of windows, or run long distances before being captured. Rarely the delirium has been so active as to simulate acute mania. Stupor may alternate with delirium. Rarely the patient lies with wide-open eyes, apparently staring fixedly at some object, but really unconscious (coma-vigil).

In ataxic cases the patient has marked twitching of the tendons, and jactitation. He is wakeful and restless, wearing himself out. The hands and lips tremble, and he keeps muttering to himself all the time.

Convulsions are rare, but may occur in children. Sometimes there are considerable hyperæsthesia and tenderness along the spine.

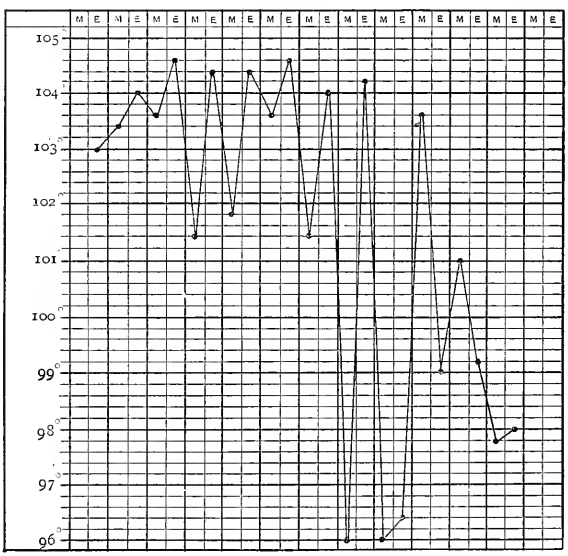
The severity of the nervous symptoms depends upon the habit of the patient as well as upon the height of the temperature and gravity of the disease. In children and neurotic individuals they may be pronounced with only moderate fever.

On the seventh or eighth day the *eruption* appears. It consists of small, very slightly elevated, rose-colored papules, which disappear upon

pressure and come out in successive crops, each papule lasting three or four days. The spots are most common over the abdomen and back, but are occasionally found elsewhere. They are usually few in number, a half-dozen or dozen, but sometimes the eruption is very copious, especially in severe cases. Sometimes it is wholly absent.

During the latter part of the second week, and throughout the third week, the symptoms are apt to be intensified. The temperature remains high or even reaches a higher point. Delirium is more decided and constant. The heart grows weak and the pulse increases in frequency. Some degree of hypostatic congestion of the lungs is usual. Diarrhœa may be troublesome; intestinal hemorrhages, announced by sudden fall of temperature and symptoms of collapse, may occur. Tympanites may become so great as to interfere with respiration and circulation. This is the period when ulceration of Peyer's patches in the intestine is deepest, and when perforation is imminent. There is rarely any desire for food, though it is taken and assimilated. Nausea and vomiting are rare. The tongue is dry, brown, sometimes glazed and fissured, and sordes often collect on the teeth.

FIG. 304.



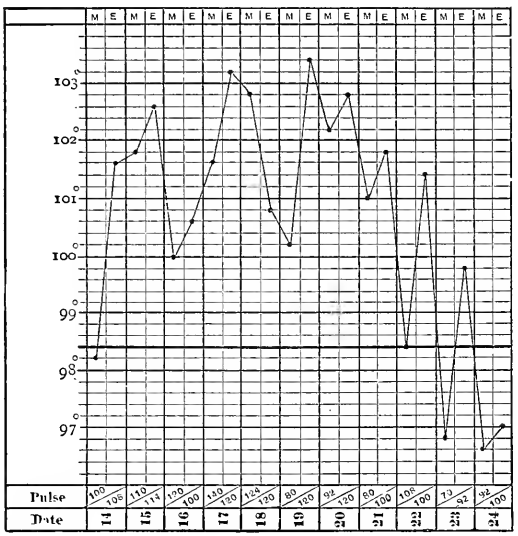
Typhoid fever in a child aged twelve years. Chart from twelfth to twenty-third day. Repeated crises. (Frequent mode of termination in children.) (Original.)

In cases ending in recovery the temperature begins to fall in the mornings; delirium grows less; sleep is more refreshing. Diarrhœa ceases, and constipation may even require treatment. The pulse does not usually improve so rapidly as the other symptoms. There is sometimes marked anæmia without leucocytosis. When the temperature sinks to normal or subnormal, convalescence has set in. This is very rapid as far as digestive symptoms are concerned, but strength returns very slowly.

It may be interrupted by a relapse, in which the original symptoms are reproduced, with high temperature, but the duration is shorter.

Varieties. It is now well known, as Osler forcibly states, "that typhoid fever is no more primarily intestinal than is smallpox primarily a cutaneous disease." Studies in bacteriology, promoted especially by Chiari, Flexner, Kraus, Nicholls, and others, enable us to divide the infection into three varieties: 1. Typhoid fever with intestinal lesions, as described above. 2. Typhoid fever with general infection or typhoid septicæmia, in which the symptoms are entirely those of an infection, and

FIG. 305.



Course of temperature in a relapse beginning on the twenty-sixth day. First attack mild. (Original.)

the diagnosis must rest upon the serum reaction and culture-methods. 3. Typhoid fever with more intense infection of other organs than the intestines. The lungs, the spleen, the kidneys, and the cerebrospinal meninges are the structures invaded, so that we may have a pneumo-, nephro-, spleno-, or cerebrospinal typhoid.

Varieties are also based upon the severity of the disease; hence we have the abortive, ambulatory, and grave forms.

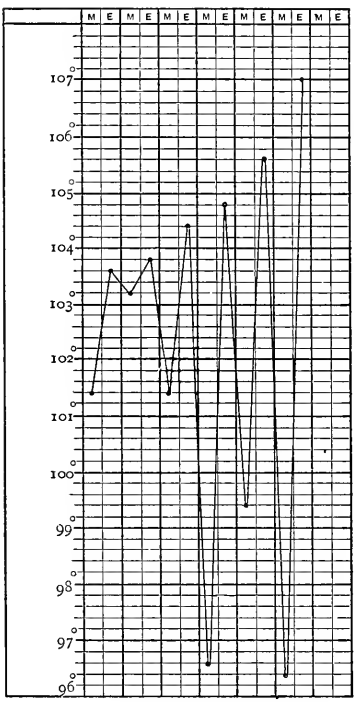
The *abortive* form is so named because of the abbreviated course of the disease. The symptoms are sufficiently well marked to make the diagnosis clear, but the type is mild, and in a week or two convalescence is established. In rare instances an afebrile form with intestinal symptoms and eruption is seen.

In the *ambulatory* form, commonly called "walking typhoid," the patient, from ignorance of the gravity of his ailment or from apparent necessity, keeps at his work until weakness and incessant headache lead him to consult a physician in his office or at a dispensary. He may then

be well into the second week of the disease. The majority of such cases prove fatal.

Grave forms are due to especial severity of some symptoms or group of symptoms, such as hyperpyrexia; profound stupor, coma, or intense ataxia; inability to take or retain sufficient nourishment; profuse diarrhœa and intestinal hemorrhage; great adynamia with weak heart and a tendency to cyanosis. In other cases the gravity of the disease results from the existence of complications.

Fig. 306.



Grave typhoid fever. Daily rigors. Died on nineteenth day. No complications. (Original.)

In the *malignant* form either the dose of the poison has been large or the organism is very weak, or both, the result being an acute toxæmia; this is not so common as in scarlatina and typhus fever.

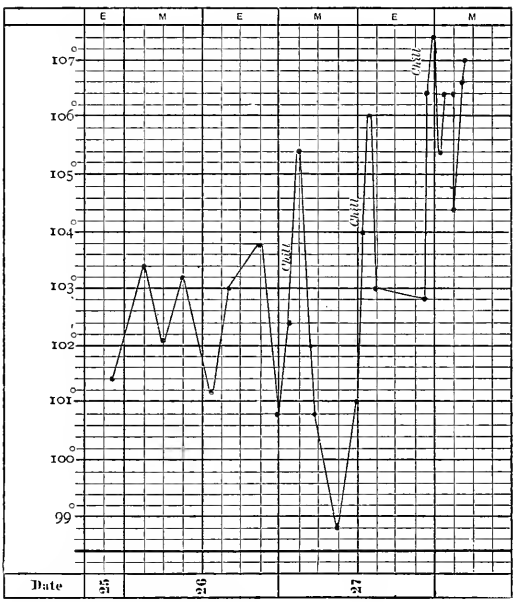
In the *pulmonary* form the onset may be so obscured by severe bronchitis or lobar pneumonia that the primary disease is not suspected at first. Severe bronchitis seems to be more common in children. Chill and initial high temperature are common in these cases.

Typhoid Fever without Intestinal Lesions. This rare form may present the typical clinical symptoms of typhoid, or may be of splenotyphoid type, or of nervous type with extreme intoxication. The first type is rare. The second type, described by Eiselt, is characterized by an excessive enlargement of the spleen, with local inflammation and remit-

ting fever. In the third the symptoms of the typhoid state with subcutaneous and visceral hemorrhage occur. Jaundice is more or less common.

Complications and Sequelæ. Typhoid fever may be accompanied by a number of complications, the most frequent and important being severe laryngitis, bronchitis, hypostatic congestion of the lungs with œdema, and true lobar pneumonia; bed-sores; parotitis; phlebitis, especially of the femoral vein; peritonitis from perforation of the bowel; jaundice; cholangitis; meningitis, acute mania; myocarditis; periostitis and osteitis. Sequelæ are not frequent. Sometimes, however, the foundation is laid for permanent ill health. There may be impairment of the senses, mental weakness, and even insanity. Paralyses, neuritis, hyperæsthesia, chorea, and epilepsy are occasional sequels.

FIG. 307.



Renal typhoid. Nephritis on the twenty-fifth day. Course of temperature during three days preceding death. (Original.)

Examination of the Blood. The infection is due to Eberth's bacillus, *Bacillus typhosus*. The bacillus is found in colonies in the spleen, liver, mesenteric glands, kidneys, and intestines. It is also found in the feces and rarely in the urine. It may be seen in the blood. It may be recognized by staining methods, although rarely. It has been isolated from the blood successfully by culture-methods. The results of Kühnau and Gwyn show that in 20 to 25 per cent. of the cases, whether severe or mild, bacilli may be obtained. Gwyn had 8 positive cases, some of which were very mild infections. Richardson found bacilli very constantly in the spots (5 out of 6 cases).

Morphology. It has flagella 3 to 5 times as long as the bacilli. It stains with the anilines, best with Löffler's method.

Serum Diagnosis. This method of diagnosis has been more successfully employed in typhoid fever than in any other infection. The methods have been previously described. The agglutinative reaction takes place as early as the eighth day, rarely as early as the third day, but sometimes not until the fifteenth or twentieth day, and may even be delayed until convalescence is established. By this means typhoid fever can be distinguished from the infection due to the bacillus of Gärtner (*Bacillus enteritidis*).[1]

The paracolon bacillus infection, as shown by Gwyn,[2] who studied a case which resembled typhoid clinically, does not give this reaction. Influenza and Malta fever, and forms of tuberculosis can also be distinguished from typhoid fever by this method.

Leucocytosis. Typhoid fever is one of the infections in which leucocytosis does not occur. In a differential count some variation from the normal is seen. The large mononuclear and transitional forms are relatively increased; the polynuclear neutrophiles are decreased. The absence of leucocytosis aids in distinguishing typhoid fever from various septic fevers and acute inflammations. On the other hand, if leucocytosis occurs in a case of typhoid fever, an inflammatory complication or mixed infection should be suspected. Perforation and peritonitis are attended by leucocytosis.

In addition to the absence of leucocytosis we find, after the second or third week, gradual reduction of the red cells, and by the time convalescence is established, a marked anæmia develops. Both the red cells and the hæmoglobin are reduced.

Culture-methods. The bacillus can be isolated from the blood, the stools, and the urine.

Bacteriological Diagnosis. Recently bacteriologists have been successful in isolating the typhoid bacillus from the blood, the stools, and the urine. Unfortunately, the methods are too complicated for clinical work. P. H. His, Jr., recovered *Bacillus typhosus* and distinguished it from members of the colon group by a combined plate and tube method.[3] For differentiation of the typhoid from the colon bacillus the method of Proskauer and Capaldi may be used.

Mark Richardson isolated bacilli in the *urine* of about 22 per cent. of the cases of typhoid examined. They were present in large numbers and in pure culture. They appeared late in the disease, and persisted into convalescence. The bacilli were always associated with albumin and casts. After disinfection of the meatus the urine is passed in two portions into sterilized test-tubes. The second portion is used. It is immediately plated upon plain agar. At the end of twenty-four hours the characteristic colonies appear. Richardson relies upon the active motility of the

[1] See Lancet, January 15, 1898.

[2] Bulletin of the Johns Hopkins Hospital, 1898, vol. ix., No. 84.

[3] P. H. His, Jr., "On a Method of Isolating and Identifying Bacillus Typhosus," etc. Journal of Experimental Medicine, vol. ii., No. 6, p. 677.

bacilli, which are set free in a typhoid colony by scarring with a platinum needle to distinguish them from the colon bacilli. He also used the dry serum reaction test.[1]

Gwyn had twelve cases in which the bacilli were present in enormous numbers, all in pure culture. In one the diagnosis was made from the urine, the Widal reaction being absent at the time. In two others the bacilli were recovered from the urine before the development of the Widal reaction. They were present in practically the same percentage as in Richardson's series. When one calculates, as in one case, 500,000,000 bacilli per cubic centimetre and sees the bacteriuria persist for weeks and years, the importance of the urine as an infective agent is apparent.[2]

Richardson and Steele in my wards at the Presbyterian Hospital have recovered the bacillus from the sputum of cases of typhoid fever with coincident bronchitis and pneumonia. The pneumococcus and the influenza bacillus are generally associated, and the sputum is hemorrhagic.

Diagnosis. A typical case of typhoid fever ought not to be mistaken for any other infection, but atypical cases are numerous.

New Diagnostic Sign of Typhoid Fever. Simon Baruch writes as follows: "As soon as the patient shows a rectal temperature above 102.5° in the morning and 103° in the evening for three successive days, especially if this be accompanied by headache, dulness, or apathy, he is placed in a full bath at 90°, which is reduced to 80°, with constant friction over the body. In three hours, the temperature still being above 102.5°, he receives another bath 5 degrees cooler. This is repeated until the temperature of the bath is 75°. If one or more of these baths fail to reduce the rectal temperature 2 degrees in half an hour, the diagnosis of typhoid fever is almost certain, and the bath-treatment is continued. The resistance of the rectal temperature to a bath of 75° for fifteen minutes with friction, is an almost certain sign of typhoid fever."[3] Baruch considers that the diagnosis of this disease should no longer be obscure, even in the first days of its course.

Local Infections. The most common sources of error are a hurried diagnosis and a willingness to accept a demonstrable local affection as sufficient to account for the condition. In this way the significance of bronchitis, pneumonia, and diarrhœa is overlooked. In the symptomatic form there will almost always be found a history of gradual onset and a degree of fever and prostration greater than should attend the purely local affection. Moreover, in the bronchitis and pneumonia which are a part of typhoid fever, there may be found tenderness with gurgling in the right iliac region, enlargement of the spleen, and epistaxis, to aid in the diagnosis; while in cases in which the diarrhœa is misleading, bronchitis, enlargement of the spleen, and epistaxis may coexist.

Examination of the blood, extended over a period of several days, is necessary to exclude the *æstivo-autumnal* type of malaria, which often resembles typhoid fever. The Widal test, the absence of leucocytosis,

[1] M. W. Richardson, "On the Presence of the Typhoid Bacillus in the Urine." Journal of Experimental Medicine, vol. iii., No. 3, p. 349.
[2] Johns Hopkins Hospital Bulletin, April, 1899.
[3] New York Medical Journal, September 2, 1893.

and the bacteriological examinations are essential methods for positively excluding the simulative infections, the consideration of which follows.

Appendicitis is more likely to be mistaken for typhoid fever than the converse. There is usually a history of constipation, though the occurrence of several inadequate movements a day may conceal the fact that there is a fecal accumulation. In appendicitis the onset is more abrupt and the local symptoms are more pronounced than in typhoid. Pain and tenderness are prominent in appendicitis, and while they may be general over the abdomen at first, they are found to be more acute in the iliac region and in the loin. Here, in place of gurgling, we find some increase of resistance on palpation, and a relatively dull note—a wooden sort of tympany—or there may be a demonstrable tumor. The patient lies with the right leg drawn up, has moderate fever, and vomiting. In fact, the attack is often introduced by chilliness and vomiting. Headache is not a prominent symptom, while bronchitis and enlargement of the spleen are absent. Leucocytosis is a valuable sign much insisted upon by the surgeons, while, of course, the presence of the Widal reaction favors typhoid fever.

Acute right-sided *salpingitis* simulates typhoid fever. It is distinguished by the history of a preceding vaginitis, endometritis, or abortion, and by the absence of diarrhœa, of enlargement of the spleen, and of the characteristic eruption. A digital examination through the vagina discovers the womb pressed to one side and fixed, and a tender mass blocking up the pelvis.

Simple continued fever is distinguished from typhoid fever of a mild type principally by the absence of bronchitis, of enlargement of the spleen, of epistaxis, and of the characteristic eruption of typhoid fever. In simple continued fever constipation is more common than looseness of the bowels, and gurgling is absent.

Typhus fever is distinguished by its sudden onset, the besotted expression of the face, with reddened eyelids and small pupils, the absence of abdominal symptoms, and the appearance on the fourth day of macules, which are subsequently converted into petechiæ. It is of shorter duration, and terminates very abruptly by crisis.

Relapsing fever differs from typhoid fever in its sudden onset with chill, pain in the epigastrium, but absence of abdominal symptoms and eruption; in the absence of marked nervous symptoms, in spite of the high fever; the short duration and termination by crisis, and the characteristic relapse at the end of a week. The conclusive test is the finding of spirilla in the blood.

Acute tuberculosis of the lung at times closely resembles typhoid fever. In both the onset is gradual, with cough and fever. In the former, however, the bronchial symptoms are more prominent, there are apt to be recurring chills and sweats, the temperature is remittent and irregular, emaciation is rapid, and constipation instead of diarrhœa is the rule.

In *peritoneal tuberculosis* there is persistent, diffuse pain in the abdomen, and the belly is swollen. If effusion occurs, the physical signs disclose its presence. The temperature is irregular and may be below normal; nervous symptoms comparable to those of typhoid are wanting.

Meningitis before the stage of effusion exhibits exaggeration of the reflexes and marked hyperæsthesia. There may also be muscular rigidity. The patient is restless, easily annoyed, and "fussy" about things that would be unnoticed by a typhoid patient. Vomiting is often present, whereas it is rare in typhoid fever. The temperature does not maintain so high an average range as in typhoid fever, and is subject to oscillations. The pulse varies greatly, and may be irregular. Leucocytosis is present, and cultures from "swabs" of the nose and nasopharynx or the cerebrospinal fluid may establish the diagnosis.

In *septic meningitis* the headache and vomiting are more persistent, the bowels are confined, and the abdominal walls are retracted. There may be double optic neuritis. In *tuberculous meningitis* the knee-jerk and other reflexes are variable, irregularly absent or present. In typhoid fever they are always present. In the former, choroidal tubercles may be seen with the ophthalmoscope. (See Eosinophile, Chapter XXXIX.) Leucocytosis is present in all forms of *tuberculosis;* in typhoid it is absent.

Typhoid fever must not be confounded with *trichinosis;* the peculiar muscular pain and œdema do not occur in the former. *Uræmia* may simulate typhoid fever when it becomes chronic; but the age, the character of the urine, and the cardiovascular symptoms are diagnostic, and, in the absence of the specific typhoid symptoms, render the diagnosis easy.

Mountain fever is an infection which has been described as peculiar to the mountains of our Western States, characterized by a continued fever with intestinal symptoms not unlike those of typhoid fever. Irregularity of the temperature range and the occurrence usually of constipation rather than diarrhœa make it difficult to separate the infection from typhoid fever on the one hand and from forms of malaria on the other. Recent observations of Woodruff, who studied the serum reaction in a large series of cases, show conclusively that the infection is typhoid fever, confirming the prior observations of Hoff, Smart, and Raymond.

Paratyphoid Fever. This infection is due to a bacillus which is intermediate between the typhoid and the colon bacillus.

The symptoms are like those of typhoid fever in the larger number of cases. In another, but smaller group of cases, the symptoms are those of gastro-enteritis with fever. The first form corresponds closely to typhoid fever in all its incidents, including the etiology and the occurrence of relapses. It is usually of shorter duration, premonitory symptoms are absent, prostration is early, myalgia is more marked, and the temperature rises more rapidly. In the gastro-intestinal form the temperature rises rapidly, after a chill, diarrhœa supervenes at once, vomiting and epigastric pain are marked, while prostration is the dominant feature. The diagnosis can only be established by the serum test. Cultures from the blood, urine, or stools would prove positively the presence of this infection.

In using the serum test, both species of the paratyphoid bacilli must be employed, and an agglutination should not be expected except in high dilution. If the blood fails to agglutinate the typhoid bacillus or agglutinates it only in very low dilutions, typhoid fever is absent.

Yellow Fever.

The infection which we are about to consider is the latest of the epidemic and contagious disorders for which a definite causal micro-organism has been discovered. It is an acute, specific, contagious, miasmatic disease, endemic and epidemic on the tropical and subtropical shores of the Atlantic Ocean, characterized by a sudden onset, a duration of a week or less, a characteristic facies, a fall in the pulse-rate preceding a fall in the temperature, and by albuminuria, jaundice, and vomiting, with a tendency to hemorrhages. The specific micro-organism is *Bacillus icteroides* described by Sanarelli. Sternberg asserts that his bacillus *X* is identical, and hence claims priority.

Yellow fever is endemic in Havana and other seaport cities of Cuba, and in Rio Janeiro, Brazil. From these centres it is liable to become epidemic and to be carried in ships and by persons and clothing to other places. In this way epidemics have developed in the seaports of the United States, especially on the shores of the Gulf of Mexico, but sometimes as far north as Philadelphia and New York. The disease becomes epidemic in the hot season and disappears upon the appearance of frost.

In countries in which the disease is endemic it is the custom to regard the native children as immune. John Guitéras, however, is strongly of the opinion that the disease is kept alive between epidemics by mild cases among these children. He has also shown that it prevails among white children before it becomes epidemic among adults.

The period of *incubation* varies from a few hours to two weeks. Guitéras states that the cases in which it extends beyond the seventh day are exceptional.

The *invasion* is abrupt, and occurs usually in the night. It is marked by chilliness oftener than by a decided chill. The temperature rises rapidly to 102° to 103° or 104° F., not often higher in favorable cases. The *pulse* is correspondingly increased in frequency at first, but very commonly begins to fall before the temperature, so that later the pulse is relatively slow. The *face* is peculiar and characteristic—it is flushed and somewhat swollen; the edges of the eyelids are reddened; the eyes are watery, glistening, and slightly but distinctly tinged with *yellow;* the pupil is small and brilliant. Guitéras says:[1] "The appearance of the face is often sufficiently characteristic on the first day of the disease to warrant a positive diagnosis." "The early manifestation of jaundice is undoubtedly the most characteristic feature of the facies of yellow fever." He also says that these phenomena are often better observed at a slight distance than on close inspection. The *tongue* is large, moist, and coated with white fur. The stomach is irritable and the epigastrium tender. Nausea with repeated vomiting occurs. The fluid is at first of a light greenish-yellow, subsequently becoming decidedly bilious. The bowels are constipated.

The *urine* almost invariably contains albumin at some time during the first three days. The albuminuria may last only a short time and may

[1] "Report of the Surgeon-General of the Marine-Hospital Service, 1888"; Keating's Cyclopedia of Diseases of Children, 1889, vol. i.

be found only in the evening; the amount of albumin is sometimes very large, and abundant blood- and tube-casts are found. The nephritis subsides rapidly, without leaving traces. The urine is acid in reaction and scanty in amount. It is sometimes suppressed.

During this febrile period the patient complains of headache, pains in the back and limbs, and intense thirst. The mind, however, is usually perfectly clear. Contrary to expectation, Guitéras asserts that the nervous symptoms are perhaps more prominent in the adult than in the child. "The loquacity, the short-cut phrases and precipitate speech, the excitement, the show of indifference with unmistakable evidences of fear—all these, that are such prominent features of the disease in the adult, are absent in the young."[1]

In from two to five days the temperature falls to or below normal, headache and pain in the limbs disappear, and the patient is cheerful and believes himself convalescent. In mild cases this is the fact, but in more severe cases the period of remission or stage of *calm* is followed in a few hours or at most a day or two by a return of symptoms. The jaundice deepens, vomiting becomes more urgent, and in adults is accompanied by much retching. The vomitus is bilious, streaked with blood, or thick and wholly black ("black vomit"); the temperature may equal or surpass that of the original paroxysm, or it may remain depressed. In any event the pulse is apt to be slow, often from 40 to 60. The urine contains albumin, blood, and casts, and there may be suppression, adding uræmia to the other toxæmia. Convulsions at this stage are usually uræmic. Hemorrhages may occur from any mucous surface. The gums are tender, swollen, and bleed easily. There may be epistaxis, hemorrhage from the ear, bowels, uterus, or vagina. Pregnant women miscarry. Ecchymoses also may form. Death may take place in coma or convulsions. If the patient lingers beyond the fifth or sixth day, he sinks into a typical typhoid state, with diarrhœa and marked adynamia, from which he may or may not emerge.

As in scarlet fever, the patient may be smitten down and die in a few hours from the time he was in apparent health. In other grave cases the temperature remains high, and rises instead of falls on the third or fourth day. The *duration* of the disease is from two to five or six days; if a typhoid state develops, it may last ten days or two weeks.

Complications are not common. Phlebitis and lymphangitis occur, and Guitéras says he has noticed hepatitis, insanity, and paralysis (probably from neuritis). Second attacks are extremely uncommon.

Examination of the Blood. *Bacillus icteroides* is a slender rod from 2 μ to 4 μ in length. It is ciliated and motile. It is found in more than half the cases, if Gram's method is employed.

Serum Diagnosis. Woodson and the Archinards have found agglutination to take place in a large proportion of cases of yellow fever. The blood, taken as early as the second day, gave a prompt reaction in from 75 to 80 per cent. of all cases. Dilutions of 1 to 40 were used, but reaction took place in dilutions as low as 1 to 50. Pothier and Lerch report successfully upon this reaction. Cultures from the blood produce

[1] Keating's Cyclopædia, loc. cit.

an organism which grows on ordinary media, does not coagulate milk, but ferments saccharine fluids.

Inoculation. Inoculation of dogs and monkeys produces a clinical picture similar to the original infection.

Diagnosis. Yellow fever is distinguished from *pernicious malarial fever* by the slow pulse, the characteristic facies, the early transient albuminuria, the early and deep jaundice, the absence of diarrhœa, the occurrence of black vomit, the tendency to hemorrhage, the clear mind, and the absence of enlarged spleen.

The diagnosis is based upon examination of the blood, the three important characteristics which Guitéras lays stress upon—the facies, the albuminuria, and the slowing of the pulse, with continuance or increase of the fever, and the usual diagnostic data of an epidemic and contagious disease. The affection must be distinguished from dengue and from various forms of malarial fever, especially the æstivo-autumnal infections. Jaundice rarely appears as early as the second or third day of the disease. Hemorrhages are rare in both dengue and malarial fever.

Malta Fever.

Malta fever is an infection that prevails in tropical countries. It is characterized by gradual onset and by repeated remissions of the *fever*. The alternating febrile and afebrile periods which characterize the disease continue from two months to two years. The most remarkable feature is the peculiar character of the temperature range, which consists of intermitting waves or undulations of fever of a distinctly remittent type. These periods of fever last from one to three weeks, followed by an apyretic period or a period of abatement lasting from two to ten days. The daily temperature range may be intermittent or remittent. The febrile course may continue six months or longer. During this time patients grow more and more prostrated, become anæmic, and usually suffer from constipation. Profuse sweats attend the decline of the daily range, and in many instances the spleen is enlarged. Neuralgias occur in various parts of the body; the joints become enlarged, and fibrous tissues may be the seat of inflammation. Hughes—who describes the disease most accurately—describes a malignant type lasting a week or ten days, and an undulatory type continuing for weeks or months. Indeed, the relapses are known to occur over a period of two years. The third is known as the intermittent type, in which there is a daily rise of temperature without other marked symptoms. The undulatory type is the most common. The infectious micro-organism is *Micrococcus melitensis*.

Diagnosis. A positive differential diagnosis is made from all forms of malaria by an examination of the blood, and from typhoid fever by the Widal test and the absence of *Bacillus typhosus* in the urine or stools of the suspected patient. The micro-organism has not been isolated from the blood, but the *serum reaction* is a valuable means of diagnosis. (See page 552.) This reaction is obtained as in cases of typhoid fever. The culture must be carefully selected. With a 1 to 10

or 1 to 50 dilution agglutination takes place when the serum of a patient with Malta fever is used. The serum of such a patient does not have

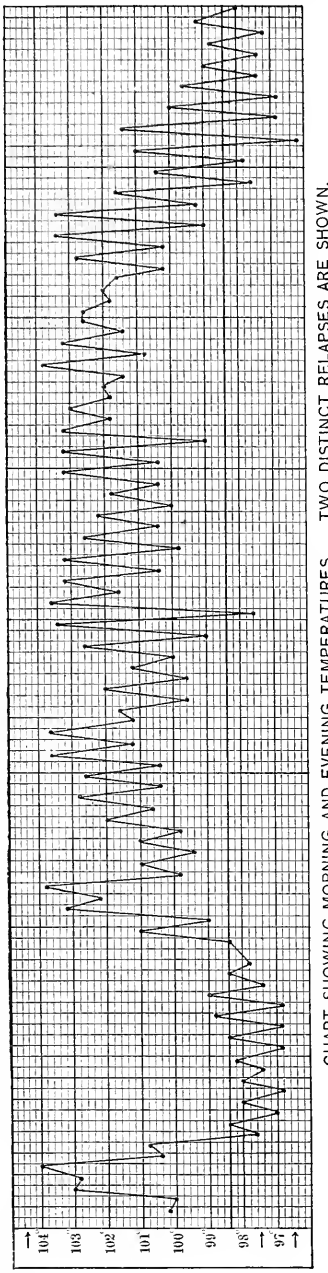
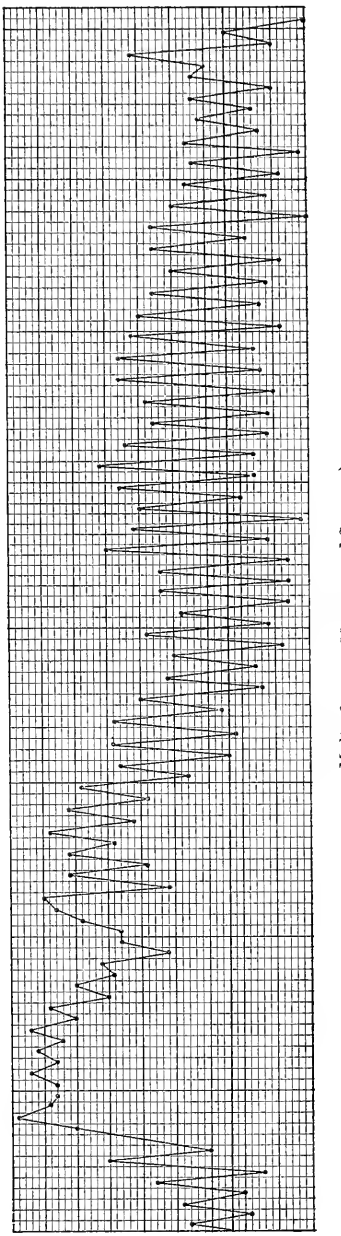


FIG. 308.—CHART SHOWING MORNING AND EVENING TEMPERATURES. TWO DISTINCT RELAPSES ARE SHOWN.



Malta fever. (MUSSER and SAILER.)

any effect upon the typhoid bacillus nor upon other organisms. Aldrich states that the reaction first occurs about the fifth day.

BACTERIOLOGICAL EXAMINATION.

Gonorrhœal Infection.

Although the infection is usually limited to the genito-urinary tract, it is well known that the gonococcus may enter the blood and infect tissues elsewhere, causing a local inflammation. We therefore see symptoms due to the primary infection; symptoms due to infection of the genito-urinary organs by direct continuity and systemic infection. The primary infection involves the adnexæ of the genital organs in the male and the female. Salpingitis, metritis, and ovaritis in females, with the occurrence occasionally of peritonitis, arise from spreading by continuity. In both sexes, cystitis, ureteritis, and pyelitis occur. The infection is usually mixed. When the gonococcus invades the blood, symptoms of septicæmia or pyæmia arise. The infection may be rapid and fatal, and may terminate ten days after the primary lesion. The occurrence of such general infection is suspected when the history of the primary infection can be secured, and in addition the micro-organism can be recovered from the blood, as was successfully done by Thayer.

In other infections the joints become involved and we have the phenomena of gonorrhœal arthritis (see Joints), the course and symptoms of which are discussed elsewhere. Endocarditis may result from gonorrhœal infection, and can only be distinguished from other forms of endocarditis by the history and the finding of micro-organisms in the blood. Myocarditis (Councilman) and pericarditis may also occur.

Diagnosis. Thayer, Blumer, Thayer and Lazear have succeeded in recovering the gonococcus from the blood in this form of septicæmia. The blood is drawn from the median basilic vein with a sterilized syringe. It is mixed with melted agar and immediately plated. Cover-slip preparations of the colonies, if the case is gonorrhœal, will show the tinctorial and morphological characteristics of the gonococcus. (See Plate XIII.–C, Fig. 4.) The diagnosis is further established by finding the gonococcus in any purulent discharge, urethral or vaginal. (See Chapter XLV., Part I.—Exudates, etc.)

INFECTIONS RECOGNIZED BY THE EXAMINATIONS OF EXCRETIONS AND SECRETIONS OR BY THE PRODUCTS OF THE INFECTIOUS INFLAMMATION.

The following infections are disclosed by the examination of the products of the infection found in the inflammatory areas (pus); in the excretions and secretions of the body; in the sputa; in the vomitus; and in the feces, or in the urine. The reader should turn to the sections describing the method of bacteriological examination of pus, sputum, and secretions. The infections referred to are: *erysipelas, pneumonia, tuberculosis, influenza, cerebrospinal meningitis, diphtheria, septicopyæmia, gonorrhœa, glanders, cholera Asiatica, dysentery, bubonic plague, leprosy, actinomycosis, tetanus, trichinosis.*

Erysipelas.

The fever of this infection, particularly in a first attack, is very marked, and rises suddenly to a considerable height and may precede the eruption. It resembles the course of a pneumococcus infection.

Erysipelas is an acute, specific, contagious, and infectious disease, characterized by a sudden onset, with a bright-red eruption which usually begins on the face near the nose or mouth and spreads over the entire face and scalp. It is attended with burning heat of the skin and great disfigurement from swelling.

The specific cause of erysipelas is *Streptococcus erysipelatosus*. It is carried to a slight extent by the air, and still more in the discharges, especially those of the nose. Repeated attacks occur in persons with chronic nasopharyngeal catarrh, carious teeth, or a sinus. This infection is apt to attack persons with open wounds (surgical erysipelas), and puerperal women, producing in these cases sloughing and septicæmia. One attack does not protect against another; on the contrary, if there is any focus in which the streptococci linger, one attack actually predisposes to another.

The *period of incubation* is usually from three days to a week. On close inquiry a history of sore throat and some enlargement of the cervical lymphatics is usually found to have preceded an attack of facial erysipelas. The *invasion* is sudden and is marked by chill. The temperature rises to 104° or 105° F., and in the next two or three days may rise still higher. Coincidently with the rise in temperature the portion of the *skin* to be affected burns, tingles, is tender to the touch, and may be reddened. The redness increases in intensity and extent, while the skin is swollen and slightly œdematous. The part of the face affected is usually the cheek in close proximity to the nose, less frequently near the mouth and ear. Vesicles and blebs often form when the inflammation is very intense. The redness disappears upon pressure, but quickly returns; sometimes it has a dusky, purplish hue.

A marked characteristic of the disease is its tendency to spread. In ordinary cases it involves one cheek, eyelid, and ear, and travels across the bridge of the nose to the other side. The inflammation is most intense while it is spreading; the advancing margin is raised, tense, and brawny; the line is thus sharply drawn between healthy and inflamed tissue. The loose tissue about the eyes swells enormously, both eyes are closed, the entire face swollen, red, and disfigured with vesicles and blebs here and there. Curiously the chin escapes. The redness and swelling begin to subside in the part first attacked, before the process has reached its height on the opposite side. As a rule, facial erysipelas does not extend beyond the face, the scalp and neck being spared. The scalp, however, is more frequently affected than the neck; occasionally erysipelas leads to extensive cellulitis of the scalp, with the production of a septic constitutional condition and much local sloughing. The submaxillary glands are more or less enlarged, sometimes so much so as to prevent the taking of solid food. On the body the eruption spreads over a greater extent than when primary on the face, hence its name, "the red runner." It may pass from

the heel to the thigh, and over the trunk, and may last for weeks. While the eruption is extending, the fever continues and is sometimes alarmingly high. The pulse is frequent and soft. Leucocytosis is present. Nocturnal delirium is not uncommon in severe cases, and nausea and vomiting are frequent. The bowels are usually constipated. The urine is high-colored, frequently contains a small amount of albumin, and actual nephritis sometimes occurs.

Prognosis. In favorable cases of facial erysipelas the process is at an end in a week or less; it may be prolonged to two weeks, subsiding by crisis or lysis. Convalescence is usually rapid; the vesicles or bullæ dry up into yellowish crusts, and the epiderm is shed in large or small pieces according to the intensity of the process.

Complications and Sequelæ. Pneumonia and nephritis are the most frequent complications. Meningitis, pericarditis, and endocarditis also occur. Erysipelas may extend inward and involve the fauces, pharynx, and larynx, producing œdema and death from suffocation. If the scalp has been involved, the hair falls out. The cervical adenitis may result in abscess; chronic nephritis may occur. Otitis media occurs occasionally, and so do keratitis and abscess of the eyelids. On the other hand, erysipelas is credited with causing the disappearance of lupus, chronic eczema, and sarcomata.

Diagnosis. **Bacteriological Diagnosis.** Examination of pus, or of discharge from the nose or pharynx, will disclose the presence of the streptococcus. (See Plate XVII., Fig. 1; and Chapter XLV., Part I.)

Herpes zoster of the face and forehead is distinguished from erysipelas by the fact that vesicles appear first, followed by erythematous redness, and that they are limited by the median line, and are preceded and accompanied by sharp neuralgic pain, whereas erysipelas affects both sides of the face, and vesicles appear at the height of the disease; the pain is much less in erysipelas.

Dermatitis. From dermatitis of various kinds it is distinguished mainly by the sharper febrile reaction, the raised border of the eruption, which begins on one side and spreads to the other. Erysipelas is rarely equally intense upon the two sides; dermatitis frequently is. In the latter the skin often exhibits a rough surface, whereas in erysipelas the skin is smooth and shiny until vesicles appear.

Chronic *erythematous eczema* occurs in middle-aged and old persons, is afebrile, accompanied by little sweating but a great deal of itching, and runs a slow course.

Pneumonia. Pneumococcus Infection.

In typical cases of pneumococcus infection the course of the *fever* is of great diagnostic significance. Its sudden rise to a great height, preceded by a rigor, is of itself suggestive. During the succeeding days of the disease the morning and evening temperature vary but little. When associated with hurried respiration and the intoxication symptoms attending this infection, even though no physical signs are present in the lungs, pneumonia can reasonably be suspected. The termination of the febrile

PLATE XVII.

Fig. 1.

STREPTOCOCCUS—ERYSIPELAS.

Fig. 2.

STREPTOCOCCUS SEPTICUS.

Fig. 3.

STAPHYLOCOCCUS.

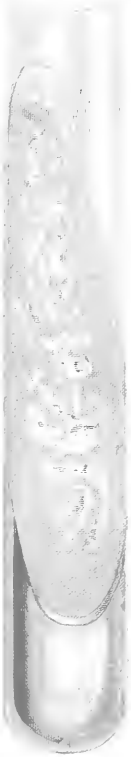
Fig. 4.

DIPHTHERIA-BACILLI.

Fig. 5.

TYPHOID-BACILLI.

Fig. 6.



TUBERCULOSIS-BACILLI.

course is characteristic of this disease. The sudden fall to normal or a subnormal temperature—known as the crisis—brings to an abrupt end the usually alarming symptoms.

Acute pneumonia, croupous or lobar pneumonia, is an infectious inflammatory disease excited by *Micrococcus lanceolatus* (*Diplococcus pneumoniæ*, pneumococcus) involving the vesicular structure of the lungs, and followed by choking of the alveoli with the products of inflammation; it is attended by severe constitutional symptoms due to the toxins of the infecting organism.

Symptoms. Mode of Onset. The invasion of pneumonia is usually sudden, and is marked by a *chill*. The temperature rises rapidly, and may reach 104° or 105° F. in the first twelve hours after the chill. With the fever, the patient complains of severe headache and *pain* in the side, and has a short, quick cough and sometimes vomiting. The pulse is moderately accelerated, and the respiration either is or soon becomes very frequent. The face is apt to be flushed, and there may be a circumscribed red spot on the cheek. The skin is hot and dry. On physical examination, within the first twenty-four hours, a small patch of consolidation is detected, which may subsequently extend over a large area.

While this is the picture of an ordinary pneumonia in its early stage, all cases are by no means so clear. In some the course resembles that of a general fever in which the pulmonary disease is a local manifestation. In such cases there may be prodromata, consisting of headache, general malaise, a slight bronchitis, and digestive disturbance. Then follows the chill.

Central Pneumonia. The fever may be high for several days before there is discoverable consolidation of the lungs, and during this time cough may be wholly or almost wholly absent. The respirations increase gradually in frequency, and finally a well-marked pneumonia can be made out. It is customary to account for these cases by the supposition that pneumonia developed in the interior of the lung, and consolidation gradually extended to the surface. In some cases the patient presents no more definite symptoms for three or four days than high fever, intense headache, and moderately accelerated respiration.

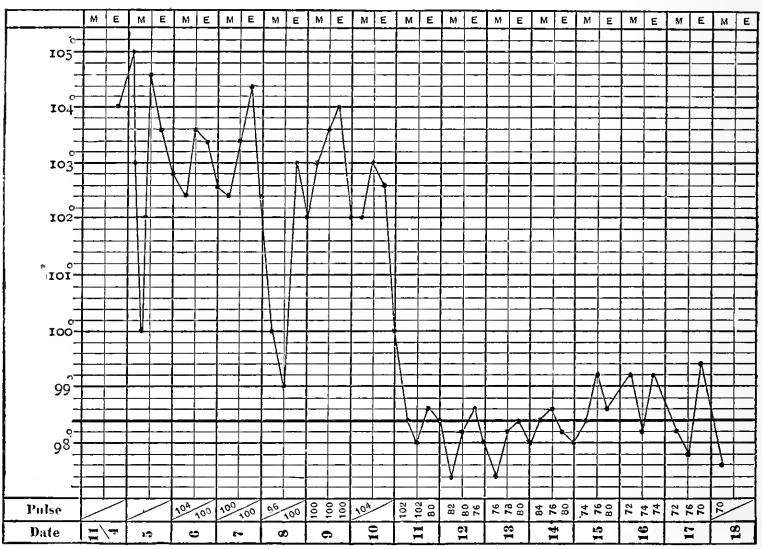
Later Stages. At the end of forty-eight hours, or, at the most, of four days, the patient is found lying in bed in the dorsal position, or on the affected side. The face is flushed, and countenance anxious, the respiration hurried, the *alæ nasi* play vigorously. The temperature varies little from the first day's rise; the chest pain has subsided, and the short dry cough is now attended by viscid expectoration. The respiration continues hurried, the pulse full and bounding. During this time the physical signs of consolidation continue and increase.

After a period of five to ten days the termination takes place by crisis, the pain in the chest abates, the cough becomes looser, and the expectoration more free, but the other symptoms persist. In addition, in some cases, delirium occurs, the pulse softens and becomes dicrotic, the urine becomes albuminous.

Respiratory Symptoms. Chest-pain, cough, hurried respiration of a peculiar type, and expectoration are characteristic. The *chest pain* is

sharp and stabbing or lancinating. It is increased by breathing. It is seated about the nipple or in the *axillary region*, at the angle of the scapula or below the diaphragm. Its seat always indicates the side affected. *Cough* is short and dry, smothered and painful; it soon becomes softer and painless as the expectoration becomes free. It may be absent in the feeble, in the aged, in alcoholic subjects, or in persons with brain disease, including insanity. Characteristic symptoms of pneumonia are the increased frequency and the type of the *respiration*. The rate in adults reaches 40, 50, or even 60 per minute, and in children 80 and 100 are not uncommon. The pulse, on the contrary, does not increase in frequency in the same proportion; hence the normal ratio of respiration to pulse of 1 to 4 becomes 1 to 3 or 1 to 2. Inspiration is short, expiration quick and often attended by an expiratory noise or grunt. The long pause may take place after inspiration instead of expiration. In children both are so short that unless the epigastrium is inspected, it may be difficult to distinguish the two.

FIG. 309.

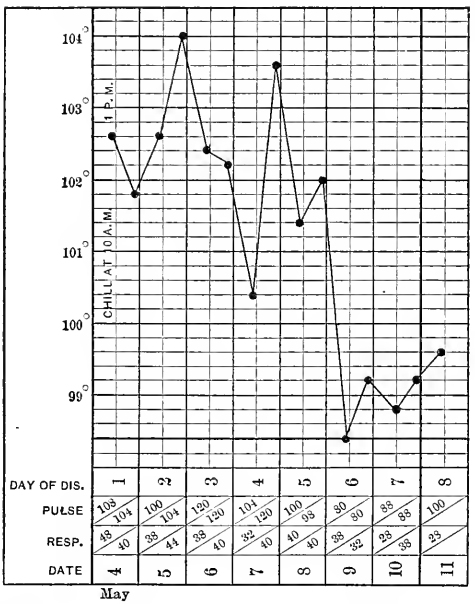


Pneumonia; sudden rise; termination by crisis; pseudocrisis on eighth day. (Original.)

In ordinary cases which run a normal course the cough is followed by *expectoration*, which is at first viscid mucus, but gradually becomes reddish brown from admixture of blood—the *rusty sputum* of pneumonia. This sputum is characteristic, almost pathognomonic. It is expelled with difficulty from the mouth, clinging to the lips or to the mustache. It can not be removed from the spit-cup by turning it upside down. It continues to be rusty until the crisis approaches, when it becomes purulent and is discharged with ease. In typhoid pneumonia it looks like prune-juice. (See Sputum.) It contains blood, alveolar epithelium, the specific micrococcus, and later, pus and small fibrinous casts.

Fever. The chill that precedes the fever is pronounced, and is always a warning to look for a pulmonic inflammation. In children a convulsion is rarely absent in frank pneumonias. During its occurrence the body temperature rises. In twelve hours it reaches 104° to 105° F. (See Figs. 309 and 310.) It remains at this point, obeying the laws of diurnal variation. The hot, dry skin, the parched lips, the dry tongue, the thirst, the anorexia, the hurried breathing, the occasional delirium, the loaded urine attest its presence. At the end of the third, or more frequently the

FIG. 310.



Pneumonia from first day; pseudocrisis on fourth day; crisis began on fifth. (Original.)

fifth, seventh, or ninth day, *crisis* takes place; the fall is abrupt, and the normal or a subnormal temperature may be reached in from five to fifteen hours. *Pseudocrisis*, as the accompanying chart indicates, may precede the true crisis by twenty-four or forty-eight hours. The decline however, may take place by lysis. Protracted fever indicates delayed resolution or the occurrence of a complication.

Cerebral Symptoms. In some cases, especially in children, the onset of the disease may be marked by a *convulsion*. This is said to occur more frequently in apical pneumonias than in pneumonias of the base. Headache and delirium are so pronounced in some cases as to simulate meningitis. This is most likely to be the case in severe apical pneumonia in children, and in double pneumonia either in children or in adults. *Delirium* may occur during the height of the fever, and occasionally is maniacal. Nocturnal delirium may be a constant symptom in very grave cases. In drunkards it may simulate delirium tremens, and may be pronounced without much fever. In the later stages of grave or fatal cases a low form of delirium, with a tendency to coma, is common.

Heart and Pulse. The pulse is small at the time of the chill, but becomes full and bounding during the fever; later it may become dicrotic. Reference has been made to the pulse-respiration ratio. The pulse varies in frequency and in character with the type of the disease. In healthy adults it is rarely over 110. In the debilitated it may be very frequent, small, and feeble; in the aged, frequent and dicrotic. Extensive consolidations reduce the amount of blood in the general circulation, cause rapid action of the heart and a small pulse, and favor death with the heart in asystole.

The heart-sounds are clear. A murmur low in pitch is often heard in the mitral and pulmonary areas. The left ventricle acts forcibly. The pulmonary second sound is accentuated. If dilatation and failure of the right heart take place, the area of dulness may extend beyond the right edge of the sternum, an epigastric impulse be noted, turgescence of the veins in the neck become marked, but above all, the previously accentuated pulmonic second sound may become weak or disappear.

Gastro-intestinal Symptoms. Vomiting frequently occurs in children at the onset, and both in them and in adults may persist and mask pulmonary symptoms. The appetite is lost. The tongue is furred, and may become dry and brown. The bowels are constipated except when complications occur. The *spleen* is enlarged. The vomiting and epigastric pain may be so pronounced as to mask the pulmonary symptoms. The occasional presence of jaundice has caused pneumonia to be mistaken for hepatitis, congestion of the liver, and even for gallstones. I saw a case of pneumonia, said to be appendicitis and peritonitis because of the characteristic pain, colic, and vomiting, followed by great abdominal tenderness in the upper abdomen.

Blood. Leucocytosis is a marked accompaniment of pneumonia, especially in cases ending favorably. The white cells may be increased from 12,000 to 40,000. An increase in the polymorphonuclear cells is always present. They fall with the crisis, or probably a day after the termination of the fever. In malignant forms there may be no leucocytosis. Increase in the fibrin network, causing the "buffy coat" of older writers, is commonly seen.

Cutaneous Symptoms. Herpes on the lips, the nose, or the genitals is of common occurrence. Sweating occurs with the crisis, or if heart-failure is imminent.

Urine. The urine is scanty and high-colored, and may contain a small amount of albumin. In some cases the chlorides are found to be absent. This is determined by acidulating the urine with a drop or two of nitric acid, and then adding one or two drops of a 10 per cent. solution of silver nitrate. If chlorides are present, a heavy white cloud of silver chloride is thrown down. The chlorides are not invariably absent, or even diminished in pneumonia, hence their reappearance, which is said to indicate beginning convalescence, loses its value as a prognostic sign.

Physical Signs. (See Diseases of the Lungs, Plate XXI.) **Consolidation.** This consists in a diminution of the amount of air and an increase of solid contents. On *inspection*, diminished movement. If extensive consolidation, enlargement of the affected side. On *palpation*,

inspection confirmed and increased vocal fremitus discovered. Both are more marked at the height of consolidation. *Percussion.* In the first stage, impaired resonance or Skodaic resonance. In the stage of hepatization, dulness or flatness, but without any wooden quality or marked resistance. *Auscultation.* In the early stage, that of congestion, the respiratory murmur is suppressed and crepitant râles are heard at the end of inspiration. On full inspiration or after cough a bronchovesicular respiration is brought out. When consolidation has taken place, the respiratory murmur is bronchial. Râles, if present, are moist subcrepitant râles due to associated bronchitis, or a few crepitant râles may persist, and a friction-sound be heard. When resolution sets in, the crepitant râle reappears, quickly followed by moist subcrepitant râles, heard both on inspiration and expiration, while dulness gradually yields to impaired resonance. The respiration loses its bronchial character and again acquires a vesicular element before becoming completely normal. It may be a week or two, or many months, even in uncomplicated cases, before the percussion-note becomes perfectly clear and râles wholly disappear.

The physical signs are modified by the intensity of the inflammation in the lung structure and by the pleural complications. In *massive pneumonia*, for instance, the auscultatory signs are absent. On percussion, the lung is absolutely flat. There is no fremitus or tubular breathing. The physical signs resemble those of pleurisy with effusion. In *central pneumonia* the physical signs may be delayed until the third or fourth day. A few râles or feeble breath-sounds over a small area may be the only indication of a possible lung process. In the *aged* the physical signs are obscure. In patients with laryngeal disease or marked obstruction in the nasopharynx the physical signs may be indefinite. Bronchial breathing may not be heard unless the patient takes a full breath or coughs. In this class of cases, as well as in those with feeble respiratory movement, as the aged, the weak, and in those suffering from some other disease, as tuberculosis, the physical signs are not made out because of the deficiency of respiratory movements. The indefinite character of the physical signs makes the diagnosis all the more difficult, because it is in this class of subjects that the general symptoms of infection are very slight. Increased respiration may be the most suggestive sign. Slight elevation of the temperature and more or less stupor may be the only other clinical symptoms.

Duration and Course. The duration of the disease is from one to two weeks. It may subside by crisis on the third, fifth, seventh, or ninth day, or gradually by lysis. Crisis is marked by a profuse sweat, a copious discharge of limpid urine, or sometimes by a few loose movements of the bowels, accompanying a fall of temperature to or below normal. Instead of clearing up, the pneumonia may progress to suppuration, abscess, or gangrene. These conditions can be made out by the character and range of temperature, the general condition of the patient, the sputum, and the physical signs. Termination in abscess or gangrene is rare. In cases proceeding to a fatal issue the strength fails, respiration becomes more labored, and expectoration increasingly difficult. The number of respirations often diminishes, but the pulse continues frequent and often

becomes small and irregular. Physical examination shows diffuse bronchitis with œdema. The heart's action is irregular and rapid. The sounds are weak and feeble; the first becomes short and snappy like the second, and later both are weak or indistinct. Death may occur abruptly from convulsion, or more frequently from asphyxia due to œdema of the lungs, which in turn sets in on account of weakness of the heart or the development of heart-clot from cardiac asystole.

Varieties. Migratory Pneumonia. Sometimes, with the reappearance of abundant râles and increased expectoration, the fever continues high, or, if the temperature has fallen to normal, again rises, the patient is disinclined to take food, has a dry, brown tongue, and is often delirious. In such cases the pneumonia is probably extending in the lung already involved or has attacked the other lung.

Typhoid pneumonia is an unfortunate name for an adynamic form of the disease with typhoid symptoms. If it arises in the course of or complicates low fevers, it is usually of the typhoid type: but it occurs also in those much exhausted, in depraved health, or exposed to unhygienic surroundings. It is found also in cases of septicæmia, in Bright's disease, and in drunkards. It is a type occurring in the negroes in the southern part of the United States.

The characteristic features of this form of pneumonia are the great physical prostration and the weak heart action. The fever is high, the respiration and pulse frequent, and delirium and vomiting are more common than in the ordinary form. The skin sometimes has a dusky hue; the tongue is heavily coated, or may be dry and brown, and sordes collect on the teeth. The sputa may be rusty, and sometimes pure blood is expectorated. The disease may rapidly prove fatal or may linger for a long time, the patient only gradually coming out of a low typhoid state. It is always dangerous.

Bilious pneumonia is the name given to a type of pneumonia occurring in persons who are already suffering from malarial poisoning. The initial chill lasts longer, and the pain in the side, from coincident pleurisy, is more marked than in ordinary pneumonia. The fever is more remittent, and jaundice and vomiting are present.

Pneumonia in infants is characterized by nervous symptoms. Repeated convulsions and active delirium may be most pronounced, followed by torpor and coma. There is no sputum and but little cough. The apex of the lung is affected.

Pneumonia in the aged is characterized by latency of symptoms. There is but little cough and expectoration. A tendency to the typhoid state, however, is pronounced. The physical signs are obscure.

Pneumonia in alcoholic subjects also develops insidiously and may be masked by the symptoms of delirium tremens. The temperature may be the only indication of infection, as there is neither pain, cough, expectoration, nor dyspnœa.

Pneumonia with other Infections. *Staphylococcus* and *Streptococcus pyogenes*, the colon bacillus, and *Bacillus pneumoniæ* (Friedländer) are often found with the pneumococcus, and may predominate, inducing a *mixed infection*. The micro-organisms which cause diphtheria, typhoid

fever, influenza, and the plague may cause a condition which resembles that of lobar pneumonia in the extent of the consolidation. *Micrococcus lanceolatus* is found in increased numbers in the sputum of these cases. There is not, however, the same intensity of pulmonary symptoms. The respirations are not so hurried. The physical signs, while extensive, are obscure, and indicate rather a heavy lung (congested) than one greatly consolidated. There are impaired resonance, feeble breathing, and a few râles in a large number of cases.

It is this form of lobar pneumonia which is difficult to distinguish from bronchopneumonia or catarrhal pneumonia—an infection which usually begins in the upper air-passages. This form of local infection is considered in the chapter on Diseases of the Lungs.

Diagnosis. The diagnosis is based upon the aggregation of special symptoms. The mode of onset, the chill, the course of the fever, the pain in the chest, the cough, the peculiar expectoration, the dyspnœa, the abnormal pulse-respiration ratio, the peculiar character of breathing, the physical signs, and leucocytosis are the phenomena of the symptom-complex. It must be remembered that in children, in the aged, in drunkards, in cases of chronic disease, the type is different. In drunkards cerebral symptoms are more marked. In children the cerebral symptoms are more prominent, the expectoration often absent. In the aged the cough, the expectoration, and the fever are not pronounced; the former may be absent; the onset is insidious. The same onset and course occur in wasting diseases, as cancer, phthisis, Bright's disease, diabetes, and organic heart disease. In this class of cases a small patch of pneumonia, difficult to determine on physical examination, may be attended by the gravest general symptoms. In all of the above cases, if there is *fever* without cause, although no pulmonary symptoms are present, the lungs must be examined repeatedly. In many such cases the physical signs are obscured because respiratory action is enfeebled by the primary condition.

Pneumonia must be distinguished from other acute inflammatory affections of the lung and pleura and from acute tuberculo-pneumonic phthisis. The evidence for each is considered in the respective sections. The presence of leucocytosis serves to distinguish it from acute tuberculosis and from typhoid fever, meningitis, and influenza. To distinguish pneumonia from pleurisy with effusion, the aspirator may be used.

Bacteriological Diagnosis. Staining and microscopical examination of the sputum reveal the characteristic micro-organism. Care must be taken to secure the sputum from the lung. By inoculation of rabbits with the sputum the disease is readily reproduced. The organism is not readily, although it frequently has been, found in the blood. (See the Sputum.)

In certain cases pneumonia may be distinguished from *cerebrospinal meningitis* by the results of spinal puncture alone; from *acute tuberculous pneumonia* by the examination of the sputum. The diagnosis in the latter instance may be postponed, as tubercle bacilli are sometimes not found until the tenth or twelfth day. (See Tuberculosis.) Typhoid fever sometimes resembles pneumonia, and must be distinguished after the first week by the results of serum diagnosis.

Pneumococcus Septicæmia.

The account we have just given of pneumonia represents but one phase of the pneumococcus infection. This infection may be attended by very grave symptoms, especially those of a toxic nature, with but little if any involvement of the lung tissue. It is well known that we may see the chill, fever, rapid pulse, and hurried respiration, with but little evidence of consolidation in the lung, but with nervous symptoms paramount. Delirium, stupor, coma, with the phenomena of the ataxic or the typhoid state, may prevail. (See pages 358 and 359.) In the ataxic state the symptoms resemble those of mania. In the typhoid form they are not unlike those of uræmia. In either instance death ensues in coma or from heart-failure with its attending symptoms. Preceding the cardiac failure the urine is diminished in amount and the secretions generally suppressed. The pneumococcus may be recovered from the blood, as has been done fairly frequent.

In other forms of this infection the localization of the process is in the pleura, as in empyema, in the pericardium, in the endocardium, and in the cerebral meninges. Pneumococcus inflammation of these structures is very common. It may develop at the same time that the lungs are affected, independently of the process in the lungs, or subsequent to it. These forms will be considered in a discussion of the various local inflammations just referred to.

It is important to remember that in pleural, pericardial, and cerebro-spinal infections the nature of the infection can be determined by *aspiration* and bacteriological examination of the fluid removed from the infected serous cavity. The pneumococcus infection can be positively diagnosticated in this manner.

These complications, which occur in the course of the disease, modify the clinical picture and obscure the diagnosis.

Tuberculosis.

The infection discussed in this section prevails to a greater degree than that of all the others combined. In some forms, as pointed out in the clinical description, *fever* is one of the gravest symptoms. In other forms the febrile process may not be pronounced. It must be remembered that the fever may be due to the specific micro-organism or its toxin, or it may be due to a mixed infection. Staphylococcus and streptococcus infections are common attendants upon the tuberculous infection. This secondary infection may disappear or may become the most prominent infection. In many instances a terminal infection ensues, and is the cause of death. Infection by the pneumococcus is the most common of these terminal infections. (See page 374.)

Tuberculosis is an infectious disease, the course of which may be acute or chronic. It is caused by *Bacillus tuberculosis*. This micro-organism sets up a specific inflammation characterized by the development of nodules or tubercles, or by a diffuse growth of tuberculous tissue. Either anatomical product may undergo caseation or sclerosis, and in either instance ulceration or calcareous degeneration may set in.

Invasion of the body by the micro-organism may give rise to general infection, with an eruption of miliary tubercles in most of the organs and structures of the body, or to a local infection. General tuberculosis is acute; local tuberculosis may be acute or chronic. In acute tuberculosis the serous membranes, the lungs, liver, kidneys, lymphatic glands and spleen, the bone-marrow, and choroid coat of the eye may be invaded in whole or in part. In chronic tuberculosis the lymph-glands, the lungs, the serous membranes, the tissues and organs of the alimentary canal, the liver, the organs of the genito-urinary system, and the brain and cord are individually invaded.

Diagnosis. The diagnosis of any form of tuberculosis is aided by the determination of the chief factors in its etiology, when this is possible.

Bacteriological Diagnosis. *First.* The discovery of *Bacillus tuberculosis* in any inflammatory area, or any product of inflammation, as serum, blood, pus, or the secretion from any gland or mucous membrane invaded by the disease, establishes at once the diagnosis of this condition. The method of determining the presence of this micro-organism is fully detailed in the various descriptions of tuberculosis, in the discussion of local diseases, and in the accounts of the examination of the sputum, exudates, and transudates. *Inoculation* of inflammatory products, as of a gland or of a fluid which has been sedimented, is a positive mode of diagnosis. Guinea-pigs are selected for inoculation.

Second. As tuberculosis is an infectious disease, discovery of the *source* of the infection is an aid in the diagnosis. Infection takes place through the inhalation of the sputum or other secretions, which when dry float in the air. It implies in a measure more or less contact with individuals previously infected. In rare cases such contact is productive of the disease by means of direct contagion. The second source of infection is the food-supply. This may occur from the consumption of milk secured from a cow infected with tuberculosis. The eating of meat of tuberculous animals may possibly lead to infection. Direct inoculation is another but rarer source of infection, and usually occurs accidentally.

Third. It is possible that tuberculosis may be *inherited.* A more prominent ætiological factor, which aids in the diagnosis of the disease, is the presence of a certain type of structure which is a marked hereditary characteristic in families, on account of which feeble resistance is offered to the invasion of the tubercle bacillus. The phthisical and phthisinoid chest which belongs to this type has been described elsewhere, and the tuberculous and scrofulous states have been outlined. (See page 139 and Chapter XXXV., Part I.) These anatomical conditions, which are inherited, undoubtedly favor the development of tuberculosis.

It is a mistake to lay much stress in the diagnosis of tuberculosis upon the age or the occupation of the individual. Tuberculosis may occur at any age. It is true, however, that at certain periods of life the tubercles are distributed more commonly in one group of organs, while in other periods they affect another group. Lymphatic, joint-, and meningeal tuberculosis is most common in the first decade of life; the mesenteric glands are particularly subject to invasion at this period.

The diagnosis of tuberculosis, whether local or general, is further aided

by a complete knowledge of the phenomena that attend the entrance of the virus into the body and the mode of diffusion throughout the body after infection has taken place. The phenomena at the point of entrance of the micro-organism are nearly always distinct.

General Tuberculosis. The *general* invasion is associated with symptoms like those of specific fevers. The local secondary effects upon the tissues are always decided. It must be borne in mind that after the exposure, which may lead to infection, either an acute form of tuberculosis of a general character may be set up, with or without marked local symptoms, or acute local tuberculosis alone may arise.

Local Tuberculosis. In local tuberculosis the disease is confined to one organ or to the lymphatic glands and the organs in the lymphatic distribution, as the bronchial glands, which are primarily affected, and to the lungs. In these structures the entire process of nodular formation, caseation or sclerosis, ulceration or calcification, may take place. The disease remains primarily local. On the other hand, it may spread by continuity of structure through the lymphatics throughout the remainder of the organ affected, leading to its ultimate destruction and the death of the patient; or general infection of the system may take place from the primary local area. The primary seat of infection may be the lungs, the larynx, the alimentary tract, or the genito-urinary organs. Primary tuberculosis of the serous membranes, of the lymph-glands, of the bones and joints, may take place.

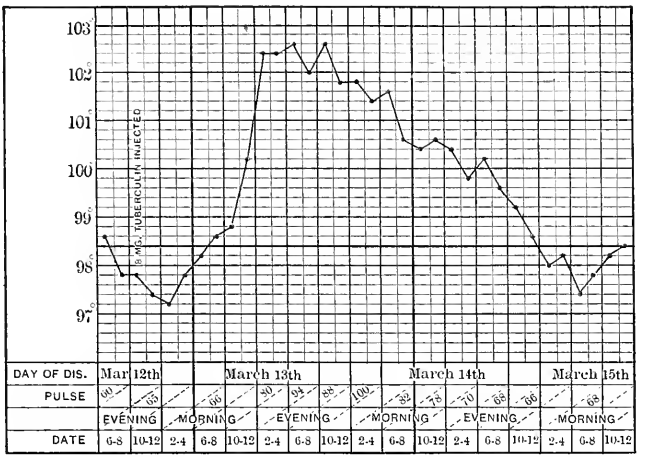
The symptomatology and diagnosis of the various forms of tuberculosis are detailed in the section devoted to the special diseases of the various organs of the body.

The Tuberculin Test. The physical signs and clinical symptoms may point to an inflammatory process in one of the many structures of the body which may be invaded by tubercle bacilli. On the other hand, failure in health, loss of weight, anæmia, and moderate fever may alone occur. The nature of the inflammatory process may be obscure. To determine more accurately whether the inflammation is tuberculous or not, or the "decline" due to tuberculosis, we can resort to the use of tuberculin. Since the researches of Koch, who introduced tuberculin as a remedy in tuberculosis, he himself as well as a number of other observers have employed this preparation to determine the presence of tuberculosis in the body. In this country Trudeau has been the earliest and most earnest exponent of this method of diagnosis. After the injection of tuberculin a group of phenomena follows, known as the tuberculin *reaction*, if tuberculosis exists anywhere in the body. It was thought the occurrence of this reaction was necessary to bring about a cure. As a therapeutic measure its value has not been upheld by experience. The invariable production of the reaction has led to its use as a diagnostic agent.

Phenomena of Reaction. About twelve hours after the injection of tuberculin the temperature rises rapidly. In the course of a few hours it has risen two or three degrees. This elevation of temperature is attended by malaise, pains in the head, back, and legs, and sometimes nausea or vomiting. The maximum temperature is maintained for two or three hours, and then a gradual decline to the normal takes place.

The normal temperature is reached in from twenty-four to thirty-six hours. The whole period of the reaction, from the time of the injection until the termination of the fever, is about forty-eight hours. With the fall of temperature to normal the constitutional symptoms subside. The accompanying chart (Fig. 311) shows the course of the fever in a typical reaction.

FIG. 311.



Typical reaction with tuberculin. (Original.)

Method. For twenty-four to forty-eight hours preceding the test the patient's temperature should be taken every two hours to determine the range at this period of the disease. The injection should be made at a time when the reaction can be observed—*i. e.*, during the period of normal or subnormal temperature if the temperature of the disease is intermittent. The hour of day selected to inject the tuberculin should be such that the reaction may be conveniently observed during the waking hours of the patient. Bedtime or the early morning hours are the most convenient.

The site of the injection is not important; usually the interscapular space is selected. The amount of tuberculin employed is of the greatest importance. The initial dose should never exceed 5 milligrammes, and it is better to use less than this, and an increasing quantity injected every second or third day. The maximum dose should not exceed 10 milligrammes. For children one-twentieth to one-tenth of a milligramme may be the initial dose. The crude tuberculin should be diluted with a 1 to 2 per cent. solution of carbolic acid at the time it is used.

At the point of injection a little redness and infiltration, with tenderness to the touch, are observed. This local reaction may also be seen at the site of former negative injections when the larger dose produces reaction. In pulmonary tuberculosis in which physical signs are obscure some auscultatory phenomena which were previously absent may be found during the period of a reaction. The test also enables one to detect tuberculosis in the pleura, pericardium, peritoneum, genito-urinary tract,

and lymphatic glands, the meninges, bones, and the skin. The test is of special value in cervical adenitis.

It must be remembered that a negative result with large doses of tuberculin is of more value than a positive one. In the former instance one can affirm that tuberculosis is absent, as well as that there is no old focus in any of the organs. It must also be remembered that the test should only be employed after all other means have failed to make a positive diagnosis.

Acute Miliary Tuberculosis.

This form of the infection has been spoken of elsewhere. (See Chapter VI., Part II.) Its course may resemble typhoid fever, septicæmia, or malignant endocarditis. It usually develops in the course of tuberculosis in some other organ of the body. The typhoid form has been described in the section indicated. It must not be forgotten that the diagnosis is rendered positive by the demonstration of the presence of tubercle bacilli in the blood, or of the occurrence of choroidal tubercles in the eye-ground. Another form is attended by marked pulmonary symptoms, which is seen in the bronchial pneumonia that occurs in children following measles and whooping-cough. (See Catarrhal Pneumonia.) Of the pulmonary symptoms, dyspnœa is the most prominent. Cyanosis is marked. The physical signs are not prominent, and may be those of bronchitis alone. Although there is impaired resonance at the base of the lungs, areas of hyper-resonance are observed above and anteriorly. Collapse of the lung may cause tubular breathing. The temperature rises to 102° or 103° F. An inverse type may be seen.

The diagnosis of acute tuberculosis is determined by the history of infection from extraneous sources or from local tuberculosis in some portion of the body, and by the presence of bacilli.

The following conditions should point to the possibility of chronic tuberculosis in some portion of the body: (1) emaciation, not otherwise explained; (2) chlorosis or anæmia; (3) weakness without cause; (4) fever—the temperature should be taken every two hours during night and day; (5) causeless sweats; (6) gastro-intestinal catarrh; (7) morning nausea; (8) signs of local inflammation in some organ of the body.

Influenza.

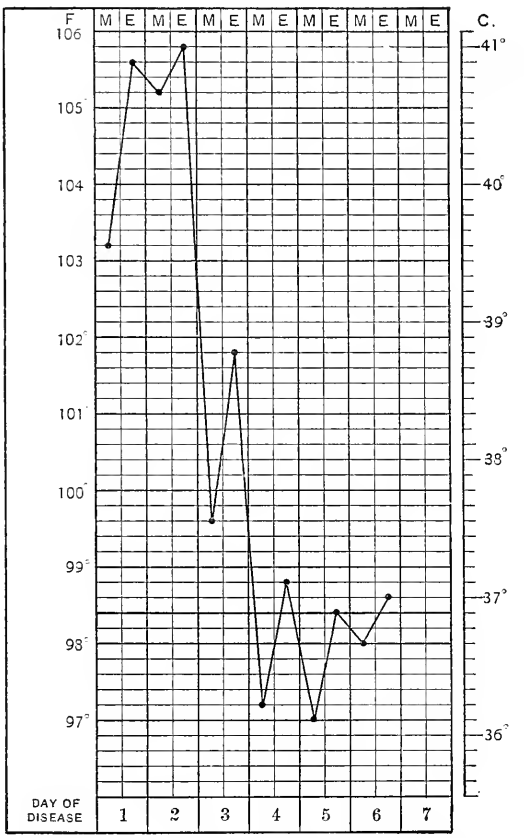
High temperature out of proportion to the local signs of inflammation in the lungs or other structures characterizes this infection. The *fever* may be continuous, remittent, or intermittent.

Influenza is a specific contagious febrile disease, occurring in widespread epidemics, having a very short period of incubation, and characterized by great prostration, marked nervous symptoms, and catarrhal inflammation of the respiratory or gastro-intestinal tracts, or both. There is great liability to relapse, and to complications, which are generally pulmonary or nervous.

The disease generally begins with the ordinary symptoms of coryza;

but the headache over the eyes and root of the nose is more severe, and may be so agonizing as to mask all other symptoms. The lacrymation, rhinitis, and tormenting cough are all usually worse than in ordinary coryza. Physical weakness, weariness, and depression of spirits are almost invariably present, and they sometimes reach an extraordinary degree. Fever is usually moderate (100° to 102° F.), but may be 104° to

FIG. 312.



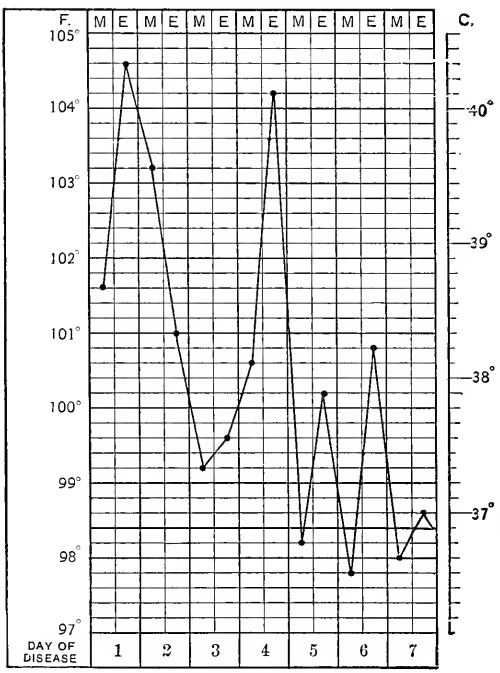
Temperature in influenza—interrupted crisis. (WILSON.)

105° F. for several days, and then gradually subside. It may terminate by crisis (Fig. 312), or may assume an intermittent or remittent type (Figs. 313 and 314). In ordinary cases the patient seeks relief first for the headache, severe aching pain in back and limbs, and extreme weakness; if these are relieved, he is apt to complain most of incessant racking cough, often due more to a tracheitis than to bronchitis. Nausea and vomiting are not uncommon, especially in the morning, at which time also the patient frequently feels worse than he does later in the day. Sleep is broken and restless, and may be accompanied by drenching perspirations. Severe neuralgic pains are common. In some cases the disease attacks the stomach and bowels especially, and vomiting and diarrhœa

are the prominent symptoms. In other cases the predominant symptoms are nervous, and great pain with prostration masks any catarrhal symptoms that may be present. Torpor and delirium may be present. Sometimes a prolonged and severe attack of asthma marks infection in susceptible persons.

The duration of the disease is from a few days to a few weeks. Convalescence is remarkably tedious, and is characterized by persistent weakness. Sweats are often annoying during this time. The heart often continues for some time to beat too frequently and to be easily excited by exertion. Relapses are common.

Fig. 313.



Influenza—intermittent type. (Wilson.)

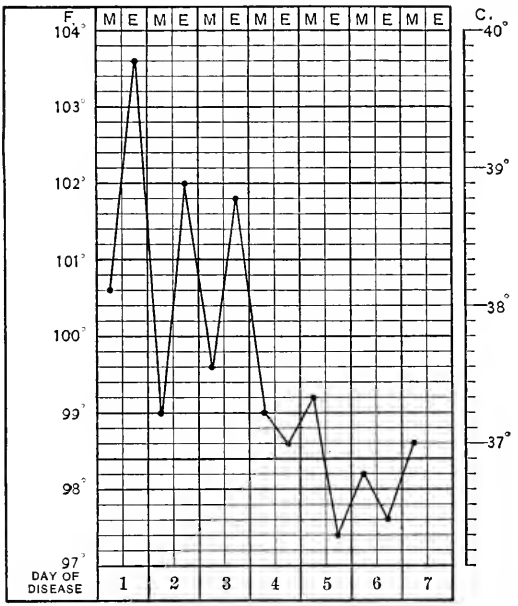
Diagnosis. **Bacteriological Diagnosis.** This is possible when the characteristic bacilli are detected by the means described in the section on Sputum. Influenza in the great majority of cases is easily recognized. In certain cases, however, it is to be differentiated from *pneumonia*, *typhoid fever*, and *cerebrospinal meningitis*.

Cases in which the disease sets in with high fever and marked chest symptoms are very apt to be mistaken for *pneumonia;* but the headache and prostration are more intense, while the respiration is not so frequent. Sweats are common, and albumin and casts in the urine are by no means rare. Physical exploration shows that both lungs are involved, though often not to the same degree. Resonance is impaired, and auscultation

shows moist crepitant and subcrepitant râles, which seem to be due to an œdematous condition of the lung tissue associated with a diffused bronchitis. A true lobar pneumonia is rarely present even as a complication.

If diarrhœa is one of the symptoms, *typhoid fever* has to be excluded. This is extremely difficult in the first two or three days. As a rule, headache, backache, nausea, and sleeplessness are at this time greater in influenza, the spleen is not so much if at all enlarged, the diarrhœa can be checked, and tenderness and pain in the right iliac fossa are absent.

FIG. 314.



Influenza—remittent type. (WILSON.)

Influenza can be distinguished from *cerebrospinal meningitis* by noting the fact that it begins with coryza, whereas the latter often sets in with chill, vomiting, and faintness; the headache in the former is usually frontal, in the latter occipital, and accompanied by stiffness of the back of the neck. Further, in cerebrospinal meningitis there are often swellings of the joints, and delirium alternating with coma, and in young subjects convulsions are common.

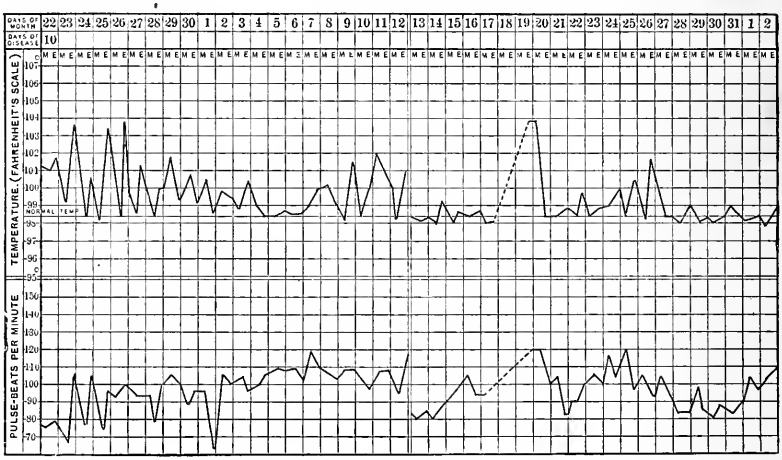
Finally, it may be said that the pronounced diagnostic feature is the preponderance of general symptoms over local inflammations. The occurrence of undue exhaustion, extreme general neuralgias and myalgias, high fever, and profuse sweats, without intense catarrh or inflammation to account for or to co-ordinate with them, is of the highest diagnostic significance. The presence of an epidemic, the contagious nature of the affection, the sudden onset, and the bacteriological diagnosis, all point to influenza.

Cerebrospinal Fever.

Cerebrospinal fever, also known as spotted fever, is an acute, specific, infectious, and mildly contagious disease, endemic and epidemic, characterized by evidences of *systemic infection*, and generally also by symptoms depending upon inflammation of the cerebral and spinal meninges—particularly intense pain in the back and head, hyperæsthesia, retraction of head and neck, delirium, coma, convulsions, and vomiting.

It is most common in cold weather, and in children under fifteen years of age. None of the epidemics show a continuous extension. The period of incubation is unknown, but is probably short. It is free from symptoms. The invasion of the disease is abrupt, although in some instances the patient may complain of rheumatoid pains in the limbs or a

FIG. 315.



Cerebrospinal meningitis, showing irregularity of pulse and temperature. (COUNCILMAN.)

joint, and headache and weakness. Usually the first symptom is a severe chill, which may awaken the patient from sleep. In other cases the initial symptom is a convulsion. Then quickly follow repeated vomiting, intense headache, sometimes accompanied by backache, retraction of the head, delirium, and extreme prostration.

In this infection more than all others the course of the *temperature* is without diagnostic significance unless it be that the absence of a characteristic course is significant. Its extraordinary irregularity is most striking when a large number of charts are examined. The fever may have the course and exacerbation of a typhoid temperature, but as a rule it is more like that of tuberculosis. It is often of very short duration, followed by a prolonged subnormal temperature. It may be high from the onset of the disease, or remain below 100° for several days, and then suddenly rise to a great height. Remissions and exacerbations may attend many of the cases. The most marked feature, apart from the irregularity of the temperature, is the inequality between the pulse and the temperature. In some instances the pulse is rapid, and the tempera-

ture is normal or subnormal, while later in the disease the pulse may be slow when the temperature rises to a considerable height. The temperature then may be said to be intermittent, remittent, or continuous; it may be intermittent at one period, continuous at another; it may be afebrile at one period and continuous at another.

Symptoms. The face is pale and livid, expressive of suffering, and the patient may toss from one side of the bed to the other, begging relief for his headache. Simple stiffness of the muscles of the neck may prevail. The pain in the head may be occipital or frontal. The pain in the back becomes more severe, and root-pains dart in all directions, but especially into the limbs or joints, which may be swollen and tender to the touch; in fact, the whole skin is hyperæsthetic and the reflexes are increased. The spinal muscles become rigid and the head is often retracted. Less frequently trismus occurs and the back is arched. Delirium is common at night. It may develop very early or appear at a late period of the disease. It is sometimes violent or low and muttering. It is often of a sportive type, the patient making absurd remarks, cracking jokes, or singing snatches of a comic song. Delirium may alternate with tonic or clonic convulsions and with stupor. The appetite is poor, the bowels constipated. A remission may occur on the third day, with temporary improvement of the symptoms.

As the attack progresses there may be strabismus, which is usually divergent, inequality of the pupils, nystagmus, ptosis, and optic neuritis. Vertigo, tinnitus, anosmia, and photophobia are common. Hyperæsthesia and delirium persist. Facial paralysis, a monoplegia, a hemiplegia, or a paraplegia may occur. The pulse becomes more frequent and the fever continues. In favorable cases improvement now begins, the headache and root-pains abating, and delirium and spasms becoming less frequent. In unfavorable cases the convulsions may become more severe and end in fatal coma, or the patient may sink into a typhoid condition, with nephritis as a complication. Coma may come on in the beginning and continue until death.

Kernig's Sign (Kernig, 1884; Netter, 1898). This sign is of value only in the diagnosis of meningitis in general. It is determined by placing the child in the dorsal position, with the legs relaxed and fully extended at the knees. When the child is raised to a sitting posture, the knees are flexed, and can not be extended on account of contracture of the posterior muscles of the thigh. In adults, if the patient is propped up, or seated on the side of the bed, and an attempt made to extend the leg on the thigh, there is contraction of the flexors. The test can be equally well performed by flexing the thigh on the abdomen until it makes a right angle. When an attempt is made to extend the leg, it will be found that the limb can not be fully extended if meningitis is present. "It is not always present in children under two years of age." (Packard.)

The *skin eruptions*, which explain the name "spotted fever," are not always present and exhibit no constant character. Herpes and petechiæ are the most frequent; in other cases the eruption is a purplish mottling, or is macular, or the eruption resembles that of measles. Herpes is most common on the nose and mouth, then on the cheek, forehead, eyes,

and ears. The blood shows a leucocytosis, the increase being due to the polynuclear leucocytes.

In the **malignant (fulminating) form** of the disease death occurs in a few hours, or in two or three days. Such cases are apt to arise early in an epidemic. The patient has a violent chill; delirium occurs early; the headache is less intense, or at any rate gives way rapidly to stupor and coma. The pulse is frequent and feeble; there may be no rise of temperature, the skin being cool, clammy, and cyanotic. Local or general convulsions may occur. The eruption may be purpuric, and even ecchymoses may occur. The urine is scanty and contains albumin and casts.

Mild cases usually occur late in epidemics, and are characterized by severe aching in the head, back, and limbs, nausea, vomiting, vertigo, and prostration. They closely resemble the nervous type of influenza, and could easily escape recognition except during an epidemic.

An abortive form, ending in recovery in two or three days, and an intermittent form, with exacerbations on alternate days, have been described.

The *duration* of the disease is from a few hours to two or three months. In ordinary favorable cases there is decided improvement toward the end of the first week, and convalescence is established in two weeks. It may become chronic and last for weeks, or, as already stated, may be fatal in a few hours. Relapses are common in some epidemics.

The most frequent *complications* are those involving the lungs and heart, particularly pneumonia and endocarditis or pericarditis. Pneumonia often occurs so early that it is difficult to decide whether it is primary with marked nervous symptoms, or is only a complication of the cerebrospinal fever. Nephritis also occurs.

The most frequent *sequels* are deafness, blindness, headache, and local palsies.

Diagnosis. The diagnosis in the presence of an epidemic is not difficult, although an absolute diagnosis can only be made by *lumbar puncture*. The fluid withdrawn is more or less cloudy if the patient has meningitis. If it is the epidemic form, microscopical examination of stained cover-slips and cultures will expose the diplococcus. In some cases fluid can not be secured, either because the spinal canal is filled with membrane or the fluid is retained in the lateral ventricles.

The fluid is turbid early in the disease; in some cases a purulent sediment forms in the bottom of the test-tube at once. In others the fluid is simply turbid, and after standing contains considerable fibrin and many cells. The fluid secured at the first puncture may be more turbid than that secured later, although the symptoms may be more severe than at first. If the acute symptoms subside, the fluid may be clear and no cells may be found. In the intermittent cases the fluid may be clear during the interval that the patient is without symptoms. In chronic cases there may be no turbidity. The *cells* in the spinal fluid are chiefly polymorphonuclear leucocytes—"pus-corpuscles." Small lymphoid cells and large endothelial cells may be present. The latter are phagocytic, and have large oval or round nuclei. They may contain leucocytes and blood-corpuscles. In the pus-corpuscles or leucocytes the

PLATE XVIII.

Fig. 1.

Fig. 2.

Cerebro-Spinal Meningitis. (Councilman.)

Fig. 1. Forty-eight hour culture of diplococcus intracellularis on Loeffler's blood-serum mixture.

Fig. 2. Abundant growth in twenty-four hour culture on fresh blood-serum. The colonies are minute, very numerous, and somewhat resemble similar cultures of the pneumococcus.

diplococci are found; they are rarely found outside of the cells. Late in the disease the pus-corpuscles do not stain sharply and are degenerated. In chronic cases the fluid contains a few pus-corpuscles which are smaller than usual and similar to lymphoid cells.

Bacteriological Diagnosis. The diagnosis of this form of meningitis can often be made by staining, and by microscopical examination of products from the nasopharynx removed from the nares with a swab. The disease is due to *Diplococcus intracellularis*, which appears in the diplococcus form as two hemispheres the size of the ordinary micrococcus. It stains with the ordinary stains for bacteria. It is decolorized by the Gram method. The staining is sometimes irregular, some of the diplococci being brightly stained, others faintly. There is some variation in the size of the organisms. Both the variation in size and staining are apparently due to degeneration. The two cocci are sharply separated usually, though sometimes they seem to be united. (Figs. 316 and 317.)

Fig. 316.

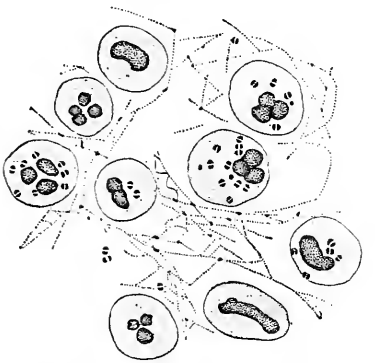


Fig. 316.—Pus-cells containing diplococci from the meninges. A few diplococci are in the exudate outside of the pus-cells. Between the pus-cells there are delicate fibrillæ of fibrin. The drawing is an accurate representation of a group of cells in the field of the microscope. (Councilman.)

Fig. 317.

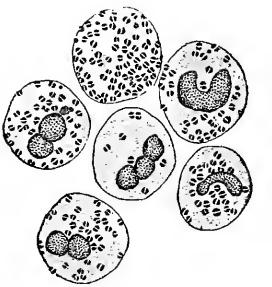


Fig. 317.—Pus-cells from an alveolus of the lung in a case of diplococcus pneumonia. The cells are swollen and contain immense numbers of diplococci. Both figures from stained cover-slips.

Cultures. Cultures should be made at the time of puncture. In the majority of cases a growth of the diplococcus is found, although rarely even in acute cases they may not grow. In chronic cases a growth is only rarely obtained. (Plate XVIII.) The micro-organism has been recovered from the blood, and from the fluid of an inflamed joint as well as from the spinal fluid.[1]

The epidemic form of meningitis must be distinguished from pneumococcus meningitis, tuberculous meningitis, and streptococcus meningitis. In the pneumococcic form the symptoms are comparatively slight and are usually preceded by pneumonia. In the streptococcic form the clinical history is like that of ordinary forms of meningitis. The evidence of an infection elsewhere is usually present. Tuberculous meningitis is recognized by the methods employed to detect tuberculosis elsewhere in a patient suffering from the usual symptoms of cerebrospinal meningitis.

[1] Osler, Philadelphia Medical Journal, 1899.

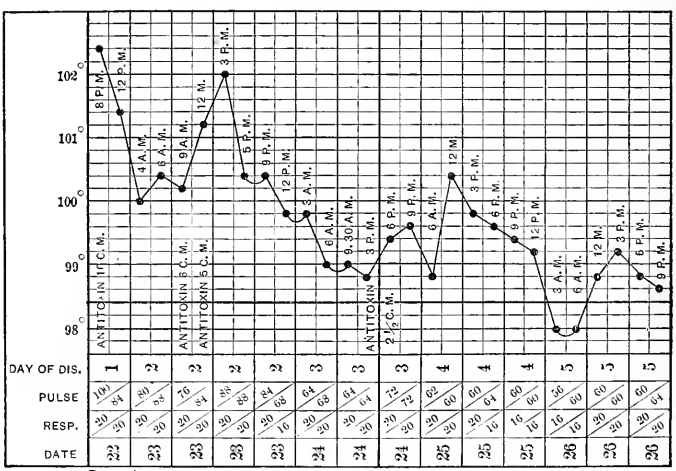
The most positive method of distinction of the various forms is by lumbar puncture. (See Chapter XLV., Part I.)

Diphtheria.

Diphtheria is an acute, specific, infectious, and contagious disease, sporadic and epidemic, occurring especially in children from one to six years of age, and characterized by insidious or abrupt onset, with moderate fever; and the development upon the fauces or upon any abraded surface of a grayish-white false membrane, which has a tendency to extend, especially to the larynx. The subsequent phenomena are those of stenosis of the larynx, toxæmia, with or without superadded uræmia, or marked cardiac weakness; it is further characterized by the liability to paralysis as a sequel.

Diphtheria is contracted by inhaling the expired breath of a diphtheritic patient, or breathing air which has been contaminated by the clothing of the patient or the discharges from his nose or throat. It may also be transmitted directly, as when a fragment of membrane is ejected by coughing and infects the mouth or eye of the physician or attendant. Moreover, air contaminated by diphtheria is contained in the sewers of large cities while the disease is endemic, and may persist in damp cellars if they have once been infected. Hence, sewer-gas and cellar-air may carry the disease. There is reason also for believing that a similar disease affects birds, fowls, and cats at times, and by them may be transmitted to man. These facts must be borne in mind in making the diagnosis.

FIG. 318.



Diphtheria. (Original.)

The specific poison is the Klebs-Löffler bacillus and its toxin.

While children from one to six years of age are especially liable to it, no age is exempt—neither the new-born babe nor the very aged. One attack does not protect a person completely against a subsequent attack.

The temperature range is variable. The infection may be intense, and yet the temperature remain subnormal, especially if the *fever* is due to the toxin, and not, as is frequently the case, to a mixed infection.

The period of *incubation* varies from a few days to two weeks, or perhaps longer in exceptional cases. As a rule it is less than a week. It is shorter when the poison is virulent and when infection has been upon abraded surfaces.

The *onset* in mild cases is deceptively free from positive symptoms. The child is languid, perhaps slightly chilly, and has a little fever, with thirst, impaired appetite, and discomfort in swallowing. Unless the nature of the trouble is suspected, the child is not thought ill enough to be kept in-doors. The throat is slightly inflamed, especially about the tonsils. The child may protest that there is no pain on swallowing. In from twelve to twenty-four hours after the onset, sometimes later, a grayish pellicle will be found upon the tonsils, and the cervical glands will be swollen. In more severe cases the disease begins with chill or chilliness, followed by a rise in the temperature to 102° or 104° F., sore throat, and sometimes vomiting, though this is not so common as in scarlatina. Convulsions and delirium may occur if the fever be high or the case malignant, but they are not common. The disgust for food makes it difficult to nourish the patient. Headache, thirst, and aching in the back and limbs may be complained of. Prostration is often very pronounced from the beginning.

Objective Symptoms. The characteristic false membrane appears at first as a grayish pellicle upon one or both tonsils, and spreads thence to the soft palate and pharynx. The membrane soon becomes thicker and whitish in color; when fully developed, it appears like white or grayish-white parchment, not lying loosely upon the surface, but imbedded in the mucous membrane, the inflamed swollen edges of which rise above the false membrane, surrounding it "as the crystal of a watch is surrounded by the rim." (J. Lewis Smith.[1]) As the membrane becomes older it may be brownish or even blackish in color if ferric chloride has been given. If it is forcibly torn from the underlying surface, hemorrhage is excited, and the membrane forms again. As the membrane loosens spontaneously there is often marked inflammatory reaction at the edges of the surrounding mucous membrane, and in the tonsils there may be decided sloughing, with a dark, gangrenous appearance.

The *temperature* usually falls on the second or third day, but this does not indicate either a favorable or an unfavorable end. A temperature slightly above normal is not uncommon in profound toxæmia.

Urine. Albumin is usually present early, and often tube-casts and renal epithelium also can be found. The submaxillary and cervical glands are swollen, and it may be difficult to open the mouth sufficiently to inspect the throat.

As pointed out by Buzzard and McDonnell, the patellar tendon reflexes are often abolished as early as the first day.

In *favorable cases* the membrane ceases to extend after three or four days; there is no extension to the larynx; the urine is free from albumin,

[1] Keating's Cyclopædia of Diseases of Children, 1889, vol. i., p. 606.

or only slightly albuminous; and the pulse-rate is not more than 100 to 120 per minute and of good force.

In *unfavorable cases* the membrane shows a tendency to extend upward into the nasal fossæ, producing a thin, irritating, excoriating discharge from the nostrils, and rendering mouth-breathing necessary; or it may extend to the ears through the Eustachian tube; into the maxillary sinus; or downward into the larynx, producing laryngeal stenosis. This is announced by hoarseness, with rapidly increasing difficulty in breathing. Inspiration is high-pitched, noisy, and difficult; the patient brings all the accessory muscles of respiration into action, the alæ of the nose play, the ribs are sucked in, and still the child pants for breath. Every now and then a paroxysm of coughing produces cyanosis.

In other unfavorable cases the throat symptoms are not dangerous, but *uræmia* develops. The urine is scanty, contains a large amount of albumin, considerable blood, and numerous epithelial and granular casts. There are œdema of the feet and puffiness of the eyelids. There is apt to be repeated vomiting; convulsions, followed by coma, and death may end the scene, or the patient may slowly be restored.

Heart. In still other cases the diphtheritic poison affects the heart. The pulse becomes feeble and very frequent, the first sound very faint; acute dilatation of the right heart may occur. There may be faintness and a tendency to cyanosis on the slightest provocation, or attacks of sinking and faintness may come without warning; in still other cases sudden exertion induces paralysis of the heart and death.

In some *malignant cases* the patient is overwhelmed by a large dose of the poison, and dies in from one to three days in collapse from acute toxæmia, without any special local symptoms to account for it. In others the false membrane extends rapidly over the fauces, pharynx, and nasal cavities to the larynx; death occurs from early obstruction, or, if it is postponed, there is extensive sloughing, with death from secondary blood-poisoning or septic pneumonia.

In exceptional cases the membrane is primary in the nares or larynx, or develops upon some abraded surface, as a burn, or in the vagina of a puerperal woman. It may also attack the mucous membrane of the eye or the seat of a recent operation. Diphtheria also occurs as a complication of other diseases, particularly scarlet fever.

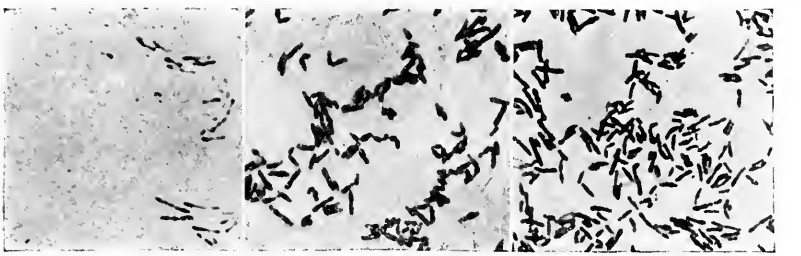
Sequelæ. The most frequent sequelæ are anæmia, albuminuria, and paralysis. The latter comes on in from one to two weeks after convalescence has set in, but it may appear much earlier, and in exceptional cases later. It may be marked simply by loss of the knee-jerk, which has been alluded to, or involve the palatal and pharyngeal muscles, causing nasal speech, difficulty in swallowing, and regurgitation of food through the nose, or there may be multiple peripheral neuritis.

Loffler's or the Klebs-Loffler Bacillus. This micro-organism is found in diphtheritic pseudomembranes, especially in the deeper portions, and has also been found in the blood.

Morphology. A bacillus 2 μ to 3 μ long by 0.5 μ to 0.8 μ broad, straight or slightly curved, with very many irregular forms. (See Fig. 319.)

The *pseudodiphtheritic bacillus* resembles the genuine in all respects, except that it is not pathogenic. It seems to be an attenuated variety of the former.

FIG. 319.



Diphtheria bacilli. × 1000. (From LOOMIS and THOMPSON.)

Diagnosis. Diphtheria is distinguished from ordinary *pharyngitis* by the presence of membrane. From *follicular tonsillitis* by the projecting mouths of the follicles containing a creamy-white exudate. Later the exudate may cover the entire surface of each tonsil and be difficult to distinguish from false membrane. The points of distinction are that in the former the exudate lies upon the surface and can be brushed off without force and without leaving a bleeding surface; whereas in diphtheria the membrane is imbedded in the mucous membrane and can not be torn from it without some violence. A raised, red, inflammatory border of mucous membrane overhanging the patch is strongly suggestive of diphtheria. In tonsillitis there is no appearance of membrane upon the soft palate or pharynx. Furthermore, in tonsillitis the onset is attended with more fever and pain in swallowing than is true in simple tonsillar diphtheria. The existence of albuminuria and swelling of the cervical glands indicates diphtheria, and the absence of knee-jerk is an important but not a constant sign of diphtheria. The presence of the Klebs-Löffler bacilli in a culture from a suspected throat is proof of the existence of diphtheria.

Septicopyæmia.

The clinical course of this infection and the bacterial causes have been considered in Chapter XXXII., Part I. It will be recalled that its phenomena may attend a number of the infections described in this and in previous chapters. When occurring in the course of pneumonia, diphtheria, typhoid fever, etc., its origin is recognized by the methods discussed in the chapter referring to these infections. Septicopyæmia caused by pyogenic organisms, the so-called "cryptogenetic sepsis," is recognized by bacteriological examination of the blood; by an examination of the morbid secretions; or by an examination of the products of inflammation. Bacteriological examination of the blood has its limitations. Usually only late in the course of the disease and in the more intense infections can the bacteria be found. Examination of the pus from foci of suppuration in the bones (osteomyelitis), in the joints (pyæmia), in the serous cavities (empyema, pericarditis, peritonitis), in the lungs (see Sputum),

and in the genito-urinary tract (see Urine), will show the infective micro-organism.

The causal micro-organism is detected by cover-slip preparations and cultures. (See Chapter XLV., Part I.)

Glanders.

Glanders is an infectious constitutional disease, transmitted from horse to man, appearing in an acute and chronic form, and characterized by an eruption, ozæna, small tumors, ulcerations, cough, and death in coma or collapse in from one to four weeks in the acute form, or in three or four months in the chronic form. The symptoms in the latter resemble at times syphilis and at times tuberculosis.

The disease is rare in man. It may be acquired by direct inoculation of an open wound with the pus from a glanderous ulcer or nasal mucous membrane, or indirectly from infected straw or other material. The raw meat of a glandered animal also has infective power.

The *fever* which attends this infection is similar to that of the infective granulomata. In severe cases the symptoms are like those of an acute septicæmia.

In *acute glanders* the onset is marked by headache, slight fever, and pains in the limbs. If a wound has been infected, it becomes painful, swollen, and behaves like any poisoned wound. Sometimes a diffused redness, resembling erysipelas, spreads from the infected point. Fagge refers to a case in which the first complaint was of pain in the side and dyspnœa, so that acute pleuropneumonia was suspected. An eruption, consisting first of papules, which rapidly become flat vesicles and then pustules or bullæ, appears in the first day or two, or sometimes not for a week or even longer. (Fagge.) The bullæ or pustules rupture and give vent to a thin, purulent discharge. There may be hard, painful lumps in the muscles, with subsequent suppuration (farcy). Ozæna is not always present. It appears in the second or third week of the disease. The nasal discharge is mucopurulent, then purulent and fetid. The nose subsequently swells and becomes red and very painful. Ulcers and even necrosis of the septum are the lesions; a similar catarrhal condition may exist in the throat, eye, larynx, and mouth, accompanied at times by ulcers and false membrane. The patient gradually sinks into a septicæmic condition, with irregular fever, dry brown tongue, albuminuria, delirium, coma, and collapse.

The *duration* of the acute form is from one to four weeks. Only one in thirty-eight cases collected by Bollinger ended in recovery.

In the *chronic form* there are ulcers upon the hand, face, forehead, or elsewhere. In other cases the lesions are abscesses in the joints which are followed by persistent fistulæ. In still other cases there is pustular eruption. Ozæna may or may not exist. In still other cases the prominent symptoms are cough, bloody expectoration, hoarseness, fever, and emaciation. Bollinger reports seventeen recoveries in a total of thirty-four cases of chronic glanders.

Diagnosis. Acute glanders is distinguished from rheumatism by the

history of the case, the occupation of the patient, the existence of an open, irritable sore, and the fact that while the joints may be painful, they are rarely red and swollen as in rheumatism. Subsequently the appearance of pustules, bullæ, and ozæna makes the case clear. The same peculiar features serve to distinguish the disease from pyæmia, malignant pustule, and other infectious diseases. In a suspected case of chronic glanders a correct diagnosis might be arrived at by inoculating a mule or a horse with the nasal mucus or pus from a farcy.

Bacteriological Diagnosis. The specific germ is *Bacillus mallei*. This is a short, non-motile micro-organism resembling the tubercle bacillus. It is 2 μ to 3 μ long, and 0.3 μ to 0.4 μ broad, frequently having spores on the ends. It stains readily, although irregularly, with all the basic aniline dyes.

STRAUSS TEST. The examination for the bacillus is readily made by the method of Strauss. A portion of the suspected tissue or a culture from the lesions is inoculated into the peritoneal cavity of a male guinea-pig. If the case is one of glanders, the testicles begin to swell in about thirty hours, and an orchitis with abscess develops. The diagnostic sign is the tumefaction of the testicles.

MALLEIN TEST. Mallein is the filtered product of the growth of the bacillus on fluid media. It is allied to tuberculin. Injection in a suspected case produces a reaction similar in its course to the tuberculin reaction if the case is one of glanders.

Cholera.

An acute, specific, infectious disease, endemic in parts of India, but occurring in epidemics elsewhere, characterized by the outpouring into the stomach and bowels of large quantities of a serous fluid resembling rice-water, which fluid is usually vomited and discharged from the intestines. It is further characterized by an algid state of collapse and by painful muscular cramps.

The specific poison of cholera is believed to be the *comma bacillus* of Koch and its ptomaïn.

The native habitat of cholera is India, particularly the neighborhood of Calcutta; here it is endemic, and thence it is liable to spread in successive epidemic waves along the lines of travel by sea and land over the whole world. It is scarcely, if at all, contagious; the poison is contained in the vomitus and in the dejections, which contaminate the drinking-water, food, and clothing. The cholera bacillus preserves its vitality for long periods of time in water, especially if the water is slightly alkaline and contains vegetable matter, and in moist clothing, as rags.

The period of *incubation* is probably short in the majority of cases, lasting only a few days. Occasionally it is two weeks. There are usually no definite symptoms during this time, but there may be a sense of weakness, with loss of appetite and dyspeptic symptoms.

First Stage. The first stage, that of premonitory diarrhœa, is better regarded as the beginning of true cholera. It is characterized by profuse watery stools of a yellow or light-yellow color and alkaline reaction.

They are accompanied by a rumbling noise in the bowels, but are passed without pain. From six to a dozen of these passages occur in twenty-four hours. The patient feels faint and exhausted after them, and may suffer from nausea, but vomiting is not usual. In severe cases there may be cramps in the calves of the legs. The voice is faint and husky, thirst intense, the tongue white and moist. The temperature is normal or slightly depressed. This stage may last from two days to a week, depending upon treatment. In some cases it is absent, and the second stage sets in abruptly.

Second Stage. This usually comes on during the night. The patient is seized with vomiting, which is at first bilious, but rapidly loses all color and becomes like rice-water. The stools likewise resemble water in which meal has been stirred, or in which rice has been soaked—a semitransparent fluid, with particles of epithelium resembling rice floating in it. This fluid seems to well up and regurgitate rather than to be vomited from the stomach, and to gush in quantities of a quart or two from the anus. Sometimes vomiting and diarrhœa occur at once. The patient has unquenchable thirst, and is tortured with painful cramps of the toes, legs, belly, and diaphragm. As the discharges continue the patient becomes more and more exhausted; the nose is pinched and twisted, the eyes sunken, the lips bluish, and the whole body may shrink beyond recognizable proportions. The skin is cold and moist, the breath icy, and the temperature under the tongue is sometimes as low as 78° F. In the vagina and rectum it may be normal or slightly above normal. The patient, however, often has a sensation of heat. The urine is very scanty, containing albumin and sugar, or it may be suppressed. The pulse is very small and feeble, its rate 100 to 120. The mind is clear, but the patient is listless, answering questions in an extremely faint voice and with manifest effort.

Third Stage. From this collapse and algid condition the patient may slowly emerge, the skin becoming less cold, the cramps less severe.

A return of the secretion of urine is a hopeful sign. The reaction, however, may simply introduce a low typhoid condition, with fever, dry brown tongue, subsultus, low muttering delirium, and coma. In some cases serum is poured out into the stomach and intestines and is retained there. The patient while walking may be seized with dizziness, faintness, extreme prostration, and early collapse. In other cases the patient is smitten with profuse vomiting and purging, dying algid and collapsed in a few hours, no reaction appearing. In favorable cases the vomiting ceases, the stools become less frequent, and are tinged with bile and have a fecal odor. The urine increases in volume, while the albumin diminishes. Convalescence is protracted. Anæmia, great debility, feeble digestion, and sometimes obstinate diarrhœa delay complete recovery. Relapses are frequent. In other cases reaction brings improvement in the gastro-intestinal symptoms, but uræmia develops, death following in convulsions or coma.

The most frequent *complications* and *sequelæ* are eruptions, chiefly erythematous, ulcerations, bed-sores, and parotitis; and between the tenth and fifteenth days of convalescence a painful tetanic spasm may be seen of the flexor muscles of the hands, forearms, legs, and feet. (Stillé.)

Diagnosis. The chief points in the diagnosis from other affections are the knowledge of exposure to cholera; the character of the vomitus and dejecta, and the bacteriological findings; the cyanosis; the rapid development of collapse, with cold skin, icy breath, torturing cramps, and greatly shrunken visage and body.

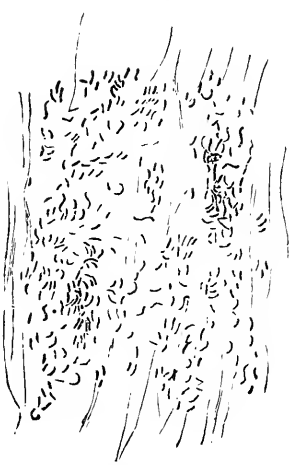
Cholera morbus differs in that the stools are turbid with bile or fecal matter, or contain blood; they never present the rice-water appearance. Moreover, the passages are frequently preceded by colicky pains. Cyanosis and collapse are extremely rare. The stools do not contain the cholera bacillus.

Other forms of acute *toxic gastro-enteritis*, whether from ptomaïn-poisoning or from corrosive poison, are to be distinguished by the history, the difference in the character of the stools, and the comparative mildness or absence of such symptoms as painful cramps in the legs, cyanosis, and collapse.

Bacteriological Diagnosis. Koch remarks:[1] "As cholera resembles in clinical symptoms cholera nostras, infantile cholera, certain forms of peritonitis, certain organic poisons, and poisoning by arsenic, it is important to attain some means of making a definite diagnosis." The bacteriological diagnosis is based upon the staining of smears, cultures, and inoculations.

Spirillum Choleræ Asiaticæ (*the Comma Bacillus*). The comma bacillus of Koch is the specific causative agent of cholera. In a disease so widespread in times of epidemics and so fatal, it is of great importance to be able to recognize the bacterium that produces it.

Fig. 320.



Cholera spirilla grown on moist linen. × 600. Cultivated from the dejections after two days. (After Koch.)

Microscopical Examination. The cholera bacillus is short, more or less bent, both shorter and thicker than the tubercle bacillus, and is generally shaped like a comma, but may form a half-circle, or two may be joined like an S. It has rounded ends, and is from 0.8 μ to 2 μ long and from 0.3 μ to 0.4 μ broad. The bacilli are often found placed end to end, and thus form a curve like a spiral. They are always present in the stools of cholera patients and sometimes in the vomitus. They are particularly abundant in the mucous floccules of the rice-water discharges, and can be obtained from the linen soiled by the same. Cover-slip preparations are made from these portions by placing a uniform film on the slip, drying it in the air, and then passing it through the flame of a Bunsen burner or spirit-lamp.

By Löffler's method a single flagellum is found on the rods. It stains with anilines, but slowly. An aqueous solution of fuchsin (Ziehl's red)

[1] Zeitschrift für Hygiene und Infectionskrankheiten, 1893, vol. xiv., No. 2.

is the best. In addition to the cholera bacilli, *Bacillus coli communis* and other intestinal bacteria are found. In thread-like strands of mucus the cholera bacilli form groups in which the bacilli all lie in the same direction. Koch holds that this mode of grouping is characteristic and diagnostic. He further holds that if *Bacilli coli* are in close proximity to numerous scattered bacteria resembling the cholera bacilli the case is one of Asiatic cholera.

The bacillus of cholera nostras and one found in cheese by Deneke resemble the comma bacillus in shape, though somewhat larger, but they have bacteriological peculiarities by which they can be differentiated.

Dysentery.

The fever which attends this infection is, from a clinical standpoint, the least characteristic symptom. It varies in part with the age of the patient. In the aged it is subnormal, normal, or moderate; in the young it is usually very high. It differs with the character of the infection; if a mixed infection prevails, the temperature is not unusual.

The term dysentery is applied to an inflammation of the intestinal tract, chiefly the colon, which is attended by the symptoms of intestinal catarrh in intense degree, with mucus and bloody discharges and the general symptoms of *fever* and prostration, followed by extreme exhaustion, and at times the occurrence of abscesses in the portal circulation, or of paralysis, arthritis, nephritis, or profound anæmia. It was formerly thought to be an epidemic, mildly contagious disease. Although of frequent occurrence sporadically, it is especially common in jails and institutions, in camps, or where people are crowded together, when at the same time hygienic conditions are most unfavorable. It usually occurs in the summer or fall, and is attributed to the drinking of impure water. A form most common in the tropics is called *tropical dysentery.* Recent investigations appear to show that catarrhal dysentery due to the above-mentioned circumstances, and "tropical" dysentery, which therefore is not necessarily confined to the tropics, are associated with inflammation and ulceration of the bowel, due to *Bacillus dysenteriæ*, the bacillus of Shiga, or *Amœba coli.*

Catarrhal Dysentery. Catarrhal dysentery may be limited to simple inflammation of the intestine or may be followed by ulceration. Its first symptoms are those of intestinal catarrh. There is indigestion, with loss of appetite, perhaps vomiting, and a slight diarrhœa. These symptoms may be the immediate effect of the inflammation. At the end of three or four days a chill may take place, showing the setting in of an infection. The diarrhœa is attended by pain, at first seated around the umbilicus; it then becomes marked in the course of the colon. The bowel movements are frequent, preceded by constant desire and attended by extreme tenesmus. The stools, which are first fecal and fluid, soon become scanty, and consist almost entirely of mucus and blood. The symptoms of local proctitis are severe; there is a sensation of a hot mass in the rectum. There may be strangury, and prolapse of the anus may ensue.

With the continuance of acute pain and frequent evacuations the skin becomes hot and dry; thirst, nausea, and occasionally vomiting occur. The temperature continues at about 103° F.; the pulse is rapid. The patient is weak and restless; the tongue is red and raw.

If the disease is severe from the start, or the course is unfavorable, the stools may contain pure blood, or they may be dark in color, and contain shreds of membrane. Pain and tenesmus disappear, and the evacuations become constant or involuntary. Restlessness is aggravated; the extremities become cold; mild delirium sets in. The tossing and restlessness are characteristic, and are attended by sighing and some dyspnœa. The pulse is rapid and feeble; the heart-sounds are weakened; the tongue becomes dry and brown, the mouth is parched, and thirst is intense; ulcers develop in the mouth and sordes collect around the teeth. The delirium increases to stupor, and from that to coma. The urine, at first high-colored and scanty, becomes bloody, and contains albumin and casts. Although the fever continues during this stage, the extremities become cool, perspiration breaks out over the forehead, and, instead of typhoid symptoms, the symptoms of collapse may ensue. If the disease is prolonged and the bowels are controlled, the symptoms of pyæmia may develop.

The anæmia that ensues is extreme, and there is great wasting. Convalescence is slow and may be attended by chronic diarrhœa. Before it is established, ulcers of the skin may form on various parts of the surface of the body. Arthritis is of common occurrence, and peripheral neuritis causing paralysis may occur during convalescence. Chronic dysentery may succeed the acute type. It is thus seen that the attacks may be of moderate severity or extremely grave; during the course of the latter, gangrene of the lower bowel may take place.

Diagnosis of Acute Dysentery. *H. D.* Exposure to cold and wet.
S. D. Rectal pain and distress.
O. D. No hepatic or vesical abscess.
P. D. Examine stools.
L. D. Serum diagnosis.

Tropical Dysentery. This form of dysentery may be due to *Bacillus dysenteriæ* or to *Amœba coli*, and may be acute or chronic. The symptoms of the acute form are like those of acute catarrhal dysentery. In the chronic form the patient wastes, the complexion is earthy, the abdomen scaphoid, and the temperature afebrile. The intestinal symptoms are like those of chronic inflammation of the bowels. Secondary abscesses do not occur so frequently as in amœbic dysentery. The blood-serum agglutinates a pure culture of the organism. The diagnosis of the nature of the dysenteric process is based on the agglutinating power.

Diagnosis. *H. D.* Climate, exposure, epidemic.
S. D. Abdominal pain, no chills, rectal tenesmus.
O. D. Facies, anæmia, arthritis.
P. D. Negative.
L. D. Serum diagnosis.
D. D. Enteritis.

Amœbic Dysentery. This differs from catarrhal dysentery in many

respects. The onset may be abrupt or gradual, as in the previous form, with symptoms of intestinal catarrh. In most of the cases a frequent and painless diarrhœa follows a period of slight ill health. The diarrhœa alternates with short periods of constipation; the stools are watery and contain mucus, but no blood. The course of the disease is irregular. There may be intermissions and exacerbations of the diarrhœa without obvious cause. Amœbic dysentery may rapidly pass from one grade to another, or become chronic. One form is the gangrenous, which may scarcely be suspected from the symptoms until autopsy shows it to have been present. True relapses are common, and the tendency to chronicity is great.

The *milder* cases are attended by weakness, emaciation, and pallor; the expression is dull; the skin is dry and sallow; the tongue pale, flabby, moist, and slightly furred; the abdomen is normal or retracted; the temperature does not rise above 100° F., and the pulse-rate ranges from 70 to 90 per minute. Sleep is disturbed by frequent evacuations of the bowels.

In the *grave form* the face is drawn, or cyanosed, or flushed; the expression anxious; the mind is clear. Anorexia, intense thirst, and sleeplessness are present. The abdomen is greatly retracted, and there may be free sweating. The temperature is normal or subnormal, the pulse small and rapid. Progressive anæmia and loss of flesh are prominent and dominate the intestinal symptoms. The skin is dry and harsh, and of a dull greenish-yellow color if the cases are protracted.

The special features of amœbic dysentery are:

1. **The Anæmia.** This is due to diminution of the red cells and the hæmoglobin, first, because of the action of the amœbæ upon the red blood-corpuscles, which they destroy; second, the direct loss of blood; and, third, malnutrition. The first is the most prominent.

2. **Diarrhœa** may be the only feature of the disease. It is characterized by great variation in character and frequency in all grades and during different periods of the disease. Intermissions and exacerbations may be observed at any time. The latter begin suddenly, and subside in the same manner. They last from two to ten days. The intermissions continue from one day to three weeks, during which the feces are soft, but contain mucus. Councilman and Lafleur have observed this periodicity to be most marked in cases complicated with hepatic abscess.

3. **The Stools.** The reaction is generally alkaline. The stools are extremely variable according to the severity of the ulceration, and also vary in number and character from day to day in individual cases. In the gangrenous form they number thirty or forty in twenty-four hours at first, then decrease, so that toward the end of fatal cases but three or four take place. At first the movements are small, and consist of mucus with more or less bright blood and small fecal masses. As ulceration advances, the stools change, they become more copious and watery, feces are absent, and blood is not so frequent. Shreddy masses of grayish or yellow color, mixed with mucus, appear. If there is sloughing, they become greenish or grayish, resembling spinach, or reddish brown and very liquid or pultaceous. The odor is penetrating and offensive. Shreddy masses of necrotic

tissue are discharged. Gray liquid movements, somewhat slimy, contain more pus than the others. Small opaque, or translucent, gelatinous grayish masses, 1 to 3 c.mm. in diameter, are found in the stools.

In the more moderate types if the attack is abrupt, the stools at the outset are like those of gangrenous dysentery; if gradual, the stools are fecal, liquid, containing mucus and streaks of blood and many gelatinous grayish masses. Stools of this character number from four to ten in twenty-four hours; and may continue thus for weeks. During the exacerbations the stools resemble those of the second period of the gangrenous form. In chronic dysentery there is not so much mucus or blood, except in exacerbations. The stools are of the consistence of thin gruel and have an earthy or dull-yellow color. Mucus is persistently present, however, in the intermissions, when the stools are soft and fecal.

Microscopical Examination. In the mucoid and bloody stools of the acute stage red blood-corpuscles, leucocytes, and large, round, or oval epithelioid cells are seen. The latter are sharply outlined and occur often in groups of three or more; the nucleus is about the size of a red blood-corpuscle, the protoplasm granular. They may be mistaken for amœbæ; but are non-motile and refract light less strongly. *Cercomonas intestinalis* is present, but bacteria are not abundant. In the later periods the cell-elements are less numerous; shreddy and muscular detritus and bacteria are observed, with elastic tissue-fibres. Charcot's crystals and phosphates are seen. In chronic dysentery the cell-elements are still fewer and amœbæ are easily detected.

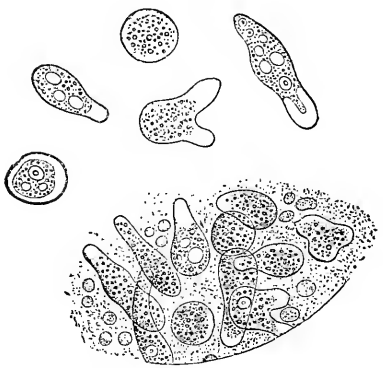
Amœba Dysenteriæ. Amœbæ are found at all periods of the disease. They vary in different cases and at different periods in proportion to the severity of the intestinal ulceration. (See section on the Feces.) They are most abundant in the grayish-yellow gelatinous masses, next in the particles of clear or opaque mucus, and least in the fluid portions of the stools. In chronic dysentery they are found in all portions. In the intermission of the diarrhœa they may be found in the particles of mucus adherent to the feces. They disappear as recovery proceeds, although they may be seen after the evacuations have become normal. They vary in size and activity, and are most common in the alkaline and neutral stools; in acid stools they are scarce and rarely motile. In the more active forms of the disease red corpuscles are seen in the stools.

For the *detection of amœbæ* the following rules should be observed: First, the stools should be passed into a warm bed-pan and kept at a temperature of 30° to 35° C. until an examination is made. Better still, a soft warm catheter should be passed up the rectum. Mucus may then be withdrawn through the eye of the instrument. Second, the stools must be examined before they become acid. Third, the gelatinous masses in the stools should be selected for examination, for they contain the amœbæ in greatest abundance. A magnifying power of 400 diameters is required, although they may be seen with less. A $\frac{1}{12}$ oil-immersion lens is the best.

Histologic Appearance. When *inactive* the amœbæ are round or slightly oblong, highly refractive, and contain clear vacuoles which vary from small points to one-third of the diameter of the amœbæ. The ectosarc

and endosarc may or may not be sharply divided. If they are, the outer is hyaline or homogeneous, the inner is more refractive and contains vacuoles. They are difficult to recognize in this condition, being easily mistaken for swollen connective-tissue cells. The amœbæ frequently enclose red corpuscles, pus-cells, blood-pigment, bacilli, and micrococci.

Fig. 321.



Amœbæ coli. (Hallopeau.)

In a fresh state the nuclei can not be made out because they resemble vacuoles. The endosarc is not granular, is composed of a dense substance, and is highly refracting. When *active*, the movement is characteristic. It may be slow or rapid, and is of two kinds, a progressive movement and one limited to the throwing out of pseudopodia. The movements appear to be rhythmical in some cases, occurring at regular intervals. The movement is sudden and characterized by change in form of the pseudopodia. The ectosarc and endosarc are usually clearly defined. The pseudopodia are hyaline and homogeneous, like the ectosarc. The amœba changes its position sometimes by enlargement of the pseudopodia, into which the inner contents of the older part follow. The movements are increased when the examinations are made on the warm stage. These amœba may be stained with various aniline dyes.

In *catarrhal dysentery* the stools are uniform in character, quantity, and frequency. The onset is sudden, and evacuations consist of bright blood and viscid, clear mucus mixed with fecal matter. Soon they are composed entirely of viscid mucus and a little blood. In a week or ten days the mucus changes and becomes grayish white in color—is less blood-stained and brown ; pultaceous or fluid fecal matter appears in the stools. As the blood and mucus disappear, formed feces return. In protracted cases there are soft, yellowish-brown or greenish stools in addition to the bloody mucoid stools. The frequency is greatest at the onset, and progressively diminishes until convalescence is established. The more frequent the evacuations the smaller the size of the stools. The mucoid stools are small, pultaceous, more bulky. On microscopical examination red and white corpuscles, cylindrical, epithelial, and oval epithelioid cells are seen. The latter are very characteristic, and occur singly or in groups.

Bacteria are more common as improvement sets in. In the pultaceous stools the cell-elements are scarce.

In *diphtheritic dysentery* the stools are watery. They resemble wheat-washings—evacuations such as are described in cases of *gangrenous dysentery*. They are grayish green or reddish brown and very offensive. Mucus is present in small amounts. At first they contain unclotted blood; later, minute dark-red clots. Shreddy and finely divided material, gray or reddish brown in color, is present, but there are no sloughs. The stools are not numerous at first, and average from seven to fifteen daily during the course of the illness; the quantity is small. Cylindrical epithelial cells are most abundant on microscopical examination. Red blood-corpuscles and leucocytes are observed, but fibrin constitutes the larger portion of the stool. In all the stools bacteria are present in great numbers.

Other Symptoms of Amœbic Dysentery. Abdominal *pain* is constant; it occurs in the early stages of both forms and in acute exacerbations. As the movements diminish, the pain decreases. In the gangrenous form pain also disappears, although the intensity of the process is increasing. In chronic cases the colic is complained of during the exacerbations; during the intervals there is a dull, aching or burning pain in the upper quadrants. In all cases the pain is cramp-like, boring or burning in character, and usually precedes and accompanies movements of the bowels. When severe it is general; but it is usually localized in the lower abdominal zone. Moderate tenderness on pressure is present in most cases along some part of the course of the large bowel. In catarrhal dysentery tenesmus is common; in the amœbic form it is infrequent. A burning sensation is generally present in the rectum and at the anus during and after the passage of feces. Nausea and vomiting occur at the outset or at irregular intervals, being caused by improper food, or due to complications. Hiccough occurs in the terminal stages.

Fever In amœbic dysentery, fever is not a prominent feature, although there is usually a moderate rise in temperature. In the gangrenous form it is normal, or may be subnormal for days. Chronic dysentery is afebrile. In exacerbations of diarrhœa slight fever may occur. Complications cause a higher temperature. If fever is present, it may be remittent or intermittent in character; or if the illness is prolonged, first continuous, then remittent, and then intermittent. If the latter, the usual morning fall is observed, although an inverse temperature may be present. Rigors occur with the complications. In the gangrenous form, sweating is observed, with subnormal temperature. In cases of abscess the fever is intermittent or remittent.

In *chronic dysentery* the skin is excessively dry. The circulation and respiration are influenced by the pyrexia. Anæmia is pronounced. When exhaustion ensues, the pulse becomes more feeble, compressible, and rapid. The urine is albuminous, and often contains casts. In the gangrenous form there may be retention of urine.

Complications. The complications of amœbic dysentery are: 1. Hepatic abscess or hepatopulmonary abscess. 2. Peritonitis. 3. Hemorrhage from the bowels.

Hepatic Abscess. This complication may develop at any period of the disease. The time of the disease when it occurs can not be determined definitely. In the subacute cases it is liable to develop from the fourth to the twelfth week. The abscess may develop on the convex surface of the right lobe of the liver near the coronary ligament. The lung also becomes involved. Councilman and Lafleur suggest that infection takes place by the peritoneum. (See Abscess of the Liver.) The symptoms of abscess of the liver will be treated under the section devoted to liver disease, but it is important to note that hepatic symptoms may occur in cases in which, on account of the mildness of the disease, the local bowel trouble may be overlooked.[1] If the association of hepatic pain with fever and discharge of mucus from the bowels is observed, it is barely possible, even if an examination of the feces can not be made, that an hepatic abscess is present. If, in addition, cough and expectoration occur, involvement of the lungs is possible.

Hepatopulmonary Abscess. The character of the expectoration points conclusively to the nature of the lung complication. After a period of dry, hacking cough, sudden expectoration of mucopurulent or bloody sputum takes place. It is of a dirty-red or brownish color, not unlike anchovy sauce. From this time on, this material is expectorated in varying quantities after a paroxysm of coughing. The expectoration is diffluent, tenacious, and frothy. It varies in color from bright red to russet-brown; it may be bile-stained; the reaction is alkaline; the odor is not putrid. At a later period it becomes more purulent, and contains less blood. The sputum separates into three layers: an upper frothy layer, a middle layer of turbid fluid, a thin layer of mucopus below. Large amounts may be coughed up in twenty-four hours. The sputa contain on examination blood-corpuscles, leucocytes, round alveolar epithelial cells, and polyhedral, fatty, degenerated cells which look like liver-cells. Elastic tissue fibres from the lungs are found with crystals of hæmatoidin and tyrosin, and Charcot's crystals. Bacteria are present. Amœbæ are constantly present. They vary in size and activity, but are larger than those seen in the stools. The sputum should be kept warm and examined as soon as possible. Abscesses form in other situations, as the jaw. (Flexner.)

Peritonitis. Peritonitis from perforation is not a common complication of amœbic dysentery, but takes place occasionally in the gangrenous form. Peritonitis without perforation may occur. The symptoms do not differ from peritonitis under other circumstances.

Hemorrhage from the bowel occurs and may be sufficiently profuse to cause death. This accident may occur in the course of amœbic abscess of the liver, as in a case reported by the author, in which there were no intestinal symptoms. Other complications which have been described under catarrhal and croupous dysentery are likely to occur in this affection.

Diagnosis. *H. D.* Hygienic surroundings; climate; temperature; food; country; clinical course.

S. D Enteritis; toxæmia.

[1] See "Amœbic Abscess of Liver." Musser and Willard, Trans. Phila. Co. Med. Soc.

O. D. Negative.
P. D. Liver abscess; pubic fistula; amœba.
L. D. Feces; sputum; pus from abscess.
D. D. Malaria.

The diagnosis of amœbic dysentery is rendered absolute by finding the amœbæ in the stools. The history and the course of the illness must also be taken into consideration, the characteristics of which have been previously detailed. The irregularity and the intermittency of the diarrhœa, the infrequency of tenesmus, the moderate fever, the reaction of the stools and their comparative freedom from bacteria are further corroborative points.

The Plague.

Plague is an acute, specific, infectious and contagious disease, occurring in epidemics, characterized by high fever, sometimes by petechiæ and other hemorrhages, and, in cases which last long enough, by buboes. The death-rate is extremely high. It is a disease of the East, being endemic in some parts of India; but epidemics have occurred in Italy, Russia, China, Turkey, England, and other parts of Europe.

Incubation. The period of incubation is from two to seven days. The invasion is marked by lassitude, languor, headache, and dizziness. The stupid aspect and staggering gait may lead to the belief that the patient is drunk. Chill or chilliness soon supervenes, followed by fever, which often rises to hyperpyrexia, and is accompanied by unquenchable thirst, and sometimes nausea and vomiting. Delirium and a typhoid condition follow, with a marked tendency to failure of the circulation and collapse. If the patient survive until the second or third day, glandular swellings develop in the groin or axilla, or at the angle of the jaw. Often they have to be sought for to be found. Sometimes they are prominent, and are followed by suppuration and even ulceration. Carbuncles are much rarer manifestations than buboes. Petechiæ, vibices, hemorrhages into the kidney, and bloody vomit occur in the worst cases.

The clinical divisions are those of *pestis minor*, in which fever, some glandular swelling and possibly suppuration occur; and *pestis major*, in the course of which buboes, septicæmia, the typhoid state, pneumonia, and other local infections occur. The cases are often ambulant, but the urine and feces contain bacilli.

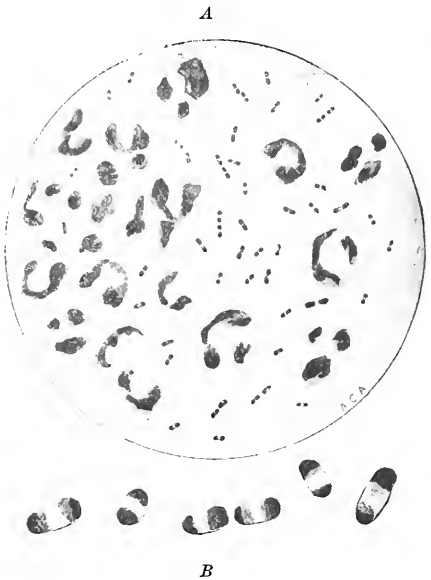
Diagnosis. *H. D.* Exposure; climate; epidemic.
S. D. Clinical course. Symptoms of toxæmia.
O. D. Buboes; carbuncles; hemorrhages.
P. D. Pneumonia if present.
L. D. Organism in pus; sputum; blood. Serum diagnosis.
D. D. Typhoid fever; other infections.

The diagnosis is based upon the history, the clinical course, and the results of bacteriological examination. The following description from Abbott enables the diagnosis to be readily made:

"This organism is described as a short, oval bacillus, usually seen single, sometimes joined end to end in pairs or threes, less commonly as

longer threads. It stains more readily at its ends than at its centre. It is sometimes capsulated; is non-spore-forming; is aërobic, and is non-motile. It is found in large numbers in the suppurating glands, in the sputum, and in much smaller numbers in the blood. (See Fig. 322.)

FIG. 322.



Bacillus of bubonic plague: *A*, in pus from suppurating bubo; *B*, the bacilli very much enlarged, to show peculiar polar staining. (ABBOTT.)

"It is demonstrable in cover-slip preparations made from the pus and in sections of the glands by the ordinary staining methods. Yersin states that it retains its color when treated by the method of Gram, while Kitasato says that it at one time stains by this method and at another it becomes decolorized. Aoyama observed that those bacilli within the suppurating glands were decolorized, while those in the blood retained the stain when treated by Gram's method."

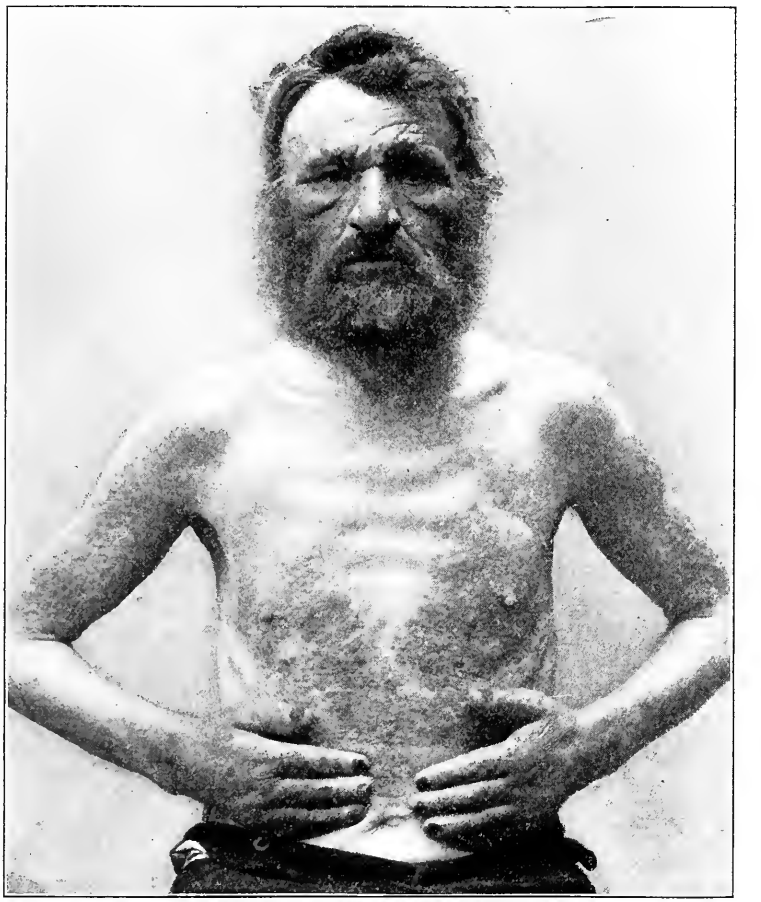
The duration is from six to ten days. If there is much suppuration, convalescence is prolonged.

Leprosy.

A chronic, specific, infectious disease, characterized by the development of tubercles, anæsthetic patches, and neuritis, and followed by ulceration and destruction of tissue. The disease occurs especially from puberty to the thirtieth year, and oftener in men than in women. It develops slowly and insidiously. Sometimes the first skin-lesion is a crop of bullæ, suggestive of pemphigus. More commonly there appear reddish or violet-colored patches, varying in size from $\frac{1}{4}$ inch to 2 or 3 inches in diameter, and becoming of a darker hue later. The next step

is the formation of nodules, which are characteristic of the disease. These may develop upon the patches already described, or in other places. They vary in size from that of a pea to that of a bird's egg or larger. They are most common upon the face and extensor surfaces of the arms, legs, fingers, and toes. The tubercles consist of an infiltration into the true skin; they are raised, firm, relatively painless, and vary in color from red to copper. The face is characteristically distorted into a fierce expression (leontiasis). The tubercles may become absorbed and leave

FIG. 323.



Leprosy.

atrophic areas, but generally they break down into eroding ulcers, which slowly burrow and increase in extent, eating off a portion of the nose, fingers, hands, and feet, and exposing muscles, tendons, nerves, blood-vessels, and bone. Tubercles form also upon nerve-trunks, and ulcers upon the mucous membranes. (See the Nose and Larynx.)

In other cases, or in combination with the tubercles, especially upon the limbs and trunk, there are *anæsthetic* areas. Ulcers may follow with-

out the previous occurrence of tubercles. With the anæsthetic patches are associated crops of *bullæ* and *neuritis*.

The further peculiarities of the disease are: its long duration, its slow progress interrupted by apparent healing of some of the ulcers; its afebrile course (the temperature is generally subnormal); its comparative painlessness, and the slight impairment of the general health.

Death results from gradual wasting, or is hastened by some intercurrent affection.

Diagnosis. *H. D.* Exposure; residence.

S. D. Painless lesions.

O. D. Skin-lesions, nodules; ulceration; anæsthesia; facial expression; afebrile.

P. D. Negative.

L. D. Bacilli in lesions.

D. D. Syphilis; hysteria.

The specific cause of the disease is *Bacillus lepræ* of Hansen. It is found in the thin pus of the ulcers and in the lesions. It consists of rods 4 μ to 6 μ long and 1 μ broad, closely resembling the tubercle bacillus, and stains in alkaline fluids, but does not bleach after exposure to acids. Staining cover-slip preparations with the Ziehl-Neelsen fluid and decolorizing in acid and alcohol bring the bacilli out quite distinctly. They may be distinguished by the fact that they yield their color more readily, and easily take aniline dyes in simple watery solution. (Von Jaksch.)

The *diagnosis* from a tubercular *syphilide* is made by the history of the case, the possibility of infection, the bacteriological examination, the slow progress, and the inadequacy of specific treatment. Anæsthesia and neuritis point to leprosy, although their occurrence in young neurotic subjects also suggests hysteria.

Actinomycosis.

The general symptoms attending this infection are like those of suppurative infections. The *fever* is irregular, often intermittent. It is a specific, infectious disease of cattle, occurring occasionally in man, attacking especially the lower jaw, lungs, and intestines, and characterized by a long duration, by the development of tumors and metastatic growths, and by *pyæmic* symptoms.

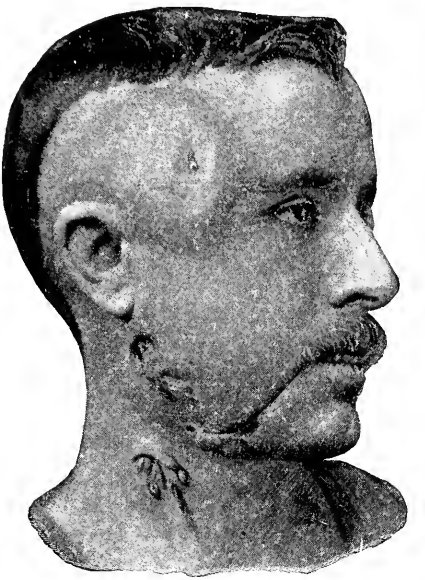
Actinomycosis is due to actinomyces, or ray-fungus (see Fig. 325), which produces in cattle the disease known as big or lumpy jaw and swelled head. The fungus is conveyed in the food or drink, and gains entrance to the body through abrasions in the mouth or a decayed tooth, or is inspired into the lungs. Israel, Ponfick, and Boström have given us the greatest amount of information in regard to this parasite. It was discovered in 1845, in human beings, by B. von Langenbeck, and in 1877, in cattle, by Bollinger.

At the seat of invasion a slowly growing, slightly painful tumor develops. Bones are affected as well as soft tissues. These become swollen and suppurate, the fungus being at all times obtainable. The fungous

masses appear to the unaided eye as particles of yellow sand, and are greasy to the touch.

Pulmonic Form. Actinomycosis of the lung may be divided into three stages; a latent stage, when the lung proper is affected; an active stage, when extension to the pleura and chest-wall takes place; and a final or chronic stage, when perforation and the formation of a thoracic fistula occur and the adjoining organs become affected.

FIG. 324.



Case of actinomycosis.

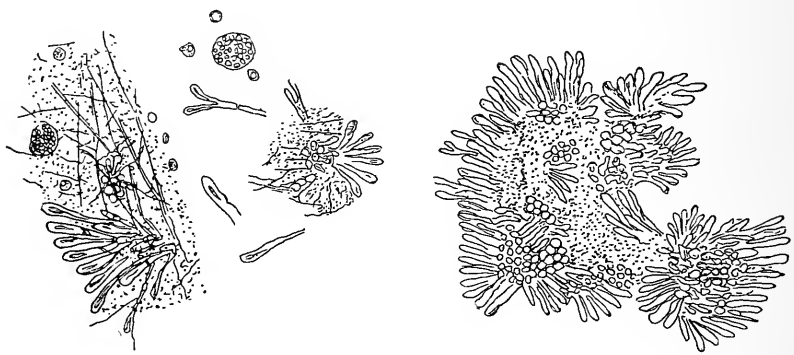
The symptoms of the *first stage* are those of chronic bronchial catarrh, with later the occurrence of the physical signs of consolidation, especially in the mammillary and axillary regions of the chest, in the middle zone of the thorax. The apices and bases are rarely affected primarily.

The symptoms of the *second stage* are those of pleurisy, with adhesions and with or without effusion. At this time the disease may extend downward to the liver and peritoneum, or the pericardium may become infected. Fever and pain accompany these processes. On physical examination, in addition to the signs of the pulmonary and pleural conditions above mentioned, swelling of the thoracic wall will be observed, not unlike that due to an empyema which is about to perforate. The swelling, which is at first dense, and hard, and red, becomes softer in small areas, and may fluctuate. Fluid, which is mucopurulent and contains the parasite, may be removed by aspiration. Repeated taps may fail before the needle finds the serous or sanguinoserous exudation in the pleura. The sputum at this time may accidentally contain the parasite, although this is rare; it is mucopurulent, but it is said never to contain elastic fibres. The course of the disease at this time may extend over many months, in contradistinction to empyema on the one hand and carcinoma on the other.

In the *final stage* ulceration of the swelling is seen in many places, a fistula forms, and the disease extends to adjacent structures.

Metastasis. Secondary infection may occur and symptoms of pyæmia develop. The masses which form upon the intestinal mucous membrane may lead to suppuration and perforation of the intestine. Metastasis to any organ may occur, with resulting local symptoms. The duration depends upon the organs involved in metastases. If metastases do not lead to early death, that result is brought about at the end of months or years by slow pyæmia, with resulting amyloid degeneration and its consequences.

FIG. 325.

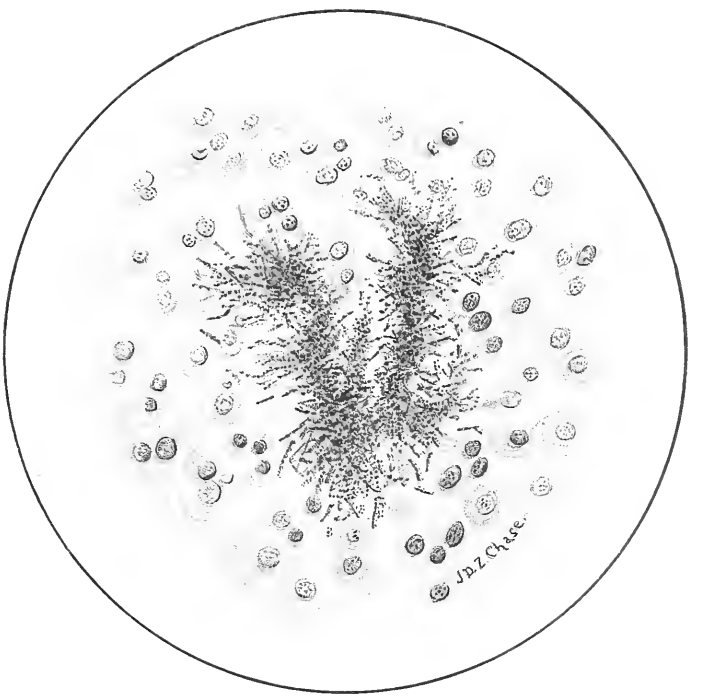


Actinomyces.

Character of the Pus. Actinomycosis is usually associated with chronic inflammation and the production of pus. The pus is peculiar. It is thin and viscid. Nodules of gray or yellow color, the size of a poppy-seed, can be seen in it with the naked eye when it is spread on a slide. With a low power these particles appear as aggregations of spherules, which with a higher power are seen to be arranged in masses radiating from a common centre. Each separate spherule is pear-shaped. They have high refractive power. The centre of the masses is occupied by a network of fibres. If the mass be broken up, numerous club-shaped forms are seen at the periphery, while at the centre a sort of detritus alone is observed. The micro-organism belongs to the class of fission-fungi, and the club-shaped bodies are the degenerated forms. (See Fig. 325.)

Gram's method of staining brings out the threads of the network most distinctly. The centre is made up of a network of minute spherical organisms, with converging, constituent threads. The whole is surrounded by a delicate envelope. The pear-shaped bodies may be defined by Weigert's process. Make a solution of 20 c.c. of absolute alcohol, 5 c.c. of concentrated acetic acid, 40 c.c. of distilled water, and sufficient French extract of litmus to color it ruby-red after repeated filtering. In this solution the cover-glass preparations are allowed to remain for an hour, and then rinsed with alcohol rapidly and placed in a 2 per cent. gentian-violet solution for three minutes. The fluid should be boiled

PLATE XVIII-a.



Section through wall of brain abscess showing character of streptothrix colony. Stained with hæmatoxylin, followed by modified Gram-Weigert method, 1/12 in. homog. immersion objective.

before use and filtered after cooling. The fungous threads are stained a ruby-red, while the central mass of actinomyces is colorless.

Diagnosis. *H. D.* Diseased teeth or mouth, exposure.

S. D. Painful indolent swelling; symptoms of empyema or bronchitis.

O. D. Swelling about jaw slowly suppurates; bone disease; metastases; pyæmia; enteritis.

P. D. Some cases; empyema; bronchopneumonia.

L. D. Pus; smears; sputum; exploratory puncture; ray fungus.

D. D. Suppurations; pulmonic affections; enteritis.

Simple *microscopical examination* is usually sufficient to determine the nature of the fungus. The recognition is more positive if we bear in mind the peculiar character of the pus in which the nodules and the club-shaped forms are seen. It must not be mistaken for the radiating leptothrix threads found in the mouth. Pure cultures have been obtained resembling macroscopically those of tubercle bacillus.

Streptothrical Infections.

The pathogenicity of streptothrices and their relation to infections and lesions described are very evident, though cultural and animal experiments are lacking in some of the earlier observations. It is clear that actinomycosis and Madura foot can no longer occupy their isolated positions as examples of streptothrical infections. The predilection of these organisms for certain regions or systems of the body is very apparent, the *lungs*, the *brain*, and the *skin* being most frequently affected; the brain, however, is more often invaded by metastasis in cases of primary pulmonary or bronchial gland infections.

The *pulmonary lesions* in which the streptothrices have been found either alone or with other organisms show considerable variety: bronchopneumonia, extensive consolidation, abscess, bronchiectasis, empyema, and necrotic bronchitis.

The lesions in the *nervous system*, practically always metastatic infections from pulmonary or bronchial gland disease, have been abscess, meningitis, or large areas of softening from thrombosis.

An *abscess* of the *kidney* was noted in one instance. Naunyn found in his case an *endocardial excrescence* containing a streptothrix.

From a clinical standpoint but little that is distinctly new is presented in streptothrical infections. The cutaneous lesions of Madura foot, the cases of Rosenbach, Ferri and Faguet, Scheele and Petruschky, the cutaneous expression of actinomycosis, the actinomycotic-like skin infection of Fullerton, show distinct differences. The pulmonary infections with their secondary accidents are all more or less hidden under the mask of a tuberculous process, herewith termed *pseudotuberculosis*, or resemble some one or other of the number of pulmonary lesions, such as *abscess*, *gangrene*, or *bronchiectasis*, but the detection of the streptothrix and its mycelial masses is as diagnostic as the demonstrations of tubercle bacilli. Not all instances reported have been primary infections. In many the streptothrix may have been a very late invader. Many instances of streptothrical infections have probably passed unrecognized,

owing to the peculiar tinctorial and cultural reactions of the micro-organism. Rullman, Ferri and Faguet, Aoyama, Scheele and Petruschky were able to determine the nature of the disease in their cases by the findings in the sputum, as was Gwyn in the case I have reported.

Tetanus.

Tetanus is an acute infectious disease of the nervous system, the essential characteristic of which is persistent tonic spasm of the muscles of the jaws (*lockjaw*) and of the spinal and thoracic muscles.

The disease begins with stiffness of the jaws, which steadily increases until within a few hours there is complete tonic spasm. The neck muscles, and then those of the spine and trunk, become rigid, so that the body is arched backward and may rest upon the heels and head (opisthotonos). The facial muscles share in the spasm, and by their contraction produce a horrid, grinning countenance (*risus sardonicus*). The contracted muscles become painful, and there is also epigastric pain. The rigidity is persistent, but is interrupted by exacerbations in which the phenomena already described are exaggerated, and, in addition, respiration is embarrassed, the face becomes livid, the skin bathed in sweat, and the patient is further distressed by increased pain in the affected muscles. The body may be bent forward (emprosthotonos) or laterally (pleurosthotonos). The temperature is not constant. It may remain normal, be moderately elevated, or hyperpyrexia may be present, especially toward and after the end in fatal cases. The spasm ceases during sleep, but subsequently returns.

FIG. 326.

A B

Tetanus bacillus: A, vegetative stage, from gelatin culture; B, spore-stage; showing pin-shape. (ABBOTT.)

Bacteriological Diagnosis. The cause of the disease is the bacillus of tetanus, which produces the convulsive poison *tetanin*. The bacillus is seen as a delicate, slender rod, with a terminal spore. It stains with aniline dyes and Gram's fluid. Cultures may be made with the pus.

Tetanus frequently follows an injury. *Trismus neonatorum* and *puerperal tetanus* are names given to special varieties which occur in new-born children and in puerperal women. Tetanus is much more common in men than in women, and Gowers states that three-fourths of the cases occur between the ages of ten and forty. It is much more common in hot than in cold countries, though cold is an exciting cause.

In traumatic and puerperal cases the disease usually develops in from

a few days to two weeks from the time of injury or childbirth or abortion. In new-born children it occurs usually during the first week. It lasts from two to six weeks, but may be fatal much earlier, or in rare cases last even longer.

Diagnosis. *H. D.* Trauma; infants; puerperium; climate; season.
S. D. Pain in muscles.
O. D. Rigidity; spasm (lockjaw).
P. D. Negative.
L. D. Smears; cultures; inoculation.
D. D. Strychnine-poisoning.

Tetanus must be distinguished from strychnine-poisoning. In the latter the jaw muscles are never involved early, if at all, and the muscles are relaxed between the paroxysms. It is distinguished from tetany by the history and the distribution of the spasm, which in tetany is confined to the extremities.

Trichinosis.

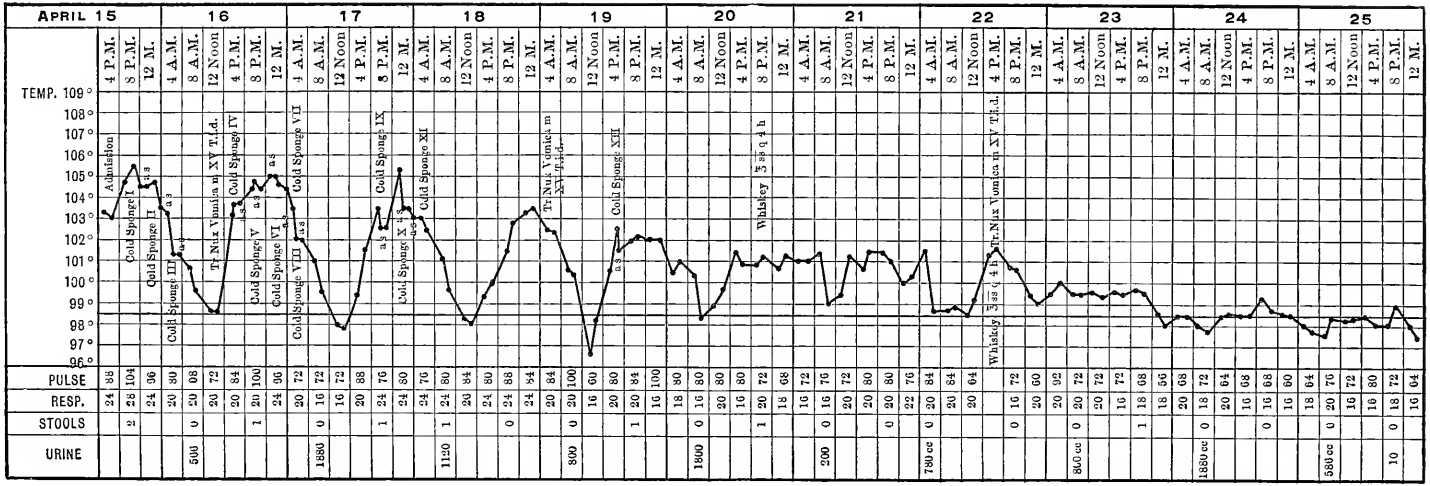
Definition. The infection is acute, caused by absorption of *Trichinæ spiralis*, and characterized by fever, gastric and intestinal irritation, followed by pain and stiffness in voluntary muscles, œdema of the eyelids, face, and feet, by profuse sweating, and by death or tardy convalescence.

The trichinæ are absorbed by human beings through raw or imperfectly cooked food, often in the form of sausage. The trichinæ are encysted when absorbed, but within forty-eight hours they are liberated in the intestine and can be found adherent to the mucous membrane. In the course of six or seven days each liberated female worm produces about 180 embryos, which immediately penetrate the walls of the intestine and travel or are carried to all parts of the body, becoming encysted in turn.

Clinical Course. Swallowing of trichinous flesh does not necessarily produce symptoms; the trichinæ may be destroyed in the stomach, or, if calcified, may pass through the intestine unchanged. When symptoms result, the severity depends upon the number of trichinæ that become liberated. The symptoms are sleeplessness, lassitude, anorexia, nausea, vomiting, tenderness over the abdomen, and diarrhœa. Headache is a constant and marked symptom of invasion. Colicky pains attend the gastro-intestinal symptoms. These symptoms may not be marked in the beginning of the disease; or they may be so severe as to cause death in two or three days. If the patient survive, toward the end of the week the voluntary muscles become stiff, painful, and contracted. The muscles feel hard and swollen. The eyelids, face, and sometimes the feet become œdematous. Depending upon the muscles involved, there are interferences with the eye movements, contractions of the jaw muscles, difficulty in breathing or in swallowing, etc. The calves of the legs are especially involved. Recurrent œdema over the affected muscles, eyelids, and face is very common and characteristic. Marked erythema of the limbs occurred in one case. Profuse sweating also is very common, and at times there are severe neuralgic pains. The later stages in fatal cases are marked by insomnia, delirium, stupor, and coma.

The *duration* varies from a few days to four or five weeks, or even

FIG. 327.



Intermittent fever in trichinosis. (OSLER.)

longer. Muscular pains may persist for months after recovery. Death results from exhaustion, or from some complication, as pneumonia or ulceration of the large intestine.

Fever. The fever is usually moderate, but it may be high. It is accompanied by malaise, with pains in the joints and muscles, preceding the true local muscle pain.

In some cases the temperature may be only moderately elevated for a few days, and then fall to normal, and even below normal, especially during convalescence. In other instances the temperature-curve may be markedly intermittent. The chart from Osler's monograph shows this peculiarity. (See Fig. 327.) Finally, the fever range is not unlike that of typhoid fever in many instances. Strümpell observes that the fever is seldom continuous for any length of time, and that its course is interrupted by frequent and prolonged intermissions. Niemeyer compares the curve to that of typhus, and Eichhorst to that of typhoid fever.

Pulse. The pulse is very frequent if trichinæ reach the heart.

The Blood. Brown, in studying Osler's cases, found an increase in the leucocytes, and on a differential count a great increase of the eosinophiles. The diagnosis of five of the six cases studied by Brown was suggested by the eosinophiles. The leucocytes were increased to 17,000 per c.mm. The eosinophiles increased from 2 per cent., the normal, to 37 per cent., and at one time to 68.2 per cent. In subsequent cases their average increase was as high as 48 per cent. Blumer, in the report of an epidemic, confirms the observation. Subsequent observations have confirmed these studies, so that *eosinophilia* is a recognized accompaniment of this parasitic invasion.

Diagnosis. *H. D.* Exposure to cause (food).

S. D. Pain in muscles.

O. D. Œdema, subcutaneous and muscular tubercles, rigidity, fever, sweating, gastro-enteritis.

L. D. Examination of muscle; leucocytosis, eosinophilia.

D. D. Differential from typhoid fever and muscular rheumatism.

The diagnosis is based upon the history, the peculiar *muscular pains* and *swellings*, the localization of the œdema, and the *leucocytosis* and *eosinophilia*. The muscles are swollen and hard, painful on pressure, and contracted. There is no involvement of the joints, an important point in the diagnosis. The œdema (see Chapter XXIV., Part I.) is seen in the eyelids and over the eyebrows. It is of common occurrence over the swollen and tender muscles.

Trichinosis is distinguished from *typhoid fever* by the presence of vomiting, and œdema of the face and eyelids, the development of muscular troubles, and by the absence of hebetude, delirium, and other typhoid symptoms, and absence of the characteristic eruption and of enlargement of the spleen. The Widal test, of course, is necessary.

Muscular rheumatism is distinguished by being limited to one part, as the lumbar region, arm, or chest; by its appearance following exposure to draught; and by the fact that it is not preceded by nausea, vomiting, and diarrhœa, nor accompanied by œdema.

CHAPTER II.

THE INTOXICATIONS.

FEBRILE INTOXICATIONS.

Sun-stroke.

SUN-STROKE (siriasis, thermic fever, insolation, heat-stroke), whether the cause be the direct action of heat upon the brain-centres, or the production of some toxic substance, is the most pronounced expression of *fever* apart from the infectious disorders. The flushed face, the pungent skin, the dyspnœa, and the rapid pulse forebode the high body temperature, which in the axilla may reach 108° to 112° F. in a very short time, and death takes place in coma from hyperpyrexia. If recovery takes place, the temperature may be moderate and continued for a few days. Nervous and cardiorespiratory phenomena are added to the picture. In some instances dyspnœa, heart-failure, and coma may follow rapidly, and death ensue in one or two hours. In other cases pain in the head, dizziness, and languor precede the stupor. Nausea and vomiting, perhaps diarrhœa, chest oppression, frequent micturition, and convulsions may precede insensibility. Unconsciousness is lost quickly or gradually, and it may be transient or pass into deep coma. Relaxation of the muscles with twitching is seen, and the pupils, at first dilated, become contracted. As the coma deepens, the heart's action becomes more rapid and feeble, the respirations hurried, shallow, and irregular, and death ensues, preceded or not by convulsions.

The diagnosis is based on the history, the mode of onset, and the hyperpyrexia. Thermic fever must be distinguished from uræmia and apoplexy.

Heat-exhaustion is readily recognized. The moist, pale, and cool skin, the soft, feeble pulse, the quiet but hurried breathing, are unattended by fever. The collapse, for such it is, is not attended by coma, and it usually responds to treatment.

Morphinism.

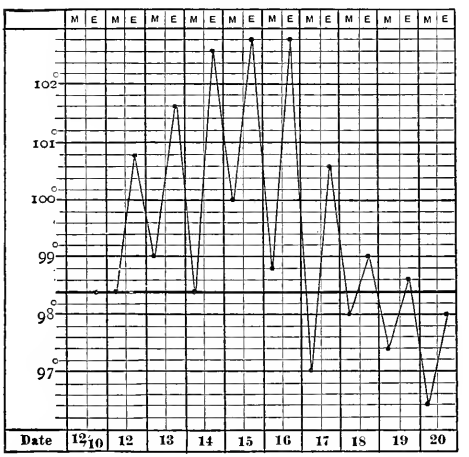
Lewin showed that morphinism is attended by fever. The fever may be continued or intermittent. When the latter, *chills* are of frequent occurrence. The diagnosis is based on the history, on the evidence of malnutrition without cause, on the general depression and lassitude, and upon the temperament of the patient, to which are added insomnia, restlessness, and itching of the skin. The peculiar sallowness of the complexion and the prematurely aged appearance are familiar. Pseu-

doneuralgic pains are common, tabetic symptoms may be present, and notably gastro-intestinal symptoms, as gastralgia, vomiting, diarrhœa (especially if the drug is withheld). Fever, it must be remembered, may be absent.

Simple Continued Fever.

A non-contagious fever, lasting from one to twelve days, not dependent upon any known specific cause, and not attended by definite local lesions. The chief feature is the continued elevation of temperature. It occurs especially in children and in those prone to ready disturbance of the heat-regulating apparatus. Great mental and physical exhaustion, prolonged bathing in the hot sun, and disturbances of digestion may cause it. Perhaps, as suggested by Guitéras, some of these cases occurring in the tropics and in very hot weather should be regarded as mild forms of thermic fever.

FIG. 328.



Simple continued fever.

The onset of the disease is abrupt. There may be a chill, or in nervous children a convulsion; but these are rare. The temperature rises rapidly from 102° to 104° F., accompanied by headache, thirst, restlessness or drowsiness, loss of appetite, a coated tongue, constipation, and occasionally nausea. The urine is scanty, and sometimes there is a heavy deposit of urates. There may also be more or less muscular soreness. Sometimes within twenty-four or forty-eight hours free perspiration takes place, with rapid subsidence of the fever. This is *ephemeral fever.*

In other cases the fever continues for a week or ten days longer. During this time the symptoms already noted continue. Sleep is disturbed and mild delirium is at times present. Respiration and pulse are not much accelerated. Sudamina upon the abdomen and herpes on the lips are common. Pale-bluish maculæ are sometimes seen. The spleen is not enlarged except in very rare cases, and there are no local evi-

dences of disease. The fever subsides more gradually than in ephemeral fever, the defervescence being marked at times by perspiration, a few loose stools, a copious deposit of urates in the urine, or by hemorrhages from the nose, rectum, uterus, or urethra.

The *diagnosis* from other fevers and febrile affections is made by the absence of any characteristic eruption, of enlargement of the spleen and liver, and of any lesion, such as endocarditis, bronchitis, or pneumonia.

FOOD INTOXICATIONS.

Among the intoxications which give rise to fever are those due to food-poisoning. Meat, milk-products, and shell-fish cause an intoxication of the system which often threatens life, and, from the suddenness of the attack and the severity of the symptoms, points to an infection rather than an intoxication.

The history of the case is often the first clue to its nature. The symptoms are those of acute gastro-intestinal irritation, to which are added, with or without afebrile periods, the symptoms of collapse.

Meat-poisoning.

In the intoxication arising from poisoning by meat, the temperature rises from 101° to 104° F., preceded usually by a brief period of chilliness. The occurrence of fever may be preceded by a period of incubation lasting from twelve to forty-eight hours. During the period of *incubation* there are malaise, loss of appetite, nausea, and colicky pains. As they increase chilliness ensues, and in some instances there is a marked rigor. Prostration occurs almost immediately, with giddiness and faintness, and the occurrence of cold perspiration. Headache and backache are liable to occur. Following the chilliness the symptoms of gastro-intestinal irritation arise, diarrhœa being more frequent than vomiting. The abdominal pain increases and the perspiration and clammy sweats become more pronounced. As further evidence of the intoxication there is an extreme degree of muscular weakness. The pulse becomes rapid, and later thready. In addition to muscular weakness, cramps in the legs and arms, followed by convulsive movements, occur, and the patient complains of paræsthesia of various forms. In milder cases the symptoms of gastro-intestinal irritation and of muscular weakness attend the fever. In the more severe cases fever is replaced by collapse.

Poisoning by Milk-products.

Gastro-intestinal and choleraic symptoms ensue. Some of the diarrhœas of infancy are types of this intoxication. The high degree of fever that occurs is well known. In cheese-poisoning the fever is not continuous as in the other forms, the temperature becoming subnormal with the onset of collapse.

Poisoning by Shell-fish.

In mussel-poisoning the symptoms are those of an acute mineral poisoning with profound nervous symptoms. Fever does not attend this condition, but collapse follows quickly. There are no gastro-intestinal symptoms.

Fish-poisoning is also unattended by fever, collapse occurring early.

AFEBRILE INTOXICATIONS.

Herein will not be considered those important *afebrile intoxications* due to disease of the *ductless glands*. The latter include some diseases of the suprarenal bodies (Addison's disease), the thyroid gland (exophthalmic goitre and myxœdema), the lymphatic glands (status lymphaticus), and the spleen, which will be considered in their respective chapters.

Alcoholism.

Acute Alcoholism. In this state the reeling gait, the incoherent speech, followed by narcosis, are generally familiar. The temperature is afebrile. Often, indeed, it is subnormal, and when equal on both sides of the body is very suggestive. The flushed face, possibly slightly dusky, and the injected eye, would lead us to suspect the presence of fever. The odor of the breath furnishes a clue. The heavy breathing, the full pulse, the dilated pupils, the stuporous rather than comatose state, are accompaniments of this intoxication. The flaccid limb of one side would point to hemiplegia from hemorrhage, especially if the coma is deeper than usual and the stupor more marked. But the possibility of *urœmia* and *apoplexy*, or both, occurring in a drunken subject must be borne in mind.

Chronic Alcoholism. When the poison is taken for a long time it acts as a tissue poison and a check upon waste. Epithelial and nerve degeneration and fibrous over-growth follow the first or poisonous irritative action; and fatty change the second. In the alcoholic, tremor of the hands and tongue is seen. The action of the muscles is unsteady. The mind is dull, the temper irritable, forgetfulness is most common, and later a dementia and epilepsy may ensue. Alcoholic neuritis, to be described later, is of frequent occurrence.

Gastro-intestinal catarrh, with poor appetite and constipation, is most liable to ensue, and later cirrhosis of the liver and kidneys; endarteritis, with its train of pathological processes, including myocarditis, and visceral scleroses also arise.

Grain-poisoning.

Three forms are seen. When the grain is contaminated by ergot, symptoms known as *ergotism* occur. Chronic ergotism may cause gangrene or a train of nervous symptoms in which convulsive movements are most prominent. In the gangrenous form the toes and fingers are the seat of mortification. The process is preceded by anæsthesia, paræsthesia, and pain. In the convulsive form there is slight fever with some weakness

and tingling sensations in the body. Cramps and contractures occur in the extremities, continuing for hours or days, and relapsing frequently. A mild delirium or the development of melancholia or dementia attends the convulsive form.

In other intoxications fever is not so pronounced. In *lathyrism* the symptoms are those of spastic paralysis, which may proceed to paraplegia. In *pellagra*, a disturbance due to maize, there are disorders of digestion, loss of sleep, general pain, and debility. The digestive symptoms are those of salivation, dyspepsia, and diarrhœa. A peculiar erythema arises. Subsequently, desiccation and desquamation of the epidermis occur, and often small boils develop. Headache, backache, spasms, and paralysis of the legs occur in the severe and chronic forms. The nervous symptoms may give way to melancholia.

Lead-poisoning.

Intoxication due to lead, or plumbism, may be acute or chronic. In the *acute* form we have symptoms of gastro-intestinal irritation with constipation and extreme colicky pains. Anæmia may develop rapidly, and pronounced nervous symptoms arise. Among the latter we have neuritis, convulsions, epilepsy, and delirium. Hemorrhages from mucous membranes may be seen, and a form of nephritis develops rapidly. The urine contains albumin and tube-casts. Fever is not a pronounced symptom.

The characteristic symptoms of *chronic* poisoning are:

a. Saturnine *cachexia*, in which anæmia is most pronounced.

b. *Colic.*

c. *Paralysis*, which may be acute, subacute, or chronic, and which usually develops without fever. The paralysis may be antibrachial, causing characteristic wrist-drop; brachial, in which the scapulohumeral form of paralysis is seen, or of the Aran-Duchenne type, resembling chronic anterior poliomyelitis. Another is the peroneal type, in which the lateral peroneal muscles, the extensor communis of the toes, and the extensor proprius of the big toe are paralyzed, causing the steppage gait. Finally, paralysis of the adductor muscles of the larynx occurs in lead-poisoning. The paralysis often extends from a local group of muscles throughout the body, presenting symptoms like those of an ascending paralysis with rapid wasting. In other instances the general paralysis occurs primarily, the wasting and loss of power going hand in hand. Fever sometimes attends a general paralysis in lead-poisoning.

d. The *cerebral* symptoms of the acute form have been mentioned. In the chronic cases they may also occur.

Optic neuritis, or neuroretinitis, is common. *Delirium*, with hallucination, may occur. *Tremor* is a common symptom. It must not be forgotten that headache, convulsions, epilepsy, and delirium may be manifestations of *lead encephalopathy* even in cases in which the history of exposure to lead is not direct.

e. Chronic lead-poisoning leads to *arterial sclerosis* and *contracted kidneys* with hypertrophy of the heart.

f. *Gout* is very common, and may be seen in both acute and chronic forms, particularly in the big toe.

g. As described in the section in which the mouth and gums are discussed, the *blue line* is the specific symptom of lead-poisoning. The reader is referred to that chapter for a description of the line. It must be remembered that in all forms of obscure nervous disease, in gastro-intestinal irritation, in arteriosclerosis, and gouty arthritis, this line must be looked for.

Arsenic-poisoning.

Acute arsenical poisoning is attended by symptoms of gastro-intestinal irritation followed by the rapid development of collapse. Fever is not a prominent symptom unless recovery is about to take place. The temperature is subnormal, but as the collapse symptoms disappear fever due to gastro-intestinal ulceration develops.

In *chronic* arsenical poisoning the fever occurs only if there is great irritation of the mucous membranes, as of the conjunctiva, mouth, or pharynx. In this form, in addition to the irritation of these mucous membranes, there may be subacute gastro-intestinal catarrh, with diarrhœa. In other instances there is profound anæmia and debility, with paræsthesia and neuralgia. In others, again, paralysis like that of lead-palsy may occur. It must not be forgotten that puffiness under the eyelids may be due to this cause.

CHAPTER III.

CONSTITUTIONAL DISEASES.

Gout.

A DISEASE characterized by specific arthritis, associated with uric acid in the blood and the deposit of sodium urate in the joints, or manifesting itself as a diathesis in which occur other inflammations of non-articular tissues and various disturbances of functions of organs, the blood also containing uric acid.

Gout is common in Europe, particularly in England, but in its articular form is rare in this country. There is an hereditary predisposition in from 50 to 60 per cent. of the cases. It results from over-eating of rich foods and the drinking of malt liquors, associated with insufficient exercise and excretion. Garrod has called attention to its association with lead-poisoning. Paroxysms are induced by indiscretions in eating or drinking, by nervous shock or great mental strain, by exposure to cold or injury, or by over-work and sexual excesses. The characteristic phenomena of gout are preceded for a variable time by acid flatulent dyspepsia, colicky pains in the stomach and bowel, constipation alternating with diarrhœa, and the excretion of scanty, heavily loaded urine. Accompanying these dyspeptic symptoms often are impairment of physical and mental vigor, irritability of temper, and hypochondriasis. In other cases the premonitory symptoms are palpitation of the heart, or dyspnœa resembling asthma, or various nervous symptoms, as drowsiness, insomnia, or headache.

Acute Articular Gout. In acute articular gout the onset is often sudden, especially in the first attack. The patient may go to bed in apparent health, but wake up early in the morning with a feeling of discomfort or uneasiness, usually in the great toe. In some cases the pain is agonizing from the first. The patient finds he is unable to put his foot to the ground without torturing pain. The ball of the great toe is hot, swollen, red, and exquisitely resentful of the slightest touch or jar of the bed. The veins are swollen and the joint stiff. There are slight fever, perhaps chilliness, thirst, coated tongue, constipation; scanty, high-colored urine depositing urates on cooling; the skin is warmer than normal, and there is slight perspiration. The pain usually abates during the day and increases at night. It is aggravated by motion and attended by painful muscular cramps. By the end of the first day or two the swelling increases and the pain lessens, owing to diminished tension of the part. Pain on motion is still great, however, and without treatment may continue for a week or two; under treatment the paroxysm subsides in four or five days. Both great toes may be attacked in the first seizure, more often alternately than simultaneously, and sometimes other joints

than those of the toe are affected. After subsidence of an attack the urine contains a larger quantity of uric acid, and the patient feels better in health and spirits than for some time. A second attack may be postponed for several years, but usually after that the intervals of freedom steadily diminish until an attack recurs every few weeks or months, and the patient may be scarcely ever free from it. Other joints than the toes, particularly those of the fingers, become involved in subsequent attacks.

The Blood. Neusser has attributed to gout and the uric acid diathesis the presence of granules, observed after staining, in the white corpuscles, but they have been found in other affections, and are not diagnostic. The nature of many otherwise obscure gouty manifestations or arthritic changes may be determined by an examination of the blood-serum. Collect the serum which accumulates in a blister and examine for uric acid. (See Blood.)

Chronic gout results from repeated acute attacks. It is characterized by deformity of the affected joints, around which are deposited chalk-stones (tophi) of sodium urate. Similar deposits occur in the helix of the ear. The first appearance is that of a clear vesicle under the skin, which subsequently becomes chalky-white and solid. The deposits of sodium urate occur not only in the cartilages of the joints, but in the ligaments and bursæ also, resulting in great impairment of motion and deformity. "In extreme cases an appearance is presented by the hand very closely resembling a bundle of French carrots with their heads forward, the nails appearing to take the place of the stalks." (Garrod.)

Gouty abscesses consist of collections of liquid and solid sodium urate, which discharge, with or without pus, through the skin. A patient may have a number of them with but slight impairment of the general health. They may even act as a helpful vent to the system.

In so-called *retrocedent gout* the external joint-manifestation is suppressed or replaced by an internal inflammation, as of one of the serous membranes.

Gout attacks the nervous system, causing headache, delirium, and sometimes apoplexy, apoplectiform seizures, epilepsy, mania, various neuralgias, and spinal symptoms. It also affects the heart and blood-vessels, causing valvulitis and chronic arteritis. The symptoms presented by the digestive organs have been mentioned. They are often premonitory of an attack. The kidneys may be affected, causing typical contracted kidney, or there may be chronic cystitis and urethritis.

Rheumatoid Arthritis.

Rheumatoid arthritis, or rheumatic gout, is an affection characterized by acute or chronic inflammation of the joints, of progressive character, and resulting in deformities. It is attended with very little fever, and occurs apart from any known systemic disease.

It may be acute or chronic. The *acute* form differs but little in its manifestations from acute rheumatic fever. Several joints are enlarged, tender, and painful. Constitutional symptoms, such as fever, loss of appetite, frequent pulse, thirst, and furred tongue, occur as in rheumatism.

Profuse acid sweats, however, are absent, and so is the tendency to serous inflammations. Moreover, while the larger joints, as in rheumatism, may be affected, the smaller ones also, especially of the fingers and toes, are inflamed and often the seat of serous effusions. Furthermore, the inflammation persists in the affected joints and does not jump from one to another. Instead of disappearing in a few weeks, it drags on for a much longer time. The pain subsides, but the swelling persists, and permanent deformity results in at least some of the joints. The muscles of the arms and legs waste and are affected with painful spasms. The disease is most common in young women exhausted by repeated pregnancies or prolonged lactation, and is favored by poverty, privation, and cold.

The *chronic* form is much more common. It also attacks most frequently young women who are exhausted or are subjected to great fatigue. There is pain, numbness, or formication in a joint, as the knee. The joint becomes tender, painful, and may be slightly swollen. This subsides after a while, but sooner or later the same joint or another one becomes affected. The process is persistent, one joint after another is attacked, and gradually all the joints may become greatly distorted, enlarged, and the seat of contractions. There may be no impairment of general health, or at most only dyspeptic symptoms. The progress

FIG. 329.

Rheumatic arthritis. (Original.)

is interrupted from time to time by remissions. Pain may be severe and subject to nocturnal exacerbations. The shape is altered by the effusion into the joint and adjacent bursa, by thickening of the tissues around the joint, growths of new bone on the joint-extremity of the bones, absorption of the articular cartilages, and growths of new cartilage in the synovial sheaths. These alterations may be accompanied by relaxation of ligaments, muscular contractures, and luxation of the joints. The joints crack and creak like rusty hinges, are sore and stiff, and the attached muscles are affected with painful cramps. (See Fig. 329.) Great enlargement of the joints at times occurs from the causes already mentioned and from infiltration of the overlying tissues. The enlargement

is rendered more conspicuous by the atrophy of adjacent muscles. (See Fig. 30.) Physicians with large experience in this affection lay stress upon the frequent involvement of the *temporomaxillary articulation*, which is never seen in gout.

In addition to the articular symptoms other phenomena attend the process. One of the more common is increased *frequency* of the *pulse*. Although the patient is afebrile, the average pulse-rate is 100 to 120, or even higher. Moreover, the pulse is soft and compressible, in contradistinction to the pulse of gout and rheumatism. It is worth noting that a return to the normal pulse-frequency is a sign that the progress of the disease is arrested, although the joint-lesions remain.

The condition of the *skin* is characteristic. It is soft and often much freckled, while the complexion is fair. C. T. Griffiths has observed the pigmentary cutaneous changes, along with neural symptoms, prior to the joint-manifestations, and describes two forms: a diffuse melasmic discoloration, and dark-brown spots resembling moles, but not raised. Moisture of the skin with clamminess is common. It is limited to the palms of the hands or may occur in the distribution of certain nerves. The sweats are not acid; they are usually local, but may be profuse. *Pain* independent of the joint-lesion is due to neuritis, and may precede the joint-trouble. It is not confined to the nerve-trunks, but affects the smaller branches which are distributed to muscles, as the group at the base of the thumb. *Numbness* and tingling are often present.

The disease is steadily progressive. In extreme cases not only are the limbs crippled, deformed, and helpless, but there is fixation of the cervical spine and of the articulations of the jaw, so that the patient can not move the head or masticate food. The first phalanx of the fingers is either flexed upon the metacarpus or extended, and the terminal phalanx in like manner is either markedly flexed or extended upon the second, or these two phalanges are held in extension, while the first phalanx is, as usual, decidedly flexed upon the metacarpus. The hand is pronated and the fingers turn toward the ulnar side. (Palmer Howard and Charcot. (See Fig. 329.) The foot is abducted and flattened, and the great toe abducted across and above the other toes. Rarely it may be beneath the other toes. The metatarsophalangeal joint is enlarged.

A variety of the disease is sometimes encountered, chiefly in old persons (senile arthritis), in which the tendency is to involve one or two joints, particularly the hip, or hip and knee. It is of slow progress, and is otherwise attended with the same deformities as the usual polyarticular form.

Diagnosis. Rheumatoid arthritis is distinguished from *gout* by the absence of heredity and by its development under the exhausting influences of repeated pregnancies, lactation, poverty, and malnutrition. Rheumatoid arthritis is progressive, with occasional remissions; gout occurs in successive attacks, with intermissions. Uric acid is absent from the blood in the former and is present in gout. Rheumatoid arthritis in the vast majority of the cases is subacute or chronic. The acute form is distinguished from acute gout by the duration of the paroxysm and the absence of intermissions; by there being less heat, swelling, and redness

of the joints, and less infiltration of the soft parts; by the fact that large and small joints are involved, and that there is no special tendency to inflammation of the great toe.

From *chronic gout* rheumatoid arthritis is distinguished by the absence of hereditary predisposition, of repeated acute attacks, and of the causes of gouty paroxysms—indulgence in sugars, acids, malt liquors, etc. Moreover, rheumatoid arthritis most frequently begins in the hands, and is symmetrical and bilateral. Gout has a predilection for the great toe, and is unilateral. Again, gout attacks well-fed males most frequently after the age of thirty years, while rheumatoid arthritis tends to attack women under the depressing influences already mentioned. It may, however, occur in both sexes, and even be associated with gout.

Rheumatic fever is distinguished from acute rheumatoid arthritis by its tendency to involve the larger joints, its erratic course, acid sweats, and heavy deposits of urates from the urine, its shorter course, its tendency to heart complications, and its subsidence without impairment of the usefulness of the joints.

Chronic articular rheumatism is distinguished by the preceding history, the tendency to seasonal exacerbations, by its involving fewer joints, and not being so symmetrical in the joints affected. It does not produce so great deformity as is common in rheumatoid arthritis, nor is it so likely to affect the vertebræ and jaws. The existence of valvular heart disease or a history of antecedent chorea is in favor of rheumatism.

The joint-affections of *tabes dorsalis* are distinguished by the associated symptoms of inco-ordination and absent knee-jerk, by their sudden onset without pain or fever, by the occurrence of large effusions into the joints, with subsequent disorganization, fractures, and dislocations.

Gonorrhœal arthritis is distinguished by the history of gonorrhœa or the existence of a discharge from the urethra, by the tendency of the disease to attack the larger joints, particularly the knee or shoulder, and to become fixed in one, not wandering from one to another. The affected joint suffers effusion, and the synovial membranes and bursæ are inflamed. The process is very chronic but indolent, and the heart rarely becomes affected.

Chronic Articular Rheumatism.

The patient has pain and stiffness in one or more joints or in the contiguous tissues. The joints most frequently affected are the shoulder and knee. The pain is more or less constant, but worse in damp weather or on the approach of a storm, and worse also at night in many cases. Conversely, it improves in warm, dry weather. There is not much if any tenderness, and rarely any swelling or elevation of temperature. The joints very frequently creak and grate on motion. In the interval between the attacks there is no impairment of the usefulness of the joints. In very chronic cases there may be some atrophy of muscles and permanent stiffness, even fibrous anchylosis. In some cases there are repeated attacks of subacute articular rheumatism, accompanied by the usual symptoms and joint-effusions.

Chronic articular rheumatism is distinguished from *chronic gout* by the

fact that there is no special tendency to involve the great toe, by the absence of the deformities resulting from gout, and the absence of deposits of sodium urate in the ears, fingers, and around the joints.

Muscular Rheumatism.

In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night, or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling; on attempting to move, bend, twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing tearing or burning pain. When the muscles are relaxed, the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to the touch and to sharp blows. Muscular rheumatism may be acute or chronic. In the latter the symptoms are very much like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and in cold, damp seasons.

The disease receives different names according to the muscle affected. The most common subvarieties are: lumbago, in which the muscles of the small of the back are affected; pleurodynia, in which the intercostal muscles suffer; and torticollis, in which the sternomastoid and trapezius are painfully contracted.

In *lumbago* the patient holds himself rigid and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful, but pressure is not. Fever is absent. There is a history of repeated attacks, or of exposure, such as lying upon damp ground. Lumbago needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In *pleurodynia* there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient breathes as little as possible, and often inclines the body toward the affected side to lessen the motion. Pleurodynia is distinguished from pleurisy by the absence of fever, cough, and, above all, of friction-sounds. In intercostal neuralgia there are painful points upon pressure, whereas in pleurodynia firm pressure is grateful, though tapping is painful.

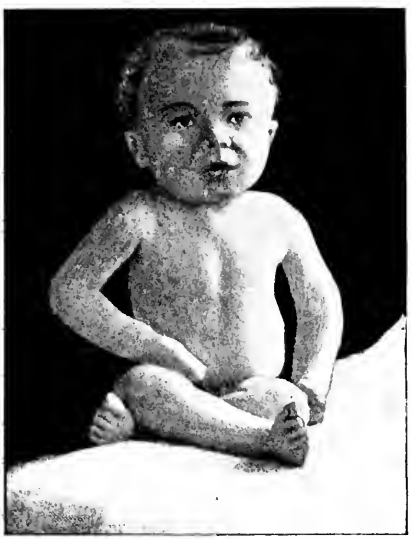
In *torticollis* the head is drawn to one side and fixed in that position. The sternomastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

In rheumatism of the *abdominal muscles* the pain may be so acute as to simulate peritonitis. The surface pain of hysteria which gives rise to so-called "hysterical peritonitis," simulating true peritonitis and rheumatism, may be noted.

Rhachitis.

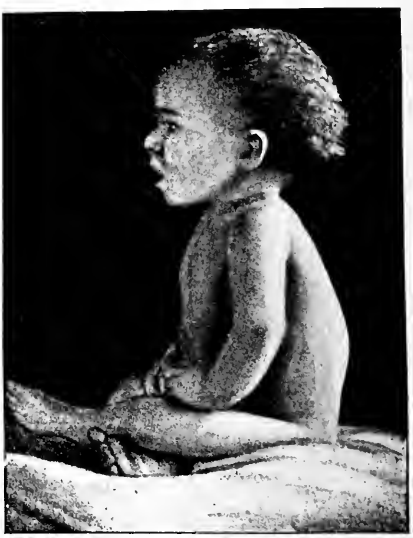
In this affection the size of the body is lessened. For its recognition it is important to know how rapidly the osseous deposits in childhood have formed. The fontanelles and the epiphyses must be examined. If the fontanelles are open after the normal time of closure, or if the epiphyses are enlarged and lack firmness, the condition points either to simple malnutrition or to rhachitis.

FIG. 330.



Rhachitis: attitude in sitting; one hand raised to exhibit swelling at the wrist. (WILLIAMS.)

FIG. 331.



Rhachitis in moderate degree in a boy aged fifteen months; showing backward excurvation of the spine. (WILLIAMS.)

Rhachitis usually develops in childhood, and is most common in children with unfavorable hygienic surroundings, who have lived upon a starchy diet and have taken cows' milk for too long a period of time. A child that has been nursed during the mother's pregnancy is apt to have the disease.

In rhachitis late development of the *teeth* is observed. If the ribs are examined, nodules will be detected at the junction of the bone with the cartilage. These may be seen, as well as felt, if the child is thin. They form the so-called *rhachitic rosary*. The thorax also is changed in shape. At the junction of the cartilages and ribs a depression takes place which is continuous with a groove passing out from the ensiform cartilage toward the axilla. This transverse curve is known as *Harrison's groove*. It may deepen with inspiration. The sternum projects, forming the so-called "pigeon-breast." (See Thorax.) Such deformity must not be confounded with a similar one seen in adenoid disease. Changes at the lower end of the radius and ulna, and sometimes at the end of the humerus, are noticed. The parts are enlarged at the junction of the

shaft and epiphyses. There may be thickening of the clavicles at the sternal ends. In the legs the lower end of the tibia becomes enlarged, and at times the upper end, or even the shaft, becomes thickened. The child becomes bow-legged, or the tibiæ and femora may arch forward. Knock-knee sometimes occurs. The bones of the vertebral column and of the pelvis are also affected. The spine is usually kyphotic, and sometimes there is lateral curvature as well. The contraction of the pelvis is such as to narrow its outlet—a matter of much importance for the future of females.

The child's head is quite characteristic. It has been mentioned that the fontanelles remain open for a long time, and areas of ossification are imperfect, so that the bone yields to the pressure of the finger. This occurs particularly at the side, and the term *craniotabes* is applied to it. The large head is square in shape, not globular, when seen from above downward. The appearance of the face is peculiar: it is proportionately very small, especially in the lower two-thirds, while the forehead is broad and square. In addition to changes in the bones a child presents other evidences of defective nutrition, such as marked pallor, flaccidity of the muscles, and general muscular weakness which results in an inaction resembling paralysis.

The disease usually progresses slowly, and is eminently chronic. A form is seen, however, in which the course is more acute. With some gastro-intestinal disturbances there are mild fever, considerable weakness, and great restlessness. Sleep is disturbed, and pain is complained of, if the child is of an age to make such complaint. *Soreness* of the body is observed on handling the child; and of its own accord, on account of the pain and soreness, it avoids all customary movements. The child lies on its back and shrinks from attempts to disturb it. The pain is not only caused by handling of the muscles, but the bones also are sore and tender. Sometimes the most marked manifestations of the more acute forms are the gastro-intestinal symptoms. It may often happen that vomiting and diarrhœa have this rhachitic condition as an underlying basis.

With the above symptoms, *perspirations* about the head are common, and occur also in chronic cases. There is usually more heat of the head than is natural, hence in sleep the child rolls the head. This rolling causes the hair on the back of the head to wear off, a characteristic of rhachitis when observed along with changes in the skeleton.

In both the acute and the chronic forms *enlargement of the liver and spleen* is observed. The enlargement is not only actual, but a false enlargement may also be seen from distortion of the organs on account of changes in the vertebræ and ribs. The abdomen is prominent, usually on account of flatulence, although the enlarged organs contribute to the swelling.

Nervous phenomena are common in the course of rhachitis. *Tetany* limited to the upper extremities, and *laryngismus stridulus* are the most frequent. Either of these complications may occur before the disease is otherwise suspected.

Diagnosis. The possible presence of rhachitis must not be overlooked

in cases of chronic vomiting in childhood. The acute form of the disease must not be confounded with scurvy, as often happens in the case of children. It must not be forgotten that scurvy may set in in the course of rhachitis. In scurvy the pain, tenderness, and weakness are limited to the lower extremities. The immobility of the extremities may go on to pseudoparalysis. The tenderness, however, is great; œdema is more pronounced, and local areas of periostitis are more common. In scurvy the gums are swollen and may be spongy or the seat of ecchymoses. The most decisive diagnostic criterion is the therapeutic test, scurvy rapidly yielding to a proper regimen.

Diabetes Insipidus.

This form of diabetes differs from the saccharine in that the urine is normal except for its low specific gravity, from 1001 to 1005. The disease may come on suddenly after mental emotion or develop gradually. The amount of urine may range from 10 to 40 pints. It is pale and watery. The solid constituents are not reduced. Urea is sometimes increased, but abnormal constituents are rare. The passage of large amounts of urine induces thirst, but otherwise the symptoms do not tally with the symptoms of diabetes mellitus. The patients are usually well nourished.

The disease is usually secondary to some organic disease of the brain, or of the abdomen, as tuberculous peritonitis, abdominal tumors, or aneurism. It usually occurs in males, and is often hereditary. It is most common in young people. Traumatism, meningitis, affections of the brain involving the sixth nerve, tumors of the brain or of the medulla are causal factors. It may follow fright, a protracted debauch, or perturbation of the nervous system from other causes.

Diagnosis. The diagnosis is not difficult. It must be distinguished from the polyuria that is seen in chronic interstitial nephritis and in amyloid disease. In hysteria, polyuria is common, although transitory. The presence of the stigmata and other hysterical manifestations establishes the diagnosis in hysteria.

Diabetes Mellitus.

The occurrence of any of the following conditions should lead to an examination of the urine for *sugar*, and an estimation of the quantity of urine passed in twenty-four hours, apart from the routine examination, which should be made in every case of chronic disease or of obscure acute disease.

1. *Muscular weakness without cause*, progressive and rapidly advancing to an extreme degree.

2. *Emaciation.* In young subjects this is rapid in cases of diabetes. In older patients it is not so striking, particularly if the gouty diathesis is present.

3. *Thirst.* This is of common occurrence in diabetes. If the amount of fluids taken be compared with the amount of urine excreted, it will

be found that the two bear a definite ratio. The thirst is greater immediately after meals, although the patient does not necessarily have indigestion.

4. *Hunger.* Excess of appetite, boulimia or polyphagia, also occurs in diabetes. The amount of food that is taken is sometimes enormous, and the ravenous manner in which it is devoured is revolting.

5. *Loss of sexual power.*

The five symptoms just mentioned, with increased frequency of micturition, are the common symptoms of diabetes mellitus. They may develop gradually. In rare instances the onset is sudden. The occurrence of these symptoms should lead at once to an examination of the renal secretion.

Three special characteristics of the urine are observed : (*a*) The *quantity* is increased, so that from 6 to 10 pints, or even as much as 30 to 40 pints are passed in twenty-four hours. (*b*) The *specific gravity* ranges from 1025 to 1045, and may even be higher. (*c*) The presence of *sugar*. The sugar is detected by the ordinary tests. (See Examination of Urine.) In addition the urine is usually of a pale color, of a sweetish odor and acid reaction.

In addition to thirst and increased appetite, some gastro-intestinal symptoms may be of diagnostic importance. Of these, first, the appearance of the *tongue* is characteristic. It is dry, red, and glazed. The dryness is increased because of the scanty flow of *saliva*. The *gums* are swollen and spongy, and marginal gingivitis and *stomatitis* are often present. There are no marked dyspeptic symptoms. *Constipation* is of common occurrence. In diabetes the other secretions of the body are diminished. *Perspiration* does not occur excepting in inflammatory complications. The *skin* is harsh and dry. As the disease progresses, the *heart's* action becomes weak and the *pulse* frequent with higher tension. The *temperature* of the body is usually below normal.

Diabetes may occur at any age, but is most frequent in adult life. In young adults the symptoms are more pronounced, and the duration shorter. In patients past middle life the disease may continue for a number of years without marked interference with the health and nutrition.

While the symptoms just mentioned should lead to an examination of the urine, diabetes mellitus may not be indicated by any of the usual objective or subjective symptoms. It may happen that none of these symptoms is sufficiently marked, and that only by routine examination of the urine, or by the occurrence of affections known to be associated with sugar in the urine, is the disease discovered.

Complications. Of the complications which should lead to the suspicion of sugar in the urine the following are the most important :

1. **Cutaneous Complications.** Boils and carbuncles should always lead to an examination of the urine. Pruritus and chronic eczema may have diabetes in the background. Gangrene of the extremities, chiefly of the feet and legs, and gangrene in other situations, are of common occurrence in the course of diabetes.

2. **Lung Complications.** Tuberculosis of both the chronic and the acute

pneumonic type is frequently associated with diabetes. Lobar pneumonia is apt to occur. In all cases of pneumonia the urine should be examined for sugar. Its presence would modify the prognosis of an otherwise favorable case. Gangrene is likely to ensue in acute and chronic lung affections. Gangrene of the lung in the course of diabetes may be latent and recognized only by the odor and the character of the expectoration, or it may run an acute febrile course.

3. **Nervous Symptoms.** *Diabetic coma* may develop in the course of the disease. In young subjects, particularly, the occurrence of coma should lead to a suspicion of diabetes. It may occur before the disease has been recognized. Coma may occur without any premonitory symptoms whatsoever, the patient reeling for a short time, and complaining of pain in the head as after a debauch. The attack may begin with fainting and prostration, followed by stupor which deepens into complete unconsciousness. It may be preceded by nausea and vomiting or by the lung complications previously mentioned. Extreme dyspnœa, agitation, pain in the head, and some delirium precede the loss of consciousness. The pulse becomes rapid and feeble. For this form of coma the term *acetonæmia* is used. The breath is of a peculiar sweetish odor, due to acetone, and this compound is detected in the urine.

Peripheral neuritis should always lead to an examination of the urine. It may be limited to one group of nerves, or may be more or less general, with symptoms like those of locomotor ataxia, as the lightning-pains, abolition of reflexes, and loss of power in the extensor muscles. Diabetic patients are also subject to neuralgia and to peripheral hyperæsthesia and paræsthesia, probably due to neuritis. The neuritis may be so extreme as to lead to paraplegia.

Eye Symptoms. A curious symptom of diabetes is the occurrence of *cataract*. This may develop at any age, and is often rapid in its course. Cataract or alterations of vision always demand an examination of the urine. Diabetic *retinitis* is sometimes present. Atrophy of the optic nerves or muscular insufficiencies may take place, the latter causing pronounced symptoms of eye-strain.

Aural Symptoms. Ringing in the ears, deafness, the occurrence of acute otitis, are phenomena which arise in the course of diabetes.

Diagnosis. Sugar in the urine occurs temporarily when there is an excess of saccharine diet, or when there is functional disorder of the liver. The sugar is small in amount, and the glycosuria is transient. The diagnosis of true diabetes is not difficult, although the disease may be overlooked unless the habit, previously insisted upon, of invariable urinary examinations is fully developed.

CHAPTER IV.

DISEASES OF THE BLOOD AND OF THE DUCTLESS GLANDS.

DISEASES OF THE BLOOD.

THE blood is a tissue, the origin, growth, and decay of the elements of which have been the source of the greatest interest. In days gone by it was the tissue held responsible for many diseases of unknown origin, so that skin eruptions, scrofula, and other affections were known as "blood diseases." At present we hold only such affections blood diseases as show a demonstrable change in the physical or morphological characteristics of the blood. There is either diminution of the red cells, increase or diminution of the white cells, or diminution of the hæmoglobin. Strictly speaking, most of the blood diseases now so called are really diseases of the blood-making organs—the lymphatic glands or the spleen. It is interesting to note that as late as 1866, J. Hughes Bennett included under diseases of the blood leucocythæmia, chlorosis and anæmia, diabetes, the infectious diseases, rheumatism, gout, and scurvy. The most recent textbook divides the blood diseases into *anœmia*, with two subdivisions, and *leukœmia*. Of course, no one thinks of considering the infectious diseases blood diseases, any more than we think of considering typhoid fever an ulceration of the intestine.

Although the blood diseases are thus limited, it is none the less true that the blood may be the only tissue by an examination of which we can determine the ailment from which the patient suffers. As has been previously related, many infections are recognized in this manner only.

The symptoms of blood affections are due to the physical change in the blood and the effect of this altered blood upon the function or the nutrition of the organs. Many *functional symptoms* thus arising may be the first indications of blood disease, as vertigo, headache, dyspnœa, or palpitation, all very common symptoms. The symptoms may be subjective or objective, or both. The recognition of the former comes from the history of the disease and the complaints of the patient. The latter, or the objective symptoms, are determined by the physical examination of the patient and the examination of the blood.

We recognize scarcely any condition at the present day due to an increase of the bulk of the blood or of the red cells. Plethora is hardly a clinical entity. The symptoms of blood diseases therefore are the symptoms of *anœmia*. In like manner, all the data obtained by inquiry are those which belong to some form of anæmia.

Anæmia.

Anæmia is a condition characterized by a reduction in the number of red blood-cells, or of their hæmoglobin, or of the albumin, or of all

combined. In general we say an anæmia is (*A*) primary, essential, or idiopathic; or (*B*) secondary, according to (*a*) the absence of a well-defined cause or (*b*) the presence of some recognizable blood-destroying condition. The anæmias may be suitably subdivided as follows:

ANÆMIA { Primary. . . . . { Chlorosis. / Pernicious anæmia. / Secondary.

The most casual observation may be sufficient for the recognition of anæmia. The color of the surface, the appearance of the mucous membranes, and the evident breathlessness of the patient are indications of diminution in the amount of blood, or of some of its constituents, as the red cells, or of the coloring-matter of these cells. On inquiry it is found that the patient is easily prostrated, that there is breathlessness on exertion (aggravated on ascending any height), that there is palpitation and perhaps cardiac oppression. The patient complains of neuralgias in various parts of the body, and especially of the neuralgia so often seen in the inframammary region of the left side. (See Pain.) Headache is a more or less constant symptom, with the peculiarity that it is increased when the patient ascends stairs, and is often throbbing or pulsating. The anæmic subject has usually a poor appetite and suffers from gastralgia, although it must be remembered that the gastric symptoms of anæmia are as often primary as secondary. Many of the symptoms which attend neurasthenia occur in the course of anæmia.

On physical examination of the patient the appearances above indicated are observed, although grave anæmias may be present, and yet the lips be bright red, the color under the nails fair, and the cheeks flushed, especially if the examination is made in the evening. Reference must be made to the chapter on the Color or Hue of the Surface for a description of the appearance in anæmia.

A study of the heart and bloodvessels usually yields the physical signs that attend anæmia. The vascular phenomena are described in the section on Diseases of the Heart. Here again it must be remembered that considerable anæmia may be present without murmurs in the bloodvessels.

The Blood. The final diagnosis rests upon an examination of the blood. Sometimes the most apparently anæmic subjects yield normal results in blood examination, while the most plethoric in appearance may be very anæmic. The various forms of anæmia give rise to blood-changes in a measure peculiar to the respective variety. The primary anæmias, or hæmolytic varieties, to which pernicious anæmia and chlorosis belong, have characteristics which will be described in the special sections.

In anæmia from hemorrhage the red corpuscles may be reduced to 1,500,000, while the hæmoglobin is reduced to a degree greater than that of the red cells. The leucocytes are increased in number, the polynuclear forms being relatively much more numerous than the other varieties.

The red corpuscles are paler than normal; their white centres are increased in size. This is known as *achromia*. There is some poikilocytosis. An excess of nucleated red corpuscles, or *blasts*, is seen in grave anæmias. If the *normoblasts* are in excess, active regeneration is in

progress; if the megaloblasts, there is reversion to embryonal regeneration, a serious import in a case of anæmia. A megaloblast-anæmia is associated with general increase in size of the red cells and an increase of the macrocytes. In fatal anæmia, as in purpura, the red cells are like those in the form just described, although nucleated red corpuscles are absent. The white cells are sometimes reduced in number; the mononuclear forms may be relatively numerous.

In the oligocythæmic forms of anæmia other than the hemorrhagic, the occurrence of poikilocytosis is constant and marked. Nucleated red corpuscles are not common, although a few of some kind are seen in the severe forms, but large nucleated cells with karyokinetic figures occur. These corpuscles have pale-staining nuclei. Achromic forms, polychromatophiles, and degenerate forms are seen. There is usually moderate leucocytosis in secondary anæmias with increase in the polymorphonuclear elements.

For clinical purposes it is necessary to make a number of divisions of anæmia, though on ætiological and pathological grounds many of them will no doubt soon be grouped together.

I. Toxic Anæmias. The poison may be developed in the body or it may be introduced from without. Toxæmia is, sometimes at least, a factor in the anæmias which develop in the course of acute infectious diseases or during convalescence from them. According to Hunter, pernicious anæmia should be classed under this head. The metallic poisons, particularly lead, mercury, arsenic, phosphorus, the potassium salts, especially the chlorate; certain of the antipyretics, notably pyrodin; and the aniline preparations, are capable of producing anæmia.

II. Parasitic Anæmias. Anæmia may be *parasitic*. 1. To this class belongs the anæmia of malaria, which is due to *Plasmodium malariæ* described by Laveran, and severe pernicious anæmia caused by *Uncinaria duodenalis*.

2. Certain intestinal worms are found associated with marked anæmias:

a. Bothriocephalus latus sometimes produces a disease closely resembling pernicious anæmia, but whether by direct destruction of the blood, or by the development of toxic products themselves destructive, is uncertain; the worms may be present in large numbers without giving rise to anæmia.

b. Uncinaria duodenalis is the cause of the anæmia known variously as Egyptian or African chlorosis, tropical anæmia, brick-burners' anæmia, and the anæmia widespread in the Carolinas, Texas, and other parts of this country.

c. Anguillula intestinalis is the cause of "Cochin-China diarrhœa" and its associated anæmia.

3. *Filaria sanguinis hominis* may produce anæmia by blocking the lymph-channels.

4. *Bilharzia hæmatobia* may produce anæmia by inducing hæmaturia.

III. Anæmia from Hemorrhage. Anæmia may be due to *hemorrhage*. In addition to accidental and *post-partum* causes, purpura, hæmophilia, menorrhagia, and metrorrhagia are frequent causes.

IV. Anæmia from Constitutional and Local Diseases. Anæmia is

often a marked symptom of *constitutional* and *local diseases*, such as tuberculosis, syphilis, cancer, rheumatism, scrofula, scurvy, rickets, Bright's disease, chronic catarrhal gastritis, and others. The anæmia here may be due to the malnutrition and interference with digestion brought about by the disease, or, as in the case of Bright's disease, in part to the direct loss of albumin, and in dyspeptic conditions to inability to take and assimilate food.

In many cases of simple symptomatic anæmia the spleen may become progressively enlarged, probably secondarily. In some cases of Hodgkin's disease the spleen is enlarged. It is not fully determined whether there is a primary splenic anæmia.

V. Anæmia of Malnutrition. Anæmia may also be the result of malnutrition from deficient or improper food, or from the poisonous influences of unsanitary surroundings.

I. Primary Anæmia—Chlorosis.

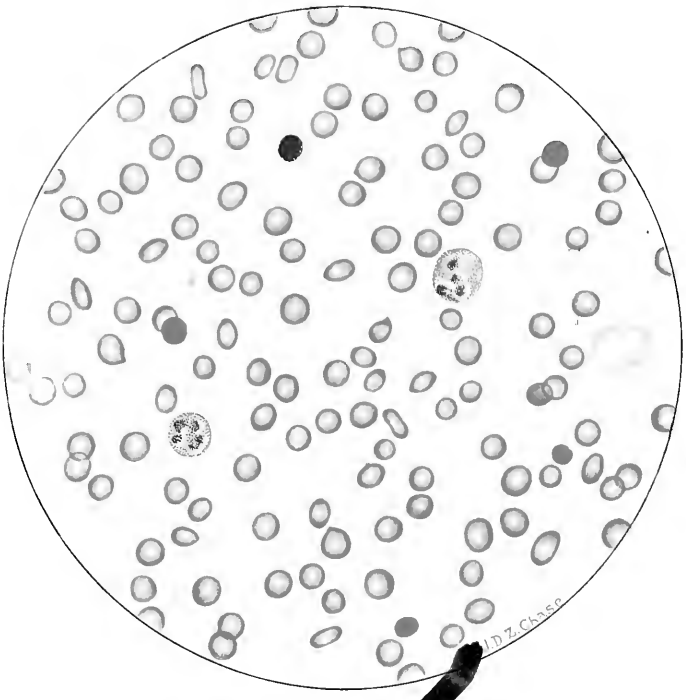
Chlorosis, or chloro-anæmia, is a form of anæmia occurring especially in young girls about the period of puberty and characterized by great pallor with a greenish tint of the skin and mucous membranes, a pearly eye, languor, weariness, suppression or irregularity of menstruation, venous hum in the vessels, dyspnœa, palpitation, dizziness, neuralgias, and an unstable condition of the nervous system. In spite of the extreme pallor there is usually but little loss of flesh. The skin may be pigmented, especially around joints. The bowels are usually constipated; the urine abundant, pale, and of low specific gravity. The digestion is disturbed, the appetite capricious, and the patients sometimes crave unwholesome things, such as earth, slate-pencils, vinegar, and the like. Hyperacidity of gastric juice is commonly present. A systolic murmur over the base of the heart is common. Gastralgia is more common than in other forms of anæmia.

The changes in the blood are very important. There is always a marked reduction in the hæmoglobin, the percentage falling sometimes to 30 or 25 per cent. of the normal. The red blood-cells are usually also reduced, but not in the same proportion as the hæmoglobin. For example, there may be 4,000,000 red cells, but only 30 per cent. of hæmoglobin. Sometimes there is no diminution in the number of red cells; the latter, however, appear pale (achromia), vary considerably in size, microcytes and occasionally poikilocytes are present, and, in severe cases, nucleated red corpuscles are found; occasionally macrocytes occur, but in general the size of the red cells is below that which is usually found. The number of leucocytes varies but little from the normal, but there may be a slight increase. Occasionally there is a rise of temperature, but it is probably due to some complication. (See Plate XVIII.–B, Fig. 1.)

The cause of chlorosis has not been determined satisfactorily. Virchow has established the existence of congenital narrowing of the blood-vessels. Sir Andrew Clark thinks it is due to the absorption of poisonous matter from the intestines; the great benefit that follows saline purgatives in many cases indicates that fecal toxæmia is a factor in these cases.

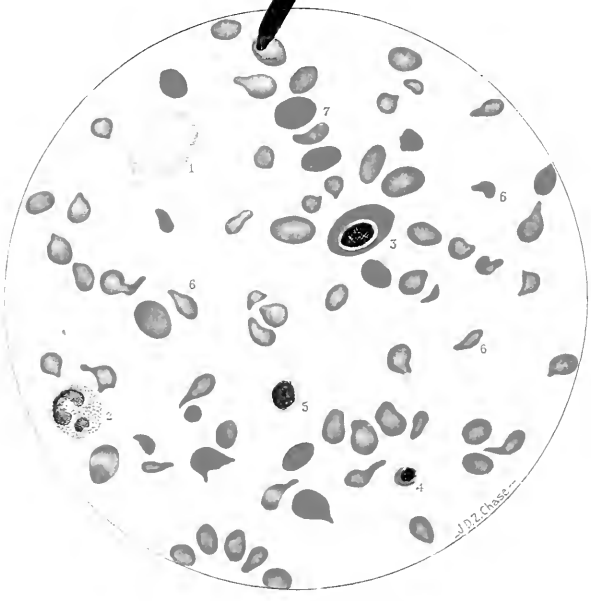
PLATE XVIII-b.

FIG 1



Chlorosis

FIG 2



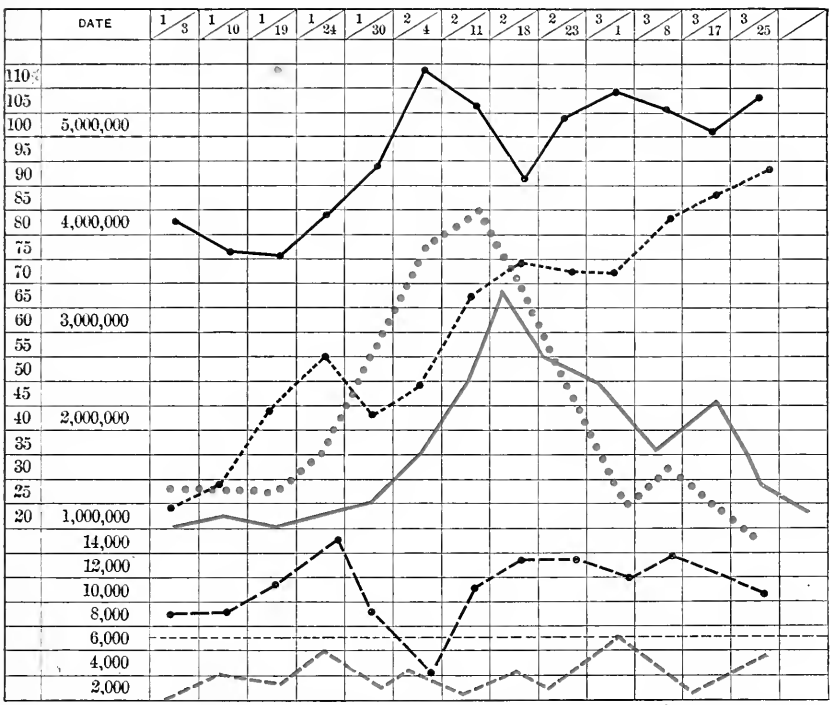
Pernicious Anæmia.

1. Large Mononuclear Lymphocyte.
2. Polymorphonuclear Leucocyte or Neutrophile.
3. Megaloblast } Nucleated Red Corpuscles.
4. Microblast }
5. Small Lymphocyte.
6. Poikilocyte.
7. Normal Red Corpuscle.

Forchheimer [1] also looks upon it as intestinal in origin. Sex and puberty are predisposing causes; but chlorosis may occur in boys, and appear in girls before puberty, and in young women considerably after that period.

FIG. 332.

Blood-charts comparing pernicious anæmia and chlorosis.



Chlorosis. Showing improvement after the third week. Continuous lines, number of red cells; small dots, percentage of hæmoglobin; large dots, number of white cells; red lines, pernicious anæmia.

The prognosis is favorable; the disease may, however, be complicated with gastric ulcer, chorea, tuberculosis, and endocarditis. Recovery is often slow and interrupted by relapses.

II. Pernicious Anæmia.

Pernicious or idiopathic anæmia is a form in which the diminution of red blood-cells reaches an extreme degree. It occurs without adequate known cause, and runs a progressive course with remissions; it usually terminates in death.

The disease usually develops slowly and insidiously, the patient presenting the ordinary symptoms of anæmia—pallor, weakness, shortness of breath, palpitation, venous murmurs, loss of appetite, and impaired

[1] Transactions of Association of American Physicians, 1893.

digestion. As the disease progresses the skin becomes of a pale lemon hue, weakness and dyspnœa increase, the patient has attacks of dizziness, faintness, and ringing in the ears; there may be slight œdema; hemorrhages occur from the nose, the bowels, and into the retina. The hemorrhages are small and distinct in the skin and mucous membranes. The urine is of low specific gravity, and usually contains an increased amount of uric acid. According to Hunter, the urine should be dark and contain a pathological amount of urobilin, some renal epithelium, a few casts containing blood-pigment, and an increased amount of iron. The bowels may be disturbed by diarrhœa. In spite of extreme exhaustion, anæmia, and widespread functional disturbance, there is no emaciation; the patient appears well nourished.

Fever. A peculiarity of the disease is the occurrence of fever of an irregular type. The temperature rarely rises higher than 102° or 103° F. in the evenings, and is followed by a morning remission. It is not usually present in the early stages of the disease, may be absent for weeks at a time when the disease is fully developed, and may cease entirely in the later stages.[1]

Blood. The *blood* appears pale and watery to the naked eye. There may be difficulty in obtaining by puncture a sufficiently large drop for examination. The specific gravity is lowered, often being 1028 instead of 1055. It has been found deficient in fibrin, iron, and nitrogen.

The blood-changes in idiopathic anæmia are characteristic, and are essential to the diagnosis of the disease. In brief they are (1) very great reduction in the number of red blood-cells; (2) an absolute diminution in the amount of hæmoglobin, but as compared with the number of red cells sometimes a proportionate increase; (3) considerable variation in the size of the cells, the average size of the cells probably being larger; (4) poikilocytosis; (5) nucleated red blood-cells; (6) degenerative cells. (See Plate XVIII.–B, Fig. 2.)

Reduction in the number of red blood-cells (oligocythæmia) reaches a more extreme degree in pernicious anæmia than in any other disease; the number often falls below 1,000,000, and in one case reported by Quincke[2] the number was only 143,000 per cubic millimetre. The shape of many of the cells is altered; they are oval, elongated, bent, or have projections of their substance (poikilocytosis). The size of the cells varies; there are microcytes and megaloblasts; but the occurrence of a distinct proportion of large nucleated red blood-cells (megaloblasts) is regarded by Ehrlich as almost diagnostic. The average size of the red cells seems to be increased, and so is the proportionate amount of hæmoglobin in each cell. The latter is a very characteristic symptom (the only one, according to Hunter). There are also red corpuscles which are stained slightly and uniformly by basic stains, "polychromatophilia," or which may show punctate stained areas—"granular degeneration"; these are regarded as degenerative by Ehrlich. The leucocytes are "usually diminished in number, showing a relative increase in the small mononuclear elements

[1] See "Idiopathic Anæmia: A Report of Three Cases." Musser, Philadelphia County Med. Soc. Trans., 1885.

[2] Deut. Arch. für klin. Med., Bd. xx.

(lymphocytes, small transparent forms), while the multinuclear elements are relatively diminished, sometimes being under 50 per cent."[1]

The blood condition is not constant, but is subject to wide variations. Von Noorden has found that in a very short time a change in the form of the blood, a "formal" crisis, may occur. A "formal" flooding of the blood with polynuclear leucocytes and nucleated red blood-cells takes place before a period of improvement; whereas, before a change for the worse and before the final stage the blood becomes poor in leucocytes and nucleated red blood-cells.[2]

Secondary sclerotic changes in the spinal cord cause late symptoms of *locomotor ataxia*.

The *ætiology* of the disease has not been determined satisfactorily. It is more common in Germany and Switzerland than in other parts of Europe or in America. It occurs most frequently after the twentieth year, and between that and the age of fifty. Excluding the influence of pregnancy and parturition, sex makes no difference. Previous exhausting disease, chronic gastric and intestinal catarrh, great physical over-exertion, exposure, great shock or fright, precede in certain cases the development of the disease. It is probably due to faulty hæmatogenesis and hæmolysis.

Petrone and Halst regard the disease as infectious and its germ identical with that found by Frankenhauser. Von Jaksch supposes that it is brought about by a living contagium. Hunter traces the cause to a poison produced by bacteria in the gastro-intestinal canal.

Diagnosis. The most important diagnostic features of the disease are extreme oligocythæmia, relatively high percentage of hæmoglobin (color-index high), great poikilocytosis, which may, however, occur in any severe anæmia, a noticeable number of large nucleated red blood-cells (gigantoblasts), an average increase in the size of the cells, and all this without emaciation or discoverable local disease that might bear a causative relation to the anæmia. In addition, retinal, subcutaneous, and submucous hemorrhages, a urine with high specific gravity, high color, with urobilin in excess, alternating with urine of low specific gravity, in the absence of organic disease, point to *pernicious* or *idiopathic* anæmia. The distinction from secondary anæmias can not be based on the blood examination alone. The history and the physical signs must play a prominent part, while all possible causes of secondary anæmia must be excluded.

The disease is not so rare as we have been led to believe. The writer has seen a large number of cases in consultation, usually believed by the attending physician to be carcinoma of the stomach or liver, heart disease, renal disease, typhoid fever, or tuberculosis.

Leucocythæmia.

Leucocythæmia, or leukæmia, is a disease of the blood-making organs, characterized by great and persistent increase in the white blood-corpuscles; by diminution in the number of red blood-cells, which are altered in shape and size and display nucleated and degenerate forms; by a lessened amount of hæmoglobin, and by changes in the spleen, lymphatic glands,

[1] W. S. Thayer, Boston Med. and Surg. Journ., February 16 and 23, 1893.
[2] Quoted by Weiss, Diagnostisches Lexikon.

or medulla of bone. As a rule, the blood in leukæmia presents one of two distinct types: (1) the *myeloid*, associated with great enlargement of the spleen, marked marrow-changes, and little or no enlargement of other lymphatic tissue, usually a very chronic type; (2) the *lymphoid*, in which some one set of lymphatic glands is enlarged, and the enlargement of the spleen sometimes equals that of the myeloid form. These cases may be acute or chronic. The disease occurs twice as frequently in men as in women, and two-thirds of the cases appear between the twentieth and fiftieth years. In women, pregnancy, parturition, and the cessation of menstruation are causative factors, while in both sexes depressing influences upon the body or mind and antecedent disease, particularly malarial fever, have a distinct influence. The first symptom generally noted is enlargement of the abdomen; subsequently the patient complains of pains in the splenic region, weakness, dyspnœa, hemorrhage, œdema, and digestive derangements. Occasionally profuse hemorrhage from trifling cause, as the drawing of a tooth, has been the earliest symptom noted. The increase of white cells and diminution of red cells are progressive, and in the lymphoid form soon become manifest in the pallor of the skin and mucous membranes, and in increasing weakness and dyspnœa. Pallor is not a constant symptom of leukæmia; the myeloid form and a high grade of color are quite consistent.

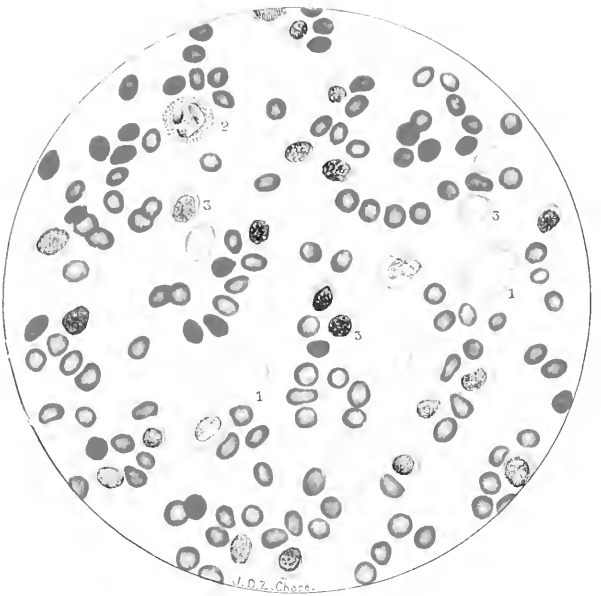
In the so-called *splenomedullary* or *myeloid form* of the disease, myelogenic leukæmia, the spleen steadily enlarges, but may attain considerable size before the patient becomes aware of it. The enlargement is not usually painful, but gives rise to a feeling of distention, weight, and dragging. There may be tenderness on palpation and pressure, and sometimes the patient complains of sharp, stabbing pains, due either to attacks of local peritonitis or to sudden enlargement of the spleen and consequent stretching of the capsule. The splenic enlargement is uniform, so that its shape and characteristic notch are unchanged. Moreover, the spleen remains in contact with the abdominal walls, lying in front of the splenic flexure of the colon, pushing aside the descending colon and small intestine, moving with respiration, and presenting the usual physical signs of a solid organ. Not infrequently the enlargement is so great as to fill the left hypochondriac and iliac regions, and reach beyond the middle line toward the right groin. Sometimes a venous hum can be heard over it. As the result of this enlargement the diaphragm is pushed upward, increasing the dyspnœa already caused by anæmia, and sometimes inducing palpitation. The gastric functions are disturbed from pressure; vomiting and other symptoms of dyspepsia are common.

A rise in temperature is a very common symptom. The fever is of irregular type, usually with nocturnal exacerbations, the temperature not often rising above 102° F. The febrile type may be intermittent or remittent, and sometimes there are periods of apyrexia. The pyrexia is said to be most marked toward the close of the disease. Gowers states that the cases in which there is most fever run a usually rapid course, marked by considerable dropsy and extensive hemorrhage.

As the disease progresses weakness increases; anæmia becomes more intense; œdema, ascites, or hydrothorax occurs; hemorrhages from the

PLATE XVIII-c

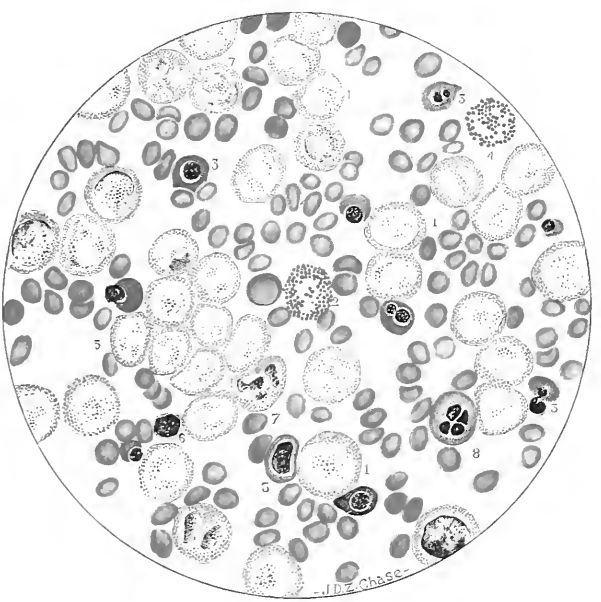
FIG. 1.



Lymphatic Leukæmia.

1 Large Mono-nuclear Lymphocyte. 2. Polymorphonuclear Leucocyte or Neutrophile.
3. Small Lymphocyte, dividing Nuclei.

FIG. 2.



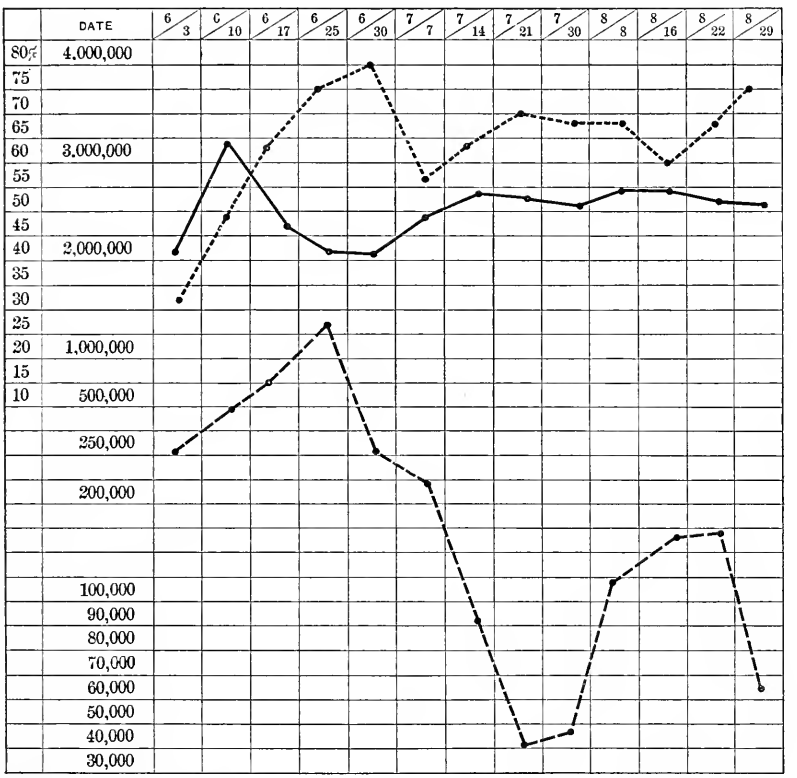
Spleno-Myelogenous Leukæmia.

1. Myelocyte. 2. Eosinophile Myelocyte. 3. Normoblastic Red Corpuscles: dividing or fragmenting nuclei.
4. Eosinophile Leucocyte. 5. Large Mono-nuclear Lymphocyte. 6. Small Lymphocyte.
7. Polymorphonuclear Leucocyte or Neutrophile. 8. Megaloblast.

nose, gums, bowels, stomach, lungs, or uterus further exhaust the patient; digestion is poor and diarrhœa is common. Headache and tinnitus are frequent symptoms, occasionally delirium and coma may occur, and deafness is not uncommon toward the close of the disease. The eyes may be the seat of leukæmic retinitis.

The *liver* is enlarged, often to a considerable degree, but without special symptoms. The same is true of the lymphatic glands and other adenoid tissue. (See Plate XVIII.–C, Fig. 1.) The marrow of the bones becomes the seat of disease in some cases, but it does not usually give rise to symptoms during life; certain bones, however, may be tender.[1]

FIG. 333.



Leukæmia. Continuous line, red cells; small dots, hæmoglobin; large dots, white cells.

The Blood. The most characteristic and important changes from a diagnostic point of view occur in the blood. The blood when drawn from the finger has often a decidedly muddy hue and may interfere with the hæmoglobin estimation, an appearance supposed at one time by Bennett to be due to admixture of pus. It coagulates slowly, is of lower specific gravity than normal, and its alkalinity is diminished. When placed under the microscope, it is at once seen that the number of white

[1] See "A Case of Leucocythæmia." Musser and Sailer, Amer. Journ. of the Med. Sciences, 1896.

cells is greatly increased, and that a large percentage of these increased cells is made up of mononuclear, finely granular cells, the myelocytes, or marrow-cells. These cells are not amœboid, a distinctly noticeable feature. The granules are fine, and neutrophilic, the nucleus is oval. As many as 1,000,000 white cells may be counted in a cubic millimetre of blood, the myelocytes constituting 30–50 per cent. of the total number. If a drop of blood is mixed with distilled water containing a small quantity of gentian-violet, the white cells are stained a decided blue and can be picked out with the greatest ease. Instead of there being 1 white cell to 300 or 500 red, the ratio falls as low as 1 : 5 or 1 : 3, or even lower. Even in periods of comparative well-being, when the total leucocyte count may be nearly or quite normal, a high percentage of myelocytes still remains and distinguishes leukæmia from other conditions in which the white cells are increased. In ordinary leucocytosis myelocytes are absent, and an increased percentage of the polymorphonuclear leucocytes is regularly seen.

Not only do the white cells greatly increase in number, but they vary considerably in size and react differently to staining fluids. Ehrlich has described five varieties of leucocytes.

The variations in the leucocytes in this disease are: (1) the small mononuclear and polymorphonuclear elements are relatively diminished; (2) the great difference in size of the multinuclear elements; (3) the presence of the myelocytic elements; (4) the presence of a normal or but slightly increased proportion of eosinophiles in so extensive an increase of leucocytes [1] and the presence among these of eosinophiles with a single round or oval nucleus, the eosinophilic myelocyte, rarely seen in any other disease. (Plate XVIII.–C, Fig. 2.) (5) Large mononuclear elements with karyokinetic figures. (Müller.) (6) "Mast-cells." Satisfactory study of these can be obtained only by cover-glass preparations.

The essential points in the diagnosis of myeloid leucocythæmia are: 1. A marked excess of leucocytes in the blood, with a high percentage of myelocytes, the ratio of white to red falling below 1 : 50 or 1 : 20. The individual leucocytes vary in size and characteristics, as already described. 2. Enlargement of the spleen. 3. The occurrence of hemorrhages and dropsies unexplainable by disease of the heart, kidneys, or other organs. 4. The symptoms of a high grade of anæmia, as dyspnœa. 5. Leukæmic retinitis. 6. Anæmic fever. 7. The presence of eosinophilic myelocytes, "mast-cells," and nucleated red blood-cells. 8. Specific gravity below 1040. 9. Excess of uric acid in the urine. 10. Charcot-Leyden crystals in the blood.

The *lymphatic form* of the disease is not so common. It is characterized by enlargement of some group of the lymphatic glands and by the great increase in the proportion of the lymphocytes, which may be of the small or large variety or of intermediate size and average about 90 per cent. of the leucocytes present. The total increase in the colorless elements is not so excessive; 300,000 per c.mm. is an extreme. Eosinophiles and nucleated red cells are rare. The myelocyte of Ehrlich is not present. Acute leukæmias are of the lymphoid type and frequently

[1] W. S. Thayer, loc. cit.

show the greatest increase in the large mononuclear elements (lymphocytes); the main features are high fever, a duration of symptoms of only a few weeks, and a rapidly progressing anæmia occasionally with nucleated red cells.

TABLE SHOWING COMPARATIVE DIFFERENCES OF BLOOD IN VARIOUS AFFECTIONS.

Leukæmia.	Normal.	Chlorosis.	Pernicious Anæmia.	Secondary Anæmia.
Red cells.	4,500,000–5,000,000.	Average, 3,500,000; rarely under 2,000,000.	Average, 1,000,000.	Average, 1,500,000 to 2,500,000.
		Poikilocytosis usually only in severe cases; few microcytes and megalocytes.	Marked poikilocytosis; microcytes and megalocytes numerous.	Poikilocytosis present; microcytes and megalocytes.
Normoblasts.	. . .	Occasional.	Fairly numerous.	Numerous.
Megaloblasts.	. . .	Rare.	Usually present in severe conditions.	Sometimes present.
Hæmoglobin.	90–95 per cent.	As low as 30–40 per cent.	Relatively higher than red cells.	Relatively lower than red cells.
White cells.	4000–10,000.	Normal or diminished. 2000–8000.	Normal or diminished.	Usually increased.
Small lymphocytes.	20–30 per cent.	Slightly increased.	Increased.	Diminished.
Large lymphocytes.	5–6 per cent.	Increased.	Increased.	Diminished.
Polymorphonuclear.	60–75 per cent.	Slightly diminished.	Diminished.	Increased.
Eosinophiles.	2–4 per cent.	Normal.	Normal.	Normal.
Myelocytes.	. . .	Rare.	Present in severe cases.	Present in severe cases.

	Pathological Leucocytosis.	Leukæmia, Splenomyelogenous.	Leukæmia, Lymphatic.
Red cells.	Normal or diminished.	2,000,000–3,000,000.	2,500,000 and less.
Normoblasts.	. . . . .	Numerous.	Comparatively rare.
Megaloblasts.	. . . . .	Present at times.	Rare.
Hæmoglobin.	Normal or diminished.	Relatively diminished.	Diminished.
White cells.	Usually not over 50,000.	Average, 200,000–400,000; 1 : 10, 1 : 5, or even 1 : 1 with red cells.	Average, 100,000 or less.
Small lymphocytes.	Diminished.	Diminished.	Increased; 90 per cent. or more.
Large lymphocytes.	Diminished.	Diminished.	Increased.
	Together about 15 per cent.	Together about 10 per cent.	Either small or large may predominate.
Polymorphonuclear.	Average, 85–90 per cent.	Diminished 30–40 per cent.	Diminished; average, 3 per cent.
Eosinophiles.	Normal or diminished.	Increased; 4–10 per cent.	Very scarce.
Myelocyte.	Occurs at times.	Increased; 35–50 per cent.	Rare; 0.3 per cent.

In Hodgkin's disease, lymphadenoma, there are marked and early glandular enlargement and progressive anæmia, the white cells are generally normal in number and in relative percentages toward the end, or if inflammatory changes supervene they may rise from 60,000 to 70,000 per c.mm. This is, however, about the limit of their increase and the relative increase of the polymorphonuclear cells is often a marked symptom. Syphilitic and tuberculous adenitis must be excluded; the spleen, though enlarged in these conditions, rarely reaches the size seen in leukæmias.

"Splenic anæmia," "splenic pseudoleukæmia," "splenomegalic primitive," or, as it has been wrongly called, the "splenic type of Hodgkin's

disease," resembles the leukæmias in general symptoms and in the presence of splenic enlargement, which is often as large as that seen in myelogenous leukæmia. Removal of the spleen has improved or even cured some cases; hence "anæmia with enlarged spleen" is a more acceptable term. The anæmia is marked and progressive, the white cells are diminished (leucopenia) and show no special variations; the reduction of hæmoglobin is excessive and the corpuscular color-index is below normal. Poikilocytosis and nucleated red cells are not present. Banti's disease, if such exists, presents the same characteristics with the addition of cirrhosis of the liver developing after the enlargment of the spleen.

Anæmia infantum pseudoleukæmia of von Jaksch, is disputed as a clinical entity; an intense anæmia, leucocytosis, large spleen and liver, a blood condition with certain characteristics of both leukæmia and pernicious anæmia or even of secondary anæmia are to be seen. It is remembered that the infantile hæmatopoietic system responds readily to any influence with a result far more striking and varied than in the adult. Many of von Jaksch's cases may be leukæmias, pernicious anæmias, or anæmias secondary to rickets or syphilis; some doubtless, according to the condition of their blood, belong to some intermediary state; poikilocytosis, presence of normoblasts and megaloblasts, lymphocytosis, and a moderate percentage of myelocytes, may coexist.

Hodgkin's Disease.

Hodgkin's disease, an extremely rare affection (pseudoleukæmia, lymphadenoma, or lymphatic anæmia), is characterized by enlargement of the lymphatic glands and other adenoid tissue; by progressive oligocythæmia, without, in most cases, much increase of leucocytes, and by the development of lymphatic tumors in unusual situations. The disease is most frequent in the first half of life and three-fourths of the cases occur in males.

The first symptom noted is *enlargement of the glands* of the neck; but sometimes the inguinal, less frequently the axillary glands, are first enlarged; rarely the tonsils are the first to be affected. The enlargement is painless and progressive, appearing first on one side of the neck and extending under the jaw to the opposite side. The tumors at first are distinct, and movable under the skin. The swollen glands may remain in this condition indefinitely for months or years; but eventually they begin to enlarge rapidly, lose their separate identity, and coalesce into large masses. Other glands in remote parts, as the axilla and groin, retroperitoneum, and arm, are affected. They may be soft and fluctuating, or very dense and hard, but heat, tenderness, suppuration, and other evidences of inflammation are absent.

The spleen becomes greatly enlarged, but rarely attains the dimensions common in leucocythæmia. Other adenoid tissue in the intestine, tonsil, and posterior nares, and even the thymus, may enlarge and give rise to pressure symptoms.

Fever is a very constant symptom, but the type is not constant. It may be intermittent, remittent, relapsing, or continuous. A curious form

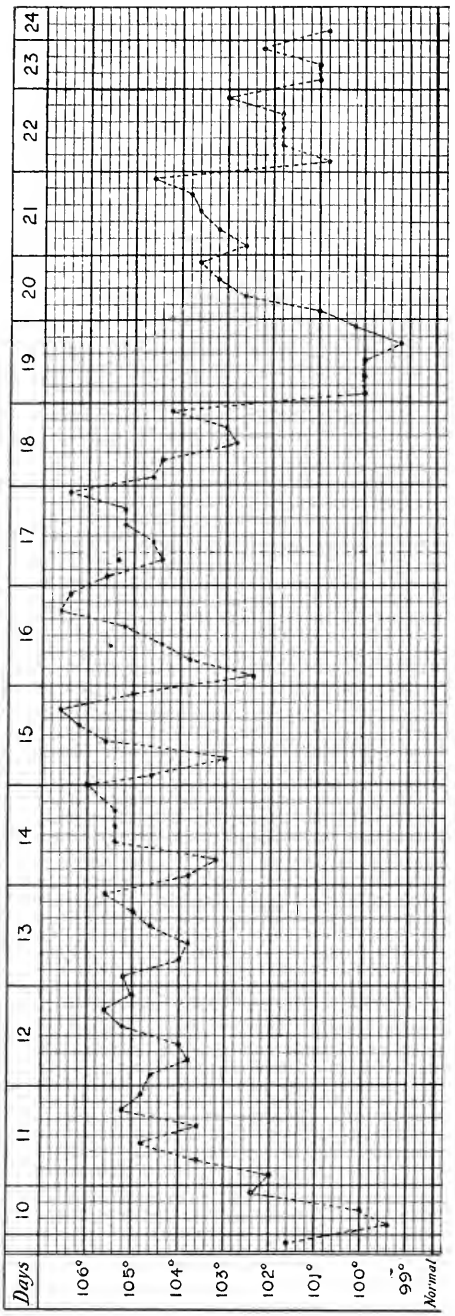


FIG. 334.

A characteristic febrile period in the recurrent fever of Hodgkin's disease.

is that known as *Ebstein's disease*, "Rückfäll," or the relapsing type in Hodgkin's disease. The accompanying chart illustrates the course of the fever during one of the paroxysms. In the interval, which may last ten days or more, the temperature is normal. The paroxysms last from

three to ten days. The onset of the disease may be marked by fever and constitutional symptoms, and the glandular enlargement appears later. On the other hand, in 3 cases reported by J. Dreschfeld,[1] all the patients enjoyed good health and were able to continue their work until a few weeks before death. In all, the symptoms appeared suddenly, and consisted of pain, weakness, pallor, loss of appetite, and pyrexia. Coincident with the rapid and extensive enlargement of the glands, anæmia may arise accompanied by the usual symptoms. Cough is often associated with anæmic dyspnœa, and in women menstruation may cease.

FIG. 335.

Hodgkin's disease. Glands in right axilla and neck much enlarged. (Original.)

Along with the general symptoms there are numerous local ones, due to pressure or to impairment of function—cerebral anæmia from pressure on the carotids; cerebral congestion from pressure on the veins of the neck; disturbance of the heart from pressure on the pneumogastric; deafness; difficulty in deglutition and mastication; and pleural, peritoneal, and pericardial effusions.

The most frequent *complications* are nephritis, fatty degeneration of the heart, pleurisy, and, less frequently, pneumonia and pericarditis.

The *duration* of the disease is from six to eighteen months. Two-thirds of fifty fatal cases referred to by Gowers[2] ended in less than two years. It is difficult to determine accurately the beginning of the disease; sometimes a long period of latency follows the early glandular swelling; sometimes a general anæmia precedes any noticeable swelling of the glands; and sometimes the disease runs an acute course, ending fatally in two or three months.

[1] British Medical Journal, April 30, 1892.
[2] Reynolds' System of Medicine, Philadelphia, 1880, vol. iii., p. 549.

Death results most frequently from exhaustion; but pressure upon the trachea producing asphyxia is not uncommon, and death has occurred from starvation, the result of occlusion of the œsophagus by pressure. The complications already mentioned are the immediate causes of death in other cases.

Diagnosis. The diagnosis is not difficult if a blood examination is made. By this means leucocythæmia is excluded. It may be distinguished from tuberculosis in the early stages when local by the site of the enlargement. In the former the submaxillary glands are involved; in the latter the glands in the anterior and posterior cervical triangles. The *tuberculin* test is required, as insisted upon by Otis, to establish tuberculous adenitis.

Purpura.

Primary purpura occurs without any known cause. It has been divided, for convenience, into *simple* and *hemorrhagic* purpura, though the two differ only in intensity.

Secondary purpura occurs in connection with a variety of febrile and constitutional diseases: (1) scurvy, (2) hæmophilia, (3) Hodgkin's disease, (4) splenic leucocythæmia, (5) pernicious anæmia, (6) chronic lesions of the kidney and liver, with or without jaundice, (7) ulcerative endocarditis, (8) malignant sarcomata, and (9) infectious diseases.

In *simple purpura* the hemorrhages are limited to the skin. They consist of: (1) bright-red spots, varying in size from that of a pin-head to that of a silver three-cent piece. These spots are under the skin and are unaffected by pressure; they fade gradually from red to yellow and disappear. (2) Larger spots or streaks called vibices. (3) Ecchymoses. The disease is said to be most common about the age of puberty. It may come on in the midst of apparent health, or it may follow an illness, as typhoid fever. Purpura occurs especially upon the legs, the standing position seeming to favor its development. The eruption comes on in successive crops. Sometimes large blebs, filled with thin blood, form under the skin, and gangrene occurs at times.

In the *hemorrhagic form*[1] hemorrhages occur from the nose, stomach, bowels, vagina, and bronchi, or into the kidney or some other viscus. Cutaneous and submucous hemorrhages also occur. The onset of these cases is sudden, though there may be a day or two of depression, lassitude, headache, and nausea. The first symptom noticed is generally, *fever*, which is apt to be moderate, then an *eruption* is detected, and for a day or two the patient may seem to be only slightly ailing. Copious epistaxis may now occur, or a hæmatemesis or hæmaturia, or all of these and other hemorrhages may occur the same day. The temperature may be only moderately raised, or it may reach 104° to 105° F., or even a higher point. The pulse at first is frequent (120 to 140), but of good volume and tension. Subsequently, in unfavorable cases, it becomes thready and very frequent. Respiration is not affected, and the mind is

[1] See "Grave Forms of Purpura Hæmorrhagica." Musser, Trans. Association of American Physicians, vol. vi.

clear; the face is pale and anxious. Hemorrhage may also occur into the choroid and brain-substance, with blindness and paralysis as sequels. It may also occur into the uvula or tonsil.

The *subjective symptoms* are pains in the loins, limbs, epigastrium, or chest. Often these pains announce a fresh hemorrhage, as into the kidney, or a fresh crop of purpuric spots. The degree of anæmia depends upon the copiousness of the hemorrhage and the length of time the disease lasts. Sometimes the hemorrhages cause great exhaustion, with a tendency to collapse. In some instances gastro-intestinal *crises* of severe pain and hemorrhage occur, with occasional *arthritis* and acute *nephritis*. The *urine* in the case of hemorrhage into the kidney, of course, contains blood; sometimes casts are also found.

Peliosis Rheumatica. Another variety of purpura is known as peliosis rheumatica, the peculiar features of which are tender and swollen joints, œdema of the subcutaneous cellular tissue, and purpura associated with urticarial wheals and intense itching (purpura urticans). The subcutaneous hemorrhages consist of petechiæ, vibices, and ecchymoses. There may be such large hemorrhages into the penis, scrotum, and uvula as to result in gangrene and slow separation of the dead tissue by ulceration. Epistaxis may occur, but copious hemorrhages from the stomach, the bowel, or into the kidney or other organs are rare. Endocarditis and pericarditis occur as complications in some cases. The duration is apt to be long, convalescence being delayed by repeated outbreaks of purpura with multiple arthritic symptoms and œdema.

Diagnosis. Primary purpura is distinguished from *scurvy* by the absence of antecedent debility and anæmia, of spongy gums, of brawny induration in the limbs, and by the fact that the hemorrhages do not usually occur around a hair-follicle. In scurvy there is a history of deprivation of vegetable food, whereas purpura may occur in the midst of robust health. As a rule, the cutaneous hemorrhages are larger in scurvy than in purpura.

Purpura is distinguished from *acute infectious diseases*, particularly typhus, cerebrospinal fever, and smallpox, by the absence of severe constitutional symptoms which characterize the graver forms of these diseases—in which alone a purpuric eruption is likely to be severe enough to cause doubt. Hemorrhages from mucous surfaces are rare in the latter.

Hæmophilia is distinguished by the history the patient gives of being a bleeder by heredity, and the fact that the bleeding has been started by some injury, wound, or operation.

It is distinguished from the hemorrhages of *leukæmia* by the absence of enlarged spleen and liver, and by the fact that there is no excess of leucocytes in the blood.

Malignant sarcoma causing hemorrhages is recognized by the previous history of anæmia and cachexia, and by the detection of primary or secondary growths.

It must not be confounded with *Raynaud's disease*, a vasomotor affection characterized by local syncope, local asphyxia, and gangrene.

Hæmophilia.

The diagnostic significance of subcutaneous hemorrhage is clearer when associated with profuse hemorrhages in other portions of the body, and when there is also a history of the occurrence of such hemorrhages in the family. Hæmophilia is a constitutional affection characterized by bleeding, which is spontaneous or occurs upon slight injury. It is nearly always hereditary, but may arise *de novo*. Males are very much more liable to it than females, the ratio being about 11 to 1. This curious disposition to bleeding may be transmitted for generations, and almost always to the males through the female members of the family—that is to say, the daughter of a bleeder is not usually affected, but she transmits the tendency to her sons, who become bleeders; so, too, the granddaughters are not bleeders, but they in turn transmit the disposition to their male offspring. It generally shows itself early in life, usually before the end of the second year, and almost invariably before puberty is passed.

The affection usually first declares itself by the occurrence of a *hemorrhage*, either spontaneous or the result of slight injury, the bleeding being far more profuse than would be natural, and in some cases absolutely uncontrollable. Legg[1] has divided hæmophilia into three degrees, according to the severity of the symptoms. The first is characterized by external and internal bleedings of every kind, and by joint-affections; the second, by spontaneous hemorrhages from mucous membranes, but no traumatic bleeding or ecchymoses, and no joint-affections; the third, by a tendency simply to ecchymoses. The first form is seen most frequently in men; the second most frequently in women; and the third in either sex.

The most frequent *seat of hemorrhage* is the nose, and next the gastro-intestinal tract. The bleeding is from the capillaries; it may prove fatal in a few hours, or last for days or weeks, with final recovery. Intense anæmia follows the prolonged hemorrhage, but the blood is replaced with remarkable rapidity. All operations, even the most trivial, are extremely dangerous in bleeders. Circumcision, extraction of teeth, and leeching are credited with the most deaths by Grandidier.

Joint-symptoms are very common. The knees, elbows, ankles, and shoulders are the ones most frequently involved. The attack may be marked by pain, redness, swelling, inflammation, and fever; or fever may be absent; or pain alone may be complained of. The attacks are liable to recur, especially in cold, damp weather, and may result in stiffened, deformed joints.

Diagnosis. The diagnosis is easy when the history of a hereditary tendency to bleed can be obtained. Osler[2] properly remarks that slight joint-trouble and petechiæ are as much a manifestation of the disease as the more severe hemorrhages. In cases in which no history can be secured, the diagnosis is made by noting a persistent liability to hemorrhage, without adequate cause, and associated with joint-affections.

[1] Hæmophilia. London, 1892.
[2] Quoted by Osler, Pepper's System of Medicine, 1885, vol. iii., p. 937.

Osler gives the following excellent summary of the affections with which hæmophilia may be confounded:

1. The umbilical hemorrhages of infants, due to jaundice or to syphilis, hæmorrhagica neonatorum, etc.

2. Purpura simplex, often seen in debilitated, rarely in healthy children, usually confined to the legs, and in some cases associated with rheumatic pains or swellings in the knees and ankles.

3. Peliosis rheumatica.

4. Purpura hæmorrhagica, morbus maculosus Werlhofii, a grave disease, characterized by extensive cutaneous ecchymoses, mucous hemorrhages, but not dependent on any local disease or on any known specific poison.

5. Infective purpura due to the action of some specific poison—smallpox, measles, scarlet fever, cerebrospinal fever, etc. The hemorrhages may be cutaneous and trivial, or may be in the most aggravated form of interstitial and mucous bleedings, as seen, for example, in black smallpox.

6. Toxic purpura, as in snake-bites and many poisons, such as phosphorus.

7. Simple hemorrhagic diathesis, under which may be included those cases in which, without any hereditary disposition or previous hemorrhagic history, there is a tendency to uncontrollable hemorrhage from a slight wound.

8. Hæmatidrosis, bloody sweat, which occurs usually in hysterical or epileptic females, and is in rare instances accompanied by mucous hemorrhages.

Scurvy.

Scorbutus, or scurvy, is a constitutional condition brought about by a long-continued diet deficient in fresh vegetables, or in infants by artificial foods. It is characterized by pallor, great physical weakness and mental sluggishness, dyspnœa, subcutaneous and submucous hemorrhages, a swollen, spongy condition of the gums, and a brawny induration, especially of the calves and hams.

The *joints* are swollen, painful, and tender in about one-third of all cases of scurvy. When to these joint-symptoms the spongy gums, the hemorrhages, the anæmia, and cachexia are added, scurvy may be suspected. The onset of the disease is gradual, and is marked by a peculiar dirty yellow or greenish *pallor* of the face, associated soon with an apathetic expression of the face, physical *weakness*, and decided lack of customary energy. The appearance is so characteristic that patients are said to detect it readily in others, though unaware of it themselves.

The *gums* swell almost always, become spongy, and bleed upon slight irritation. They are dark cherry-red in color and look not unlike a split cherry. Sometimes they swell so as almost to hide the teeth completely and even to protrude the lips. The breath has a heavy, sickening odor, and the teeth sometimes drop out of their sockets. In some cases the eye and its surroundings are the only parts exhibiting signs of scurvy at this time. "The integument around one or both orbits is puffed up into a bruise-colored swelling. The conjunctivæ covering the sclerotic is

tumid and of a brilliant red color throughout, and about an eighth of an inch in thickness or in elevation above the cornea, the latter being at the bottom of a circular trench or well."[1] The condition is not inflammatory. These cases often terminate fatally.

Sleep and digestion are good, but rheumatoid pains may be complained of. Other prominent subjective symptoms are fatigue on slight exertion, dyspnœa, faintness, and despondency. In the course of a week or two petechiæ appear upon the lower extremities, especially around a hair as the centre. (See page 211.) Depending upon the severity of the case there are also bullæ, vibices, and ecchymoses. Brawny induration, due to deep effusion of blood, occurs, especially in the calves and hams, producing considerable pain on flexure of the knees.

There is no fever apart from complications. The pulse is frequent, weak, and small, and the first sound of the heart and the impulse may be very faint.

In addition to the cutaneous and gingival bleeding, hemorrhages occur from the nose and other mucous surfaces, and effusions take place into the lungs, intestines, pericardium, and pleura, associated with inflammatory products. There may be no physical signs of the lungs to account for the dyspnœa; or some dulness and bronchial breathing, or a few râles, may be detected.

A very peculiar symptom, and sometimes the earliest, is *hemeralopia*, or night-blindness; the patient can see during the day but not by moonlight, and except with artificial light is totally blind at night.

So-called *scurvy-rickets* is more or less common in infants fed on artificial food exclusively or on sterilized milk. It is therefore limited to the first four or five years. The symptoms of scurvy are added to those of rhachitis. In 15 cases I have seen, the most pronounced features were those of weakness, anæmia, polyuria, restlessness, the scorbutic gums, local periostitis, particularly of the tibia, sometimes periarticular inflammation, and always a general tenderness of the body, as in rhachitis. In all of them the presence of a dietetic cause confirms the diagnosis.

DISEASES OF THE THYROID GLAND.

The thyroid gland may be enlarged from simple hypertrophy or from fibrocystic disease. The characteristics of these affections are considered in Chapter XX., Part I. Enlargement may also be due to abscess and morbid growths, which are considered at the end of that chapter.

Enlargement of the Thyroid.

Enlargement of the thyroid can be detected without much difficulty. It may be limited to one lobe, or both lobes may be affected. It may vary in size from a small localized swelling to large masses which fill the median and lateral sides of the neck, pressing upon the trachea and extending into the thorax. On palpation the swelling may be

[1] Buzzard, Reynolds' System of Medicine, 1880, vol. i., p. 451.

soft or hard. In the fibrous forms the swelling is not very large and is very much indurated. In the cystic forms of the thyroid enlargement, fluctuation may often be detected ; it may be localized to a small area of the lobe, or may be detected over the entire affected lobe. In some cases, on palpation, a purring or thrill is transmitted to the fingers. The thrill is synchronous with the heart's action and due to increased vascularity of the gland. Auscultation under these circumstances reveals a systolic murmur.

Causes. Enlargement of the thyroid gland may be due to simple hypertrophy, to fibrocystic enlargement, or to enlargement in which the vascularity is more prominent, as in exophthalmic goitre. 1. In simple *hypertrophy* the enlargement is often intermittent, increasing in size at each menstrual period, or coming on in pregnancy to disappear after labor. It may then disappear entirely or return at the menopause. 2. The *fibrocystic* enlargement which occurs in countries in endemic form is persistent. 3. The enlargement of exophthalmia generally continues throughout the course of the disease. (See below.)

Exophthalmic Goitre.

Exophthalmic goitre, Graves' or Basedow's disease, is far more frequent in women than in men. It may develop at any age, but is most common in early adult life. A neurotic heredity, exhausting disease, general debility, and anæmia are predisposing causes, while sudden fright or shock is the most common exciting cause. Graves' disease begins slowly.

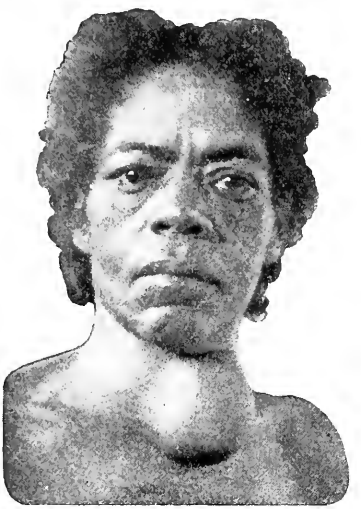
Of the three classic symptoms, *rapidity of the heart's action*, with palpitation, *enlargement of the thyroid*, and *prominence of the eyes* (exophthalmos), the first is the essential symptom. It is also usually the earliest. Either enlargement of the thyroid or exophthalmos may be absent for months or years, and in some instances throughout the disease.

Tachycardia. Attacks of palpitation may recur at intervals for a long time before their true nature is suspected. In these attacks the behavior of the heart is much like that which occurs under the influence of fright or great excitement. The frequency may not be over 100 or 120 in the early attacks, the rate being normal in the intervals. In the later and severe attacks, however, the pulse beats 160 or 180 or even 200 per minute. It is small and regular. The heart beats with increased force ; the sounds are loud, sharp, and clear, occasionally being heard several feet from the patient. In time the heart becomes hypertrophied and dilated, and there is often a loud, basic, systolic murmur. The larger arteries and even sometimes the smaller ones show the vascular disturbance by increased pulsation, sometimes with thrill.

The Thyroid Gland. The thyroid is usually the next to become affected. It enlarges slowly from vascular dilatation, the swelling at first subsiding in the intervals between attacks, but subsequently persisting. The right lobe may be larger than the left. The enlargement is painless, soft, and compressible. It may pulsate with or without thrill, and over it can be heard hæmic murmurs.

The Eyes. Prominence of the eyes is the most conspicuous feature of well-marked cases. Like enlargement of the thyroid, it varies in degree, and rarely is wholly absent. The protrusion allows the white sclerotic to show above and below the cornea, giving the eyes an unnatural, startled, staring appearance. The protrusion may be so great that the eyelids can not close; more commonly they close, but when the eyeball is simply directed downward the upper eyelids do not follow but remain spasmodically elevated or lag behind the movement of the eyeball. (Von Graefe's sign.) The eyeball may become inflamed and even slough from undue exposure. In rare instances one eyeball alone is

FIG. 336.



Exophthalmic goitre. (Original.)

affected, and in these cases the lobe of the thyroid of the opposite side is enlarged. Stelwag's sign (widening of the palpebral fissures) is the third ocular sign of significance in exophthalmic goitre. Finally, Möbius calls attention to the frequency of insufficiency of the internal recti muscles.

In addition to these characteristic symptoms *loss of flesh* and *strength*, moderate pyrexia of irregular type, impaired appetite, diarrhœa, and despondency are observed. The *diarrhœa* is of the nervous type—increased peristalsis without local catarrh. The menstruation is apt to be disturbed. Tinnitus aurium, headache, and vertigo are not uncommon, and sometimes there is profuse *sweating*. A restless, nervous excitement (Charcot) is very common. *Muscular tremor* (Marie), occurring on voluntary movement, is frequently observed, and with diarrhœa is almost as common as the three primary symptoms. *Œdema* of the feet is often seen if there is coexisting mitral disease. Transitory vasomotor œdema of the eyelids, the face, hands, and the supraclavicular and infraclavicular regions occurs. It is usually circumscribed, and may not pit on pressure. Transient erythemas are not uncommon.

Prognosis. Graves' disease, as a rule, runs a chronic course, lasting for years. A few cases that have run an acute course of a few weeks, some ending in recovery and some in death, have, however, been reported. Moreover, there may be recurring attacks with apparent recovery in the intervals. Death results from gradual weakening of the heart and its direct and indirect effects. It may be hastened also by uncontrollable diarrhœa, acute mania, and epilepsy. The disease may also be complicated with hemorrhages, and these may be the immediate cause of death.

Enlargement of the thyroid gland from the above-mentioned causes must be distinguished from enlargement due to *abscess*, *cancer*, *sarcoma*, or *adenoma*. Abscess usually follows infectious diseases; in the writer's case it followed typhoid fever. With carcinoma and sarcoma there are anæmia, gradual loss of flesh, and the usual clinical phenomena of these processes. It must also be distinguished from other tumors in this region. It particularly must not be confounded with enlargement on the right side due to an innominate aneurism. (See Aneurism.)

Myxœdema.

Myxœdema is a disease of mature life and occurs most frequently in women. The swelling is general and involves the face. The arms are more markedly swollen, however, than the fingers; the legs more than the feet. Usually the swelling of the legs and arms is irregular. In some cases *supraclavicular paddings* are marked. These paddings must not be confounded with the pseudolipomata described by Verneuil as occurring in these situations. The swelling is due to infiltration of the connective tissue with mucin, and arises from some affection of the thyroid gland. The *gland is absent* functionally or actually. The hard, indurating, non-pitting swelling is associated with striking change in the appearance of the face, particularly the nose and forehead. The nose becomes thickened, the forehead more prominent and overhanging. The outline of the face is rounded, and the term "full-moon" is applied to it. The *skin* is thickened, dry, and rough, somewhat translucent, pale or yellow in color, and of a doughy consistence, but with a moderate degree of elasticity. The perspiration is diminished. The hands change in shape, become square or spade-shaped, and the fingers clubbed. The nails become brittle and distorted; the hair dry, harsh, and may fall out.

With these remarkable changes in the exterior, marked nervous and mental symptoms arise. *Speech* is thick and hesitating, the memory feeble. The *intellect* is dull and irresponsive; the temper irritable. *Sensibility* is impaired, particularly the pain sense. Patients have been burned without their knowledge, as happened in one of the writer's cases. Abnormal sensations of heat and chilliness are complained of, as well as other paræsthesias. The patient is anæmic, the temperature is subnormal, the heart's action weak, the respiration sluggish. Breathlessness on slight exertion is pronounced, and exertion itself is very difficult; while there is a greater sense of fatigue than the exertion and the condition of the organs would warrant. The muscularity is enfeebled. There are impairment of appetite, indigestion, and flatulence.

The *urine* may become albuminous, but for a long time is not characteristic save in amount and specific gravity. The former is increased, the latter lowered.

FIG. 337.

A typical case of myxœdema. (STARR.)

As the case advances mental and physical failure becomes more pronounced, the patient becomes subject to hallucination, and extremely irritable. Stupor sets in; death may take place in coma or from uræmia. Myxœdema is a disease of mature life, and occurs most frequently in women.

The following varieties are seen: (1) spontaneous myxœdema of the adult; (2) infantile myxœdema; (3) operative myxœdema; and (4) endemic myxœdema or cretinism. In infantile myxœdema the functions of the thyroid body are suppressed during the period of the individual's development. Typical cases justify the name of myxœdematous idiocy.

DISEASES OF THE SUPRARENAL GLANDS.

Tumors of the suprarenal glands are considered in the discussion on Physical Diagnosis of Diseases within the Abdomen, Chapter XXXVI.

Addison's Disease. (See Plate II., page 209.)

The most marked form of *bronzing* is seen in Addison's disease—an affection characterized by a gradual loss of strength without much loss of

flesh; by gastric uneasiness and occasional vomiting; feeble circulation, and a bronze hue of the skin.

Social History. The disease occurs most frequently during the active period of life, from the age of twenty to forty years, and nearly twice as often in males as in females.

Asthenia. The disease begins insidiously with gradual and progressive *loss* of *strength*. It becomes evident from the patient's languor, weariness on slight exertion, and inaptitude for mental effort that he is suffering from some exhausting disease. The most characteristic symptom is the extreme prostration without obvious cause. Any exertion requires great effort and may induce *syncope*.

Gastric Symptoms. The appetite is impaired or lost, there is more or less discomfort at the epigastrium, and occasional vomiting.

Perhaps at this time a close inspection may show some discoloration of the skin, but usually this appears later. By degrees the gastric symptoms become more prominent, and vomiting may be so frequent as to shorten life materially. Finally the patient is unable to leave the bed. Dull pains in the head, back, and abdomen are not uncommon; neuralgic pains in the limbs may be complained of; and Osler states that there is tenderness on pressure in the lumbar region in a considerable proportion of cases.

The *pulse* is extremely small and feeble; in the later stages it may be absent at the wrist.

Bronzing. The discoloration of the skin is the most striking symptom of the disease when it is well marked. The external surfaces are changed in hue, and delicate portions of the skin underneath the clothing are also bronzed. The discoloration is not removed by pressure. The areas are irregular in shape. The skin is soft and pliable. The pigment which causes the discoloration is deposited in the rete Malpighia. The pigmentation is never seen in the cornea or in the nails. The axilla, the flexure of joints, the median line, the areola about the nipple and other normal areas of pigment deposit are the common sites. Bronzed areas in sharply circumscribed patches are also seen in the mucous membrane of the lips and cheeks. Sometimes the whole body becomes of a walnut-juice color, a bronzing which is deeper in exposed surfaces. At times only portions of the body are discolored, in which case the dark hue shades off gradually into the normal hue of the skin. Wilks[1] states that in all the cases which he has seen the scalp, finger-nails, soles of the feet, and palms of the hands escaped pigmentation.

Nevertheless, discoloration of the skin is not an invariable symptom of the disease; in some cases it is entirely absent. These cases, especially if associated with much vomiting, run a more acute course than the others, lasting only a few weeks. Such cases have been mistaken for *typhoid fever*. On the other hand, diseases of the suprarenal capsules not usually associated with the Addison symptom-complex, as carcinoma, are attended by pigmentation. However, in about an equal proportion of cases it is absent. At times the bronzing and other characteristic symptoms of Addison's disease are associated with tuberculosis in other organs. Con-

[1] Reynolds' System of Medicine, Philadelphia, 1880, vol. iii., p. 561.

versely, in cases of phthisis in which there is bronzing, tuberculous disease of the suprarenal capsules may be suspected, and the gravity of the prognosis increased.

Diseases of the spleen are considered in Chapter XXXVI, Physical Diagnosis of Diseases within the Abdomen.

Diseases of the lymphatic glands other than leukæmia and Hodgkin's disease, are considered in Chapter XXV., Part I.

CHAPTER V.

DISEASES OF THE NOSE AND LARYNX.

THE NOSE.

THE symptoms of disease of the nose are: (1) physiological; (2) anatomical; or, (3) those due to the morbid process.

Physiological Symptoms. Impairment of the sense of smell, anosmia, and symptoms of obstruction may occur. *Obstruction* causes retention of secretions and invites infection. Putrefaction and fermentation set in and give rise to offensive *odors*. In addition the patient becomes a *mouth-breather*. The appearance of the face is altered; the voice changes, snoring is common, mastication is interfered with, and the amount of air that enters the lungs diminishes. (See the Lungs, Chapter VI., Part II.)

Symptoms due to the Anatomical Structure. The nose is an open space or a series of air-spaces lined with *mucous membrane*. The membrane is subject to affections that are common to all mucous membranes, and the subjective and objective symptoms are similar to those that arise in other organs, modified by the function and anatomical arrangement.

The abundance of bloodvessels and glands is the cause of one of the symptoms—namely, the *discharge*. Moreover the difficulty of removing the discharge from the various cavities in the nose in which it is pent up, leads to *obstruction*. Anatomical defects and growths produce the same effect. Because the air is constantly passing over the parts, discharges are prone to dry out, and hence *crusts* and *scabs* form. The *vascularity* of the structures of the nose is another source of symptoms. The bloodvessels are richly supplied with nerves, which cause them to contract or dilate on comparatively slight provocation by reflex action. Chilliness of the body, or of local areas of the body, chilling of the extremities, and other peripheral impressions, are followed by congestion of the nasal mucous membrane which may go on to inflammation. The vascularity predisposes to hemorrhage.

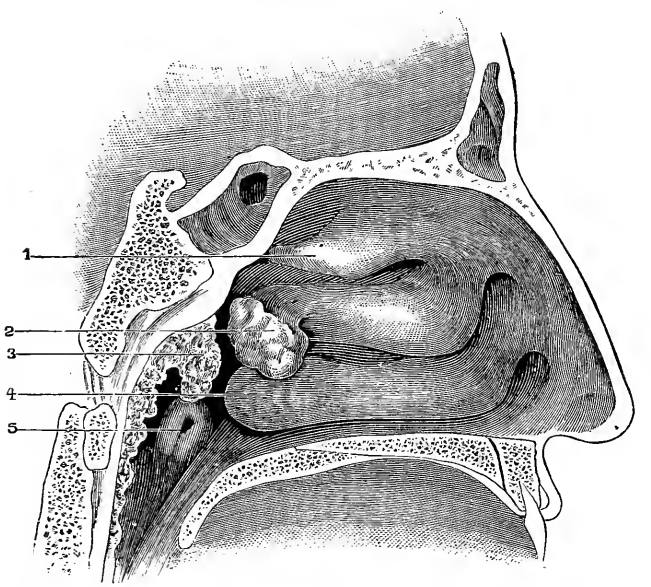
The nose is richly supplied with *sensory nerves* (in addition to the olfactory), on account of which we have the neuroses previously described. The irritation may be derived from air laden with unusual material, as fumes of a chemical nature, emanations from animals or plants, and certain substances not yet isolated, which are decidedly irritating. There is often local irritation from polyps and adenoid growths, and foreign bodies, or enlarged bone. The nerves are connected by a mechanism directly with the centres in the medulla, particularly with the pneumogastric centre. The effect of the above-mentioned peripheral nasal irritation may be felt reflexly in the area of distribution of that

nerve; hence an unpleasant odor may bring on sudden nausea or vomiting. But of more striking and frequent pathological significance is the occurrence of asthma, or sudden dyspnœa, from reflex excitation of the pulmonary division of the pneumogastric nerve.

Morbid Processes. The morbid processes are congestions; inflammations, which may be infectious, or toxic as from vapors; infections, either primary or symptomatic of some general disease; and morbid growths. Acute inflammations are significant of the exanthematous diseases, particularly measles. As Meigs has pointed out, an acute inflammation with great obstruction of the nares and an abundant, puriform discharge, is a complication or symptom of Bright's disease which may portend the onset of uræmia. Chronic inflammations may be due to syphilis or other chronic infection.

Diagnosis. The facts obtained by the *historical diagnosis* give but little information in the differential diagnosis of nasal diseases, except for the lesions which simulate one another but which have a different origin. Often only in this manner can we recognize syphilitic lesions.

FIG. 338.



Vertical section through nasal cavities. (Diagrammatic.) (SEILER.)

1. Superior turbinated bone. 2. Middle turbinated bone, with posterior hypertrophy. 3. Section of hypertrophied pharyngeal tonsil. 4. Inferior turbinated bone. 5. Orifice of Eustachian tube.

The *subjective diagnosis* demands a study of the symptoms of pain, cough, and disturbance of the sense of smell. The first two have been considered in the chapters devoted to these symptoms.

Disturbance of the Sense of Smell. (See the Nerves.) *Loss of smell*, or *anosmia*, occurs to a moderate degree in all the inflammatory and obstructive diseases of the nose. The intensity depends upon the degree of change in the mucous membrane. It may also be due to disease of the

nerves or the olfactory centre in the brain. *Parosmia* is the perception of abnormal odors, and may be a neurosis or solely a psychical difficulty and hence purely subjective, or there may be inability to distinguish an odor when presented to the nostril. All odors may appear the same, or agreeable odors may seem to the patient very disagreeable. In addition, the patient may complain of the perception of an odor in connection with the nasal disease with which he is affected. Parosmia is due to an involvement of the olfactory nerves.

DISEASES OF THE NOSE.

The subjective and objective symptoms previously described are due in general to inflammations, malformations, morbid growths, and foreign bodies. They are recognized by their subjective and objective signs, by rhinoscopic inspection, and by bacteriological and microscopical examinations. The inflammations may be acute or chronic, primary or secondary. When secondary, both acute and chronic inflammations may be due to infections. To the acute varieties belong the acute catarrh of measles, glanders, hay fever, or influenza; to the chronic belong syphilis and tuberculosis.

Simple Acute Rhinitis.

Acute coryza, or "cold in the head," is ushered in with a feeling of lassitude, aching in the back and limbs, feverishness, and a sense of fulness in the nostrils, with sneezing. After twenty-four hours an irritating discharge begins. During this time the malaise has increased. The pain in the forehead and cheeks has become more pronounced, and a nasal twang is given to the voice. The fever continues, the temperature reaching 101° F. in the more pronounced cases, with thirst and loss of appetite. At the height of the fever, in twenty-four or forty-eight hours, a crop of herpes very often develops on the lips. The general symptoms then subside and the local symptoms change. The discharge becomes thick and purulent, the fulness continues, but the pain is diminished. The inflammation often extends up to the tear-ducts and to the eyelids. The latter are congested and smart very much. Very frequently, also, the inflammation extends to the pharynx, causing soreness of the throat and stiffness of the neck, and the larynx even may be involved. A slight deafness may result from the inflammation extending into the Eustachian tube.

Hay Fever.

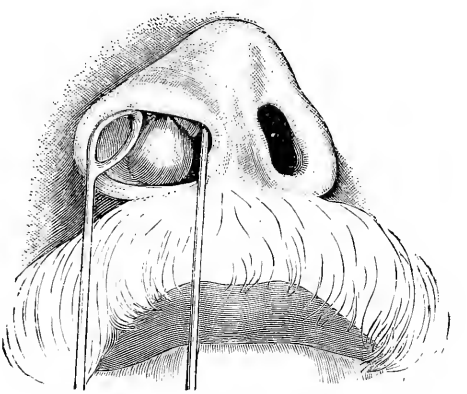
Hay fever is an acute affection ushered in by paroxysmal sneezing, itching, and smarting of the inner canthus of each eye, or of the throat or nose. After hours or days of sneezing, coryza develops. The disease continues for a varying length of time, and is more pronounced at certain seasons of the year, particularly the late fall. Coughing may be an additional symptom, and paroxysms of asthma may develop which are hard to distinguish from true bronchial asthma. The attack may be excited

by vegetable emanations, particularly the pollen of plants, but other emanations may also induce it. Certain conditions of the nasal mucous membrane predispose to the attack. Local inflammation of the nose or obstructive diseases from hypertrophies are primarily present. To the exciting cause and the local predisposing cause may also be added a neurotic factor. The disease affects families of nervous constitution, and may occur through several generations. It is more common in this country than abroad, and dwellers in cities are more subject to it than residents in the country. *Asthma* may be due to disease of the nose, but the only proof that it is of nasal origin is that it disappears after the nose has been treated for the various ailments that are supposed to cause it.

Chronic Rhinitis.

Four varieties are distinguished, to all of which the term *nasal catarrh* is applied. In one there is hypertrophy of the turbinated bones; in the second there is extension of the disease to the post-pharynx—chronic

FIG. 339.



Dilated nostril, showing anterior hypertrophy. (SEILER.)

post-nasal catarrh; in the third there is absolute dryness of the mucous membrane—rhinitis sicca, or dry catarrh; in the fourth there is atrophy of the mucous membrane—atrophic rhinitis, or ozæna.

Chronic Hypertrophic Rhinitis.

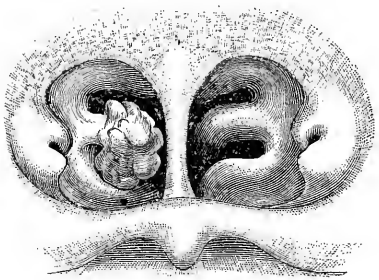
The affection comes on gradually after repeated acute attacks of coryza. The only symptoms may be slight fulness in the nose and a little hoarseness of the voice. In more advanced stages the symptoms of stenosis are marked, the patient snores and breathes through the mouth, and the voice becomes nasal. There is a constant discharge of mucopus backward into the pharynx, causing hawking. The hearing is frequently impaired, as well as the taste and smell. The discharge often affects the larynx, causing an irritating cough. The hypertrophied tissue on the turbinated bones,

and the pressure of the bone on the septum may lead to reflex attacks of asthma.

Chronic Post-nasal Catarrh.

Chronic post-nasal catarrh is an extension of the rhinitis into the pharynx. It is distinguished by discomfort or pain in the soft palate and posterior nares. There are tingling and a sense of fulness at the root of the nose, with frontal headache; the patient complains of a bad

FIG. 340.



Rhinoscopic image from a case of posterior hypertrophy on the middle turbinated bone. (SEILER.)

taste in the back of the mouth, and of constant flow of thick secretion into the pharynx, causing snoring and hawking. The same perversion of the senses of taste, smell, hearing, and of the voice occurs as in acute rhinitis. Headache seems to be due to the condition of the pharynx. (See Atrophic Rhinitis.)

Dry Catarrh, or Rhinitis Sicca.

Dry catarrh, or rhinitis sicca, is also chronic in its course, and is accompanied by tingling and dryness of the nostrils. A faint, musty odor is detected, but there is no discharge or sense of obstruction. In severe cases there may be sharp pain in the nose extending to the forehead.

Syphilitic Coryza.

Syphilitic coryza is seen in infants and young children affected with hereditary syphilis. The nostrils are swollen and red at the edges, sometimes completely occluded, causing oral respiration and inability to take the breast or bottle.

Pustules, fissures, and ulcers are found in the nose and at the margin of the orifices. They are also seen in the pharynx and larynx. Hemorrhages may occur. Other evidences of hereditary syphilis are present.

Atrophic Rhinitis, or Ozæna.

The *odor* is characteristic, and is diagnostic if syphilis is excluded. A sense of dryness is complained of. Occasional obstruction arises from accumulation of crusts, otherwise the passage is unduly open. There

are constant hawking and spitting of brownish-green crusts, which are often blood-tinged. Frontal headaches may occur in paroxysms. The patient is often depressed in spirits. The bridge of the nose may fall in slightly.

The Auxiliary Cavities of the Nose.

The Antrum. The antrum is subject to abscess, cysts and polypi, tumors, and to the invasion of parasites.

Abscess. An odor somewhat like that of ozæna, a putrid taste, nausea, anorexia, pain in the cheek and at the root of the nose, often neuralgia in the frontal region, and malaise are present. A very characteristic symptom is the discharge of pus from one nostril on leaning the head forward. There is often a decayed tooth on the same side in the upper jaw.

The Sinuses. The frontal, ethmoidal, and sphenoidal sinuses are subject to inflammation, abscess, traumatism, and the irritation of foreign bodies, usually parasites.

The frontal sinuses are the only ones that exhibit external symptoms. When these cavities are inflamed, the patient complains of pain and tenderness over the frontal protuberances; if the process goes on to the formation of abscess, there may be redness and swelling and finally fluctuation. If the communication is not closed, a fetid discharge from the middle meatus takes place.

When the sphenoidal and ethmoidal sinuses are affected, there are no external symptoms unless the enlargement is so great as to affect the orbit. There is deep-seated pain. Pus is seen exuding into the superior meatus and flowing backward into the pharynx. Parasites cause intense pain and lead to abscess, caries, and necrosis. *Rhinoscopic* examination, in disease of the antrum, shows rough hypertrophic enlargement on the under surface of the middle turbinated bone and a flow of pus into the middle meatus. Sometimes a probe can be passed into the antrum from the nose. Often an exploratory puncture is necessary. *When the foramen is obstructed*, there is a dull aching pain in the upper jaw, with deformity of the orbit, face, hard palate, and nostril. Fluctuation can usually be found at some point after a time.

The *lacrymal duct* and *sac* are often the seat of inflammation by extension, causing pain, obstruction in the nose, and epiphora. On examination pus will be seen flowing forward over the inferior meatus. When the lacrymal probe is introduced, the ducts are found to be painful and obstructed, and pus exudes.

THE LARYNX.

The structural composition of the larynx does not differ from that of other parts of the respiratory passage. Mucous membrane, connective tissue, cartilages, and muscle are similar to the same tissue situated elsewhere.

The result of their anatomical association in the larynx is the establishment of the functions of that organ, the formation of the voice, and the admission of air. Now, the morbid processes that affect the larynx do

not differ from morbid processes elsewhere in which similar tissues are involved. Each tissue is liable to congestion, to inflammation, to degeneration, to new-growth formation; the joints may become anchylosed, the muscles either paralyzed or the seat of spasm, and we have therefore all the symptoms common to morbid processes in each class of tissue. We also meet with other symptoms, which result from the anatomical position of the larynx and of its functions. The cords can not vibrate, or the muscles and articulations can not move, and *dysphonia* or *aphonia* occurs. The narrow chink of the glottis soon becomes occluded, giving rise to *dyspnœa*. Obstruction to the pathway or pain from inflammation or ulceration causes *dysphagia*. The sensitiveness of the mucous membrane provokes *cough* on the slightest provocation.

The larynx is a highly specialized organ, and is well innervated. Large central nuclei, connected by a large nerve which passes over a circuitous route and which anastomoses with other nerve-cords, preside over the function of phonation. Affections of the central nuclei, affections of the nerve-trunk or of adjacent structures exerting pressure upon the trunk, have their expression in disorder of the larynx, particularly if phonation is disturbed. In other words, the phenomena of laryngeal disease may be symptomatic of affections of the brain or of the nerve-trunk, as well as of the larynx. (See Nervous Diseases.)

Owing to the anatomical position and special function of the organ the symptoms of disease of the larynx are very striking, pointing at once to the seat of trouble. Laryngeal affections are not likely to be mistaken for disease of contiguous parts, although retropharyngeal abscess, abscess at the side of the pharynx, disease of the thyroid gland, and inflammation of the lymphatics or cellular tissue in the neck may cause symptoms suggestive of laryngeal disease.

Finally morbid processes in the larynx determined by the symptoms and physical appearances may be symptomatic of the following general processes: acute inflammation, erysipelas, typhoid fever, smallpox, or measles; chronic inflammation or ulceration, of the rheumatic or gouty diathesis, syphilis, or tuberculosis; scars, of syphilis; anchylosis, of rheumatic gout. The laryngeal symptoms of brain disease and of affections of the nerve-trunk have been referred to.

The practical point of all this is that affections of the larynx are not due to *primary* disease of that organ alone, but are often *secondary* either to general processes or to local morbid processes elsewhere. Therefore when laryngeal symptoms or lesions are observed, seek beyond the larynx, as well as in it, for their cause.

Diagnosis. It is seen by the above that facts obtained in the *historical diagnosis* rather suggest the cause of the affection than the disease itself.

The *subjective symptoms* do not aid much in the differential diagnosis. The value of pain, of dyspnœa, dysphonia, and cough are considered in the chapters devoted to the discussion of these symptoms.

The *objective symptom* is of the greatest value and particularly enables one to distinguish the various forms of paralysis, ulcerations, and morbid growths.

The *laboratory diagnosis* aids us to recognize the various infections, especially diphtheria.

Perichondritis.

Inflammation about the cartilages, or perichondritis, is usually phlegmonous in character, and leads to the formation of abscess. The collateral œdema is so great as to cause some obstruction, with cough and hoarseness. On palpation the larynx is extremely tender. The pain is increased by movement of the larynx, as in speaking or swallowing. If the inflammation involves the arytenoid cartilages, pain extends toward the ear, the vestibule is swollen, and the cartilage fixed. On the other hand, when the cricoid is diseased, the swallowing of solid food is painful on account of interference with the muscular attachments, there is dyspnœa, and the posterior crico-arytenoid muscles are paralyzed.

Suppurative inflammation of the thyroid cartilage may rupture externally or internally. In the latter case the abscess can be seen in the larynx. The discharge of pus and necrosed cartilage confirms the diagnosis. The bare cartilage can be felt with a probe, giving further proof of the presence of the disease. The pain may extend to the ears in carcinoma; it is propagated by the auricular branches of the vagus.

Laryngismus Stridulus.

In this form of dyspnœa the act of breathing ceases midway in an inspiration, and is attended by a characteristic sound. The disorder is seen usually in poorly nourished children. It is of frequent occurrence in *rickets*, its presence suggesting that disease when other manifestations of it are obscure. The symptoms occur suddenly and are very alarming. The child awakes in the night, and after a few short whistling inspirations suddenly stops breathing. Terror is depicted on the countenance; the eyes stare; the face is pallid at first, but rapidly becomes livid. The *alæ nasi* are extended, the head is thrown back, and the spine arched. A cold perspiration breaks out over the forehead. Carpopedal spasms may occur and urine and feces be discharged involuntarily. In a few seconds, or, at most, two minutes, the child draws two or more deep, noisy inspirations, successively lessening in depth and sound, when color returns to the face, the cyanosis gradually disappears, and the child becomes tranquil. In mild forms the child "catches its breath." It holds its breath, and then makes a noisy inspiration.

Attacks of laryngismus stridulus are more rare in adults. They may occur in hysterical subjects. A series of long, harsh, whistling or stridulous inspirations, followed by short, noisy expirations, occur during the attack. Rarely is there complete closure of the glottis.

In both children and adults general convulsions may occur during the attack, or carpopedal spasms alone may be seen. In adults the convulsions occur only in hysterical subjects.

Diagnosis. The diagnosis of laryngismus stridulus is based upon the absence of laryngeal symptoms prior to the attack, the absence of cough or hoarseness, and complete disappearance of all laryngeal symptoms

when the attack subsides. The absence of pain and fever and of laryngoscopic signs is noteworthy. This applies, of course, to spasm occurring independently of laryngeal disease.

Inflammation of the Epiglottis.

When the epiglottis is the seat of acute inflammation, there is great dysphagia on account of pain or on account of the obstruction. The sensation of a lump in the throat at the base of the tongue or the top of the larynx is complained of, and there is pain on swallowing. The pain becomes very intense at times. Fluids can not be taken, for the fluid enters the larynx when the patient attempts to swallow, because the epiglottis fails to perform its function. The voice is usually clear throughout the attack, and the general symptoms are not marked.

When the epiglottis is fixed or ulcerated, as also in some forms of ulceration of the larynx, food enters the larynx and produces dysphagia.

Acute Laryngitis.

Acute laryngitis is an inflammation of the larynx characterized by a sensation of fulness and dryness, with cough, hoarseness, and at times dyspnœa. Several varieties are observed: (1) simple acute laryngitis, (2) laryngitis with great stenosis, (3) laryngitis with membrane (laryngeal diphtheria), (4) laryngitis with spasm.

Simple Acute Laryngitis. The causes are exposure to cold, the inhalation of acrid vapors. Over-strain, as in singers or excessive use of the voice, particularly in the cold air. It may be symptomatic of the eruptive fevers, as measles, smallpox, or of erysipelas. Its occurrence in the course of chronic diseases must be looked upon with alarm, particularly in cases of Bright's disease, if dropsy is present in other situations.

The attack begins with a feeling of chilliness, followed by *fever* of varying degree, but usually mild. The patient complains of a feeling of pressure and dryness in the larynx, or as if a foreign body were present. Some *pain* gradually develops at the height of the attack, but is never so severe as to require an anodyne. From the first there is *cough*, dry, hacking and slightly painful. In the more intense forms the cough is continuous, disturbing the patient night and day. Paroxysms occur when the patient speaks, or takes food. First the cough is dry; within a short time it becomes moist, and expectoration of clear, transparent mucus takes place. The mucus may be tinged with blood. At the end of forty-eight hours the expectoration becomes more yellowish and opaque. The voice may be merely hoarse, or may be lost entirely. Sometimes *aphonia* without general symptoms occurs in acute laryngitis.

In *laryngitis sicca*, cough and dyspnœa occur in paroxysms and are not relieved until a dry secretion is coughed up. The paroxysms take place at night or in the early morning, and may cause retching and vomiting. It is seen in adults.

Acute Laryngitis with Stenosis. No doubt some of the cases of so-called membranous croup in children are cases of acute laryngitis,

with swelling and occlusion of the glottis from congestion and the presence of tough secretion. Œdema may or may not be present. The attack begins with catarrhal symptoms. The child is languid, refuses to eat, is thirsty, and has some chilliness and rise of temperature. With the slight cough, which may be shrill, there are hoarseness and some difficulty in breathing, but no pain on swallowing. On the second day, or after the lapse of four or five days, during which time mild fever continues, the catarrhal symptoms become more marked. The voice is more hoarse or may be suppressed. The harsh, clanging cough becomes toneless, and soon the sound is suppressed. Dyspnœa is most severe, and the breathing is hurried and noisy, attended by loud whistling inspiration and snoring expiration. The stenosis is inspiratory, and may become very intense during the day or in the succeeding twenty-four hours. It is attended with violent efforts at breathing and the occurrence of cyanosis in its most aggravated form. The larynx moves up and down; the head is thrown back. There is recession at the root of the neck, along the margins of the ribs, and at the epigastrium. The lower portion of the sternum may be drawn in. Duskiness of the extremities and of the lips is observed as the stenosis becomes more marked, and finally deepens into cyanosis. It may be relieved from time to time by removal of the obstruction, by cough, vomiting, or change of position. Another paroxysm soon comes on, and with each paroxysm the lividity becomes more and more marked, the respirations continue hurried. The face becomes pale, the extremities cold, and a cold sweat bathes the brow. Restlessness is characteristic. The child tosses about in the bed or from the bed to the arms of the nurse. The heart's action increases in frequency and becomes weaker each hour as the stenosis advances. As exhaustion ensues and the symptoms of obstruction become more marked, stupor deepening into unconsciousness develops. Convulsions may occur at the end. The attacks rarely recur if the patient once recovers. They follow exposure to cold.

If recovery takes place, the dyspnœa gradually subsides, the cyanosis fades, and the restlessness disappears. Relief is followed by a prolonged sleep, but the voice may remain hoarse or suppressed, and the cough continue many days.

Laryngeal Diphtheria. The same symptoms are seen in membranous croup and laryngeal diphtheria. In the latter affection there may be a history of exposure or of infection. At the commencement of the attack the diphtheritic patches may be seen in the fauces or nares. If a membrane can be secured and a bacteriological examination made, the diagnosis of diphtheria with stenosis is positive. Enlarged glands in the neck, with marked physical depression, a moderate degree or entire absence of fever, and the occurrence of early albuminuria, also point to diphtheria. The distinction between the two affections is nevertheless quite difficult, and as long as there is a shadow of doubt, the case should, for prophylactic reasons, be considered one of diphtheria.

Acute Laryngitis, with Spasm—False Croup or Spasmodic Laryngitis. Another form of laryngitis associated with spasm of the larynx is seen in children. The catarrhal symptoms are mild, so that the child

seems to be well during the day. Fever is absent, and a slight cough or huskiness alone calls attention to the larynx. After the first three or four hours of quiet sleep the child suddenly awakes with a barking cough, sits up and struggles for breath. The dyspnœa continues from a few minutes to an hour or so, gradually lessening, to disappear entirely as the child lapses into sleep. Throughout the next day the child seems as well as on the previous day, and the succeeding night is seized with another attack of "croup." This may occur once or twice during the night. It seems to be influenced by the weather. Damp days and an east wind are provocative of an attack. It recurs frequently during the same season.

Œdema of the Larynx. This condition develops in the course of acute laryngitis, quite frequently in chronic diseases of the larynx, particularly if ulceration is present, and as a complication of erysipelas and diphtheria. In some cases of Bright's disease it may develop suddenly.

Symptoms of laryngeal stenosis may occur suddenly. The voice becomes husky and suppressed, the dyspnœa is very extreme so that in a few hours grave symptoms of obstruction arise. There is no cough. The patient complains of the sensation of a foreign body, and tries to grasp it.

Acute Submucous Laryngitis. The inflammation extends to the submucous cellular tissue. It arises in the course of acute laryngitis, and is the form seen in traumatism, or from burns and scalds. The symptoms are those of intense laryngitis, with stridor. They increase in severity until stenosis arises. If the lower surface of the cords is affected, death occurs from asphyxia. Sometimes the inflammation is circumscribed and is followed by development of an abscess.

The *chronic form of submucous inflammation* of the larynx is usually seen in drunkards, and is recognized usually by the laryngoscopic examination. The symptoms are those of slight stenosis.

Diagnosis. Acute affections of the larynx are distinguished from other diseases without much difficulty. To recognize the various forms of acute laryngitis, however, is not easy. In all there is a certain degree of laryngeal stenosis, and practically the question to be answered is, Which form of stenosis is present? The accompanying table shows the differential points of diagnosis. It is seen that the age, the occurrence of previous attacks, the character of the general symptoms, the history of previous laryngeal disease, the association of faucial disease, the presence or absence of membrane, and the results of laryngoscopic examination must be considered before making a positive diagnosis.

Simple Acute Laryngitis—"Catarrh of Larynx."	*Acute Laryngitis with Stenosis.*
Gradual onset of laryngitis, with dyspnœa very slight or absent.	Gradual onset of laryngitis, but dyspnœa develops to great severity.
All ages.	Children.
Fever of varying degree.	Fever of varying degree.
Dry irritating cough.	Dry cough, often paroxysmal.
May be hoarseness.	Hoarseness.
Pharynx reddened.	Pharynx reddened.
Gradual increase and decline.	Gradual increase, and either death of patient or decline of dyspnœa.
Larynx red and slightly swollen, as seen by laryngoscope.	Same, but swelling much greater.

Acute Laryngitis with Spasm—Spasmodic Croup.

May be slight hoarseness or cough, or none. Suddenly, in night, child awakes with intense dyspnœa and crowing inspiration.

Children.
Temporary high fever.
Slight brassy cough during day.
May be slight hoarseness in day. Very hoarse in attack.

Lasts a few minutes to one hour. May recur, or no attack until next night.
Slight redness, or nothing seen by laryngoscope.

Œdema of Larynx.

Some inflammatory disease of larynx exists.
Rapid development of dyspnœa, increasing to great severity.

All ages.
Depends on cause.
No cough.
No hoarseness.

Increases steadily to climax, then death, or decline of dyspnœa.

Epiglottis and aryteno-epiglottic folds swollen, pale, and waxy.

Foreign Bodies.

During eating or while holding object in mouth, sudden dyspnœa, varying in intensity according to object.

All ages.
No fever.
Irritative, expulsive cough.
May be hoarseness or not.

Cough persists till removal of body, or occasionally the larynx becomes accustomed to its presence, and cough ceases.
See the foreign body.

Laryngismus Stridulus—"Child-crowing."

No laryngitis. Sudden attacks of dyspnœa with crowing inspiration, either day or night. Very severe. May be general convulsions.
Children or hysterical adults.
No fever.
No cough.
No hoarseness.

Occurs often in rhachitic and hysterical cases.
Ends suddenly, in at most two minutes, and recurs often.
Nothing seen in larynx.

Membranous Laryngitis—Croup; Diphtheria.

Epidemic.
Gradually developing hoarseness and croupy cough, with low fever and lassitude, then development of dyspnœa, gradually and without intermission, as a rule.
Children.
Low fever and depression.
Croupy cough, later suppressed.
Very hoarse.
Fauces red and often with membrane; albuminuria; paralysis.
Increases steadily, broken by intense paroxysms. Either death or gradual improvement.
Red, swollen, with membrane.

Pertussis—Whooping-cough.

Epidemic.

Bronchitis, with cough developing in from one to three weeks. Then dyspnœa caused by severe paroxysms of coughing—absent between them,
Children.
Only the fever due to bronchitis.
Intense paroxysm of coughing.
No hoarseness.
Hemorrhages in various places from strain or emphysema.
May be death from exhaustion, or gradual improvement.

Nothing seen, unless slight laryngitis.

Chronic Laryngitis.

Chronic hoarseness may be due to chronic laryngitis. This affection either originates in an acute attack or comes on slowly. Prolonged use of the voice in a higher key than natural or in the open air, the abuse of alcohol, and constant exposure, are exciting causes. It may be symptomatic of syphilis or tuberculosis, and frequently results from inflammation of the upper air-passages, and particularly from chronic pharyngitis. It occurs more frequently after middle life, and usually in the male sex.

Prolonged or excessive use of the voice gives rise to discomfort with dryness and tickling in the throat. At first the secretion of mucus is very slight, but after hawking and coughing it increases in amount. Hoarseness occurs, and if the patient is careless or persists in the harmful occupation, complete aphonia may result. The voice is clearest in the morning, after expectoration of the nocturnal mucus, but becomes husky toward night. The aphonia may occur in paroxysms, and is relieved by coughing up a dry secretion. The cough is never severe. The sputum is small in amount, glairy, often in little balls or crusts.

Paralyses of the Laryngeal Muscles.

They are divided for convenience into groups. The *symptom* is dysphonia, which, with laryngoscopic appearances, leads to the recognition of the particular form of the paralysis.

1. Paralysis of the Tensors of the Cord. The cricothyroid muscle is paralyzed; the superior laryngeal nerve which supplies the muscle is affected. The *voice* is deep and rough, and incapable of producing high tones. Usually the whole nerve is involved, and the result is *anæsthesia* of the larynx and *paralysis of the epiglottis.*

Laryngeal Examination. The *epiglottis* is fixed, and falls back against the tongue. The *glottis* forms a wavy line.

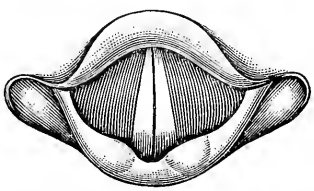
Causal Disease. The condition occurs almost exclusively after diphtheria.

2. Paralysis of the Closers of the Glottis, or Adductors of the Cords. The muscles involved are the crico-arytenoideus lateralis, arytenoideus transversus, and the thyro-arytenoideus internus and externus. The nerve is the recurrent laryngeal.

The symptoms are *complete aphonia*, coming on suddenly, and often disappearing as suddenly.

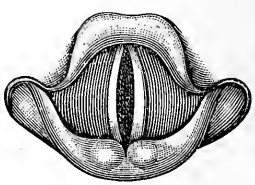
Laryngeal Examination. During phonation the cords remain in the inspiratory position. The paralysis may affect one or both sides.

Fig. 341.



Paralysis of the arytenoideus transversus in phonation. (Gottstein.)

Fig. 342.



Paralysis of the thyro-arytenoideus internus in phonation. (Gottstein.)

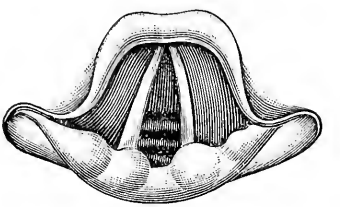
Sometimes the arytenoideus transversus alone is affected. Then there is hoarseness or aphonia. The anterior portions of the cords come together in phonation, but the posterior portions do not, leaving a triangular opening posteriorly. (See Fig. 341.) In other cases the thyro-arytenoideus internus alone is affected. There is then dysphonia or aphonia, as before, but the cords come together at both extremities and remain apart in the middle, forming an oval opening. (See Fig. 342.)

CAUSAL DISEASE. These paralyses occur in hysteria, catarrh, or severe over-strain of the voice.

3. Paralysis of the Openers of the Glottis, or Abductors of the Cords. The muscle affected is the crico-arytenoideus posticus; the nerve is the recurrent laryngeal.

SYMPTOMS. When one side is affected, the respiration is free, but there is stridor or forced inspiration. The voice is harsh.

FIG. 343.



Paralysis of the left recurrent nerve; inspiration. (GOTTSTEIN.)

LARYNGEAL EXAMINATION. One cord remains in the median line. (See Fig. 343.) When both sides are affected, inspiratory dyspnœa with stridor gradually develops. The voice is nearly normal. The glottis forms a narrow cleft which becomes still narrower on inspection.

4. Complete Paralysis of the Recurrent Laryngeal Nerve. **Unilateral Paralysis.** A weak, toneless voice which breaks into a falsetto when the patient endeavors to speak loud.

LARYNGEAL EXAMINATION. The cord and arytenoid body are in the cadaveric position, viz., half-way between the phonating and the inspiratory positions. In phonation the other cord passes beyond the median line, and the glottis is slanting. The edge of the paralyzed cord is excavated.

Bilateral Paralysis. Aphonia and inability to cough and expectorate.

LARYNGEAL EXAMINATION. Both cords are in the cadaveric position and their edges excavated. The adductors are usually paralyzed before the abductors, and one can see all the intermediate stages by close observation.

CAUSAL DISEASE. The conditions that give rise to the paralysis are numerous. It may arise from simple catarrh or from hysteria. More often it is due to pressure on the vagus or recurrent laryngeal, or some disease affecting these nerves or their roots.

The causes of pressure are: aneurism of the subclavian or aorta, mediastinal tumor, tuberculous bronchial glands, the apex of a tuberculous lung, cancer of the œsophagus, goitre, and carcinoma of the pleura.

The diseases are: diphtheria, tumor, softening or hemorrhage into the brain, bulbar paralysis, neuritis, typhus, cholera, variola, articular rheumatism, toxæmia (?), sclerosis of the cord, progressive muscular atrophy, and paralytic dementia.

Tumors of the Larynx.

Both benign and malignant growths are seen. At first *dysphonia* or *aphonia* takes place. The impairment of voice may continue for a long period of time before *dyspnœa* arises. This develops very gradually, and in some few cases is attended by an irritative *cough*. The general symptoms are not marked in benign cases. In the malignant forms they are pronounced, but characterized by the development of cachexia later than in carcinoma elsewhere.

The *diagnosis* of malignant disease of the larynx is based upon the association of symptoms of laryngeal disease with pain, and with the characteristic appearances found on inspection, on its occurring after the middle period of life and lasting only from six to nine months with the development of cachexia and emaciation without fever. Enlargement of the cervical glands points to cancer. Simple and syphilitic perichondritis must be excluded.

Tuberculosis of the Larynx.

The occurrence of primary laryngeal tuberculosis is doubtful. It can not be proved clinically, and the majority of cases at least are secondary to tuberculosis of the lungs. The manifestations of tuberculosis of the larynx may be either a simple persistent catarrh, an infiltration, or an ulceration. (See pages 443 and 444.) The symptoms vary according to the lesion:

a. **Catarrh.** There is a slight hoarseness and the voice tires easily. Often paræsthesia or peculiar sensations in the larynx are present. Cough, when due to this alone and not to the process in the lungs, is short and dry.

b. **Infiltration.** At first the symptoms are those of simple catarrh; then the alteration of the voice increases even to aphonia; there is a feeling of dryness or soreness in the larynx, with dysphagia. The cough is very slight and is usually wholly disguised by the cough due to the disease in the lungs. There is some difficulty in expectoration.

c. **Ulceration.** The symptoms are the same as those of infiltration, but the dysphagia and pain are greater.

Diagnosis. Tuberculous ulcer occurs most frequently in the male sex, and during the period ranging from eighteen to thirty years of age. If the symptoms develop in the course of phthisis, or in case that affection can not be recognized, if there is a history of infection or exposure, and if bacilli are found in the sputum, the diagnosis is not difficult. A portion of the diseased mass may be removed for microscopical examination or inoculation. In examining the secretion for tubercle bacilli, it is to be remembered that the exudate may have been brought up from the lungs. The examination in cases of phthisis is of little practical value, except to determine whether the ulceration present may be syphilitic and grafted upon a tuberculous disease of the lungs. Enlargement of the glands of the neck is often present, but is not diagnostic.

Fever is present, and, indeed, may be an important diagnostic feature in doubtful cases. The temperature should be taken every two hours,

for the morning or evening exacerbations may not be present. Emaciation ensues, and sooner or later the hectic phenomena and signs of tuberculosis in other structures arise. When tuberculous ulceration of the larynx occurs in the course of local pulmonary tuberculosis, the disease runs a much more rapid course.

The *laryngeal symptoms* are not diagnostic. Pain may be the most distinct. The laryngoscopic findings are more characteristic. Local anæmia with paræsthesia, paresis of the cords, and short cough, and an obstinate diffuse catarrh are suspicious symptoms. The peculiar ridged infiltration between the arytenoids is almost invariably tuberculous.

Isolated thickenings anywhere in the larynx, tapering gradually into the normal tissue, can only be tuberculous or syphilitic. The regularity and number of the lesions, with anæmia and absence of inflammatory signs, usually suffice to distinguish the tuberculous from the syphilitic form. The ulcers are non-erosive. Syphilitic ulcers do not often occur anywhere but on the edge and lingual side of the epiglottis and on the cords. They extend more rapidly than the tuberculous, and may be continuous with ulceration in the pharynx. The area of ulceration may extend to the base of the tongue, which is very frequent in tuberculous disease. In syphilitic ulceration scars or cicatrices are seen, but they are absent in the tuberculous form. Laryngoscopic examination in tuberculous ulceration is difficult, as it causes great pain; in syphilis comparatively little pain attends examination. (See the Infections.)

Syphilitic Affections of the Larynx.

Mucous patches, papules, infiltrations, or gummata may be present in the larynx for some time without causing symptoms. Usually a change in the voice is the first symptom noticed, due either to the catarrh or to ulcers, scars, infiltrations, or gummata affecting the cords. There is often a feeling of pressure or a tickling sensation. Pain is not usual, and when present is very slight. Dysphagia occurs only when the epiglottis is extensively ulcerated. There is little or no cough.

The *diagnosis* rests upon the history of infection, the objective signs of syphilis indicated by pigmentation or recent eruption, scars, periostitis or nodes on the bone, and enlarged glands. The laryngeal symptoms are not diagnostic, save that pain is absent in spite of extensive ulceration, while difficulty of deglutition, on account of food entering the larynx, is of frequent occurrence. The laryngoscopic appearances, as indicated above, are characteristic. In obscure cases the distinctions spoken of in tuberculosis are of diagnostic value.

Although the patient may be broken down and cachectic, the febrile range is not high, unless perichondritis occurs, or pneumonia sets in on account of food in the air-passages.

The Larynx in Other Diseases.

Laryngeal symptoms due to lesions of the nervous system are found under the following circumstances. (See Cerebral Localization.)

Cerebral Hemorrhage. 1. **Aphasia.** The movement of the muscles is normal, but they can not be controlled by the will. It is caused by hemorrhage in the cortex or along the course of connective fibres.

2. **Recurrent paralysis.** Due to hemorrhage in the medulla.

3. **Symptoms of bulbar paralysis.** Same cause.

Encephalomalacia.. (Softening.) When in the brain, aphasias result; when in the medulla, bulbar symptoms.

Tumors of Cerebrum. The symptoms are, according to location, aphonia, aphasia, or paralysis of the cords.

Bulbar Paralysis. We have, of course, the other symptoms of the disease. The voice becomes weak and monotonous, devoid of modulation. High tones are impossible. The disturbance progresses to hoarseness and finally aphonia. Particles of food and drink enter the larynx. Paresis or paralysis of the cords results.

Multiple Sclerosis. The speech is low, uncertain and scanning, later hoarse. Laughing and crying are accompanied by peculiar yawning inspirations. On laryngoscopical examination slight paresis of the cords is seen.

Posterior Sclerosis (*Tabes*). The muscles act very slowly. Sometimes symptoms of irritation, as tickling or burning in the larynx, with a dry cough, occasionally severe paroxysms of coughing, even to spasm of the larynx, occur—"laryngeal crises." In rare cases a phonetic spasm has been observed. Less often paresis or paralyses of the various muscles occur, most frequently the posticus, next the recurrent. Sensibility may or may not be disturbed.

Amyotrophic Lateral Sclerosis. There is a mixture of bulbar with spinal symptoms. (See Sclerosis.)

Progressive Muscular Atrophy. The same mixture of symptoms occurs very late.

Paralytic Dementia. There may be disturbances in articulation, with paresis and paralysis of the cords.

Chorea. There may be a tremor of the cords from under-tension, but probably no true choreic movements.

CHAPTER VI.

DISEASES OF THE LUNGS AND PLEURA.

Pathology. Disease of the lungs may be due to alterations in (*a*) the connective tissue, (*b*) the channels, (*c*) the muscles, and (*d*) the nervous mechanism. The pleura is subject to diseases common to serous membranes.

Alterations in the Connective Tissue. The connective tissue, composing the structure of the lungs independently of the canals, although liable to the same morbid processes that affect it in other situations, is not often subjected to irritants that cause acute inflammation. Its chronic inflammations, in the large majority of cases, occur secondarily to processes in the channels.

Alterations in the Channels. Diseases of the lungs are usually diseases of their channels, due to morbid alterations of them (1) by processes common to the structure of such channels and (2) by obstruction of them.

1. Morbid Processes of the Channels and the Symptoms Produced. The *air-tubes* are lined with mucous membrane, which is subject to the morbid processes that attack such lining: congestion or acute and chronic inflammation. A flux is the characteristic symptom. The muscle and the elastic tissue of the canal become involved in the process. The former undergoes spasm, with or without inflammation of the mucous membrane (asthma). Grave consequences do not arise until degeneration takes place, with loss of the power of confining the air or driving it out and resulting emphysema. In the *blood-canal*, *hyperæmia* (congestion), *embolism* and *thrombosis*, and secondary *œdema* may take place; in the *lymph-channels*, *inflammation* (acute and chronic pleurisy), and *transudation* (hydrothorax or hæmothorax) occur. The *symptoms* that arise in each or all of the above processes—pain, local discomfort, mucous or purulent discharge, serous or purulent exudation and fever—are not different from those which are found in diseases of similar tissues in other localities. (Compare with affections of mucous or serous membranes in other organs.)

2. Obstruction of the Channels and the Symptoms Produced. In addition to these, there is a group of symptoms due to obstruction of the various channels, causing interference with the function of the lungs. The symptoms are purely mechanical. Obstruction of either the *bronchial tubes* or *bloodvessels* gives rise to *dyspnœa*. This is as pronounced in asthma or capillary bronchitis as it is in embolic obstruction (fat-embolism) or congestion of and stasis in the bloodvessels. It occurs when the canals are occluded by extrinsic causes—the presence of foreign bodies in the bronchi or the presence of pleural effusions. As a sequence we

have another vivid picture—the development of *cyanosis* from interference with aëration.

Alterations in the Muscles and the Symptoms Produced. *Inactivity* of the muscles, from *pain*, from *debility*, or from *paralysis* through any cause practically occludes the canals by lessening the movement of their normal contents. The amount of air therefore is diminished and dyspnœa results.

Alterations in the Nervous Mechanism and the Symptoms Produced. Various forms may exist. (*a*) A *central affection*, causing pulmonic symptoms from the following causes: 1. The influence of the higher centres upon the lower pulmonary centre, as seen in hysterical cough, or emotional cough, and in asthma—*respiratory neuroses*. 2. *Disease* affecting the region of the centre, as in *tumor* or in *bulbar* or glosso-labio-laryngeal *paralysis*. 3. *Irritants* acting upon the centre, as urea, exciting uræmic asthma. (*b*) An *affection* of the *nerve-trunk*, as from the pressure of an aneurism or morbid growth. (*c*) *Reflex influences* through the pneumogastric and correlated nerves. The asthma of nasal disease, or of peripheral irritation, and reflex cough (neuroses) are of this nature.

Differential Diagnosis. It is not usually difficult to distinguish diseases of the lungs from affections of other structures. Pleurisy and pleurodynia, it is true, are often differentiated with difficulty. We may be called upon to decide between pleurisy and subdiaphragmatic inflammation, between a pleural and an hepatic inflammation, between a pleuritis and a pericarditis and between cardiac and pulmonary disease, especially when in the presence of both conditions we desire to determine which is primary.

Chronic affections of the lungs, of the mediastinum, and of the great vessels must be distinguished from one another; an aneurism or mediastinal disease, from chronic phthisis. With care in ascertaining the history and the subjective and objective symptoms the distinction may not be difficult.

It often happens in pulmonary disease that some of the pronounced symptoms may point strongly to an infection other than that of the lungs; thus the cerebral symptoms of pneumonia may be attributed to meningitis, or the fever present thought to be due to typhoid fever. On the other hand, the presence of a pulmonary affection, as tuberculosis, may explain the nature of a morbid process in other organs or structures. Hence, in all cases in which there is a possibility of secondary tuberculosis the lungs should be examined in order to determine if they are the seat of the primary disease. In this way the true nature of a meningitis, of a peritonitis, or of another tubercular affection may be recognized. In secondary anæmia and in protracted debility of unknown source the lungs always should be examined. It must be borne in mind that in chronic diseases, such as chronic renal disease, chronic arthritis, diabetes, etc., pulmonary tuberculosis may set in most insidiously. In the same diseases, pneumonia frequently is a terminal infection, and likewise runs an insidious course. In the extremes of life pulmonary infections, such as pneumonia, present symptoms out of the usual run. In infancy and childhood the cerebral symptoms may mask the pulmonary

symptoms; in senility the absence of cough or expectoration may lead to the dismissal of all thought of pulmonary disease. In short, the lungs should be examined in all affections.

This injunction is to be observed particularly inasmuch as lung diseases are often secondary to other diseases: phthisis to tuberculosis elsewhere: pneumonia or pleurisy to all infectious disorders, to Bright's disease, cancer, and diabetes. The possibility of a hydrothorax, secondary to causes of transudation, must always be borne in mind.

Relative Value of Subjective and Objective Symptoms. The subjective symptoms are few, and are common to so many pulmonary diseases that they are of little diagnostic value. The objective symptoms obtained by applying the laws of physics to the lungs are of greater service. The effect of the occlusion of the channels is mechanical or physical; hence a physical change in the lung follows. The objective symptoms occur when the air-movement is checked and no sounds are heard; when abnormal breath-sounds and new sounds (râles) are created; and when air is replaced by solid structure. The objective signs of these conditions are determined by inspection, palpation, percussion, and auscultation.

DISEASES OF THE LUNGS AND BRONCHI.

Classification. Affections of the lungs may be divided into (*A*) the neuroses, (*B*) the congestions, (*C*) the inflammations, (*D*) the degenerations, (*E*) the morbid growths, and (*F*) those due to gross parasitic invasion. Influences operating through the pneumogastric and phrenic nerves may be responsible for respiratory neuroses. The congestions are so intimately associated with vascular phenomena that the latter may be included in the process. The inflammations are limited to the bronchi, to the alveoli, and to the connective tissues surrounding both. The intimate relation of the small bronchi, the alveoli, and their surrounding connective tissues implies their conjoint involvement in many processes.

A. The Neuroses.
B. The Congestions.
   1. Active, including *hemorrhagic* infarct.
   2. Passive.
      Subsidiary: *hemorrhage.*
C. The Inflammations, chiefly *infections.*
   1. The Bronchi.
      Acute.
      Chronic.
   2. Bronchi and alveoli.
      Bronchopneumonia (an infection).
   3. Bronchi, alveoli, and connective tissue.
      Pneumonia.
      Tuberculosis.
      Abscess of the lung.
      Gangrene.
      Chronic interstitial pneumonia—pneumonokoniosis.
      Syphilis of the lung.

D. The Degenerations.
   Emphysema.
   Bronchial dilatation.
E. Morbid Growths.
F. Gross Parasites.
   Hydatid disease.

THE NEUROSES.

The neuroses are affections of the lungs unattended by structural change. To this class belong the varieties of rapid breathing, of slow breathing, of cough and of dyspnœa which appear to arise without structural change, and which are discussed exhaustively in the section devoted to the subjective symptoms. Of the neuroses, *asthma* is the most common form. Reference need not be made further to the respiratory neuroses, but it should be borne in mind that their presence may or may not be unattended by organic change in the lungs. On the other hand, we are likely to find the general phenomena or stigmata which are associated with neuroses of other organs, as well as the lungs. Hence, the condition of neurasthenia is likely to be present on the one hand, or the numerous stigmata of hysteria may be found on the other.

Asthma.

Asthma is a chronic disease caused by spasmodic narrowing of the bronchial tubes, and characterized by paroxysmal attacks of dyspnœa, diminished respiratory movement of the chest, and prolonged expiration, attended by a wheezing sound and sibilant râles, and which end abruptly with the expectoration of tenacious mucus. The attack may be limited to a single night, or may be prolonged for days, with nocturnal exacerbations.

Symptoms. Premonitory symptoms are said to occur in about one-half the cases. These are for the most part nervous, such as headache, neuralgia, irritability of temper, vertigo, and drowsiness. Hyde Salter found that there were premonitory symptoms in 111 out of 226 cases collected by him. In 63 they were nervous, in 8 they consisted of profuse diuresis, and in 14 they were connected with the digestive system.

The attack itself usually begins during sleep, and often at a regular time. It may, however, begin during the day, and at a certain hour, independently of sleep. The onset is manifested by tightness across the chest and more or less difficulty in breathing. This dyspnœa increases rapidly and often reaches an extreme degree. The face becomes pale and anxious, and may be covered with a cold perspiration; the lips are dusky from insufficient oxygenation of the blood. The patient feels smothered, and makes frantic efforts to get his breath, rushing to an open window, no matter how cold the weather, or, if unable to leave the bed, sitting up with the hands pressed upon the bed so as to give purchase to the accessory muscles of respiration. Notwithstanding the great respiratory efforts made, the chest moves but little, because the lungs are already distended to the extent of a full inspiration. The patient is unable to expel the contained air, owing to the spasm of the bronchial tubes.

The frequency of respiration is diminished, sometimes to one-half the normal; the rhythm is also altered, inspiration being short and gasping, and followed without pause by expiration, which is much prolonged and accompanied by a wheezing sound audible to bystanders.

There is an increased amount of air in the thorax, and inability to remove it. The duration of an attack of asthma varies from half an hour to a day or two. In patients with chronic bronchitis it may be prolonged for a week or two, with remissions during the day. It may subside abruptly or by degrees.

Subsidence of an attack is marked by expectoration, the sputa having special characteristics. (See under Sputum.) At first it is made up of rounded gelatinous masses, which when unfolded in water are seen to be made of spirals. Later it becomes mucopurulent. Curschmann's spirals and the Charcot-Leyden crystals are nearly always found. The leucocytes are increased, and 25 per cent. of them are eosinophiles.

The causative factors in asthma are various. About twice as many males as females are affected, and there is a marked hereditary tendency in some families. There is probably some special peculiarity in asthmatic patients, but just what it is, has not been determined. It may reside in the lungs, and may be part of a general constitutional irritability. (Salter.) Bronchitis, emphysema, and heart disease act as causes, and also syphilis, malarial poisoning, and chronic Bright's disease.

The above description applies to spasmodic asthma, the dyspnœa up to this time having been paroxysmal. Sooner or later it becomes constant, and then other changes take place in the lungs. First, there is persistent bronchitis; second, the presence of emphysema. Indeed in many cases it is difficult to ascertain the exact sequence of affections. In emphysema of the lungs, dyspnœa is constant, but on exposure to cold or on account of an attack of indigestion more severe paroxysms may occur, as well as asthmatic attacks, although the patient is not an asthmatic. On the other hand, a patient may have had asthma for a number of years, during which attacks of dyspnœa occurred only in paroxysms. As time passes, the paroxysms become more and more frequent, and emphysema develops. With the advent of emphysema the dyspnœa becomes more constant.

Physical Signs. On *inspection* the chest is enlarged, barrel-shaped, its movement is lessened and strikingly out of proportion to the muscular exertions. The diaphragm is lowered.

On *percussion* hyper-resonance is elicited; on *auscultation* expiration is faint and short, and inspiration is prolonged, and sibilant and sonorous râles are heard, more marked on expiration.

THE CONGESTIONS.

Active Congestion of the Lungs.

In active congestion there is an increased amount of blood, which diminishes the air-space by encroachment and causes more or less consolidation.

Symptoms. Dyspnœa, cough, and frothy, bloody expectoration attend

the fluxion. No cases have yet been reported in which bacteriological examination of the sputum was made. Of course, *Micrococcus lanceolatus* is not found in the sputum.

Physical Signs. Increased fremitus, impaired resonance or dulness, and bronchial breathing are present. They are observed on both sides, usually at the bases.

Diagnosis. If the above signs and symptoms develop suddenly—within twenty-four hours—a fluxion to the lung has in all probability taken place. If the patient is subject to heart disease, or if he has been exposed to and has inhaled hot vapors or irritants, the probability of fluxion is increased. The occurrence of fever would point to pneumonia as the cause of the objective and subjective symptoms.

Passive Congestion of the Lungs.

Mechanical congestion occurs when the flow of blood to the heart is obstructed, as in organic valvular disease or insufficiency. Rarely the pressure of tumors on the pulmonary veins acts in a similar manner.

Hypostatic congestion occurs in fevers, as protracted typhoid, and in prolonged general exhaustion or adynamia. Ascites or other affections below the diaphragm, which lessen the respiratory excursion, cause this form.

Symptoms. Dyspnœa, cough, and expectoration of blood-stained sputum are common. The sputum contains alveolar cells, often pigmented, but no micro-organisms.

Physical Signs. Consolidation is present, manifesting itself by slight dulness and feeble or bronchial breathing; the bronchial mucous membrane is also congested, giving rise to abundant large râles. The affection is bilateral and usually confined to the posterior portions of the bases.

Œdema of the Lungs.

The air-cells and alveolar walls are filled with serous exudation, as in œdema of the skin. The condition is frequently due to the weakness of the heart, which occurs at the end of long-continued diseases of an exhaustive nature, particularly if the heart is over-taxed. It occurs, therefore, in the terminal stages of chronic Bright's disease, of organic heart disease, of the anæmias and cachexias. Both congestion and œdema occur in cerebral affections.

Symptoms. They are those of congestion in a more aggravated form. Dyspnœa, cough, and the expectoration of large quantities of a seromucoid fluid are seen. In cases of myocarditis or acute dilatation of the heart, in valvulitis with failing compensation, œdema of the lungs often takes place suddenly. It may follow some unusual exertion. Its onset is attended with more or less collapse, increased pulse-rate, hurried, oppressed, noisy breathing, cyanosis, and an anxious expression.

Physical Signs. These are an unusual number of small râles throughout the chest, apparently created in the air-sacs, and imperfect resonance, showing that some lobules are collapsed.

Pulmonary Embolism and Thrombosis.

Pulmonary embolism consists in plugging of the pulmonary artery or its branches by coagula formed in the right heart or in the veins. The symptoms depend upon the size of the occluded vessel and upon the nature of the embolus—*i. e.*, whether septic or not. If the artery itself is plugged, death takes place suddenly or after a short interval, with symptoms of syncope or asphyxia.

Plugging of a Large Branch. **Symptoms.** The first symptom is generally intense dyspnœa, which may amount to an agonizing craving for air. Pain in the chest, which may or may not be acute, is complained of, and may be referred to the seat of the embolus. Cough is not a common symptom, and may be altogether absent. The breathing is considerably altered; it is usually increased in frequency, and may be much hurried; it may or may not be shallow, and while the patient can take a deep inspiration, it does not give relief to his dyspnœa. At times it is irregular and gasping.

The face is pale or may be cyanosed, and is apt to be bathed in perspiration. The veins are swollen and prominent. The heart's action is irregular and may be tumultuous. Exophthalmos has been observed. The temperature falls below normal, but a febrile rise may occur later. The intellect is unclouded.

Physical Signs. These are indefinite. The respiratory murmur is roughened and exaggerated in most, but not in all cases. Fox states that râles are very rarely heard. Collapse, œdema, and bronchitis are possible results. A systolic blowing murmur may be heard over the heart and pulmonary artery, and in protracted cases albuminuria and œdema may be met with.

Septic Embolus. When the embolus is septic, a *septic pneumonia* or *metastatic abscesses* are probable results in cases not immediately fatal.

Hemorrhagic Infarcts. **Symptoms.** When the emboli produce hemorrhagic infarcts, the symptoms are milder, and consist principally in dyspnœa, pulmonary hemorrhage, and palpitation. The onset is sudden and accompanied by a fall in temperature.

Physical Signs. These indicate consolidation, if the pneumonia or infarcted area is of moderate size. The consolidation may be discovered at the root of the lungs in the interscapular region.

Cardiac Embolus. Hæmoptysis is a common symptom when the embolus has arisen in the heart. The amount of blood varies from a copious expectoration to the rusty sputum seen in pneumonia; it may persist for weeks. Pleurisy and pleural effusion are frequent complications; chills occur sometimes, and pneumonia, with corresponding rise of temperature, may develop.

The most important points in diagnosis are the sudden onset of the dyspnœa and other pulmonary symptoms, and the detection of a condition which would give rise to emboli, such as puerperal fever or heart disease.

THE INFLAMMATIONS.

Inflammations of the bronchi are distinguished from other diseases of the lungs chiefly by the difference in the physical signs. Except in capillary bronchitis, the general and subjective symptoms are not so severe as in other affections.

We are aided in the recognition of bronchial affections, first, by the fact that they are bilateral; second, that the bases are usually affected; third, that there is diminution of fremitus determined by palpation; fourth, that there is absence of dulness on percussion; fifth, that râles are more pronounced in proportion to other physical signs, and more general than in other lung affections.

Acute Bronchitis.

This is an acute inflammation of the mucous membrane of the larger or middle-sized bronchial tubes. It occurs most frequently by extension of the catarrhal inflammation from the nose and throat; but in some persons it develops so suddenly that it appears to be primary in the tubes. The duration of the disease is from a few days to several weeks. It is never fatal except in the very old and very young, or in those who are much debilitated.

Symptoms. The patient complains of soreness or rawness underneath the sternum, especially at its upper part. There are frequently a feeling of tickling in the throat, and a sense of weight or oppression in the chest. Chest pain is due to myalgia or the strain upon the muscles from coughing. The *cough* is at first hard and dry, and often produces pain of a tearing character in the muscles of the chest and abdomen. The cough is apt to be worse when the patient first lies down, and again on rising, especially after a night's rest. *Fever* is usually slight and of short duration. The respirations are accelerated, but not markedly, and there is no dyspnœa. The *expectoration* is at first a white, frothy, viscid mucus, subsequently becoming more abundant and mucopurulent. The cough and expectoration usually last for some time after fever has subsided.

Physical Signs. In uncomplicated cases there are no changes in the physical structure of the lungs. On examination of the chest the *percussion-note* is found to be clear; the *respiratory murmur* more roughened and harsher than normal, but not bronchovesicular or bronchial; accompanying breathing there are heard sibilant and sonorous *râles*, and in the later stages some large and medium-sized mucous râles. The râles vary in position from time to time, and especially after coughing. *Vocal resonance* and *fremitus* are unaltered. A *rhonchal fremitus* may be produced by sonorous râles.

Diagnosis. The diagnosis of acute bronchitis is easily made by noting the fact that the disease runs an acute course marked by fever, cough, and expectoration; and that the physical signs are negative, except as to roughening of the respiratory murmur and the existence of bronchial râles, heard on both sides of the chest.

From *croupous pneumonia* and *chronic local tuberculosis* of the lungs,

acute bronchitis is distinguished by the absence of dulness on percussion, of bronchial breathing, and of increase in the vocal resonance and fremitus; by the absence, in other words, of the ordinary signs of consolidation. From pneumonia it is further distinguished by the milder character of the subjective symptoms, and by the fact that physical signs are almost always bilateral, while in pneumonia they generally are unilateral. Tuberculosis is further distinguished by its slow progress and by its involvement of the apices preferably, whereas bronchitis is more marked at the bases, and by the occurrence, sooner or later, of hectic fever and emaciation, which are absent in bronchitis. Doubt will exist only at first; the progress of the case will in time make everything clear. Systematic examination of the sputum is an important diagnostic aid, and will lead to the differentiation of many cases of bronchitis from tuberculosis and from pneumonia. In infants and children especially, bronchitis is at times so rebellious to treatment that tuberculosis is suspected.

In *bronchopneumonia* (catarrhal pneumonia) there is a diffuse bronchitis associated with small areas of pneumonic consolidation. Bronchopneumonia is distinguished by having graver general symptoms and by the presence of small areas over which there are dulness on percussion and bronchial breathing, associated with the physical signs of bronchitis already noted.

Acute miliary tuberculosis of the lungs is very easily mistaken for bronchitis, because dulness, if present, amounts to nothing more than tympanitic dulness, because the signs are diffused through both lungs, and because the respiratory murmur is fainter than normal and only slightly roughened. Close inspection of the patient will, however, make it evident that his condition is worse than could be accounted for by bronchitis alone. The fever is higher, the respirations are more frequent, pallor, with a dusky or faintly cyanotic hue intermingled, is common, and perspiration is more pronounced. A primary focus or a source of infection may be discovered.

Acute bronchitis may be mistaken for *spasmodic laryngitis* (croup). It is distinguished by the fact that the spasms are less pronounced in bronchitis, and there is fever in addition to the physical signs. In bronchitis the breathing is rarely so stridulous as in laryngeal spasm.

Whooping-cough can not be distinguished positively from bronchitis before the characteristic whoop appears; but it may be suspected when the child has been exposed to contagion, and when the coryza and redness of the fauces persist in spite of treatment.

In the diagnosis of bronchitis it is often more difficult to determine the primary cause than it is to distinguish bronchitis from other pulmonary affections. Yet the former is the more important; and it must be borne in mind that bronchitis is a frequent accompaniment of many febrile diseases, such as typhoid fever, measles, and whooping-cough; of diseases of the heart and kidneys, and of septic diseases and blood disorders. The primary will not be likely to be mistaken for the secondary disorder if one is upon his guard and insists upon finding a cause for each case that presents itself.

Measles can usually be diagnosticated from the first by the coryza, but

especially by the Koplik spots and the red spots upon the anterior half-arches of the soft palate, which appear usually several days before the eruption upon the body.

Bronchitis is a common and important early symptom of *typhoid fever*. The latter disease may be suspected when the fever, prostration, and headache are greater than they would be in bronchitis alone, and, especially if these symptoms coexist with a loose condition of the bowels, chilliness, and occasional nose-bleed.

Chronic Bronchitis.

Symptoms. This occurs most frequently in middle or later life. Its special feature is long duration, without fever and with comparatively little impairment of the general health. *Cough* is not constant; it is entirely absent for periods; it then returns, perhaps with increased severity, and lingers indefinitely.

Chronic bronchitis in its milder form consists in what is often called "winter cough." It attacks especially persons past middle life who have emphysema. It appears with the cold weather, and lasts until the following summer. The cough is not severe, though sometimes paroxysmal, and *expectoration* is scanty, non-purulent, and may be confined to the morning. Dyspnœa is not marked unless there is considerable emphysema. Acute exacerbations occur from time to time, and the tendency of the disease is to become worse from year to year, and to be more continuous, even persisting all summer.

In *dry catarrh*, or *catarrhe sec* of Laennec, paroxysms of cough occur on the slightest provocation, with the expectoration of small hard pellets, or without any expectoration. The patients with this form are emphysematous.

Physical Signs. These are those of bronchitis of the larger and middle-sized tubes. Large moist *râles* are more or less abundant, depending upon the degree of swelling of the mucous membrane, and the quantity and fluidity of the secretions. The *respiratory murmur* is roughened and less intense than normal.

W. Fox says that in chronic bronchitis there is commonly *hyper-resonance* from coexisting emphysema, but in acute exacerbations the bases may be dull from congestion or œdema. *Respiration* is harsh, and in some cases of senile bronchitis expiration may be both prolonged and high pitched when other signs of dilatation of bronchial tubes are absent. The *percussion-note* is clear.

The subjective symptoms of the patient consist, in ordinary cases, of a moderate amount of *dyspnœa* and *tightness across the chest*. At the onset of a fresh attack the symptoms may be those of acute bronchitis. The cough is paroxysmal, somewhat resembling that of whooping-cough, but without the characteristic whoop. It is usually severest on lying down and when rising in the morning.

The *sputa* of the severe forms of chronic bronchitis are usually copious and mucopurulent, the latter predominating. They vary in color from yellowish white to ashy, greenish, or black when the lungs are anthracotic

or collapsed. The quantity and character of the sputa vary more than in acute bronchitis. Sometimes they are very copious, consisting of serum mixed with mucus, constituting *bronchorrhœa*. More commonly they are scanty, glairy, and tenacious.

Diagnosis. This is made by noting the long duration of the disease without impairment of the general health, its relation to season, and the absence of physical signs of involvement of lung tissue.

Chronic bronchitis may be the result of repeated acute attacks, or, rarely, of only one. It is frequently found in association with gout, chronic heart disease, chronic endarteritis, Bright's disease, emphysema, asthma, and chronic alcoholism. It may alternate with other gouty affections, as articular inflammation or eczema, being relieved when the other manifestations are more marked. It also accompanies tuberculosis of the lungs. Climate and season have a marked influence; the disease is worse in damp, cold climates and in the winter months.

Chronic bronchitis can be diagnosticated from the cough of *aneurism* by the absence of the stridulous breathing, due to paralysis of one-half of the vocal cords, and by the local signs of a tumor of the vessel. Other *tumors* may cause cough by pressure, and the possibility of their existence should therefore be borne in mind.

Capillary Bronchitis, or Suffocative Catarrh.

This is bronchitis of the smaller tubes. It occurs most frequently as an extension of the catarrhal process from the larger tubes, but sometimes seems to attack the smaller tubes from the beginning, or coincidently with the larger tubes. Infants, young children, and the aged are most liable to it.

Symptoms. The disease begins with a succession of chills or chilliness, followed by high fever. The temperature may rise to 104° F. The skin is hot, the face flushed. The head and neck and the upper portion of the trunk may be covered with perspiration. The pulse rapidly increases in frequency.

The aspect of the patient from the first shows that the illness is graver than ordinary bronchitis. The face expresses anxiety, and in children the *alæ nasi* dilate in respiration, which is both accelerated and difficult (*dyspnœa*). The respirations may be as many as 80 to the minute, the pulse not being correspondingly rapid. Dyspnœa is more or less constant, but becomes urgent in paroxysms, and the patient may have to be propped up in bed to enable him to breathe (*orthopnœa*). It is *expiratory:* inspiration may be free and easy, or difficult; but expiration is always difficult and prolonged. In children the pause in the act of breathing takes place at the end of inspiration, instead of expiration.

Cough is more frequent and violent than in ordinary bronchitis, and the *expectoration* is viscid and difficult to raise. As the disease progresses, dyspnœa becomes more intense, and signs of insufficient aëration of the blood make their appearance (*cyanosis*). The lips and finger-nails become bluish, and the extremities cool and clammy. If the patient is unable to expel the tenacious secretions from his bronchial tubes, the further

progress of the case is that of rapidly developing cyanosis; the breathing continues frequent, but is shallow and more labored. Children often have convulsions, followed by coma and death, while old persons sink into coma without preceding convulsions.

The *sputum* contains mucus, pus, occasionally blood-cells, granular matter, and sometimes fibrinous casts of the tubes. The micro-organisms found are *Micrococcus lanceolatus*, *Streptococcus pyogenes*, and *Staphylococcus aureus et albus*. Mixed infections are usually present.

Physical Signs (Plate XIX.). These are those of bronchitis of the larger and smaller tubes. Sibilant and sonorous *râles*, if present at first, give way to fine subcrepitant and crepitant râles, which speedily become moist and very abundant. As in ordinary bronchitis, the bases of the lungs posteriorly are the parts most involved. The *percussion-note* of both lungs remains clear, but there is apt to be *increased resistance*. The *fremitus* may be lessened in some areas, increased in others. If an area of *dulness* appears, it may be due to pneumonia or collapse of the lung; if the former, there is usually an access of fever.

Plastic Bronchitis.

This is a form of bronchitis, usually chronic, the characteristic feature of which is the expectoration of fibrinous casts, which, when unravelled under water, are found to be solid casts of the smaller bronchial tubes. The casts are often tree-like in shape, showing that a bronchial tube and its smaller subdivisions have been occluded by the casts.

Persons of all ages are liable to plastic bronchitis, but it affects males about twice as often as females.

The subjective symptoms are *cough* and *dyspnœa; hæmoptysis* occurs in about one-third of the cases. (Biermer.[1]) The cough occurs in paroxysms, which are frequent and severe; relief follows expectoration of the casts.

Hemorrhage may appear only as streaks of blood upon the casts, or may be considerable and follow their dislodgement. The *casts* themselves when ejected are usually coated with mucus, so that they appear as solid masses of sputum; their arrangement into cylinders may not be suspected until they are agitated in water. The size of the cylinder varies from that of the little finger to that of a bodkin, but they do not often exceed the size of a goose-quill. The larger casts may be hollow, but the smaller ones are solid, and are arranged in layers. They are whitish or gray in color, and firm in consistence, but become softer as the disease improves. Microscopically, the casts are nearly structureless, consisting of a fibrillated base, with pus and mucous corpuscles, a few gland-cells, and, occasionally, blood-cells in the outer layers. Charcot-Leyden crystals and Curschmann's spirals are found.

The acute form is rare, and out of ten cases accepted by Biermer six proved fatal. The disease begins with *fever*, dyspnœa appears early, severe paroxysms of cough occur, sometimes hemorrhage. Death results

[1] Virchow, Handbuch der spec. Path. u. Ther., Bd. v., Abth. 1.

PLATE XIX.

FIG. 1.—Anterior Aspect.

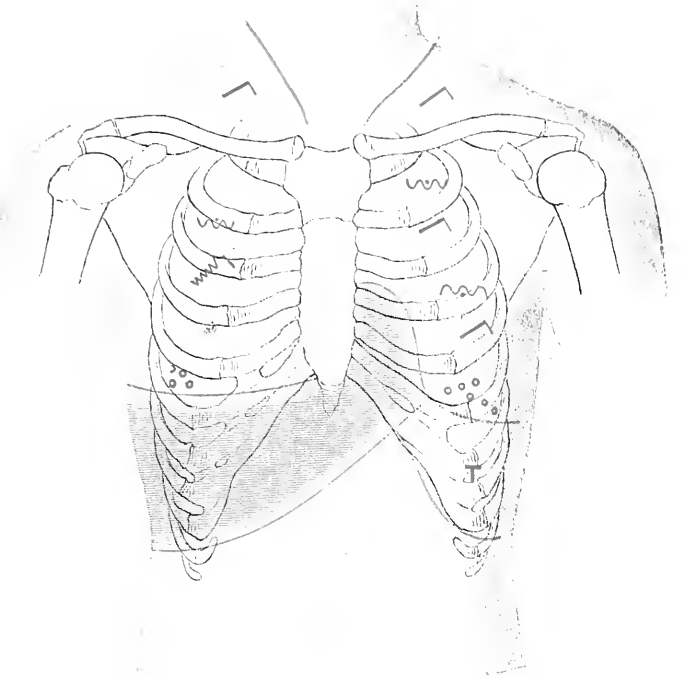
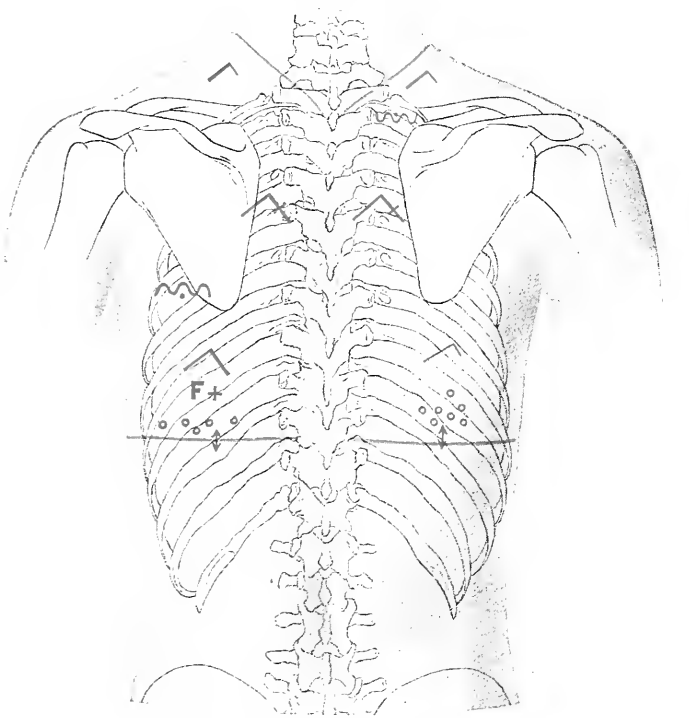


FIG. 2.—Posterior Aspect.



Capillary Bronchitis (early stage).

Rough or sharp breath sounds—expiration in places prolonged. Sonorous, sibilant and small moist rales. Local increase of fremitus.

from asphyxia. Grave symptoms are excessive dyspnœa, scanty expectoration, and drowsiness. Copious expectoration is a favorable sign.

Physical Signs. The casts obstruct the bronchial tubes. There is less air entering the part, hence there are *diminished fremitus* and *respiratory murmur* over the portions of lung supplied by the obstructed tubes. If collapse ensues, there is *dulness* on percussion; if the casts are dislodged, the murmur becomes normal or but slightly roughened. In unaffected portions of the lung resonance is clear or exaggerated, and the respiratory murmur remains unaltered.

Fuller says[1] that the upper portions of the lung are oftener affected than the lower portions.

Fetid or Putrid Bronchitis.

This name is applied to the condition in which the sputa have a highly offensive odor and are copious and semi-putrid. The odor is said by some to be due to microscopical sloughs, and by others to a special bacillus.

Putrid bronchitis may accompany (1) dilatation of the bronchial tubes; (2) chronic pneumonia; (3) phthisis or (4) empyema with a fistulous communication with a bronchus; or (5) it may occur independently.

Symptoms. The subjective symptoms are cough, irregular fever, and occasional chills. The *sputa* of fetid bronchitis have an odor of gangrene or feces. On standing they separate into three layers. The upper one consists of a greenish fluid or frothy layer; the second is sero-albuminous; and the third a thick granular deposit in which are masses the size of peas (Dittrich's plugs), and flakes consisting of granular detritus, and containing fatty crystals and bacteria, *Oïdium albicans*, and crystals of leucin and tyrosin. (See Sputum.)

Physical Signs. These are those of chronic bronchitis, or of bronchitis and the conditions with which it may be associated (*q. v.*).

Diagnosis. This form of bronchitis is diagnosticated from *gangrene* by the absence of physical signs of disintegration of lung tissue and by the absence from the sputum of fragments of lung tissue and elastic fibres. Nevertheless, gangrene of the lung may be the final result of putrid bronchitis.

Infectious Bronchitis.

In addition to the bronchitis that attends the infectious disorders mentioned above, three forms are seen of an infectious nature which are properly classified among the infectious diseases. It is proper to refer to them now, as bronchitis is usually the most pronounced local manifestation. They are *influenza*, *whooping-cough*, and *hay fever*. The last only will be spoken of at present.

Hay Fever.

Hay fever is a specific catarrh of the respiratory passages, caused by the pollen of certain plants, principally the grasses. The attack begins

[1] Quoted by Peacock in Diseases of the Chest.

with *itching, burning,* and *lacrymation,* and *pain* in the *brow* or *eyeballs.* Subsequently there are *itching* or *pricking* of the *nasal mucous membrane,* frequent *sneezing,* and an irritating *watery discharge.* Themucous membrane of the nose is red and swollen. A similar condition obtains in the throat when it is affected. If the disease attacks the bronchial mucous membrane, a bronchitis is set up, which, if it differs at all from ordinary bronchitis, is more persistent and attended by greater dyspnœa, with asthmatic attacks.

Collapse of the Lung.

Collapse of the lung is a condition produced by exhaustion of air from the air-vesicles. It may affect alveoli here and there, or even a large section of the lung. Formerly such collapse was invariably looked upon as pneumonia, until Legendre and Bailly proved by forcible inflation that the air-vesicles had simply collapsed from absence of air. Collapse occurs most frequently in the course of bronchitis and in cases with feeble respiratory power. The bronchial twigs supplying certain air-vesicles, or tubes supplying sections of lung, become occluded to such a degree that no air can enter. The air already contained in the vesicles then becomes exhausted gradually until the vesicles are completely airless. The vesicles or sections of lung involved then return to the fœtal condition. When the collapse is congenital, the term *atelectasis* is preferable. Anything which induces great muscular weakness predisposes to collapse of the lung; hence, in the aged and feeble, in wasting diseases, and in low febrile diseases of long standing, collapse is very apt to occur. But bronchitis is the most frequent and direct cause. The secretions which are poured out, and the swelling of the mucous membrane, occlude the tubes, and if the patient has not sufficient strength to expel the secretions, and by forced inspiration to expand the collapsing vesicles, collapse ensues.

Symptoms. Subjective symptoms are those of dyspnœa and insufficient oxygenation of the blood.

Physical Signs. If the symptoms are developed suddenly, and are accompanied by the appearance of dull areas in the lung without bronchial breathing, the diagnosis is tolerably certain; but when scattered lobules only are involved, the physical signs of collapse are absent, and its existence must be a matter of inference.

In life the diagnosis of the condition consequently is difficult. The site of collapse, being airless, is, of course, *dull on percussion.* The *respiratory murmur* is more likely to be *faint* or *absent* than to be increased in intensity or approach the bronchial. Nevertheless there is sometimes heard a faint bronchovesicular expiration.

When œdema is superadded to collapse, moist crepitant *râles* are heard, difficult if not impossible to distinguish from those of pneumonia. Respiration is embarrassed, and is accompanied by sucking-in of the lower part of the chest in inspiration. Sometimes the plug of mucus which occludes the tubes becomes dislodged while the physician is auscultating, and then the respiratory murmur will be heard, accompanied by a suc-

cession of crepitant râles, which disappear after a few inspirations. The dull areas, as a rule, are less persistent than those of pneumonia; thus it may be found at successive examinations that one area has cleared up and another has become dull. Stress is laid by some writers upon the signs of emphysema surrounding collapsed areas. But this does not give assistance in the cases in which most help is required—cases in which there is diffuse bronchitis with more or less œdema.

Diagnosis. From *lobar pneumonia* the diagnosis is easily made by the difference in the physical signs, and by the absence in pulmonary collapse of inflammatory symptoms, by the lower temperature, and the difference in onset.

The diagnosis from *bronchopneumonia*, or *catarrhal pneumonia*, is beset with greater difficulties. But here also the low temperature, and the fact that the physical signs and the location of the dull areas are subject to rapid changes, are of aid in diagnosis.

Bronchopneumonia, or Catarrhal Pneumonia.

This is a pneumonia occurring secondarily to bronchitis, and characterized by the development of areas of consolidation in both lungs and the persistence of a bronchitis of the middle-sized or smaller tubes. In proportion as the areas of consolidation are large, the symptoms and physical signs approach those of lobar pneumonia. It is more common in children and in debilitated persons. It is the chief form in infants. It is frequently *secondary* to measles, diphtheria, scarlet fever, and pertussis.

As *aspiration pneumonia*, it occurs when food, septic particles, blood, or tissue enter the lungs during the loss of sensibility of the larynx in apoplectic, uræmic, or other forms of coma, and in operations about the upper air-passages and mouth. It is a fatal complication of tracheotomy. It is frequently of *tuberculous* origin.

Symptoms. Catarrhal pneumonia, except the aspiration form, develops gradually, and it may not always be easy to mark the point at which the bronchitis which precedes merges into pneumonia; but as a rule there is more or less *chilliness* (rarely a decided chill) and an access of *fever*. There is usually greater *prostration* than in the lobar form, in proportion to the amount of pneumonia present. The *pulse* is more *frequent* and more likely to be *feeble*. *Cough* and *expectoration* are marked symptoms. The *sputum* is tenacious and glairy, not rusty. It contains streptococci and staphylococci in much greater numbers than are found in ordinary bronchitis; fatty epithelial cells, epithelium, fat-globules, and diplococci.

Dyspnœa is more extreme than in lobar pneumonia. The respirations are excessively rapid—60 to 80 per minute; *cyanosis* rapidly ensues. The finger-tips become blue, the face dusky. The fever does not rise as high as in the lobar form. At first the skin is hot and dry; later it becomes cold and clammy, and in the tuberculous form sweats are common. The duration of the disease is usually much longer than in lobar pneumonia.

In the common form seen in infants the symptoms of *asphyxia* set in at variable periods in the course of the disease. General cyanosis super-

venes. Stupor sets in, the hurried respirations grow shorter and more gasping, the pulse becomes excessively rapid and feeble, the extremities cool and clammy; with the stupor the cough abates and the breathing becomes more shallow. The lungs fill with fluid mucus, and the child drowns in its own secretions, or cardiac paralysis sets in after dilatation of the right heart.

Examination of the *sputum* shows an abundance of the streptococci and staphylococci and the special micro-organism which belongs to the primary infection, as that of influenza, diphtheria, or tuberculosis.

Physical Signs. (Plate XX.) These are those of bronchitis, with here and there larger or smaller areas of *consolidation*, over which the râles are finer and closer set; the *percussion-note* is *dull*, and the *respiratory murmur* is *bronchial* or *bronchovesicular*. An entire lobe may be consolidated. Areas of collapse and portions more or less œdematous combine to make the more complex physical signs. While both lungs are affected, they are not usually so to the same extent. It is said that the apices are more prone to involvement in this than in the lobar form; and some writers (Osler) look upon it as almost, if not always, of tuberculous origin.

Diagnosis. The affection is distinguished by (1) its pathological antecedents and causal relations; (2) its gradual onset; (3) its distribution in both lungs; (4) the preponderance of physical signs of bronchitis over those of consolidation; (5) the extreme dyspnœa and cyanosis with a lower temperature than in lobar pneumonia; (6) the onset of carbon dioxide poisoning; (7) the long duration and gradual decline. The *tuberculous* form is distinguished by (1) the history of exposure to infection or of a focus of infection in the body, glands, or joints; (2) the longer course; (3) the delayed asphyxia; (4) the rapid emaciation; (5) the diffused sweats; (6) the physical signs of consolidation and subsequently of cavity at the apex; and absolutely by (7) tubercle bacilli in the expectoration, coughed up or vomited. I have seen a child aged fifteen months, of a tuberculous mother, completely recover. The tuberculous form is common in colored infants.

Lobar Pneumonia, or Croupous Pneumonia.

For consideration of this disease, the reader is referred to the section on Infectious Diseases, Chapter I., Part II. (Plate XXI.)

Chronic Interstitial Pneumonia.

Cirrhosis, fibroid phthisis, and chronic interstitial pneumonia are names given to a condition of chronic induration of the lung, caused by interstitial over-growth of fibrous tissue. Obliteration of the air-vesicles and contraction of the lung result from the over-growth. The bronchi are frequently dilated, and cavities and gangrene may occur. The disease is rare except as the result of tuberculosis, but it may follow pneumonia and pleurisy, and it is said to be caused by inhalation of fine particles of steel

PLATE XX.

FIG. 1.

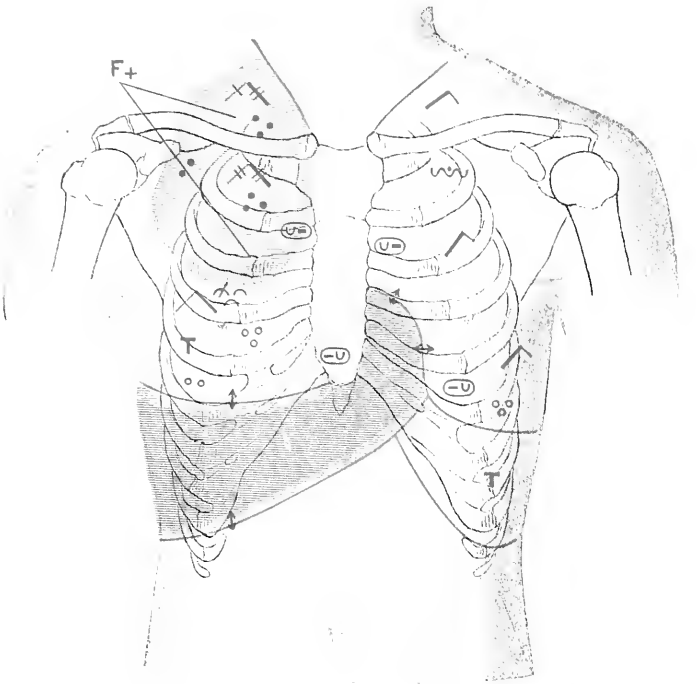
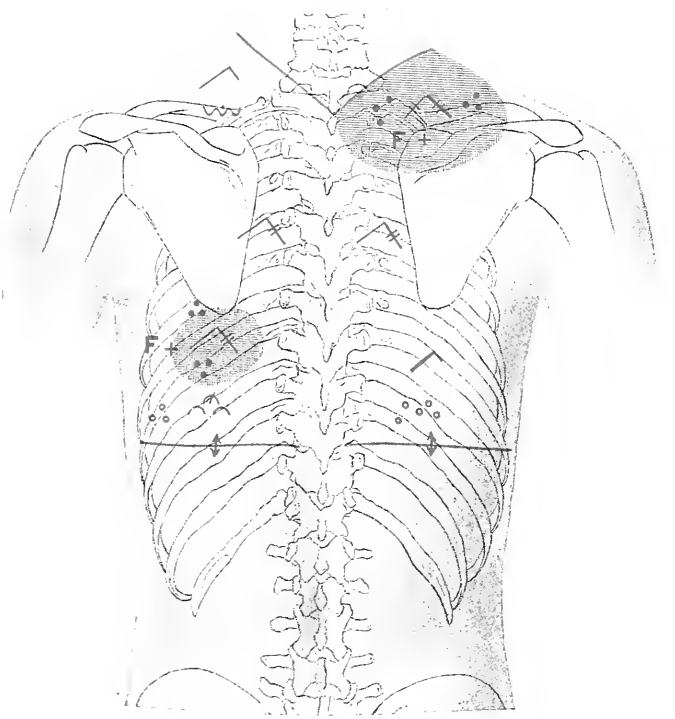


FIG. 2.



Bronchopneumonia.

Consolidation in the right upper and the left lower lobes. Physical signs of bronchitis over both lungs.

PLATE XXI.

FIG. 1.

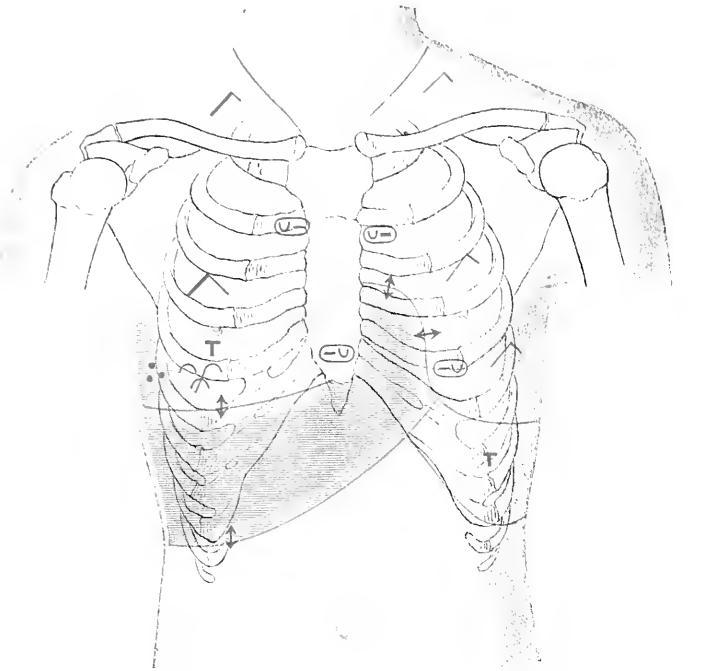
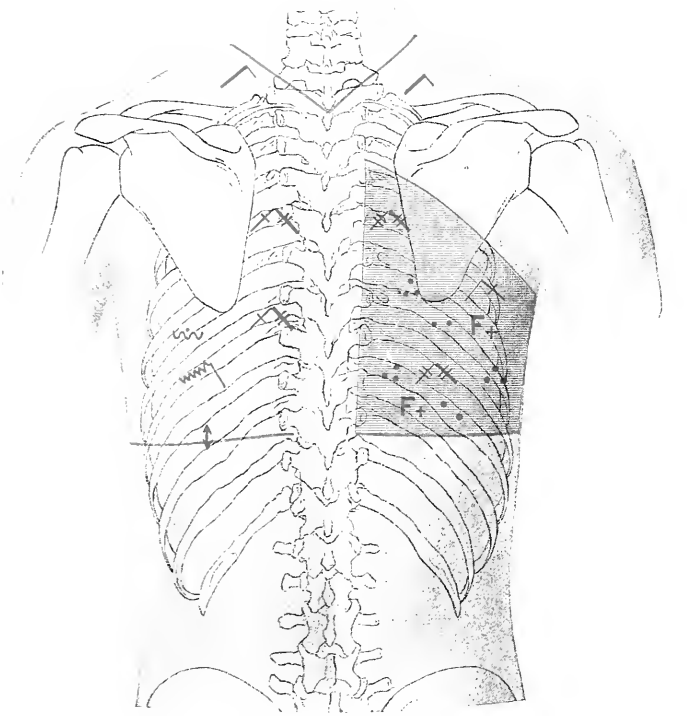


FIG. 2.



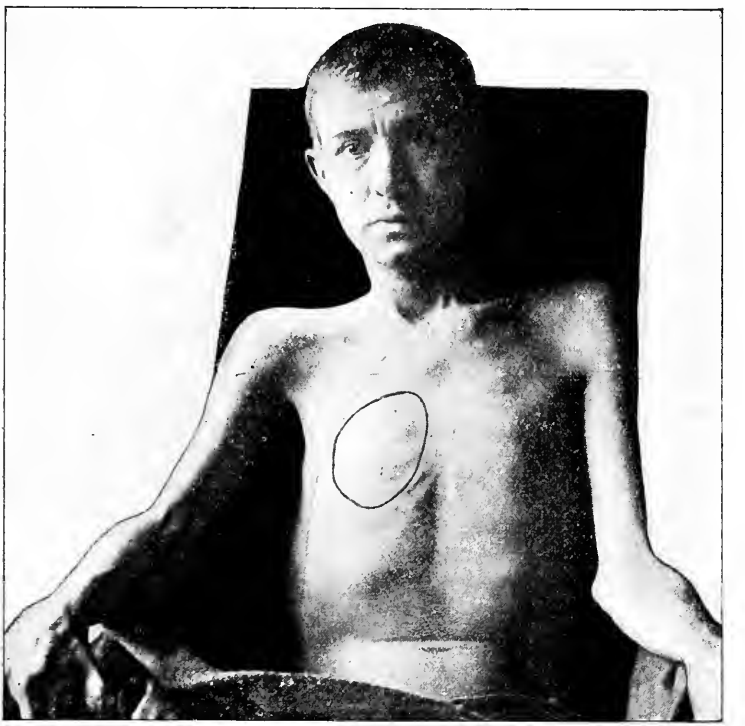
Lobar Pneumonia.

Consolidation of the right lower lobe. Transmitted bronchial breathing and signs of bronchitis over the left lung posteriorly.

or cotton. *Pneumonokoniosis* is the term, first employed by Zenker, for the chronic interstitial pneumonia due to the inhalation of dust.

Symptoms. The disease runs a very chronic course, attended by *cough*, and mucopurulent and sometimes bloody *expectoration*, even *hemorrhage;* but there is no fever and not much loss of flesh. *Dyspnœa* occurs on ascending heights only. Dilatation of the right heart is likely to ensue, with cardiac murmurs and increased lateral dulness and increase of dyspnœa. Death is hastened by the disease, and is often brought on by acute pneumonia.

FIG. 344.



Fibroid (tuberculous) phthisis; right apex. Heart displaced as indicated by oval. (Original.)

Physical Signs. (See Plates, Bronchiectasis.) **Inspection.** The disease is *unilateral.* The chest-wall is *retracted.* The ribs are drawn together, so that the interspaces are obliterated. The shoulder is drawn over the sunken thorax. The *spinal column* is *curved.* The *heart* is *displaced;* it is drawn toward the affected side. If the right lung is the seat of disease, an impulse is seen to the right of the sternum; if the left, the præcordial area of impulse is increased and extends upward. There is *no expansion* whatever (immobility) of the affected apex or base. The healthy lung is the seat of compensatory emphysema. (See Fig. 344.)

Palpation. Inspection is confirmed. *Fremitus* is *increased*, especially at the apex. At the base, pleural thickening lessens the fremitus.

Percussion. The physical signs show *increased density* of lung tissue, with *dulness* on percussion, or, over a dilated bronchus, a tympanitic or amphoric note.

Auscultation. The *respiratory murmur* is *bronchial*, or, over a dilated bronchus, has a hollow sound. At the base breath-sounds are feeble, distant, or absent. *Râles* are also heard.

Pneumonokoniosis (also known as *anthracosis*, coal-miners' disease; *siderosis*, from metallic dust; *chalicosis*, from mineral dust, as in stone-cutters' phthisis). In this form there is a history of *exposure* to the irritating particles for a considerable period, during which time *cough* develops, gradually increases, and the *general health fails*. *Emphysema* simultaneously arises, causing *dyspnœa*. The patients wheeze, cough in paroxysms, and expectorate *sputum* which contains the dust-particles. In anthracosis it is black. On microscopical examination the special dust-particles are often found. The symptoms of emphysema and chronic bronchitis predominate. Tuberculous infection may take place late in the disease.

Pulmonary Tuberculosis.

For convenience of diagnosis the specific inflammation of the lungs caused by *Bacillus tuberculosis* will be considered in this section. If a strict ætiological classification were followed, it would be considered among the infectious diseases.

Clinically, we see tuberculosis in the lungs manifesting itself in one of the forms of acute pneumonic phthisis, acute miliary tuberculosis, and chronic ulcerative phthisis. (See Chapter I., Part II.)

Tuberculosis of the lungs, pulmonary phthisis, and consumption are names applied to an infectious and mildly contagious disease of the lungs, caused by the tubercle bacillus, appearing in an acute and chronic form, and characterized by cough, fever, sweats, more or less rapid emaciation, purulent expectoration containing elastic fibres and tubercle bacilli, and by peculiar physical signs.

Acute Pulmonary Tuberculosis, Acute Phthisis, Acute Pneumonic Phthisis, Galloping Consumption. This disease may be primary or may be secondary to a localized area in the lung, causing rapid infection, or to tuberculous pleurisy, tuberculous peritonitis, or to tuberculosis of some other organ.

Symptoms. Its onset is usually marked by *cough*, *fever* with or without chills, *dyspnœa*, and sometimes *hæmoptysis*. The fever rises to 103° or 104° F., and is of a continued type (lobar pneumonic form), or rapidly assumes a hectic type, accompanied by restlessness and exhausting night-sweats, anorexia, and rapid emaciation. *Prostration* is extreme, but the mind is at first clear and the spirits cheerful. *Cough* increases; the *expectoration*, at first mucoid and scanty, but often tinged with blood, becomes more copious and mucopurulent. The *bowels* may be loosened or constipated. The *urine* may show the diazo reaction.

When death takes place without more decided pulmonary symptoms, the tuberculosis has been secondary to tuberculosis elsewhere, or death is the result of a general miliary tuberculosis.

PLATE XXII.

FIG. 1.—Anterior Aspect.

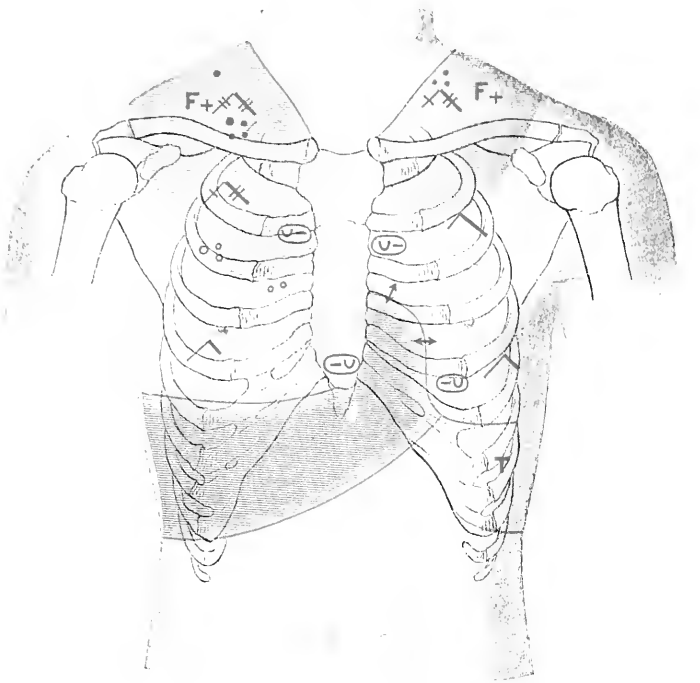
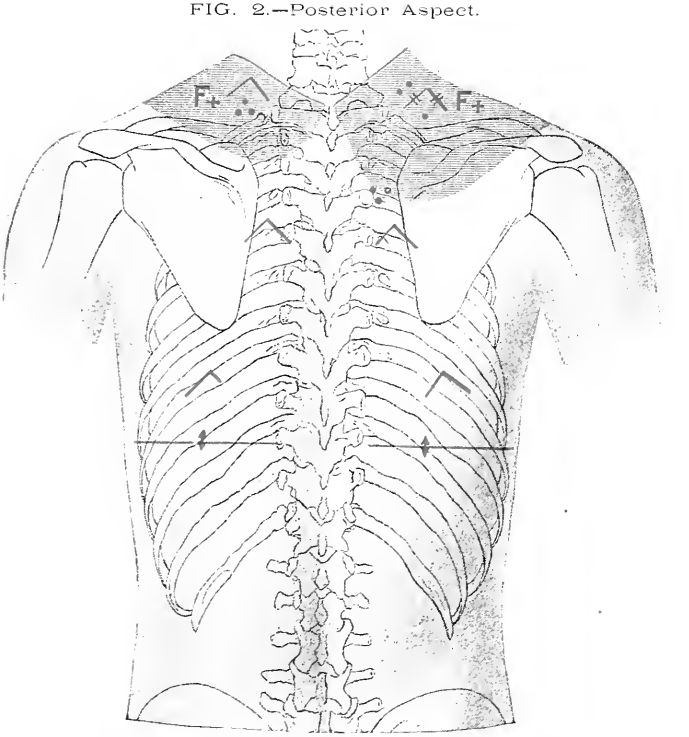


FIG. 2.—Posterior Aspect.



Acute Pulmonary Tuberculosis.

Consolidation of the entire right upper lobe and of the left apex.

When the acute pulmonary tuberculosis is primary, the character of the disease is soon made clear by the early development of consolidation of the lungs, usually of an apex first, rapidly followed by softening and the formation of cavities. The *sputum* becomes mucopurulent, is frequently streaked with blood, and pure blood is often coughed up. The sputum contains yellow elastic tissue and abundant tubercle bacilli. The patient often presents a *cachectic* appearance; emaciation has been very rapid, and has reached an extreme degree; there is frequently a red flush about the cheek-bones, which, with the bright eyes, contrasts strongly with the hollow cheeks and temples, and the white, wasted hands and clubbed fingers with bluish nails. *Cyanosis* is shown in the dusky countenance and blue finger-tips. The *exhaustion* becomes extreme. The *sweats* are profuse. The appetite is lost. *Diarrhœa* may set in. *Remissions* may take place, even in acute cases; for a time the fever and more aggravated pulmonary symptoms are in abeyance. The typhoid state ensues in some cases. *Death* takes place from exhaustion and heart-clot or from meningeal tuberculosis. The *duration* is from two to six weeks.

The patient's *mental attitude* is often peculiarly and characteristically hopeful. He expresses himself as better each day, though he is occasionally subject to despondency, and is sure that if he could only gain a little strength he would soon be well.

Sometimes, especially in children, the disease is *latent*. The patient suffers from weariness, the cheeks flush easily, the pulse is readily disturbed, there are nocturnal fever and occasional sweats. Emaciation proceeds very gradually, and a long time may elapse before disease is demonstrable.

In a few cases the *cerebral symptoms* are so pronounced as to mask the pulmonary, and in other cases there is actual coincident involvement of the cerebral meninges.

Physical Signs. (Plate XXII.) These are those of consolidation, often without conjoint pleurisy. The apex is usually first invaded. There are *diminished movement, increased fremitus,* and *dulness on percussion.* At first the *breathing* is *bronchovesicular.* It rapidly becomes *bronchial.* At first small moist *râles* are detected. Later they become large and gurgling. A *pleural friction*-sound may be heard. It may be heard first above the spine of the scapula behind, above the clavicle in front, or high up in the axilla. The upper lobe of the right lung may be affected at first, or the anterior portion of the middle lobe. The physical signs may be observed first in the axillary region of either side. The consolidation extends to the remainder of the lung, being preceded by physical signs indicating gradual encroachment upon the air-containing structure. The respiratory murmur is harsh, but soon becomes bronchovesicular and then bronchial (lobar pneumonic form). As consolidation progresses in the middle and lower portions of the affected lung, signs of cavity or multiple cavities appear in the upper. (The whole of a lobe may be the seat of small cavities filled with mucopurulent or purulent fluid.) *Cavernous breathing* and *pectoriloquy,* or the *bronchial sniff* of consolidation, become more pronounced. The dull note of consolida-

tion is relieved by a *dull tympanitic* or full tympanitic note. Now moist râles of all degrees are heard (*bronchopneumonic form*). Above they are gurgling; below, small and large moist râles. If the progress is not too rapid throughout the lung first affected, signs of invasion are found in the remaining lung, usually at a point corresponding to the primary focus in the original lung. The apex therefore is first invaded in most cases. Infection of the second may begin earlier than the signs in the first lung would lead one to anticipate. The rapid invasion of one lung compels compensatory emphysema of the other. The increased movement, with harsh or puerile breathing, without change in fremitus or in pitch and tone on percussion, masks many small consolidations.

Diagnosis. In the earliest stages, before the invasion of new territory is pronounced, the cases are involved in doubt. Pulmonary tuberculosis may be confounded with pneumonia until the sputum is secured and bacilli are found.

In *pneumonia* we have the pronounced rigor, the rapid rise of temperature, the altered pulse-respiration ratio, the hot, dry skin, the sticky, viscid sputum, containing the pneumococcus, the peculiar changes in the urine, leucocytosis, the occurrence of herpes, and the termination by crisis, to point to the nature of the process. Emaciation is not marked; there are no such profuse sweats as the repeated drenchings we see in pneumonic phthisis; anæmia is not so pronounced. Then cavity formation does not take place, or at least occurs rarely. In pneumonia the fever is of a continued type; in phthisis it is often intermittent or remittent. The sputum is more purulent in acute pneumonic phthisis. Finally, the history of exposure to infection, the primary occurrence of tuberculosis elsewhere, the secondary occurrence of tuberculosis in other organs after the lung-invasion, the longer duration—aid in determining the true affection. Inoculation of animals may be resorted to in doubtful cases.

Acute Miliary Tuberculosis of the Lungs. **Symptoms.** These are high *fever*, rapid *emaciation*, *hurried breathing*, *rapid pulse*, *duskiness* of face and extremities, more or less *stupor*, *delirium*, and the development of the typhoid state, with *prostration* and the occurrence of profuse *sweats*. Intestinal symptoms, as *flatulence* and *distention*, may be pronounced, and *diarrhœa* may form a prominent feature.

The onset is abrupt or may follow a period of malaise. In some instances the tuberculous process is more advanced in some situations than in others, giving rise to special local symptoms. Thus, recently, a patient was admitted to the Presbyterian Hospital with stupor and moderate delirium. He had fever, rapid pulse and breathing, and a peculiar dry, harsh skin. There were albuminuria, casts and blood in the urine, and it was thought he had uræmia. The temperature range was irregularly intermittent. The diagnosis was established later because of the development of undoubted secondary tuberculosis in other organs. At the autopsy general tuberculosis was found, with primary tuberculous ulceration in the bladder, the ureters, and renal pelves.

Physical Signs. These are negative or are those of bronchitis. There is *resonance* or *hyper-resonance* on percussion. The latter is not uncommon.

Diagnosis. Hurried breathing and cyanosis are distinctive features, out of all proportion to the physical signs, and, on this account, of diagnostic significance. Acute miliary tuberculosis must be distinguished from typhoid fever, septicæmia or pyæmia, and malignant endocarditis. It is distinguished from *typhoid fever* by the absence of successive stages in the course of the disease; in typhoid fever the evolution of the disease is more characteristic than its symptoms. The headache of the first week, finally disappearing, is noteworthy. The special range of temperature, the onset, the fastigium, and the defervescence at definite periods in the evolution of the disease, are of diagnostic value. Cyanosis is more constant and marked in tuberculosis. The skin and capillaries have more tone in typhoid fever than in tuberculosis, at least in the first two weeks. Hyperæmia follows irritation in typhoid; pallor, with duskiness, in tuberculosis. The eruption, with its specific mode of development, belongs to typhoid fever alone. The stools, the enlarged spleen, the vascular tone are suggestive of typhoid fever. The spleen enlarges earlier in the disease in typhoid fever. Bacteriological examination may be of service. The occurrence of intestinal hemorrhage, pointing as it does to typhoid fever, is a welcome sign in cases in which the diagnosis is obscure. I have never seen it in tuberculosis. In typhoid fever the reflexes (knee-jerk) are never absent; in tuberculosis, if the meninges are involved, they are variable, present one day, absent the next. The Widal test is important. The diazo reaction in typhoid fever is of some service, although it also occurs in tuberculosis. (See Urine.) It does not come on until later than the fifth day in typhoid. It disappears at a certain time in the involution of typhoid; it continues indefinitely in tuberculosis. (See Chapter I., Part II.)

The distinction of tuberculosis from *septicæmia* or *pyæmia* and *malignant endocarditis* is often difficult. We must search for local areas of septic or pyæmic infection. The ears, the teeth, the bones, the veins, the heart, the pelvic organs in females, the rectum, the genito-urinary tract, all must be carefully examined. Hemorrhagic infarcts, or metastatic abscesses, may be found which point to the original conditions. The eye-ground may show hemorrhages. The skin and mucous membranes may exhibit minute capillary hemorrhages or infarcts. They are the size of a pin-head, do not disappear on pressure, and are not elevated. The spleen is more likely to be enlarged in the septic affections. The respirations are not so rapid as in tuberculosis. Cyanosis is a distinctive feature of tuberculosis. The physical signs of endocarditis may be determined, and subsequently embolism or thrombosis prove the nature of the process.

Chronic Tuberculosis, Chronic Ulcerative Phthisis. Chronic tuberculosis or phthisis is much more common than acute tuberculosis, from which it is distinguished by its slow progress and by periods of remission, during which the disease may be arrested temporarily or permanently.

Symptoms. The disease may begin in a variety of ways. The most common mode of origin is in an ordinary *bronchitis* with which pleurisy is occasionally associated. Previous to this the patient may have been in good health, but generally the health has been impaired for some time.

The bronchitis may be simple or part of influenza, measles, whooping-cough, or some other specific disease.

The bronchitis usually proves obstinate, and by and by the patient will be found to have *lost strength*, and usually some *weight*. There is often a slight evening *rise of temperature*, and occasionally *nocturnal perspirations*. The appetite is impaired, and *anorexia* may exist. *Cough* is rarely absent, especially during the night or on waking in the morning; it may, however, be so slight as apparently to have escaped the notice of the patient. When characteristic, it is dry and hacking. *Expectoration* is scanty and mucoid, but occasionally it may be tinged with blood. It should be remembered that children and old persons sometimes do not expectorate, and that, as a rule, women are more inclined to suppress expectoration than men. No tubercle bacilli may be found in the sputum after repeated examination; but if examinations are continued, they will appear sooner or later.

Instead of developing after a bronchitis, as we have just described, it may set in suddenly under the guise of a pneumonia, more frequently of the catarrhal form. The symptoms and physical signs do not differ essentially from those of pneumonia, except that the expectoration is more likely to be profuse, mucopurulent, and blood-streaked, and bacilli are found in it; the fever is more hectic in type, and night-sweats are common. After the patient convalesces from such an attack he continues weak, does not gain flesh readily, still has a cough with expectoration, and evening fever with occasional night-sweats.

In some cases fever, emaciation, and weakness progress for some time before pulmonary symptoms arise.

In still other cases the invasion of the disease is by sudden *hæmoptysis*, which is oftener copious than not. Several such hemorrhages may occur in rapid succession, or there may be only one. Moreover, its disappearance may not be followed, or at least not immediately, by any further pulmonary symptoms; more commonly, however, it is followed by fever, cough, and expectoration.

In still other, but rarer cases, the pulmonary disease is latent, being marked by *gastric* or *peritoneal symptoms*, or by a general *anæmia*.

By whatever path invasion comes, the physician should be on the look-out for it, especially in a young adult predisposed by heredity or environment to tuberculosis. The recognition of the disease in its early stage requires the greatest skill, which in turn is recompensed with the highest reward, since the disease is then curable.

The further progress of a case of tuberculosis of the lungs, after consolidation has once become manifest, is very variable. It may be arrested at this point permanently, cure resulting from cicatrization. More frequently there is temporary arrest of the process; fever lessens or ceases entirely, the pulse resumes its normal rate, appetite improves, and there is a gain in flesh and strength. Cough and expectoration are more likely to persist than the other symptoms, but with the other improvement they diminish in frequency and copiousness.

After a longer or shorter time, as the result of reinfection from the old focus excited by acute bronchitis or by some depressing influence, the

PLATE XXIII.

FIG. 1.—Anterior Aspect.

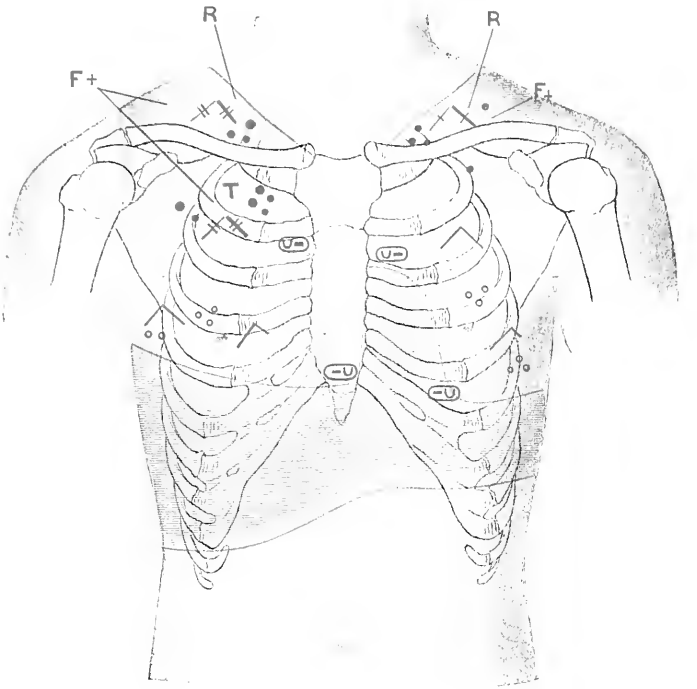
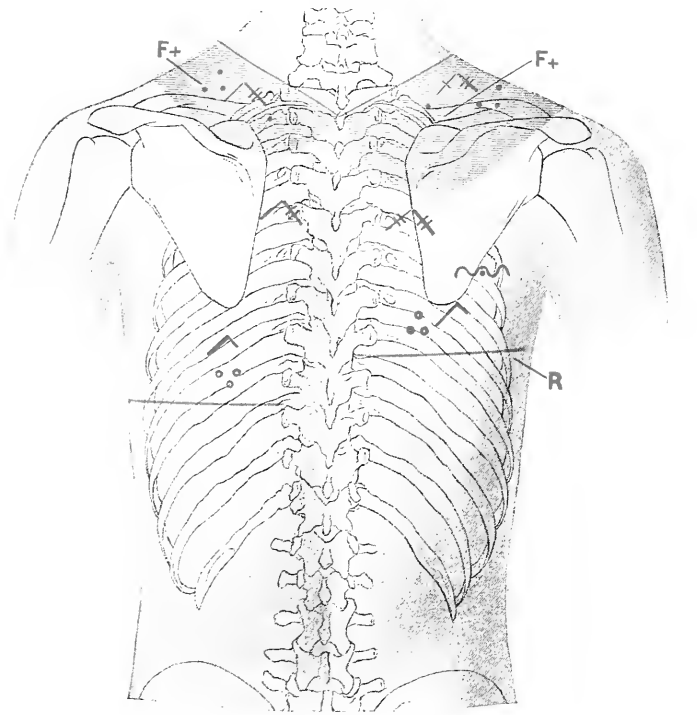


FIG. 2.—Posterior Aspect.



Chronic Pulmonary Tuberculosis.

Consolidation with cavity formation. Chronic pleurisy with loss of respiratory movement of lung margins. Retraction.

tuberculosis is relighted, so to speak, and runs much the same course, the lung being left more diseased and the general health worse after every such attack. Nevertheless, there may be long intervals between such attacks, the patient in the meantime continuing in fair health. Thus the disease may linger or recur for years, the patient not ill enough to be confined to the house, and not well enough to withstand hard work or great exposure. Slowly, by ulceration and suppuration, the lung tissue is wasted and cavities are formed. Before there are large cavities at an apex the base of the same lung becomes consolidated by the production of tuberculous material, and before one lung is extensively diseased the apex of the opposite lung is attacked, the process being repeated in it if the patient lives long enough. Instead of reinfection from an old focus, new infection may take place, giving rise to the old train of symptoms, or setting up some acute disease. During this time the patient is liable to an attack of acute pneumonia, pleurisy, bronchitis, or general miliary tuberculosis. He is also liable to sudden death by hemorrhage. In a number of cases the intestines and peritoneum become affected, and abdominal pain and diarrhœa are superadded as symptoms.

As a rule, the patient *gradually sinks*. The later stages are marked by increasing cough and dyspnœa, which are very distressing and prevent sleep. Expectoration is more copious, purulent, and is raised with increasing difficulty.

The appetite is poor and capricious, or anorexia is complete. The heart becomes more and more feeble, the fever is *hectic* and accompanied by *exhausting night-sweats*, the feet and limbs swell, and acute cramp-like pains are felt in the legs, probably caused by thrombosis of the veins.

Emaciation is extreme, scarcely anything but skin and bone being left. *Death* occurs from perforation of an intestinal or gastric ulcer, from hemorrhage, or more commonly from exhaustion, and from asphyxia caused by œdema of the lungs.

Physical Signs. (Plate XXIII.) These depend upon the lesions. It is often possible to detect all stages of the tuberculous process, from early consolidation to large cavity, in the same patient. When the disease begins as an obstinate bronchitis, there is found at the apex of the lung a small area over which, on percussion, there is *increased resistance*, with *slight impairment of resonance*, as compared with the other side; the *respiratory murmur* is *bronchovesicular*, sometimes jerky in rhythm, and the *vocal resonance* and *fremitus* slightly increased or unaltered. Such physical signs are met with more frequently at the right apex than at the left, and oftener in the suprascapular fossa than anteriorly. The next most frequent seat is probably between the clavicle and second rib anteriorly. When phthisis sets in as a catarrhal pneumonia, consolidation is, as a rule, found at the apex. After convalescing from the attack, the patient has an area of consolidation usually at an apex of the lung. Over this area, in addition to the usual signs of consolidation (bronchial or feeble breathing, dulness, etc.), moist or dry subcrepitant *râles* are heard. As the case progresses there are fewer râles, but the signs of consolidation are still present, though there is no further exten-

sion of the process. Often after a cavity has been formed the disease is arrested or progresses very slowly. When softening begins, the *percussion-note* continues *dull* and the *breathing bronchial;* but it is often difficult to make out the quality of the breath-sounds because they are feeble and obscured by numerous *moist crackling râles* and *moist subcrepitant râles* from disintegration of lung tissue and bronchitis. After the patient has coughed several times and expectorated, and then takes a long breath, the quality of the breathing becomes perceptible. As the lung tissue is further softened and removed by expectoration cavities are formed. These, if large enough and superficial, give a *tympanitic note* on percussion, and, if there is communication with a bronchus, a *cracked-pot sound.* The *breath-sounds* are *hollow* and the *râles* are *bubbling and gurgling,* or large and mucous.

The normal vocal resonance is replaced by *bronchophony* and *pectoriloquy.* Tactile *fremitus* may or may not be increased. (See Cavities.) But if the walls of the cavity are thick from indurated tissue, the percussion-note will be dull and the breathing bronchial. If the tissue composing the wall is less thick and dense, percussion produces a wooden sort of resonance. If much normal lung tissue intervenes, the percussion-note will be clear.

As tuberculosis of the lungs progresses, the clavicles and ribs become more and more prominent from the loss of fat, and *local flattening of the chest,* with *impaired expansion,* marks the seat of the disease.

Résumé of the Diagnostic Features of Pulmonary Tuberculosis. The striking phenomena of tuberculosis which are considered in the diagnosis are emaciation, anæmia, fever, cough, dyspnœa, chest pain, hemorrhage, the expectoration, and the objective symptoms. Of less diagnostic value, but important as collateral data, are the aspect, the occurrence of vomiting and diarrhœa, and of symptoms of secondary tuberculosis in other organs. Age and occupation may, to a certain extent, aid in the diagnosis.

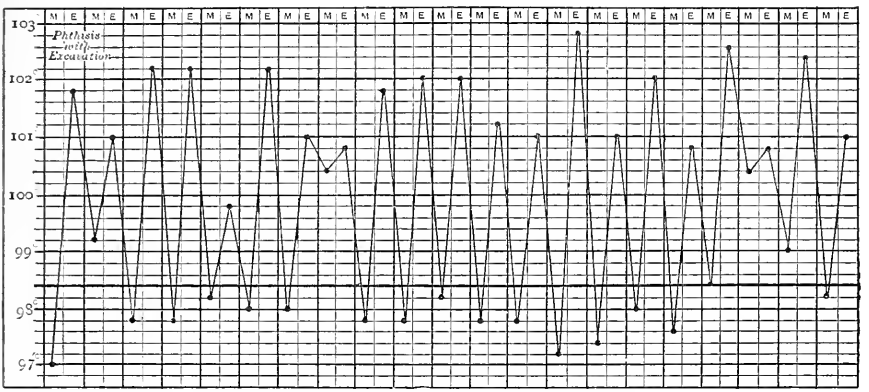
Symptoms. Emaciation. This is always seen, even in acute forms of tuberculosis. It is rapid in the acute; slow and progressive in the chronic forms. In the latter there may be a temporary improvement in this respect. It must not be confounded with *muscular atrophy,* and the emaciation of *carcinoma, diabetes, anorexia nervosa,* and other exhausting diseases.

Anæmia. This is always pronounced. It may be associated with leucocytosis if there is cavity formation. The reduction of red cells and diminution of hæmoglobin are marked.

Fever. This symptom is always present. The temperature should be taken every two hours for a time, to determine accurately the degree and course. It may be intermitting, remitting, or continuous. It may be intermitting in some acute forms, the morning fall reaching or going below normal. The difference between morning and evening temperature may not be more than a degree. In the acute form it is high and continuous, and soon may be attended by the typhoid state. In the more chronic cases it may be intermittent at first, then continuous, and finally intermittent again. In the later stages the intermitting fever is due to a

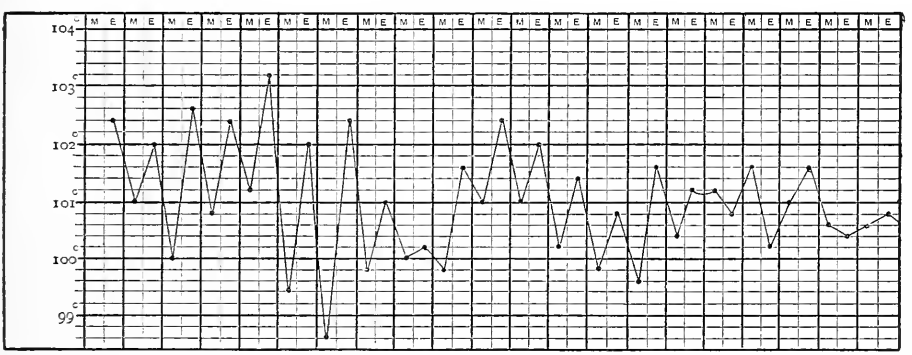
mixed infection, or sapræmia, due to the purulent contents (staphylococcus and streptococcus infection) of the lung cavities.[1] (See Fig. 345 and Fig. 346.) The intermittent fever of the early stages has frequently been mistaken for *malaria*. (See Fever.) The occurrence of fever in a patient who has been losing flesh, and is otherwise in poor health, excludes cancer and diabetes and other afebrile causes, and points strongly to tuberculosis. It must not be forgotten that in chronic tuberculosis in the aged the temperature may not rise above 100° F.; often, indeed, it is subnormal.

FIG. 345.



Intermitting fever of tuberculosis. (Original.)

FIG. 346.



Continued fever of tuberculosis. (Original.)

We must consider, therefore, that fever, the cause of which is not obvious, may be due to tuberculosis; and that if, when such probable causal conditions as gastro-intestinal catarrh or infectious disorders (malaria and syphilis) and suppurations are eliminated, the fever still persists, then the fever is probably of tuberculous origin.

[1] Leyden has pointed out that intermitting fever is part of the tuberculous process, and not a streptococcus or staphylococcus infection as formerly held, because pus micro-organisms are not found in the purulent contents of cavities, and because in other forms of tuberculosis, as empyema or joint-disease, they are notably absent, and yet such form of fever exists.—Deutsche med. Wochenschrift, Sept. 14, 1894.

Sweats. Frequent sweating may be the first symptom complained of by the patient. It may occur with the tripod of symptoms of the intermitting febrile range—chill, fever, and sweat. It would be likely to occur at night under these circumstances. It may occur at any time, however. "Night-sweats" are alarming to the mind of the laity, and are really of diagnostic significance. The perspiration awakens the patient at night because it is so profuse. It may be only moderate, not rousing the patient until morning. It may be general or local. Local sweats are confined to the head and neck.

Cough. Cough is one of the earliest symptoms. It may be the only symptom for some time. It is often dry and hacking at first, and may continue so for a long time. Later it is accompanied by mucoid and then mucopurulent sputa, which contain the characteristic elements. (See Sputum.)

Dyspnœa. This is almost always present. The degree varies with the association of fever. When the latter is present, dyspnœa is more pronounced. It is more pronounced in acute cases. In miliary tuberculosis the frequency of respirations that attends the dyspnœa is out of all proportion to the physical signs. In this form cyanosis is more marked. In chronic localized phthisis the dyspnœa may occur only on exertion, after eating, or upon excitement. The bloodless lips may have a constant bluish hue. The fingers are dusky and become "clubbed." In the later stages the dyspnœa is constant and in proportion to the extent of involvement of the lungs and the degree of fever. Although of diagnostic significance only when associated with other symptoms, it is most distressing, and is the cause of constant demand for relief.

Chest Pain. This is due to localized pleurisy or to myalgia. The latter may be seated in muscles strained by coughing. Pleuritic pains may occur in any situation, and vary in position from time to time. They may be due to extensive inflammation or to tuberculous pleurisy. Constantly recurring and unilateral chest pains, with or without signs of pleurisy, with cough and emaciation, are significant of the disorder under consideration. (See Pain.)

Hemorrhage. This symptom is alarming, and, in the large majority of cases, is due to pulmonary tuberculosis. It may mark the onset of the acute disease, and continue irregularly throughout its course or it may recur several times before the advent of the more common symptoms of the chronic form. It may occur at intervals of a few months or a year before emaciation, cough, and characteristic expectoration set in, or before bacilli are found in the sputum. Each attack is usually attended by fever, and is followed by anæmia and prostration. If hemorrhage of the lungs (see Symptoms) occurs in a young adult without cause (as aneurism or cardiac disease, etc.), it must be looked upon with suspicion. The likelihood of tuberculosis is increased if the bleeding occurs in a patient of tuberculous aspect in whom a family history of tuberculosis is found, or who has been exposed to infection. In the aged it may occur from a localized area of disease. Hemorrhage is also common in the late stages of tuberculosis. It is not at this period of diagnostic value as to the primary cause. It is usually due to erosion of an artery in a cavity.

Hemorrhage also occurs in tuberculosis during the quiescent period. The progress of the disease is arrested. The discharge of blood is accompanied by the expectoration of pulmonoliths, calculi formed by the degeneration of caseous areas.

VOMITING (see Gastro-intestinal Disease). This is a symptom which is often present in the early stages of tuberculosis of the lungs, and frequently masks the true condition. The vomiting may lead to the belief that a LOCAL GASTRIC CATARRH OR DIARRHŒA is to blame for the general symptoms. The occurrence of fever with the gastric symptoms should lead to an examination of the lungs.

The occurrence of diarrhœa and symptoms of tuberculosis in other organs may thoroughly establish the diagnosis in tuberculosis of the lungs with otherwise obscure pulmonary symptoms. The intestinal discharges may contain tubercle bacilli, or they may be found in the urine, in joint-suppuration, or glandular enlargement.

THE SPUTUM (*q. v.*). The diagnosis is absolute when tubercle bacilli are found in the expectoration. Nummular sputa are more common in phthisical excavation. The sputum is discharged in tough coin-shaped masses, which sink when expectorated into a vessel containing water. Fragments of lung tissue (yellow elastic) point to tuberculosis, but are possible under other circumstances.

Physical Signs. The aspect of the patient is always suggestive, and is an aid to the recognition of the condition. The tuberculous or *phthisical chest*, the long neck and arms, the pale face, the occasional *hectic flush*, the *clubbed fingers*, the *emaciation* of the many subjects we see in our infirmaries, fix in our minds a composite picture the recognition of which goes far to diagnosticate the insidious disease.

The objective signs point to an invasion of air-containing structure by solid material, with collapse of lobules, leading to consolidation, followed by cavity formation, and in both stages by the occurrence of pleurisy. *Local contraction* (*flattening*) and *impaired movement* at an apex, with *inspiratory depression* above the clavicles, with suppressed breath-sounds and *prolonged expiration*, with *impaired resonance*, are the earliest signs of tuberculosis. In the chronic cases, contraction, impaired movement, dulness, and increased resistance from thickened pleura may override the signs of consolidation. No one physical sign is of diagnostic significance. The combination of signs, and the orderly procession by which they advance as the physical conditions progress, are the most diagnostic.

In the diagnosis of pulmonary tuberculosis the physical examination must be directed to a determination of the size of the lung, and of the extent of its expansion, by which we judge of the amount of air entering the lung, as well as to the presence of consolidation.

The tuberculous process is usually associated with diminution in the bulk of the lung. We can estimate the *size and the degree of expansion* by inspection, palpation, and percussion. The so-called diaphragm-phenomenon is studied and the *x*-rays employed. Any diminution in the excursion of the shadow of the diaphragm is evidence of diminished bulk of the lung or of diminished expansion. By palpation, with mensuration, measurements are taken. By percussion we estimate the lung boundaries.

The degree of expansion can be determined by securing the limits of liver-dulness and cardiac and splenic dulness in ordinary breathing, and then at the end of full inspiration and expiration. Valuable information is thus secured. Of course, in employing inspection and palpation the two sides of the lung must be compared. Percussion enables one to determine fairly early the presence of consolidation. In thin subjects the change in the note is more readily elicited than in fat or muscular subjects.

On auscultation in the early stage of tuberculosis roughness of respiratory murmur with prolonged expiration, feeble respiratory murmur, and jerking or cog-wheel respiration are common signs. These signs change gradually into bronchovesicular and then bronchial types of breathing. Crackling râles or clicking sounds and consonating râles attending these modifications of breath-sounds are of the greatest diagnostic importance. They must be brought out frequently by cough and then full inspiration.

The Site of the Lesion. The situation of the physical signs is diagnostic. Percussion should be directed especially over those parts of the lung in which an infection is liable to occur, as the clavicular and subclavicular spaces, the anterior border of the upper lobe, the tongue-like part of the left upper lobe, which overlaps the heart, the supraspinous space, the upper interscapular region, and the upper borders of the lower lobes posteriorly. The latter is best secured by having the patient place the hand of the arm of the side percussed on the shoulder of the opposite side. The scapula is thus removed from the surface of the lung to be examined.

It is necessary also to consider carefully the general conditions. We inquire the age; adolescence and early adult life being the common periods in which pulmonary tuberculosis develops. The occupation, the history of exposure to the disease, the history of predisposition to tuberculosis in the family, the history of previous, now arrested, tuberculosis, as in joint-disease, or glandular tuberculosis (scrofula), are data deserving special consideration, as they may furnish corroborative evidence of the presence of the disease.

Diagnosis. The presence of tuberculosis is presumed in a patient with pulmonary symptoms—as a hereditary predisposition, abnormalities in the form of the chest, and imperfect development or hypoplasia of the circulatory organs. If the patient is under weight and has a poor appetite, and at the same time is undergoing unusual strain or anxiety, the possibility of tuberculosis is increased. Often before the physical signs of tuberculosis can be established, the shrewd physician will fear recurrence of tuberculosis if there are signs of anæmia, progressive loss of weight, slight fever, disturbed digestion, a frequent pulse, and persistent and localized bronchial catarrh. The examination of the lungs, the examination of the sputa, and the tuberculin test must be employed as soon and as often as practicable. (See Diagnosis of Tuberculosis,[1] Chapter I., Part II.)

The diagnosis is established by finding tubercle bacilli in the sputum.

[1] Several undoubted instances are recorded in which hospital residents and young physicians working in laboratories in which tuberculosis is studied, or constantly examining sputum, have been infected in the course of their studies.

Their absence, in spite of the most careful search, is against the tuberculous origin of the disease. (See Diagnosis of Tuberculosis, Chapter I., Part II.)

In other chapters the differential diagnosis of tuberculosis and other diseases will be pointed out. It must not be forgotten that the disease may set in as the terminal affection in many diseases. Thus, in diabetes, in insanity, in chronic cerebral or spinal disease, and in other affections, tuberculosis may develop insidiously, and finally cause death.

It must be distinguished from *chronic gastric disorders*, and particularly *anorexia nervosa*. It must not be confounded with *malaria*. It must be distinguished from *simple anæmia*, the cause of which may be recognized with difficulty. It must be distinguished from *chronic bronchitis* with bronchiectasis, from *pulmonary gangrene* and *carcinoma*. Finally, it must not be mistaken for *cancer of the œsophagus* and *aneurism of the aorta*, two divergent conditions which may have pulmonary symptoms simulating phthisis.

Gangrene of the Lung.

Gangrene is a rare disease of the lung, and, like abscess, always secondary. It may be produced by any cause which so obstructs the circulation that a portion of the lung dies in bulk. The gangrene may be circumscribed or diffuse: it results most frequently from pneumonia, but may be due to injury, to a general septic condition, or to embolism. It is rather frequently met with in the insane, possibly owing to particles of food which have found their way into the lung. Aspiration bronchopneumonia, bronchiectatic and tuberculous cavities, sometimes lead to gangrene. Gangrene in the lung, as elsewhere, occurs in diabetes.

Symptoms. When it occurs in the insane or is of embolic origin, gangrene of the lung may remain latent, and in septicæmia it may be overlooked, on account of the general symptoms. In well-marked cases, however, the symptoms are characteristic. Symptoms and physical signs of pulmonary disease precede the specific symptoms of gangrene. With the onset of a moderate *fever*, *hæmoptysis* may occur at once or be preceded by the *expectoration* of a brownish purulent *sputa* having a most intense and persistent gangrenous odor. It contains fragments of lung tissue, altered blood, and putrid débris. (See Sputum.) It separates into the three characteristic layers in a conical glass. The *fetor of the breath* and the characteristic sputum are diagnostic.

Physical Signs.—The disease usually occupies the lower or middle lobe of the lung. The physical signs are those of *cavity*. The disease could with difficulty be distinguished from abscess were it not for the characteristic sputum, though in gangrene there is greater tendency to a general septic condition, with profuse sweats and collapse.

Abscess of the Lung.

Abscess of the lung may originate in causes outside the lung or in causes within the lung. To the former class belong those produced by suppurating bronchial glands, abscess of the mediastinum opening into

the lung, cancer of the œsophagus with ulceration, and abscess of the liver, suppurating hydatid cyst, or subdiaphragmatic abscess in general, bursting into the lung. Intrapulmonary causes are tubercle, septic emboli, in which case the abscesses are multiple and subpleural, and pneumonia. In the aspiration form of lobular pneumonia abscesses occur. Rarer causes are the presence of tumors and obstruction of the bronchi.

Abscess of the lung is therefore always secondary. Its *diagnosis* depends upon the demonstration of a *consolidation* in which a *cavity* subsequently forms, taken in connection with the history pointing to a definite cause. The *sputa* are copious, purulent, often odorless, sometimes offensive, but always without the fetor of gangrene. They contain elastic fibre, but no bacilli except in tuberculous cases. (See Sputum.) In embolic abscess the signs of pleural friction can only be detected at times. Of course, the constitutional symptoms of suppuration are present.

THE DEGENERATIONS.

Emphysema of the Lung.

Emphysema consists in an "excessive, permanent, and unnatural distention of the air-cells," or in "extravasation of air into the interlobular or subpleural cellular tissue." (Laennec.)

Emphysema may be unilateral or bilateral. Local and unilateral forms are usually compensatory. Bilateral emphysema may be hypertrophic or atrophic.

It is more common in men than in women. Its symptoms are more common in childhood and after middle age. Two factors are essential in its causation. First, defective development of the elastic tissue of the lungs. Second, increased intra-alveolar air-pressure. The latter is due to a number of causes. In childhood, no doubt, nasal and nasopharyngeal obstructions are operative. In adults occupations which necessitate continuous and severe muscular effort, especially if coupled with forced expiration with closed glottis, act as causes. Such occupations are blacksmithing and playing upon wind instruments. Diseases which cause much coughing or respiratory effort, such as chronic bronchitis and whooping-cough, act in the same manner. Chronic mitral valvular disease and the lessened elasticity of the lung tissue of advancing age both favor congestion of the lung, and thereby predispose to emphysema. The disease is hereditary; several members of a family may be affected. It occurs in many in childhood, is in abeyance in adult life, and reappears in old age.

Symptoms. The prominent symptoms in hypertrophic emphysema are dyspnœa, cyanosis, and cough, with expectoration from associated bronchitis. There is no fever. The *dyspnœa* is in proportion to the degree of emphysema, and is aggravated by the coexistence of bronchitis, asthma, and eccentric hypertrophy of the right ventricle, which are very frequent complications in cases of long standing. When the degree of emphysema is only moderate, dyspnœa is not complained of except upon climbing or walking briskly, or after a hearty meal. But when the degree of em-

PLATE XXIV.

FIG. 1.—Anterior Aspect.

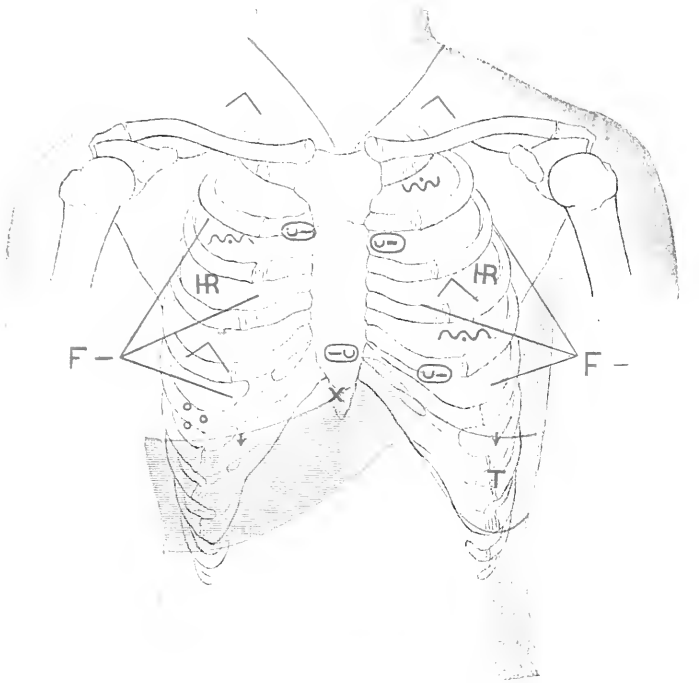
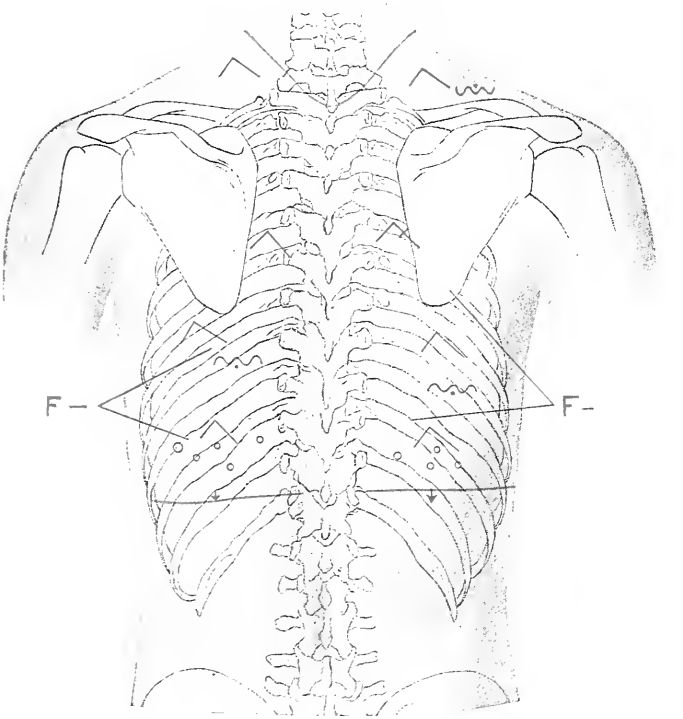


FIG. 2.—Posterior Aspect.



Emphysema.

Hyperresonance. Enlargement of lungs and diminished respiratory movement of margins. Diminished fremitus. Signs of bronchitis.

physema is great, dyspnœa is constant; it interferes with all exertion, frequently necessitates orthopnœa, and prevents continuous speech, so that patients speak in broken sentences or syllables.

Cyanosis is marked. The livid lip is common in asylums for old men. The face is of a dingy pale color, but becomes bluish on exertion. The extremities are also dusky, and the blueness is general in severe cases. This cyanosis, the round shoulders, and the drawn, chronically anxious expression, if I may so term it, make it easy to pick out the emphysematous subjects in a ward of chronic cases.

Respiration is not accelerated, and may be diminished in frequency. It is often accompanied by *wheezing* when chronic bronchitis coexists.

The *cough* varies greatly in frequency; it may be altogether absent, since its presence simply indicates an associated bronchitis. This bronchitis may for years be present only in the winter. In children it may be associated with asthma. It may arise on changes of the weather; finally it becomes chronic. The expectoration is that of chronic bronchitis (*q. v.*). It is rarely stained with blood.

The general health suffers by loss of strength and capacity for physical and mental work, rather than by loss of flesh.

Physical Signs. (Plate XXIV.) The physical signs of emphysema depend upon its degree and upon whether it is complicated with chronic bronchitis or not.

Inspection. The patients are large-chested, stoop-shouldered, and short-breathed, and have an anxious expression of countenance. In well-marked cases the *chest* is *barrel-shaped.* (See under Inspection.) There is *little movement of the chest* in respiration, because the lung is already in a condition of full inspiration (expiratory dyspnœa).

Palpation. *Vocal fremitus* is usually diminished.

Percussion. The *percussion-note* is abnormally *clear* and may even be *tympanitic. Hyper-resonance* is typical of the disease. When the distention is extreme, the note may be woodeny. The lungs are enlarged. The *heart-dulness* becomes *obliterated* by the overlapping lung. The upper margin of the liver falls one or two interspaces below normal. The resonance extends higher above the clavicles than normal.

Auscultation. The *inspiration* is found to be *distant* and *feebler* than normal, while the *expiration* is *prolonged,* and may become three or four times the length of the inspiration. *Grazing* or *rubbing sounds* have been described and attributed to the friction of distended vesicles against the pleura. Other adventitious sounds are due to an associated bronchitis, pleurisy, or tuberculosis. But bronchitis is such a common accompaniment of emphysema that the *râles* of the former become almost symptomatic of the latter. Their character in emphysema does not differ from that in chronic bronchitis (*q. v.*).

Physical Signs of the Heart. The *apex-beat* is *absent.* There is *epigastric pulsation* or systolic shock. The normal area of heart-dulness is encroached upon by the distended lung, and the *heart* itself is *pushed to the right,* the apex-beat being frequently at the xiphoid cartilage. If the emphysema attains a very high degree, there may be no perceptible dulness, except on very strong percussion over the cardiac region. The

heart-sounds appear *feebler* and *more distant* than normal. The right ventricle becomes dilated and hypertrophied as the result of the pulmonary congestion produced by emphysema. The *pulmonary second sound* is *accentuated*. A *tricuspid regurgitant murmur* may be heard. Venous congestions are common in the later stages. Albuminuria is common. Œdema of the feet and limbs may occur, but general anasarca is rare.

Diagnosis. This is based upon the history (heredity, occupation, long duration), the occurrence of dyspnœa and cyanosis, and of winter cough or chronic bronchitis, and upon the physical signs.

Emphysema can be distinguished from *pleural effusion* and from *aneurism*, which may cause dyspnœa, by the universal hyper-resonance on percussion. Pleural effusion, which also causes bulging, is usually unilateral, and the percussion-note is flat. The area of dulness of the heart and aorta is diminished in emphysema.

Pneumothorax, which most resembles emphysema in its physical signs, develops suddenly, affects one side, and gives a hollow, tympanitic note on percussion. The succussion-splash, metallic tinkling, and coin-test have no counterpart in emphysema; moreover, the antecedent history and mode of development are different.

Atrophic emphysema is due to the degeneration of age. The lung is reduced in size, the diameters of the chest are lessened, the ribs are oblique, and there is atrophy of the chest muscles. The patients have dyspnœa and exhibit other signs of senility.

In *interlobular emphysema* the physical signs are the same as those of vesicular emphysema, but it develops suddenly and is liable to be followed by emphysema (intercellular) of the neck, which on palpation gives a peculiar crepitation. The friction-sound and crackling which have been described as occasional adventitious sounds in vesicular emphysema are more commonly heard in the interlobular form. It is caused by rupture of the air-cells, and hence occurs in diseases in which a great strain is put upon them—especially in whooping-cough, but also occasionally in pulmonary hemorrhage and pneumonia; violent coughing and laughing, and great straining, as in child-labor, are capable of producing it.

Bronchiectasis.

Dilatation of the bronchi occurs secondarily to affections which tend to weaken the walls of the tubes and to lessen their elasticity. Hence it is found in chronic bronchitis with emphysema, in chronic phthisis, in catarrhal pneumonia in children, in chronic obstruction from external pressure or foreign bodies. (See Obstructions.) It also occurs when the lungs contract in fibroid pneumonia or in pleural thickening. It occurs in two principal forms: the *simple*, in which the affected tubes are uniformly dilated; and the *saccular*, in which larger or smaller pouches are formed. It is commoner in males than in females, and probably begins most frequently in adult or middle life. One lung only is affected in about one-half the cases, and when both lungs are affected (chronic bronchitis and emphysema), it is not often to the same degree.

Subjective Symptoms. These consist of cough, expectoration, and a

PLATE XXV.

FIG. 1.—Anterior Aspect.

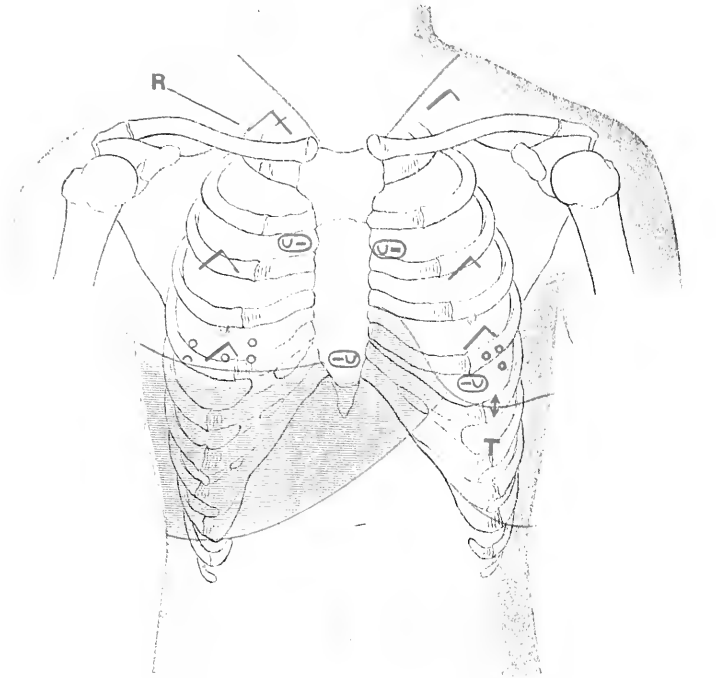
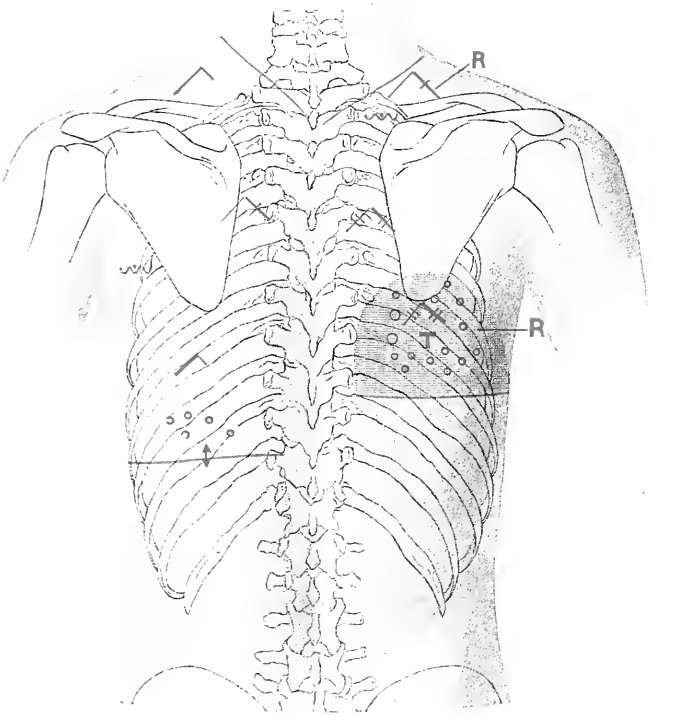


FIG. 2.—Posterior Aspect.



Bronchiectasis.

Chronic pleurisy with induration of the right lower lobe and bronchiectasis. Vicarious emphysema of the left lung. Bronchitis.

variable amount of dyspnœa. Eventually there may be some loss of flesh and strength.

The *cough* is usually paroxysmal. It may occur only in the morning after the dilated tube fills and it may follow change in position. A paroxysm is followed by copious expectoration, sometimes amounting to a pint and a half in twenty-four hours. The *sputa* are grayish brown and mucopurulent, faintly or extremely fetid. They contain mucus, pus, casts of the tubules, and various salts. Charcot-Leyden and fatty crystals, vibrios, leptothrix, and bacteria (Fox) can be found on microscopical examination. Elastic fibres are found only if the tubes are ulcerated. In a conical glass the sputum separates into three layers—a frothy brown top, a thin mucoid layer in the middle, and a granular layer below. Hemorrhage may occur periodically even when tuberculosis is absent. It was a feature in 14 of the 35 cases reported by Fowler and Godlee. *Dyspnœa* is not usually severe, except when the dilatation is complicated by disease of the heart or lungs, or by an acute attack of bronchitis.

Physical Signs. (Plate XXV.) The physical signs differ according to the extent and variety of the dilatation. In simple dilatation there may be nothing different from the signs found in chronic bronchitis, except a tendency to more *bronchial respiration*, with *râles* having a metallic quality. Percussion will vary according to the degree of alteration of the lung tissue surrounding the affected bronchi, and according to the extent of the dilatation and its proximity to the surface. In the *simple forms* the *percussion-note*, if altered, is somewhat *less resonant* and *higher in pitch*, whereas in *saccular dilatations*, favorably situated for percussion, the note is *tympanitic* if the pouch is empty. On auscultation in simple dilatation the *breathing* approaches the *bronchial* and is accompanied by bronchial râles. In saccular dilatation the sounds are practically those of a cavity, respiration varying from bronchial to *amphoric*. *Vocal resonance* and *tactile fremitus* are usually both *increased*, but the latter may be diminished.

Diagnosis. The diagnosis of simple dilatation from *chronic bronchitis* may be impossible, but copious and fetid expectoration indicates the former. The diagnosis of the saccular form from *tuberculosis* of the lung *with cavity* is difficult. Wilson Fox says that severe cases are usually associated with consolidation of the lung or with tubercle; but even without the presence of the latter they often present phthisical symptoms—retraction of the chest, with the physical signs of excavation, pains in the side, hæmoptysis, pyrexia, nocturnal perspiration, and diarrhœa—which may all coexist with only an induration of the lung and dilatation of the bronchi. The diagnosis must be made by noting the persistence of the physical signs, which change but little and are not progressive as are those of tuberculosis; the protracted course of the disease; the character of the sputum, and the comparatively slight impairment of the general health.

The Morbid Growths of the Lungs.

Cancer of the Lungs. The new growth may be primary or secondary. The latter is most common. Of primary cancer, the epithelioma is most

common; encephaloid and scirrhus come next. Secondary new growths succeed disease in the abdominal organs, the genito-urinary tract, the bones, the breast, and the eye.

Symptoms. The general symptoms of carcinoma accompany the thoracic symptoms. *Chest pain, dyspnœa, cough,* and a peculiar expectoration belong to the latter. The pain is due to associated pleurisy; the dyspnœa is paroxysmal. (See Dyspnœa from Pressure on Bronchi.) The *expectoration* is dark like prune-juice. Signs of intrathoracic pressure are seen. The external thoracic veins are enlarged. The face and arms may be cyanosed, or one arm only may be affected. The heart may be dislocated, the trachea changed in its course; compression of trachea and bronchus causes dyspnœa.

Physical Signs. In primary cancer the affection is unilateral; in secondary forms, bilateral. The *physical signs* are those of *pleural effusion* or of local *consolidation*. The consolidation may be massive and not partake of the shape of a lobe. Often signs of effusion and consolidation are combined (enlargement, immobility, absent fremitus, but bronchial breathing). In the secondary forms the disease is bilateral. The signs are mixed. They indicate diminished air in the lung structure. Care must be taken not to overlook the pleural effusion which accompanies the process, the removal of which gives temporary relief. In both forms external lymphatic glands, particularly the cervical, may be enlarged.

Diagnosis. The diagnosis is based upon: (1) the age (after forty); (2) the occurrence of emaciation; (3) the duration of the disease, often rapid, rarely beyond eight months; (4) the presence of primary disease elsewhere; (5) the presence of moderate fever; (6) the signs of intrathoracic pressure; (7) the involvement of lymphatic glands; (8) the occurrence of irregular areas of consolidation and of pleural effusion, alone or combined; (9) the characteristic expectoration; (10) dyspnœa due to pressure on the bronchus or trachea; (11) the absence of bacilli from the sputum.

An effusion can often be recognized only after puncture. Hæmothorax is not necessarily present.

Sarcoma. This is sometimes primary, but more often secondary. The *symptoms* and the *physical signs* are much the same as in carcinoma. The age (usually under forty) may be an aid in *diagnosis*.

Gross Parasites of the Lungs.

Hydatid Disease of the Lungs. The lungs are affected in about 11 per cent. of the cases of hydatid disease.

Symptoms. According to Wilson Fox, these consist of *dyspnœa, pain in the chest, cough,* occasional *hæmoptysis,* and sometimes the *expectoration* of hydatids, the *sputa* being otherwise bronchitic, or presenting the characteristics of pneumonia or gangrene when these complications are present. Gradually *weakness* increases, sometimes with *pyrexia*, which, when combined with *emaciation*, may impart to the case a considerable resemblance to phthisis. *Pressure-symptoms* occasionally occur.

Physical Signs. These are either those of *consolidation* of the lung or of *pleural effusion*, together with certain peculiarities depending on the

size and site of the tumor. Graham states that they are more frequent in the right lung and more common at the base, causing marked bulging of the thoracic wall. When the physical signs are those of pleural effusion, localization of the fluid to a definite area takes place, and hence is not related to the shape of the pleural cavity. The *breathing* may be *tubular;* there is condensed lung between the hydatid and the thoracic wall.

Diagnosis. The symptoms present—cough, dyspnœa, anæmia, emaciation, and clubbing of fingers—too often lead to the diagnosis of *phthisis.* Hæmoptysis occurs in many cases. The temperature is normal—an important point in diagnosis. If the cyst ruptures, the sputum is diagnostic. Complications often mask the diagnosis. It must be distinguished from *pleurisy,* localized *empyema, pulmonary abscess,* phthisis, *actinomycosis,* and *mediastinal tumors.*

DISEASES OF THE PLEURA.

The large lymph-structures which cover the lung and line the inside of the thorax are often the seat of disease. It is usually of an inflammatory nature.

Acute Pleurisy.

Pleurisy, or pleuritis, is the most common affection of the pleura. It may be, as to its distribution, bilateral or unilateral; as to its extent, local or general; as to the nature of the inflammation, plastic, serous, or purulent. The inflammation is rarely primary. It may arise in the course of general disease, or it may be the result of the extension of inflammation, chiefly of an infectious nature, from neighboring structures.

1. *Disease* of the *ribs* or *vertebræ,* of the *mediastinum,* of the *aorta,* and of the *œsophagus,* gives rise to various forms of pleurisy, depending upon the nature of the primary affection.

2. *Diseases below the diaphragm; abscess of the liver; perforative inflammation* of other viscera adjacent to the diaphragm; *abscess of the spleen or pancreas; pus in the pelvis or about the appendix,* may give rise to purulent pleurisy by the pus burrowing upward or by infection through the lymph-channels.

3. *Disease of the lungs.* In the large majority of cases pleurisy in some form occurs in the course of pulmonary disease. In all surface inflammations of the lungs there is associated pleurisy. It is seen in pneumonia, in tuberculosis, in gangrene, and in abscess.

Pleurisy may be *simple* or *purulent.* Empyema is always due to infection from the exterior, as the ribs; from the lungs (pneumonia); suppuration below the diaphragm; or to general infective processes, as septicæmia, pyæmia, and tuberculosis.

The *general diseases* in the course of which pleuritis arises are usually infective, or of such nature as to cause irritating products to circulate in the blood. Of the former, the most common is tuberculosis; the next most common are septicæmia and scarlatina; while to the latter class belong Bright's disease, gout, diabetes, rheumatism, and scurvy. Puru-

lent pleurisy is more common in children than in adults; in males than in females; and more common in tuberculous pleurisy and pyæmia than in rheumatism and Bright's disease.

Three stages usually occur in acute pleurisy, although the morbid process may be arrested in the first stage.

The First Stage—Dry Pleurisy.

Symptoms. The onset of the disease is usually abrupt, and is marked by *fever*, which may or may not be preceded by *chill*, and is followed by pain in the side, *dyspnœa*, and *cough*. The *pain* is sharp, stabbing, or tearing in character, and is usually, but not always, referred to the seat of pleurisy. This is most frequently on a level with the nipple, or a little below this, and more often anteriorly or in the axilla than posteriorly. The pain is caused by the rubbing together of the inflamed surfaces of the pleura, and hence is excited by respiration and cough. For this reason the patient is inclined to *restrict the motion* of the affected side as much as possible; he does this by leaning over toward that side and by pressing his elbow in against the chest-wall. Pain is usually the first symptom noticed by the patient. The *cough* is dry and painful. Fever is moderate.

Physical Signs. The physical sign in primary cases is a *friction-sound* heard on inspiration and expiration. This friction-sound may be a nest of fine, dry, crepitant râles, which are very superficial, and appear to be just under the ear; or a coarse rubbing sound, heard over a larger surface, and resembling a bronchial rhonchus, from which it can be distinguished by its persistence after the patient has coughed. The lungs themselves present nothing abnormal.

If the inflamed surfaces become glued together by plastic lymph, recovery usually occurs very soon, though pain often persists for a long time in lessened degree, and the pleurisy is liable to be relighted.

The Second Stage, or Stage of Effusion. When effusion takes place, the two layers of the pleura become separated; hence pain and friction-sound cease, and physical exploration shows that a collection of fluid intervenes between the chest-wall and the lung.

Symptoms. During this stage there occur slight or moderate *fever*, sometimes intermittent in character, with recurrent *chills;* considerable *dyspnœa*, occasionally amounting to *orthopnœa* when the effusion is very extensive; and dry *cough*, which adds greatly to the dyspnœa. There is frequently some evidence of insufficient oxygenation of the blood; when this amounts to *cyanosis*, the condition is one of great danger. The *urine* presents changes in amount; in advancing effusion lessening very much; but increasing in amount with the decline of the fluid. Pleurisy may be complicated with bronchitis, pneumonia, and pericarditis.

Physical Signs. (Plates XXVI. and XXVII.) In this stage are seen (1) *enlargement of the affected side*, increase in semicircumference, with fulness of interspaces; (2) *diminution of movement;* (3) *absence of vocal fremitus and resonance;* (4) *dulness* or *flatness* (deadness) on percussion, with *great increase in the resistance* to the pleximeter finger; (5) *absent or greatly diminished respiratory murmur;* (6) *displacement of organs.*

The dead *percussion-note* being caused by fluid, it follows that its upper

PLATE XXVI.

FIG. 1.—Anterior Aspect.

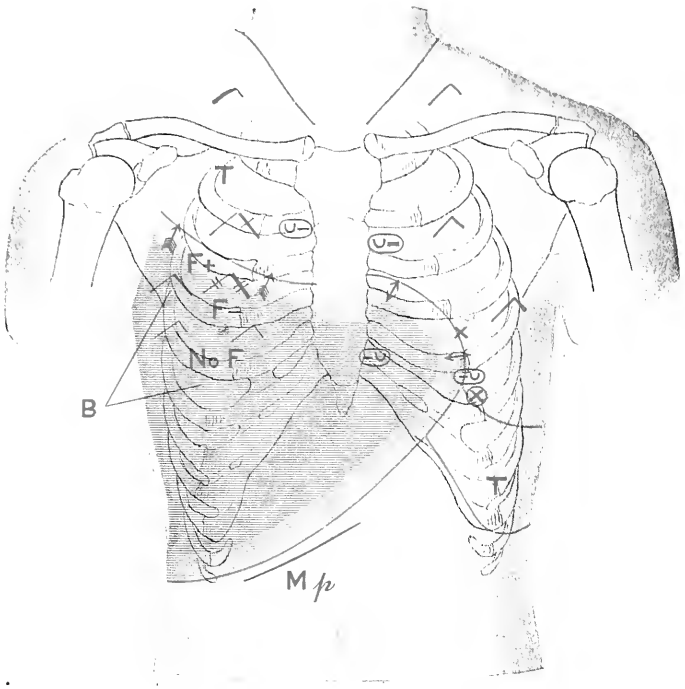
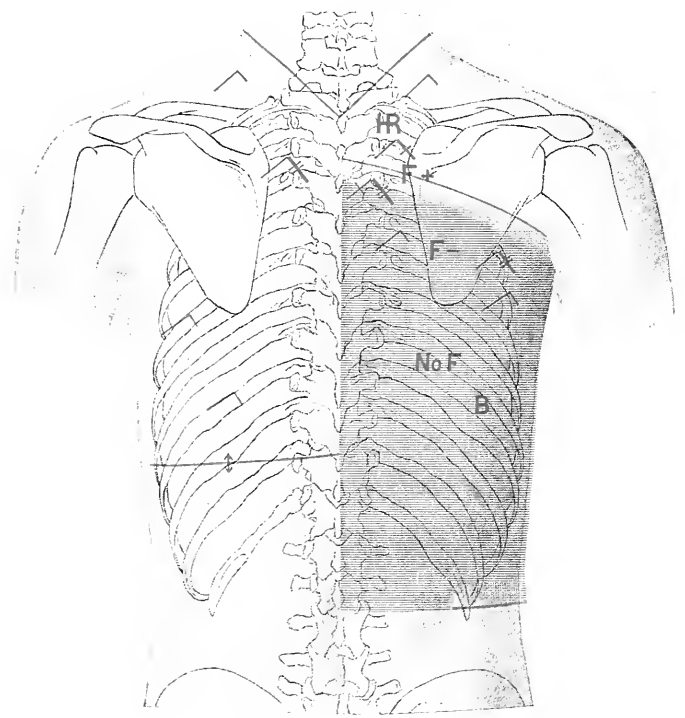


FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (right-sided).

PLATE XXVII.

FIG. 1.—Anterior Aspect.

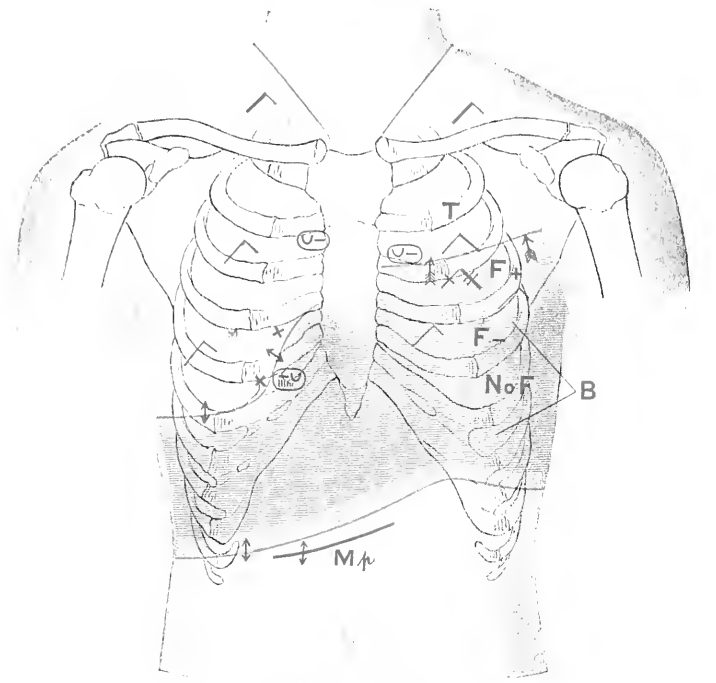
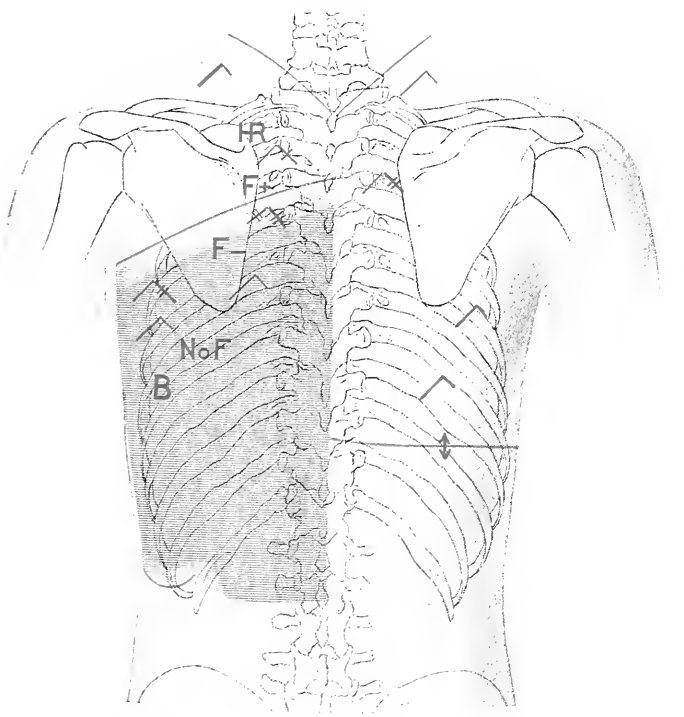


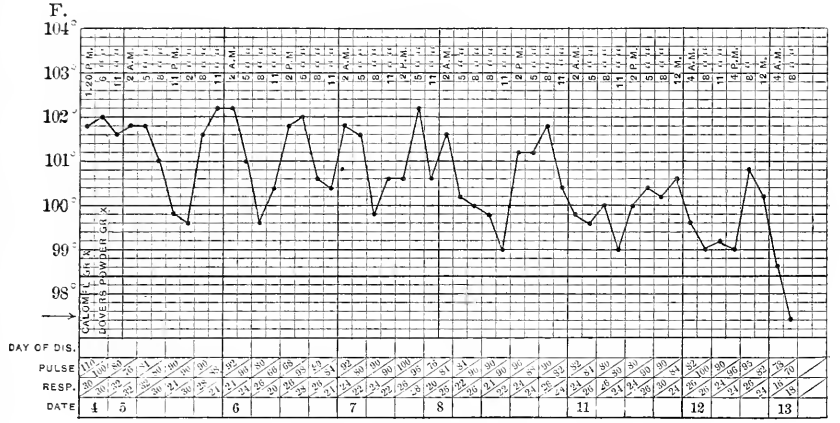
FIG. 2.—Posterior Aspect.



Pleurisy with Effusion (left-sided).

level will change with the position of the patient if the fluid is free. If the upper level is at the third interspace when the patient is sitting up, it will fall to the fourth or lower when he is lying down. This change of level can not be appreciated when the effusion is very large. Moreover, above the line of dulness the percussion-note is hyper-resonant or tympanitic—*Skoda's resonance*. Toward the spine on the affected side there may be partial resonance and bronchial breathing, because here the lung is compressed against the vertebræ. In large effusions the tympanitic resonance in the second interspace does not change when the mouth is opened—that is, "Williams' tracheal tone" can often be elicited. The upper limit of dulness in large pleural effusions is higher at the spine and slopes downward, and is lowest in front. This parabolic line is only obtained when the patient is in the erect posture. In moderate effusions

FIG. 347.



Pleurisy with effusion. Recovery. (Two days omitted.) (Original.)

the line of dulness is lowest near the spinal column, rises in the middle of the scapula and slopes downward, assuming the shape of the letter S as it passes toward the front. (Garland.) The patient should take deep breaths before the percussion is performed. At the left base in front the semilunar space is obliterated, dulness continuing to the margin of the ribs. In small effusions the dulness may be limited by the posterior axillary line, resonance being present in the lateral and anterior regions.

On *auscultation* below the upper level of the effusion, posteriorly, the voice frequently has a metallic quality resembling the bleating of a goat —*ægophony*. It occurs usually when the effusion is moderate, and may be heard only over a limited area. It is commonly heard at or above the angle of the scapula. Bronchophony may be heard when tubular breathing is present.

While the respiratory murmur is, as a rule, absent, breath-sounds may be heard, and are then weak and distant, or bronchial. In such cases there may be adhesions. Bronchial breathing may be present along the spine in small effusions, and in large effusions in the interscapular region. Bronchial breathing, tubular in character, is said to be almost constant in

children. It may also occur when pneumonia coexists. In one of the cases in my ward the signs were like those of a large cavity at the right base, but the immobility, the absent fremitus, the enlargement, and the exploratory puncture disproved its presence.

At the level of the fluid a friction-sound may persist. Above the level of fluid anteriorly, the breath-sound may be bronchial or bronchovesicular, associated sometimes with fine râles, due to compression and slight œdema.

Displacement of Organs. If the effusion is on the left side, the mediastinum and heart become displaced to the right, and the apex-beat may be found in the epigastrium or even to the right of it. The occurrence of displacement of the heart must also be judged by the position of maximum intensity of the heart-sounds, as the heart may be behind the sternum. At the same time the semilunar space (Traube's line) is lower than usual or entirely effaced. On the left side inaction of the diaphragm may be observed, and the tissues at the costal margin fall in with each inspiration. If the effusion is on the right side, the diaphragm, and with it the liver, is depressed, and the mediastinal contents are moved to the left.

Empyema. The above-mentioned physical signs apply chiefly to serous effusions. They are also present in effusions of pus. Other physical phenomena, however, and different general symptoms distinguish the two kinds of effusions, although it must be confessed that aspiration must often be resorted to before a positive diagnosis can be made.

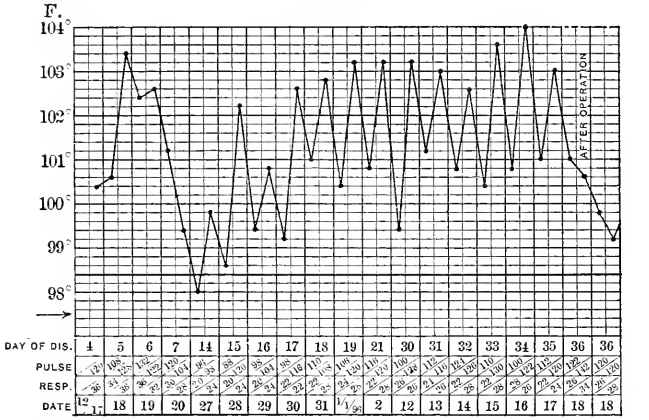
Symptoms. The general symptoms are more marked in empyema than in simple serous effusion. The *temperature* is higher from the onset. It soon becomes intermittent or remittent. *Chills* or chilliness may attend the beginning of each febrile paroxysm, and *sweats* occur with the daily fall of temperature, or at irregular periods during the twenty-four hours. The *heart's action* is more rapid and the *pulse* more feeble, soon becoming dicrotic. Examination of the *urine* may aid in the distinction of the two forms of the effusion. *Albumosuria* occurs in purulent pleurisy. It must be remembered that albumosuria occurs in suppuration from other causes. Thus, in phthisis with suppuration of a cavity, pleural effusion may develop. The albumosuria that attends the primary process must not be mistaken for that which occurs in empyema. *Indican* is also present in excess in the urine in suppuration. Before a positive conclusion is arrived at, two or more examinations of the urine should be made. Examination of the *blood* may aid in arriving at a conclusion. In purulent effusion there is usually leucocytosis.

Physical Signs. The physical signs of empyema are the same as those of other effusions within the pleura. In addition, especially in children, local *œdema* of the chest-wall may be found. Another sign was pointed out by Bacelli, and is held by others to be of diagnostic significance. In purulent effusions the fremitus produced by the *whispering voice* is not transmitted to the hand laid over the effusion, whereas in serous effusions such vibrations are transmitted. In lobulated empyema the diagnosis is very difficult. In one of my cases dulness continuous with that of the heart extended to the second rib and laterally to the post-axillary line. The dulness occupied three interspaces. Additional physical signs were

immobility, prominence of interspaces, localized above the heart, absent fremitus and resonance. There were no breath-sounds, but an abundance of râles, apparently very superficial. The râles complicated the physical signs. Martin removed 2 ounces of pus from a small abscess above the heart and between the lobes.

In empyema a local area may become more prominent and the surface assume an inflammatory appearance. It is an indication of discharge of the abscess through the chest-wall. It is usually found in the fifth interspace in front, or below the angle of the scapula behind—*empyema necessitatis*. (For a microscopical and chemical description of the "Effusion within the Pleural Sac," and of the morphological elements of the purulent effusions, see Chapter XLV., Part I.)

FIG. 348.



Empyema following pneumonia. (Fever absent from seventh to fourteenth day.) (Original.)

Notwithstanding the positive physical signs of effusion the character of the effusion may not be recognized until perforation into a bronchus has taken place. The peculiar character of the expectoration that attends this accident is described in the section on Sputum.

Hydrothorax. This is an accumulation resulting from a transudate. (For character of the fluid, see Chapter XLV., Part I.) It occurs in the course of diseases which produce anasarca, as failing organic heart disease, chronic Bright's disease, and debilitating diseases, as scurvy. Locally, it may attend carcinoma of the pleura or obstructive disease of vessels within the mediastinum.

Symptoms. The general symptoms belong to the primary disorder. *Dyspnœa* may develop gradually and even amount to orthopnœa. It is distinguished from inflammatory effusions by the character of the fluid, by the absence of the general symptoms of inflammation, by its insidious development, and by its bilateral distribution.

Physical Signs. The physical signs of hydrothorax are those of *effusion* in acute pleurisy.

Hæmothorax. The transudation of blood into the cavity of the pleura occurs rarely from the rupture of an aneurism into the sac. The fluid is then pure blood. Serous effusions in which a large amount of

blood is found point to primary carcinoma of the pleura, or to tuberculous disease. Both specific processes of this serous membrane may occur, however, without transudation of serobloody fluid.

Thickened Pleura. Chronic inflammation, with thickening of the pleura from excessive development of connective tissue, occurs in tuberculosis and in cases of combined pleuritis and peritonitis. The thickening of the pleura is usually more marked at the base.

Subjective symptoms of cough and dyspnœa are present. The degree of *cough* depends upon the condition of the lung. If there is bronchitis or tuberculosis, the cough is excessive. The amount of *dyspnœa* depends upon the degree of compression of the lung by the thickened pleura.

Physical Signs. (Plate XXVIII.) These are pronounced, and are those of effusion, but without enlargement of the chest. There are marked *contraction* and *diminution in movement* of the affected side. The *fremitus is absent.* There is *dulness* on percussion, or even *flatness.* The *breath-sounds* are *distant* or are *absent.* Along the vertebræ, especially opposite the angle of the scapula, *bronchial breathing* may be heard.

Tuberculous Pleurisy. The affection may be acute or chronic. It may occur primarily, it may be a part of general tuberculous infection, or it may occur secondarily to disease of the lungs. It may give rise to all forms of the inflammatory process, to (1) dry pleurisy; (2) pleurisy with effusion; (3) pleurisy with great thickening. Often the distinction between tuberculous pleurisy and pleurisy due to other causes can not be determined positively. If the pleurisy be associated with tuberculosis in other organs, or if the patient be of tuberculous habit and be exposed to infection, or if there has been a history of previous tuberculosis, the pleuritic infection is probably of tuberculous origin. If the affection is bilateral and associated with peritoneal inflammation, and if at the same time no other cause exists for serous membrane inflammation, the probability of its tuberculous origin is very strong.

Pulsating Pleural Effusion. Wilson has made the most recent studies of this rare affection. The effusion within the pleura pulsates synchronously with the ventricular systole; the pulsation is detected usually by inspection and palpation. In some instances its presence is only determined by palpation. It may be confined to two or three interspaces, or occupy the anterior aspect of the thorax and the axillary region on the left side. Rarely the pulsation is behind. It is usually situated on the left side. The original effusion is purulent in the large majority of cases. The physical signs and general symptoms of empyema are present. Nevertheless, the disease simulates aneurism of the aorta. The latter affection, however, is accompanied by vascular symptoms and physical signs in the course of the aorta. Pulsating empyema is distinct in movement from the pulsation of the aorta and occupies a different anatomical site.

Diaphragmatic Pleurisy.

In diaphragmatic pleurisy there is intense *pain in the epigastrium.* Guenеau de Mussy[1] regards a pain along the tenth rib, extending from

[1] Arch. gén. de Méd., 1853, vol. xi. Quoted by Fox.

PLATE XXVIII.

FIG. 1.—Anterior Aspect.

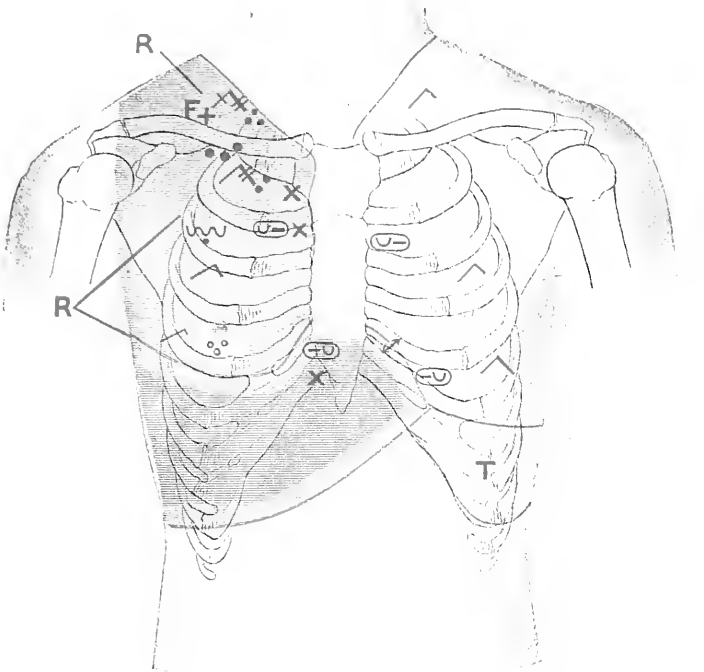
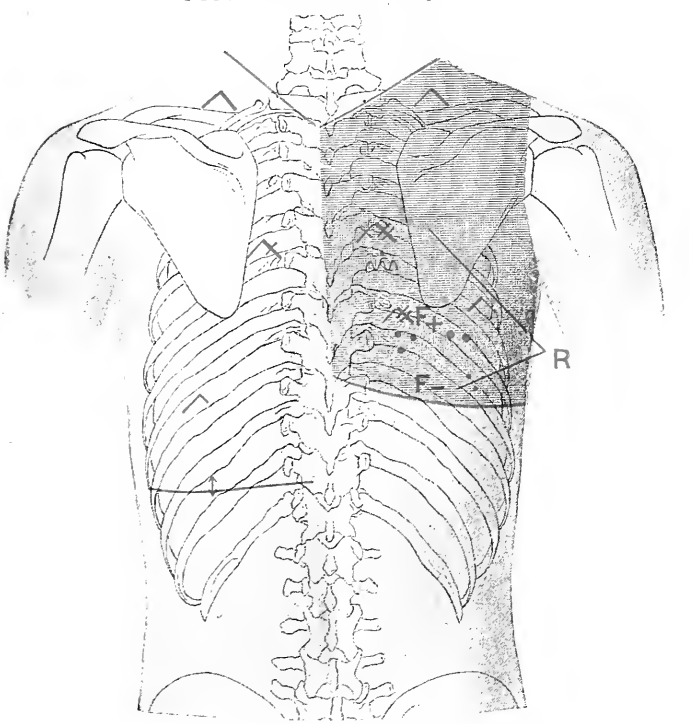


FIG. 2.—Posterior Aspect.



Fibroid Phthisis with Chronic Pleurisy.

Heart drawn toward the right and aorta uncovered by retraction of lung margin. Vicarious emphysema of left lung.

the anterior extremity to the sternum and xiphoid cartilage, as pathognomonic. Other symptoms are *nausea, vomiting,* and *hiccough.* The *dyspnœa* often amounts to orthopnœa, or the patient sits stooping forward. The anxiety of the patient is very great. The *fever* is usually higher than in ordinary pleurisy, and there may be delirium. Effusion may lessen the pain. Peritonitis may occur at the same time or be secondary to the pleurisy.

Diagnostic Features of Acute Pleurisy. The special features of diagnostic importance that are observed in the course of pleurisy are the subjective symptoms of pain, dyspnœa, cough, and fever, the physical signs of effusion within the pleura, and the results of exploratory puncture.

Symptoms. *Pain:* The pain is short, sharp, lancinating, and is usually recognized quite readily by its character and location. It must be distinguished from the pain due to pleurodynia and intercostal neuralgia. The pain of pleurisy is associated with cough and is increased by breathing. It causes diminution of movement of the affected side. The patient is compelled to sit up in bed, or lie on the side which is the seat of pain. *Cough:* In the first stage the cough is short, suppressed, dry, and painful. It is constant. In the second stage it changes in character. There is no pain, there is no expectoration. It is frequent and irritating, and of a peculiar sound which is difficult to describe, and yet, when once heard, is most suggestive in subsequent cases. It is short and lacks resonant quality, as if the fluid in the chest stopped the sound-waves. *Dyspnœa* in the first stage is due to pain, in the second stage to the large effusion which encroaches upon the normal air-space. It is not diagnostic.

Physical Signs. The most decisive signs of pleural effusion are *diminution or absence of movement, enlargement of the affected side, absence of fremitus, flatness* on percussion, *fulness of intercostal spaces,* and the *displacement of organs.* The latter is of the greatest diagnostic importance in the distinction between consolidation and effusions. The results of *exploratory puncture* lead to positive conclusions usually, although it must not be forgotten that effusions may be loculated, and therefore missed by the aspirating-needle. Again, the enormously thickened pleura may intervene between the exudate and the surface of the chest, and prevent withdrawal of the fluid. Finally, effusions may complicate inflammatory processes, as pneumonia, tuberculosis, or abscess of the lungs. Securing fluid for diagnosis by aspiration, therefore, does not necessarily exclude these conditions, and hence, before the process is decided to be within the pleura alone, the sputum and other conditions must be taken into consideration.

Differential Diagnosis. Acute plastic pleurisy is diagnosticated from *acute pneumonia* by the friction-sound and the maintenance of the clear percussion-note and the normal respiratory murmur, with unaltered vocal resonance and fremitus. When effusion takes place, the chest is enlarged and immobile, especially on the affected side; the interspaces are filled out and the diaphragm is depressed; these changes do not occur in pneumonia. Moreover, the percussion-note in pleural effusion is flat, with greatly increased resistance; the shape of the upper line of dulness is

diagnostic; the respiratory murmur is feeble and distant, or entirely absent, except along the spine, where the compressed lung yields bronchial breathing, and also above the line of effusion, where the lung yields exaggerated breathing. In pneumonia, on the other hand, the percussion-note is dull, without greatly increased resistance, and the breath-sounds are bronchial. In addition, in pleurisy, the vocal resonance and fremitus are usually almost if not entirely absent, and posteriorly at the level of the effusion ægophony may be detected. In pneumonia, on the contrary, vocal resonance and fremitus are increased in intensity. In pleurisy with effusion the movable organs are dislocated and Traube's line is obliterated.

Finally, the fever of pneumonia is much higher and more continuous than that of pleurisy, the respirations are more frequent, the cough is looser, and in typical cases is followed by rusty sputa. (Compare the temperature chart in article on Pneumonia.) A crucial test is aspiration with a hypodermatic needle; in pleural effusion, serum is withdrawn; in pneumonia, a few drops of thick blood.

In *pleurodynia* there is also severe pain in one side; but the pain is more continuous than that of pleurisy, and consists of a constant aching or a burning sensation. It is made worse by twisting or turning, as well as by breathing. The side is also tender to the touch. The pain is not so sharply localized as that of pleurisy, and may leave one side and affect the other. It is unaccompanied by fever or friction-sound, and is frequently found in rheumatic subjects.

In *intercostal neuralgia* there is the same absence of fever and friction-sound. The pain, however, is sharply localized, as in pleurisy, but is of the darting, neuralgic character, and is associated with tenderness at the points of exit of the intercostal nerves. It is most common in women, especially in those having uterine disturbances. It is more frequent on the left side, and just beneath the mammary gland.

Chronic Pleurisy.

Chronic dry, or plastic, pleurisy is the result of an acute attack, or develops insidiously if tuberculous.

Physical Signs. Chronic pleurisy causes great *deformity of the chest* from contraction, and *compensatory emphysema* of the healthy lung. The *heart is dislocated* or can not be found on physical examination, because it is overlapped by lung or is drawn behind the sternum. There is considerable *spinal curvature, dislocation of the scapula, deformity of the shoulder*, and indrawing and overlapping of the ribs at the base of the chest.

Chronic Pleurisy with Effusion. This results from an acute attack of pleurisy, in which the fluid remains unabsorbed, or from a series of attacks.

Symptoms. So far as subjective symptoms go it may remain latent; patients so affected not infrequently go about their work with comparatively little dyspnœa. There may be an evening rise of temperature and acceleration of the pulse. Chronic effusions are more likely to be purulent in children than in adults. When empyema results, the fever

PLATE XXIX.

FIG. 1.—Anterior Aspect.

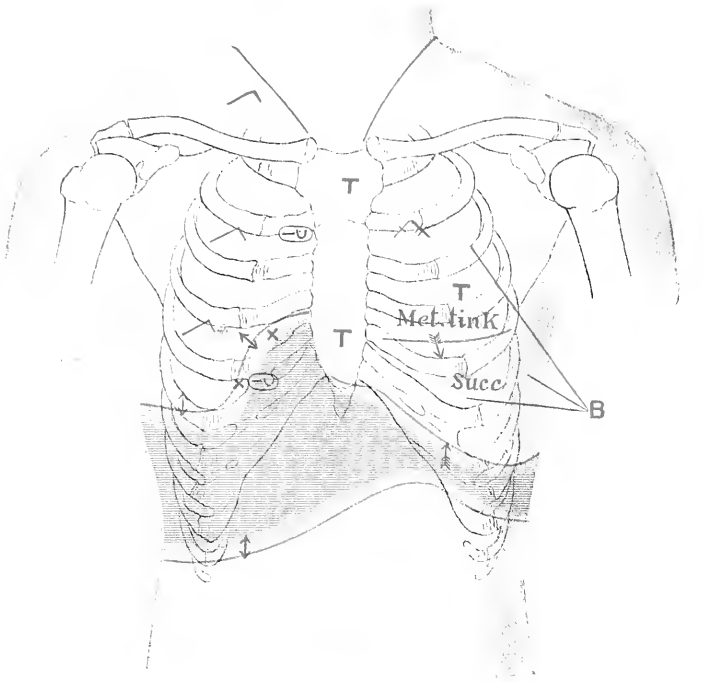
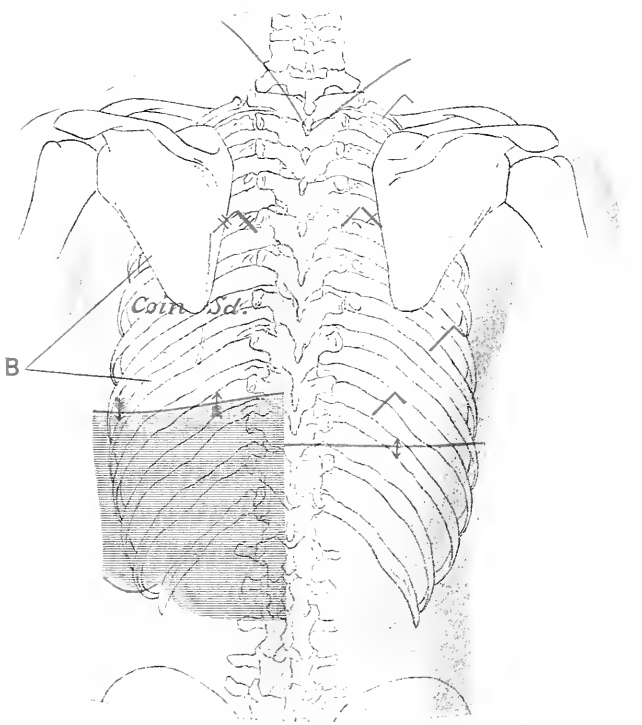


FIG. 2.—Posterior Aspect.



Pneumothorax (left-sided).

becomes hectic; there are chills and sweats, pyæmia develops, and death is likely to occur from some intercurrent suppuration, as cerebral abscess.

Physical Signs. The physical signs are the same as in acute effusion.

After chronic effusion the chest is rarely restored to its original shape, even if the effusion is finally absorbed. The affected side becomes motionless and retracted. In course of time the spine may be bent. The opposite lung becomes hypertrophied. The patient is usually in precarious health, liable to acute attacks of pain in the affected side, and liable also to be carried off by phthisis or some intercurrent affection. Rarely the patient may maintain good health; complete cure is even possible, with restoration of the retracted side to, or almost to, normal dimensions, especially in children.

Pneumothorax.

Pneumothorax consists in an accumulation of air in the pleural cavity, accompanied or followed by an outpouring of fluid, which may be serous or purulent, constituting respectively *hydropneumothorax* and *pyopneumothorax*.

Pneumothorax may originate: (1) in causes external to the chest, by perforation of the chest-wall and pleura; (2) in perforation of the lungs, bronchi, or œsophagus; (3) it may be caused by gases developed from an existing effusion.

The most frequent cause is tuberculous disease of the lung, and next, an empyema; out of 121 cases collected by Saussier, 81 were due to phthisis and 29 to empyema. Pneumothorax may occur very early in tuberculosis of the lung, and may even be the first symptom of that disease. (See cases referred to by Fox and recorded by Louis and Chomel.) The left side is affected not quite twice as often as the right; the disease is usually unilateral.

Symptoms. The onset of the condition is usually sudden. During a paroxysm of coughing or vomiting, or without immediate cause, there is an escape of air into the pleura, and in the majority of cases the patient at once complains of *acute pain in the chest* and *excessive dyspnœa* with great *dread of impending suffocation*. The patient often sinks into *collapse* from shock, but sudden death is rare. If the escape of air into the pleura is gradual, there will be less pain and dyspnœa.

Physical Signs. (Plate XXIX.) The *chest is distended*, especially on the affected side; the *percussion-note* is a *bell-like tympany* except when the distention is excessive and the air contained is under great tension, when the note is proportionately duller and higher in pitch; the *diaphragm is depressed* and the *heart displaced* unless adhesions prevent it. In left pneumothorax it may beat on the right side, the whole mediastinum being pushed to the right; in right pneumothorax the mediastinum may be pushed to the left nipple; hence there is resonance over the normal cardiac region. The pitch of the percussion-note may be raised when the mouth is closed, and lowered when it is open (*Wintrich's change of note*), and a *cracked-pot sound* can be elicited in some cases, but this occurs only when the communication with the pleura remains open.

A valuable sign of pneumothorax is the *coin-test*, or, as Trousseau named it, the *bruit d'airain*. A silver coin is laid upon the chest and struck with another, while the auscultator applies the stethoscope opposite to the point struck or over any part of the side distended by air. The ringing coin-sound is reproduced with great intensity. It is pathognomonic, and the outlines of the cavity can be traced by it.

When fluid is present, as it usually is, there will be the ordinary signs of a pleural effusion, which have been sufficiently dwelt upon. The fluid is more mobile in pneumothorax, however, than in simple pleurisy, so that its level changes more quickly with change of posture of the patient, and *Hippocratic succussion* is readily obtained. This movable dulness is a very valuable sign—indeed, being almost pathognomonic.

The lung being compressed against the spine by the air, as it is by the fluid in pleurisy, the *breath-sounds* are *feeble* or *absent*, except over the root of the lung, where the breathing is bronchial. But if the lung is not completely collapsed, *amphoric breathing* may be heard, the air-chamber of the pleura acting as a consonance-box; it may be heard with both inspiration and expiration, or only with expiration.

Metallic tinkling is a sound believed to be due to the vibration of bubbling bronchial râles re-echoed through the air-chamber, or to drops of fluid falling from above upon the surface of the effusion. Re-echoing, with metallic quality, may also accompany the heart-sounds, and in cases in which the respiratory murmur is amphoric the vocal resonance is of the same character. *Vocal fremitus* is generally *absent*.

Differential Diagnosis. Pneumothorax is most likely to be confounded with (1) emphysema; (2) tuberculosis of the lungs with large cavities; (3) cases of pleural effusion in which the lung is markedly hyper-resonant above the upper level of the fluid; and (4) abscess below the diaphragm containing air (*pyopneumothorax subphrenicus*).

Emphysema can be distinguished by its slow onset, its relatively slight impairment of the general health, by the fact that it is bilateral, whereas pneumothorax is almost always unilateral, and by the existence of feeble breathing with greatly prolonged expiration. Amphoric breathing and resonance, metallic tinkling, and signs of fluid are all absent in emphysema.

When the pneumothorax is circumscribed, the physical signs resemble those of *pulmonary cavity*. But over a large cavity the chest is usually flattened. Cracked-pot sound and alteration in pitch upon opening and closing the mouth are more common in cavity than in pneumothorax. Displacement of viscera does not necessarily occur in phthisical cavity, the coin-test is negative, and succussion can not be produced. Fremitus is absent in pneumothorax and increased over a cavity.

The hyper-resonance above a *pleural effusion* develops with a very different clinical history, is accompanied by increase of fremitus with bronchial or, at times, amphoric breathing, and changes when the patient's mouth is open or closed. The percussion-note usually lacks the metallic quality heard in pneumothorax, metallic tinkling is absent, the coin-test is negative.

Pneumothorax must be distinguished from abscess below the dia-

phragm containing air (*pyopneumothorax subphrenicus*). Often the distinction is difficult. The constitutional symptoms of suppuration are present. Leyden points out the importance of remembering the sequence of events in the development of the disease. When the abscess is situated below the diaphragm, abdominal symptoms precede its development, and early in the course of the disease there is absence of respiratory symptoms. If the patient has had gastric ulcer, this would point to subphrenic abscess, as most of the cases of subphrenic abscess are secondary to gastric ulcer. Moreover, in subphrenic abscess the heart is not displaced nor do the interspaces bulge. Indeed, the viscera below the diaphragm are more likely to be displaced than are those above it. In pneumothorax, according to Leyden, the respiration is normal under the clavicle, and the transitions from the normal to the metallic and amphoric sounds lower down are abrupt. In pyopneumothorax on the left side the semilunar space disappears. In subphrenic abscess the amphoric sounds laterally or posteriorly may be above and below the diaphragm, or they may be loudest at the epigastrium. In addition, in *pyopneumothorax subphrenicus*, as Mason points out, adhesions of the lung to the diaphragm and parietes can be made out, particularly if the case has been under observation in its earlier stages and dry pleurisy has been discovered. Abscess in this location and slight fluctuation are likely to develop with associated effusion. The limited extent of the effusion is of diagnostic import in favor of subdiaphragmatic inflammation.

CHAPTER VII.

DISEASES OF THE HEART, THE BLOODVESSELS, AND THE MEDIASTINUM.

DISEASES OF THE HEART.

Symptoms. The symptoms of disease of the heart are due to the anatomical structure of the organ, to its physiological offices, and to the morbid process. The heart is a hollow muscular structure which hangs in a cavity, the pericardial sac, and encloses other cavities—the two auricles and two ventricles—separated from each other by valves. Both sets of cavities are lined by serous membrane. The serous membranes are subject to similar lesions, and present similar signs as diseased serous membranes elsewhere. In inflammation of the external membrane —the pericardium—the surfaces rub together and create a sound of friction. The external serous cavity may also become filled with the products of exudation or transudation. Physical signs are produced. They are the physical signs of a localized increase of contents as determined by inspection, palpation, and percussion, and of physical interference with the heart's action. The heart muscle is also subject to the same morbid processes as other muscular structures. They are hypertrophy and atrophy; inflammation, acute and chronic, with over-growth of connective tissue; and degenerations. The *symptoms* are likewise the same. Increase or diminution in the power of the muscle is associated with corresponding change in size, which is determined by physical signs. Above all, however, such change modifies the heart's action so that strength or weakness of the muscle shows itself in excessive or deficient vascular pressure. The latter is more particularly an object of observation because of the congestions, dropsies, and cyanosis that ensue.

The heart is constantly subjected to internal pressure. Dilatation of the cavities or of a portion of a cavity (aneurism) follows previous disease of the muscle or increase of internal pressure, and causes physical signs of enlargement. Degeneration of the heart muscle, nearly always secondary to deficiency of vascular supply, is also attended by symptoms of weakness and physical signs of enlargement (dilatation) or of diminution in size (atrophy). When dilatation occurs, the orifices of the cavities enlarge, the valves can not close them, and the symptoms and physical signs of incompetency and of blood-regurgitation result.

The serous membrane that lines the cavities of the heart and, with the subserous tissues, makes up the structure of the valves, is subject to inflammations, the signs of which are common to all serous inflammations. The swellings and outgrowths that attend such inflammation occlude the orifices and prevent closing of the valves. There is produced

a physical interference with the heart's function which is recognizable by physical signs. The successful effort of the heart muscle to overcome such defects on the one hand (hypertrophy), or its failure on the other (dilatation), again leads to the production of symptoms and signs. The serous membranes, and hence the valves, are exposed to causes which excite inflammation. By virtue of the position of the heart at the centre of the circulation, the blood, infectious or irritative, as in rheumatism and Bright's disease, constantly bathes this vulnerable structure. For the same anatomical reason, symptoms arise not common to serous membrane inflammation—that is, *embolic* phenomena. (See Symptoms of Morbid Processes.) Hence, the physical signs (objective symptoms) of cardiac disease may be due to primary and secondary morbid anatomical changes. They may be due (1) to valvulitis as indicated by signs of (*a*) obstruction or regurgitation at the valve-orifice, or (*b*) of embolic phenomena; (2) to secondary changes in the heart muscle as seen in (*a*) change in the size and strength of the organ (hypertrophy or dilatation), and (*b*) in consequence of the latter, signs of congestion, œdema, cyanosis, etc.

It is the function of the heart to propel the blood. It has been shown how interference with the action of the heart muscle and with the consequent flow of blood through the cavities and orifices modifies this function. The functional power is increased or diminished by the physical changes. The evidence of increased power is increased force of the heart-beat, and increased pressure in the arteries (pulse).

Diminished power shows itself in symptoms of diminished blood-supply to parts, and in stagnation of the blood that is sent to the periphery. The former is more pronounced in cerebral anæmia, and physiological weakness of organs or of the organism as a whole; the latter, in congestion and dropsies.

The functional activity of the heart is controlled by a nervous mechanism, any alteration of which alters cardiac action and consequently produces symptoms. Just as with the larynx, a break in the cardiac mechanism may be in the centres in the medulla, in the centres in the heart muscle, or in the sympathetic nerves to and from the heart. The rich anastomosis of these nerves exposes the heart to disturbance by reflex influences. We should suppose such extensive innervation would invite frequent cardiac perturbation. In a measure it does, but, fortunately, so perfect is this mechanism that the inhibitory fibres control such perturbation to a large extent, and we do not see such pronounced symptoms as occur in disease of the larynx. The symptoms which point to disturbance of the cardiac mechanism are alterations in the rhythm of the heart. Its action may on this account be increased or diminished in frequency, or it may be irregular or intermittent. Such alterations of rhythm may be due to organic disease of the centres, notably the pneumogastric from apoplexy, softening or tumor in the medulla, or to stimulation or depression of the centres by toxic substances in the blood, as in anæmia, acetonæmia, or autogenetic or other toxæmias, or by nicotine or other extraneous material. The altered rhythm may be, and most frequently is, of reflex origin. It may be due to disease of the nerves, as the pneumogastric or sympathetic, from pressure upon the nerve-trunk

by a tumor or inflammatory growth. The most pronounced symptom of altered rhythm of which the patient is cognizant is palpitation. The exciting cause of this, as well as of other rhythmical changes, must, in the great majority of cases, be sought for beyond the domain of the heart.

While the symptoms or signs of cardiac disease are often due to morbid processes in the heart or its membranes, it must be remembered that grave and persistent subjective and objective symptoms may be caused by, or at least associated with, disease of contiguous structures outside of the pericardium. The symptoms are not excited through the nervous system, but are produced by mechanical encroachment upon the organ, as in pleurisy with effusion, mediastinal disease, and disease of subdiaphragmatic viscera. They will be referred to in the study of objective symptoms. Care must be taken never to overlook the possibility of their presence.

In the study of the symptomatology of cardiac disease the student must bear in mind two things: (1) that the cause of the morbid processes and of the symptoms (pain and palpitation) may be elsewhere than in the heart; and (2) that the ultimate object of the examination is to determine the muscular power of the heart. He will soon learn that with that power intact the functions can be performed notwithstanding the presence of marked physical abnormalities.

The recognition of disease of the heart is usually not attended by much difficulty except in some special lesions. The non-recognition of cardiac disease is due to faults in the examination. The physician is too often satisfied with the recognition of the remote process, as a congestion or functional weakness in some organ. Safety lies, as has been often said, in the examination of the organs of the body. Often, for instance, indigestion from gastric catarrh is not relieved, for the cause, mitral regurgitation, is not recognized.

The Data Obtained by Inquiry.

Diagnosis. The historical diagnosis furnishes facts of ætiology which clear up doubts about an obscure case. Excesses of all kinds, infections, and intoxications, are causal, and hence diagnostic.

The subjective symptoms in diseases of the heart and bloodvessels are common to many affections and most of them are considered in the section on Subjective Diagnosis. They are pain, palpitation, dyspnœa, cough, arrhythmia, and pulsation of the vessels. They are not so frequently found in other affections, but are sufficiently common to be treated of in a separate chapter.

The subjective symptoms therefore are: (*a*) Cardiovascular—pain, palpitation, arrhythmia, and pulsation. (*b*) Pulmonary—dyspnœa and cough. (*c*) Gastro-intestinal—the symptoms of gastritis and enteritis. (*d*) Laryngeal—pain, aphonia, and cough. (*e*) Renal symptoms of congestion of the kidney, of chronic inflammation (cyanotic induration), and renal embolism. (*f*) Hepatic—pain, fulness, and pulsation of the liver. (*g*) Nervous—vertigo, chorea, convulsions, coma, and disorder of special senses.

The physical diagnosis establishes or renders final the conclusions as to the nature of a cardiac lesion.

The laboratory diagnosis enables us to discover the effects of cardiac disease and practically establishes a diagnosis of infectious endocarditis.

Pericarditis.

Inflammation of the Pericardium. The inflammation may be *acute* or *chronic*. It is also divided according to the nature of the inflammation into *simple fibrinous* inflammation and inflammation with *effusion*. The effusion may be serous (really serofibrinous), bloody, or purulent, depending upon the nature of the inflammation. Pericarditis may be *primary* or *secondary*. The primary form is of extremely rare occurrence. Indeed, it may well be doubted whether, in common with the inflammations of serous membranes in general, pericarditis is ever primary, or so-called idiopathic, in origin.

Causes. 1. Extension from Neighboring Structures. Extension of the inflammation from infected tissues in the vicinity is a common cause of pericarditis. It may follow a pleurisy and partake of the nature of the primary pleural inflammation. It often attends empyema, either from extension of the infection to the pericardium or from rupture into the pericardial sac. It may follow all forms of inflammation of the mediastinum, and sometimes results from extension of infection from a tuberculous peribronchial gland. Disease of the ribs adjacent to the pericardium may set up pericarditis, acute or chronic. It attends aortic aneurism at times, but more frequently infectious endocarditis and myocarditis. Inflammations below the diaphragm frequently give rise to pericarditis. Peritonitis, when general or local; subdiaphragmatic abscess; suppurative gastritis, with perforation of the stomach; abscess of the liver; suppurating hydatid and other forms of suppuration below the diaphragm belong to the latter.

2. General Infections.—The general diseases causing inflammation of the pericardium are those which affect serous membranes. They are: infectious diseases, particularly scarlet fever, measles, erysipelas, and typhoid fever. All forms of septicæmia may be attended by inflammation of the pericardium. Tuberculosis is a frequent cause of pericarditis. Inflammation of this membrane frequently arises in the course of rheumatism. It may occur in the course of the disease, or attend some of the affections which are themselves manifestations of rheumatism, such as acute tonsillitis. In the course of certain dyscrasias the pericardium is frequently the seat of inflammation because more vulnerable. This is particularly the case in scurvy. It occurs also in Bright's disease, and may be the first manifestation to the patient of this disease, particularly in the chronic form of nephritis. It occurs in the course of gout.

The various forms of pericarditis may occur at any age, although that which attends scarlatina and rheumatism occurs in early life, while late in life it is an attendant upon chronic Bright's disease and gout.

Acute Fibrinous or Plastic Pericarditis. This is the most common form. It attends Bright's disease, rheumatism, and tuberculosis. It may

be wanting entirely in symptoms. A routine examination of the heart may reveal its presence.

In the course of one of the primary causal diseases, if the temperature rises a little higher than it should, or convalescence is delayed, pericarditis should be suspected. Again, if the pulse is more rapid and quicker than is customary at the period of disease the examination is made, or out of proportion to the temperature, the disease should be suspected. There may be altered rhythm or tumultuous action. In other instances the patient may complain of pain in the region of the heart. It is usually localized in the fourth or fifth interspace. It is not very severe and not influenced by pressure. Sometimes the pain is complained of at the xiphoid cartilage. In rare instances it may resemble that of angina pectoris. The pain and the occurrence of fever further call attention to the heart.

Physical Diagnosis. INSPECTION. Nothing unusual is observed, although the heart may be seen to beat more violently against the chest-wall. The impulse is diffused.

PALPATION. A friction-fremitus may be detected, due to the rubbing together of the roughened pericardial surfaces. It is not always present. It may be felt when the whole hand is laid over the præcordia, or by palpation with the tips of the fingers. It is most marked over the right ventricle, particularly in the fourth interspace, and is increased when the patient leans forward.

AUSCULTATION. A friction-sound is usually present. It may be present while the fremitus is absent; but, on the other hand, if the fremitus is present, we can always hear the friction. It is heard over the region where the fremitus is felt.

Point of Maximum Intensity. It may be heard along the course of the sternum. It is usually heard in the third or fourth interspace, but may be heard as high as the second, adjacent to the sternum in either interspace. Sometimes it is heard at the second costal cartilage on the right, rarely at the apex. The point of maximum intensity may vary with the position of the patient.

Time. It is both systolic and diastolic. In some cases it may be only systolic in time, or it may be of a galloping nature, representing three sounds during the cardiac cycle. Again the to-and-fro sound is not synchronous with the systolic and diastolic sound, although it occurs but once in the cardiac cycle. It may begin after systole, and be completed before the end of the diastole. The impression that it is a superadded sound is most positive.

Direction of Transmission. It is localized, and not transmitted.

Character. A friction-sound is a to-and-fro rubbing, scratching, or grating noise; it gives the impression of being near the ear. It may be modified by the pressure of the stethoscope by the position of the patient, and by the respiratory movements, which may press the heart firmly against the chest. It may be heard in the erect and disappear in the recumbent posture.

Diagnosis. Acute pericarditis is overlooked because it is not sought for. In the larger number of cases there have been no indications of disease of the pericardium. The diagnosis is usually easy.

Differential Diagnosis. The *pericardial friction* may be mistaken for an *organic heart-murmur* or for *pleural* or *pleuropericardial friction*. It is often difficult to distinguish the to-and-fro friction from the murmurs of double aortic disease. If attention is paid to the general and local phenomena, the mistake is not likely to be made. The location of the murmurs in organic heart disease, the direction of the transmission, the character of the murmur, the peculiar character of the pulse, and the secondary effects upon the muscle of the heart, point to the valvular lesion.

The pleuropericardial friction which simulates pericardial friction usually occurs in the course of phthisis or pleuropneumonia. It is modified by respiratory movement: (1) it may disappear, or at least diminish, if the breath is held; (2) a full expiration may cause its disappearance. While it is of cardiac rhythm, it is modified by the respiratory rhythm, so that on inspiration it is usually more marked. The pleuropericardial friction is not so strikingly modified by position.

Pleural Friction. This is of respiratory rhythm and ceases with cessation of breathing. The pericardial friction persists even if the breath is held.

Pericarditis with Effusion. As with fibrinous so with this form, it is frequently overlooked. This is because it very often develops without symptoms. In plastic pericarditis we have referred to the occurrence of *pain*. This may occur before the effusion in the latter form, but is usually moderate. As with dry pericarditis, however, it may, in rare instances, be very severe, anginose in character, and be increased by pressure over the heart or on the pit of the stomach.

Symptoms. The symptoms are usually due to the special character of the inflammation and the presence of fluid in the pericardium.

1. General Symptoms. In non-suppurative cases the symptoms are usually cerebral. Delirium may be moderate or maniacal. It must not be confounded with the delirium which occurs in the course of acute rheumatism with hyperpyrexia. Choreiform movements have been described in many cases. They may, however, be of rheumatic origin. Other cerebral symptoms, as hemiplegia and convulsive attacks, occur in the course of pericarditis, probably due to an associated endocarditis, causing embolism. In some cases albuminuria is found.

The general symptoms of pericardial effusion depend upon the nature of the primary disease and the character of the fluid. In *tuberculous pericarditis*, emaciation, irregular fever, sweats, and prostration ensue. In *purulent pericarditis* there may be recurring chills with a temperature-range decidedly intermitting, along with other phenomena of purulent accumulation. In a case seen in 1895 the patient was extremely debilitated and prostrated on account of pneumonia following influenza. He was extremely anæmic, and the blood-count showed diminution of red cells to one-half without other change. Every fourth day after a chill, the temperature would rise to 103° or 104° F. A friction-sound was detected after the second chill. It disappeared, but the physical signs of effusion were not positive. From the first the heart's action was so weak that the sounds were scarcely discernible. At the autopsy, 4 or 5 ounces

of pus were found in the pericardial sac. The purulent accumulation was the only lesion to account for the symptoms, and, we would say now, was no doubt a pneumococcus infection.

2. Local Symptoms. The local symptoms are due to the accumulation of fluid within the pericardium. *Dyspnœa* is the most common. The degree depends upon the amount of effusion. If the latter is large, there may be extreme orthopnœa; if the effusion is present for a considerable time, it may give rise to no symptoms. *Dysphagia* may occur in large effusions on account of pressure upon the œsophagus. *Altered cardiac rhythm.* The effect of the effusion upon the heart is to interfere with its action. Although usually regular, on the slightest exertion or the least excitement it palpitates violently or becomes irregular. The heart's action is increased in frequency; when the effusion is very large, it may be not only irregular, but also intermittent. *Aphonia* may occur from pressure upon the recurrent laryngeal nerve. *Cough* of an irritative character is sometimes noted. The *pulsus paradoxus* may be present.

Physical Diagnosis. (Plate XXX., Fig. 1.) Inspection. There is bulging of the præcordia, particularly in children. The ribs and interspaces are prominent. In adults the interspaces are on a level with or distended beyond the surface of the ribs, and are sometimes widened. The enlargement may extend to the anterolateral region of the left chest. A large effusion interferes with expansion of the lung on the left side, and hence movement is diminished. The epigastrium may be prominent, on account of displacement downward of the diaphragm and liver. The apex-beat is absent or faintly seen, displaced upward and to the left. It does not extend as near the left border of dulness as in dilatation. It may be seen in the fourth interspace, or a faint impulse may be observed in the second and third interspaces beyond the midclavicular line.

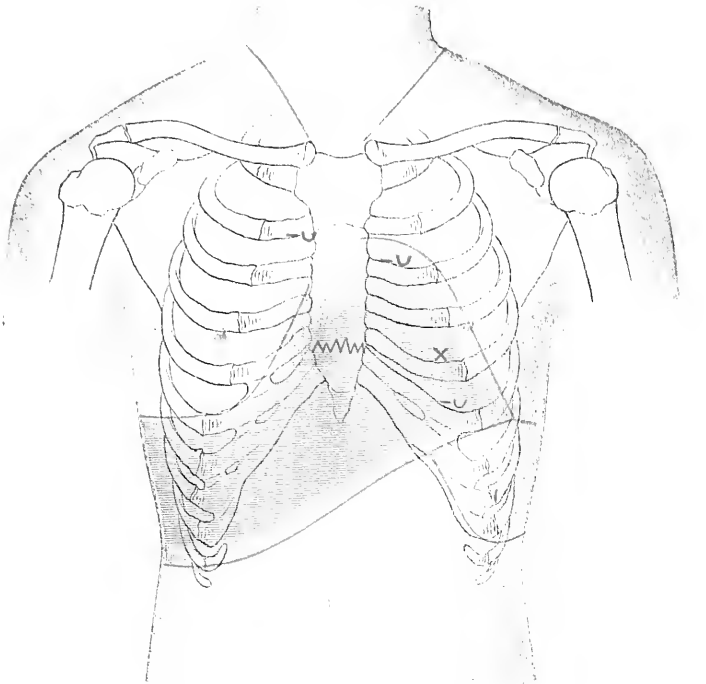
Palpation. The impulse is feeble and diminishes in force as the effusion increases. The position of the apex as determined by inspection is confirmed. Ewart points out that the first rib is palpable at its sternal attachment in pericardial effusion. The pericardial friction which may have been present at first disappears with the effusion. Fluctuation may be detected in large effusions. The liver in large effusions is depressed and readily palpable.

Percussion. The area of præcordial dulness is increased. There is increase of the lateral boundaries and great increase of *absolute dulness.* The relative dulness is generally absent. The increase of dulness is usually in all directions, although increase upward and to the left only is very common. It may extend as high as the second rib. As pointed out by Rotch, dulness in the fifth right interspace in the angle formed by the right border of the heart and the right lobe of the liver is common in effusion. It may be an early sign. Ebstein calls this region the *cardiohepatic triangle,* and points out that the dulness is absolute in effusion, although impaired in normal states because of proximity to the liver.

Pulmonary resonance is modified posteriorly in large effusions. The dulness in large effusion includes the axillary region, so that it may simulate pneumonia or pleural effusion. The dulness, however, does not

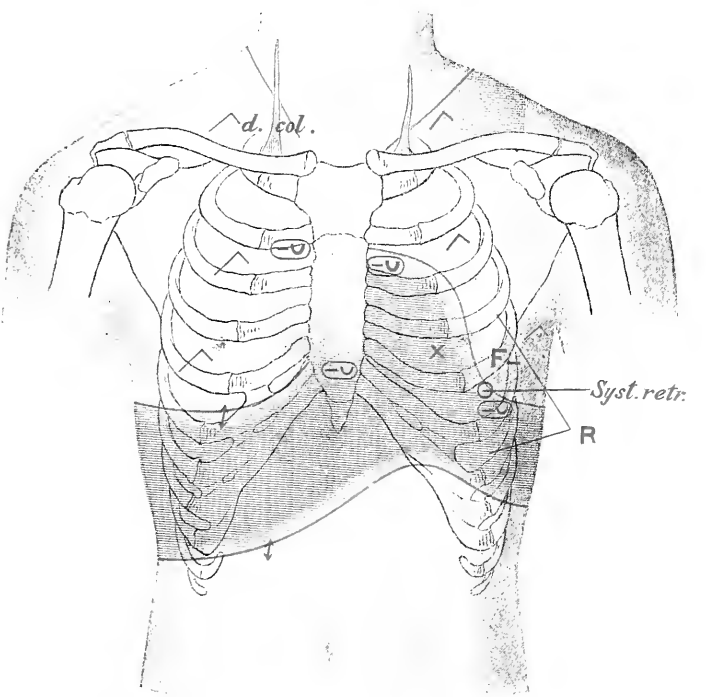
PLATE XXX.

FIG. 1.



Pericarditis with Effusion.

FIG. 2.



Adherent Pericardium. Chronic Left-sided Pleurisy.

extend below the eighth rib in this region, whereas in pleural effusion dulness always extends to the bottom of the pleural sac. In a large pericardial effusion the semilunar space of Traube is obliterated.

Auscultation. The heart-sounds are feeble and distant. They may be scarcely heard at all over the præcordial region. The sounds at the base of the heart are diminished in intensity. If a friction-sound was heard at the beginning, it disappears entirely as the effusion is poured out. In moderate effusions the friction may be heard when the erect posture is assumed.

It must not be forgotten that the physical signs, and especially the change in impulse and the area of præcordial dulness, are modified by the position of the effusion. Accumulations occur behind the heart or above it, and in these situations interfere least with the displacement or the enfeeblement of the apex-beat. The area of dulness, however, is increased upward.

In cases of large effusion the compression of the lung may cause bronchial breathing heard posteriorly or in the axillary region. In a case under my care the diagnosis of pericardial effusion was readily made, but the enormous effusion so markedly simulated an effusion into the pleural cavity that both serous cavities were believed to contain fluid. Aspiration was performed in the sixth interspace in the anterior axillary line. The fluid was removed from the pericardium, as was afterward determined. During life pressure-signs—laryngeal stridor, difficulty of deglutition, and extreme dyspnœa—were present. Early vomiting, epigastric pain and tenderness, slight delirium, albuminuria, and an excessively weak, rapid pulse occurred in the course of the disease. The patient was a male, twenty years of age. The effusion was due to tuberculous pericarditis, secondary to tuberculosis of the bronchial glands. The physical signs were prominence of the præcordia; bulging of the interspaces on the left side; diminished expansion of the left side—anteriorly, laterally, and posteriorly; increased expansion at the extreme apex of the lung. The vocal fremitus was absent below the second interspace in front, below the third in the axilla, and diminished below the spine of the scapula behind. There was dulness from the second left rib in front to the margin of the thorax; from the fourth to the eighth rib in the axilla; below the eighth rib, tympany. The dulness extended beyond the margin of the sternum on the right side, almost to the right nipple-line, in the fourth and fifth interspaces. Posteriorly, dulness from the middle of the scapula to the base of the thorax, except along the vertebræ, where, from the seventh to the ninth rib, there was tympany. On auscultation the physical signs of pericardial effusion were marked. In the axilla the breath-sounds were absent. There were bronchial breathing and bronchophony behind from the spine of the scapula to the base along the vertebræ. They were most marked opposite the angle of the scapula, where the above-noted tympany was observed. In the midscapular line the breath-sounds diminished from above downward, and were absent at the base. It is seen that the physical signs of pleural effusion were present posteriorly and laterally, due to the enormous effusion. At the autopsy the pericardium was found to contain 64 ounces of fluid.

Pleural effusions may be excluded in similar cases by the absence of

dulness in the axillary region below the eighth rib; by increase in dulness beyond the right edge of the sternum; and at the same time by the absence of signs indicating dislocation of the heart to the right.

Diagnosis. Pericardial effusion must be distinguished from *dilatation* of the heart. Although feeble and diffuse, the expansile shock of the impulse is more distinct than in dilatation. This distinction is generally not difficult if the patient has been under observation during the development of the disease. The impulse is not always absent in dilatation. Fluctuation may be detected. The area of dulness in dilatation does not extend upward except in cases in which the right auricle is enlarged. The dulness does not extend downward in dilatation without a similar displacement of the apex-impulse. The shape of the dulness differs. In dilatation the dulness is square in shape; in effusion it is triangular or pear-shaped, with the base downward. Normally, and in case of enlargement of the heart, the cardiohepatic angle is a right angle, sometimes even an acute angle; in pericardial effusion this angle becomes markedly obtuse. In dilatation the sounds are accentuated, and are of a valvular character; in effusion they are muffled. Dilatation does not cause the pressure-symptoms that occur in effusion. In pericardial effusion Bamberger's sign is of importance. When the patient is sitting upright, an area of dulness about the size of a silver dollar can be marked out at the angle of the scapula. Over it, dulness, increased fremitus, and bronchial breathing are made out. If the patient leans forward, the dulness and the other signs of consolidation disappear, to return when he sits upright. In children, pseudopleuritic signs are often present posteriorly—dulness, pleuritic friction, broncho-ægophony—but will disappear if the patient is put in the knee-chest posture. It is of diagnostic significance to have change of the rhythm and the character of the sound from day to day, or of its degree of loudness on movement of the patient.

The friction-sound may return after absorption of the effusion. It may disappear entirely and all signs of pericardial inflammation subside. Adhesion of the two layers of the pericardium may take place in plastic pericarditis and pericarditis with effusion.

Effusions into the pericardial sac of serum, blood, or air may take place without previous inflammation.

Hydropericardium. This may occur in the course of general dropsy from kidney or heart disease. It may not prove fatal of itself, but when associated with effusion in the pleural sac it contributes to the orthopnœa, which may cause death. Rarely after scarlet fever, effusion into the pericardial sac may be the only dropsical symptom. The physical signs are those of effusion. It is not attended by fever. It is frequently overlooked, because investigation beyond the pleura is not made after an effusion into that cavity has been found.

Hæmopericardium. This occurs on account of rupture of an aneurism of the first part of the aorta, of the heart itself, or of the coronary arteries. Wounds of the pericardium and heart cause hæmopericardium. The extension of the ulceration of malignant endocarditis to the surface may cause gradual effusion of blood.[1] The physical signs are those of

[1] See Keating, Transactions of the Philadelphia Pathological Society, 1878.

effusion. Death usually takes place before there has been time to make a sufficiently accurate examination to determine its presence. Rapid heart-failure due to compression is the cause of death. In the case referred to above, and in cases of rupture of the heart, the patient may live for many hours with dyspnœa and progressive weakening of the heart. In tuberculosis and cancer the effusion is frequently blood-stained.

Pneumopericardium. This occurs very rarely, and is due to perforation from without by a stab-wound, or perforation from the lung, œsophagus, or stomach. A purulent exudation may undergo decomposition, causing an accumulation of gas. If it arises from perforation, acute pericarditis is set up. The accumulation of gas causes tympany over the movable area of percussion-dulness. The most striking sign is noted on auscultation. Churning, splashing, or metallic sounds are heard, drowning the feeble heart-sounds. Death usually occurs quickly.

Adherent Pericardium. (Plate XXX., Fig. 2.) Chronic adhesive pericarditis may follow the acute form or, particularly if tuberculous, it may develop independently and progress slowly.

Inspection and Palpation. Indrawing of the interspaces may be seen at the time of the systole of the ventricles; even the ribs are said to be drawn in. This indrawing is most marked at the apex, and must not be confounded with the retraction that occurs in the third and fourth interspaces with the ventricular systole. The recession is synchronous with the systolic shock. In some cases the systolic movement over the præcordia is of an undulatory character. Walter Broadbent calls attention to systolic retraction of the left back in the region of the eleventh or twelfth rib as a valuable sign. The apex is displaced outward and the area of impulse is increased. The increase in area of impulse is due to the hypertrophy which always attends universal adhesion of the pericardium. After the systole there is frequently felt a quick rebound, known as the diastolic shock, which is said to be characteristic of pericardial adhesions.

In pericardial adhesions Friedreich's sign, *collapse of the cervical veins* during the diastole of the heart, is seen. We may also see inspiratory swelling. (Kussmaul.) In addition, the *pulsus paradoxus* is significant of the presence of pericardial adhesions, or rather of the dilatation that succeeds the adhesions. The pulse is small and feeble during inspiration, assuming greater strength during the period of expiration.

Percussion. The area of cardiac dulness is increased usually upward, extending as high as the first interspace. The area of dulness is frequently not modified by respiration—that is, it is not lessened when the patient takes a full breath, when the lungs should expand over the præcordial region. This is particularly the case when there is pleuritis associated with pericarditis, a common association in the large majority of cases.

Auscultation. On auscultation the signs vary. The sounds are due to hypertrophy or to dilatation; and it must not be forgotten that they frequently arise on account of pericardial adhesions. In the former condition the first and second sounds are accentuated; in the latter, a murmur may be heard at the apex, loud and systolic in time.

In pericardial adhesions the physical signs depend upon the condition of the heart muscle at the time of the examination. At first we have the

physical signs of hypertrophy, with retraction of the interspaces, particularly at the apex, or the space at the xiphoid cartilage. This is particularly the case in young subjects. In the later period of the disease the physical signs of dilatation arise, indicated by increase in transverse dulness, enfeeblement of impulse and of sounds, with the development of a murmur at the apex, undulation of the veins in the neck, and the *pulsus paradoxus*. The physical signs of associated pleurisy aid in the recognition of adherent pericardium. Diminution of the breath-sounds, increase in the area of cardiac dulness, lessened fremitus in the neighborhood of the heart pointing to pleural thickening, serve as further evidence. Sansom considers the presence of pulmonary tuberculosis of value, as pointing to the occurrence of pericardial adhesions, for the associated pleural adhesions are likely to be attended by tuberculous pericarditis.

I have learned to suspect adhesive pericarditis in a young subject the victim of valvulitis, when the symptoms do not yield to treatment—in short, when the heart is not affected by digitalis. Unfortunately, the physical signs are often not conclusive.

The *subjective* symptoms of adherent pericardium are those of dilatation or hypertrophy of the heart, whichever is in excess.

Diagnosis. Briefly, fixation of the heart is the physical condition the signs of which are well summarized by A. O. J. Kelly as follows: In the diagnosis of adherent pericardium, most help will be derived from: a weak or absent apex-beat, especially in cases in which there is no increase in the area of cardiac dulness; systolic retraction of a considerable area about the apex; systolic retraction of the base of the left chest posteriorly; arrest of the normal respiratory movements in the epigastric angle; imperfect descent of the apex-beat during inspiration; inadequate or entire absence of shifting of the apex-beat, with change in the posture (lateral posture) of the patient; absence of change in the limits of the cardiac dulness during the respiratory phases; absence of increase of the cardiac dulness to the right, despite marked engorgement of the veins of the neck; a diastolic shock or rebound of the heart; evidences of dilatation or hypertrophy of the heart in the absence of valvular or other disease that might cause it; absence of the characteristic changes in the heart in the presence of definite valvular disease—that is, absence of the usual hypertrophy of the right ventricle in mitral disease and of the left ventricle in aortic disease; absence of pericardial effusion in the presence of pleural and peritoneal effusions; paradoxic pulse—inspiratory diminution in the force and volume of the pulse; diastolic collapse and inspiratory swelling of the veins of the neck.

In unusual cases the first evidence of adherent pericardium is ascites—a symptom-complex for which Pick has proposed the designation, "pericarditic pseudocirrhosis of the liver." In reality, the condition is a manifestation of multiple inflammation of the serous membranes, "*multiple serositis*," or *polyorromenitis*, of the Italians and others. The pericardium, pleuræ, and peritoneum (especially about the liver—Zuckergussleber) may be involved consecutively.

Multiple serositis, or chronic adhesive pericarditis, with ascites is recognized by attention to the following facts: a history of a previous attack

of acute pericarditis, pleuritis, or perihepatitis; the early occurrence and subsequent disappearance of the œdema of the legs; marked ascites, with little or no œdema of the legs; enlarged liver early in the course of the disease (in some cases the liver appears not to have been enlarged); small and distorted, but otherwise smooth liver in the later stages of the disease; absent or very late enlargement of the spleen; repeated attacks of pain, tenderness, rigidity, and possibly palpable and audible frictions in the right hypochondriac region—attributable to attacks of perihepatitis; rapid recurrence of the ascites after tapping, and the physical signs of adherent pericardium—without which, it may be said, the disease is incapable of diagnosis.

Obliterative pericarditis with ascites may be distinguished from cirrhosis of the liver: by the symptoms and signs of adherent pericardium; by the absence of the ætiological factors of cirrhosis of the liver; by the slow, insidious, protracted, and intermittent course of the disease; by the long periods of quiescence during which the ascites may remain stationary and the patient in good condition; by the entire absence or the transient presence of slight jaundice; by the absence of symptoms of portal congestion other than ascites; in some cases, by the association of an enlarged, smooth, and firm liver with marked ascites; and by the fact that in many cases the patient survives a large number of tappings.

Indurative mediastinopericarditis with adhesion may occur with or without fibrous inflammation and adhesion of the structures in the anterior mediastinum. The pericardium is adherent and thickened. Rarely the anterior mediastinum alone is a mass of fibrous inflammation. Peritonitis and perihepatitis may be found. The entire process is usually tuberculous.

The *symptoms* are dyspnœa, venous engorgement, cyanosis, enlargement of the liver, ascites, and dropsy.

The *physical signs* are those of extreme cardiac dilatation; the *pulsus paradoxus;* collapsing jugular veins during diastole, due to the dragging upon the innominate veins and cava by the fibrous adhesions, or to stretching and narrowing of the aortic arch by these adhesions; or inspiratory swelling of the veins of the neck. A friction-sound, systolic in time, heard over the sternum, increased when the arm is held up—*mediastinal friction,* so called—has been described in this affection.

Endocarditis.

Endocarditis may be acute or chronic. In either form it is usually secondary. The acute form is divided into simple and so-called malignant, infectious, or mycotic endocarditis.

Simple Endocarditis. Acute endocarditis rarely occurs primarily. It usually occurs secondarily to general morbid processes. The pathological antecedents are acute rheumatism, tonsillitis, whooping-cough, scarlet fever, gonorrhœa, rarely smallpox and typhoid fever. It is of common occurrence in pneumonia and tuberculosis. It is frequent in chorea. In the simple form it occurs in septic inflammations and in

debilitating diseases, as cancer. It may occur in gout and develop in the course of Bright's disease.

Symptoms. The symptoms of simple endocarditis are scarcely observed during the early course of the disease. The process is latent, and there are no indications of cardiac disease. The physical signs alone betray its presence. Unless these are sought for, the disease is overlooked. The subjective symptoms are negative. In the course of rheumatism or chorea, or during convalescence from the former, the patient may complain of palpitation, and increased frequency and irregularity of the heart. At the same time there may be a rise in temperature, not attended by any increase of the rheumatic symptoms; this should direct attention to the possibility of a cardiac complication.

The rise is not marked, and may not assert itself during the severity of the disease.

Physical Signs. On examination a murmur is detected in one of the cardiac areas. The murmur is soft, low in pitch, and follows the laws of transmission according to its situation. Instead of a distinct murmur a roughening of the first sound alone may be heard. Preceding the murmur the heart's action may be quickened and arrhythmical; the first sound may change in character from day to day or be accentuated; the second sound may be reduplicated at the apex and accentuated. The new sounds may disappear at first when the patient sits up; later they persist. The murmur must not be mistaken for the murmur at the apex in cardiac dilatation; or the murmur which may be heard in the course of fevers; or the murmur of anæmia, which may rapidly develop in rheumatism and other affections.

Malignant Endocarditis. Unlike simple endocarditis, the malignant form very rarely develops in the course of rheumatism and chorea. (See the Infections.) It occurs more frequently in pneumonia than in any other disease. It arises in the course of erysipelas, septicæmia, puerperal fever, and gonorrhœa. It may occur in dysentery. It is usually a streptococcus infection, but may be due to the staphylococcus, pneumococcus, gonococcus, typhoid bacillus, etc.

Symptoms. The symptoms are: (1) those due to the morbid process —the infection; (2) the physical signs; (3) those due to emboli. The *general symptoms* due to the specific morbid process are septic in nature. The febrile phenomena may be one of four groups: 1. The fever is paroxysmal. Chills and fever occur daily or at intervals of two or three days, resembling types of malarial fever. Each paroxysm is attended by profuse sweats. Rapid exhaustion ensues. The fever, instead of being distinctly intermittent, may be irregularly intermittent. 2. The fever is excessive and continued, and a typhoid state frequently sets in. The temperature is irregular; extreme prostration, low delirium, sordes, subsultus, and other symptoms of that state arise. 3. The fever is moderate and continued. Physical examination, however, reveals the presence of marked endocarditis. In this group chronic heart disease has usually preceded the affection. The duration may be prolonged. 4. The fever may be remittent. Petechial *rashes* and *erythema* are common, so that, as pointed out by Osler, the disease may resemble the eruptive

fevers. The *sweating* is profuse, contributing to the profound exhaustion which usually ensues. A septic *diarrhœa* occurs. In a few rapidly fatal cases *jaundice* has occurred. Again, the symptoms may be almost exclusively cerebral, resembling cerebrospinal or basilar meningitis.

The *embolic phenomena* are due to escape into the blood-current of soft vegetations from the valves of the left heart (for the right heart is rarely affected), which are carried by the blood-stream to distant points of the circulation. Emboli occur in the brain, producing convulsions, aphasia, or hemiplegia; they occur in the retina, causing some visual defect which may be accurately recognized by ophthalmoscopic examination. They occur in the lungs, producing local pain, hæmoptysis, possibly localized dulness, bronchial breathing, and râles. Emboli occur in the kidneys, producing bloody urine and renal pain. In nearly all cases the spleen is the seat of embolism, and in some instances infarctions may take place in this organ alone. The spleen is always enlarged, and the infarct may cause pain and increased tenderness on pressure. Emboli in the skin and mucous membranes present the most striking phenomena. The hemorrhages underneath the skin are minute. Emboli are seen in the extremities, but may also be found on the trunk. Occurring in the long bones, they cause local pain and tenderness. They occur in the mucous membranes, as those of the mouth and tongue. Emboli are seen in the bulbar conjunctivæ and in the conjunctivæ of the lids.

Physical Signs. Repeated examinations are necessary in some cases to determine the presence of a murmur, or to decide whether a previously existing organic lesion is the seat of an acute process. Variations in the character of the murmur from day to day are characteristic of malignant endocarditis. In organic *heart disease* with dilatation and failure of compensation, irregular fever followed by embolic phenomena points to the occurrence of an infectious process on the antecedent valvulitis.

Diagnosis. The diagnosis rests upon proof that an infection is present, and is made by the methods described in Chapter I., Part II., which should be reviewed by the reader. The history of an infection in some part of the body is most important in the diagnosis. The presence of the infection, as well as its nature, may be disclosed by an examination of the blood. When embolic phenomena are present, the diagnosis is made without much difficulty.

The more pronounced general symptoms distinguish it from *simple endocarditis*. The temperature range, the septic and typhoid symptoms, belong to the malignant form.

The more prolonged cases with moderately continuous fever, without apparent primary cause, are frequently confounded with *typhoid fever*. This is readily appreciated when the symptoms of the two are compared. The following symptoms are common to both malignant endocarditis and typhoid fever: a continued type of fever, with the symptoms of the typhoid state, including delirium; enlargement of the spleen, diarrhœa, and abdominal tenderness; there may be infarctions, although they are extremely rare in typhoid fever, and only occur late in the disease; progressive exhaustion. But in endocarditis the onset may be more abrupt, and prostration and anæmia may develop very early, and sweating may

be profuse. The fever does not present the regularity of type that is seen in the development of typhoid. In endocarditis there is more chest oppression and dyspnœa early in the disease than in typhoid fever. In endocarditis the source of the infection may be discovered in the genito-urinary organs, the lungs, the bones, etc. The diazo reaction is found in typhoid fever after the fifth day, but rarely, if ever, in endocarditis. A polynuclear leukocytosis is practically present in malignant endocarditis, while leukopenia is characteristic of typhoid. The results of bacteriological examination of the blood, urine, feces, petechiæ or rose-spots, and especially of *serum diagnosis*, distinguish the two affections. This ought to be of value in endocarditis, because the process is usually due to a staphylococcus or streptococcus infection. Either micro-organism may be found in any suppurations which may possibly be present, or in the blood. In a child recently seen by me in the relapse of an attack of typhoid fever, malignant endocarditis was thought to be present because of a loud and rough murmur at the pulmonary orifice. Fortunately the murmur was present in the apyretic period, and as the child was anæmic its exaggeration was ascribed to the fever.

Malignant endocarditis must be distinguished from *cerebrospinal fever* and from *smallpox* of the hemorrhagic type. We must rely on the local cardiac symptoms and physical signs, and the preponderance of these over the other symptoms. Of course, the prevalence of an epidemic and a history of exposure are of service in the distinction between the diseases. Examination of the blood excludes the forms of *malaria* which formerly were mistaken for endocarditis.

There is probably no disease which is more frequently overlooked. It has been mistaken for cancer of the stomach with secondary infection, tuberculosis, pernicious anæmia, Bright's disease with terminal infection, as well as the infections just considered.

Probably the local infection, which is causal and most frequently not sought for, is a latent gonococcus infection.

Chronic Endocarditis. Chronic endocarditis may follow the acute form or develop in the course of atheroma or of endarteritis due to alcoholism, the poison of syphilis or of gout. If associated with endarteritis, the endocardial change may be part of the general degenerative changes which occur in the aging process. It may be of dynamic origin, often following prolonged heavy muscular exertion, by which the valves, particularly at the aortic orifice, have been subjected to strain. The process is slow and insidious, and leads to the changes in the valve-segments which constitute chronic valvular disease.

Symptoms. The symptoms of chronic, or sclerotic, endocarditis are the symptoms of *chronic valvular disease*. *Insufficiency* or *obstruction*, or both combined, take place at the affected valve-orifice. The outflow of blood is retarded in obstruction. Backward flow, or regurgitation, takes place in the opposite direction from the normal blood-current in insufficiency.

When there is obstruction, *hypertrophy* usually develops to meet it.

Compensation. If the obstruction is moderate, and the person remains in good health, the hypertrophy is sufficient to overcome the obstruction.

In this manner the effect of the valve-lesion is compensated. On the other hand, when blood is permitted to flow by regurgitation backward into the cavity—that is, in the opposite direction to its usual course— it meets a blood-current flowing to this cavity in the normal direction, and the result is over-distention, or over-filling, of the cavity. Dilatation ensues, and may persist. If the regurgitation takes place suddenly, the dilatation continues ; if gradually, as in chronic endocarditis, the dilatation is attended with hypertrophy. Thus, when there is regurgitation from the left ventricle into the left auricle, on account of incompetency at the mitral orifice, the auricle becomes over-distended with blood, for it is filling with blood from the pulmonary veins at the same time. This over-distention can only be overcome by some hypertrophy. When this is not sufficient, the blood is obstructed in the pulmonary circulation, with the consequences hereafter to be mentioned.

The symptoms of chronic endocarditis are latent if the lesions are compensated for, while by *physical diagnosis* alone are they recognized. The physical signs are those of *chronic valvulitis*. The character of the signs depends upon the lesion of the affected valve. If compensation is not established, symptoms of failure occur or *dilatation* of the heart arises.

Disease of the Coronary Arteries.

Atheroma, associated with the process in other vessels, or distinctly localized to the coronary arteries, affects these vessels. Its causal factors are those of endarteritis elsewhere. Its influence on the nutrition of the heart, either by sudden obstruction of the vessels by an embolus or by their gradual closure, with or without thrombus, is apparent.

Symptoms. If an atheromatous coronary artery is suddenly obstructed by an *embolus*, death may be instantaneous. This is a common cause of *sudden death*.

If a *thrombus* forms, it is followed by anæmic infarction, myocarditis, and mural aneurism with the usual symptoms.

The onset of symptoms may be sudden, although the process (occlusion) has been in progress for an indefinite time. (1) Præcordial oppression or dyspnœa or angina pectoris may be the first indication. (2) Succeeding this or immediately, dilatation of the heart, dyspnœa, and venous stasis occur. The heart's action is persistently rapid and may be arrhythmical. (3) The presence of an *aneurism* may be made out. If there has not been previous valvulitis, no murmurs are heard until dilatation ensues. The patient may live three or four weeks, or as many months.

In a large group of cases occlusion is so gradual as to lead to *myocarditis* (*q. v.*) only with the attending symptoms.

Diagnosis. Unfortunately, too often the diagnosis can only be provisional. Coronary artery disease may be suspected in sudden death if there has been a history of previous attacks of angina, if there is evidence of arterial disease elsewhere, and if dyspnœa or anginoid symptoms preceded the fatal termination.

Thrombosis secondary to atheroma may be suspected if a patient, in whom there is no valvular disease, no pulmonary or renal disease, is

seized with angina pectoris or dyspnœa; providing tachycardia and arrhythmia follow, and in a short time cardiac dilatation, venous stasis, etc. In a male, aged forty-three years, without syphilis, but with a history of antecedent rheumatism, an attack of angina pectoris followed some unusual exertion. Prior to this he had been in the most perfect health. The attack was followed by dyspnœa and remarkably rapid heart action without apparent cause. The physical signs of acute congestion of the lower lobe of the right lung followed within twenty-four hours of the attack of angina. The patient was ill three months. He improved somewhat, but rapidity of the heart's action and some stasis in the lung persisted. Gradually cardiac dilatation ensued, with a murmur in the tricuspid area. Death took place from pulmonary congestion. At the autopsy the coronary arteries were atheromatous; the left was filled with an old thrombus; there were extensive myocarditis and an aneurism of the left ventricle.

In another case, male, aged seventy-two years, with general atheroma but no valvulitis, sudden præcordial distress, tachycardia, and persistent dyspnœa were followed by cardiac dilatation, mitral incompetency, and general anasarca.

I have said elsewhere that a persistently rapid pulse uninfluenced by digitalis, indicates pericardial adhesion in the young; the same pulse uninfluenced by treatment points to coronary artery disease in the middle-aged and senile.

Myocarditis.

Acute Myocarditis. Myocarditis may be acute or chronic. The entire muscle or only a portion may be affected. General myocarditis is always acute. The local form may be acute or chronic, depending upon the degree of the primary cause. The local variety is usually due to a thrombus in the terminal endings of the coronary artery, which cuts off the blood-supply.

Ætiology. Pathological antecedents of acute general myocarditis are the infections, particularly typhoid and typhus fever, pneumonia, diphtheria, and septic infections. Chronic myocarditis is usually associated with atheroma, one of the causes of which occurs in the later stages of Bright's disease. (See Atheroma.) The result of myocarditis, when acute, is dilatation of the heart, fatty heart, or aneurism of the heart. Chronic myocarditis is followed by fatty heart, by dilatation, by the so-called fibroid heart or fibrous myocarditis, and by aneurism. The above facts in ætiology are important in diagnosis.

Symptoms. The symptoms of *acute* myocarditis are vague. In the course of, or in the convalescence from an infection the patient may complain of some oppression in the præcordia and suffer from dyspnœa; attacks of syncope may occur, and sighing may be frequent. The pulse becomes more rapid and weak, but is usually not irregular. The circulation is much depressed, the hands may be cold, the face pallid. These symptoms may be accounted for by the extreme exhaustion alone that follows fever. No doubt some myocarditis accounting for the symptoms exists in all cases, particularly if there is prolonged high temperature.

Often the patient does not complain of any cardiac symptoms. Death takes place suddenly, either in the course of the disease or after it has spent its force, from acute dilatation or cardiac paralysis. This is particularly true in pneumonia and diphtheria. In the latter affection the sudden appearance of cardiac symptoms, dyspnœa, cyanosis, and cold extremities may be due to paralysis of the heart.

Physical Signs. Enfeeblement of the heart-sounds, sometimes with accentuation of the mitral first sound, is observed. The impulse and apex-beat are scarcely perceptible or absent altogether. If acute dilatation supervenes, the area of dulness may be increased.

Chronic Myocarditis. Chronic myocarditis may exist without physical signs and may be devoid of clinical expression.

Symptoms. 1. A person may have chronic myocarditis in an advanced degree without symptoms. Death may be due to other causes, as an intercurrent affection, an infection, a terminal nephritis, or one of the many phases of endarteritis. The final termination may be "cardiac" and yet the myocarditis may not be the cause of the symptoms or of the dissolution. Thus coronary artery disease with obstruction by thrombus frequently causes death in cases of latent, and sometimes of advanced, myocarditis.

2. Often the presence of a previously unsuspected myocarditis is not made known either by signs or symptoms except in or during the course of an infection, as pneumonia, or of a toxæmia, as gout. An arrhythmia, an unsuspected dilatation, syncope, or bradycardia, may be brought out by the toxæmia or the infection.

3. The clinical expression may be effaced by the phenomena of an associated or primary valvulitis, a nephritis, an emphysema, or an arteriosclerosis.

Combined with the cardiac symptoms of myocarditis, to be referred to later, we may have the symptoms of chronic nephritis, of pulmonary emphysema, of advanced valvulitis with failure of compensation. Again, the symptoms of endarteritis in other parts may obtain, alternating in character and constancy and interchanging with symptoms of the aforementioned conditions. A cerebral thrombosis, the same condition of an extremity, uræmia, apoplexy, or perhaps phenomena of coronary artery disease (previously referred to) may close the scene. Such shifting of scenes and complexity of phenomena require a clear conception of the associated or concomitant processes in senile or sclerotic myocarditis, and intensely increase the interest in its study. It is well to dwell upon these associate and more frequently dominating features, for only by realizing the pathological lesion in the background—the endocarditis and secondary myocarditis—can the nature of the phenomena be recognized.

4. The clinical expression may, on the other hand, be simply the phenomena of senility.

5. The symptoms, singly or combined, usually the latter, compel the patient to seek relief. They are the *cardiac symptoms of myocarditis.*

1. Heart-tire. As seen in cardiac debility, this is out of proportion to the evidence of senile decay elsewhere. There are breathlessness, œdema of the feet, syncopal attacks or anginal attacks. Local anæmia occurs,

hence the functional activity of organs, as the brain, is in abeyance. Digestive disturbances, renal insufficiency and cerebral inactivity occur singly or combined. The syncope occurs on the slightest exertion, as straining at stool, rising up in bed, or turning from side to side; the breathlessness on any attempt to walk, often, even to use the upper extremities. Weakness out of proportion to the seemingly strong appearance of the patient is characteristic. *Tremor cordis* may be present; palpitation is not infrequent. Tachycardia is the rule and the pulse is small and feeble.

2. **Dilatation of the Heart.** The dropsies, the effusions, the congestions, the enlargement of organs, the functional derangements, coming and going, soon permanent, with the physical signs of cardiac dilatation, are described under dilatation. If symptoms of dilatation arise, it is unusual to have anginal symptoms. If digitalis is administered to the degree of inducing a hypertrophy, cardiac pain not unlike that of angina is caused. After long dilatation its immediate symptoms may be relieved and then those of secondary sclerosis of other organs, as of the kidneys, arise.

3. **Angina Pectoris.** *a.* A person with myocarditis may have one attack of angina terminating in death without premonitions of cardiac disease. They are the cases in which there is often found, associated with the myocarditis, extensive coronary artery disease, death being due to thrombosis.

b. The paroxysms may continue for twenty-four or forty-eight hours before death closes the tragic scene. Arrhythmia frequently attends the breast-pang. It may have preceded the angina for months or years. Dyspnœa might also, although not necessarily, have been a forerunner, occurring either on exertion or at rest. It is not common to see the forms of dyspnœa which will be described later, attended by shock and so often terminating fatally, associated. Nor do we usually see in these subjects of this rapidly terminating form of angina the cardiac asthma of dilatation nor the nocturnal form of dyspnœa due probably to the same cause. We do see dyspnœa from pulmonary congestion developing in the status. When the *angina vera* is replaced by *angina sine dolore*, we have asthmatic attacks in most of the cases. In rare instances the first paroxysm is followed by rapidly recurring attacks, increasing weakness of the heart, and death in cardiac asystole or the ingravescent asystole of Balfour. Houchard describes the condition as *l'état de mal angineux*.

c. The paroxysms continue for several years. During the interval the patient will suffer, as in the preceding cases, from arrhythmia and the forms of dyspnœa observed in that class. No other cardiac symptoms arise except in very rare instances. Pronounced dilatation may occur with the marked symptoms belonging thereto. On the other hand, it is usually the case that other symptoms or accidents of endarteritis do not occur. Thus an anginal subject does not die of apoplexy; again, a prolonged intermission may occur in the attacks of pain, but if arrhythmia has been present, it does not subside. The dyspnœa, except on exertion (dyspnœa of dilatation), may be in abeyance. Indeed, subsidence of the pain is usually due to moderate dilatation as indicated by the symptoms and physical signs.[1] One of my patients, suffering for one year from

[1] Musser, "Angina Pectoris," Trans. Assoc. of Amer. Phys., vol. x., p. 85.

angina, arrhythmia, and dyspnœa, and having the physical signs of myocarditis, had a severe attack of gangrenous appendicitis, for which he underwent operation, and from which he had a long convalescence, during which time he was free from pain and dyspnœa for six months, when the attacks recurred, followed by death in one of the paroxysms.

d. Occasionally we see angina *sine dolore*, at first described by Gairdner, the incident occurring in myocarditis with moderate dilatation.

4. Dyspnœa. *a.* With Dilatation. We see the dyspnœa of dilatation, if that condition complicate myocarditis, with or without pulmonary congestion or pleural effusion. It is constant, or it is increased by exertion, or it is paroxysmal and hence usually nocturnal. In the latter instance there are cyanosis and orthopnœa, perhaps due to a temporary increase of the pulmonary congestion. It must not be confounded with the toxic dyspnœa of nephritis so often concomitant.

It is often the first premonition of myocardial trouble; it may be during a hard pull on the bicycle or on a mountain climb. After some physical effort, dyspnœa occurs with præcordial cardiac sensations of fulness or of sinking or with palpitation or syncopal sensations. After a brief rest, it may disappear and not recur for months, or a slight exertion again brings it about. Frequent recurrences become a warning, and very soon arrhythmia, angina, or the congestions of dilatation ensue. At first it may be due to temporary acute dilatation or to arrhythmical action of the papillary muscles with asystole.

b. Myocardial Dyspnœa. Dyspnœa associated with the aforementioned localized substernal distress without true paroxysms of angina. Often, in your presence, the patient will have paroxysms, probably induced by the psychical excitement attendant upon the consultation. A paroxysm occurs independently of such influence, however, possibly following the taking of food. The patient takes ineffectually breath after breath without satisfying air-hunger until a final full breath satisfies him for a time. "There," remarked M., after repeated labored breathing, "I have gotten over it. I can now breathe easily." The patient's features are such, with the ashy countenance and the cardiac signs, as point unmistakably to the myocardial condition. One can almost make a diagnosis by the facies and the intermittent dyspnœa. In many of these cases the dyspnœa is relieved after some physical exertion. It must not be confounded with the curious form of psychical dyspnœa occurring in certain forms of indigestion, from gastric neurasthenia.

c. Dyspnœa with Angina. The often-repeated story, "I walk half a square and then must seek support to get my breath and relief to the breast anguish." Usually if the angina occurred when at rest there was no dyspnœa. This was not always the case, however, and the interruption to breathing was created by, or coincident with anginal paroxysms. This is one of the true forms of myocardial dyspnœa.

d. Dyspnœa due to Asystole. Angina is one of the tragic terminations of a career which has been fraught with the high and often severe duties in the world's battle. Another tragic end is that attended by the paroxysms of dyspnœa and shock we are about to describe. Often called "heart-failure" or "œdema of the lungs" or, wrongly, "conges-

tion of the lungs," one paroxysm frequently ending the battle. Again, the attacks may recur at intervals of weeks or months, the fatal paroxysm not occurring until a year or two after the original attack. I have seen this continue in rare instances over three years. In one patient, a woman aged seventy-nine, the attacks recurred for five years.

The attacks are characteristic. The patient is suddenly seized with dyspnœa. The respirations are labored, but not much increased. A rattling sound attends it, readily found to be due to fluid in the tubes, as the general large and small râles show. The clear fluid pours from the mouth or is discharged easily, if the attack is not to be fatal; with difficulty or not at all, if death is imminent. The respiratory distress is extreme. The patient sits upright; the face is pale; the brow is covered with sweat; the hands are cold and pallid. There is little if any lividity. The temperature is subnormal; the pulse rapid, thready, irregular, perhaps not perceptible at the wrist. The patient is in a state of profound shock. Because of the lung-noises, the heart-sounds can not be heard, or only as a confused jumble, or with irregular and ineffectual systoles. Either no change in the cardiac dulness is distinguished or it may be lessened.

In the interval, after full recovery, the patient is fairly free from dyspnœa. There is probably some arrhythmia; rarely any angina, and dilatation does not supervene. As in angina, the occurrence of one attack is usually the beginning of the end. I have never seen any patient thus affected live more than five years, and usually death occurs within the year from the onset, generally from an attack similar to the original, such attacks having grown more frequent toward the end. Of course, a progressive weakness develops, and nephritis may become a dominant feature.

These attacks of cardiac asystole differ from those of acute dilatation. The difference is mainly in the following particulars. Had the patient been examined before the attack, signs of myocarditis would have been present in the acute dyspnœa of that affection, whereas dilatation could occur without the presence of fibromyocarditis. In the former, shock and œdema of the lungs are dominating features. In dilatation, marked cyanosis is prominent, tachycardia and arrhythmia are extreme, while the lungs are the seat of congestion rather than œdema. In dilatation there is congestion of the spleen, liver, and kidneys. The urine gives evidence at once of the congestion, whereas it is not necessarily nor instantly changed in myocardial dyspnœa.

It seems academic to discuss the diagnosis of œdema and congestion of the lungs, for it is usually not practicable. It must be admitted that very frequently it can not be done. Nevertheless, it seems well to attempt it, for, as one appears to be a feature of myocardial dyspnœa (œdema) and the other of dilatation (congestion) and as the treatment of each is different, it is well to have an idea of their distinction. I may say in passing that, as I have stood at the bedside of these cases, my feelings were that the œdema was largely from impairment of innervation of the pneumogastric nerve, whereas the pulmonary congestion was vascular.

Œdema of the lungs is attended by shock, by an abundant, frothy expectoration, by dyspnœa without much cyanosis, and by signs of the

outpouring of fluid in the large bronchi rather than in the air-vesicles. Congestion of the lungs is attended by cyanosis, by some cardiac distress, as palpitation, by congestion of other organs, and by the physical signs of moderate filling of air-vesicles with fluid and by frothy, bloody, but not abundant expectoration. There does not seem to be the same fear of impending death in dilatation. While air-hunger is the same in both—orthopnœa marked—it is a necessary attendant of myocardial dyspnœa, but not of the congestion of dilatation.

5. **Tachycardia.** Frequently an unduly rapidly acting heart is due to myocarditis. Usually there is some arrhythmia, but it is remarkable how little suffering may occur, although the heart is beating at the rate of 150 to 180 per minute. Such heart-hurry must be distinguished from toxic and from reflex forms of tachycardia. It must be remembered that it is not to be confounded with palpitation, the phenomena of which give rise to actual mental suffering and to local distress. It is one of the essential symptoms of Graves' disease and a common accompaniment of mitral stenosis. Even in the absence of these conditions tachycardia may be a symptom of myocardial strain, and may be relieved by judicious management. The great fact to be remembered is that tachycardia may be the terminal cardiac symptom of myocarditis, death resulting in from forty-eight hours to one month after the onset, the end being attended by *ingravescent asystole*, congestions of the internal organs proceeding slowly to embarrass further the causal failing circulation. A patient with tachycardia may suffer from *tremor cordis*, the sensation of fluttering followed by a systolic shock belonging thereto alone giving rise to distressing symptoms.

6. **Bradycardia.** Time forbids a description of the interesting syndrome of Stokes-Adams. It is now well recognized that with this slow pulse, syncope, convulsions, and pseudo-apoplexy may arise. Patients with this combination of symptoms rarely suffer from angina or from severe arrhythmia. A fatal syncope is the cause of death. A progressive dilatation rarely takes place. The syncope may be hastened by exertion or by excitement.

7. **Arrhythmia.** Finally, myocarditis may have clinical cardiac expression in arrhythmia alone. Palpitation may attend, but does not commonly do so; delirium cordis, however, is not infrequent, and death may come after many years from asystole, sudden or ingravescent. In the marked cases early in the ailment it is not uncommon to see dilatation with dropsies, congestion and the physical signs of mitral insufficiency. Improvement may take place for a time, followed by recurrences, and death occur from either nephritis, dropsies and cardiac asthenia, or from a terminal infection. Death from angina is rare; indeed, occurrence of the paroxysms is rare. Dyspnœa, however, is common when signs of dilatation arise.

Objective Diagnosis. The most common objective sign is the sallow, pallid complexion. This is most striking. When such persons present themselves, it is safe to infer that the endarteritis has involved the coronary arteries and that the myocardium has suffered.

With the sallow countenance premature age manifests itself in the color of the hair, the baggy eyelids, and the abundance of wrinkles.

Physical Diagnosis. *The Arteries.* The palpable, often tortuous vessels show thick walls and high tension. The temporal arteries are prominent and the other indication of degeneration, *arcus senilis*, is present. The pulse is like that of endarteritis. The artery is readily palpable, isolated with ease, showing high tension, and while apparently strong because of a quality of fulness which with the hard vessel gives a feeling of strength, yet it is actually feeble. The arteries are fibrous but not necessarily atheromatous. Indeed, the patient with pronounced atheroma will probably not have symptoms at least of endarteritis and will die of cerebral thrombosis or of senility alone, or of some intercurrent or terminal infection. Our middle-aged patient with endarteritis does not necessarily end with advanced atheroma, but dies of apoplexy, nephritis, angina, or sudden heart asystole.

The Heart. The physical signs of the heart are those of the (*a*) myocarditis alone, or those of (*b*) myocarditis plus some hypertrophy, or those of (*c*) myocarditis plus dilatation.

The physical signs of *myocarditis* are those of feeble or absent impulse; or if palpable, of apex-impulse displaced to the left; of marked increase in the area of absolute cardiac dulness and of characteristic auscultatory phenomena. The latter phenomena are those either of a systolic shock, greater than the force of the impulse would lead us to believe to be present, or of feeble muscular sound. In the early stages the former exists, and continues later if hypertrophy coexists. From the first, or at least early, we have gallop rhythm, or reduplication of the systolic sounds. This reduplication may be heard over the right heart or more distinctly over the left heart; sometimes it is heard all over the præcordia. My impression is that it is more commonly heard in the parasternal line at about the fourth rib. It may be more marked in the supine position and is generally more marked after exertion. It may disappear after a stimulant is taken or if the heart is stimulated by fever. Reduplication of the second sound also obtains, but is less frequent in the myocarditis of coronary artery disease than in that due to valvulitis or nephritis. In uncomplicated cases murmurs are not heard until late in the disease. Sometimes, however, we hear a systolic murmur at the fourth rib, greater in the recumbent posture. This murmur is soft, low in pitch and so often heard in the parasternal line that it is my custom to call this line the myocardial line. However, it may be at the tricuspid or even in the pulmonary area—when it is probably, although not necessarily, hæmic.

The papillary muscles are the source of the auscultatory phenomena of myocarditis whereby leaflets do not coapt synchronously, or areas of the muscle contract asynchronously.

When *dilatation* supervenes, the physical signs change in keeping with the physical condition of the heart. The auscultatory signs of insufficiency at the mitral and tricuspid orifices are predominant, rarely at the aortic; accentuation of the pulmonary second sound is a frequent sign, with low-tension pulse and engorgement of the venous side of the circulation.

The myocarditic heart of *fatty degeneration*—the fatty heart—presents some similar signs. A visible impulse is wanting and any palpable im-

pulse is felt as a slap against the chest-wall. The area of dulness is normal or slightly increased; a loud hæmic murmur at any orifice, but most frequently at the pulmonary and aortic valve, may be heard. Just as frequently the sounds are almost inaudible, only the second sound at the base being conducted to the ear. There is pulsation of the vessels of the neck out of proportion to the apparent force of the heart.

We may say, then, that myocarditis may exist (1) without definite physical signs—a rare occurrence; (2) with signs of moderate cardiac hypertrophy, marked reduplication being the only physical sign and of significance only when coupled with signs of endarteritis; (3) with physical signs of dilatation; (4) with physical signs of fatty degeneration; (5) with the physical signs mentioned, which in the aggregate are of great significance.

Aneurism of the Heart.

Aneurism of the valves, following endocarditis, can not be recognized during life. Aneurism of the walls usually results from chronic myocarditis. The aneurism develops in the left ventricle at the apex. The symptoms are indefinite. In rare cases a marked bulging has been noted in the region of the apex, and the tumor may perforate the chest-wall. A projection beyond the normal line of cardiac dulness may be detected by stethoscopic or plessimetric percussion. The symptoms are those of myocarditis and of dilatation of the heart. (See Diagnosis.)

Rupture of the heart is one of the causes of sudden death, often without previous symptoms. The accident takes place during exertion. Quain collected 100 cases, in 71 of which death took place without warning. In other instances there was a sense of anguish and suffocation in the cardiac region. The physical signs of slowly developing pericardial effusion may be obtained if the leakage from rupture is slow in progress.

Chronic Valvular Disease.

Valvular disease includes valvulitis and valvular incompetence; there is either obstruction or regurgitation at the orifices affected. Valvulitis may exist with or without symptoms; valvular incompetency is always accompanied by symptoms. Valvulitis implies organic disease of the valves; valvular incompetency indicates regurgitation through orifices whose valves, whether healthy or diseased, are unable to effect perfect closure.

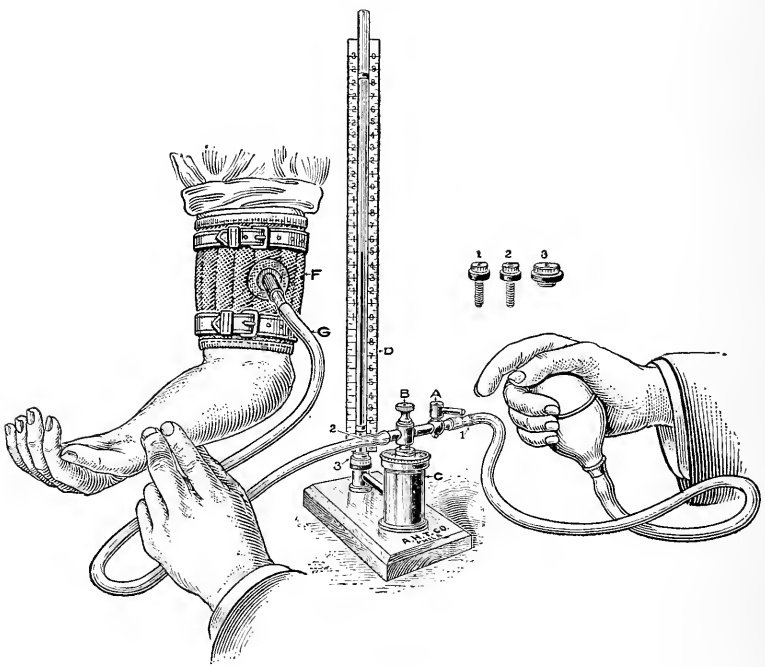
Valvulitis may be recognized by physical signs of (1) the lesion, (2) the secondary effects of the lesion on the heart and circulation—hypertrophy or dilatation.

Valvular incompetence occurs usually in dilatation, and may be secondary to valvulitis. It is recognized by both signs and symptoms. Valvular disease is without symptoms so long as the heart muscle enlarges sufficiently to keep in balance the impaired circulation; compensation is then said to be complete. When compensation is broken, we have the subjective symptoms enumerated above, all in consequence of dilatation

of the heart. It may be said that valvulitis is of no significance so long as compensation is perfect.

To review—valvulitis may be attended by physical signs in the heart and vessels only, or by its own physical signs, the physical signs of dilatation, and the symptoms of the latter. In the consideration of valvular disease it is more profitable to take up the symptoms of each valve-lesion, bearing in mind that two or more of the valves may be diseased at the same time, or that both obstruction and regurgitation may be present at the same time at the same valve-orifice. The severity of the valvular

FIG. 349.



Stanton's blood-pressure indicator.

defect, as well as the amount of work performed by the heart and coincident strain on the arterial system may be estimated accurately by means of an instrument that has appeared recently and which is shown in the accompanying illustration (Fig. 349).[1]

[1] With this apparatus, which is constructed on the general plan of the Riva-Rocci instrument (see Fig. 106 and p. 415), the highest (systolic) pressure is obtained by noting the height of the mercurial column at the moment when the pulse reappears after compression, while the lowest (diastolic) pressure corresponds to the point on the scale at which the greatest oscillations occur in the mercurial column. This instrument differs from others of its kind chiefly in the greater width of the compressing armlet, which is much broader than any hitherto employed; the mode of attachment of the manometric tube secures a firm and unyielding support; the rubber tubing is non-distensible; and the manometer is of special construction. The rubber armlet H (3½ inches wide) is prevented from expanding outward by a cuff, F, of double-thick canvas, reinforced by the insertion of tin strips, and is held by two straps which completely encircle the compressing band. The arm-piece is applied above the flexed elbow by overlapping the ends smoothly. The rubber armlet is connected by stiff-walled tubing, G, to the manometer. The manometer

Aortic Regurgitation: Insufficiency or Incompetence. This may exist for a long time without presenting any symptoms. It occurs more frequently in men than in women, and is more common in the later periods of life. It may be due to congenital malformation, to acute endocarditis, or, as is most frequently the case, to chronic endocarditis, particularly when it follows strain or undue exertion. Alcoholism and syphilis are also frequent antecedents. In rare cases it follows rupture of the valves. Relative insufficiency or incompetence is of very rare occurrence. Insufficiency is frequently combined with obstruction.

The blood falls directly into the left ventricle during the diastole. There is, first, a relative diminution in the amount of blood in the artery; and, second, an increased amount of blood in the ventricle, because the regurgitated column of blood meets the blood from the auricle which is filling the chamber at the same time. Dilatation of the left ventricle ensues, and is followed by hypertrophy. Dilated hypertrophy thus arises. The heart becomes enormously enlarged. This is one of the conditions in which enormous cardiac enlargement takes place—so-called *cor bovinum*. If this valve-lesion occurs late in life and from the above mentioned causes, it is attended by more or less sclerosis of the arteries.

Symptoms. They may be entirely absent so long as perfect compensation exists. This is particularly the case when there is but little general arterial sclerosis. Coincident lesions of other valves tend to break the compensation. The earlier symptoms are those due to arterial anæmia, particularly anæmia of the brain. They are headache, dizziness, and flashes of light before the eyes. The patient has an anæmic appearance, and soon begins to suffer from shortness of breath. This at first develops upon slight exertion. Palpitation and oppression about the chest are complained of, readily excited by undue exertion. Pain is a common symptom. It may be in the region of the præcordia, of a dull, aching character, and radiate to the neck and down the arms, particularly on the left side. The anginoid pains may be followed by attacks of true angina pectoris. The latter are more common in aortic regurgitation than in any other valve-lesion.

consists of the metal chamber C, and a horizontal metal tube inserted into its base, to which is screwed the vertical glass tube D, the calibre of which is approximately $\frac{1}{10}$ the calibre of C. The cap of C, which screws on, is provided with a metal T, one end connecting with the armlet and the other with the rubber-bulb, while the vertical limb opens into C. The stopcock A shuts off the rubber hand-bulb from the rest of the system, the screw-valve B allowing gradual escape of the air. By removing the vertical glass tube and screwing in the caps 1, 2, and 3, as indicated in the figure, the instrument is rendered transportable.

The armlet and cuff having been applied, the valve B is screwed tight and the cock A opened (arm parallel to limb of T). With the fingers of the left hand on the pulse, the rubber bulb is held in the right hand in the manner shown, leaving the thumb and index finger free. The bulb is repeatedly compressed until the pulse is no longer palpable, when, with the thumb and index finger of the right hand, the cock A is closed (turned at right angles). The screw-valve B is now slowly turned, and with the escape of air the mercurial column falls. The point at which the pulse reappears is mentally noted as the *high* or *systolic pressure*. At or even above this point oscillations appear in the mercurial column which increase as the column falls until they reach a maximum, and then again diminish. The pressure at which the greatest oscillation occurs is noted as the diastolic pressure. By unscrewing the valve B completely all the air is allowed to escape from the apparatus.

(See also "A Practical Clinical Method for Determining Blood-pressure," etc. William B. Stanton, University of Pa. Med. Bulletin, Feb., 1903, and the discussion.)

As *compensation* fails, venous stasis occurs and the dyspnœa increases. The latter is worse at night and compels the patient to sleep in a semi-erect posture. Congestion of the lungs takes place, giving rise to cough. Hemorrhage occurs, but not so frequently as in mitral disease. Œdema of the feet sets in, but general anasarca is not common. Œdema of the feet may be due to the attendant anæmia.

In aortic insufficiency *sudden death* is of common occurrence. This may take place at night during an attack of dyspnœa, or occur suddenly upon the slightest exertion, such as straining at stool, or ascending a height, or walking more quickly than usual.

Physical Signs. (Plate XXXI., Fig. 1.) INSPECTION. The apex-beat is downward, outward, and to the left. It may be as low as the seventh interspace, and as far out as the anterior axillary line. The area of cardiac impulse is increased. It occupies the whole præcordia, and heaving of the lower half of the chest may be seen. In young subjects there is præcordial bulging.

PALPATION. The impulse is strong and heaving. After compensation fails it is indefinite and wavy. A thrill, diastolic in time, may be felt if the hand is placed above the middle of the sternum.

PERCUSSION. The area of dulness is increased. The extent is greater than that in any other valve-lesion, and the enlargement is more particularly downward and to the left.

AUSCULTATION. At the second costal cartilage on the right a murmur is heard, *diastolic* in time. This may be its seat of maximum intensity. (See Fig. 128.) It is transmitted along the course of the sternum toward the apex. In some instances the seat of maximum intensity is at the fourth left costal cartilage, or even at the apex. The second aortic sound is absent in the large majority of cases. However, both murmur and second sound may be heard at the same time.

ASSOCIATE MURMURS. Other murmurs also may be associated with aortic regurgitation, not always due to disease of the aortic valves:

1. A *systolic* murmur at the second costal cartilage on the right, transmitted into the vessels of the neck, short, rough, and high in pitch. It is due to roughening of the valve-segments, to atheroma of the aorta, to aneurismal dilatation of the aorta or associated aortic stenosis.

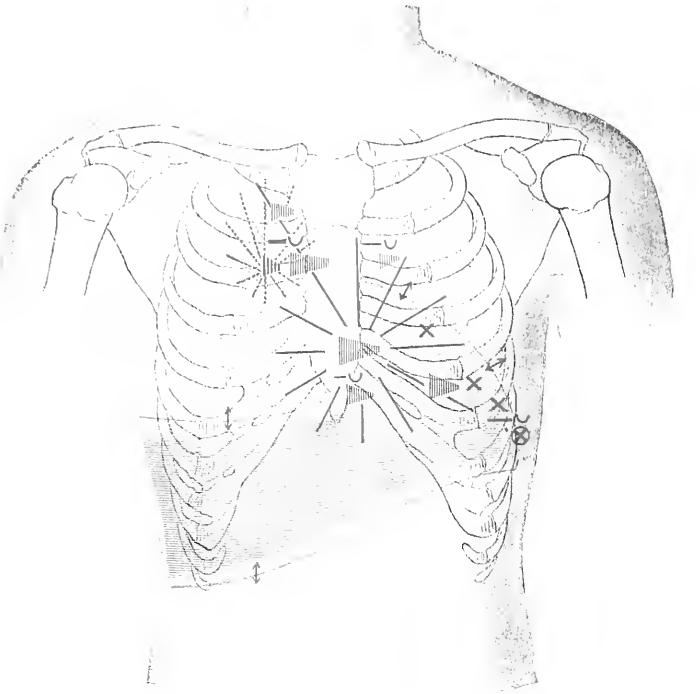
2. *Flint's Murmur.* A murmur at the apex, rumbling in character, localized to this area, usually *presystolic* in time. It is the murmur described by Flint, who attributed it to flapping of the mitral segments, which during diastole are not forced back against the heart-wall, on account of the dilatation of the ventricle. They remain in the blood-current and produce relative narrowing.

3. A *systolic* murmur in the mitral area, low in pitch, due to dilatation of the ventricle and consecutive incompetency of the mitral valves. This occurs when failure in compensation takes place.

EXAMINATION OF THE ARTERIES. Pulsation of the peripheral vessels is more common in aortic regurgitation than in any other valve-lesion. The carotids throb, the temporals pulsate, the brachial and radial arteries are conspicuous. Pulsation of the retinal arteries is seen with the ophthalmoscope, and has often led to recognition of the disease

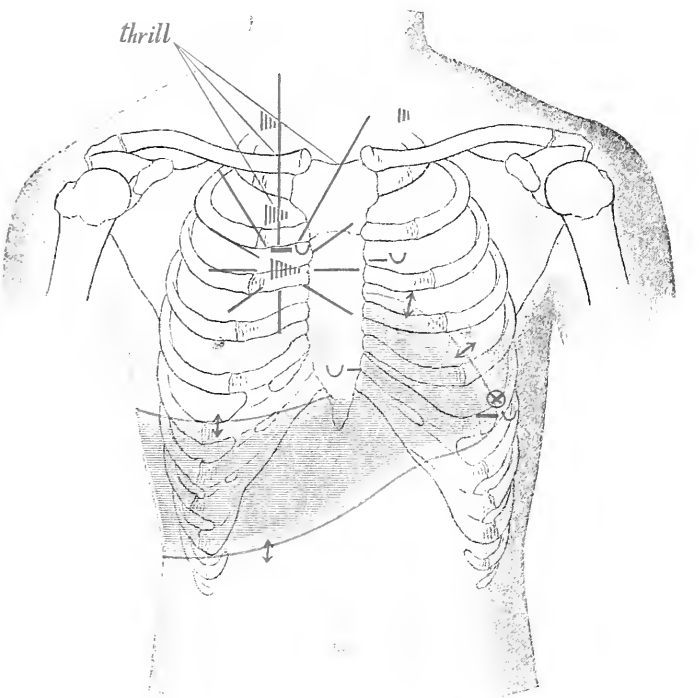
PLATE XXXI.

FIG. 1.



Aortic Regurgitation.

FIG. 2.



Aortic Obstruction.

by the ophthalmologist who had been consulted for other conditions. Pulsation of vessels in the fauces is occasionally seen. The pulsation is of a jerking character; in the neck it may simulate the pulsation of an aneurism. The aorta can be seen and felt at the suprasternal notch. The abdominal aorta pulsates vigorously in the epigastrium.

The *pulse* is significant in aortic regurgitation. The so-called water-hammer, or Corrigan's, pulse is observed. The pulse is quick, of large volume, and jerking, and after striking the finger immediately recedes. It is most marked when the arm is extended vertically.

On *auscultation* of the arteries double murmurs may be heard in the carotids and subclavians, and in rare instances they are present in the femorals. (See Pulse.)

The Capillary Pulse. This is seen beneath the finger-nails, or in the mucous membrane of the lips, or on the surface of the skin, as the forehead, when a line is drawn across it. The hyperæmia produced on either side of the line becomes alternately red and pale. Capillary pulse also occurs in anæmia, and at times in neurasthenia.

Diagnosis. No valvular lesion is more easily recognized when the arterial symptoms and signs are also present. The interpretation of a diastolic murmur at the base of the heart is difficult. It may be due to congenital disease, to aneurism, or to conditions of relaxation, as in anæmia or when there is unusual loss of tone in the bloodvessels. Cabot has noted a diastolic murmur, as seen in aortic regurgitation, in cases of anæmia, in adherent pericardium, and in dilatation of the aorta; and I have seen it come and go in exophthalmic goitre and in myocarditis.

Aortic Obstruction. Aortic obstruction occurs in the aged, and with atheroma of the arteries. It causes some diminution in the amount of blood in the peripheral circulation, resulting in poor nutrition and the development of anæmia.

Symptoms. Anæmia develops first, and embolic phenomena may occur later. When the obstruction is pronounced, the blood-supply in the arteries is diminished. Cerebral anæmia takes place, causing dizziness and fainting. Sleep is more disturbed than in other valve affections, because of the cerebral anæmia.

Embolic Symptoms. The symptoms may be latent until the occurrence of embolism. This accident is not uncommon, on account of the position of the aortic valve. The emboli are distributed throughout the arterial circuit, and may lodge in the brain, kidneys, or spleen.

Cardiac Symptoms. Palpitation and cardiac pain occur, but are not so common as in aortic regurgitation. When compensation fails, dilatation of the left ventricle ensues, followed by pulmonary congestion and stasis in the systemic circulation.

Physical Signs. (Plate XXXI., Fig. 2.) There is hypertrophy of the left ventricle.

Inspection. The apex-beat is displaced downward and outward. The impulse is strong during the period of hypertrophy. When compensation fails, the physical signs of dilatation ensue. In many cases, from the very first there may be considerable hypertrophy without the visible impulse,

because of associated emphysema, which is common in old men with this lesion.

Palpation. At the base of the heart and in the aortic area a thrill, systolic in time, may be felt. When present, it is usually very distinct, and is transmitted along the course of the vessels. The impulse is slow and heaving, if hypertrophy is present; if dilatation, feeble and indistinct.

Percussion. In the earlier stages the area of dulness is increased to the left and downward. After compensation is broken, dilatation with increased area of dulness ensues.

Auscultation. A murmur is heard of maximum intensity at the second costal cartilage to the right, *systolic* in time, and transmitted in the course of the bloodvessels. (See Fig. 125.) It is usually harsh and loud, but may be musical. As the heart weakens, the intensity of the murmur lessens and its roughness disappears. It becomes soft and low in pitch. The second sound, if there is no regurgitation, is muffled or may be absent. The *pulse* is slow, small, and regular (*pulsus tardus*). The tension is usually increased.

Diagnosis. A systolic murmur at the aortic orifice may be due to aortic obstruction, atheroma or dilatation of the aorta, ulcerative aortitis, or anæmia. Such a murmur is also common in aortic insufficiency without narrowing of the orifice, and is due to stiffened valve leaflets projecting into the blood-current. Huchard describes a murmur in this situation, with vibratory thrill, due to aberrant chordæ tendineæ. The murmur of aortic stenosis is distinguished from the others by its character, by the presence of thrill, by the character of the pulse, and by its association with hypertrophy of the left ventricle. A murmur due to atheroma of the aorta, particularly in the course of renal disease, is also associated with hypertrophy of the left ventricle. The diagnosis from aortic obstruction is often difficult or impossible. Slowness of the pulse is more characteristic of aortic obstruction. The murmur of anæmia is softer and low in pitch. There is no thrill, and the left ventricle is not hypertrophied. Anæmic murmurs may be heard elsewhere. In atheroma the second sound is usually accentuated, and in anæmia also it is intensified.

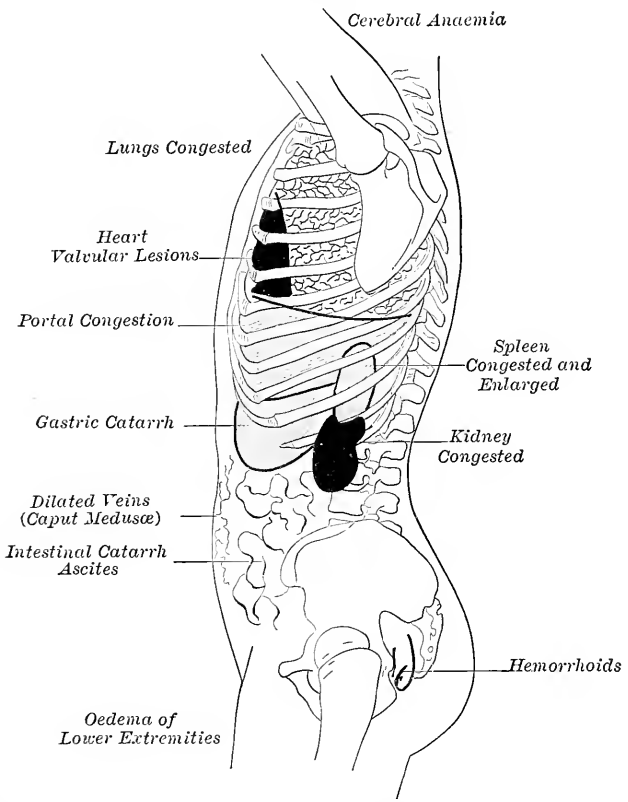
Mitral Regurgitation. The regurgitation may be due to disease of the valves (organic) from previous endocarditis; or to inability of the segments to close the orifice (incompetence), enlarged from dilatation. Incompetence occurs in dilatation of the left ventricle. It takes place when the muscle is weak in fevers and in anæmia. It is thus seen that the murmur of mitral insufficiency is one of the most commonly observed of all valve-murmurs. Its ready production and often equally ready removal with treatment make it the least serious. It must not be forgotten that insufficiency from disease of the valves and from disease of the muscles must, if possible, be distinguished from each other. The history of the case is essential in determining the diagnosis.

Mitral insufficiency or regurgitation has more serious effect upon the pulmonic and arterial circulation than any other valvular disease. These effects must be understood in order to appreciate the symptoms of mitral incompetence, which are as follows:

1. With each systolic contraction, on account of the insufficiency of the mitral leaflets, the blood flows back to the auricle, where it soon meets a volume of blood coming from the lungs. The combined volumes of blood over-distend the auricle. Dilatation ensues, and because of increased work to get rid of the increased contents, hypertrophy follows. Dilated hypertrophy of the left auricle is the first effect.

2. As a result of the above, a larger amount of blood is forced from the left auricle into the left ventricle; in order to remove the increased amount of fluid, dilatation and subsequent hypertrophy of this chamber follow.

FIG. 350.



Showing the congestions following valvulitis with failure of compensation.

3. On account of the over-distended auricle the pulmonary veins are not fully emptied during the diastole of that chamber. The veins are therefore engorged and interfere with the flow of blood through the pulmonary circuit. In consequence of the impeded flow of blood the vessels in the pulmonary circuit are dilated and over-distended. The right ventricle is compelled to act more vigorously, and even then can not empty itself freely. Dilatation and hypertrophy of the right ventricle ensue.

4. This condition causes obstruction to the flow of the blood from

the right auricle to the right ventricle: dilatation and hypertrophy of the right auricle follow.

If perfect *compensation* ensues through hypertrophy of both ventricles, engorgement in the lungs may not be observed. Moreover, the left ventricle is allowed to send out sufficient blood to supply the wants of the system. This compensation may continue for years. If it fails, either from increase in the valve-lesion, or valvular incompetence, or from weakening of the muscle, a normal amount of blood is not distributed throughout the aortic area, but is thrown back upon (1) the left auricle; (2) the pulmonary circulation; (3) the right heart; and (4) the systemic veins. For a time the pulmonary circuit alone is engorged, subsequently the systemic veins become congested because of dilatation of the right auricle and incompetence of the tricuspid valves. We then have the secondary effects of stasis upon the various organs of the body, with cyanotic induration and the development of dropsies.

Mitral incompetence without disease of the valves is of frequent occurrence in emphysema of the lungs and in Bright's disease, and is a condition which always attends hypertrophy and dilatation, or may take place from various causes. (See Hypertrophy and Dilatation.)

Symptoms. As to the general symptoms, in a large number of cases perfect compensation may continue for a long time; no subjective symptoms arise, nor are there symptoms due to dilatation.

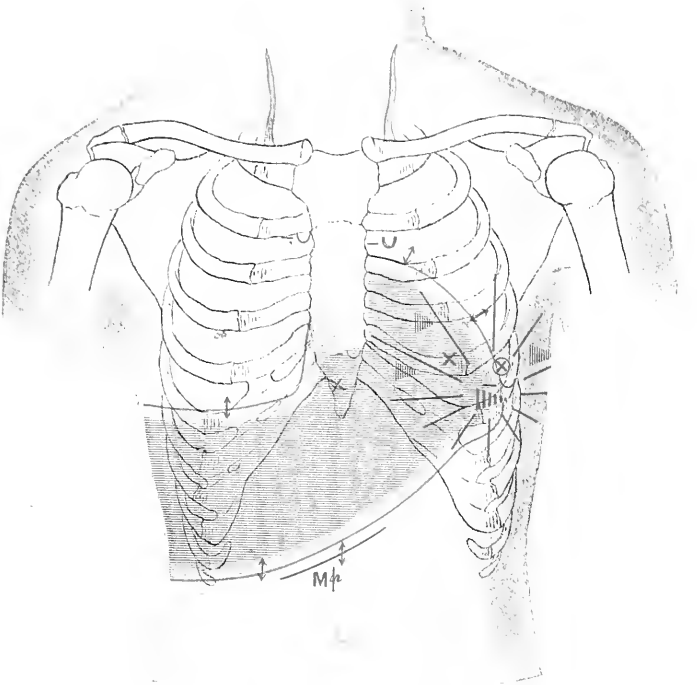
Compensation Moderate. If compensation is not perfectly effected from the first, or is broken suddenly or gradually, the symptoms of dilatation arise. In patients in whom compensation remains only fairly good we have the characteristic appearances of heart disease. It is to this class of patients that the general descriptions of heart disease apply. The face is pale and pinched, the lips and ears dusky, the capillaries of the cheeks enlarged, the finger-nails clubbed, particularly in children; shortness of breath on exertion may be the only symptom complained of, and this may exist for years. The patients are, however, liable to attacks of bronchitis and of pulmonary hemorrhage.

Cardiac Symptoms. Compensation Lost. Palpitation may occur in this as in other forms of heart disease, and from the same cause. When the compensation is broken, symptoms referable to the heart and to engorgement of systemic and pulmonary veins occur. Of the former, palpitation with a sense of oppression is the most common; pain is rare.

Venous engorgement leads to *congestion, cyanosis,* and *dropsies.* We now have the symptoms of dilated right heart superadded. The lungs are the first to be congested. Dyspnœa becomes constant and is aggravated by exertion. Cough is present, excited by exertion or speaking. With the cough there is bloody expectoration. Cyanosis occurs, and congestion of other organs follows. The liver is enlarged; obstruction in the portal area is prominent; chronic gastritis or gastro-intestinal catarrh ensues. The spleen is enlarged; ascites develops, and hemorrhoids and congestion in the rest of the portal area are seen. The kidneys are congested; the urine is scanty, albuminous, and contains casts and blood-corpuscles. At the same time that the internal viscera are congested dropsies take place, beginning in the feet and extending to

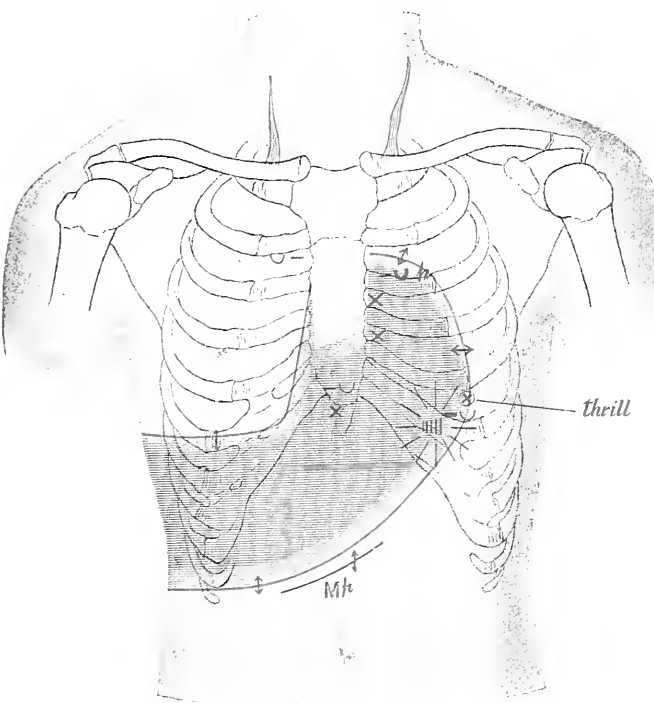
PLATE XXXII.

FIG 1.



Mitral Regurgitation.

FIG. 2.



Mitral Stenosis.

the rest of the body. Dropsy may have been present in the feet before symptoms of portal congestion ensued.

The patient may be relieved and compensation continue for a long time. Frequent attacks of dilatation of this character may take place, their recurrence being due to lack of care in hygienic matters, or failure of health from other causes. Finally, however, the compensation can not be restored; the stases persist; the dropsies become more marked, and the symptoms of cyanotic induration and secondary scleroses of the internal organs follow. It must not be forgotten that this is the chief form of organic heart disease seen in children.

Physical Signs. (Plate XXXII., Fig. 1.) INSPECTION. On inspection the præcordial area appears prominent; the apex-beat is displaced to the left and rarely downward. It may extend beyond the anterior axillary line. The cervical veins pulsate and are distended. The area of impulse is increased.

PALPATION. The character of the impulse depends upon the stage of the disease at which the case is examined. At the time of full compensation it is strong and even; when this is broken, it is feeble and diffuse. A thrill is uncommon; but when present, is systolic in time and felt at the apex.

THE BLOODVESSELS. The amount of blood in the arteries is diminished. There is notable absence of visible pulsation in the arteries. The pulse at first is full and regular. It is notably small in volume and soft. A soon as failure of compensation takes place the pulse becomes irregular. The irregularity may be that of time as well as of volume.

PERCUSSION. The area of dulness is increased transversely, but especially to the right. The transverse diameter of the heart is much increased because of dilatation of both chambers. The area of dulness may extend beyond the right margin of the sternum to the extent of an inch or more and to the left as far as the midclavicular line, sometimes to the anterior axillary line. The cardiohepatic triangle is preserved.

AUSCULTATION. At the apex, the mitral area, a murmur is heard. The point of maximum intensity is in this region. It is *systolic* in time; and may replace the first sound entirely. It may be soft and low in pitch, or rough, high in pitch, even musical in character. It is transmitted to the axilla and the angle of the scapula. (See Fig. 123.) In some instances it may be heard loudest along the left border of the sternum. The pulmonary second sound is accentuated; the accentuation is loudest in the pulmonary area at the second left interspace. It may be very loud over the right ventricle, between the parasternal line and the left edge of the sternum. The murmur of mitral insufficiency is modified by the position of the patient and intensified after exertion. It may be present when the patient is lying down, and disappear in an erect posture. It may disappear when the patient is quiet and return after exertion. Other murmurs are sometimes heard:

1. A presystolic murmur, soft or rumbling, due to associated mitral stenosis. 2. When dilatation ensues, a low-pitched systolic murmur is heard at the ensiform cartilage and at the lower left border of the sternum. It is due to tricuspid regurgitation.

Diagnosis. Of special diagnostic significance are: the position of the murmur and the direction of its transmission; accentuation of the pulmonary second sound; enlargement of the transverse diameter of the heart, due to dilatation of both ventricles.

The diagnosis is usually easy if the physical signs are sought for. Very often examination of the heart is neglected, and the patient is treated for the symptoms that arise from congestion of the viscera. We have often seen chronic gastritis or gastro-intestinal catarrh, due to mitral insufficiency, not relieved because the primary lesions had not been ascertained. In the same way cardiac cough or dyspnœa may be overlooked. It is important in the diagnosis to determine, if possible, the nature of the insufficiency, whether it is due to disease or incompetency of the valves. As previously mentioned, the history is possibly the only means by which a diagnosis can be made. If a mitral murmur ensues in old people in whom there has been physical cause for the development of dilatation and hypertrophy, as in emphysema or arteriosclerosis, it is usually due to relative incompetency of the valve. It must not be forgotten that the mitral area is the seat of a number of murmurs due to various causes. (See Auscultation, page 432.)

Mitral Stenosis. Obstruction to the flow of blood from the auricle to the ventricle is due to valvulitis, or endocarditis, and particularly the endocarditis of early life. It is of much more frequent occurrence in women than aortic disease. It is much more often seen in young adults and children, because its ætiological factors, rheumatism and chorea, are then more prevalent.

On account of the obstruction at the orifice changes ensue in the auricle. These changes depend in a measure upon the nature of the lesion. In the so-called buttonhole contraction they are very marked. The orifice may be so small in rare cases as to admit only a small probe. Dilatation and hypertrophy of the left auricle ensue if the valve-changes take place gradually. The walls of the auricle are thickened to three or four times their natural size. On account of the dilatation of this auricle the outflow from the pulmonary veins is impeded, which in turn obstructs the circulation of blood through the lungs. As a consequence, dilatation and hypertrophy of the right ventricle occur. As a result of this we have, later on, the occurrence of relative incompetency at the tricuspid orifice with engorgement of the systemic veins. The left ventricle does not take part in any changes. It retains its normal size, but it may look small in comparison with the right ventricle.

Symptoms. If hypertrophy of the right ventricle ensues, the compensation may be sufficient to prevent the occurrence of symptoms for many years. The disease may exist for a number of years without discomfort to the patient. Because of its rheumatic origin a fresh endocarditis may develop, particularly as most of the subjects are young. The old valve-lesion invites infection, and so a recurrent form of endocarditis is induced. If fresh endocarditis occurs, embolic symptoms are likely to follow. Embolism takes place particularly in the brain, causing hemiplegia or aphasia. When failure of compensation takes place, the symptoms

described in mitral incompeteney arise. They are the symptoms of dilatation of the heart, and may recur frequently during a long period of years.

Dropsy, however, is not so common as in mitral regurgitation. Visceral stases are common when compensation fails, and in many cases we find enlargement of the liver continuing for a long period. Ascites may in rare cases be the only manifestation of mitral obstruction.

Physical Signs. (Plate XXXII., Fig. 2.) The physical signs of mitral obstruction are more striking and more diagnostic of the lesion than the physical signs of any other form of organic heart disease.

Inspection. As the disease develops in children with soft ribs the local deformities are marked. For the same reason præcordial bulging is more prominent. Hypertrophy of the right ventricle causes the sternum and the fourth, fifth, and sixth costal cartilages to protrude. The apex-impulse is not usually displaced, certainly not beyond the mid-clavicular line. The impulse is not marked at the apex. In the third and fourth interspaces a visible impulse is seen along the margin of the sternum. After dilatation the extent of impulse diminishes and the veins of the neck become engorged, the blood regurgitating into them during the systole.

Palpation. In the large majority of cases a distinct fremitus or thrill is felt—more marked in the fourth or fifth interspace, inside of the nipple-line. It is diastolic or presystolic in time and is usually localized to a small area; is increased during expiration, and is of a twisting, grating, or grinding character. It is made up of a series of small shocks increasing in intensity, culminating in a sudden, sharp shock which occurs at the time of the impulse. The presystolic thrill and systolic shock are pathognomonic, and may be present when other signs, as the murmur, are absent or indistinct. The cardiac impulse is felt strongest at the lower margin of the sternum and in the third and fourth interspaces, in some cases even in the second. It is due to an enlarged and dilated right ventricle.

The Pulse. With perfect compensation the pulse is slow, regular, and of good tension, although small. If the mitral orifice is much narrowed, the pulse is small, weak, and irregular in force and rhythm. When compensation fails and the right heart is dilated, the pulse becomes rapid, quick, weak, small in size, and irregular in force and rhythm. The dilatation may be so great that the right auricle and over-distended veins may press upon the aorta or the innominate and subclavian arteries. The pulse on that side will be lessened in volume.[1]

Percussion. The area of cardiac dulness is increased upward and to the right and left of the margin of the sternum. Sometimes it extends upward as high as the second rib; this increase is quite characteristic.

Auscultation. At the apex, or just inside the position of the apex-beat, a murmur is heard, its point of maximum intensity distinctly localized to this spot. It is usually not transmitted. (See Fig. 126.) It is of a churning and grinding character, or vibratory and purring. It is usually high in pitch and rough. It occurs synchronously with the thrill, and terminates with a loud shock that is heard simultaneously with the

[1] Popoff, British Medical Journal, 1893.

first sound. It is therefore *presystolic* in time. As has been said of the thrill, so it may be said of this murmur, that it is the only murmur that is pathognomonic of a special lesion. It indicates narrowing of the mitral orifice. The only exception in which the lesion is absent, although the murmur is present, is found in the class of cases described by Flint, referred to in the section on aortic regurgitation. The first sound is loud, clear, and abrupt; it may be thumping.

The murmur of mitral stenosis may occupy the entire period of the diastole, but in the large majority of cases it occurs in the latter half only, during which the auricular systole occurs. In some instances it is heard in the middle of the diastole.

Associate Murmurs. 1. Synchronous with the above adventitious sound a systolic murmur may be heard at the apex, soft and low in pitch, which may be transmitted into the axilla. It is usually due to associated mitral regurgitation. 2. At the lower portion of the sternum a systolic murmur may be heard, due to dilatation and incompetency at the tricuspid orifice. Murmurs in the aortic region are not usually heard.

Presystolic Murmur not due to Valvulitis. A presystolic murmur without mitral obstruction may occur in aortic regurgitation and in adherent pericardium.

The *second sound* at the pulmonary orifice is usually *accentuated*. It is heard in the second and third interspaces along the left edge of the sternum. It may be heard at the apex. *Reduplication* of the first sound is often observed. Reduplication of the second sound is very common. After compensation is broken, other murmurs may be heard, and the presystolic murmur changes in character. It may disappear entirely and be replaced by a sharp first sound. The short, high-pitched systolic shock may continue, although the murmur disappears. It disappears probably because the left auricle has become weakened. The tricuspid murmur continues during this period.

Diagnosis. The points of distinction of mitral obstruction are (1) the position of the murmur; (2) its restricted area; (3) its peculiar character; (4) the systolic shock which takes the place of the first sound; (5) the thrill; (6) the impulse and increased area of dulness upward; (7) accentuated pulmonary second sound; (8) reduplication; (9) the absence of the pulse of aortic regurgitation and of hypertrophy of the left ventricle.

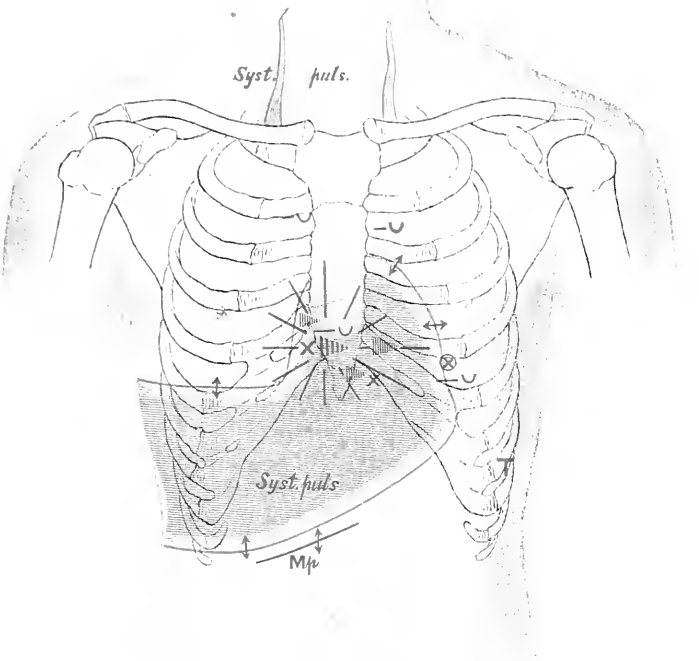
Tricuspid Regurgitation or Incompetence. Structural disease at the tricuspid orifice is of comparatively rare occurrence. Incompetence is more frequent, and is due to dilatation with relative insufficiency of the valve-orifice. It occurs secondarily to obstructive lung diseases, as emphysema and cirrhosis, and is secondary to obstruction and regurgitation at the mitral orifice which lead to stasis in the lungs.

Symptoms. The symptoms were detailed in speaking of the mitral valve affections. They are those of obstruction in the pulmonary circulation and engorgement of the systemic veins.

Physical Signs. (Plate XXXIII., Fig. 1.) Inspection. The physical signs of dilatation of the right heart are seen. An impulse in the epigastrium is noted. This is seen especially between the xiphoid cartilage

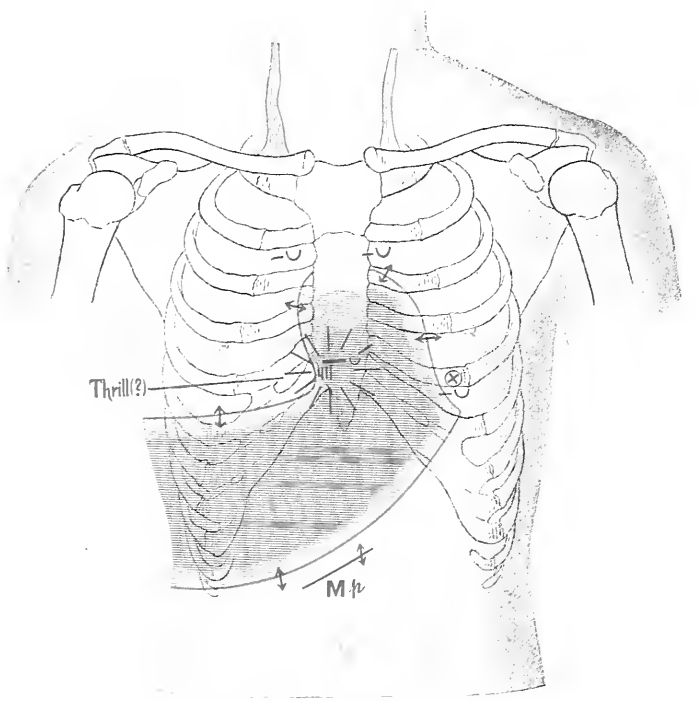
PLATE XXXIII.

FIG. 1.



Tricuspid Regurgitation.

FIG. 2.



Tricuspid Stenosis.

and the left margin of the ribs. Pulsation to the right of the sternum and in the second and third intercostal spaces may also be observed. The veins of the neck are also seen to pulsate. In addition to the wavy pulsation, regurgitation of the blood into the right auricle causes transmission of the pulse-wave into the veins. The pulsation is systolic in time. It is more marked in the right jugular than in the left, and in the external than in the internal veins. With the pulsation, regurgitation is readily observed by emptying the external jugular vein. Place the finger firmly on the vein just above the clavicle, move it along the course of the vein in the direction of the inferior maxillary bone. The vein is thus emptied of blood, and with each systole of the heart it will be seen to fill from below in rhythmical pulsation. The veins are increased in size. This is more noticeable during the act of coughing or when the patient holds his breath in full inspiration. In rare instances the pulsation is transmitted to the subclavian and axillary veins.

Palpation. By palpation the above conditions are also determined. The impulse over the lower sternum and in the epigastrium is noted to be forcible. The regurgitant pulsation is transmitted to the descending vena cava as well as to the ascending. The hepatic veins also distend during the systole. So-called *pulsation of the liver* is produced. With one hand on the fifth and sixth costal cartilages and the other over the liver in the axillary region, rhythmical expansile pulsation may be recognized. It is not of common occurrence, but is absolutely diagnostic of regurgitation at the tricuspid orifice. It must not be confounded with a pulsation transmitted to the liver from the heart or the aorta.

Percussion. The area of cardiac dulness is increased transversely and upward, as described in mitral stenosis. It extends often far beyond the right edge of the sternum.

Auscultation. A murmur is heard at the xiphoid cartilage, the lower end of the sternum, or the head of the fourth rib. It is *systolic* in time, usually low in pitch, and is heard clearly to the left of the sternum, within an inch of the apex, and to the right of the sternum and the outer limits of percussion-dulness. (See Fig. 124.) It is not transmitted further. Other murmurs, due to the primary organic disease, are heard. If the heart is weak, the lesion may not be productive of a murmur. The pulmonary second sound is accentuated.

Tricuspid Stenosis. Stenosis at this valve-orifice is generally of congenital origin. In rare instances it may be secondary to lesions in the left heart. It is accompanied by dilatation of the right auricle.

Physical Signs. The physical signs (Plate XXXIII., Fig. 2) are the same as in stenosis at the mitral orifice, except for the alteration in their position. In some instances a presystolic thrill has been observed, and with it a presystolic murmur at the lower end of the sternum or toward the right of it. The area of dulness is increased as in right-sided dilatation. Cyanosis is a prominent symptom and may be intense.

Diseases of the pulmonary valve are extremely rare and are almost always congenital.

Pulmonary Insufficiency. (Plate XXXIV., Fig. 1.) The *physical signs* are due to regurgitation into the right ventricle. The maximum intensity is in the second pulmonary interspace, and the murmur is transmitted down the sternum. It can not be distinguished from aortic regurgitation, except by the pulse.

Pulmonary Stenosis. (Plate XXXIV., Fig. 2.) In stenosis of the pulmonary valve a systolic murmur and thrill are detected to the left of the sternum in the second interspace. The murmur is not transmitted to the vessels of the neck. The pulmonary second sound is weak. The effect on the heart is the production of right-sided hypertrophy.

Combined Valvular Lesions. (Plate XXXV.)

It must not be forgotten that there may be disease causing both obstruction and regurgitation at the same time and at the same orifice, or that two or more valves may be the seat of disease in the same individual. It is not impossible, for instance, to have aortic obstruction and regurgitation, mitral obstruction and regurgitation, and tricuspid regurgitation. Aortic obstruction or insufficiency is frequently combined with mitral insufficiency. Aortic and mitral insufficiency occur together most frequently in children; aortic obstruction and mitral obstruction in adults.

When more than one valve is diseased, the site of the various lesions is determined by the time, the position of maximum intensity, and the direction of transmission of the murmurs. Students often experience difficulty here. A systolic murmur may be heard in the aortic area and in the mitral area at the same time. If it is observed that each progressively weakens as the stethoscope is moved toward the middle of the præcordial area, it may be inferred that the systolic murmur is due to two lesions. As previously intimated, the direction of the transmission of the murmur further aids in the diagnosis.

ENLARGEMENT OF THE HEART.

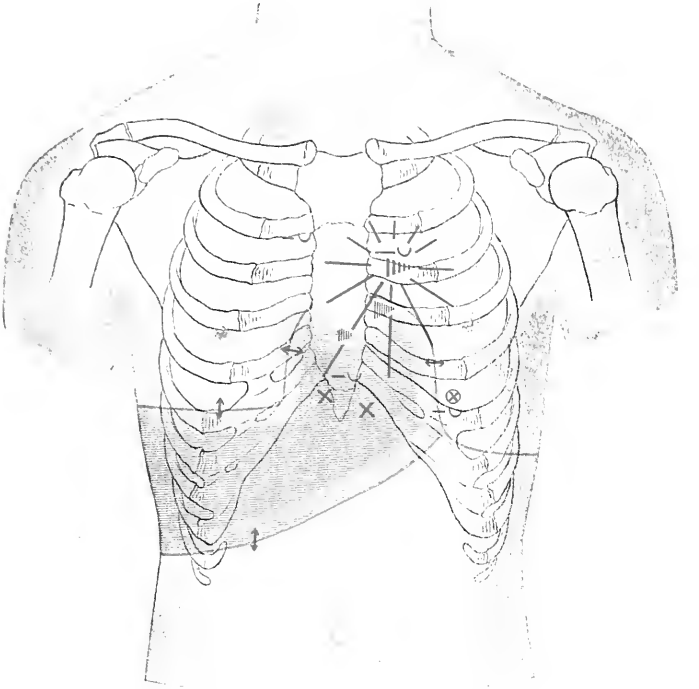
Enlargement of the heart is due to *hypertrophy* or to *dilatation*. In hypertrophy there is increased thickness of the muscular walls. This may be general or limited to the walls of one chamber. Hypertrophy is further divided into simple hypertrophy, in which the cavity or cavities are of normal size, and eccentric hypertrophy, in which, with increase in the wall, there is enlargement of the cavities. This is hypertrophy with dilatation. The left ventricle is most frequently the seat of hypertrophy when one chamber is involved. The cause of hypertrophy is obstruction to the flow of blood; increased work is followed by increased size of the muscle.

General hypertrophy occurs from over-action, from diseases of the heart itself, or from affections of the bloodvessels.

A. *Diseases of the Heart.* 1. Disease of the aortic valves. 2. Pericardial adhesions. 3. Myocarditis of the fibrous variety. 4. Neuroses with over-action and frequent palpitation, as in exophthalmic goitre and

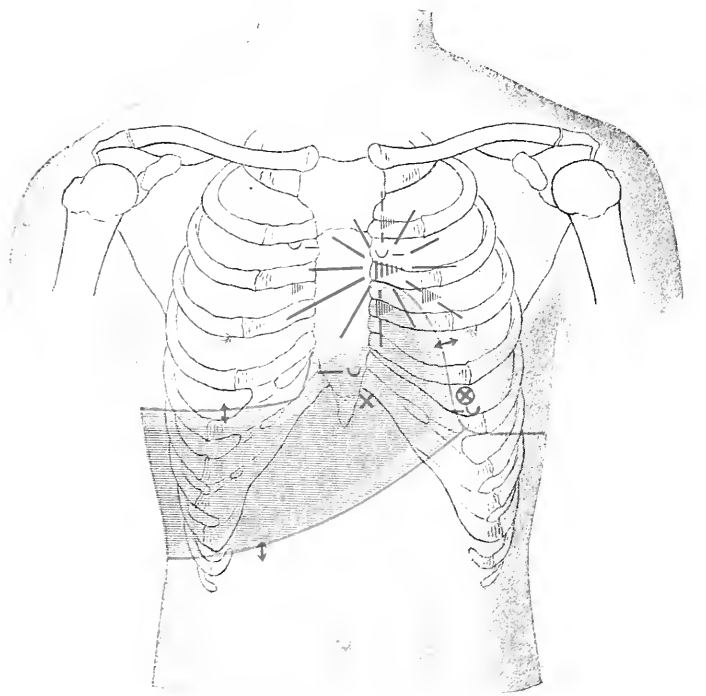
PLATE XXXIV.

FIG. 1.



Pulmonary Insufficiency.

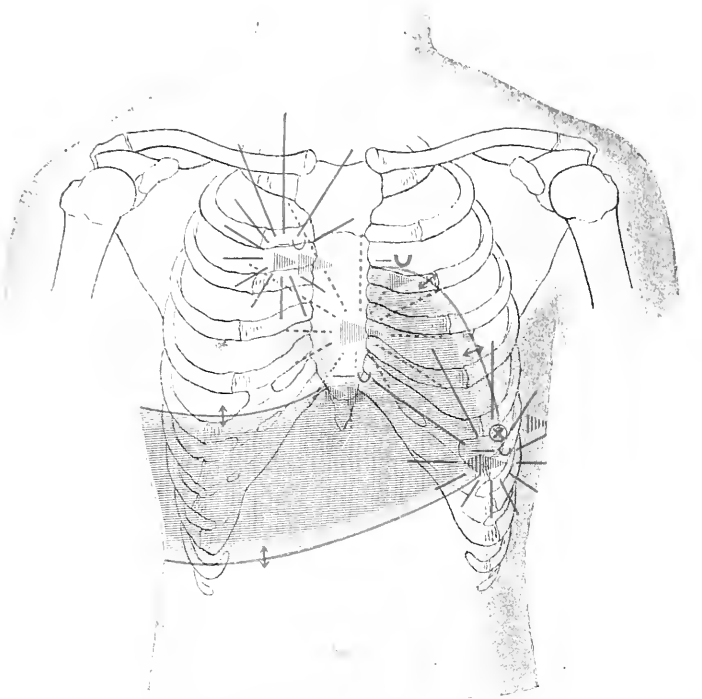
FIG. 2.



Pulmonary Stenosis.

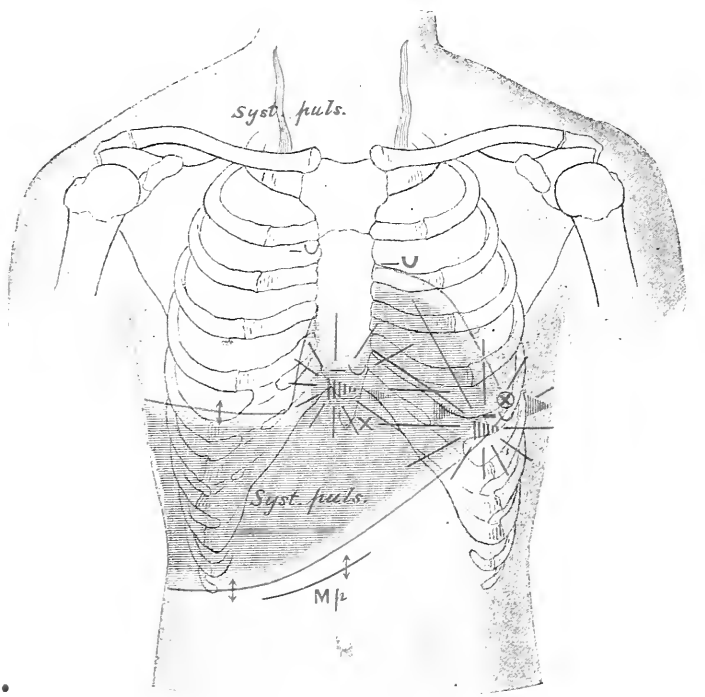
PLATE XXXV.

FIG. 1.



Combined Mitral and Aortic Insufficiency and Stenosis.

FIG. 2.



Combined Mitral and Tricuspid Insufficiency.

PLATE XXXVI.

FIG. 1.

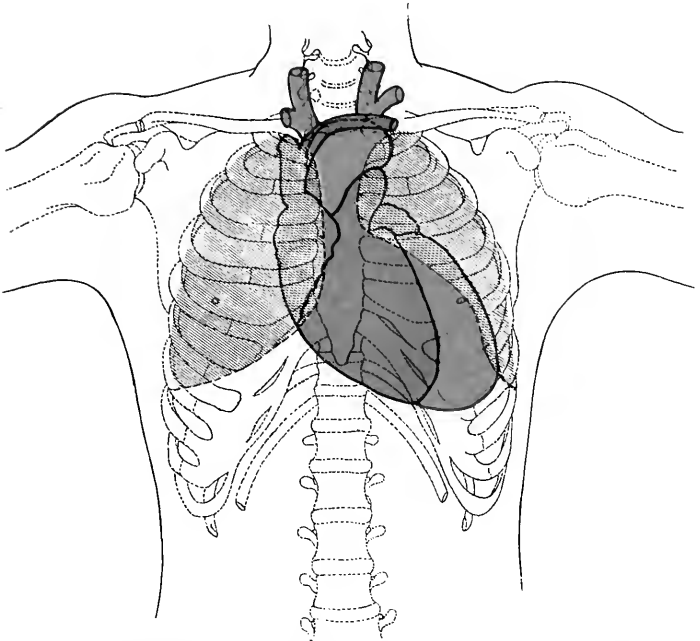


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in aortic insufficiency. RED = the left side of the heart (marked hypertrophy and dilatation). BLUE = the right side. The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2.

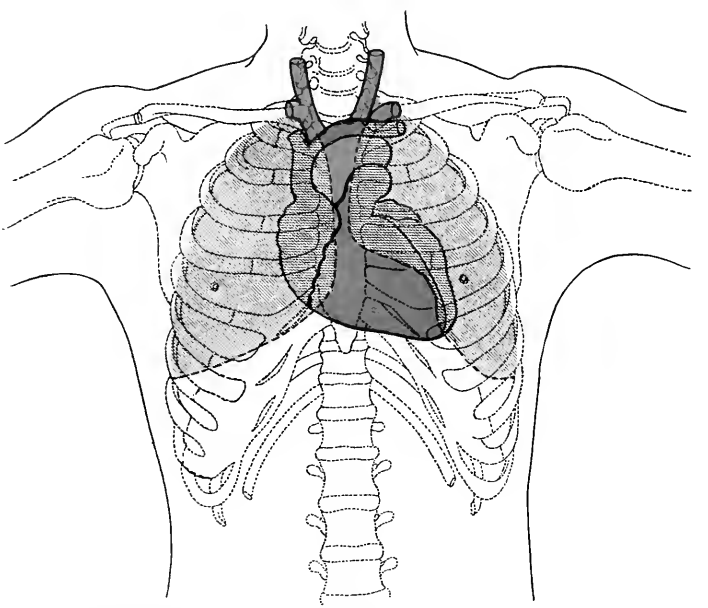


Diagram to illustrate the size and position of the normal heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness. RED = the left side of the heart. BLUE = the right side. The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

PLATE XXXVI-a.

FIG. 1.

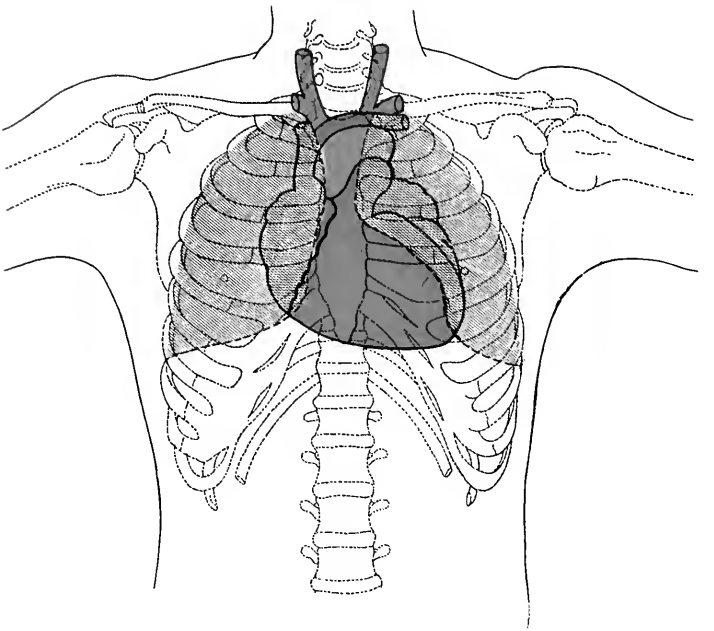


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral stenosis. RED = the left side of the heart (hypertrophy and dilatation of the auricle). BLUE = the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

FIG. 2.

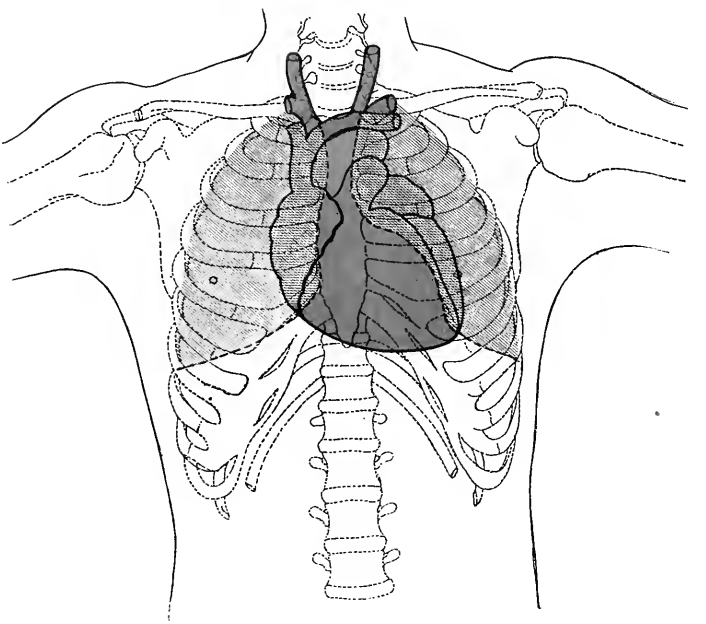


Diagram to illustrate the size and position of the heart and adjacent bloodvessels, the parts of the heart covered and uncovered by the lungs, and the superficial and the deep cardiac dulness in mitral insufficiency. RED = the left side of the heart (hypertrophy and dilatation). BLUE = the right side (hypertrophy). The DARK area represents the part of the heart uncovered, the LIGHT area the part covered, by the lungs.

from the effects of tea, tobacco, and alcohol. In pericardial adhesions and myocarditis hypertrophy arises because of the inability of the heart to do the work expected of it. There is no obstruction in the course of the vessels or at the orifices. The struggle to keep up causes the hypertrophy. In neuroses there is absence of obstruction, but the rapid action causes hypertrophy. (See Plates XXXVI. and XXXVI.–A.)

B. *Affections of the bloodvessels* which cause hypertrophy are: 1. General arterial sclerosis. 2. Increased arterial tension due to contraction of the peripheral arteries, as in Bright's disease, and in toxæmias from lead, the poison of gout and of syphilis. 3. Increased blood-pressure from prolonged muscular exertion. 4. Narrowing of the aorta from external pressure and from congenital stenosis or the development of an aneurism.

Hypertrophy of the Left Ventricle. Disease of the aortic valves, mitral valvulitis with regurgitation, and those diseases of the vessels which cause general hypertrophy also cause left ventricle hypertrophy.

Hypertrophy of the Right Ventricle. Obstruction to the flow of blood in the pulmonary area is the usual cause of hypertrophy of the right ventricle. This obstruction occurs in lesions of the mitral valve, causing pulmonary stenosis; and in disease of the lungs causing compression of the bloodvessels, as in emphysema or cirrhosis. It occurs if there is disease of the right heart with obstruction of the valves. Thus, in obstruction at the pulmonary orifice the right ventricle undergoes secondary hypertrophy.

Hypertrophy of the Auricles. Simple hypertrophy of the left auricle with dilatation develops in mitral stenosis. Hypertrophy of the right auricle occurs in tricuspid obstruction and in right-sided dilatation with tricuspid regurgitation.

Symptoms. *General Hypertrophy.* The symptoms of hypertrophy of the heart are *general* and *local*. The former are not common. They are due to increased tension in the cerebral vessels because of increased force of the heart, usually causing congestive headaches, noises in the ears, flashes of light, and flushing of the face.

General symptoms arise in hypertrophy of the left ventricle because the increased force causes reactive spasm of peripheral vessels, and hence increased tension in the vascular system. In Bright's disease, for instance, or heightened arterial tension from other causes, endarteritis develops in the large vessels on account of the strain put upon them. This is seen particularly in the aorta and its divisions. Whether atheroma is primary or secondary, its presence, with hypertrophy of the left ventricle, indicates that rupture of the vessel somewhere in the periphery may take place. This occurs most frequently in the brain, causing apoplexy.

Locally, the patient complains of fulness and discomfort, particularly marked when lying on the left side. In the hypertrophy that accompanies the tobacco-heart, or the irritable heart of soldiers, there may be some pain. On the other hand, the organ may be enormously enlarged without the patient complaining of discomfort about the heart. Palpitation is not of common occurrence except in neurasthenic subjects.

Physical Signs. The hypertrophy causes præcordial bulging, if it has

developed early in life, when the ribs are soft. The intercostal spaces are widened and the area of impulse is much increased. The normal impulse is changed in position. It is downward and to the left, often extending as far as the axilla in hypertrophy of the left ventricle.

Palpation. The impulse is forcible and heaving. The observer's head is visibly raised with each systole when placed upon the chest for auscultation. The impulse is slow. This slow, heaving impulse distinguishes it from the forcible impulse of dilated hypertrophy, which is sudden and abrupt. Inspection is confirmed as to the position of the apex. In moderate hypertrophy the apex extends to the sixth interspace in the midclavicular line. In extreme hypertrophy it may extend to the seventh interspace. The heart may be apparently hypertrophied in fibrous and fatty myocarditis; on the other hand, the impulse may be absent in emphysema, in fatty over-growth of the heart, and in persons with thick chest-walls.

The Pulse. The frequency of the pulse is not affected. It is full, regular, and strong. The tension is increased. In dilated hypertrophy the pulse is full but soft, and more rapid than in simple hypertrophy. When failure of the heart takes place, the pulse increases in frequency and becomes intermittent and irregular. When valve-lesions are present, the pulse is modified accordingly.

Percussion. The area of dulness is increased both upward and transversely. It may begin as high as the second interspace and extend two inches beyond the left midclavicular line, and an inch beyond the right edge of the sternum transversely. In simple hypertrophy the area is ovoid.

Auscultation. When the valves are healthy, prolongation of the first sounds occurs. They are also at times duller than in health. The dull, prolonged first sounds distinguish hypertrophy from dilatation, for in the latter they are clear and sharp. The second sounds are clear and loud. The degree of accentuation depends upon the state of the peripheral arteries. If there is heightened tension, the second sound may be reduplicated. If valvular disease is present, the sounds are modified.

Hypertrophy of the Left Ventricle. The symptoms of general hypertrophy obtain in left ventricle hypertrophy.

Hypertrophy of the Right Ventricle. Increased pulmonary tension from resistance in the pulmonary circulation may always be looked for in this condition. If there is complete compensation, no symptoms are observed, or only dyspnœa on unusual exertion. Hypertrophy of this ventricle persists for a long period without the grave local changes in the heart, or secondary changes in the peripheral vessels that occur in left ventricle hypertrophy. In dilated hypertrophy, when the dilatation is in excess, tricuspid regurgitation takes place, with the development of venous stases. Induration of the lungs may succeed the persistent engorgement of the capillaries. Pulmonary congestions and apoplexy may also occur.

Physical Signs. The physical signs of hypertrophy of the right ventricle have been referred to under the various valve affections. There is bulging of the lower part of the sternum and cartilages. The epigastric impulse in the angle between the ensiform cartilage and the ribs has been

referred to. The impulse may be in the sixth interspace. It is diffuse; it may extend upward as in mitral stenosis. Cardiac dulness is increased toward the right an inch or more beyond the border of the sternum. The heart-sounds are not much changed unless there is dilatation. The tricuspid sound is clear and sharp when this occurs. The pulmonary second sound is accentuated, and reduplication may take place. The radial pulse is small. If there is tricuspid regurgitation, the physical signs that attend it are present.

Hypertrophy of the Left Auricle. This is present in mitral stenosis, but can not be determined by physical signs, save possibly by greater increase of dulness to the left of the sternum in the second and third interspaces. Barr states that dulness above the "suprasternal-mammillary line" toward the left clavicle indicates enlargement of the left auricle, as in mitral stenosis. The line above mentioned is drawn from the middle of the suprasternal notch to the normal site of the left nipple on the fourth rib.

Hypertrophy of the Right Auricle. Hypertrophy of the right auricle with dilatation occurs under the same circumstances as hypertrophy of the ventricle. The right usually dilates more than the left auricle in left ventricle hypertrophy. There is increased area of dulness in the third and fourth right interspaces; abnormal pulsation is sometimes observed in this situation before the systole, with the signs of tricuspid regurgitation.

Diagnosis. The forcible impulse in nervous palpitation of the heart must not be confounded with true hypertrophy, although it must not be forgotten that hypertrophy frequently follows neurotic palpitation, as in the smoker's heart, or in exophthalmic goitre. The enlargement must not be confounded with enlargement of the area of cardiac dulness in the præcordial region from other causes, such as pericardial effusion; aneurism and mediastinal tumor, pushing the heart against the chest-wall; disease of the lungs, on account of which they are withdrawn from the surface of the heart, as in phthisis or chronic pleurisy; and displacement of the heart from pressure, as in effusion on the left side of the chest or in disease below the diaphragm. The cause of hypertrophy should be ascertained, for it is a valuable aid in diagnosis. It must not be forgotten that emphysema of the lung may mask a considerable hypertrophy of the heart by causing diminution of the area of dulness.

Dilatation of the Heart. Enlargement due to dilatation of the heart is common. The condition usually succeeds hypertrophy. Thickening of the muscles attends dilatation of the cavities, as in dilated or eccentric hypertrophy. The dilatation occurs because of increased pressure within the cavities or because of weakening of the heart-walls, the pressure within being normal.

1. Increased pressure within the walls is due to an increased amount of blood within the chamber from regurgitation, or from an obstacle to the outward flow of blood. Simple hypertrophy occurs first in many cases; in others, hypertrophy with dilatation; in not a few, dilatation takes place at once. In dilatation the chamber does not empty itself during the systole. It is seen physiologically after the exertion of ascending a great height. It may remain within the bounds of physiological action.

The dilatation may be temporary, as is demonstrated after running violently, by increased epigastric pulsation and increased cardiac dulness. The tricuspid valves temporarily become incompetent, owing to their safety-valve action. The latter may continue after the acute strain, the heart always showing symptoms of the condition; or it may disappear entirely. An excessive dilatation results in heart-strain, with cardiac distress and dyspnœa, symptoms due to over-distention and paralysis of the heart. (See Symptoms.) Dilatation occurs in all forms of heart-lesions previously described. The most typical form is seen in aortic regurgitation when the left ventricle is affected and in mitral regurgitation when the left auricle becomes the seat of dilatation.

2. Disease of the heart-walls, lessening the resisting power, the normal pressure within the cavities being maintained, invites dilatation. Acute dilatation may ensue in myocarditis and in infections. It occurs in scarlatinal dropsy, typhoid fever, rheumatic fever, and erysipelas. Certain changes which the heart muscle undergoes in acute endocarditis and pericarditis may lead to dilatation. In anæmia and chlorosis the same process may take place. In chronic myocarditis dilatation takes place at the apex. When pericardial adhesions are present, the fibrous overgrowth invades the interstices of the myocardium, thereby weakening the heart muscle. Dilatation may follow.

Symptoms. The symptoms of dilatation are the reverse of those of hypertrophy. When compensation fails, the blood is not expelled from the chambers in systole, so that the cavity is over-distended with blood which accumulates during the diastole. Weakening of the muscles also favors the development of dilatation. As soon as dilatation becomes permanent, incompetence of the valves takes place. In aortic obstruction, endarteritis the left side is first affected. It may be compensated for by hypertrophy of the right side. When this fails, venous engorgement and dropsy ensue. The symptoms have been described under chronic valvular disease. In *acute dilatation* there is a sudden occurrence of dyspnœa, and pain, or at least præcordial oppression may be complained of. The heart's action increases in frequency. The pulse is rapid, feeble, irregular, and may be barely palpable at the wrist.

Physical Signs. INSPECTION. The apex is displaced to the left, even so far as the axillary line, but rarely downward unless the hypertrophy precedes the dilatation. The impulse is diffused and undulatory in appearance. The apex-beat is defined with extreme difficulty. It may be visible when the patient leans forward, yet not felt.

With the diffuse area of impulse a quick apex-beat may be felt—much weakened, however. When the right ventricle is dilated, the impulse is seen and felt to the right or left of the xiphoid cartilage, and there is a wavy pulsation along the left edge of the sternum in the fourth, fifth, and sixth interspaces. If the dilatation is extreme, involving the right auricle, a pulsation at the third right interspace close to the sternum may be felt. Tricuspid regurgitation is then present.

The area of dulness is increased in the same directions as in hypertrophy, if the two coexist. In general, it may be said that the increase extends outward to the right or left, the direction corresponding to the

ventricle affected. It is increased upward along the left edge of the sternum in left auricle dilatation. (See Mitral Valvulitis.) When the whole heart is dilated, the increase of dulness is in a transverse direction on both sides. The apex is rounded or square, not pointed as in hypertrophy; indeed, it retains the oval shape of the dulness of a normal heart. As dilatation occurs so frequently in emphysema of the lungs, the modification of the percussion-sound must be remembered.

Auscultation. The systolic sounds are short and sharp. They are high-pitched and resemble the diastolic. The latter may become enfeebled when the dilatation becomes excessive. The right and left first sounds may differ somewhat in intensity, and reduplication may occur. The sounds may be obscured by murmurs. The murmurs are due to previous valve disease or to incompetency on account of dilatation. The action of the heart is irregular and intermittent. The pulse is correspondingly small. In dilatation the alteration of the rhythm is extreme. There may be *embryocardia* or fœtal heart rhythm, in which the first and second sounds are alike and the long pause is shortened. More frequently there is galloping rhythm of the heart. It must not be forgotten that, as dilatation ensues, murmurs of various valve-lesions may disappear, particularly the murmur of mitral stenosis. On the other hand, in the earlier stages particularly, murmurs develop on account of incompetence at the auriculoventricular orifices, in addition to the primary organic murmur. These murmurs in turn may disappear if the dilatation is controlled by careful treatment.

DISEASES OF THE ARTERIES.

Arterial Sclerosis or Arteriocapillary Fibrosis.

This occurs as the result of the wear and tear of life and as the accompaniment of age. The time of its onset depends upon the quality of the arterial tissue which the individual inherited, and upon the amount of wear and tear. It may occur early in life, and entire families may show this tendency. Very frequently the sclerosis develops from intoxications of the system, on account of which persistent spasm of the small vessels is set up; for blood of an impaired quality is passed with greater difficulty through the capillaries, as was taught by Bright. The blood-tension is raised thereby. Alcohol, lead, and the poisons of gout and syphilis lead to this condition. The poisons of gout and syphilis may set up directly an inflammation and degeneration of the arteries. In renal disease arterial sclerosis is of common occurrence. The relation to the renal lesion differs. It may be primary or secondary. When primary, the morbid cause operates upon the kidneys as well as the arteries. When secondary, a morbid poison is retained within the system by the diseased kidneys, the action of which is such as to cause peripheral spasm and heightened tension.

Over-filling of the bloodvessels from excessive eating and drinking is thought by some to cause arterial sclerosis through constant over-distention of the vessels. In over-work of the vessels and excessive strain there is

either heightened tension or increased peripheral resistance, the effect upon the bloodvessels being the same in either case. The result of the above causes is thickening of the intima, followed by changes in the media and adventitia, terminating in endarteritis deformans of the large arteries.

Symptoms. The symptoms vary. They may be general or local. The disease may be present although the patient dies from other causes. Local symptoms are due to rupture of the vessels, as in apoplexy from cerebral hemorrhage, or to their obstruction, as the coronary artery, or to rupture of an aneurism.

Physical Signs. Arteriosclerosis is recognized by inspection, palpation, and auscultation of the bloodvessels, and by observation of the condition of the heart. The superficial bloodvessels are elongated and tortuous, and pulsate visibly. On palpation the artery feels hard to the touch; it resists compression; it is corded or rounded underneath the finger, and readily rolled about. The pulse shows at once high tension; the wave is slow in ascent, continues long underneath the finger, and subsides slowly. If in the interval of the beats the vessel remains full, the pulse, as previously noted, is obliterated with difficulty. Sphygmographic tracings are characteristic. (See Pulse.) If after pressure on the radial artery it can still be felt beyond the point of compression, its walls are sclerosed; whereas, if after such compression the artery is obliterated beyond the point of compression, the hardness and firmness of the pulse previously observed are due to vascular tension and not to thickened walls. The two conditions should be distinguished. Hypertrophy of the heart occurs early in the course of the sclerosis, on account of peripheral resistance. The hypertrophy involves the left ventricle, and is not attended by dilatation. The apex-beat is beyond the midclavicular line and is usually displaced downward; the impulse is heaving and forcible. The second sound at the aortic cartilage is characteristic. It is clear and ringing; it is heard in the course of the bloodvessels, and is most distinct at or just beyond the apex. Right-sided hypertrophy and dilatation are not generally present. Auscultation of the larger arteries, as the carotids, the abdominal aorta, and femorals, shows a systolic murmur usually rough and high in pitch. All the above-mentioned conditions may be present, and yet the patient remain in apparent good health. The hypertrophy seems to compensate for the arterial occlusion. There may be no renal disease, or moderate renal cirrhosis may be present, indicated by transient albuminuria, polyuria, and hyaline tube-casts. The subsequent symptoms are due largely to closure of one or more vessels in the peripheral circulation, to the development of an aneurism or dilatation of the aorta, to failing hypertrophy of the heart, or to the development of renal cirrhosis.

The blocking of peripheral arteries is due to embolism or thrombosis, more frequently the latter, and to rupture of peripheral vessels, or in all probability miliary aneurisms. When occlusion of the vessels takes place in arteries which supply the extremities, gangrene may occur. Sometimes the occlusion is due to simple narrowing of the vessels alone. Gangrene of the feet is frequently the result of diseased arteries. If the occlusion takes place in the vessels of the brain, various secondary lesions are

produced. Acute and chronic softening occur in more or less general occlusion from sclerosis of the smaller arteries. Hemiplegia, monoplegia, or aphasia may occur—temporarily, if relieved by collateral circulation, or permanently from embolism, thrombosis, or rupture of the vessels. Hence, apoplexy is almost always due to primary disease of the arteries, in the walls of which, in the large majority of cases, miliary aneurisms have existed. If the coronary arteries are blocked, thrombosis with sudden death takes place, or chronic myocarditis may develop with subsequent aneurism and rupture. Angina pectoris, with or without thrombosis of the coronary artery, is always associated with arterial sclerosis.

Failure of the hypertrophied heart leads to dilatation with all the symptoms previously described, including cyanosis, visceral congestions, and dropsies. The murmur at the apex, due to incompetency from dilatation, may simulate chronic valvular disease, although the latter may never have been present. The sclerosis may advance more rapidly in the kidneys than in the other portions of the circulation; later, on account of the contracted kidney, symptoms of interstitial nephritis may arise.

Aneurism.

A true aneurism is formed by the distention of one or more of the arterial coats. It is usually saccular, but may be cylindrical or fusiform. The fusiform and saccular are the forms most commonly seen. False aneurism, or dissecting aneurism, arises from laceration of the internal coat of the artery. The blood dissects between the layers. It occurs in the aorta. It may begin at the heart and separate the coats as far down as the iliac arteries. *Arteriovenous aneurism* is seen when communication between an artery and a vein has been set up. If a sac intervenes, it is called a *varicose aneurism*. Sometimes communication is direct, the vein becoming dilated, tortuous, and pulsating. It is known as an *aneurismal varix*.

An aneurism may occur in the course of arterial sclerosis from diffuse distention of the coats. Its typical form is seen in dilatation of the aorta with one or more sacculated aneurisms on its surface.

Sacculated aneurism occurs from rupture of the tunica media, independently of more extensive disease of the arteries, and in arterial sclerosis. The most common seat is the ascending portion of the aorta. It occurs early in the course of arterial sclerosis. Such form of aneurism is seen in the smaller vessels. Aneurisms also arise after the lodgement of an embolus permanently plugging the vessel, the proximal end of the vessel becoming dilated.

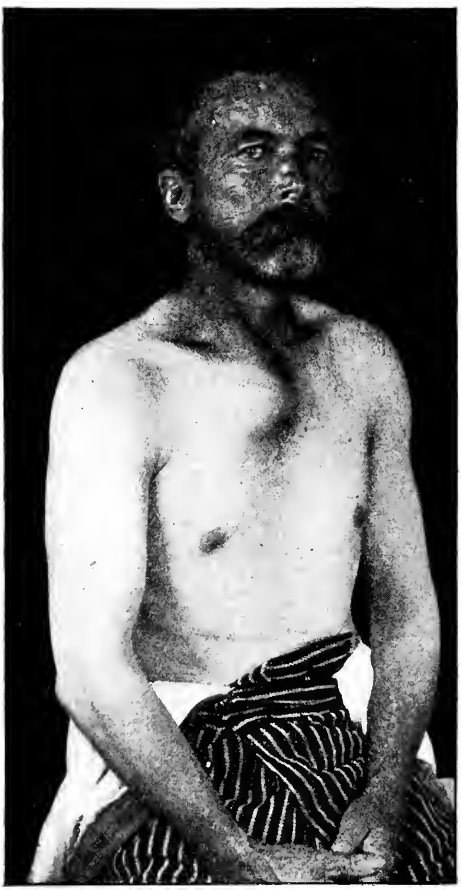
Mycotic aneurism, first described by Osler and exhaustively by Eppinger, occurs in malignant endocarditis. The aneurisms are small in size and multiple, and not recognized during life. They arise from the injury produced by the local infection of bacteria in different portions of the vascular system.

Aneurism of the Thoracic Aorta. The causes which produce arterial sclerosis are operative in the thoracic portion of the aorta—chiefly physical over-work, alcohol, syphilis, and gout. It may be

situated just beyond the aortic ring, at the junction of the ascending and transverse aorta, in the transverse, or at the beginning of the descending portion of the thoracic aorta. The larger aneurisms are at the two bends of the aorta.

Symptoms. The symptoms of aneurism are largely due to pressure, and depend upon the position of the aneurism and the direction of its growth.

FIG. 351.



Aneurism of ascending portion of arch of aorta. Tumor in first and second interspaces, extending into neck. Portion of sternum atrophied. (Original.)

Aneurisms, however, may exist *without symptoms* or *appreciable physical signs.* Even in a patient who has been under careful observation sudden death may take place from rupture of a concealed aneurism, the presence of which had not been suspected during life. On the other hand, cases occur with characteristic pressure-symptoms and with no physical signs.

Aneurisms of the *ascending portion of the arch* cause dislocation of the

heart outward or toward the right pleura or forward. They appear at the second or third right interspace, causing erosion of the ribs and sternum. The vena cava is compressed, causing enlargement of the veins of the head and arms; the subclavian vein may be compressed alone, causing enlargement and œdema of the right arm. Localized œdema may result, confined to the thorax. (See Œdema.) If the aneurism is large, the inferior vena cava may be pressed upon, causing œdema of the feet. The right laryngeal nerve may be involved, causing aphonia and dyspnœa. Pain attends the aneurismal process.

Aneurisms of the *transverse portion of the aorta* project below, forward, or backward. When forward, they produce tumors behind the manubrium, which from pressure cause destruction of the bone; if the aneurism projects backward, marked pressure-symptoms are produced. When the trachea is pressed upon, it causes dyspnœa and cough which is paroxysmal. (See Dyspnœa.) The œsophagus may be pressed upon, causing dysphagia. The left recurrent laryngeal nerve may be pressed upon, causing paralysis of the corresponding vocal cord, with aphonia; or there may arise hoarseness or a peculiar monotonous quality and inability to reach a high note. (See Larynx.) Pressure on a bronchus may produce bronchorrhœa and dilatation, which in turn may lead to localized abscess. The growth may extend upward, involving the coats of the innominate and carotid arteries on the right side, or carotid and subclavian on the left, markedly interfering with the pulse of the two sides. Pressure on the sympathetic nerve is likely to take place in this situation, with contraction of one of the pupils, although at first it is sometimes dilated. The thoracic duct is sometimes compressed, leading to rapid wasting.

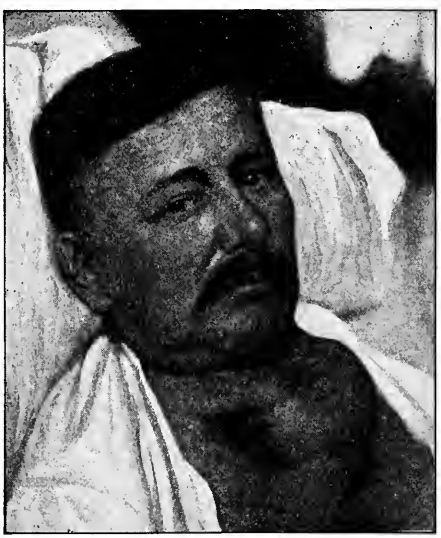
In the *descending portion* the pressure-signs of aneurism are often not so marked. The vertebræ are likely to be pressed upon in this situation. The pain, therefore, is most intense. The œsophagus and left bronchus are compressed. Dysphagia and bronchiectasis, the latter causing bronchorrhœa with subsequent gangrene, are likely to occur. The *cough* and the fever in bronchorrhœa, together with emaciation, simulate phthisis, for which aneurism is often mistaken. The physical signs of phthisis are usually pronounced in this situation, and, with the presence of bacilli in the sputum, render the diagnosis easy. In these cases rupture takes place into the bronchus or into the œsophagus. In one of my cases, which had been treated for tuberculosis because of small hemorrhages, with the conditions above mentioned, death took place from rupture into the bronchus, causing sudden profuse hemorrhage. When the aneurism is adherent to the œsophagus and slowly ulcerating into it, rupture may take place, followed by instantaneous death. The vertebræ may be eroded and symptoms of spinal compression arise.

I once saw an autopsy performed by a medico-legal expert on a case of sudden death from gastric hemorrhage. The source of the hemorrhage could not be ascertained. There was blood in the stomach. When he was about to give up the search, the œsophagus and aorta were suggested for examination. A small aneurism was found which had ulcerated and then ruptured into the gullet. In another case the aneurism had ruptured into the pleural sac, causing internal concealed hemorrhage and death.

Special Symptoms. While pressure-symptoms are the most striking symptoms of this affection, *pain*, which is usually due to pressure, must be referred to. It is an important and a constant symptom. It is sharp and lancinating, and may occur in paroxysms. It is more severe and constant when bone is eroded by pressure on the vertebræ, or on the ribs and sternum. The gnawing pain that attends ulceration of bone subsides after perforation has taken place. Anginal attacks may attend the neuralgic pains just described. Pain sometimes follows the course of the nerves, extending down the arm or to the neck, or along the course of the intercostal nerves.

Cough. The cough is peculiar. It is paroxysmal in many cases and of a brazen, ringing character, indicating its laryngeal origin, due to pressure upon the recurrent laryngeal nerves. It is frequently paroxysmal when the pressure is directed upon the windpipe or bronchus. In the former instance the cough is dry, in the latter tracheal and bronchial. It is attended by a thin, watery expectoration which, if bronchiectasis with fermentation ensues, becomes thick and ropy. *Dyspnœa* occurs more

Fig. 352.

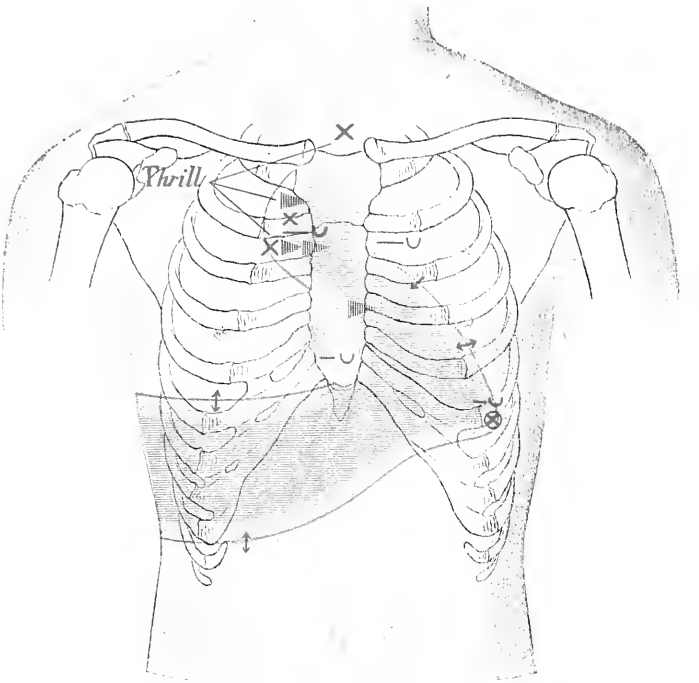


Aneurism of ascending and transverse portions of aorta projecting forward, destroying ribs and sternum. The skin ulcerated, and gradual external leakage took place. The bleeding continued in small amounts for a long time. (Original.)

frequently in aneurism of the transverse portion, due (1) to pressure on the recurrent laryngeal nerves; (2) to compression of the trachea; (3) to compression of the left bronchus. Marked stridor attends the first form. When one of the recurrent laryngeal nerves, more particularly the left, is pressed upon, there is spasm or paralysis of the muscles of the vocal cord, causing hoarseness and loss of voice. Laryngoscopic examination should not be neglected, for paralysis of the abductor muscles without symptoms may be present.

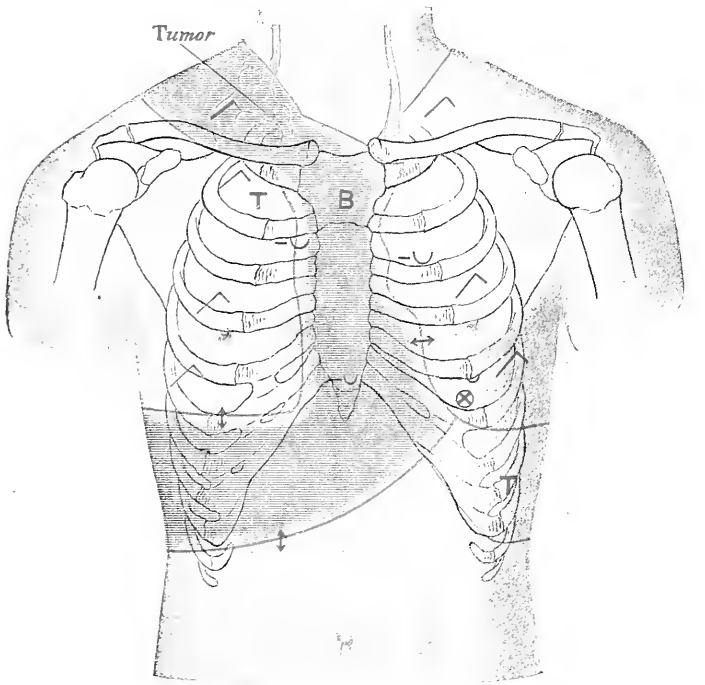
PLATE XXXVII.

FIG. 1.



Aneurism of the Arch of the Aorta.

FIG. 2.

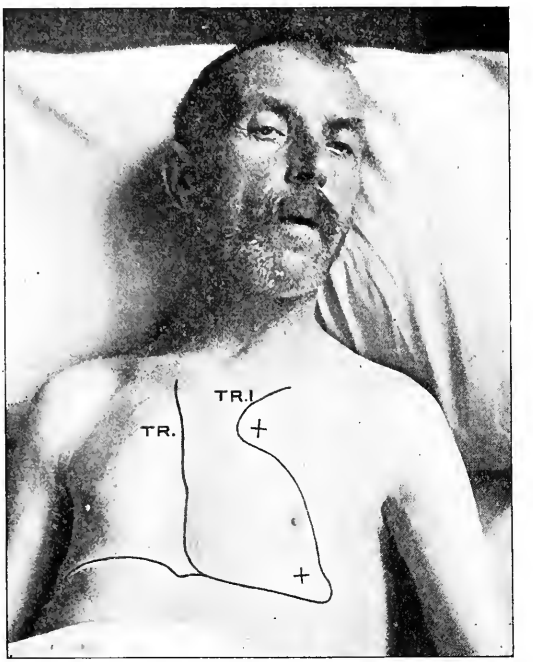


Tumor of the Anterior Mediastinum.

Hemorrhage. The hemorrhage may be gradual when there is slight leakage into the trachea at the point of compression. The amount of blood lost is small. It may take place externally. (See Fig. 352.) Profuse hemorrhages, causing sudden death, occur from rupture into the trachea or bronchus, and from perforation into the lung. With regard to difficulty of deglutition, it may be said that the œsophageal sound should never be passed in suspected cases of aneurism, on account of the danger of rupturing the sac.

Clubbed Fingers. In intrathoracic aneurism clubbing of the fingers and incurvation of the nails of one hand are sometimes, although comparatively rarely, seen.

FIG. 353.



Suspected aneurism. General endarteritis and valvulitis. (Original.)

TR. = Thrill and impulse. + = Murmur. Outline area of dulness. TR'. = In first interspace, thrill and murmur.

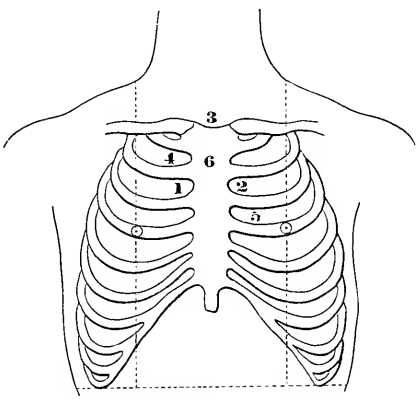
Compression and pressure on the *sympathetic system of nerves* have been referred to. If the pressure is moderate, sufficient only to irritate or stimulate the vasodilator fibres, dilatation of the pupil, usually associated with unilateral pallor of the face, results. If the pressure is sufficient to paralyze or destroy the vasodilator fibres, the pupil becomes contracted, and one side of the face hyperæmic and usually the seat of increased perspiration. Associated with the contraction of the pupil, narrowing of the palpebral fissure and exophthalmos may be observed; the latter occurs rarely, and is attributed in some cases to venous engorgement.

Physical Signs. (Plate XXXVII., Fig. 1.) INSPECTION. In health the position of the aorta can not be recognized. Pulsation may be seen

at the episternal notch in rare instances, particularly in women, independently of disease of the aorta; it is due to nervous palpitation. An aneurism may exist without external visible signs. On the other hand, pulsation may be seen at either side of the sternum above the level of the third rib, most commonly in the second interspace on the right side. The impulse may be seen alone without visible swelling; the chest must be viewed from different situations in order to detect it. An oblique light falling on the surface is sometimes necessary. When the innominate artery is involved, the pulsation is observed in the neck, above the sternoclavicular junction or above the sternum.

With the abnormal impulse a swelling or *tumor* is often present. It may be sufficiently large to press the upper portion of the sternum and adjacent ribs forward. In other instances a tumor the size of the half of a lemon may be seen along the edge of the sternum. The most frequent site is the first and second right, or the second left interspace. The skin over the tumor, as in the case of which an illustration is given, may ulcerate and be the seat of persistent small hemorrhages. The *apex-beat* of the heart is displaced downward and outward from pressure.

Fig. 354.



Possible positions of impulse in aneurism, arranged in order of frequency.

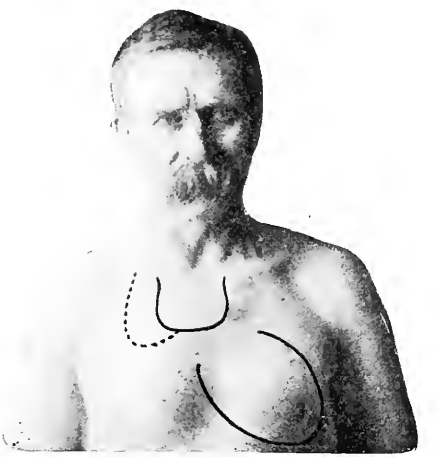
If the aneurism is seated in the ascending portion of the aorta, just beyond the aortic ring, a pulsating tumor may be seen in the third interspace at the right edge of the sternum. If in the ascending portion, beyond the heart, the tumor is in the first or second interspace along the right edge of the sternum. If the aneurism is in the transverse portion of the aorta, the upper portion of the sternum is frequently made to protrude, or the tumor projects upward into the fossa of the neck. If in the descending portion, it is in the second or third interspace on the left side. In this portion of the aorta a tumor is seen in the left scapular region in rare instances.

Palpation. Palpation must be employed by the usual method; bimanual palpation must also be used, one hand placed upon the sternum and the other upon the vertebræ. Moderate pressure should be exerted.

Palpation should also be employed at different periods of respiration. At times signs are only yielded at the end of complete expiration. It must further be said that palpation must be employed both with the tips of the fingers and with the palm of the hand applied to the surface.

By palpation the area and degree of pulsation are determined. If the aneurism is large or has perforated, the impulse is expansile and heaving in character. The sac may be soft and fluctuating, but usually presents considerable resistance. In addition to the systolic impulse the diastolic shock is also felt. This is a most conclusive physical sign. A thrill is frequently present, systolic in time, usually due to dilatation of the arch; at times, to sacculated aneurism. Without visible tumor, pulsation and thrill may be felt in the suprasternal notch, if the head is bent forward, so that the tissues are relaxed, and the fingers pushed down toward the aorta. When the aneurism is filled or filling with clot, the tumor may be seen and felt, but no impulse will be transmitted to the hand, or thrill be felt by the fingers.

FIG. 355.



Aneurism of aorta.
Area of absolute dulness, dark line. Area of relative dulness, broken line. (Original.)

PERCUSSION. Percussion furnishes the most reliable evidence of the presence of an aneurism or aneurismal dilatation in cases in which the tumor is not too deep-seated or small in size. The dulness may be relative only. (See Cardiac Percussion.) The area of dulness is increased somewhere in the course of the aorta. It may be observed projecting outward at the right edge of the sternum when the ascending portion of the aorta is the seat of disease, or over the entire upper part of the sternum extending toward the left, when the transverse portion is diseased. It may be observed as an extension of cardiac dulness upward in the second and third interspaces. Sometimes dulness is detected in the scapular regions, particularly of the left side. The percussion-tone is flat, and there is marked sense of resistance. Percussion must be employed with the patient in the upright and in the recumbent posture. Eichhorst states that he has made a provisional diagnosis of aneurism in 3 cases, from the

occurrence, during percussion of the anterior chest, of severe paroxysms of coughing, and of the complaint on the part of the patient of severe pain over a localized area. The cough was also induced by palpation of these areas. The necropsy confirmed the diagnosis in each case, and Eichhorst believes that these signs are of considerable diagnostic importance.

Respiratory Percussion. The character of the note and the shape of the dulness must be noted at the end of full inspiration and of full expiration.

Auscultatory percussion is of the utmost value, and the method of percussion taught by Sansom and Ewart must be carefully followed. An aneurismal tumor may be present without thrill or murmur, but yields signs of dulness on percussion.

Auscultation. As just stated, murmurs may not always be present. They depend upon the amount of fibrin in the sac. When present, the murmur is usually systolic in time, heard with maximum intensity usually over the abnormal area of impulse or tumor, or over the increasing area of dulness. It is transmitted in the direction of the vessels, and may be heard louder in the vessels of the neck and along the course of the aorta. Often a double murmur is heard, the diastolic sound being frequently due to associated regurgitation at the aortic orifice. Sometimes the diastolic murmur alone may be heard. Increase in intensity or accentuation of the aortic second sound is pronounced. The sound is ringing in character, and is rarely absent in large aneurisms.

Gerhardt has described a rare arteriodiastolic murmur in the left interscapular region, due to beating of the aneurismal sac against the left bronchus. Glasgow has described a systolic thud or shock in the brachial artery similar to that which occurs in aortic insufficiency.

The Peripheral Vessels in Aneurism. The pulse in the two radial arteries may show a marked difference both in volume and in time. The difference may indicate the position of the aneurism. If the pulse of the right radial is smaller than the left, the aneurism may be in or near the innominate artery; if the opposite, it is near or includes the orifice of the left subclavian. In the same way the difference in time may also aid in determining the location. Osler refers to obliteration of the pulse in the abdominal aorta and its branches. In one case he could not feel throbbing in the aorta and the femorals, although the circulation was unimpaired. The aneurism was in the descending portion of the aorta, and its pulsation was seen in the left scapular region. The sac was sufficiently large to act as a reservoir which filled during the ventricular systole and emptied in a continuous instead of an intermittent stream, the effect of the ventricular systole being lost.

Tracheal Tugging. Tracheal tugging may be obtained in one of two ways. By the old method the patient should be sitting or standing, while the observer sits or stands to one side, and faces him. With the hand farthest from the patient steadying the head, the observer gently but firmly grasps the surface of the cricoid cartilage with the thumb and finger of the other hand, while the head is slightly thrown back. The head is then flexed, so that the neck is no longer stretched. The patient is then told to hold his breath completely, and any up-and-down move-

ment of the trachea is immediately transmitted to the observer's fingers. One must not mistake the transmitted pulsation in the cervical vessels for such movement; and great care should be exercised to see that the breathing is entirely stopped.

In the other method, as proposed and practised by Ewart,[1] the observer stands behind the patient, steadying the latter's head against his body, and the cricoid is firmly held between the tips of the first or middle fingers. The writer, after considerable experience, prefers this second method, on account of delicacy of touch, firmness of grasp, and comfort to the patient.

Similar to this, which is known as Oliver's sign, is Cardarelli's sign—lateral movement of the larynx. It differs from Oliver's only in the direction of the movement of the larynx and is of similar diagnostic value. In some cases lateral or anteroposterior deviation of the trachea in the neck may be observed, and in other cases the upward and downward movement of the larynx and trachea during deglutition is much restricted. Hall has lately referred to a tracheal diastolic shock which he regards as important in the diagnosis of aneurism.

Diagnosis. The special points of diagnosis are: the ætiological factors; the antecedent pathological conditions, as arterial sclerosis; the occurrence of pain; the occurrence of pressure-symptoms; and the physical signs. These have been sufficiently dwelt upon, and it is not necessary to consider them again. It must not be forgotten that aneurism may be present without diagnostic physical signs, and, on the other hand, the pressure-symptoms may also be in abeyance. If one of the two is present in a male subject past forty, with a previous history of syphilis, gout, alcoholism, or muscular strain, the probability is that an aneurism exists.

Cases are often seen that present physical signs of aneurism which are due to valvulitis and occasionally to pericarditis. The remarkable case from which Fig. 353 was taken simulated aneurism during life, in thrill, impulse, and area of dulness. It was thought that the transverse and upper part of the descending aorta were the seat of an aneurism. Aortic and mitral valvulitis were known to exist. At the autopsy were found chronic endarteritis, large *dilatation of the pulmonary artery* (9 cm. in circumference), aortic and mitral valvulitis, chronic fibrous pericarditis, dilated and hypertrophied left auricle and ventricle, but no hypertrophy or dilatation of the right heart.

The pressure-symptoms always point to some form of intrathoracic disease as the cause of this group of symptoms. Thus in cancerous disease of the lymphatic glands, or other tumors within the mediastinum, pressure-symptoms exactly simulating aneurism may be present and also the physical signs of a tumor. In tumor, however, there is usually absence of certain ætiological factors of aneurism, such as strain, syphilis, etc., and the tumor, if primary, is very common in young subjects. The tumor rarely projects externally, and still more rarely pulsates. If pulsation is present, it is not of the expansile character seen in aneurism, nor is there as decided a systolic shock when the ear is held against the chest. By the same method we observe the shock of the heart-sounds,

[1] British Medical Journal, March 19, 1892.

which are notably lessened or absent in tumors from causes other than aneurism. In deep-seated tumors with pressure symptoms the condition of the arteries, apart from aneurism, is of diagnostic importance. Accentuation of the aortic second sound, with hypertrophy of the heart, points to aneurism. The presence of tracheal tugging is also a valuable diagnostic point in its favor. In tumor, and especially in cancer, there are emaciation and the development of a cachexia, which, as is well known, is most pronounced in cancer of the œsophagus. Most intrathoracic tumors are fatal within a year of the onset of symptoms, while aneurism may last a long time. Cancer of the œsophagus, from its frequent point of election near the left bronchus, often simulates the pressure-symptoms of aneurism.

Schneele and Porter have described procedures for the recognition of aortic aneurism which they regard as important. A blind rubber bougie is passed down the œsophagus until it is opposite the arch of the aorta or the site of the suspected aneurism. It is then filled with water which may be colored, and a glass tube is attached to the outer end. In health little or no impulse and consequent variation in the height of the fluid are to be noted; in aneurism considerable variations may occur. It has also been held that œsophageal auscultation practised with a solid œsophageal sound results in a murmur being rendered more distinct, and that one ordinarily inaudible may be heard in this manner. It is not difficult to conceive that serious and even fatal results might follow these procedures.

Aneurism must be distinguished from the pulsation of the aorta which is seen in aortic regurgitation. This pulsation is usually associated with dilatation, the latter causing increased dulness, which may add further to the confusion. Exaggerated pulsation without dilatation may, as Bramwell has reported, be the cause of dulness and pulsation over the aorta. The subjects are under forty, neurotic, and usually anæmic.

It is not, as a rule, difficult to distinguish between *pulsating empyema* and aneurism. Wilson points out that aneurism bears a definite relation to the central long axis of the chest. The area of dulness of aneurism is circumscribed, and is usually the seat of murmurs or other sounds synchronous with the rhythm of the heart. The signs of pulsating empyema are usually upon the left side and at a distance from the median line. The percussion-dulness is at the base of the chest and quite extensive. Arterial murmurs are not present. The pulsation is influenced by pressure and by respiratory movements.

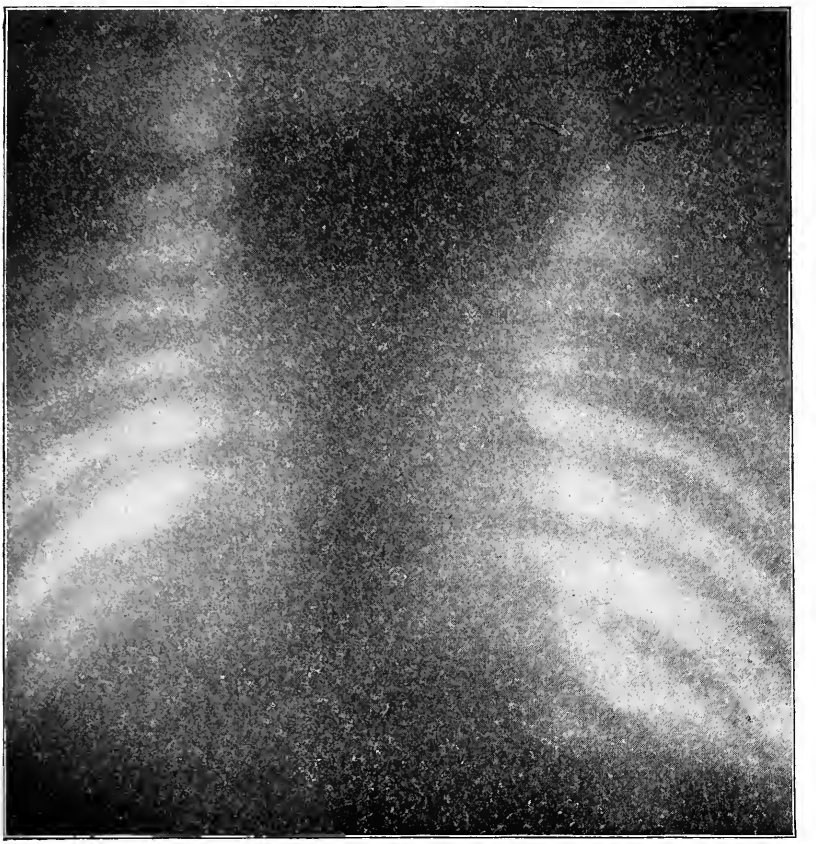
In *mediastinal cancer* we are aided by the discovery of enlargement of the glands in the axilla, neck, or elsewhere, or by a history of the growth in some other area.

Aneurism must not be confounded with *phthisis*. The diseased vessel may occlude a bronchus and cause collapse and bronchial dilatation; hemorrhage may occur; bronchorrhœa and cough always ensue. Fever is not marked, which fact, with tracheal tugging, vascular physical signs, and the absence of tubercle bacilli, points to aneurism.

X-RAY EXAMINATION. By virtue of the large amount of blood and fibrin in an aneurism, the tumor is not pervious to the *x*-rays, and in con-

sequence is readily seen by fluoroscopic examination. Williams and others have been very successful in recognizing an aneurism even when it

FIG. 356



X-ray appearance in aneurism. (PEPPER and LEONARD.)

could not be made out by physical signs. Such examination should be resorted to in all cases. (Fig. 356.)

DISEASES OF THE MEDIASTINUM.

Inflammation of the mediastinum may be limited to the glands or the connective tissue, or may involve both. Moderate inflammation of the glands, lymphadenitis, occurs in bronchitis and pneumonia, particularly if bronchitis is of specific origin, as in measles or influenza. It is said that such inflammation is of common occurrence in whooping-cough, and may be the exciting cause of the paroxysms. DeMussy and Guitéras have found physical signs of enlargement, characterized by dulness in the upper part of the interscapular region, in cases of this disease and of influenza. Other authorities, as Osler, dispute the possibility of this occur-

rence, or at least of its recognition by physical signs. Tuberculous inflammation of the lymphatic glands of the mediastinum may, however, give rise to local physical signs. Abscess of the glands can not be distinguished during life.

Tumors of the Mediastinum.

Cancer and sarcoma are the most frequent forms of tumor in this locality. Hare found the proportion in 520 cases to be as follows: 134 of cancer, 98 of sarcoma, 21 of lymphoma, 7 of fibroma, 11 of dermoid cyst, 8 of hydatid cyst, and the remainder of lipoma, gumma, and enchondroma. With the application of more correct histological methods we now know that sarcoma is more common than carcinoma. The tumor is most frequently found in the anterior mediastinum when one region alone is affected. The disease may be either primary or secondary. In sarcoma it is usually primary. Males are chiefly affected, and most often between thirty and forty. The tumor may originate in the thymus gland, the lymphatic glands, the pleura, or the œsophagus; the most frequently in the thymus gland.

Symptoms. The symptoms of mediastinal tumor are chiefly due to pressure. *Dyspnœa* is early and constant, and may be laryngeal, or tracheal from pressure on that tube. In some instances encroachment upon the heart or the vessels causes dyspnœa. Again, the dyspnœa may be due to a pleural effusion which accompanies the growths. Cough of a peculiar character occurs. It is laryngeal, and of a dry, brassy quality. Aphonia may arise from pressure upon the recurrent laryngeal nerves. (See Diseases of the Larynx.) If the bloodvessels are pressed upon, symptoms of obstruction occur, depending upon the vessel occluded. Œdema of the upper extremities may occur. If the œsophagus is pressed upon, there is difficulty in deglutition. In some instances the sympathetic nerve is pressed upon, causing hyperæmias and pupillary changes.

Physical Signs. The physical signs (Plate XXXVII., Fig. 2) are those of a tumor in the anterior portion of the chest, frequently in the præcordial area, which may or may not pulsate; dislocation of the heart, not limited to any position; great dulness and resistance; frequently conduction of lung-sounds and heart-sounds to some distance; at times a systolic murmur; increased size and pulsation of the veins; and physical signs from pressure. (See Aneurism.) It must be remembered that *pain* is more common in aneurism; *fever* and *emaciation* in mediastinal growths.

Tumors of the anterior mediastinum present, in front, the physical signs of a prominence more or less marked, often including projection of the sternum; an irregular area of dulness; rarely transmitted pulsation; more frequently transmitted heart-sounds and lung-sounds. It is the form in which phenomena from pressure upon the veins are most marked. Symptoms from arterial pressure (difference in pulse), pressure on the vagus and sympathetic are less frequent. *Dyspnœa* may occur.

Tumors of the middle and posterior mediastinum are characterized by

pressure upon the bronchi and structures adjacent thereto; hence we have symptoms from pressure upon the œsophagus, aorta, and the nerves. *Dyspnœa* and *cough* are the most pronounced symptoms, while phenomena from pressure on the vagus, cardiac palpitation, vomiting, etc., are not uncommon. *Emaciation* and cachexia are more marked than in tumors in other regions. Pepper and Stengel consider that fever attends growths in this region with greater frequency. (For differential diagnosis from aneurism, see Aneurism, p. 963.)

Tumors of pleural origin have symptoms of acute or subacute pleuritis, with or without effusion. The fluid secured by puncture is usually bloody, rarely chylous, and may contain suspicious vacuolated epithelial cells. A mass may be suspected if there is great resistance to the trocar. If the tumor ulcerate into the lung, the sputa may contain characteristic groups of cells, while hemorrhagic oozing may be suspicious.

CHAPTER VIII.

DISEASES OF THE MOUTH, INCLUDING THE TONGUE THE FAUCES, PHARYNX AND ŒSOPHAGUS.

THE MOUTH.

THE mouth is affected by comparatively few diseases, and most of these are the result of infection or of trauma, or, rarely, are trophoneurotic. The cavity is a good breeding-place for all forms of organisms, and were it not for the secretions and constant cleansing of the mouth by the passage of food and its physiological labors, diseases would be very common. Indeed, it is possible that such diseases do not occur at all unless there is such perversion of the normal secretion as destroys its antiseptic or antimicrobic qualities. We know but little specifically concerning the changes in the secretions. Clinically, we do know, however, that in conditions of poor nutrition, in wasting diseases generally, and probably in connection with the rheumatic diathesis, there is such change in the secretions as permits pathogenic micro-organisms to exercise their influence upon the mucous membrane. The result of their action is seen in various forms of *inflammation.*

Diagnosis. All methods of diagnosis are employed In the *historical diagnosis,* hygienic conditions, food and methods of feeding should be investigated, and the former occurrence of infections are inquired for; these are factors that aid in so much of diagnosis as pertains to ætiology and therapeutics. For the *subjective diagnosis* but few symptoms present themselves, practically only the symptoms of inflammation or of diminished secretion. The *objective diagnosis* is detailed in full in this chapter. By *laboratory diagnosis* we learn the nature of the infections, notably diphtheria, and the condition of the saliva. (See Section VI.) It is readily seen that greater reliance must be placed on objective and laboratory than upon any other method of diagnosis.

Stomatitis.

The *inflammation* is not limited to the mouth, but extends to structures within the mouth, as the gums, and may invade the tongue. It is recognized by the subjective and objective signs common to such inflammations. There is pain, and hence the child (for it usually occurs in children) refuses to nurse or take the bottle, or cries when food is given. The pain is accompanied by *fetor* of the breath. This occurs in all forms of stomatitis.

The usual signs of inflammation are rarely general, being, as a rule, localized to small areas which may rapidly become ulcerated. The mucous membrane is red and hot in the *catarrhal* form; the color

extends to the gums, lips, and tongue. The follicles are enlarged. The tongue becomes red and smooth, or may be covered with a white coating, through which the prominent red fungiform papillæ project. There is increased secretion, which dribbles from the mouth, or is constantly discharged by older patients. The ever red mucous membrane is swollen; the breath is hot and fetid.

A recurring form is seen in gouty subjects. Pain is not marked, but the heat, redness, and burning are severe. Superficial glossitis and salivation are present. The saliva is highly acid, and causes a dermatitis on the chin. Other mucous membranes, as the vaginal, are involved at the same time. An acid mucoid discharge sets up painful irritation at the vaginal outlet.

Aphthous Stomatitis. The areas of intense inflammation are sometimes followed by ulceration. In *aphthous stomatitis* small yellowish-white spots appear, at first discrete, but soon dotted over the mucous membrane inside of the cheeks, in the roof of the mouth, along the sides of the gums, and on the tongue. They subsequently break down into shallow ulcers with raised red margins.

Aphthous ulceration is seen in *foot-and-mouth disease.* It is characterized by greater swelling, with softening and ulceration of the soft parts, than in other forms of stomatitis. There is a history of infection, profuse diarrhœa, followed by constipation, and considerable physical depression.

In young infants that are not well cared for, one not infrequently sees *shallow ulcers*, called "Bednar's aphthæ," on the soft palate or the posterior part of the hard palate. These are due to rough cleansing of the mouth, to the use of bad rubber nipples, or, perhaps, to the pressure of the tongue in nursing and infection of areas thus abraded. They cause no general symptoms, but may interfere with nursing.

Ulcerative Stomatitis. The disease occurs in ill-nourished subjects, and is often intercurrent with exhaustive disease, as chronic diarrhœa. It may be seen in epidemic forms in camps and in penal and other institutions, on account of unsanitary conditions. In ulcerative stomatitis the inflammation is chiefly confined to the gums. They are swollen, red, and covered with ulcers. The gums in which teeth remain are affected, and the ulcers are usually at the gingival border. Gums without teeth are not affected. The ulcers are covered with yellowish material. The flow of saliva is much increased, and it is acid in reaction. The submaxillary glands are enlarged. The fetor of the breath is great.

Parasitic Stomatitis—Thrush. The affection is characterized by raised white patches looking like small curds of milk. The patches vary in size, and on the tongue may cover an area as large as a three-cent piece. (See Thrush.) The white patches are distinguished from milk-curds because they can not be removed with the napkin or brush. The parasite has been called *Oïdium albicans* (see Laboratory Diagnosis, Fig. 209); but Forchheimer prefers to group it under the saccharomyces.

Stomatitis Materna. Painful ulcers occur in the mucous membrane of the lips and cheeks of nursing women. They are solitary and interfere with mastication.

Gangrenous Stomatitis. The affection appears as a gangrenous in-

flammation of the gums, mucous membrane, and deeper tissues of the cheek. At first a small, dark-red, hard spot is seen, which increases in size and becomes of a purplish color. The cheek rapidly becomes swollen, tense, and brawny. On the surface of the more indurated portions a bleb forms which soon breaks with rapid ulceration. The ulcer is dark and gangrenous and soon perforates the cheek. It extends to the jaw and is followed by necrosis of that bone. The characteristic odor of gangrene attends the process. Gangrenous stomatitis is always secondary to depraved, depressed, or debilitated states of the system. Cases may occur simultaneously in asylums for children in which the hygienic conditions are bad and the food-supply poor.

Mercurial Stomatitis. Mercurial stomatitis, or ptyalism, particularly affects the gums. It also involves the salivary glands. The inflammation is caused by mercury. It may occur from the medicinal use of the drug, particularly in persons who are unduly susceptible, or are not particular in regard to mouth-cleansing. The inflammation is painful and attended by profuse discharge of saliva, hence the name, *salivation*. The tongue is swollen, marked on the sides by the teeth, and may be protruded with difficulty on account of its size. It is tender to the touch, and covered with a heavy, creamy coating. The gums are swollen, red, sore, and bleed on the slightest touch. Ulcers along the border occur, may become diffused, and in some instances extend to the jaw. The teeth become loosened. The fetor of the breath is heavy, offensive, and of a metallic character, The inflammation is usually preceded by a metallic taste in the mouth, and the patient notices pain on mastication, which increases in severity as the inflammation develops. In mild cases it is limited to the gums, in others the tongue and salivary glands and the mucous membrane of the mouth are affected.

Leprosy of the Mouth.

Leprosy frequently invades the mouth. The nodular and ulcerative lesions are seen. It is always associated with the characteristic lesions of the skin. Scrapings or sections would show the characteristic microorganism.

Glanders.

Glanders may invade the mouth from the nasopharyngeal space.

Actinomycosis.

Actinomycosis results from the entrance of the ray-fungus through carious teeth or an abraded mucous membrane. Often there is first disease of the alveolus, as pyorrhœa, or a periosteal abscess; then the jaw is involved. It may be preceded by a general stomatitis.

Ulcers of the Mouth.

Solitary *ulcers* are seen in *herpes*, secondary to gastric or uterine disturbances, and syphilis. The herpetic ulcers are of frequent occurrence

at the menstrual period or during the course of lactation. The tendency to their formation is often hereditary. I have seen them occur at the menstrual period or in pregnancy in the women of three generations.

In the second stage of syphilis *mucous patches* are seen as bright-red, symmetrical, oval or crescentic patches or erosions, occurring on the mucous membrane, sometimes on the tongue and fauces. They are generally covered with a scanty grayish-white secretion, and are not usually painful.

Sublingual Ulcer. This form occurs on the frænum of the tongue. It is seen in whooping-cough, and is due to the rubbing of the tongue against the teeth in the act of coughing.

Scleroderma.

This rare trophoneurosis occasionally invades the mouth. It is characterized by a submucous infiltration of cartilaginous hardness, the surface of which is denuded of epithelium or covered with crusts. The invasion is through the nostrils or the nasopharynx. Later the infiltration changes to a yellowish-red or a tendinous-like scar.

THE TONGUE.

Inflammation of the Tongue.

Acute glossitis is a rare affection, more common in adults than in children, and more frequent in men than in women. It occurs more frequently in the summer. The onset is rapid. After a short period of tenderness on mastication the movements of the tongue are stiff and painful, or there are pains in the muscles of the neck and submaxillary region. In a few hours the tongue swells; it rapidly enlarges, and at the end of fifteen to twenty hours is three times its natural size, protrudes from the mouth, is indented by the teeth, and is almost immovable, feeling heavy, painful, and tender. It is coated with a thick fur on the dorsum. Salivation accompanies these symptoms, speech is impossible, dysphagia extreme, and dyspnœa not unusual. The glands underneath the jaw are swollen. The temperature rises to 101° F., rarely above it, even if the case is severe.

Death may occur in a few hours from suffocation, or after a longer interval from diffuse suppuration, gangrene, exhausting septic fever, or pneumonia. Gangrene is more frequent than spontaneous resolution. If resolution is to be established, the swelling begins to subside in three or four days. Small ulcers form on the surface of the tongue, and by the end of a week its normal appearance is regained. The fever and distressing symptoms subside with the local swelling. It is said to be due to colds, to bites and stings of animals, to mercury, and to corrosive and acrid substances. It may occur in fevers. The diagnosis is easy. It must be distinguished from the acute œdematous swelling due to salivary calculus or affections of the floor of the mouth.

Acute Ranula. Acute ranula sometimes causes considerable swelling of the tongue, simulating acute glossitis.

Hemiglossitis. Hemiglossitis sometimes occurs. The local symptoms are less in degree. I saw a case in which the inflammation was limited to half the side of the tongue on the posterior surface. It went on to suppuration, but was not attended by serious symptoms, except discomfort in eating. It was preceded by a definite nodule in the substance of the inflamed part.

Glossitis from mercurial poisoning has been described in connection with stomatitis.

Chronic Superficial Inflammation. The surface is smooth and deprived of papillæ over the affected area, which is redder than natural. The margin of the raw patch is sharply defined, but the area has no depth. The epidermis alone is removed. When associated with dyspepsia, it covers a considerable area of the surface of the tongue. The tongue may be deprived of papillæ on the anterior part of the dorsum while the fungiform papillæ remain. It is enlarged and the borders marked by the teeth. The surface looks glossy. The tongue feels stiff and uncomfortable. Movement is irksome, irritating foods are painful; spirits and tobacco cause distress. Indiscretions in diet and slight traumatism quickly produce fresh inflammation. One observer, Hack, has described a form of hereditary glossitis peculiar to women. He observed a row of long, oval areas, caused by previous inflammation. They commenced in early childhood. The tongue was smooth over remaining large areas, with red excoriations here and there. There was no syphilis.

Another form of glossitis is that seen in pernicious anæmia. The papillæ are large, red, and very tender, so that eating, and especially the use of acids, are very painful. The red points contrast with the pallid tongue.

Sequelæ of Glossitis. *Indentations* occur when the tongue is swollen, as in mercurial and other forms of glossitis. The borders of the tongue are indented by the pressure of the teeth; but in states of debility a flabby tongue with indented borders is often seen. Sometimes the swelling is so great that the pressure of the teeth causes ulceration.

Furrows, or *grooves and wrinkles*, are seen on the dorsal aspect of the tongue. They are not necessarily tokens of disease; in many healthy persons they are of constant occurrence. Furrows vary from a few lines to an inch or more in length. They may be longitudinal, transverse, or irregular. The median furrow is liable to become ulcerated on slight provocation. The edges of the fissures are smooth and without papillæ or fur. Other furrows are directed horizontally and vary in depth. They may be curved and forked. They are more frequent in older persons, especially if the tongue is too large to lie within the circle of the teeth. They are an evidence of past inflammation, or rarely of hypertrophy. They resemble the median furrows as regards smoothness and absence of fur.

Inflammatory furrows occur in chronic superficial inflammation, but more commonly after chronic inflammation with enlargement. The furrows are sometimes so abundant that the surface of the tongue looks like the eyelid. The raised areas become sore, irritated by a foreign body (food) or a tooth. They are an indirect result of inflammation.

True inflammatory furrows, described by Wunderlich, occur. *Dissecting glossitis* is only a more aggravated form of superficial glossitis, or may be due to syphilis. *Fissures* and *clefts* are frequently caused by the rubbing and deep indentation of a rough and jagged tooth. The area around the fissure is inflamed, its base indurated, and the sides and bottom ulcerated. It is recognized by its relation with the offending tooth. It may be mistaken for syphilis.

Syphilis of the Tongue.

Fissures are found on the borders of the tongue; in *secondary syphilis* they are almost certain to occur if the teeth irritate the border. They may be due to the ulceration of a mucous tubercle which is developed upon the border of the tongue. The ulcer is stellate, and gradually deepens until it becomes a foul fissure. Two processes cause the ulceration—the specific infection and the irritation of the teeth.

Syphilitic *ulcers* are not so angry as non-syphilitic sores and fissures, which may occur in poor health. They may be sensitive, however, on account of their location on the tongue. The absence of active inflammation, the large number of sores and fissures, and the association with other lesions of the disease upon the tongue, cheeks, and lips, point to their syphilitic origin. Tertiary syphilitic ulcers are more pronounced and deeper than other forms. They may be as long as two or three inches; they are sinuous and branched.

Gummata may occur on the tongue at the same time. The gummata may be circumscribed or linear, and may break down and ulcerate.

Sclerosis of the tongue, as described by Fournier, follows the healing of these ulcers. It is curious to note that the lymphatic glands are seldom enlarged in association with syphilitic fissures.

Diagnosis. The fissures must be distinguished from *carcinoma* and *tuberculosis*. In carcinoma there is a distinct tumor, which may become fissured. Tuberculous ulceration is a sign of the presence of tubercle in other organs. The tuberculous fissures are small, at first single; tubercle, however, rarely begins as a fissure, but as an ulcer on the tip or borders of the tongue. The ulcers are stellate or irregularly branched; they are shallow at first, and deepen later, but do not widen in a corresponding manner. The lymphatic glands are always involved. (See Tuberculous Ulcer.)

Atrophy of the Tongue.

Atrophy of the tongue is very unusual. Hemiatrophy may occur as the effect of central or peripheral causes, as softening, hemorrhage, or tumors in the region of the hypoglossal nucleus. Other centres near the nucleus are affected, hence other forms of paralysis are seen, due to the lesions of the medulla. These are seen in progressive muscular atrophy and bulbar paralysis, and in hemiplegia. It is not difficult to recognize atrophy on inspection. The functions of the tongue are not affected.

Hypertrophy of the Tongue.

Enlargement of the tongue, or *macroglossia*, is generally congenital, but may occur late in life. The tongue enlarges, and presents pressure-symptoms due to such enlargement. Hypertrophy of the tongue is sometimes seen in idiots and cretins. The hypertrophy is more frequently the result of lymphatic obstruction, on account of which there is lymphstasis. The diagnosis is easy. Inflammatory hypertrophy occurs in stomatitis, and syphilitic hypertrophy occurs with gummata.

Angina Ludovici.

Angina Ludovici is characterized by slight inflammatory congestion of the throat out of proportion to the symptoms of the inflammation in the external structures. Woodeny induration of the connective tissue, which will not pit on pressure; spreading of this induration, which is circumscribed, so that it is bound sharply by unaffected cellular tissue, are characteristic. The induration may extend from the rami of the jaws to the cheek. There is a hard swelling in the *tongue* and along the lower jaw, causing thickening of the floor of the mouth. This is observed by palpating with the finger in the mouth. The glands are not affected. For a long time the nature of this affection was not known. It is now believed to be due to actinomyces.[1]

THE FAUCES AND PHARYNX.

The passage-way between the mouth and the respiratory passages is lined with mucous membrane, which is subject to the same diseases as mucous membranes in general. The symptoms thereof are similar to the symptoms of mucous membrane inflammation elsewhere. The large muscles of the pharynx which aid in deglutition are subject to affections which belong to muscular tissue generally, hence *rheumatic inflammation* and loss of power of muscle, or *paralysis*, occurs.

Paralysis of the pharynx has not the same practical importance in the diagnosis of central lesions as paralysis of other structures, such as parts of the larynx. This is due to the fact that the nerve-supply of the pharynx is derived from a nerve (glossopharyngeal) which supplies other structures, paralysis of which is more evident than pharyngeal paralysis, more readily ascertained, and which causes more pronounced symptoms. (See Cranial Nerves.) From its exposed situation the pharynx is particularly liable to *infection* from micro-organisms. The infection may extend from the mouth, or from the nares above, or the micro-organisms may affect it primarily.

The fauces and pharynx may be the seat of morbid processes which occur secondarily to diseases in other portions of the body with a moderate degree of frequency. Inflammations of the mucous membrane of the pharynx are of rheumatic or gouty origin in a large number of cases.

[1] See Parker, Lancet, 1879; and Anderson, Transactions of the Medico-Chirurgical Society, 1891.

Indeed, gouty inflammation of the pharynx seems to be more common than gouty inflammations of the mucous membranes in other situations. The large majority of subacute or chronic pharyngeal inflammations are secondary to dyspepsia. They also occur from extension of the disease from cavities related to the pharynx.

Affections of the tonsils are usually more common in rheumatic states, and bear some relationship to the rheumatic diathesis. Inflammation of the tonsils may follow acute rheumatism or may alternate with it. A patient who is predisposed to rheumatism may at one season have tonsillar inflammation, at another rheumatism. The writer has seen tonsillitis immediately followed by rheumatism, and then the latter replaced by the former.

The not infrequent occurrence of endocarditis with tonsillitis has been used as evidence of the rheumatic nature of the tonsillar disease, and it is very probable that the infection which causes rheumatism frequently enters through the tonsils. Packard considers that the tonsils may reduce the virulence of infections entering through them, and that this attenuation results in the production of rheumatic symptoms rather than of ordinary septicæmia.

There is also good evidence that tuberculosis sometimes has its primary seat in the tonsils, and may be disseminated from them, when it is especially likely to involve the cervical glands secondarily. In these primary cases it is a food infection sometimes. Secondary tuberculosis of the tonsils is apparently not uncommon.

Apart from what has just been said, diseases of the pharynx bear little, if any, diagnostic relationship to disease elsewhere. While there may be cyanosis of the mucous membrane, or tuberculous ulceration, or other changes which we have noted, the signs of the primary disease are so much more marked that we need not rely upon the appearance of the pharynx or symptoms of pharyngeal disease for diagnostic purposes. The only general affection which may be diagnosticated from the appearance of the pharynx alone is measles. In obscure cases of sudden fever, with nasal catarrh, the appearance of the eruption in the situation just indicated may lead to the recognition of measles when the external eruption is not apparent. For the purposes of the therapeutist it should be borne in mind that symptoms referable to the pharynx are frequently due to disease in the nares, particularly in that portion of the pharynx which is not open to direct inspection—the nasopharynx.

The *general symptoms* of pharyngeal disease are not marked, except in diphtheria, in erysipelas, in retropharyngeal abscess, and in affections of the tonsils. In the latter the general symptoms appear to be out of proportion to the local process. The high fever, the intense headache and backache, and rapid pulse, seem to point to a process which in extent and severity should far surpass that which occurs in the tonsils.

As the structure is a passage-way or channel, affections of the pharynx are liable to obstruct it, causing symptoms of *occlusion*. As it is a channel for the passage of air, obstruction in the pharynx will lead to *dyspnœa*. (See Chapter IX.) In addition to its function as a simple channel, the pharynx is concerned in deglutition. When, therefore, there is obstruction

of the pharynx, *deglutition* is made difficult, or may even become impossible.

Attention can not be too strongly directed to the investigation of the *nasopharynx* in children who are poorly developed physically and mentally. This combination of symptoms is often due to adenoid vegetations of the nasopharynx.

Diagnosis. The diagnosis of diseases of the pharynx is based upon the facts gleaned by all methods of diagnosis.

Historical diagnosis confirms other methods in the diagnosis of pharyngitis and tonsillitis (rheumatism), and the infections.

In the *subjective diagnosis* we consider cough (Chapter X.), pain (Chapter VIII.), dyspnœa (Chapter IX.), and dysphagia (Chapter XIII.).

The *objective* and *physical* diagnosis are considered in full, for by these methods and a consideration of the clinical course of the disease the diagnosis is most completely established.

By *laboratory diagnosis* we establish the presence or absence of infections, as diphtheria, tuberculosis, and leptothrix.

THE TONSILS.

Acute Tonsillitis.

Acute inflammation of the tonsils may affect the follicles, to which form the term *follicular tonsillitis* is applied, or it may be limited to the mucous membrane, when it is known as *catarrhal* or *erythematous tonsillitis.* If with the catarrhal inflammations vesicles appear on the surface of the mucous membrane, the term *herpetic tonsillitis* is used. When the inflammation extends to the stroma of the glands it goes on to suppuration. It is characteristic of all forms of acute tonsillitis to recur frequently in the same subject. The relationship to rheumatism, which applies to both the acute and the suppurative forms, has been spoken of. The various forms of tonsillitis occur at any period of life, although it is least common under ten years of age; the suppurative form occurs most frequently in adolescence. Tonsillitis occurs in both sexes. It may follow exposure to wet and cold, although patients who are subject to the attacks bear exposure well unless they are at the same time unduly fatigued. The *follicular* form of tonsillitis is apparently associated with faulty drainage or other unhygienic conditions, and hence it is possible that noxious emanations act as an exciting cause. Several persons of the same family may be affected at one time, so that it is often difficult to distinguish the cases from diphtheria. The disease, however, is usually not contagious. Persons brought in contact with the family, but who do not reside in the same house, escape the disease; this applies as well to children, who would, if the cases were diphtheritic, be most liable to become infected; actual epidemics of tonsillitis, due usually to the streptococcus, occur in rare instances, however, and in such cases the diagnosis must rest upon bacteriological examination. The disease occurs more commonly in the spring than in any other season of the year, more especially in cold and wet seasons.

Symptoms.—In *follicular tonsillitis,* with or without a rigor, but always

with chilly sensations, the temperature rises rapidly to a great height. The subjective sensation of fever is quickly noticeable to the patient, and is generally more pronounced than in other affections. With the chill and during the rise of temperature there are some frontal headache and severe pain in the back and limbs. The pain in the back is most excruciating. In a short time the patient complains of pain in the throat. Swallowing is difficult, and there is a sense of fulness. The throat is dry and burning. On examination the tonsils are found to be swollen, and a yellowish-white exudate is seen in the crypts. In twenty-four hours the points may coalesce to form a patch. The glands expand slightly, and may extend only slightly beyond the arches, or, in younger subjects, one-quarter of the way into the lumen of the fauces. Sometimes one gland is affected before the other. The difficulty in deglutition increases and the voice becomes nasal. There is usually some enlargement of the cervical glands. The general symptoms continue for forty-eight hours, the temperature remains at 105° F., and the pulse is very rapid. After the first twenty-four hours the pain in the back lessens. The tongue is coated and the breath heavy. The urine is loaded with urates. At the end of the fifth day the fever, which subsides gradually, has disappeared. The local symptoms, however, may remain longer—that is, the tonsils are still enlarged and the exudate disappears slowly. Sometimes the prostration and general symptoms are so severe that after the fever has subsided convalescence may be protracted.

Albuminuria, due in all probability to the fever, frequently occurs; in some cases, undoubtedly, acute nephritis attends the attack and retards the convalescence. In a case under the writer's care the patient first had acute rheumatism; this was replaced by a severe attack of tonsillitis, during which albumin, blood, and granular casts were found in the urine. The swelling of the tonsils subsided in due time, but the nephritis continued for a long period, finally ending, however, in complete recovery.

In *herpetic tonsillitis* the severe pain and intense general symptoms are out of proportion to the local lesion.

In *suppurative tonsillitis* the constitutional disturbance is also very great. The temperature rises high, 104° to 105° F., and the pulse is very rapid, varying from 110 to 130 in the adult. The inflammation usually begins in one tonsil, and the other may be involved later. The tonsils at first are enlarged and firm and red. There is swelling of the surrounding tissues. In twenty-four hours deglutition becomes almost impossible, and there is salivation. At the end of forty-eight hours the patient presents a striking appearance. The glands of the neck are enlarged, the patient is unable to open his mouth, the voice is nasal or almost suppressed; there is dribbling of saliva from the mouth. The face may have a dusky hue in spite of the capillary congestion due to the fever. There is constant desire to discharge saliva and accumulated secretions from the back part of the mouth. The patient can not lie down. The pain is extreme, and is aggravated by swallowing. It is sometimes of a throbbing character, and often shoots to the ears—indeed, earache may be the chief complaint. The patient does not take food,

and exhaustion soon ensues. During the twenty-four hours before rupture takes place the previously reddened face becomes blanched from exhaustion. The fever is continuous during this time, with great rapidity of the pulse. The patient may be delirious. Sometimes the delirium is marked and the patient resists efforts to keep him in bed.

The suffering is out of proportion to the severity of the case. About the fourth or fifth day suppuration ceases, and if the finger can be inserted into the mouth between the almost closed teeth, fluctuation is detected. In cases in which the mouth is opened a little more freely, in addition to the swelling of the tonsils below the arches, marked swelling and projection forward of the half-arches may be seen. The fluctuation may be detected through the anterior fold of the palate, and, if lancing is to be performed, the pus can only be reached through this structure. In short, a peritonsillitis takes place. After spontaneous rupture, which usually occurs into the mouth, instant relief is experienced. Rupture may take place into the pharynx and cause suffocation from entrance of pus into the larynx. In rare cases it has opened into the carotid artery, causing instant death from hemorrhage.

Diagnosis. The diagnostic features of acute tonsillitis are the sudden high fever, severe backache and headache, pain in the throat, and albuminuria. The characteristic appearance of the face, the salivation and pain, with suppressed voice and difficult deglutition, should distinguish it from trismus or *tetanus*. In both, the jaws are closed. It must not be confounded with *smallpox*, which it resembles during the first twenty-four hours.

Cases of follicular tonsillitis are frequently mistaken for *diphtheria*. The follicular inflammation in tonsillitis is limited to the gland, on which patches of a yellowish-gray color, easily removed without leaving bleeding surfaces, are seen. In diphtheria the membrane is of an ashy-gray color, not in points or small patches, or separated by red tonsillar tissue; it extends to the pillars of the fauces, and may appear on the uvula. There are, nevertheless, many cases which are doubtful, and in which a bacteriological diagnosis must be resorted to. (See Bacteriological Examination.) A history of exposure sometimes helps to arrive at a conclusion. The cases that particularly increase our anxiety are those of adults who are subject to attacks of follicular tonsillitis. In the grave and extensive forms of diphtheria with asthenic symptoms (septicæmia) the diagnosis is not difficult.

Enlargement of the Tonsils—Chronic Tonsillitis.

The tonsils may be enlarged on account of repeated attacks of acute inflammation or from chronic inflammation. They do not appear to cause serious symptoms unless associated with adenoid vegetations in the nasopharynx. They may interfere with hearing, however, and with breathing, and cause snoring at night. Fetor of the breath may be noted, particularly if the secretion lodges in the crypts. The latter may be recognized by its characteristic yellowish color and by its odor on removal. The enlarged tonsils are irregular in contour.

Foreign Bodies in the Tonsils.

Foreign bodies in the tonsils are not of common occurrence. They give rise to local symptoms, as the sensation of the presence of a mass causing repeated efforts at swallowing. If calculi are present, the patient may complain of a rough sensation. Calculi follow frequent attacks of quinsy. *Hydatids* are sometimes located in the tonsils.

Adenoid Vegetations of the Nasopharynx.

Adenoid vegetations cause more or less obstruction in the nasopharynx. The symptoms may be classed as primary and secondary. The former are local, and due to the foreign substance *per se;* the latter are local and general. The former are catarrhal; the latter the result of stenosis.

Local Symptoms. In a large number of cases there is *discharge* from the nose. This may be mucopurulent or be associated with crusts. If the discharge is not constant, the child is subject to coryza, with its customary discharge, on the slightest provocation. Even without the chronic purulent nasal discharge, mucus and blood may be passed at night and be found on the pillow in the morning.

The *hearing* is frequently impaired. There may be simply dulness of hearing, or it may amount to marked deafness, either because of pressure of the adenoid vegetations or extension of secondary inflammation to the Eustachian tubes. The senses of *taste* and *smell* are often much impaired. There is increase in the secretion of pharyngeal mucus, which in older persons causes difficult expectoration.

Rhinoscopic Examination. The roof of the pharynx is covered with rounded or villous projections, often concealing the posterior nares. Rarely the villi may be seen projecting below the soft palate. In children the examination is difficult, and hence digital exploration must be used under an anæsthetic. The finger readily detects the masses, which sometimes are soft, at other times tough and of fibrous or cartilaginous consistency.

FIG. 357.

Appearance in adenoid disease. (DAWSON-WILLIAMS.)

The Appearance. The *expression of the face* is characteristic. It is dull and stupid, and may be drawn. (Figs. 357, 358, 359.) The *mouth*

is kept open in breathing. The lips are thickened, dry, and may be cracked. The palatal arch is high and narrowed.

The *nostrils* are flattened laterally. Rarely they may be depressed. In one instance, which the writer saw with Harrison Allen, the exterior of the nose suggested inherited syphilis, especially because of our knowledge of the possible presence of the disease. There were no other evidences of hereditary syphilis in the child or in any member of the family.

The *voice* is thick and muffled, becoming indistinct upon the occurrence of slight cold.

The Chest. While there is a general lack of physical development, the appearance of the chest is most striking; the cases have been fre-

FIG. 358. FIG. 359.

Types of the "adenoid" facies. (POSEY and WRIGHT.)

quently mistaken for rickets. In this country adenoid vegetations are a common cause of chest deformity, whereas in England and on the continent rickets is the most frequent cause. The ribs are prominent in front, the sternum is angulated forward at the manubrio-gladiolar junction and grooved at the gladiolar-xiphoid junction. A saucer-shaped depression is found at the lower costal cartilages. The ribs behind are closely compressed, so that the intercostal spaces at the lower part of the chest are obliterated. The chicken-breast appearance is most striking, with the depression in the lower portions of the chest. During inspiration the diaphragm may be drawn inward in the middle and lateral thoracic region.

In addition to the "chicken-" or "pigeon-breast" the more advanced deformity known as the "funnel-breast" or *Trichterbrust* is seen. In children who suffer from asthma and bronchitis the chest becomes emphysematous.

Mental and Nervous Symptoms. Headache, listlessness, and indisposition for mental exertion are marked. The patients are usually backward in their studies and are unable to fix their attention for any length

of time upon any subject. The child is forgetful and can not study without effort. *Aprosexia* is the term applied to this condition.

Choreiform *spasm* of the face occurs in connection with it. *Enuresis* is a frequent associate symptom. The child is subject to frequent attacks of *indigestion*. I have seen the following occur in many cases: Prior to operation the child had an abnormally poor appetite and was subject to frequent attacks of indigestion, characterized by vomiting, with fever. After the operation the appetite improved and continued good, and the indigestion ceased entirely. The cases had been under observation for a number of years before and after the operation. The indigestion seems to have been due to the fact that, owing to the obstruction, the child would have to eat rapidly in order to keep the lumen of the mouth free for breathing purposes. The rapid eating, of course, prevented proper mouth digestion, and hence the occurrence of gastric catarrh.

Symptoms from Embarrassed Respiration. In addition to mouth-breathing, the patient snores at night, and sleep is always disturbed. The respirations are irregular, with a pause between, followed by noisy inspirations. The difficulty of breathing is the cause of restlessness, and the child will often wake up in the night with dyspnœa. *Night-restlessness*, with *dyspnœa* and irregular respirations should therefore suggest obstruction in the nasopharynx.

Diagnosis. Diagnosis is based upon the facies, which is very characteristic, and the physical examination. In children digital examination is necessary. The finger can readily detect small flat bodies or grape-like masses in the nasopharynx.

The student can not become too familiar with the symptoms and signs of adenoid disease of the nasopharynx. There is no doubt that in large cities this affection is of more common occurrence and more disastrous in its results than any other that we have to deal with in children. It may be said that in children in poor health, anæmic, with impaired digestion, and lack of muscular and physical development, if the causes are not due to impure air and improper diet, or to improper sanitation generally, it is almost certain that there is disease of the nasopharynx. The writer has seen a large number of cases in recent years in his practice, and has had the satisfaction of seeing the entire picture of the child change after proper operations. It may be said in passing that this change does not take place at once, but after three to twelve months the child will be fully restored in physique, if during that time attention is paid to proper exercise and the development of the chest. Notwithstanding all this, however, the natural shape of the chest and appearance of the face are only resumed gradually.

Inflammations of the Pharynx.

Acute Pharyngitis. Inflammation of the pharynx, or sore throat, follows cold or exposure, particularly after the patients have been physically depressed. The acute inflammation may be associated with rheumatism or gout, and often involves the tonsils as well as the pharynx. The symptoms are pain on swallowing, with dryness and a constant

desire to hawk and cough, on account of the tickling sensation. There may be slight laryngitis and inflammation of the Eustachian tubes, with deafness. Stiffness of the neck and enlargement of the cervical glands attend the local inflammation. The general symptoms are not marked. The attack is ushered in by chilliness and slight fever. On examination the mucous membrane is seen to be congested, dry, and glistening, and covered in spots with sticky secretions. The uvula may be swollen considerably. When the submucous tissues are involved, the parts are more swollen and there is great dyspnœa. The dysphagia is more marked, although the pain is not any greater. The fever is higher. The larynx is always involved, causing aphonia.

Phlegmonous Inflammation. A diffused inflammation of this character occurs. The writer saw one case with dyspnœa, nervous symptoms, and high temperature, simulating severe pneumonia. Pneumonia was thought to be present because there were congestion and œdema of the lungs. This occurred during the prevalence of a recent epidemic of influenza. The disease began in the pharynx; the tissues were swollen and infiltrated. The early symptoms were pharyngeal. The dysphagia was extreme, and there was an abundant mucopurulent expectoration which did not contain pneumococci. Death took place on the ninth day from exhaustion. The autopsy showed a high degree of congestion of the lungs, and phlegmonous inflammation of the pharynx, larynx, and trachea. While, therefore, the recognition of an acute phlegmonous inflammation is not difficult, it must not be forgotten that it is a grave disease, which may present such marked pulmonary and systemic symptoms as to lead to the suspicion of pneumonia.

Angina Ludovici. Angina Ludovici is an inflammation of the cellular tissue of the floor of the mouth and neck. It is probably a form of actinomycosis. The swelling is most marked below the jaw of one side. The symptoms are intense and both local and general. There are general septic symptoms from the outset. With the swelling there are œdema and board-like induration. Redness and the rapid formation of an abscess occur rarely. The throat is not affected. Death takes place from reflex suffocation or in coma. (See the Mouth.)

Rheumatic Pharyngitis. Rheumatic pharyngitis is of short duration, without unusual objective symptoms. Pain is intense, deglutition difficult. The usual concomitants of rheumatism are present. It frequently gives place to torticollis, lumbago, or rheumatism in some other situation.

Chronic Pharyngitis. Chronic pharyngitis follows acute attacks, and is a frequent accompaniment of nasal catarrh. It is common in smokers and alcoholic subjects; the use of the voice in loud tones, as by clergymen, auctioneers, etc., is also a cause. It is a frequent attendant upon indigestion, due probably to the eructations. The objective signs are relaxation of the mucous membrane, with dilatation of the veins. The membrane is covered with a thick secretion, which is dry and glistening. In the granular form the wall of the pharynx is covered with millet-seed projections and is congested. Tough mucus is seen in small areas.

Retropharyngeal Abscess. The inflammation may begin in the submucous connective tissue, and a retropharyngeal abscess forms. There are

high fever and dysphagia, with stiffness of the neck and enlarged glands. On examination a projection into the pharynx can be seen or distinctly felt on the posterior wall. The disease may be difficult of recognition in infants, in whom it is not possible to get a good view of the pharynx. On the other hand, it may be simulated by disease of the cervical vertebræ, in which there may be stiffness, difficulty in deglutition, and possibly a tumor. It must not be forgotten that retropharyngeal abscess may result from caries of the cervical vertebræ. In children the abscess is attended with dyspnœa and alteration in the voice, so that laryngeal disease may be suspected. I recall a case of retropharyngeal abscess in which the dyspnœa was so severe as to suggest croup; in fact, preparations for tracheotomy were made, when sudden rupture of the abscess revealed the nature of the disease. Fortunately the child had been kept in the upright position, so that pus was discharged into the mouth, or suffocation would have ensued.

INFLAMMATION OF THE PAROTID GLAND.

First, specific inflammation or parotitis (see Mumps); second, symptomatic parotitis occurs in typhoid fever, pneumonia, pyæmia, and septicæmia. The process is intense, characterized by swelling, redness, and heat over the parotid gland. There are pain and difficulty of mastication; suppuration rapidly ensues in the septic form. It is thought to be an unfavorable symptom, but I have seen two cases in typhoid fever get well. In a case of septicæmia it did not advance to suppuration. Stephen Paget has described a symptomatic inflammation in disease of the abdomen and pelvis. He collected 101 cases, 50 of which were due to injury, disease, or temporary derangement of the genital organs, as by slight blows, or in females to the introduction of a pessary. It may occur before the menstrual period or during pregnancy. Septicæmia or pyæmia does not attend the process—indeed, many of the cases are afebrile. In 78 cases, 45 suppurated and 33 resolved without suppuration.

Gowers describes a case of parotitis which occurred in the course of fatal peripheral neuritis.

THE ŒSOPHAGUS.

Inflammations of the Œsophagus.

Acute Inflammation. Acute inflammation is recognized by severe pain on swallowing. It is associated with the sensation of a foreign body in the lower portion of the throat. There may be tenderness on pressure along the course of the œsophagus. The pain is aggravated by speaking. The pain may extend along the vertebral column to the cardiac end of the stomach, and is usually of a burning or raw character. When the inflammation is due to traumatism, as the swallowing of acids or other caustics, the mouth and pharynx show the effects of the inflammation, and, in addition, there is agonizing burning pain at the root of

the neck and between the shoulders. The inflammation is usually attended by erosion of the mucous membrane, and hence not only frothy mucus of a glairy character is expectorated, but also blood and shreds of membrane. The effect of the corrosive poisoning on the general system is marked. There is great prostration. Because of the accompanying gastritis there is intense thirst. Acute inflammation of the œsophagus may end in ulceration or in resolution. The traumatic inflammation is followed by chronic inflammation, which ultimately results in stricture.

Chronic Inflammation. Chronic inflammation is attended by pain in the act of swallowing; liquids are swallowed readily, but solids with great difficulty. Viscid mucus is expectorated, usually in large amounts.

Abscess of the Œsophagus.

The acute inflammation may terminate in abscess. The abscess usually develops slowly, with pain on swallowing and on movements of the neck. When the abscess is high up in the gullet, it may present on the exterior of the neck. If it is situated outside of the œsophagus, and is secondary to disease of the vertebræ, it is slow and chronic in its course; fever and rigors attend its development.

Ulcer of the Œsophagus.

Ulcer, excepting in connection with acute inflammation, occurs but rarely. The so-called "decubital" ulcer is most common. This forms in the later stages of life in old people or in those who are greatly reduced by long illness, and the symptoms are usually marked. Its site is opposite the cricoid cartilage, the position where the œsophagus is most subject to injury. In rare cases typhoidal ulceration occurs, and may give rise to ultimate stricture. Somewhat more commonly a *peptic* ulcer may form in the lower part of the œsophagus. The symptoms with these forms are chiefly localized pain and pain on swallowing, occasionally complicated by hemorrhage, or rarely by perforation. They may lead to stricture. Syphilitic ulceration is not uncommon, and in rare instances tuberculous ulcers are found. These forms usually cause no marked symptoms unless they lead to stenosis, of which syphilis is a prolific cause.

Stricture of the Œsophagus.

Stricture due to the healing of ulcers, usually following traumatic inflammation, is recognized, first, by the gradual development of the symptoms, by the painless nature of the obstruction in the large majority of cases, and by its seat. It is readily found by introducing a bougie; the patient can sometimes localize the area in the upper portion of the œsophagus. The difficulty of deglutition continues over such a long period that nutrition is but slowly interfered with, but gradual emaciation with coincident anæmia develops eventually.

Carcinoma of the Œsophagus.

In cancer of the œsophagus dysphagia is the most prominent symptom. It comes on gradually. The patient expectorates a considerable quantity of frothy mucus, often containing blood, and revealing, on careful examination, cancerous tissue at times. Pain is not generally very severe. Cough is usually present, due to pressure of the cancerous mass on the recurrent laryngeal or pneumogastric nerve. Sometimes the cancer develops in the anterior wall, and ulcerates into the trachea or bronchus. When this complication takes place, the cough is violent. Dyspnœa from pressure is likely to occur. Perforation of the œsophagus into the air-passages is followed by pulmonary abscess or gangrene, or the sudden appearance of dyspnœa, and shortly the onset of aspiration pneumonia. When ulceration causes a pulmonary œsophageal fistula, the condition may simulate that of phthisis.

The difficulty of deglutition due to cancer must be distinguished from that of traumatic or syphilitic stricture and from spasmodic stricture and paralysis of the œsophagus. The history of the case aids in the recognition of traumatic or syphilitic stricture, while the ready passage of a bougie indicates that the difficulty is spasm or paralysis. Cancer usually occurs late in life and is attended with rapid emaciation. Its complications, more common than in other obstructions, are attended with fever and rapid prostration. Cancer may be distinguished from disease outside of the œsophagus by the condition of the stomach beyond the point of stricture. If there is cancer, atrophy is more likely to take place, the change in size being recognized by distending the stomach with air or fluids. The excessive secretion of ropy mucus, salivation included, is, according to Harrison Allen, pathognomonic of disease in the pharyngolarynx or in the œsophagus, at or above the level of the left bronchus. This secretion may be an early indication of cancer of the œsophagus. It may occur in aneurism.

Sarcoma of the œsophagus is very rare. It occurs most frequently in males and presents symptoms like those of carcinoma.

Dilatation of the Œsophagus.[1] Primary dilatation of the œsophagus is an extremely rare affection. The chief symptom is the regurgitation of food, which is neutral or alkaline, and may be returned some time after the act of swallowing. The patient sometimes complains of a sensation of distention along the course of the œsophagus, with heat and burning. The odor of the breath is fetid. If the œsophagus is not deflected, a bougie can be passed.

If the dilatation is secondary, the amount of dysphagia depends upon the obstruction. Food, however, is not returned immediately. After remaining an indefinite time, not longer than two hours, it is regurgitated unchanged. Bougies, of course, do not pass. In sacculated dilatation, which usually takes place in the posterior wall near the pharynx, a bougie may sometimes pass, and at other times may be caught in the sac. The sac may be enlarged, so as to retain a considerable amount of food, which is regurgitated some time after it is swallowed. A sacculated

[1] "Foreign Bodies in the Œsophagus." Allen, New York Med. Jour., Aug. 17, 1895.

diverticulum, from traction on the outside of the œsophagus, may occur when there is disease of the glands of the neck or mediastinum, with adhesions to the œsophagus.

Sacculated diverticulum in the upper portion of the œsophagus is often readily recognized by discovering the alternate presence and absence of a mass when the sac is full or empty.

In the lower part of the œsophagus, the diagnosis between sacculated diverticulum and a simple dilated diverticulum is best made by using Jung's method. His apparatus consists of (1) a small tube which is introduced into the dilated area; (2) a large stomach-tube with small perforations extending well up the sides; and (3) a small stomach-tube which has its openings only at the end. No. 1 is introduced, then No. 2 is passed into the stomach and a small amount of colored fluid poured through No. 1. If the difficulty is a sacculated diverticulum, the fluid will remain in the sac; if it is a dilatation or simple diverticulum, the fluid will pass through the perforations in tube No. 2 into the stomach. If No. 3 is now passed into the stomach through No. 2, the fluid may be siphoned out. The *x-rays* may also be used to demonstrate the nature of a widening of the lumen.

Rupture of the Œsophagus.

This occurs in very rare instances as a result of disease of its walls, or in some instances even from violent vomiting. It causes extreme pain with severe shock. Hemorrhage may occur through the mouth or bowel; pleural effusion is likely to be seen, and the condition soon results in death.

Functional Affections of the Œsophagus.

The functional affections are quite as common as organic disease. They are of longer duration, but are unattended by the same grave effects upon the general system. *Spasm* is one of the most frequent affections. It may be so intense as to lead to temporary stricture. It usually occurs in women. The attack comes on suddenly during the act of swallowing food. The food is at once regurgitated. After subsidence of the perturbation, swallowing can be accomplished, if done slowly. Spasm usually occurs in hysteria. The patient may have had some slight accident in the performance of the ordinary act of deglutition, out of which grew the idea that swallowing can not be accomplished. In consequence, the further acts are performed with trepidation, and slight emotional disturbance at the table may cause a recurrence of the sudden spasm.

Unfortunately, calling attention to the act of swallowing always has the effect of embarrassing the patient, and the taking of a meal under unusual circumstances is sure to be attended by complete dysphagia. Sometimes the idea is conceived that certain forms of food alone can not be swallowed. It is usually thought that solid food gives the distress. Mitchell says that dysphagia occurs early in cases of hysteria; unless relieved, the hysterical symptoms are likely to be transferred to the stomach. I saw a female patient who, after an ordinary choking attack,

for several years could not swallow food in the presence of strangers, or after the slightest emotional disturbance, or if hurried. The spasm disappeared after treatment with bougies.

In *paralysis*, difficulty of deglutition is the main symptom. The course of œsophageal paralysis depends upon its cause. The larynx is usually affected at the same time, so that laryngeal symptoms are present. Paralysis generally comes on very gradually. It may be due to cerebral hemorrhage, tumor, bulbar paralysis, or to general paralysis of the insane. The bougie passes easily, and does not cause irritation. In paralysis there is no regurgitation of food.

CHAPTER IX.

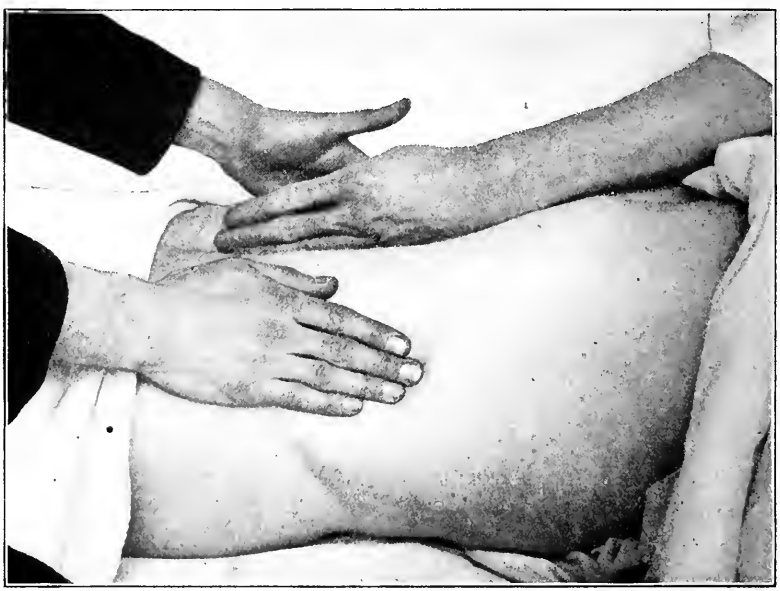
DISEASES OF THE PERITONEUM AND RETROPERITONEAL GLANDS, THE STOMACH AND INTESTINES.

DISEASES OF THE PERITONEUM.

Ascites.

Ascites is accumulation of fluid in the peritoneal cavity and may be due to general or local causes. The *local causes* are, first, *simple*, *cancerous*, or *tuberculous* inflammation of the peritoneum; second, portal obstruction from disease of the liver, as cirrhosis, or disease of the portal vein from compression or inflammation; and third, abdominal tumors, which are often attended by ascites, particularly solid tumors of the ovary. The *general causes* of ascites include all those that give rise to dropsy.

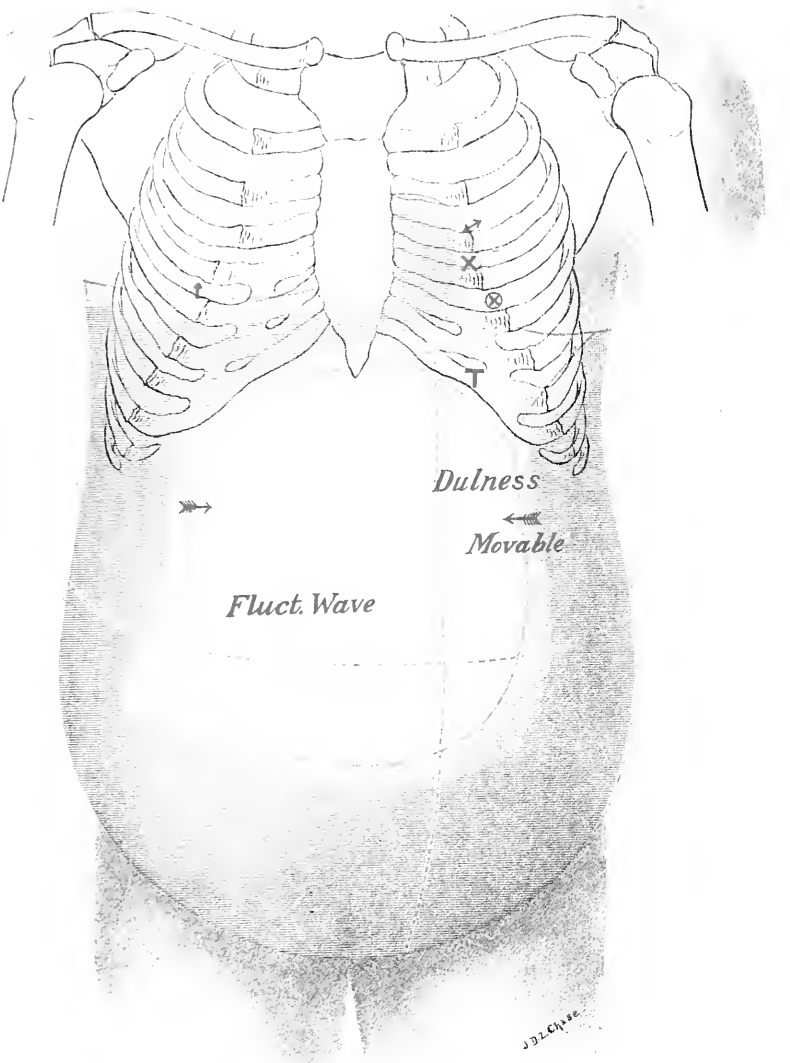
Fig. 360.



Bimanual palpation to determine the presence of fluctuation.

Inspection. (Plate XXXVII.) The surface of the abdomen is usually smooth, but when the effusion is large, the skin becomes tense and *lineæ albicantes* may be present; there may be pouting of the umbilicus. When the ascites is due to disease of the liver or of the portal vein, the

PLATE XXXVIII.



Ascites.

Blue shading shows level of dulness in recumbent posture.
Dotted lines indicate change of level of fluid in other postures.

superficial abdominal veins become enlarged, although such enlargement may be produced by any effusion persisting for a long period of time. When the patient is lying upon his back, the front of the abdomen is flattened and the flanks bulge; when he turns upon his side, the flank that is uppermost becomes hollowed out and the front of the belly prominent. This is the appearance when a moderately large effusion has existed long enough to stretch the lateral abdominal muscles; but when the effusion is enormous, all parts of the belly are distended and the abdomen is barrel-shaped; in such a case the shape is not affected by posture.

Palpation. Fluctuation can usually be detected by applying one hand —the left, for example—firmly against one side of the abdomen while the opposite wall is gently tapped with the middle finger of the other—the right—hand at a point corresponding to the supposed level of the fluid. (Fig. 360.) At first the hand should be placed on the flank, and if fluctuation is not perceived, the hand should be brought forward toward the median line with each successive blow of the percussing finger. Sometimes light percussion will elicit the sign, at others more forcible percussion is required; the faintest tap is often sufficient. Care is necessary not to confound the undulatory vibration of the abdominal walls, produced by percussion, with the wave of true fluctuation; to guard against this error, the former must be cut off by the hand of an assistant placed vertically in the median line. In order to ascertain the position of solid organs in ascites we employ the procedure known as *dipping*, which consists in suddenly dipping down with the tips of the fingers over the organ sought for. The fluid is thus displaced, and the edge or surface of the organ is readily felt.

Percussion. *Dulness* is found over the space occupied by the fluid, which of course corresponds to the dependent portions of the abdomen; sometimes, as Tyson has pointed out, the colon gives rise to tympany in the flanks. In the dorsal position the dulness occupies the flanks and may extend around the lower portion of the abdomen; in the erect posture it may reach to the umbilicus in the median line and to the same level in the midclavicular line.

FIG. 361.

Ascites. Upper limits of dulness indicated by the dotted line. Umbilicus prominent. (Original.)

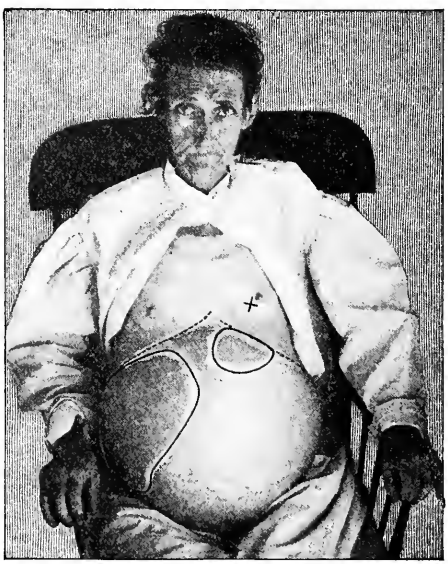
Aspiration. (See Chapter IX., Part I.) It is important to ascertain whether the fluid is *serous* or mixed with *blood* or *chyle*, and this can be done only by aspiration. *Hemorrhagic effusions* are usually found in

cancer and in tuberculosis, although in both of these diseases the fluid may consist of pure serum; in ruptured tubal pregnancy the fluid is always hemorrhagic. Disease of the lymphatics and of the thoracic duct with perforation in rare instances leads to the production of a chylous, milky ascitic transudate. *Chylous ascites* may also be due to the presence of filaria. It should be remembered that *lipæmia*, which sometimes develops as the result of excessive or long-continued ingestion of milk, may be responsible for the turbidity of an effusion into a serous cavity.

The **subjective symptoms** are those due to the primary disease and to mechanical pressure.

Differential Diagnosis. Ascites must be distinguished from enlargement of the abdomen due to *pregnancy*, enlargement due to *ovarian tumor*, and enlargement due to an *over-distended bladder*. During the last month or two of gestation, especially when the woman has previously borne children, the enlargement is general. In ovarian tumor the swelling at

FIG. 362.



Ascites from sarcoma of ovary. Dislocation of liver and spleen. × is apex-beat, not lifted because of fallen abdominal organs. (Original.)

first takes place to the right or left of the median line; when the tumor is very large, the signs may be in the central portion of the abdomen, but the flanks are always tympanitic on percussion. A vaginal examination usually reveals the local disease. A distended bladder should always be thought of, and catheterization performed in doubtful cases. *Pancreatic cysts* may be mistaken for ascites, and large *hydatid cysts* connected with the liver may simulate an accumulation of fluid in the peritoneal cavity. The history and the appearance of the fluid obtained by aspiration point to the correct diagnosis.

Peritonitis.

Acute General Peritonitis. Inflammation of the peritoneum may be acute or chronic, general or localized, primary or secondary. Acute general peritonitis is rarely primary; more commonly it represents a terminal infection in the last stage of chronic Bright's disease or other dyscrasia. A rheumatic form is said to follow exposure to cold, and when the inflammation develops as the result of an injury, the term traumatic peritonitis is used. In the great majority of cases peritonitis is secondary to inflammation in one of the organs covered by the peritoneum or to perforation of an abdominal viscus. Thus, it may follow inflammation of any portion of the gastro-intestinal tract or of the pelvic viscera, or suppurative inflammation of the spleen, liver, or pancreas.

Peritonitis an Infection. In every instance the primary inflammation in the organs mentioned is due to some micro-organism, as the staphylococcus, streptococcus, or *Bacillus coli communis*, and the peritoneal inflammation to subsequent extension of the infection. In perforative peritonitis, as in ulcer of the stomach or bowels, the element of infection also plays the most important part in the process. Inflammation of the gall-bladder may eventuate in perforation with resulting peritonitis; and rupture of an abscess in the liver, spleen, or kidneys, with the evacuation of pus into the peritoneal cavity, is also followed by general peritonitis; but the most common forms are due to appendicitis or to disease of the Fallopian tubes. Acute peritonitis may develop by direct infection in the course of tuberculosis or other systemic infection.

Symptoms. The onset of peritonitis depends in a measure on the cause, that of the perforative form being quite sudden. The disease is ushered in by *chills* and *rigor* with intense abdominal pain, at first localized but rapidly becoming general. The *pain* is constant although subject to exacerbations, and is aggravated by movement and by pressure. The patient assumes the dorsal decubitus with the knees drawn up, in order to relieve the tension of the abdominal muscles. Owing to the pain attending the contractions of the diaphragm the respirations are shallow and hurried, illustrating the superior thoracic type of breathing, and the patients are unable to take a full breath without suffering; while the act of speaking or coughing greatly adds to their distress. The *location of the pain* depends on the seat of the primary infection, which is usually in the right or left lower quadrant where the Fallopian tubes and the appendix are found. The pain of perforating gastric ulcer may be referred to the back, or to the chest or shoulders.

The general symptoms are marked. After the initial chill the *temperature* rises to 104° or 105° F., and then continues at a moderate elevation in ordinary cases. In septic peritonitis, however, the temperature may continue at the initial level or even rise above it, and in rapidly progressing cases a temperature of 105° or 106° F. on the second or third day is not uncommon. The difference between morning and evening temperature is not great except in the case of abscess. The onset of pain and physical signs of distention and rigidity is usually followed at once by *vomiting*, which is painful and at first complete, the vomited matter

consisting of stomach contents, then of yellowish, bile-stained fluid, and finally of greenish material. Later, actual vomiting is replaced by simple regurgitation of fluid, so that the slightest movement or the ingestion of a small amount of fluid is followed by passive regurgitation, unassisted by the action of the diaphragm, of the characteristic greenish-colored fluid, sometimes continuing almost uninterruptedly for from twenty-four to forty-eight hours. The bowels are constipated; they may be loose at first, but *constipation* is characteristic, the intestines being paralyzed from over-distention and from œdema of their walls due to the inflammatory process.

Physical Signs. The *appearance* of the patient at the height of the disease is characteristic: the features are pinched, the eyes sunken, and the expression of the face anxious. (See Expression.) The tissues of the body show the wasting effects of the continual vomiting, and the signs of collapse are marked. The *tongue* at first is furred, but later becomes dry, and often cracked and red. The abdomen is extremely sensitive, so that the patient is unable to bear the weight of clothing or external applications.

Distention gradually develops and the percussion-note becomes tympanitic. The distention may become so great as to push up the diaphragm, causing the above-mentioned superior thoracic type of breathing, and to dislocate the heart so that the apex-beat appears in the fourth interspace. The splenic dulness may be altogether obliterated, and the liver-dulness diminished in size; it has even been asserted that in some instances the latter also becomes obliterated, but recent observations have shown that the obliteration is confined to the anterior aspect of the abdomen; in the axillary region the hepatic dulness, although diminished in extent, persists even when the distention is enormous. The obliteration referred to could only take place in perforative peritonitis. Osler points out that in pneumoperitoneum from perforation the anterior hepatic dulness may be obliterated, although dulness persists in the lateral region on account of the effusion of fluid. When a patient with gas in the peritoneum is turned on the left side, a clear note is heard beneath the seventh and eighth ribs, corresponding to the hepatic region. The abdominal muscles are more or less rigidly contracted.

Spasm of the muscle overlying the primary inflammatory focus takes place at once and is a valuable indication of the source of the infection. In some cases, usually when the inflammation is due to the streptococcus, the abdomen shows but little distention or may even be flattened, with board-like rigidity; the pain is not marked, and tenderness may be absent.

The *pulse* is rapid and feeble and soon becomes thready, ranging from 110 to 150 beats in the minute. During the early stages it may be small and hard—the peculiar wiry pulse of the early stage of peritonitis to which numerous clinicians have called attention.

The *urine* is scanty, and micturition may be frequent and painful, particularly if the inflammation began in the pelvic organs. In suppurative peritonitis the urine usually contains a large quantity of *indican*.

Course and Prognosis. In severe cases death may take place in from

thirty-six to forty-eight hours, but, as a rule, life is prolonged for five or six days, or even longer. The vomiting persists, collapse with falling temperature ensues, the pulse becomes rapid and thready; except when the septicæmia is marked, the mind remains clear throughout the entire attack, and the patient dies of paralysis of the heart. Septicæmia manifests itself by a dusky color of the face, rapid and irregular pulse, slight delirium, dry brown tongue, and other evidences of the typhoid state. In protracted cases some effusion may take place into the peritoneal cavity, and dulness which alters with the position of the patient is noted in the flanks. If recovery takes place, particularly in tuberculous peritonitis, the affection sometimes becomes circumscribed, and in that case the dulness is not movable.

Diagnosis. It is essential in making a diagnosis to ascertain, if possible, the primary source of the infection or inflammation. To determine this, we inquire into the age, sex, and history of previous disease. In young male adults appendicitis is first to be thought of; in females, inflammation of the pelvic organs. In chlorotic subjects, if the pain is high up, a history of ulcer of the stomach must be inquired for. Later in life, particularly if there has been jaundice, the possible history of frequent attacks of gallstones and of hepatic disturbances must be ascertained. All forms of intestinal obstruction must be sought for. Frequently, however, a definite cause can not be ascertained. Peritonitis occurring in the course of typhoid fever is usually due to perforation, and its recognition, as a rule, presents no difficulties; but the mental state of the patient may be such that he does not complain of pain. In such cases local tenderness, rigidity, and the sudden occurrence of a leucocytosis are almost distinctive signs.

The most important conditions to be borne in mind are: *enterocolitis*, *acute hemorrhagic pancreatitis*, *intestinal obstruction*, and *rheumatism of the abdominal muscles.*

The distinction of acute peritonitis from *enterocolitis* is usually not difficult if due attention is paid to the development of the case. The pain is less severe and more colicky in enterocolitis; the general tenderness is not so great as in peritonitis, and the distention does not interfere with respiration to such a marked degree. Diarrhœa is more common in enterocolitis; collapse, if present, is less pronounced.

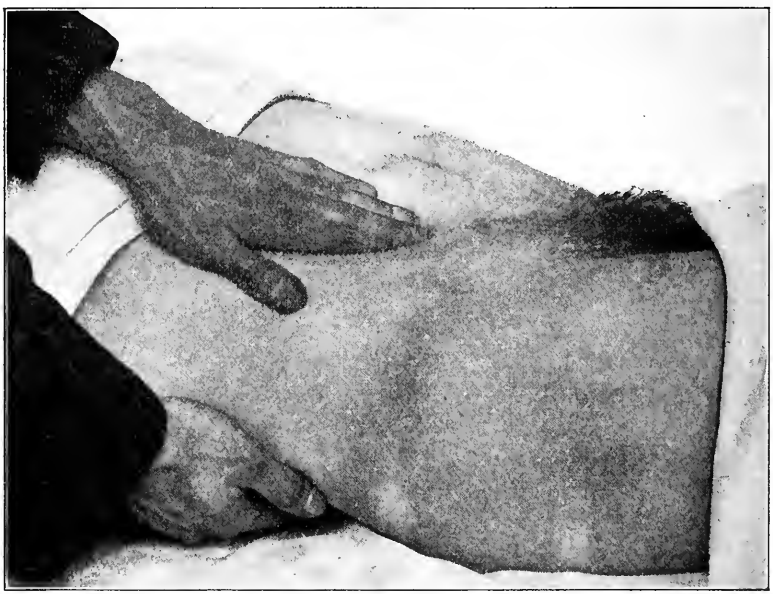
Acute hemorrhagic pancreatitis may simulate peritonitis by the sudden onset and the intensity of the pain and by the occurrence of shock.

The diagnosis from *obstruction of the bowel* is difficult in the absence of a distinct history; but in peritonitis stercoraceous vomiting does not occur until late, and the tympanites and pain are more general. Peritonitis frequently accompanies or follows intestinal obstruction. A tumor, if one be present, may point to the true nature of the case; or a discharge from the rectum may reveal invagination as the exciting cause.

Rheumatism of the Abdominal Walls. There is no history of sudden acute pain followed by general pain; the fever is less marked; the respirations are not interfered with, the pulse is not so rapid, and symptoms of collapse do not supervene. A rheumatic pharyngitis or inflammation of muscles in some other portion of the body may occur simultaneously.

Peritonitis is simulated by a condition to which the name *hysterical peritonitis* has been applied. It occurs in hysterical subjects, and every feature of the true form is imitated. The mode of onset, the decubitus, the difficulty in micturition, and the local distention and tenderness of the abdomen are characteristic of both. In a few cases that I have seen the vomiting differed from that of true peritonitis, both in the mode of ejection and in the character of the fluid. It must not be forgotten that the temperature may even be elevated, and collapse may take place in the hysterical form. In the cases that I have seen, the abdominal facies did not develop; on the contrary, the facies of hysteria, with the self-interest

FIG. 363.



Bimanual palpation to determine if pus is localized in the retrocæcal region.

exhibited by the patient, and the precision with which symptoms were narrated, coupled with emotional or other manifestations of hysteria, suggested the true nature of the affection. The case is judged by the history of other manifestations of hysteria and the presence of permanent stigmata of the disease; there is always a positive absence of cause and of disease in any of the abdominal viscera. Sometimes if the patient's attention is diverted, the tenderness on pressure may not be complained of. I am not familiar with the results of examination of the urine in this form of peritonitis; indican should not necessarily be increased, as it is found to be in acute suppurative peritonitis.

Local Circumscribed Peritonitis. The causes of localized peritonitis are those of general peritonitis—extension of inflammation from, or perforation of neighboring viscera. After perforation has taken place the process remains localized if the inflammation progresses rapidly enough to bring about immediate isolation of the perforated area from the gen-

eral cavity of the peritoneum. Accordingly the regions of predilection for the development of local or circumscribed peritonitis, with or without perforation, are the same as those that have been indicated as the usual starting-point of general peritonitis. But although the inflammatory focus may be retained by a limiting wall, the process may nevertheless, after suppuration has began, undergo gradual extension and the pus may burrow in various directions.

Circumscribed peritonitis in the lower half of the abdomen is due to disease of the vermiform appendix or of the Fallopian tubes; the local signs are, first, those of pain and tenderness; second, the development of tumor.

Subdiaphragmatic Abscess. Subdiaphragmatic or perigastric abscess is a collection of pus walled in by the stomach, spleen, diaphragm, colon, and the abdominal walls, and may be associated with local peritonitis in the upper half of the abdomen. The most common cause is irritation from a gastric ulcer that has nearly or quite perforated and has formed adhesions with the surrounding viscera. This was the cause in 41 out of 52 cases analyzed by Fenwick, while in 6 it was associated with cancer, and in 4 with abscess commencing externally. *Pain* in the epigastrium was the most prominent symptom; some of the patients were dyspeptic, and vomiting occurred also; while, singularly enough, hæmatemesis is mentioned in only 2 cases. *Fever* is quite marked. Fenwick thinks that in every case of perigastric abscess, except in persons affected with phthisis, cancer, or some other chronic exhausting malady, the first formation of the abscess will be accompanied either by collapse and signs of general peritonitis, or by sudden and severe pain in the epigastrium with indications of local peritonitis.

The *physical signs* are not pronounced; a tumor, according to the same author, is rarely distinguishable except when the condition is caused by cancer. The percussion-note, when a tumor is present, is at first dull but later becomes tympanitic, and the tumor is not movable on inspiration or external pressure; there may, however, be arching outward of the ribs. Palpation is difficult on account of the tension of the abdominal muscles. The degree of displacement of surrounding viscera depends on the size of the abscess and the extent of the adhesions. The breathing is embarrassed by the upward pressure of the diaphragm against the lung and heart, and the dulness may extend as high as the angle of the scapula, simulating a pleural effusion. It should be borne in mind in this connection that the lower limit of the lungs in health corresponds to the eighth or ninth interspace. There may also be dulness in the axillary region; and if the abscess is on the right side, it may simulate an enlarged liver and cause a considerable extension of the hepatic dulness anteriorly, laterally, or posteriorly. If the local peritonitis in the epigastric region is secondary to disease of the pancreas, it may be limited to the lesser peritoneum and cause the physical signs of effusion into that cavity (see Diseases of the Pancreas); while subdiaphragmatic abscess is not limited to the lesser peritoneum and must be recognized by the history of a previous lesion capable of causing perforation and by the general symptoms of abscess.

Subphrenic Pyopneumothorax. Sometimes gas is formed in con-

nection with the abscess, and amphoric sounds are heard on auscultation and percussion both in the abdomen and in the thorax. In this condition, to which the name *pyopneumothorax subphrenicus* has been given, the abdomen becomes tense, tender, prominent, and tympanitic on percussion (see page 918). It must be distinguished from *left-sided pneumothorax*, which pushes the left wing of the diaphragm down and therefore increases the area of dulness, while the left lobe of the liver and the spleen become palpable. In subphrenic pyopneumothorax, on the contrary, the liver and spleen are not palpable, nor can their limits be outlined by percussion. In left pneumothorax the heart is dislocated to the right and the cardiac area is tympanitic on percussion instead of dull, while the impulse is seen in the epigastrium or even to the right of the sternum; in subphrenic pyopneumothorax, on the other hand, the heart is displaced upward and the impulse appears in the nipple-line, while only the lower half of the cardiac area is tympanitic. Pyopneumothorax subphrenicus must not be mistaken for *dilatation of the stomach*.

Chronic Peritonitis. Symptoms of chronic diffuse peritonitis may follow an acute attack, or may occur in the course of tuberculosis. The intestines and peritoneum are matted together. General pain and tenderness, with a prolonged period of ill health, attend the diffuse form. (See Tuberculous Peritonitis.) If there is considerable fibrous proliferation, even though not cancerous or tuberculous, the abdomen becomes retracted, the muscles rigid, the note over the abdomen modified or dull tympanitic, especially in the upper half of the abdomen and over the liver. Sometimes a fremitus can be felt. The patients are under weight and without strength. The pain may continue a long time. The disease finally results, at least clinically, in such compensation that the patient is able to continue his usual occupation. Owing to the formation of adhesions the patients have painful sensations of a dragging character, the significance of which is not serious, however, unless, as has been seen in intestinal obstruction, coils of intestine become caught in these adhesions.

Cancer of the Peritoneum.

Cancer of the peritoneum is a disease of old age and occurs more frequently among women than among men. Although sometimes primary, in the majority of cases it follows cancer in other organs, as the stomach, liver, or uterus. The indurated and nodular omentum forms an irregular mass lying transversely across the upper zone of the abdomen and may be painful on pressure. A tumor of the same physical characters is seen in tuberculous peritonitis, and I have seen several such tumors in the aged without apparent cause, unless it were proliferative peritonitis. (See Tumor.) Ascites is usually present and the enlargement of the abdomen is in striking contrast to the emaciation of the rest of the body; the ascitic fluid is bloody. Sometimes pain is the most prominent symptom. Progressive emaciation, chronic ascites without cause, and a localized tumor without the occurrence of fever are strongly suggestive of carcinoma; and if these symptoms are present without signs of cancer in some

other organ, as the stomach, rectum, or uterus, there is probably primary cancer of the peritoneum.

Retroperitoneal sarcoma, or Lobstein's cancer, may be central or lateral; The growth is very large, deep-seated, and usually fixed; it can be detected above the sacrum by rectal examination; ascites and the general symptoms of cancer are present. The growth is crossed in front by the intestines, which transmit to the palpating hand a peculiar sensation like the fremitus of a hydatid cyst, as in Barrow's case. Lockwood's cases were thought to be solid ovarian tumors. Sarcoma of the glands and tissues in the retroperitoneal space, according to J. Dutton Steele, is slightly more common in males than in females, and more common in the first decade and after the fiftieth year. The duration is about nine months. Of the 65 cases collected by Steele, 39 per cent. were spindle-cell sarcoma, 34 per cent. round-cell sarcoma, 14 per cent. lympho-sarcoma, and 13 per cent. were mixed cases. The growth originates in the lymph-glands or in fibrous connective tissue surrounding the kidneys, the spinal column, the pelvis, or the sheaths of bloodvessels. The onset is insidious, the first appreciable symptoms being the presence of a tumor, or the effects of pressure upon the vessels, nerves, or viscera of the abdominal cavity, the nature of which depends upon the site of the tumor; varicocele is a frequent concomitant of the disease. It is often impossible to distinguish retroperitoneal sarcoma from malignant disease of the kidney or of the suprarenal bodies. The diagnostic features are (*a*) the rapid growth; (*b*) the position of the colon, which is pushed in front of the tumor, particularly if it is lateral; (*c*) the pressure-symptoms; (*d*) movability of the tumor both with respiration and independently, and sometimes fluctuation.

Tuberculosis of the Peritoneum.

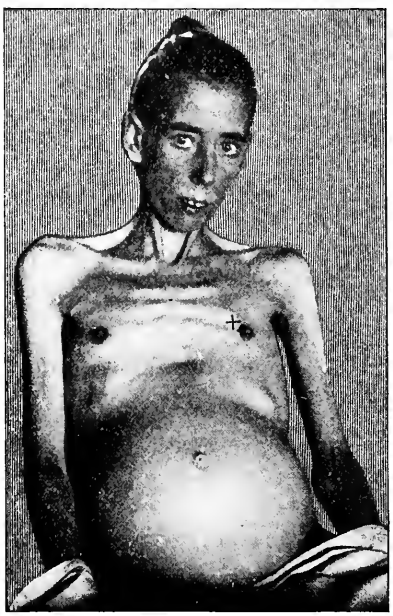
The tuberculous process in the peritoneum may be acute or chronic. While the symptoms are often acute and alarming, in the larger proportion of cases the process is more *chronic* and is attended by characteristic local, and by general symptoms resembling those of typhoid fever—indeed, a true typhoid state may develop in the course of the disease. In rare instances the malady runs its course without producing any symptoms either local or general, In protracted and moderately severe cases the *fever* is moderate and may be continued or remitting in type; in old people the temperature is frequently subnormal. (See Fig. 366.) In more severe cases the fever is high, but always irregular in type, although the latter may also approach the remittent form. Profuse sweating is characteristic of the disease, and the emaciation is more or less rapid.

The Local Symptoms. The cases may be divided into four classes, according as they exhibit: (1) abdominal enlargement with effusion; (2) enlargement with tumors; (3) a combination of the two; or (4) enlargement without evidence of fluid or tumor in the abdomen. In the forms included in the second and fourth subdivisions the abdomen may subsequently undergo retraction.

1. **Enlargement with Effusion.** The local symptoms and the physical signs are those of ascites; but the distention is never so great as in ascites from cirrhosis of the liver. Often the fluid is confined by adhesions in the right or left quadrant of the abdomen, in which situation fulness and fluctuation may be readily detected.

2. **Enlargement with Tumors.** (Plate XXXIX.) The tumors are usually found in the upper zone of the abdomen, and may be localized in either quadrant, or extend from the right to the left. They are usually due to tuberculosis of the omentum, with secondary contraction. In some instances an indurated tumor, somewhat tender on pressure, may

FIG. 364.

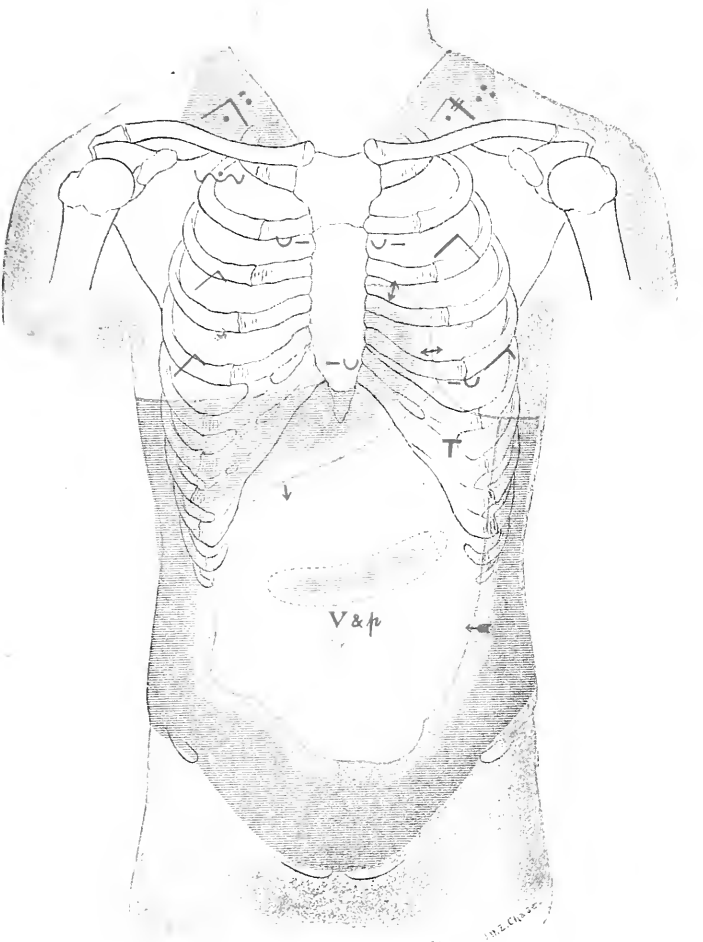


Tuberculous peritonitis; pulmonary tuberculosis. + site of cardiac impulse. (Original.)

extend across the abdomen midway between the xiphoid cartilage and the umbilicus, or at the level of the umbilicus, varying from two to four inches in width and sometimes continuous with the liver-dulness. In other instances more distinctly localized masses may be felt either to the right or to the left of the umbilicus. They may be movable and vary with the position of the patient. I have never seen tuberculous masses in the lower quadrants. In children with *tabes mesenterica* the tumors may be made out close to the vertebral column in the median line, extending to the brim of the pelvis, although at the lower portion they are not so distinct. The intensity of the dulness over the tumors depends upon their relation to the bowels and the degree of intestinal distention. Instead of dulness a modified tympany or muffled resonance may be observed.

3. **Cases in which Effusion and Tumors are Present at the Same Time.**

PLATE XXXIX.



Tuberculosis of the Peritoneum.

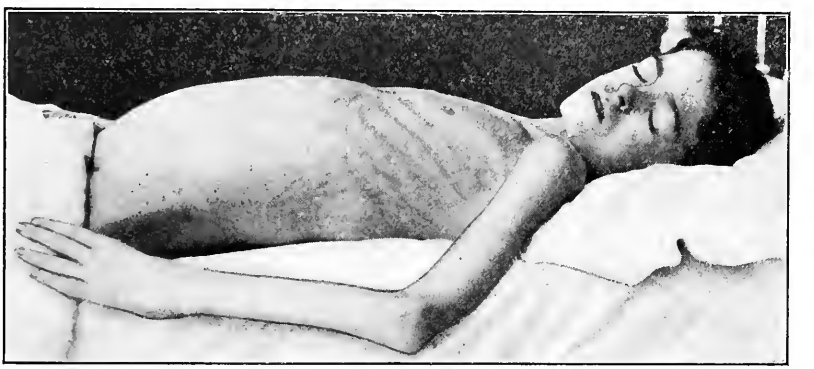
Abdominal exudate (not freely movable); omental tumor. Consolidation at apices.

These present symptoms common to the two conditions, although the tumors are not so distinctly defined.

4. Absence of Effusion and Tumors. When effusion and tumors are not present, the increased thickness of the peritoneum and of the intestinal walls produces a modified dulness over the entire abdomen. When retraction takes place, the resonance is of a woodeny character, the abdomen is more or less tender, and an ill-defined induration to which the term *carreau* is applied may be present.

The only other distinctive symptoms, apart from those described, are the symptoms, if any be present, of tuberculosis in other situations. The appetite is usually impaired, there is some atonic dyspepsia, vomiting may occur at regular intervals, and the bowels may be constipated, although in my experience they have usually been relaxed. Anæmia develops, and the skin becomes harsh and dry; the emaciation is often extreme, and the body covered with eruptions and boils; œdema is sometimes present over the ankles. Death results from exhaustion and from tuberculosis in other localities.

FIG. 365.



Tuberculous peritonitis. Note the emaciation, the length of the arm, the enlargement of the wrist, the size of the hand, and the tapering fingers.

Diagnosis. Cases belonging to the first and fourth classes above mentioned probably present the greatest difficulties. Age is an important factor; peritoneal tumors, with or without effusion, in young subjects are almost always due to tuberculosis; in the aged they must be distinguished from carcinoma or chronic peritonitis from other causes. The existence of diarrhœa is rather against carcinoma. In males the primary lesion often develops in the testicles. Sacculated peritoneal effusions may be confounded with ovarian or other abdominal tumors, especially if the tubercles develop primarily in the tubes or uterus. In a recent case the autopsy disclosed a large caseating ulcer inside the uterus, with tuberculous disease of the Fallopian tubes and of the peritoneum. The right tube was chiefly affected. The effusion during life was encysted and confined to the right lower quadrant, was not movable with the patient, and fluctuation could be made out both by external palpation and on bimanual examination *per vaginam*. The fact that the normal resonance in the flank was replaced by dulness was the only evidence that the

condition was not a growth. As Osler has pointed out, the association with salpingitis must arouse suspicion, particularly if at the same time signs of tuberculosis are found in some other part of the body, as the apex of the lungs or the pleura. The history of the case and the irregular development of the disease associated with gastro-intestinal disturbance rather than disturbance of uterine function are points in favor of tuberculosis.

The diagnosis may be obscured by the presence of tympanites, which is of frequent occurrence.

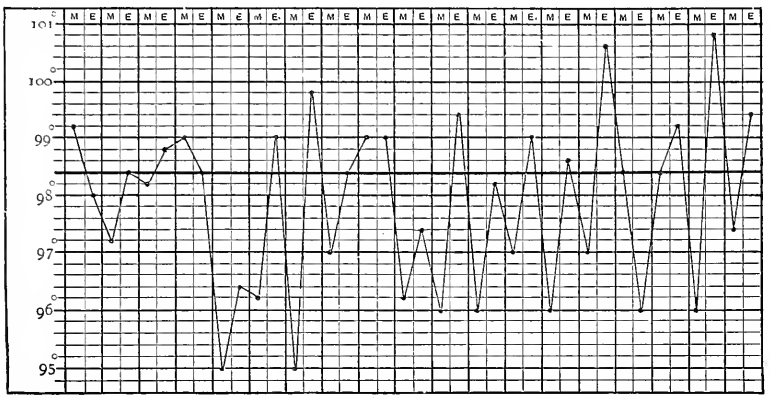
In not a few instances the local physical signs are apparently due to perihepatitis. In the case of a child under my care the local signs during life were of this character, and the symptoms were simply loss of appetite, with discomfort, weight, and fulness below the sternum. Both the right and left lobes of the liver were covered with enormously thickened tuberculous peritoneum, while the lower portion of the abdomen was the seat of simple plastic peritonitis.

Suppurative peritonitis may be closely simulated by acute tuberculous peritonitis, although usually the course of the latter is more prolonged and the fluctuations of temperature are less pronounced; otherwise it can not be distinguished from acute general peritonitis save by the absence of any of the recognized causes of that condition. A history of exposure to tuberculous infection, or the presence of tuberculosis in some other portion of the body, may be of service in determining the nature of a given case; thus associate tuberculosis of other serous membranes, as the pleura or pericardium, often develops in a short time an associated process that does not take place in ordinary peritonitis. There is diarrhœa in most cases—at least it has been present in the few instances that I have seen of this form of tuberculosis. Nevertheless, the diagnosis is sometimes impossible. Henry has called renewed attention to the occurrence of inflammation about the navel as a sign of tuberculous peritonitis; he believes the peri-umbilical erythema is pathognomonic of the affection.

Appendicitis. Acute tuberculosis of the peritoneum may also simulate appendicitis, first, in the local symptoms and physical signs; and, second, in the occurrence of subsequent infection of the peritoneum. In acute tuberculous appendicitis, however, the signs of a tumor are not so marked as in true appendicitis; although in one instance Keen operated upon a patient of mine, a healthy laborer in a rolling-mill, who had the classical symptoms of appendicitis. At the operation the appendix was found to be perforated and hanging in an encapsulated abscess. A fecal fistula ensued which did not heal, and within two months the patient died of general tuberculosis. The appendix was the seat of primary tuberculous ulceration. In another instance the appendicitis arose in the course of tuberculosis. In a third instance the patient, aged forty-five years, was admitted to my wards in the Philadelphia Hospital with high fever and pain in the abdomen, at first more marked along the margin of the liver. By the end of twenty-four hours the pain became more intense in the right lower quadrant of the abdomen; tenderness at McBurney's point was distinct; the painful area was enlarged and dull on percussion, and

the surface slightly œdematous. Fluctuation could not be detected. Extension of the leg was painful. Rapid general peritonitis ensued during which the surgeon saw him, but declined to operate until the subsidence of the attack. When the attack subsided, the local signs of tumor were not present. The fever persisted irregularly for a short time, while

FIG. 366.



Tuberculous peritonitis. Subnormal temperature. (Original.)

the more acute peritoneal symptoms subsided; then the right pleura became infected, and cough ensued with expectoration of mucopurulent fluid, which, however, did not contain tubercle bacilli. Subsequently the left pleura and the pericardium became involved. The entire course of the disease was marked by diarrhœa, profuse sweats, rapid emaciation, and exhaustion. Death took place at the end of five weeks, and at the autopsy general serous tuberculosis was found.

THE STOMACH.

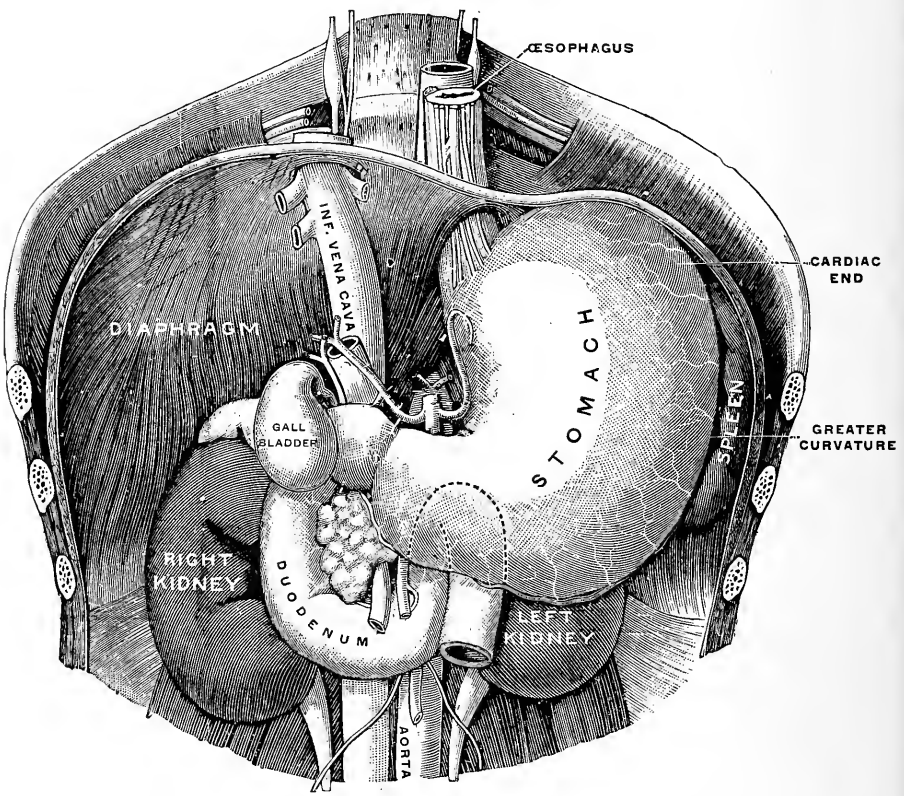
The stomach is a canal in which the food is detained for the purpose of solution and digestion. Its walls are made up of mucous membrane, muscle, and peritoneum. It is richly supplied with bloodvessels. Because of its great functional activity it has an abundant nerve-supply. It is, moreover, surrounded by rich plexuses of sympathetic nerves, through which and its special nerve, the pneumogastric, it is in intimate relation with every organ of the body.

The Symptomatology. The local symptoms of disease of the stomach are dependent upon: (1) the morbid process; (2) the effect of the process upon the anatomical structure and size of the organ (atrophy, dilatation, tumor); (3) the effect upon its function.

1. The Morbid Process. The symptoms due to the *morbid process* are not different from the symptoms of similar morbid processes elsewhere, save that they are modified by the function of the organ or its special construction. Hence, congestions are attended by discharge of mucus; inflammations by pain and by a flow of mucus and pus; ulcers

by pain and the accidents of ulceration (hemorrhage); malignant disease by pain and swelling (tumor), and its accidents, hemorrhage and obstruction; while each condition is attended by characteristic general phenomena. But the stomach is highly sensitive and resents the intrusion of disease or of that which (1) causes disease or (2) irritates the affected part. Expression of this resentment is shown in hyperæsthetic symptoms—*pain;* in the abolition or derangement of function—*indigestion;* and in the great pathological reflex act of the stomach—*vomiting.*

FIG. 367.



Stomach and duodenum, the liver and most of the intestines having been removed. The pyloric end of the stomach should be conceived as turned directly backward. (TESTUT.)

It will be seen later that this may be a symptom of any local morbid process affecting the organ, either directly by the disease which is its exciting cause, both of which are operative in irritant inflammations; or indirectly because the process has set up undue sensitiveness. In the latter instance any such material as food, which the stomach is accustomed to receive, becomes as much an irritant as mucus, pus, or blood.

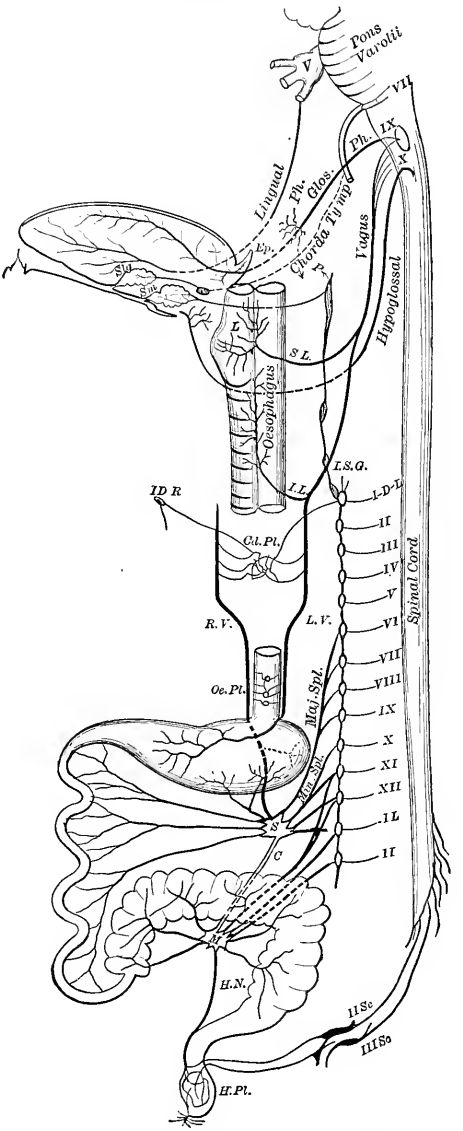
2. **Anatomical Symptoms.** The morbid processes modify the anatomical structure and lead to other morbid conditions, as we see when dilatation succeeds inflammation or obstruction at the orifices. The symp-

toms of the secondary conditions are the same as elsewhere—in atrophy, diminution in size; in dilatation, increase in size, with retention and fermentation, and finally discharge of the contents by vomiting.

NERVE MECHANISM. In the study of the symptomatology of gastric diseases the anatomical relation of the organ through its vascular and nervous connections must be considered. The student is sufficiently familiar with physiology and pathology to know that each organ has a representative in the central nerve-mass, the brain, and that disease in one organ will influence the function of, and create morbid symptoms in another organ with which it has intimate nervous connections.

The central representative or centre is influential in proportion to the power and activity of its peripheral adjunct, and is, moreover, influenced by the higher, psychical centres, which it in turn modifies. It influences or modifies lower centres—(1) functional, (2) vasomotor, (3) motor, and (4) sensory. The result of this mechanism in the case of the stomach is: 1. That functional alteration or organic disease of (*a*) the gastric centre, or (*b*) of centres of higher control, or (*c*) of the nerve that connects the centre and the organ—pneumogastric nerve—produces gastric symptoms. 2. That gastric diseases produce reflex symptoms in other organs, as cardiac palpitation. 3. That disease of other organs produces gastric symptoms or disease; instance the vomiting of pregnancy, of renal calculus, or of disease of the testicle, and the gastritis of kidney disease. Thus vomiting is caused by emotion (higher centre) influencing the pneumogastric (lower centre); by a tumor pressing upon or destroying the pneumogastric centre; or by a tumor, such as an aneurism, pressing on the pneumogastric

FIG. 368.



Showing the innervation of the digestive system.

nerve. I have taken the simplest illustrations. When we come to the study of gastric neuroses, the extraordinary influence of the nervous mechanism will be appreciated; and when hysteria is studied, the physiology of its extreme gastric symptoms will be recognized. When the mechanism and clinical course of vomiting are studied, it will be found among other causes to be frequently due to affections of the blood, the poisons of which irritate either cerebral centres or nerve-plexuses in the stomach.

Vascular Mechanism. Gastric diseases also arise because of some disturbance in the *vascular supply*. Thus in heart disease with venous stasis the gastric veins become the seat of congestion, with consequent gastric catarrh; and hepatic disease causes portal congestion and gastric catarrh.

3. Functional Symptoms. Any local disease of the stomach must influence its function; therefore, conversely, functional symptoms must be present in all local diseases. But functional disorder may be present without local anatomical change; the impairment is nearly always induced through the influences of the nervous system. The functions of the stomach are to digest and to absorb some of the products of digestion. The digestive function is both motor and chemical, and its completeness depends upon the food being thoroughly mixed with and dissolved in the gastric juice. The symptoms, therefore, must be due to changes (1) in the motor, (2) in the secretory, and (3) in the absorptive functions of the organ. The gastric functions may be increased or diminished; the former are primary and usually temporary aberrations; the latter succeed the former and are permanent. The functional symptoms, therefore, are the symptoms of what we know as *indigestion* or *dyspepsia*. They are described in the account of the subjective symptoms and also in the section on Gastric Neuroses.

4. Toxic Symptoms. The toxic symptoms arising in gastric disease are worthy of a few words. They are nervous symptoms due to the absorption of ptomaïns or products of imperfect assimilation. If absorption of the toxins takes place suddenly and in large amounts, coma and convulsions occur (Kussmaul's symptom); if gradually, hypochondriasis, melancholia, mental depression, and with these symptoms vasomotor phenomena of various kinds arise.

It follows, therefore, that in unravelling the symptomatology of gastric disease we must first note: (*A*) The *subjective symptoms* due to (1) to morbid processes, (2) to alterations of function, (3) to alterations of size (sense of fulness, etc.). (*B*) The *objective symptoms* due (1) to morbid processes, (2) to alterations of function, (3) to alterations of size.

Diagnosis from Disease of Contiguous Organs Functionally Related. The student will soon learn that functional diseases of the stomach can not be readily differentiated from diseases in other organs functionally related. He will find that to draw hard-and-fast lines between gastric and intestinal indigestion, or between so-called disordered gastric and hepatic function, is generally impossible. Organs that are closely related in physiological function, and have a common nerve- and blood-supply, can not be dealt with separately in conditions of disordered function.

Hence, indigestion and biliousness, or simple acute gastritis and duodenitis, are beyond the pale of close discrimination—in fact, the symptoms of each blend in a manner.

From what has been written above, it may be inferred that in order to judge correctly of the nature of gastric lesions we must ascertain (1) whether the gastric symptoms are dependent upon disease of other organs —particularly the eye, nose, and genitalia, the heart and kidneys—by an examination of each organ; and (2) whether other symptoms that may be present are caused by the gastric disease.

The Stomach in Other Diseases. Diseases of the stomach frequently mask other diseases; in other words, patients will complain of gastric symptoms that are but concomitant phenomena, behind which there are graver conditions. Thus in disease of the kidney, in phthisis, in chronic bronchitis, in emphysema, in valvular disease of the heart, catarrh of the mucous membrane of the stomach is of frequent occurrence, and is entirely due to the primary disease.

Tuberculosis. In tuberculosis the local gastric symptoms are often the most prominent feature. Thus in the earlier stages of phthisis loss of appetite and vomiting are of constant occurrence. The dyspeptic symptoms in a large number of cases precede the pulmonary symptoms, and may be so pronounced as to mask them entirely. The patients are usually delicate and anæmic; they complain of loss of appetite and mild indigestion, and are feeble and languid; there is some regurgitation of food. The case is treated as chronic catarrhal gastritis, but does not improve. On examining the lungs the physician is surprised to find a small area of consolidation, and upon inquiry may find that subjective symptoms of tuberculosis have been present for a considerable time. Every practitioner has seen scores of phthisical patients who, even when the disease is far advanced, believe that their symptoms are entirely due to disorder of the stomach. In addition to the early catarrh that precedes tuberculosis, symptoms of other gastric disease may occur. The well-known association of simple ulcer and phthisis is familiar; both occur at the same time of life, yet the presence of gastric symptoms may prevent investigation into those of pulmonary origin.

Anæmia and Chlorosis. In anæmia and chlorosis changes in the digestive tract are common. The functions of the stomach are impaired by the general blood condition. Here, too, we frequently have the association of ulcer with the general condition, although the danger of overlooking either is not so great as in tuberculosis.

Valvular Affections of the Heart. In valvular affections of the heart chronic catarrh of the stomach may result from venous congestion. As the symptoms may point to the gastric condition alone, it is necessary in all cases of chronic gastric catarrh to examine carefully into the condition of the heart. Over and over again patients apply for treatment, not on account of cardiac symptoms, but because of gastric disorder. They will be treated in vain if the primary cardiac affection is not discovered. Many cases of gastric catarrh have been cured by the use of digitalis.

Disease of the Kidneys. In disease of the kidneys the stomach is frequently involved. Vomiting and other symptoms of gastric disorder

may occur long before dropsy or any objective sign that would lead to a correct diagnosis. The gastric symptoms are due to chronic uræmia. Gastric symptoms also occur in other conditions of the genito-urinary tract, particularly in long-standing retention from chronic obstruction. Renal tumors may cause only disturbances of digestion, while gastric symptoms due to movable kidney are well known. The symptoms in the latter condition arise, first, from mechanical causes, as pressure of the kidney on the pylorus, and, secondly, from the influence on the nervous system.

Disease of the Liver. The intimate relationship between the liver and the stomach is such that when one is the seat of serious functional disturbance the other is likely to be affected, and it is often impossible to determine with absolute certainty which organ is the primary seat of disorder. The abuse of alcohol frequently induces chronic gastritis, just as it causes cirrhosis of the liver. On the other hand, cirrhosis of the liver is frequently the cause of chronic gastritis secondary to the portal congestion.

Diseases of the Nervous System. Attention has been called repeatedly to the relationship existing between disease of the central nervous system and disturbance of the gastric functions. (See Vomiting.) In sclerosis of the posterior columns of the cord this interdependence is more striking than in any other spinal disease. Not only do we have gastralgia and gastric crises; but mild indigestion, with hyperæsthesia and slight gastralgia, may be the first symptom of locomotor ataxia.

Diabetes. Diabetes may continue its course for a long period of time, during which the patient is thought to have stomach trouble, before an examination of the urine reveals the true nature of the case.

Gout and Rheumatism. Opinions differ as to the relationship of gout and rheumatism to gastric disorder. Some writers believe that a specific gouty inflammation of the stomach, due to the uric acid diathesis, is of frequent occurrence, and that one of the prominent manifestations of gout is dyspepsia in all its forms. The French consider gastric disturbances to be frequent expressions of the rheumatic diathesis. The relationship of the two, however, is thus far not fully developed; although it is unusual in these conditions to overlook the presence of either diathesis when symptoms of gastric disturbance occur. It is essential to bear in mind that gastric disturbances are more likely to occur in persons of a rheumatic or gouty diathesis than in healthy individuals, and that their successful management depends upon the recognition of the fundamental diathesis. It is more than probable that gastric disorder, along with defective metabolism, is primary in both affections.

General Examination. The objective examination has thus far been confined to a study of the stomach. The student will infer from the preceding chapters that in order, on the one hand, that the possible cause of the gastric disorder may be determined, or, on the other, the effect of gastric disorder upon the other organs ascertained, the latter must be examined carefully. Moreover, valuable data in the recognition of gastric affections and the diagnosis of the various forms are secured by such examination. The general appearance of the patient, the state of nutri-

tion, and the degree of strength furnish suggestive facts in the diagnosis. As well said by Stockton: "The preoccupied and dejected manner observed in those suffering from continued gastric flatulence; the restless, discomposed behavior, the stooped posture and half-surprised expression often seen in the victims of gastralgia; the emaciated, weak, and cachectic appearance frequently accompanying chronic food stagnation, are good examples of the value of the general appearance in the diagnosis."

It must be remembered that any local source of irritation distant from the stomach, as the eyes, the nose and pharynx, the uterus and ovaries, and the rectum, may be the primary cause of gastric disorder. The study of the hepatic and intestinal functions assists in the diagnosis. Examination of the urine and the blood may enable us to determine the nature of a gastric morbid process. Even the study of the skin is of importance. "A sallow, earthy-colored skin, showing improper secretion; a dry, harsh skin, with too rapid loss of epithelium, showing poor nutrition; a skin showing œdema, poor capillary circulation, lividity, or acne; certain forms of eczema, excess of pigment, or syphilides may afford important information as to the digestion, inasmuch as some of these may be the results and others accompaniments of gastric disturbance." (Stockton.)

The Blood. Examination of the blood enables us to determine the degree of the anæmia which may be the cause of digestive failure. The examination must be exhaustive. If a leucocytosis is present and there is no complicating condition, the gastric neuroses may be excluded. In carcinoma there is not only a severe secondary anæmia, but also poikilocytosis and a multinuclear leucocytosis. Such changes are without doubt the result of interference with the digestion because of motor inactivity. Post-digestive leucocytosis usually does not occur in carcinoma, and its absence is contributory evidence that carcinoma is present. It is, however, a very uncertain sign, as shown by Osler and McCrae and others.

The Urine. No study of a gastric disorder is complete without an exhaustive examination of the urine. For diagnostic, but chiefly for therapeutic purposes, the presence of renal insufficiency, hyperlithuria, indicanuria, glycosuria, peptonuria, and albuminuria must be ascertained.

The Reaction. The reaction of the urine is modified by the state of the stomach. In health the urine is alkaline after a full meal of ordinary character, but when HCl is absent from the gastric contents, this normal alkalinity does not occur. Alkalinity is rarely seen in gastric carcinoma.

The Chlorides. The chlorides are lessened when a small amount of food is taken; a similar cause diminishes the quantity of urea. Both are decreased in carcinoma and in some benign diseases of the stomach. But the chlorides are diminished in carcinoma without a proportionate lessening of the urea. It is this disproportion which, as pointed out by Nothnagel, is of diagnostic value in carcinoma ventriculi.

DISEASES OF THE STOMACH CHARACTERIZED BY FEVER, WITH PAIN AND VOMITING.

Acute Gastritis.

The simple variety of acute gastritis varies, according to the cause, from a slight attack of vomiting after indiscretion in diet with ordinary symptoms of indigestion to the more severe forms ushered in by chill and attended with fever.

In the mild forms there is a sense of fulness and discomfort in the epigastrium, attended with nausea. The appetite is lost, there may be disgust for food, and the flow of saliva is increased. There is excessive acidity. On examination the epigastrium is found to be tender. The onset of the attack is attended with giddiness, flashes of light before the eyes, frontal headache, and some prostration. The pulse is increased in frequency. When the nausea is most pronounced, the face is pale and the extremities cold; vomiting then occurs, the ejected matter consisting of ingesta only slightly changed, with mucus and watery fluid. The taste is very bitter, and the vomitus is often colored green from bile-pigment. Another attack of vomiting may be sufficient to give relief, or it may be repeated for twenty-four to forty-eight hours every hour or two. After the stomach is relieved of food, mucus and bile alone are vomited.

Examination of Stomach Contents. In ordinary cases the reaction of the vomited matter is neutral or faintly acid. No free hydrochloric acid is present, but later fatty acids are found; pepsin is diminished. In some cases the quantity of HCl may be increased as the result of a less severe irritation.

Twelve or twenty-four hours after the gastric symptoms intestinal symptoms may arise. Borborygmi and colicky pains are complained of, followed by diarrhœa, with some tenesmus. Herpes labialis may occur, and some writers speak of a peculiar odor being exhaled from the skin. The more severe cases are ushered in with chill followed by fever. The local symptoms are much aggravated. The tongue is furred, and the breath foul. The vomiting is frequent and severe. The skin is livid and the pulse becomes rapid.

Diagnosis. Acute cases attended by fever may be mistaken for meningitis, peritonitis, or hepatitis. Identical gastric symptoms may usher in an attack of pneumonia. The possibilities of a mistake are to be borne in mind, and in all cases of vomiting with fever due regard must be paid to the possibility of the gastritis being merely symptomatic. The same group of symptoms that belongs to gastritis also accompanies the exanthematous diseases, as well as diphtheria, dysentery, pyæmia, and puerperal fever. The gastric symptoms may be of reflex origin, or due to the action of fever, poison, or ptomaïns on the stomach. Ewald uses the term "sympathetic gastritis" when the symptoms are the same as in the simple variety, masked, however, by the primary disease. Sometimes, however, as in the eruptive fevers, attention is directed to the state of the stomach, to the exclusion of other conditions, until, to the surprise of the student, the appearance of an eruption or inflammation reveals the true nature of the case.

In cases of gastritis, therefore, endeavor to find a local cause for the symptoms. If there is no history of indiscretion in diet, of exposure, of exhaustion, or mental shock that might have arrested digestion, then inquire for a history of exposure to contagious diseases and look for the earlier evidences of exanthemata. If the result of the examination is still unsatisfactory, examine the condition of each individual organ, particularly bearing in mind meningitis, pneumonia, peritonitis, nephritis, and the general infections.

Mycotic and *pseudomembranous gastritis* may occur secondarily to typhoid fever, pneumonia, pyæmia, and smallpox. In very rare instances actual diphtheria of the stomach has been observed. The fermentation incident to the presence of yeast-fungi and sarcinæ in dilatation causes irritation, and these organisms may perhaps irritate the viscus directly. The mucous membrane may be covered with patches in areas or throughout its whole extent. Rarely tuberculous inflammation with ulceration takes place. Many other micro-organisms are frequently present, and though they are chiefly non-pathogenic they may contribute to the trouble and often cause fermentation and consequent irritation. Klebs found *Bacillus gastricus* with numerous spores in the tubules as the cause of a gastritis.

The mucous membrane itself usually escapes infection by micro-organisms because of the character of its secretion. The acid gastric juice is antagonistic to micro-organisms and causes their death. Tuberculosis, for instance, rarely attacks the stomach for this reason.

Phlegmonous Gastritis.

This is a very rare affection, in which the inflammation is seated in the submucosa and leads to perforation. The onset is sudden. The chief local symptom is intense pain in the epigastrium, with a burning sensation. The anorexia is absolute, the tongue is dry, and the acidity is increased. The fever is high and accompanied by delirium and chills. The pulse is small, rapid, and irregular. The matters vomited first consist of mucus, then pus. The patient is extremely restless and anxious, even delirious, and early passes into coma. Death takes place from collapse. It is impossible to make an absolute diagnosis, as local peritonitis and abscess of the liver are characterized by the same symptoms. In *abscess* a tumor may form in the epigastrium. The disease may be idiopathic, but frequently occurs in septicæmia, and follows trauma. The subjects are often alcoholics.

Toxic Gastritis.

This form of gastritis is allied to the former in the severity of the general symptoms. It is the result of the swallowing of irritating poisons, of which phosphorus, arsenic, bichloride of mercury, and caustic acids and alkalies are the most common. It is attended by inflammation of the mouth, œsophagus, and stomach. The flow of saliva is excessive, the patients are unable to swallow and constantly vomit blood, which is often mixed with shreds of mucous membrane. The patient is restless,

and may have convulsions; collapse soon develops. In mild cases, in which the local effects of the corrosive substances have been mitigated by proper antidotes, sloughing takes place and ulcers remain on the mucous membrane, which, after healing, result in deformity or stenosis of the œsophagus.

Some cases are attended by other symptoms peculiar to the special poison. Thus in arsenic-poisoning there are choleraic symptoms; in phosphorus-poisoning the symptoms come on late after the ingestion of the drug, and are attended by jaundice and symptoms of acute yellow atrophy.

DISEASES OF THE STOMACH CHARACTERIZED BY INDIGESTION.

Functional Disorders of the Stomach.

The Neuroses. Functional disturbances of the stomach are due to impairment of the motor power of the stomach, and to impairment of the secretory and sensory functions. The following table of Ewald is a classification of the various neuroses midway between the symptomatic and the ætiologic:

THE NEUROSES OF THE STOMACH.

1. CONDITIONS OF IRRITATION.

a. Sensory.	*b. Secretory.*	*c. Motor.*
Hyperæsthesia.	Hyperacidity.	Eructation.
Nausea.	(Hyperchlorhydria.)	Pyrosis.
Hyperorexia.	Hypersecretion.	Vomiting.
Anorexia *ex* hyperæsthesia.		Colic.
Parorexia.		Tormina ventriculi.
Gastralgia.		

2. CONDITIONS OF DEPRESSION.

Polyphagia.	Anacidity.	Atony.
Anæsthesia.		Insufficiency of the pylorus and cardia.

3. MIXED FORMS.

Gastro-intestinal neurasthenia (dyspepsia nervosa).

4. REFLEXES FROM OTHER ORGANS UPON THE GASTRIC NERVES.

Reflexes from the brain, eyes, spinal cord, kidneys, liver, sexual organs, and intestines manifest themselves in the forms mentioned in 1 and 2.

It must not be supposed that each of the above-named symptoms occurs in an individual, or that functional disturbances may be limited to alterations in the sensory and secretory, or in the motor apparatus respectively. As Ewald remarks, they occur not as distinct independent diseases, but usually in groups, "either appearing simultaneously or closely following one another during the course of the malady, passing before us like an ever-changing scene." They may arise directly from disease of the stomach, or reflexly from disease of other organs, the brain, the spinal cord, uterus, kidneys, liver, eyes, or nose.

Ætiology. Gastric neuroses are of most frequent occurrence in women, especially during the years of sexual activity. The accidents of child-

birth are predisposing factors. In both sexes they are of most frequent occurrence after the age of twenty because individuals are subjected to causes that lead to neuroses at this period of life. The gastric neuroses occur in all classes. They are more likely to occur in those who are poorly nourished or anæmic, although persons who are distinctly robust may also suffer. While more common among the residents of cities, they may occur in farmers and others accustomed to an open-air life. Although we are oftenest called upon to treat them among the better classes, a large number of cases are also seen among the poor. To analyze more closely the predisposing causes, we have to study individually all conditions and circumstances in life that lead to wear and tear, as in business or social affairs. The causes which Beard and others have forcibly pointed out as factors in the production of neurasthenia are especially prevalent in this country.

In men, excessive devotion to business or dissipation; in women, excesses in social life or the restraint of home cares, with, unhappily too often, the irritation of marital relations, are the predisposing factors which lead to the development of this class of cases. Often patients in the large cities are subject to the neuroses in the spring after the dissipations of the winter. Behind this excess there is, no doubt, in the majority of cases, a nervous temperament that is responsible for the bringing out of the symptoms, particularly if the patients live in an unhygienic way in regard to exercise, ventilation, drainage, and diet.

Symptoms. With the gastric neuroses other symptoms of *neurasthenia* are present, which may lead the patient to seek advice: headaches of various kinds, changes in the mental condition, vertigo, insomnia, neuralgias, and all forms of paræsthesia. Intimately connected with the neurasthenic state is that of hysteria, and therefore in gastric neuroses *hysterical manifestations* are most common. It may be impossible completely to define the border-line between neurasthenia and hysteria, and the gastric symptoms of the former are the gastric symptoms of the latter. While, therefore, general neurasthenic symptoms are prominent, in order to reach a diagnosis upon which proper lines of treatment can be based, the condition of the individual must be viewed as a whole, and no one symptom or group of symptoms exaggerated in our minds.

Varieties. Ewald has divided the neuroses into those which arise from (*a*) *irritation*, those which arise from (*b*) *depression*, and (*c*) those in which both are combined—*mixed neuroses*.

a 1. **Sensory Neuroses of Irritation.** HYPERÆSTHESIA. The first result of irritation is *hyperæsthesia* of the stomach, indicated by a feeling of *fulness* and *tension*, with *nausea*. The sensation is often somewhat like the normal sense of hunger, and is also seen in chronic gastritis, as well as in hysteria, meningeal irritation, cerebral tumors, and other diseases of the nervous system. The increased irritability is such that the mildest irritant excites discomfort or a painful sensation. There is a continuous sensation of heat or cold, of gnawing, or pulling, or burning in the organ. The local sensation reflexly influences the physical life of the patient, so that *hypochondriasis* in some form attends it. The sensations may be relieved by food, to become worse when the stomach is empty, although

in the larger number of cases the trouble is aggravated during digestion. The sensations are likely to be aggravated by fasting a longer period than usual, or by restriction of the diet. Excesses may aggravate them, and, on the other hand, they may follow debilitating states. Some foods, such as shell-fish, crabs and lobsters, or oysters and strawberries, are likely to increase the peculiar sensations in the epigastrium, exciting mild depression, or burning, or even nausea. The excitation from these foods is usually due to idiosyncrasies which also are responsible for pruritus, erythema, and urticaria with headache and some elevation of temperature.

Perversions of the Sense of Hunger. *Hyperorexia.* When hunger is exaggerated, it is known as boulimia, or hyperorexia. It may be temporary or permanent. When permanent, it is obstinate, weakening, and exceedingly unpleasant. It may occur alone or be a symptom of various diseases of the nervous system, as manifest disease of the brain, neurasthenia, hysteria, and psychoses. It complicates such disorders as diabetes, and may be of temporary duration in convalescence from acute disease. The disorder may accompany migraine, hypochondriasis, and exophthalmic goitre. Analogous to it is perversion of the appetite, as seen in pregnancy, during childhood, and in mental disorders.

Anorexia. Loss of appetite, or repugnance to food. In the first instance, there is simply loss of appetite; in the second, there is repugnance to food or nausea at the sight of it. Loss of appetite accompanies all forms of dyspepsia. In the gastric neuroses anorexia occurs spontaneously, or is due to hyperæsthesia of the stomach, and may therefore arise from central or peripheral conditions of irritation. It is commonly seen following central nerve perturbation. The patient is hungry and sits down to the meal fully prepared to satisfy himself. The first mouthful is at once followed by anorexia, which may almost amount to nausea. On account of these symptoms the patient eats less and less of solid food, which soon results in disturbance of nutrition affecting the centres. Conversely, profound mental disturbance may be an exciting cause, so that after the death of a friend or shock of any kind the patient is unable to take food. Loss of appetite may be the only manifestation of the gastric neurosis, but owing to the serious interference with nutrition other local or general symptoms soon result. Fenwick points out that its relationship to emaciation and enfeeblement is such that grave organic diseases may be simulated. Thus the patient's condition may be mistaken for phthisis, and a general examination alone be sufficient to clear up the mistake.

Gastralgia. *Pain* in the stomach occurs in organic disease, as in ulcer or cancer, or forms of gastritis; but it also attends a gastric neurosis, and may be the only symptom of this neurasthenic state. Such pain is functional, and is found in anæmic, neurotic women, although it may occur in all classes. The pain is sudden, referred to the epigastrium, usually without regularity, though at times distinctly periodic. There may not be any definite relationship between the attack of pain and the taking of food, although the pain is most apt to occur when the stomach is empty. Some kinds of food may aggravate it, though, in general,

eating relieves the pain. If the epigastrium is examined, it will be found free from tenderness—indeed, pressure with the palm of the hand may give relief. The pain is of an agonizing character, sometimes sharply localized, or again diffuse. It may even resemble the girdle-sensation. On account of the severity of the pain the patient may double himself up to relax the abdominal muscles. The breathing is short, and speaking is done in a whisper. The attack is attended by more or less collapse, and the patient may complain of a sensation of impending death. There is pallor of the face, which is distorted with pain, and the brow is covered with perspiration. The pain may radiate along the spinal nerves in close proximity to the stomach, and there is often vigorous pulsation of the abdominal aorta. The attack may last but a few minutes or continue for hours. It sometimes terminates suddenly with vomiting, or is relieved as soon as food is taken. After the attack the patient is exhausted and relaxed, and passes an abundance of urine of low specific gravity.

The gastralgias that are due to disease of the central nervous system are often most puzzling. Rosenthal has written exhaustively on this subject. Types of gastralgia of this character are seen in the *gastric crises* of tabes, first described by Charcot. Recent observers have found that it is due to sclerotic degeneration of the vagus nucleus. The patient is suddenly seized with severe pains beginning in the groin and ascending along both sides of the abdomen to the epigastrium, where they remain localized. Pain in the shoulders occurs at the same time. The pains are suggestive of locomotor ataxia in their lightning-like rapidity. With the pain the heart's action is increased in rapidity and force. There is no rise in temperature. At the same time there is uninterrupted and painful vomiting, attended by nausea and vertigo. The gastric pain may continue uninterruptedly for two or three days. The frequency of the attacks is variable; they may recur at long periods, or as frequently as once a month or once a week. The symptom belongs to the pre-ataxic period, so called, but is almost sure to continue throughout the whole course of the disease. The nature of the stomach contents sometimes bears no relation to the pain, but HCl is often in excess. Another special characteristic is the sudden relief without apparent cause.

Neurasthenic Gastralgia. Neurasthenic gastralgia occurs in patients who are suffering from neurasthenia, and is divided by Rosenthal into two forms, the one irritative, the other depressant; these are connected by transitional forms. The early symptoms of neurasthenia (*q. v.*), particularly of the irritative form, with the presence of sensitive points in the nape of the neck and between the scapulæ or often lower down on the vertebræ, with neuralgias and paræsthesias in the upper and lower extremities, are attended by periodical gastralgia. The gastralgia is characterized by a boring sensation which, during the attack, radiates over the lower ribs to the median line. It is accompanied by vasomotor symptoms and symptoms of cerebral anæmia. In the *depressant* form the patient complains of weight and fulness, or a dragging sensation after eating, which is constant instead of paroxysmal. The neuralgic pains are not so marked, motor exhaustion is not so prominent, and the pain

in the back is not so intense as in other varieties. In both instances on deep pressure over the region of the nerve-plexuses that accompany the bloodvessels in the abdomen there is sharp pain radiating to the epigastrium. Burkart considers these painful points to be present in all cases, while Richter believes that pressure over the stomach and abdomen is not painful. With such pain there is usually increased pulsation of the abdominal aorta, particularly during the paroxysm. In neurasthenic gastralgias there are increased sensitiveness to the electrical current and increased irritability of the sensory nerves of the trunk, sometimes extending to the limbs.

Neurasthenic gastralgia must be distinguished from the gastralgia of organic disease and the gastralgia of hysteria. The gastralgia of organic disease is recognized by a study of the secretion when fasting. In organic disease there is retarded digestion, or the signs of ulcer are present; in gastric neuroses digestion is completed in the normal limit of time—seven hours—and there is often no disturbance of the chemical condition of the gastric secretions, though hyperchlorhydria of purely nervous origin undoubtedly occurs. Hysterical gastralgias are recognized by the presence of the usual symptoms of hysteria, in which the psychical factors occupy a prominent place, associated with convulsions, paralyses, pupillary inequalities, hemi-anæsthesia, and electrical insensibility. Most characteristic, however, is the alternation of hysterical gastralgias with neuralgia, or neuroses in other organs.

a **2. Secretory Neuroses of Irritation.** HYPERCHLORHYDRIA, or hyperacidity due to excess of HCl, is an extremely common condition. It is most frequently due to organic disease of the stomach, such as benign stenosis of the pylorus or ulcer; but it may be a pure neurosis accompanying neurasthenia, hypochondriasis, melancholia, or hysteria; or may be due to the reflex irritation of gallstones, renal calculus, and similar conditions. The diagnosis between organic disease and neurosis is often very difficult. The symptoms of hyperchlorhydria are heartburn and acid eructations, with burning or boring pains in the epigastrium, which are usually worse when the stomach is empty and relieved by taking albuminous food, but often rather aggravated by starches. Physical examination usually shows epigastric tenderness, which may be severe, and often slight or moderate enlargement of the stomach. The contents after a test-meal usually have a high acidity (60 to 120 or more), starch digestion is very imperfect, and there is an abnormally large amount of material. Many writers have noted that symptoms of hyperchlorhydria may be present when the HCl is not in excess. Strauss and his students believe that the excessive secretion in these cases is shown by a lively starch reaction and a low specific gravity (below 1010). The *diagnosis* of the nervous form chiefly concerns the distinction from acid gastritis, ulcer, and motor insufficiency of the stomach. When there is much mucus in the stomach contents, acid gastritis is easily diagnosticated and may be considered to be present when there are evident causes of gastritis. In many cases, however, it is practically impossible to say whether it is a gastritis or a neurosis, and the diagnosis must be based upon the general condition of the patient rather than upon the

local signs. Ulcer and motor insufficiency are distinguished by the signs mentioned in considering those conditions. In some cases ulcer can be diagnosticated only by observing a rapid recovery under treatment. This is very uncommon in nervous hyperchlorhydria, but it is not an absolutely distinctive feature.

Gastroxynsis is a gastric neurosis in which, after mental over-exertion or profound emotional disturbance, there is severe headache with sudden vomiting of acid fluid, continuing for a considerable time. It is closely allied to migraine, and is a form of periodical hypersecretion.

Hypersecretion is probably an accompaniment of practically all instances of hyperchlorhydria, whether of nervous or of organic origin. In ordinary cases it is recognized largely by the fact that the contents removed after a test-meal are both very acid and of large amount; often, even more than was introduced is removed. The symptoms in such cases are produced conjointly by the increase in acidity and the excess in the quantity of gastric juice. There are, however, cases in which the hypersecretion is evidently the chief factor. These were first described by Reichmann under the name of gastrosuccorrhœa, and belong under two classes: the periodical and the chronic. Periodical hypersecretion is characterized by the occurrence of general physical prostration, with more or less epigastric pain and tenderness and vomiting. The vomitus is at first food and gastric juice and perhaps mucus; later it consists of large amounts of a highly acid gastric juice, clear or mixed with bile. The attacks last from a day to two weeks, and often produce profound prostration. They really constitute a form of cyclical vomiting or gastric crisis, the distinctive feature being the high acidity of the vomitus and the great quantity of secretion produced by the stomach.

The chronic form presents usually the symptoms of hyperchlorhydria with the added feature that the patients often vomit considerable amounts of clear, highly acid gastric juice when fasting, and the stomach, when it should be empty, as in the early morning, shows the presence of from 100 to 300 c.c. of the same excessively acid gastric juice, free from food remnants. The affection is chronic and persistently present. The periodic form is apparently usually a neurosis, and the chronic form is thought to be so in some instances, but organic disease must be carefully looked for in these cases. In most instances it is due to moderate pyloric stenosis, to pyloric spasm, or to motor weakness. Doyen in particular has insisted upon the importance of erosions and ulcers in producing pyloric spasm and gastrosuccorrhœa.

a 3. **Motor Neuroses of Irritation.** Eructations. Eructations and belching are phenomena of the gastric neuroses of motor origin. They usually occur in hysterical subjects rather than in neurasthenics. In the latter they are associated with other sensations, particularly oppression and tension in the epigastrium. In hysteria they occur alone. There is increase in the contractility of the stomach, the pyloric sphincter contracts powerfully, and the stomach is distended; gas is expelled at the cardiac end of the stomach. Eructations may be due to paralysis of the cardiac end of the stomach rather than to contraction of the pyloric end. As a rule they occur involuntarily, and must not be confounded with the

pseudohysterical vomiting described by Bristowe, in which the gas is raised from the œsophagus by contraction of the muscles of the neck. Hysterical eructation is very frequently of œsophageal origin. The belching is loud and may occur in paroxysms. The gas is odorless, and hence is distinguished from the gas of dyspepsia and fermentation; it is in all probability the result of the swallowing of air.

Pyrosis. Pyrosis, or heartburn, is the raising of sour masses from the stomach. The stomach contents are not necessarily hyperacid. If the material is acid, like normal gastric juice, or hyperacid, the regurgitation causes severe acrid and burning sensations. The neurosis is probably due to heightened contractility of the muscular coat of the stomach with pyloric contraction, which overcomes the weaker cardia.

Pneumatosis. Excess of gas in the stomach. When the stomach is over-distended, the diaphragm is pushed up and presses against the heart. The patients are accordingly seized with severe dyspnœa; at first inspiration is difficult, and finally both inspiration and expiration are impeded. Palpitation of the heart and pulsation of the peripheral arteries take place. There are fulness of the head and a sensation of impending death; the patient may even become unconscious. Relief can only be afforded by belching, when the attack rapidly subsides. Introducing a stomach-tube gives immediate relief.

Nervous Vomiting. (See Subjective Symptoms and Gastroxynsis.)

Tormina Ventriculi. Peristaltic unrest. Characterized by borborygmi and gurgling, beginning immediately after eating; the sounds are heard at a considerable distance, and are a source of great annoyance. Peristaltic unrest is a common symptom of the gastric neuroses.

Rumination. Merycismus. Rumination is a rare condition in which the patients regurgitate and chew the cud like ruminants.

b 1. **Secretory Neuroses of Depression.** Anacidity. Anacidity of the gastric juice as a neurosis is found in hysterical persons and in neurasthenics. (See chemical examination—Absence of Hydrochloric Acid.)

b 2. **Sensory Neuroses of Depression.** Anæsthesia. In conditions of depression *polyphagia* or *acoria*, the want of a feeling of satiation, occurs; if gluttony is excluded, it is a morbid condition of extreme rarity.

b 3. **Motor Neuroses of Depression.** Atony, or Atonic Dyspepsia, accompanies gastritis, and occurs also as a primary neurosis. The nerve-centres regulating peristalsis are disordered. The primary disturbance of innervation may be local or central. The movement of the chyme is tardy or insufficient. Atony should be applied to disturbance of the motor function only, or, as Rosenbach states it, to insufficiency of the stomach. The symptoms develop gradually. At first oppression during digestion occurs, with swelling and fulness of the stomach.

There is mental and physical torpor during the time of the digestive act. The symptoms become aggravated, and eructations occur, vomiting begins, and gradually the fermentative symptoms become most pronounced. At this period the condition is best described by the term putrid, or fermentative dyspepsia. By the usual tests the motor power of the stomach is found to be diminished. The secretions are often reduced, though they may frequently be excessive.

Relaxations at Orifices. *Relaxation of the Cardiac and Pyloric Ends of the Stomach from Conditions Resembling Paralysis.* When the cardiac end is relaxed, eructations and regurgitations occur. If large quantities of the material from the stomach are regurgitated and expectorated, the condition is pathological, and may lead to serious disturbance of nutrition. The condition may exist for years without producing bad results. It must not be confounded with the regurgitation from diverticula of the œsophagus.

The relaxation is often the result of repeated voluntary regurgitation in hypochondriasis. Relaxation of the pylorus is said to cause rapid discharge of food into the intestine and a consequent lienteric diarrhœa. The diagnosis is always questionable.

c **Mixed Neuroses.** Nervous Dyspepsia. According to Ewald, this is the true gastric neurasthenia, which combines all forms of gastric neuroses. The clinical picture is made up of a combination of various neurosal symptoms. Leube considers nervous dyspepsia a group of symptoms of a cerebral nature due to abnormal irritability of the sensory nerves of the stomach during the normal digestive processes, and consisting of hyperæsthesia and nausea, hyperorexia, anorexia, parorexia, and gastralgia. The secretory power of the stomach varies greatly, being sometimes normal, sometimes increased, and sometimes subnormal or absent. Although the anatomical or physiological explanation of the condition is difficult, the clinical symptoms are those of irritation or paralysis, the manifestations of which are intermingled, sometimes one and sometimes the other being most prominent. (See table, page 1018.)

The one characteristic feature is that the symptoms are mild. Nervous vomiting and boulimia do not occur with severe forms of gastralgia. Symptoms of intestinal indigestion are usually associated in a mild degree. Constipation is of the most common occurrence, although in some cases there is diarrhœa. In other cases the intestinal indigestion is much aggravated, with mild gastric disturbances and anorexia, distaste for food, furred tongue and mild nausea, constipation, and colicky pain, either diffuse or in separate painful spots. The abdomen is distended and tympanitic, sometimes to a marked degree. It is called *flatulent dyspepsia.* Along with the gastric and intestinal symptoms, the general nervous symptoms to which the term neurasthenia is applied are present. These nervous manifestations sometimes precede the local gastric symptoms, but as the latter develop the former become more aggravated. The dyspeptic conditions, as Ewald puts it, rest on a neurotic basis, or are such as may occur in the form of reflex neuroses in chlorosis, menstrual disorders, uterine and ovarian disease, and after intense physical or psychical excitement. It is impossible to say to what extent the symptoms depend upon anatomical changes and alterations of secretion. Cases have been described in which severe digestive disturbances were present for as long as ten years and yet post-mortem examination showed no anatomical changes. The general teaching, however, is that in most cases, even though alterations of structure or function may be absent in the beginning, they appear later as a result of the neurasthenic disturbance.

Diagnosis. There are no characteristic symptoms, and the student

must bear in mind that it may be necessary to make several examinations and listen to the story of the subjective symptoms frequently before a conclusion can be arrived at. This is all the more necessary because of the frequency with which organic lesions and neurasthenic conditions are present at the same time. The course of the disease must be observed for a long time, all possible causal factors investigated, and all the general signs of neurasthenia carefully considered. In addition, it may be necessary to use therapeutic tests. If the possible organic diseases are not relieved by such measures, there must be a deeper basis for the gastric symptoms. Just as in neurasthenia and neurasthenic states elsewhere, the peculiarities, idiosyncrasies, and all the associations in the life of the individual must be considered in connection with the general and local symptoms of the neurasthenic state. Great stress must be placed upon the study of individual symptoms, their mutual relationship, and their changeable occurrence. In gastric neurasthenia gastralgia is more diffuse than the pain of ulcer or cancer of the stomach. It is not so much dependent upon ingestion of food as either of the other forms, particularly that which accompanies ulceration. In gastric neurasthenia vomiting is rare; when it occurs, the material is composed of mucus mixed with bile and food in various stages of digestion, and is never bloody, nor does it contain decomposed masses. Hysterical vomiting occurs with ease and regularity as compared with that of neurasthenia, in which the vomitus is bitter from the presence of peptones. The stools in gastric neurasthenia are changeable in character, and do not contain undigested remnants of food, or mucus, or blood, while the shape of the feces is variable.

Differential Diagnosis. Neoplasms, ulcers, strictures, and dilatation are distinguished by physical signs or characteristic symptoms. In gastric neurasthenia the stomach should be empty seven hours after taking a meal. The results of the chemical examination are not sufficiently definite for diagnostic purposes; for at times the same chemical changes are present as in ulcer, carcinoma, and chronic catarrh. The diagnosis, as previously intimated, is based chiefly on prolonged observation and a carefully taken history, and on the general condition of the patient. The cases must not be mistaken for costal neuralgia, although it is not often that one is led astray. Reflex gastric neuroses as indigestion, gastralgia, or vomiting are seen. The types are interchangeable, although vomiting occurs in the more acute reflexes, indigestion in the more chronic. The cerebral disorders that give rise to vomiting are meningitis, abscess, and tumor. The vomiting may be transitory or persistent, and there is usually hypersecretion of the gastric juice; the vomiting may usher in the disease or develop during its course. When vomiting is of long standing, the possibility of a reflex origin should always be borne in mind. (See Vomiting.)

Gastralgia is sometimes a reflex symptom of a lesion in the cervical and dorsal portions of the cord, not only in tabes dorsalis, but also in disseminated sclerosis; the attack of pain and vomiting is known as a *gastric crisis.*

Chronic dyspepsia is frequently the expression of reflex irritation in

diseases of the sexual organs, as amenorrhœa and dysmenorrhœa during the climacteric period, and in chronic inflammations of the uterus. Malpositions and tumors, pelvic exudates with traction, ulcers, and ovarian tumors, are often attended with the so-called *dyspepsia uterina* of Kisch.

Chronic Gastritis.

Causes. 1. Previous attacks of acute gastritis.

2. The local irritation of badly cooked or poorly masticated food, and of alcoholic and other beverages.

3. The local irritation of urea in chronic Bright's disease, and of products of putrefaction in constipation.

4. Chronic gastritis is of frequent occurrence in anæmia and in venous congestion from any cause, but particularly from disease of the heart or diseases that interfere with the portal circulation. It occurs secondarily to diabetes, gout, rheumatism, nephritis, and tuberculosis.

5. It is a constant attendant upon local disease of the stomach, as cancer, dilatation, and ulcer, and of local disturbance of the circulation.

6. Neurasthenia is undoubtedly a prolific cause of chronic gastritis. It may produce only atony or functional disturbance at first; but these ultimately lead to gastritis.

The symptoms are those of *chronic indigestion.* There is a dry, pasty, or salty taste in the mouth, especially in the morning. The tongue is coated over its entire surface, or has red patches at the base; its papillæ are always swollen and its edges marked by the teeth. Aphthæ recur frequently. The lips are dry and often chapped. The appetite is diminished or capricious. Although there is no great thirst, the patients crave fluids with their meals, and acid drinks are grateful. After eating there is a feeling of oppression and distention in the epigastrium, frequently followed by belching. The gaseous eructations may be odorless or foul; rancid regurgitation with pyrosis is frequent. The acidity is due to fatty acids and not to hydrochloric acid, as in hypersecretion. Vomiting is frequently present, but occurs irregularly, and is usually preceded by nausea. The most characteristic form is that in which mucus is vomited in the morning on rising. Constipation usually exists; it may alternate with diarrhœa. There are flatulence and rumbling in the intestines.

General Symptoms. The nervous symptoms are the most pronounced. The mental activity is diminished, there is a feeling of languor or torpor, especially after eating. Headache is frequent after eating, and the patient may become morose and hypochondriacal. Attacks of vertigo are common. Itching of the skin and coldness of the extremities are not rare. Sleep is deeper and longer than is natural, but is disturbed by dreams, and is not refreshing. Yawning is frequent. Pharyngitis is usually present, with hacking cough and expectoration, or the hawking of mucus. The pulse may be weak and irregular, and at times there is an evening rise of temperature. The urine is scanty, high-colored, and usually loaded with urates.

Three chief forms are seen: (1) subacid gastritis; (2) acid gastritis; (3) atrophic gastritis. The last is likely to result from long-standing

cases of the other forms and constitutes the organic form of the condition called by Einhorn *achylia gastrica*. It is important to distinguish this condition from the neurotic form of achylia. In both there is entire absence of acidity and of digestive ferments, and the test-meal is returned unchanged in appearance except for some maceration. The general health is not infrequently unaffected in both varieties; but this is more commonly the case in the neurotic form, while the organic cases are characterized by absence of neurasthenic characteristics, a prolonged history of gastric disturbance and often severe intestinal symptoms, chiefly diarrhœa. Severe anæmia is often present and the general depression of health may be extreme. The diagnosis from cancer may be very difficult in these cases. It rests chiefly upon the absence of lactic acid, of Oppler-Boas bacilli, and of pus from the stomach contents, the lack of evidence of tumor or of pyloric obstruction, and the absence of leucocytosis and actual cachexia.

In *subacid gastritis* the stomach contents contain little or no free HCl, but some of the combined acid, the test-meal is usually poorly digested, and volatile acids are present. In one form called "mucous gastritis" large amounts of mucus are found in the contents after a test-meal. Acid gastritis is a form that is not always recognized, but which certainly exists. It is characterized by the usual symptoms of gastritis with those of hyperchlorhydria superadded, and the contents after a meal are found to be excessively acid. The frequent presence of excessive amounts of mucus, the history of a cause, and the general character of the patient stamp the affection as a gastritis and not a neurosis.

Diagnosis. The diagnostic features of chronic gastritis are: first, long duration; second, persistence of local symptoms; third, recurrence of local symptoms after food, the symptoms being aggravated by stimulants or stimulating food; fourth, moderate pain; fifth, absence of cachexia; sixth, absence of tumor; seventh, flatulence. Hemorrhage is rare and slight when it occurs; there may or may not be vomiting; while the quantity of hydrochloric acid is variable. Finally the cause is usually definite.

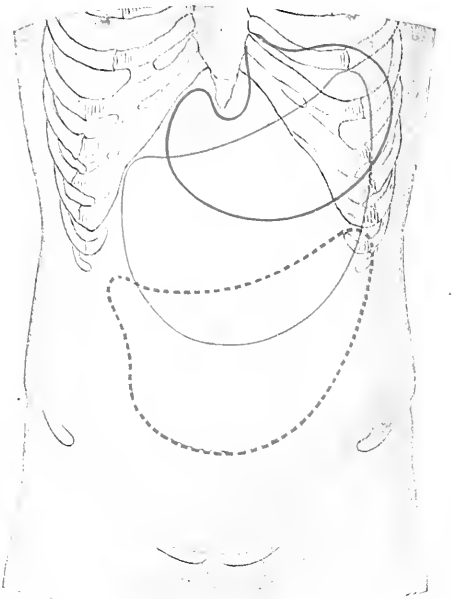
Dilatation of the Stomach (Gastrectasia). (See Plate XL., Fig. 1.)

Dilatation of the stomach is caused by obstruction at the pyloric orifice, either from cancer, the cicatrix of an ulcer, or fibrous stricture. In a mild form, so-called "atonic dilatation," it certainly follows the relaxation and degeneration of the walls of the stomach that occur in chronic gastritis and in the conditions causing chronic gastritis. It may attend paralysis of the stomach. Excessive eating and drinking are the only probable causes independent of organic disease. Hence we have (1) obstructive and (2) atonic dilatation.

The dilatation may be *acute*. The term *acute paralytic distention* is also applied to this condition. The cases are by no means so rare as was once thought. They follow blows or operations upon the abdomen, or occur as a result of the relaxation and distention of the walls that appear

PLATE XL.

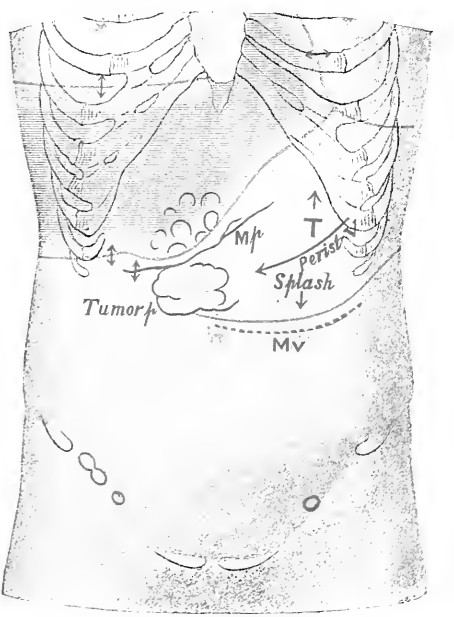
FIG. 1.



Normal Position and Displacements of the Stomach.

Solid red line—Normal position of distended stomach.
Blue line—Atonic dilatation.
Dotted red line—Gastroptosis.

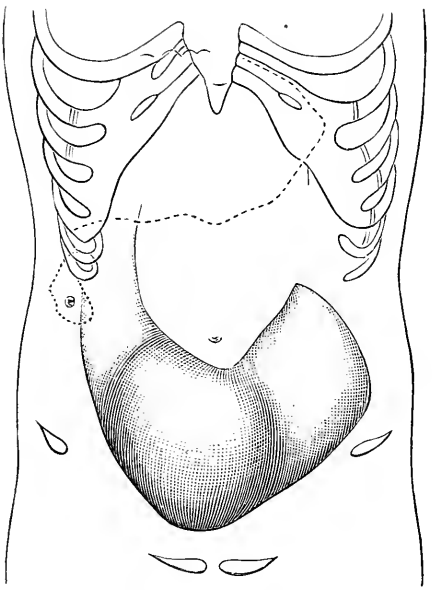
FIG. 2.



Carcinoma of the Stomach with Pyloric Stenosis.
Metastases in the Liver.

in acute diseases. There is sudden enlargement of the upper portion of the abdomen, with pressure upon the surrounding structures; the heart is dislocated and its action much interfered with; collapse follows, and may end in death. At first there may be some belching, but the patient is soon unable to remove the gas, and suffers from extreme discomfort, palpitation, and dyspnœa. The vomiting may occur at once or later. It is persistent and excessive. On physical examination the stomach yields the same physical signs as in chronic dilatation.

Fig. 369.



Dilatation of stomach.

Chronic dilatation develops slowly. The *symptoms* are superimposed upon the causal disease. There is marked dyspepsia, with flatulence, pyrosis, and other symptoms of fermentation. The tongue is pale and furred, or red, smooth, and shiny; or it may be soft and flabby. If frequent vomiting has attended the causal disease, it now occurs at longer intervals; the amount is excessive, greater than the normal stomach would hold, and is made up of partially digested and fermented food and large amounts of mucus. The stomach contents contain sarcinæ, torulæ, and other products of fermentation, and have a disgustingly foul and sickening odor. In atonic dilatation the HCl is usually diminished; in pyloric obstruction it is almost always much increased. In health large amounts of volatile acids are present, but lactic acid, except when introduced with the food, is practically always absent. The stomach contents when allowed to stand separate into three layers—the upper frothy and containing mucus and fermenting food; the middle layer clear and watery; the lower finely divided and consisting of more or less completely digested food. The patient loses flesh and strength; becomes irritable, depressed,

and more or less melancholy; and is subject to vertigo and to attacks of nocturnal asthma. The nervous symptoms of chronic gastritis are also present. Sleeplessness is quite common. In some cases there is excessive thirst because of the small amount of nutriment and fluid absorbed. Cardiac palpitation and irregularity are common, and dyspnœa may occur on account of the distention. Tetany has been observed in cases of dilatation, especially after lavage.

Physical Examination. The diagnosis is not complete without *physical examination.* On *inspection* the abdomen appears large and prominent, and the outline of the stomach can sometimes be seen. Peristaltic movements of the organ are often visible. The movement is from left to right. The heart is lifted upward. On *palpation* the peristalsis can be felt, and with one hand on the stomach, tapping with the other, a *splashing sound* can be elicited. Or the hand may be placed over the stomach while the patient is standing, and the body quickly shaken. On palpation the striking or pushing hand should be compressed over the false ribs. A tumor can sometimes be felt in the region of the pylorus, or below the umbilicus. On *percussion*, when the stomach contains gas, a tympanitic note is heard. After the patient has taken a glass of water, dulness may be detected between gastric and intestinal tympany when the patient stands up. The dull note disappears when he resumes the recumbent posture. While the stomach is empty tympany is not so marked in the upright as in the recumbent posture, because the viscus is dragged back or down. The tympany extends high up in the chest on the left side, so that Traube's half-moon space becomes enlarged, and may extend as high as the fourth interspace on the left side. Cardiac dulness is increased and the apex of the heart is lifted upward and to the left. In the axillary region the tympany may extend as high as the sixth rib, so that unless very careful light percussion is performed, the splenic dulness can not be brought out. The lower limit extends below the transverse umbilical line, and may even extend midway to the pubes. If there is *gastroptosis*, the stomach tympany falls to a lower level. On *auscultation* succussion can easily be elicited. Sometimes the sound is sizzling, as if there was effervescence. Heart-sounds may be transmitted clear and metallic over the tympanitic stomach. With *auscultatory percussion* the border of the stomach can often be defined accurately. Percussion must be commenced far away from the stomach-limit and conducted toward it. (See Examination of the Abdomen.)

The most important point is to determine whether the case is one of atonic or of obstructive dilatation. The chief distinctive features of the former are less marked pain, less violent vomiting, absence of peristaltic waves; the return flow of water in washing the stomach is slow and forceless, the dilatation is usually less marked, and the health is not so profoundly affected. In pyloric obstruction there is often a previous history of ulcer or a small mass may be felt at the pylorus, peristaltic waves are frequently seen, the vomiting is forcible, and the return flow through the tube is very strong. The stomach is usually more markedly dilated, and there is more pronounced retention, while the general health suffers severely, the skin becomes dry and wrinkled, the patient loses

flesh, has a good deal of pain, usually most pronounced at night, and falls into a condition of inanition.

Stenosis of the Pylorus.

Usually, obstruction is caused by malignant disease. Hypertrophic stenosis occurs in rare instances and leads to dilatation, as indicated above. The condition may be congenital or acquired.

Acquired stenosis may be the result of chronic gastritis, or develop independently, sometimes as part of a general proliferation of connective tissue.[1] If to the physical signs of tumor of the pylorus be added the signs and symptoms of dilatation, we have the clinical picture of hypertrophic stenosis of the pylorus. It is extremely rare to find complete obstruction.

Congenital hypertrophic stenosis, as Metzler and Caudley point out, has for its characteristic features: (1) vomiting, occurring without apparent cause and persisting in spite of treatment; (2) the absence of bile from the vomited matter; (3) obstinate constipation; (4) marasmus; (5) the presence of a tumor in the region of the pylorus; (6) the absence of abdominal distention except from dilatation of the stomach itself in some instances; and (7) the absence of signs or symptoms of gastritis and of the more common forms of intestinal obstruction. The diagnosis depends entirely on the characteristic symptoms arising during the first few weeks of life and on the presence of a tumor.

DISEASES OF THE STOMACH CHARACTERIZED BY PAIN AND VOMITING.

Cancer of the Stomach.

The clinical symptoms are varied. Gastric cancer may occur without any symptoms whatever, and be discovered after death from other causes. On the other hand, general marasmus and cachexia may be present without local symptoms. In some cases the gastric symptoms are slight and obscured by the symptoms of secondary growth in the liver or peritoneum.

Some cases of carcinoma may give a history pointing to prolonged chronic gastritis, but the most common and distinctive feature is a sudden onset of gastric symptoms without evident cause in a person beyond middle life. Loss of appetite is marked, and in spite of careful treatment there is loss of flesh and strength. The vomiting gradually becomes more frequent. The general appearance of the vomitus is at first like that of chronic gastritis. Soon it becomes streaked with blood, or a moderately large hemorrhage may take place. The vomited matter is often dark in color, like coffee-grounds in appearance. The relation of vomiting to the time of taking meals depends upon the seat of the disease. If it is at the cardiac end of the stomach, the vomiting may take place at once; if in the greater curvature, within twenty minutes or one hour and a half after taking food; if at the pyloric orifice, the

[1] See author's case, Phila. Path. Soc. Trans., 1881–83, vol. xi., p. 216.

vomiting is delayed several hours. As the disease advances and obstruction becomes more complete at the cardiac orifice, food is immediately regurgitated, unless secondary dilatation of the œsophagus takes place. When there is gastric dilatation, the vomiting may take place at longer intervals and be characteristic of the vomitus of dilatation. Constipation is the rule.

Tumor. After the symptoms of chronic gastritis have continued for some time without relief, a tumor may be detected if it is sufficiently large and is situated favorably for examination. (See Tumors of Abdomen.) If the growth is situated at the cardiac orifice of the stomach or on the lesser curvature, it is often impossible to detect it; if at the pyloric orifice, the tumor is found to the right of the median line above the umbilicus, but may be forced down by the weight of the stomach and felt at the umbilicus. (See Plate XL., Fig. 2.) When dilatation follows pyloric tumor, it may be still lower down, as in a case of the writer's, in which it was found two inches below and to the right of the umbilicus. In tumor of the greater curvature the mass is detected below the margin of the ribs on the left side, and may be as low down as the umbilicus. If the greater curvature is involved, the organ may contract, and hence the physical signs indicating the lower border of the stomach are found higher up than in health.

It is necessary to exclude tumors due to other causes. This is sometimes difficult—indeed, so far as the location and physical characters are concerned, often impossible. The most pronounced diagnostic feature of tumor of the pylorus is the occurrence of secondary dilatation of the stomach. For a differential diagnosis of tumors in this region, see Palpation of Abdomen.

Symptoms due to Metastasis. The *liver* is the most frequent seat of secondary growths. The organ enlarges, and its surface becomes covered with nodules. (See Plate XL., Fig. 2.) Jaundice occurs in rare instances. The enlarged liver may cover the stomach and hide the local mass. The *inguinal* glands may enlarge. At times there is enlargement of the *supraclavicular glands*, suggestive also of intra-abdominal carcinoma from other causes.

The general symptoms are those of *emaciation* and *cachexia*. The *emaciation* is extreme, and in some cases may be out of proportion to the local symptoms.

The symptoms of *cachexia* are those of emaciation and anæmia. The *anæmia* becomes profound. The pallor of the face is striking; often it is of a yellowish and straw-colored hue. It must not be confounded with jaundice—examination of the conjunctivæ is usually sufficient to distinguish the two. The skin is flabby, and the subcutaneous fat is entirely lost; the emaciation is not so marked as in cancer of the œsophagus, except when there is complete cardiac stricture. The nutrition of the skin suffers, boils are common, and ulcers may occur in the terminal stages. Subcutaneous hemorrhages are seen on the backs of the hands, and feet, and on the legs and arms. There is slight œdema over the ankles.

General atrophy of the internal organs takes place, so that the heart

becomes small; it loses its strength; the patient becomes weaker and weaker, the pulse rapid and feeble.

If *fever* occurs in the course of the disease, it is usually due to secondary accidents, as suppuration in a tumor, or perforation with septic peritonitis. The usual course of the temperature is normal until the latter stages, when it is subnormal.

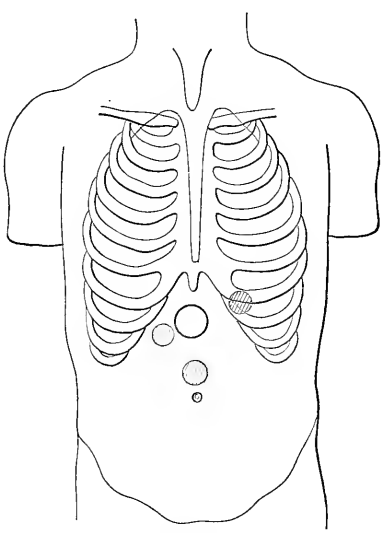
Differential Diagnosis of Gastric Cancer, Gastric Ulcer, and Chronic Gastritis. (*Welch.*)

Gastric Cancer.	Gastric Ulcer.	Chronic Catarrhal Gastritis.
1. Tumor is present in three-fourths of the cases.	Tumor rare.	No tumor.
2. Rare under forty years of age.	May occur at any age after childhood. Over one-half of the cases under forty years of age.	May occur at any age.
3. Average duration about one year, rarely over two years.	Duration indefinite; may be for several years.	Duration indefinite.
4. Gastric hemorrhage frequent, but rarely profuse; most common in the cachectic stage.	Gastric hemorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	Gastric hemorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach; lactic acid present.	Free hydrochloric acid usually present in excess in the gastric contents.	Free hydrochloric acid may be present or absent.
7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	Absent.	Absent.
8. Secondary cancers may be recognized in the liver, the peritoneum, the lymphatic glands, and, rarely, in other parts of the body.	Absent.	Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less relieved by vomiting, and less localized than in ulcer.	Pain is often paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localized than in cancer.	The pain or distress induced by taking food is usually less severe than in cancer or ulcer. Fixed points of tenderness usually absent.
11. Causation not known.	Causation not known.	Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement, or only temporary improvement, in the course of the disease.	Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Examination of the Stomach Contents. Hydrochloric acid is absent in most instances, its absence depending largely but apparently not entirely on an associated atrophy of the gastric tubules. Lactic acid, on the other hand, is commonly present even in the earliest stages, and when associated with absence of HCl is very diagnostic. For an accurate diagnosis repeated examinations must be made. (See page 595.) Other general and local conditions, as fevers on the one hand, or dilatation on the other, are attended by occasional absence of hydrochloric acid; in carcinoma it is the persistent absence of HCl and presence of lactic acid that are diagnostic. Other important signs are the presence of Oppler-Boas bacilli and, according to Strauss, of pus. Pepsin is usually diminished in this as well as in most other conditions almost in direct proportion to the diminution of HCl, while the milk-curdling ferment disappears last of all.

The Urine. Indican is increased in amount, acetone and diacetic acids may be present in the urine; otherwise there is no change.

FIG. 370.



Site of pain in gastric ulcer is marked at ○.

Diagnosis. In the diagnosis of gastric cancer the following must be borne in mind: 1. The age of the patient. 2. The occurrence of causeless dyspepsia without relief. 3. Rapid loss of flesh and strength, with cachexia. 4. The occurrence of pain in the epigastrium, continuous, increased by food, but not relieved by vomiting as in ulcer, and not distinctly localized. 5. Tumor—hard, circumscribed, followed by the physical signs of dilatation, if at the pylorus. 6. Vomiting is necessarily associated with the taking of food, in which fragments of cancer may be found; blood-cells are common; they may be detected on microscopical examination, or by the test for hæmin. 7. Examination of stomach contents: (*a*) except in dilatation the fasting stomach is empty; (*b*) hydro-

chloric acid is often absent, whereas lactic acid is present; (c) delayed absorption is present, indicated by motor tests. 8. Hemorrhage. In small amounts, usually of characteristic, coffee-grounds appearance. 9. Metastases—above the left clavicle, in the liver, in the inguinal glands, rarely in the lungs and peritoneum. 10. Eichhorst speaks of persistent itching of the skin and insomnia as characteristic symptoms. 11. Finally, the comparatively short duration of the case; rarely does it extend over a period of more than two years.

Cases of cancer of the stomach may present only symptoms of anæmia. In this manner the disease has been confounded with *pernicious anæmia*. The most important distinction seems to be, as pointed out by Henry, that in cancer the red cells never fall below 1,500,000 per cubic millimetre, while in pernicious anæmia they practically always do at some stage of the disease. The diagnosis is, however, at times almost impossible.

Ulcer of the Stomach.

Simple round ulcer of the stomach may occur at any age, but is most common in young anæmic women. It may be the result of an erosion of hemorrhagic infarcts by the gastric juice.

Symptoms. The symptoms are variable. The cases have been divided by Welch into four classes: (1) those in which there are no symptoms whatever, the ulcer being found after death from other diseases; (2) no symptoms until the sudden occurrence of hemorrhage or perforation; (3) the symptoms of chronic gastritis or gastralgia only; (4) typical cases, with the characteristic symptoms, *pain*, *hemorrhage*, and *vomiting*. The symptoms of gastric ulcer may develop suddenly.

Pain. The pain is localized; it is usually confined to a small area in the epigastrium. It may be seated behind the cartilages of the sixth and seventh ribs, or may be complained of in the back, between the eighth and ninth dorsal, and extending as low down as the first and second lumbar vertebræ. It is of a burning or gnawing character, is increased by eating, and comes on in from two to ten minutes after the ingestion of food. It is relieved by vomiting, or after the act of digestion is completed; but a persistent dull pain or a feeling of soreness remains. In addition to the ordinary pains, there may be attacks of gastralgia. The pain is increased by pressure. It may be modified by the position of the patient. It may be relieved by lying on the back when the ulcer is in the anterior wall; or by lying on the abdomen when in the posterior wall. Boas has pointed out that there is in many cases, beside the extreme epigastric tenderness, a point of great tenderness in the back, on the left side near the spines of the tenth to the twelfth dorsal vertebræ.

Vomiting. Vomiting occurs shortly after the ingestion of food. It is not attended by retching. The vomited matter may contain blood.

The vomited matter and the contents of the stomach contain hydrochloric acid, which may be in excess. Eichhorst thinks it is always in excess.

Hemorrhage. Blood in the vomitus gives the latter a brown or reddish color. It may be detected by the usual methods. Hemorrhage may

occur, however, independently of the act of vomiting. It varies in amount from half a pint to a quart. It may be so severe as to cause collapse. Sometimes, instead of being discharged as a profuse hemorrhage, the blood may gradually ooze from the ulcer and collect in the stomach before being vomited. It is then altered by the acid gastric juice. Sometimes the blood is not vomited, but passed in the stool, which is then tarry. Tarry stools also follow the vomiting of blood. In the course of ulcer a hemorrhage may be so severe that death takes place before vomiting occurs. The stomach is then found to be filled with blood.

The stomach bougie should not be used after hemorrhage or when there is good reason to suspect the existence of an ulcer; the nature of the contents must be determined by an examination of the vomited matter.

It is always very important to determine that blood supposed to have been vomited has not come from the lungs. In the latter case the blood is usually bright red and frothy, the lungs contain moist râles, and usually show some of the physical signs of tuberculosis.

A blood-count in cases of ulcer often shows a chloro-anæmia, particularly after hemorrhage. This is not distinctive of ulcer, however, as it is seen always in chlorosis and in many cases of phthisis, secondary syphilis, and in other conditions.

General Symptoms. In a case of long standing, the face is anxious and the lines are sharpened. If there is much hemorrhage, anæmia ensues. There is not much wasting and no fever. Chronic dyspepsia and constipation may be present during the intervals in which the severe symptoms are in abeyance. The period of abeyance varies in duration, and the symptoms may come on without cause, as in gastric crises, during which time the vomiting may persist for two or three days. I saw a girl of twenty years with most severe gastric hemorrhage and classical symptoms of ulcer. With careful treatment she improved. After marriage she remained well until pregnancy. During the first periods of this condition vomiting was extreme; it then subsided, whereupon, without warning, a gastric crisis took place. The vomiting of blood continued for many days, and the symptoms of gastric ulcer remained for a month.

One of the characteristic features of the disease is the recurrence of symptoms after a long period of abeyance. A patient under my care during the last ten years has had three undoubted attacks. It is possible that during each period ulcers healed, to be followed after a time by the development of new ulcers.

Diagnosis. The diagnostic features are: 1. The age. 2. The long duration. 3. The occurrence of emaciation up to a certain point only; most of the patients are underweight and have a gaunt look, particularly males. 4. The characteristic pain. 5. The vomiting. 6. The hemorrhage. 7. The periods of relief from symptoms. 8. The absence of marked nervous symptoms which attend gastric neuroses. 9. The absence of dilatation of the stomach. 10. The hyperacidity of the gastric juice.

The Accidents of Ulcer of the Stomach. 1. The occurrence of perforation. Sudden severe pain, with collapse. The pain is usually in

the epigastrium, but may be in the back as high as the seventh or eighth dorsal vertebra.

2. Hemorrhage, which may cause death immediately, with either vomiting of blood or retention in the stomach.

3. With healing of the ulcer, stenosis at the pyloric orifice may take place, with subsequent dilatation of the stomach.

Syphilis of the Stomach.

Until recently syphilis of the stomach has been generally considered to be extremely rare, but records of its discovery post mortem by Flexner and others, and of well-authenticated clinical cases, chiefly reported by Dieulafoy, Einhorn, and Dalgleish, have lately been so numerous as to make it evident that it must always be held in mind in cases of ulcer that are rebellious to treatment, or in the presence of a mass that does not show distinct characteristics of cancer. Syphilis, clinically, usually presents the signs of ulcer or of a mass which is likely to be mistaken for carcinoma. The diagnosis of syphilitic ulcer depends upon a history of syphilis, or the presence of other signs of the disease, the lack of success with the usual treatment and the rapid results with specific treatment.

The distinction between syphilis and cancer can be made only tentatively until the effect of treatment is seen. The main points are a history of syphilis, absence of a decided cancerous cachexia, and the effects of treatment.

Einhorn has described cases of syphilitic stenosis of the pylorus in which the diagnosis was made by the points mentioned.

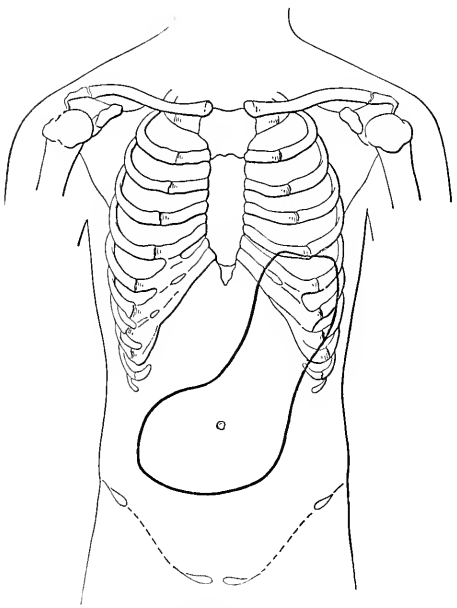
Gastroptosis.

Although downward displacement of the stomach from its normal position in the epigastrium forms part of the general symptom-complex known as enteroptosis (*q. v.*), Glénard's disease, or descent of all the abdominal viscera and one or both kidneys, competent authorities have recently cast a doubt on the propriety of assuming displacement of the colon in all cases in which the existence of gastroptosis has been demonstrated by inflation, unless the intestines themselves have been distended and thus shown to be involved in the general descent of the abdominal contents. A good deal of study has recently been bestowed on the subject, notably in this country, with the result of showing the great importance of recognizing the condition, and the possibilities of rational therapeutic measures for its relief.

Etiology. A number of theories have been advanced, some to be speedily discarded, to explain the causation of gastroptosis. Dismissing those that have been found untenable, such as the presence of adhesions between viscera, primary descent of the colon—coloptosis, congenital predisposition, which play a certain part but fail to explain more than a small proportion of the cases, it may be stated that the underlying causal condition is "anything that constricts the thorax and enlarges the abdominal cavity" (Steele and Francine), in other words, pressure on the

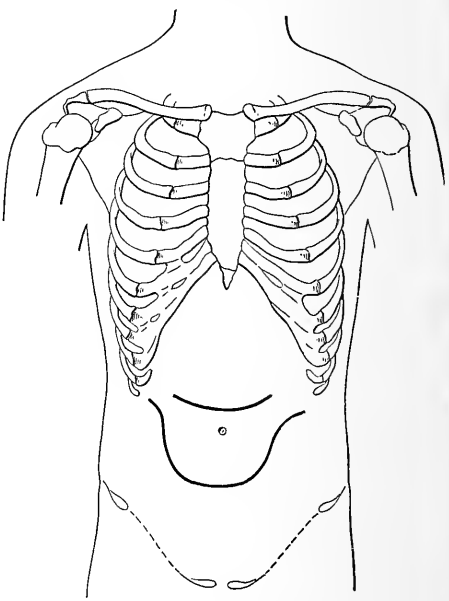
stomach from above, or removal of the support from below by the sinking down of the contents of the lower half of the abdomen. A variety of factors may bring about such a state of affairs: tight lacing and the pressure of the waist-band, pressure from a neoplasm of the liver, dilatation and pyloric obstruction, general muscular debility and emaciation after an acute systemic disease, relaxation of the abdominal walls and diastasis of the recti (Webster) after repeated pregnancies, and finally, traumatism and sudden muscular over-exertion. Steele divides the cases into primary gastroptosis with pyloric dilatation and secondary gastroptosis with general dilatation, the gastrectasis in the latter group being probably the basal condition.

FIG. 371.



Dilatation with pyloric obstruction.

FIG. 372.



Gastroptosis, with general dilatation.

It is evident from the causes enumerated that women are the chief sufferers from the disease; but as the available statistics are hardly numerous enough to furnish a trustworthy basis for calculation, and the condition may, in addition, exist without producing symptoms, the incidence is difficult to determine. The proportion as regards sex is probably about 7 to 1 in favor of women, except in the secondary and much rarer form, gastroptosis with general dilatation, in which the relation is reversed. The disease is most common in early adult and middle life; but the fact that a few cases in children have been reported by Hemmeter would seem to indicate that more careful physical examination might result in a considerable lowering of the age limit. Persons whose occupations require them to stand for a number of consecutive hours, as saleswomen, and motormen and conductors on trolley-

ears, the constant jarring of which is possibly a contributory factor, appear to possess a special tendency to the disorder.

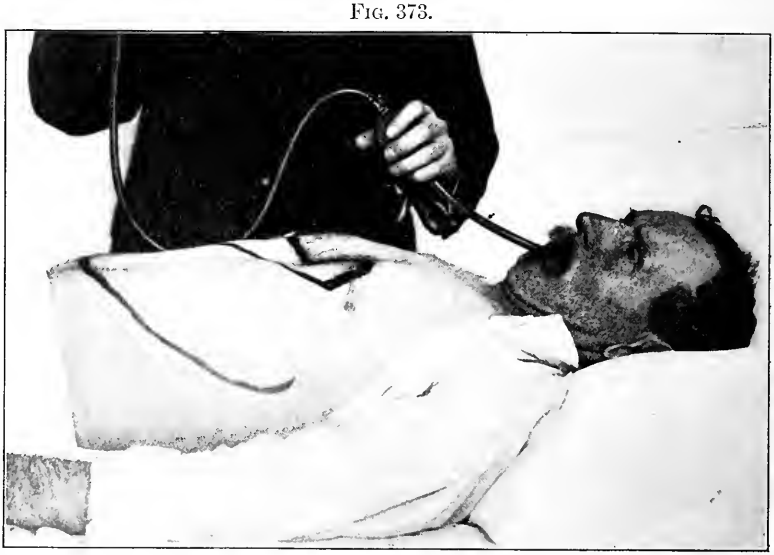
Symptoms. It is to be remembered that gastroptosis may exist without producing any subjective symptoms, its presence being discovered accidentally by a routine physical examination or at the autopsy. In many cases the dominant features are those of the accompanying neurasthenia, which should be interpreted as secondary to the anatomical disturbance, as undoubted cases of gastroptosis have been reported by competent observers, especially in men, in which there was no suspicion of neurasthenia. Sooner or later in the course of the disease the effects of dilatation and deficient motor power make themselves felt, and it is for the relief of symptoms pointing to these conditions that the patients usually present themselves. They complain of fulness in the epigastrium, distress and flatulence after meals; nausea, eructations of gas, and pyrosis, sometimes going on to palpitation of the heart and dyspnœa; headache, malaise, somnolence, inability to work and other symptoms of auto-intoxication. Pain is not a prominent or even a constant feature, and when present is probably due to some morbid process within the stomach; one theory that has been advanced is that it is the expression of an insult to the solar plexus. In cases marked by extreme dilatation with fermentation of the gastric contents, the overloaded viscus may seek relief in vomiting; otherwise, however, that symptom is not as a rule complained of. Finally the patients complain of a train of symptoms that strongly resemble the familiar features of uterine displacement, a sense of dragging and weight with sacral pain, which not infrequently are attributed to some gynecological disorder. According to Steele, chronic jaundice is an occasional accompaniment of gastroptosis and appears to be due to the presence of adhesions causing kinking and obstruction of the bile-ducts.

Physical Signs. The patients are usually emaciated and more or less anæmic, the chlorotic type predominating. The frequent association of chlorosis and gastroptosis led Meinert to the theory that there is a causal connection between the two conditions, and that all cases of chlorosis are primarily caused by descent of the stomach, but the theory is now regarded as untenable. The belly in most cases is flat; when the relaxation, which is quite characteristic, is extreme, the abdomen may be pendulous; in men, however, the tissues of the body may be quite firm. The epigastrium is flattened and there is a prominence somewhere below the umbilicus corresponding to the abnormal position of the stomach. The pulsations of the abdominal aorta can usually be felt with ease and may be perceptible to the patient. The transverse band that is felt running across the abdomen is now generally believed to correspond to the pancreas: indeed, its identity with that structure was recently demonstrated at an operation by W. F. Hamilton. Other signs of enteroptosis, Glénard's belt sign, diastasis of the recti muscles, and others that will be referred to in the proper place, should be looked for.

Inflation. For a positive diagnosis of gastroptosis the physical demonstration of the gastric area in an abnormal position, by actual distention of the viscus with a gaseous medium supplemented by percussion and

auscultatory percussion, is absolutely indispensable, and the most practicable as well as the most effective means of accomplishing this object is by simple inflation through a stomach-tube with an ordinary atomizer bulb. The carbon dioxide method has the sole advantage of enabling one to dispense with the introduction of a stomach-tube, and the distinct disadvantage that the gas rapidly escapes through the œsophagus and into the small intestine, so that the distention does not last long enough for a thorough examination to be made. With ordinary inflation, if the air is pumped in rapidly at first, spasm is induced at the pylorus and prevents the premature escape of the distending medium into the small intestine. (Pepper and Stengel.) The stomach should first be emptied in the usual manner, after which an atomizer bulb is fitted to the stomach-tube, without removing the latter, and the distention carried to a point just short

Fig. 373.



Inflation of stomach.

of causing discomfort. Owing to the lax condition of the abdominal walls, the outline of the stomach is usually at once visible, or it can be determined by palpation, or, as some authorities advise, by auscultatory percussion. The percussion-note over the distended viscus is of course tympanitic. The following sign may be sufficient: "when the bulb of the syringe is compressed, forcing the air through the tube, a peculiar metallic ring can be heard with the stethoscope over the stomach. When the bell of the instrument is moved along beyond the stomach limits, the sound instantly loses its metallic character. The same authority advises that the air be allowed to escape before the tube is withdrawn, in order to prevent the distress which often accompanies the escape of the air from the stomach, probably on account of a spasm at the cardiac orifice. When gastroptosis is present, the greater curvature is found below the level of the umbilicus, and in extreme cases may

extend almost to the pubes. In total descent of the stomach the entire outline occupies a position below the median transverse line of the body.

Blood. Except for the frequent presence of chlorosis already referred to, or a mild simple anæmia, there are no changes in the blood-count.

Urine. Examination of the urine may reveal the presence of constituents pointing to intestinal fermentation, otherwise the secretion is normal.

Gastric Contents. The findings are not distinctive and probably have no connection with the gastroptosis; the chief value of an analysis is derived from the therapeutic indications which it affords.

Diagnosis. In the light of recent researches gastroptosis must be regarded as a much more common condition than was formerly supposed, and it is therefore important to make a thorough physical examination of all cases presenting the symptoms of dilatation and deficiency of motor power, which include most cases of so-called gastric neurasthenia. If inflation is resorted to, a mistake in diagnosis is hardly conceivable. The condition for which gastroptosis is most likely to be mistaken, and with which indeed it is frequently associated, is displacement of the womb; in many supposed gynecological cases a complete cessation of the reflex nervous symptoms might be brought about by rational treatment directed to the correction of a displacement of the abdominal rather than of the pelvic viscera.

DISEASES OF THE INTESTINES.

The intestine is a canal of varying dimensions, the physiological office of which is to propel material received from the stomach and to permit of the digestion and absorption of that which is to serve for the nutrition of the body. The canal is richly supplied with bloodvessels and lymphatics. It is made up of mucous membrane, muscle, and peritoneum. For the purpose of digestion fluids are secreted either from the intestinal glands or from the large neighboring glands which discharge into the canal. Diseases that affect the canal impair or cause an abeyance of the physiological offices. As these offices—absorption and digestion—are essential to nutrition, it is not surprising that the body-weight and strength are impaired. We know too little about the function of digestion to utilize such knowledge in diagnosis. Intestinal digestion is also dependent upon the healthy performance of the functions of the liver and pancreas. It is difficult to draw fine lines of distinction even in health, and intestinal pathology is closely interwoven with hepatic and pancreatic pathology.

Alterations of the function of the intestine as a canal give rise to distinctive symptoms. Either its movements are too frequent and rapid, causing *diarrhœa*, or too sluggish, causing *constipation*. Obstruction of the canal leads to symptoms common to such a condition (see Morbid Process), modified by the physiological duties and the anatomical structure of the canal.

The morbid processes are hyperæmia, inflammation, the degenerations, and new growths. The symptoms that attend these processes are not

different from the symptoms that attend such processes in similar structures elsewhere. It must not be forgotten that the function of the canal is influenced by each process. On account of the process we may have *pain* and *fever;* on account of the impaired function, *pain, flatulence, diarrhœa* or *constipation, change in the character of the stools,* and *impaired nutrition.* Some of the above morbid processes may lead to the mechanical condition, *obstruction.*

The morbid alterations of the intestinal tract are ascertained from data obtained by *inquiry* and by *observation.* The data obtained by inquiry include the subjective symptoms—pain, and discomfort from flatulence. By observation the general condition of the patient, the presence of tenderness, alterations in the size and shape of the abdomen, and other physical phenomena are observed. The feces are carefully studied for the purpose of determining functional disturbances in the bowel, the presence of ingredients due to some morbid process, as serum, blood, pus, or mucus, or of extraneous matter, as worms or foreign substances. The feces are studied with the naked eye, with the microscope, and by bacteriological methods.

One symptom may be the chief manifestation of a disease, as pain in lead colic, diarrhœa in several morbid disorders, constipation in others. In the discussion of the special symptoms a consideration of the diseases of which the symptom is the main expression will be taken up.

Parasites. The intestine is the recipient of material for nutrition. Parasitic forms of animal life, or their ova or spores, may enter the intestine with the food. They either remain in the intestinal tract or wander into other structures. They include animal and vegetable organisms, such as forms of protozoa, vermes, and fungi. While the canal is open to infection by various micro-organisms, it is the natural habitat of others, which may become deleterious agencies when the conditions of their environment are changed. Thus *Bacillus coli communis* is, in man with normal epithelial structure and normal secretions, an innocuous parasite which, when inflammation sets in, may become virulent.

The symptoms produced by the *protozoa* and *fungi,* or by their products, the ptomaïns, are of an infectious or toxic nature. Inflammation is produced locally.

The symptoms produced by *worms* in the intestinal canal, are: (1) reflex; (2) symptoms due to catarrhal inflammation; (3) symptoms due to the action of the parasite on the blood—anæmia; (4) symptoms due to wandering of the parasite, as in trichinosis. (See Feces.)

Symptoms of the Tæniæ and Bothriocephali. There may be no symptoms save discharge of the parasite or portions of it by the rectum. In other cases the symptoms of intestinal dyspepsia or intestinal catarrh are observed. Headache, giddiness, lassitude, and itching at the nose and at the anus are said to be present. The patient becomes hypochondriacal. Convulsive disorders occur. Hysteria, forms of epilepsy, grinding of the teeth at night, and restlessness attend the presence of the parasite in the intestine. In all convulsive disorders the possibility of worms as a cause must be remembered.

Symptoms of Ascarides. (1) Gastro-intestinal catarrh; (2) symp-

toms of obstruction (rare); (3) symptoms due to wandering—as to the hepatic duct, to the stomach, or to the vagina; (4) nervous symptoms of reflex origin; (5) the worm or its ova in the feces.

Symptoms of Oxyuris Vermicularis. (1) Gastro-intestinal dyspepsia or catarrh; (2) itching or heat at the anus, worse in bed; (3) vesical and rectal tenesmus; (4) erythema about the anus; (5) priapism; (6) vulvitis and vaginitis; (7) the worms in the feces.

The Strongylus. The symptoms are local, with the symptoms of profound anæmia. The discovery of the ova in the feces distinguishes this form of anæmia from other varieties.

Uncinaria Duodenale. The symptoms are those of grave secondary anæmia and of chronic gastritis.

The symptoms due to the presence of *Trichina spiralis* and filaria are discussed in their appropriate sections. (See Blood and Infectious Diseases.)

The Intestines in Other Diseases. The relationship of intestinal disorders to affections of other viscera will be discussed with each symptom. It must not be forgotten that derangement of this tract may have its origin in local causes or in causes remote from the intestinal tract, or in some general condition of the individual. Thus diarrhœa may be due to inflammation which is primarily local, or which may be secondary to infection. Nothing is more common than to see diarrhœa in a general infection such as septicæmia. In exophthalmic goitre the diarrhœa is not due to a local cause, but to some as yet unknown nerve disorder. Constipation may be due to central brain disease, to a general condition like diabetes, or it may have a local origin.

It must be remembered that the diagnosis of an intestinal lesion is never complete without determining its causes. Thus enteritis and ulceration occur in typhoid fever, in cholera, and in other infectious disorders, all of which are to be passed in review in making up a diagnosis. Diarrhœa is a symptom in Bright's disease, and the causal relationship must always be borne in mind.

Differential Diagnosis. Intestinal disease or disorders are not usually confounded with disease of other structures. It is worthy of remark, as a fact that is sometimes overlooked, that symptoms of intestinal obstruction are frequently due to peritonitis. Tumors of the intestine must be distinguished from tumors of the peritoneum, the stomach, pancreas, and liver, and the uterus and ovaries. The history, the seat and physical character of the tumor, and the associate symptoms point to the true condition.

Embolism. The intestines are supplied by the mesenteric arteries. Their branches may become the seat of emboli. The symptoms are sudden pain, intestinal hemorrhage, and discharge of a portion of intestine. The patients are the subjects of atheroma or heart disease.

Intestinal Indigestion.

Intestinal indigestion is said to be due to alterations in or diminution of the bile, the pancreatic, or the intestinal secretion. It is almost always attended by gastric indigestion, and may not be readily distinguishable from it.

Acute Intestinal Indigestion. Acute intestinal indigestion is due to the irritation of food not properly digested in the stomach. It is attended with colic, flatulence, and borborygmi. Some fever may develop, and diarrhœa may ensue. In the mild forms the tongue is coated, there are loss of appetite and some general pains. There is epigastric distress or pain in the right upper quadrant. Flatulence and constipation occur. The stools are often clay-colored, or may not be changed. Slight jaundice occurs, and there is an abundance of lithates in the urine. Accompanying gastric indigestion modifies the symptoms slightly.

Chronic Intestinal Indigestion. The symptoms are more marked and pronounced in chronic intestinal indigestion. The *local symptoms* are as follows: Pain beginning from two to six hours after eating, and complained of in the region of the liver, below the sternum, or in the umbilical region. It is dull and continues two or three hours, or until the next meal is taken. There is some tenderness. With the pain there are tympanites, borborygmi, and a sense of fulness in the abdomen; the bowels are constipated, and the stools are hard and dry. The constipation alternates with diarrhœa, and undigested particles of food are passed. The appetite is not lost, but is variable. Hemorrhoids are often present.

The *general symptoms* are marked, and are referred to the nervous system and the condition of the blood. There are great depression and hypochondriasis. The patients sleep badly, suffer from unpleasant dreams and tinnitus aurium; there are spots before the eyes and more or less constant headache. They complain of pain in the back and limbs, and hyperæsthesia and anæsthesia are present. There is inaptitude for mental exertion. Frequently the patient has sudden attacks, apparently due to the action of toxins, as sudden fainting followed by collapse, or vertigo. During these attacks there are great palpitation and tachycardia. The extremities are cold, and there are cold sweats over the body. Independently of the attacks, the patient is subject to palpitation and some dyspnœa. The urine is always high-colored, acid in reaction, and full of urates and uric acid; oxalate of lime may be present, and there is usually albuminuria from the irritation of uric acid. The patient early becomes anæmic, because of the auto-intoxication and defective assimilation. There is some emaciation; in some cases the emaciation is rapid. The complexion is sallow. If there is an abundance of oxalates, the patient complains of weight and heaviness about the loins. It is always important in cases of chronic diarrhœa to look for achylia gastrica, particularly if the diarrhœal passages tend to occur soon after taking food. The stools may in other cases contain much undigested fat, indicating probable pancreatic disease. On the other hand, with loss of appetite, furred tongue, frontal headache, and drowsiness, the stools may be clay-colored and the bowels costive; this indicates obstruction to the outflow of bile.

Appendicitis.

This is by far the most important affection of the intestinal tract. It is of frequent occurrence compared with intestinal obstruction, and if recognized in time, is amenable to relief in a very large percentage of the

cases; whereas intestinal obstruction is more frequently fatal. We see 25 cases, at least, of appendicitis in all its forms to one case of any form of obstruction. Its importance therefore is readily recognized. Appendicitis occurs most frequently in the young—in the large proportion of cases in persons under thirty. I have seen it as early as two years of age, although from the fifteenth to the thirtieth year it is more frequent than at any other period. The symptoms vary, but clinically may be divided into those of appendicitis without perforation and appendicitis with perforation. Appendicitis without perforation is characterized by relapses, and is known also as *recurring appendicitis.*

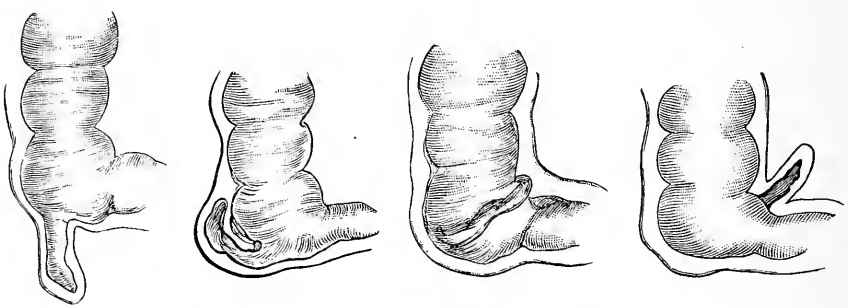
Appendicitis without Perforation. Cases of catarrhal appendicitis probably occur, although I am not prepared to say that catarrhal inflammation of the appendix gives rise to marked local symptoms; for in cases on the post-mortem table in which the lesions of catarrh were found, there had not been any symptoms during life, either due to intestinal catarrh or pointing to appendicitis in any form. Moreover, many cases in which the attacks of appendicitis had at first been slight, finally developed into appendicitis with perforation. In the milder cases, if operative measures are resorted to during the intervals between the attacks, the appendix is always found to contain a fluid loaded with micro-organisms capable of causing purulent inflammation, as the staphylococcus or streptococcus. Clinically, therefore, all forms of appendicitis should be considered infectious, with, on the one hand, escape of the contents into the bowel, and natural relief of the symptoms; or, on the other, complete obstruction with perforation. After removal of the appendix in cases of recurring appendicitis, I have always found pus or a mucopurulent material which was charged with streptococci or staphylococci, as well as *Bacillus coli communis,* natural to the intestinal canal in this region.

Symptoms of the Attack. After exposure to cold rarely, frequently after an indiscretion in diet, the patient is seized with pain referred to the right lower quadrant of the abdomen. It is paroxysmal in character, increasing in intensity, and may be described by the patient as colicky. The pain is usually such as to require the patient to take to bed and attempt to obtain relief by local applications; but it may be so slight that the patient pays but little attention to it. He may even go about his business during the attack and seek professional advice at the office of a physician. Such cases as these are usually diagnosticated as ordinary cholera morbus or intestinal indigestion. The attack may be only moderately severe, particularly if there is diarrhœa. With the onset of the pain vomiting usually occurs. The bowels may be open or they may be confined. Vomiting may not occur if there is diarrhœa, but when present, is usually attended by some nausea, although this is not marked; the vomiting is complete, there is no retching. It occurs at intervals, between which there is comparative comfort; first, the contents of the stomach, and then mucus are ejected. If the patients are to get well, vomiting does not return unless excited by food. If peritonitis supervenes, vomiting returns in the course of two or three days. In bed the patient lies on his back with his right leg flexed.

Even with a mild degree of pain the skin is hot and the temperature slightly raised. In the cases in which the pain is more severe the general reaction is greater, the temperature rises rapidly to 102° or 103° F. The skin is hot and dry, the face flushed. The pulse is full and strong and in a young adult rises to 90 and 95. In some cases the patient complains more of the fever than of the pain after the first severity of the attack has subsided. The tongue is coated and the appetite is lost.

On *physical examination* the area which was the seat of pain is found to be tender. When examined with the tip of the finger pressing firmly, more marked tenderness can usually be found at a point midway between the anterior superior spine of the ilium and the umbilicus, known as McBurney's point, which corresponds to the site of the diseased appendix. The swollen tender appendix may occasionally be palpable. On *inspection* the affected area is slightly or may be considerably enlarged.

FIG. 374. FIG. 375. FIG. 376. FIG. 377.



Some positions of appendix and relation to peritoneum.

Comparison must be made with the opposite side. It will be seen that the usual depression in front of the anterior spine, or the cavity toward the loin, is not so deep as on the opposite side. In front the surface may be even with the plane of the ilium. On *palpation*, in addition to tenderness and pain at the point previously indicated, fulness and enlargement can be distinguished. There are resistance to pressure and more or less rigidity of the abdominal muscles. On careful *measurement* the semi-circumference will be found in most instances to be larger than the semi-circumference of the opposite side. When bimanual palpation is performed, the left hand being placed in the loin behind and the right over the abdominal surface, resistance, induration, and rigidity can more easily be detected. On *percussion* there is change in the note compared with that of the opposite side, and change in the percussion-note during the course of the disease. This is particularly the case if the symptoms go on to perforation. On careful deep percussion a dull tympanitic tone is elicited, or a distinct area of dulness can be mapped out, but in some instances the distended cæcum yields tympany, which is greater than on the opposite side.

The *pain* is usually referred to the region above mentioned. The pain may be in the lower quadrant on the *left* side instead of the right, in those cases in which the appendix dips into the pelvis. It may also be

referred to the bladder or genitals, and be attended with vesical tenesmus and frequent micturition. The character of the pain and the bladder symptoms are such as to simulate an attack of renal colic, with the passage of sand. On account of the location the pain may be attributed to the Fallopian tube or ovary, and thought to be due either to disease of those organs or to dysmenorrhœa. It is not likely to be mistaken for the pain of dysmenorrhœa if the patient is subject to pain at the usual monthly period. If, however, the physiological and the pathological congestion should take place at the same time, a mistake in diagnosis might occur, particularly as increased abdominal pain may cause a uterine discharge. The occurrence of fever would exclude dysmenorrhœa in cases in which this symptom was present. The pain and leg-flexion simulate hip-joint disease.

After the first twenty-four hours, during which the above-described symptoms take place, the fever continues. There is anorexia, but vomiting occurs only at long intervals if at all. The local symptoms continue, although usually modified by the treatment. Both general and local symptoms frequently subside after a free movement of the bowels, which occasionally takes place spontaneously. In other cases constipation continues a week or ten days, and even a longer period.

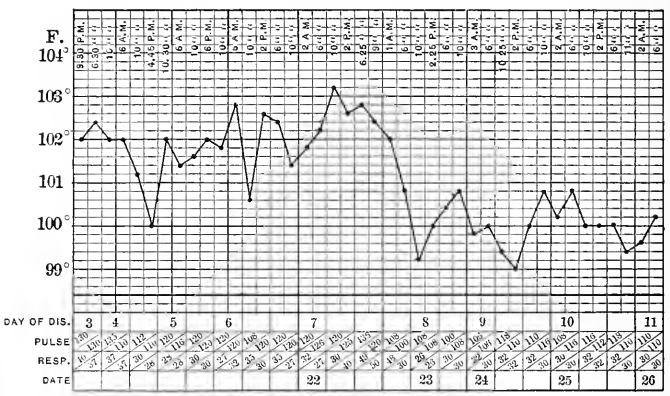
After five or six days at the furthest the fever subsides, the local distention lessens, the paroxysms of pain disappear, and convalescence ensues. Localized tenderness may, however, persist for a considerable period of time, and diarrhœa, or at least two or three evacuations each day, for a week or more. In rare instances peritonitis supervenes without the occurrence of perforation. Its onset under these circumstances is gradual, but the symptoms are like those of peritonitis under other circumstances, infection having taken place directly through the appendix.

When the fever continues, with mild diarrhœa, intestinal pain, and flatulence, the case may be mistaken for *typhoid fever*. The temperature is, however, more remittent in character in appendicitis, and the diarrhœa is not characteristic of typhoid fever. The characteristic eruption fever does not appear, the spleen is not enlarged, and the symptoms of the typhoid state do not ensue. The diazo reaction, the bacteriological examination of the stools, and the serum test may aid in forming a conclusion.

Recurrent Appendicitis. Frequent attacks of mild appendicitis occur; sometimes so frequently as every three months, or at intervals of a year. The attacks are similar to the attacks just described, although the duration is shorter. The local symptoms in some instances are more marked, because there has been a localized peritonitis previously. The induration is greater, and dulness more marked. In some instances the attacks are comparatively mild, continuing but twenty-four hours, and are described as attacks of colic. Often they are treated by the patient himself by household remedies alone. The patient spends a night in agony, with cramps, but the next day follows his usual habits. It is possible that there has been no fever with the attacks; but in all cases of recurrent appendicitis which I have seen, fever, although often slight, has been a constant accompaniment.

Appendicitis with Perforation. Before perforation takes place, the patient may have had symptoms of the mildest form of appendicitis for two or three days; or the attack may have extended over a long period of time, without any symptoms except colicky pain. As observations are not made, the presence of fever can not in such a case be utilized as a diagnostic feature. The perforation may take place early in the course

FIG. 378.



Acute appendicitis, with perforation and abscess. Female, aged eight years. Operation on seventh day.

of an acute attack, and result in *localized peritonitis* and *abscess*, or in *diffuse peritonitis*. If the latter, after the characteristic symptoms of appendicitis the symptoms of intense peritonitis set in. The abdomen rapidly becomes distended, the characteristic vomiting ensues, and collapse develops. Perforation under these circumstances has occurred within the first twenty-four or at most within forty-eight hours. Local inflammation about the appendix does not take place, and the local signs of an inflammatory tumor are not present, although tenderness at the special point can be elicited.

Abscess. If the perforation is more gradual, and there has been time for the occurrence of local inflammation about the appendix, by which pus is prevented from infecting the general peritoneum; or if perforation takes place behind, in the connective tissue which surrounds the mass, in which situation there is always inflammation, the local signs of abscess or of inflammatory tumor occur. There is swelling of the affected side; the normal outline is effaced. The area is indurated, and the early pronounced rigidity gradually gives way to a boggy sensation, with the appearance of œdema of the skin. This can be elicited by pressure over parts that are hard and resisting, as the spine of the ilium. Fluctuation can often be detected by bimanual palpation. Dulness is present, although in some instances it may be very slight, there being scarcely an appreciable change in pitch. Both light and deep percussion must be performed, and the note compared with the results of percussion over the opposite region. Palpatory percussion may alone indicate the departure

from normal. Examination per rectum may yield much information. An induration may be felt about the brim of the pelvis or in the rectal fossa, which fluctuates and may eventually soften. More accurate results may be obtained by bimanual palpation, with the index finger of one hand in the rectum and the other hand applied to the abdomen. Unless symptoms of general peritonitis with rapid infection of the system develop, the signs of abscess become more and more marked. The surface becomes reddened, and pointing may take place toward the groin or opposite the spine of the ilium. Sometimes the swelling increases in the direction of the loin, and the abscess may point in that situation.

As the abscess develops the general symptoms change. They now become the symptoms of *suppuration*. The fever is remitting or intermitting. There may be chills. Sweats are common, and there are loss of appetite and diarrhœa. Until recently it was customary, on account of the prevalent tardiness in operating, to see abscess develop in some other situation, or symptoms occur from burrowing of the pus in various directions. It may extend upward along the back of the colon, underneath the diaphragm, and thence to the pleura and lung, and be expectorated. The abscess may open into the rectum or into the bladder. If the local inflammation is virulent, even if general peritonitis has not taken place, the symptoms of *septicæmia* may rapidly ensue. This sometimes occurs quite early in the disease. There may be vomiting and septic diarrhœa, with slight delirium at night. An excessively rapid and feeble pulse is seen; in one instance it was irregular. Extreme prostration ensues, followed by symptoms of the typhoid state.

Gangrenous appendicitis is most treacherous. The early symptoms are like those of an acute attack; later all symptoms subside. Unless the temperature is taken or the physical examination is very painstaking, the patient is allowed to get up. The course may be afebrile. In a few days or a week an abscess forms about the slough, and then the usual phenomena of suppuration set in, or perforation may occur.

Diagnosis. It is clear that in cases of appendicitis we must attempt to recognize: (1) the inflammation before perforation has taken place; (2) the occurrence of perforation; (3) the occurrence of peritonitis due to either of the two conditions; (4) the occurrence of abscess (paratyphlitis and perityphlitis); and (5) the occurrence of septicæmia.

Appendicitis must be distinguished from *perinephric abscess* and the abscess which follows perforation of the intestine or cæcum at this point. Perinephritis can scarcely be distinguished from appendicitis unless there have been a previous history of renal calculus and pronounced evidence of disease of that organ preceding the formation of the abscess.

Hip-joint disease must be distinguished from appendicitis. The leg is flexed, the patient complains of pain about the region of the hip; unless careful observation has been made in the beginning of the attack, the early march of appendicitis may not be recognized. The two are confounded after abscess formation. The flexed leg of appendicitis can be extended under ether, and examination then shows the joint to be free from disease.

Fenwick says that *acute tuberculous peritonitis* may be confounded with perforation of the appendix. In both there may be pain and tenderness in the hypogastrium, dulness on percussion, and fever. In tuberculous peritonitis the onset is more gradual, the pain and tenderness more general, and there is no distinct tumor or increased tension in the hypogastrium. If there is dulness on percussion, the line generally varies with the position of the patient. Diarrhœa is urgent, and there are, in most cases, some signs of consolidation of the lungs. The absence of tumor in the right iliac region and in front of the rectum is the chief point of distinction; for when perforation occurs in phthisical subjects there is generally very slight pain, and severe diarrhœa is often the only prominent symptom. The appendicitis itself may be of *tuberculous origin*, as in several cases reported by the writer.

Inflammations of the Cæcum and Colon.

Typhlitis is an inflammation of the cæcum due to accumulation of fecal or foreign substances. The inflammation may remain as a localized enteritis, or may be followed by ulceration. In the majority of cases the ulceration is due to pressure by the contained foreign material or feces. The inflammation occurs in early life usually. The patients have been subject to constipation. The attack may follow some error in diet. There are pain in the right iliac fossa, constipation, and nausea. Moderate fever develops. On examination a fulness is discovered in the right iliac region, and the right thigh may be flexed; the cæcal region is tender to pressure, and a doughy sausage-shaped tumor may be outlined. The more severe symptoms last two or three days, but the local tenderness may continue a week or even longer. The tumor gradually disappears. If ulceration takes place, inflammation about the cæcum ensues. An abscess forms gradually in the flank. Perityphlitis is the term applied to this secondary abscess, although, as the term has been confused with paratyphlitis, it is better not to use it in this connection.

Pericæcal Abscess. Abscess about the head of the cæcum is due (1) to appendicitis, of which sufficient mention has been made; (2) to perforation of the cæcum, from typhlitis; (3) to perforation, on account of cancer of the intestine; (4) abscess secondary to kidney disease, perinephric abscess; (5) to abscess secondary to disease of the vertebræ. The physical signs are those of abscess due to perforation of the appendix. Abscess may occur behind the cæcum in cases of caries of the vertebræ and in some rare instances of empyema in which the pus has dissected downward. The symptoms are the local symptoms of abscess and the general symptoms of suppuration.

Fecal abscess, arising from ulceration of the colon, may be suspected, according to Fenwick, where there is a localized abdominal swelling, immovable in respiration or by a moderate amount of pressure with the fingers, the size and shape being altered when diarrhœa occurs, and when percussion over the tumor gives a tympanitic, or a more forcible stroke a dull sound, or when an emphysematous sensation is communicated to the fingers.

DISEASES CHARACTERIZED BY PAIN AND DIARRHŒA.

Acute Intestinal Catarrh.

Cause. Exposure to cold or the direct irritation of mechanical or chemical substances within the intestine. Irritating food that is not digested, or that can not be digested because of the quantity, tainted meats and unripe fruit usually excite an attack. Water saturated with impurities, or such as the individual is not accustomed to, may excite an attack. Strangers in a new locality are frequently subject to a diarrhœa until accustomed to the drinking-water, which in the native does not excite catarrh. Toxic substances, as poisons or drugs, or toxic substances the result of putrefaction, as ptomaïns, are frequent exciting causes. Extension of inflammation from neighboring structures by infection, as in peritonitis, sets up a catarrh. Local diseases of the intestine, as ileus, intussusception, hernia, and ulcers of all forms are attended by catarrh of the intestine. It also occurs in cachectic states of the system, as cancer, anæmia, and Bright's disease. In disease of the heart and blood-vessels, or of the liver and spleen, where the disturbance of the circulation causes a congestion, catarrhal inflammation occurs. It is of common occurrence in the infectious diseases, and particularly in septicæmia and pyæmia.

Symptoms. *Diarrhœa* is the chief symptom, varying with the cause and the extent of the catarrhal inflammation. As has been previously indicated, the stools differ in frequency and color in the various types. They contain undigested matter; sometimes worms. Colicky pains about the umbilicus, with borborygmi and frequent desire to go to stool, attend each evacuation. The fever is of the remittent type, and is attended with some prostration. The urine is scanty and high colored. The symptoms vary somewhat with the location of the inflammation, although the exact locality can not be distinctly defined. Symptoms of proctitis, pain with tormina and tenesmus, however, enable the localization to be made to that portion of the bowel. These symptoms are more common than in inflammation apparently limited to the small intestine, while in colitis the violence of the rectal symptoms stands between that of enteritis and that of proctitis.

Diagnosis. The diagnosis of acute intestinal catarrh is not difficult. It is more difficult to determine the actual cause. If the attack occurs suddenly after the eating of improper food or the drinking of impure water, the irritation is probably due to that cause, and may be determined by the character of the feces. If they contain undigested food, the diarrhœa is probably due to indigestion. Catarrh from cold usually follows exposure, and is generally not very severe. To estimate the cause from poison or drugs, the condition of the rest of the intestinal tract must be investigated and other symptoms of poisoning must be inquired for. In arsenical poisoning there is always vomiting, and the discharges are of a choleraic nature. Collapse rapidly ensues. The other symptoms of arsenical poisoning must be inquired for and the history of exposure ascertained if possible. Intestinal catarrh due to infectious diseases is attended by the

symptoms of the respective affections, each of which is usually readily recognized. The intestinal catarrh which occurs on account of local disease of the bowel, as hernia, stricture, etc., is preceded or attended by the local symptoms of these diseases. In like manner we judge of the nature of the diarrhœa that occurs in the course of tuberculosis or syphilis and in the course of organic heart disease or of liver disease. In each instance the possible influence of morbid processes present in other structures must be very carefully estimated.

Varieties of Acute Intestinal Catarrh. Divisions have been made in accordance with the symptoms which distinguish the various localities of the intestine in which the inflammation is most marked.

Catarrh of the Duodenum. This partakes of the nature and has the symptoms of mild gastro-intestinal catarrh, and is characterized by the occurrence of jaundice due to catarrhal inflammation of the biliary passages.

Catarrhal Enteritis. Colicky pains and rumbling are experienced. There is usually gastritis at the same time. The feces are mixed with mucus. Over the right lower quadrant there is tenderness on pressure.

Catarrhal Typhlitis. Pain in the right lower quadrant with tumor, dulness on percussion, and tenderness are present. (See Typhlitis.)

Catarrhal Colitis. The large intestine is most frequently affected. Pain and tenderness occur along the course of the bowel. The evacuations contain large amounts of mucus not intimately mixed with feces, or may consist of mucus; there is tenesmus. The association with gastro-enteroptosis and with neurasthenia must be borne in mind.

Catarrhal Proctitis. Proctitis gives rise frequently to small stools, tenesmus, pain in the left lower quadrant, with tenderness about the anus, and spasms of the sphincter. The stools contain a good deal of mucus and blood.

Cholera Infantum.

This affection occurs in children during the hot season. It is promoted by unfavorable hygienic surroundings, and is due to infected milk or food. At first there is catarrhal diarrhœa. This may continue for twenty-four hours, then vomiting and diarrhœa ensue. The stools are liquid and large in amount. At first they may contain milk-curds. The vomiting is excited by anything taken into the mouth, or by odors, or by movement of the little patient. The watery discharges are almost constant. They may be preceded by greenish or yellowish-green stools for twenty-four hours. The stools are acid in reaction, and their odor is sour. At first there is colicky pain; but when the watery discharges begin, there is only a little tenesmus. The stools irritate the skin and cause eczema. The rectum may become prolapsed. The abdomen is first distended with gas, but soon becomes retracted.

In a short time, twenty-four hours or even less, collapse ensues. Previous to the collapse the skin is hot and dry; the patient is restless. The thirst is intense, the mouth dry. The body-temperature is 103° to 104° F. With collapse the extremities become cold, the skin cool. The axillary temperature is lowered and the rectal temperature increased to 105° to

106° F. The restlessness continues, the fontanelles become depressed, the eyes sunken, the face pinched, the brows drawn. The urine diminishes in amount or may be suppressed altogether. Brain symptoms ensue. So-called hydrocephaloid symptoms follow—rolling of the head, strabismus, turning in of the thumbs, and, later, convulsions. Stupor followed by coma develops in the fatal cases. If the patient does not die in collapse, marasmus develops; ulceration of the cornea may take place; there are œdema and blood extravasation under the skin. The child emaciates and withers. On account of the weak heart and exhaustion pulmonary atelectasis or bronchopneumonia may occur. The age, the season, the presence of catarrh, with collapse and other symptoms, render the diagnosis easy.

Cholera Morbus.

The attack is characterized by sudden vomiting, followed in a short time by purging. The vomiting may be preceded by pain, or both may occur at the same time. At first the pain is seated in the epigastrium, subsequently above the navel. It is very severe and paroxysmal in character, compelling the patient to double up if lying in bed. A cold perspiration breaks out on the forehead, the extremities become cold, the face anxious, the pulse rapid. At first the patient vomits undigested food, then watery, greenish-colored fluid. The latter is bitter. Purging sets in at once or within an hour. The bowel-movements follow an attack of pain. The first passage is fecal, and may contain undigested food; the subsequent passages are watery and profuse. There are severe attacks of burning and tenesmus; the abdomen is tender around the navel and in the epigastrium. After an evacuation there is slight relief, but soon another paroxysm of pain comes on. The vomiting is excessive, and retching may be present in the intervals. Ice, or water, or anything taken into the stomach excites pain and causes vomiting. The attack subsides in twelve to twenty-four hours, and is followed by exhaustion; in rare cases collapse; and in others the attack is followed by gastro-intestinal catarrh.

Cholera Nostras.

The symptoms are those of a severe gastro-enteritis, sudden vomiting, and diarrhœa. The attack usually begins in the night. The vomiting is not different from that of *cholera morbus*. The watery and brownish-colored stools become colorless and have the appearance of rice-water. Pain attends the attack, rapid prostration ensues, the extremities become cold, and collapse takes place. With the collapse there are cramps in the legs. Other muscles of the body may become cramped. The disease occurs in epidemics during the hot season, and may be mistaken for Asiatic cholera. It can be distinguished from the milder forms of cholera which precede the occurrence of the epidemic only by the absence of the comma bacillus. The bacillus of cholera nostras is found in the stools. (See Feces.)

Enterocolitis.

In enterocolitis of young children the more intense inflammation succeeds a mild intestinal catarrh. There are increased languor, great fretfulness, and fever. The early catarrh is attended by green acid stools, with lumps of casein. The tongue is furred and moist at first. It soon becomes red and dry; vomiting ensues. The stools are offensive and increase in frequency, and, in addition to the appearance first indicated, contain mucus and blood. Death may take place within the first week on account of exhaustion from the vomiting and diarrhœa.

If the disease is protracted, it is attended by great wasting, symptoms of hydrocephalus, skin eruptions, hypostatic pneumonia, and extremely feeble circulation.

Chronic Intestinal Catarrh.

The disease usually follows an acute attack, or may be chronic from the start. It may follow gastric hyperacidity and dilatation of the stomach, and is not uncommonly due to gastric achylia. It arises secondarily to portal congestion in disease of the liver and in chronic disease of the heart or of the lungs. It occurs in malaria and in the scorbutic cachexia.

The symptom is diarrhœa alternating with constipation, or diarrhœa alone. Stools may contain undigested food, or pus, mucus, and blood in small amounts. Diarrhœa may be present in the morning only under these circumstances. If the feces are examined, the eggs of parasites or infusoria may be found. The local abdominal symptoms of rumbling, flatulence, and tormina are present. There are reflex symptoms of cardiac palpitation and dyspnœa (asthma). Rush of blood to the head may occur. Often these symptoms are relieved by the passage of flatus. Chronic catarrhal gastritis usually accompanies the intestinal catarrh. The general symptoms of *anæmia*, *emaciation*, and *neurasthenia* are present. Hemorrhoids are common.

Amyloid Degeneration of the Intestines.

The symptoms are those of diarrhœa, persistent but mild in character, associated with symptoms of amyloid disease in other organs. With enlargement of the liver and spleen changes in the urine due to amyloid disease are present. The occurrence of these symptoms in a patient with syphilis, or especially in a child with bone disease or tuberculosis, points to the nature of the case.

Ulceration of the Intestines.

Duodenal Ulcer. Ulcer of the duodenum often occurs in young subjects in whom there are symptoms of chlorosis or anæmia. The causes are the same as those of gastric ulcer. It may follow boils, erysipelas, or pemphigus, and differs in one ætiological respect from ulcer of the stomach in that it occurs more frequently in the male sex. The symptoms

are obscure, and may be wanting entirely, the patient probably complaining only of intestinal indigestion. In other cases they are like those of gastric ulcer. In typical cases the symptoms are those of pain situated below the xiphoid or to the right of the median line in the region of the pylorus. The pain occurs some time after eating, and may be relieved by vomiting. There is localized tenderness on pressure. Hemorrhage may take place from the stomach, or blood be found in the stools alone. It differs from gastric ulcer only in the possible difference in location of the pain, the occurrence of intestinal indigestion and hemorrhage, and the fact that the pain comes on one to two hours after eating. It may have the same consequences as gastric ulcer—hemorrhage, perforation, subphrenic or abdominal abscess, or in rare cases stenosis of the duodenum.

Duodenal ulcer is *diagnosticated* by the occurrence of melæna, which may be excessive and cause syncope and vomiting, with no blood in the vomitus; by pain, which may be in the right hypochondrium or between the navel and the right costal border; by gastralgic attacks, and by dyspepsia with constipation.

General Ulceration. Ulceration of the intestine may be due to a specific infection, and hence be symptomatic of typhoid fever, syphilis, or tuberculosis. It is nearly always present in the first-mentioned disease, and of frequent occurrence in the latter. Follicular ulceration occurs in enterocolitis in children. Ulcers due to the presence of feces occur in typhlitis and chronic constipation. The sacculi of the colon become filled with scybalous masses, the pressure of which produces ulcers. Tenderness is experienced along the course of the colon, particularly on palpation of the fecal masses, which may be felt through the abdominal wall. A *chronic ulcerative colitis* is the form that succeeds the diarrhœas which occur during camp-life, or that are set up in communities where people are crowded and live amid unhygienic surroundings. It is the form that attends scurvy, and is frequently seen in chronic Bright's disease. It may be succeeded by dilatation of the colon, by hypertrophy of the muscular walls, or by contraction of the bowel. The persistent diarrhœa leads to profound emaciation, extreme prostration, sallow complexion, with markedly impaired nutrition of the skin. Such forms of diarrhœa were seen during the War of the Rebellion, particularly in soldiers held in captivity. The diarrhœa may first be of a lienteric character, and later alternate with constipation. Stools contain blood and mucus.

Ulcers of the intestinal tract may occur from other causes, and diarrhœa may be the predominant symptom. They may be due to *cancer;* the malignant nodules may ulcerate within the lumen of the bowel. The bowel may be perforated from the exterior, on account of suppuration somewhere along its course, as in appendicitis, pancreatitis, or tuberculous peritonitis.

Symptoms. The symptoms of intestinal ulcer are usually those of diarrhœa. Ulceration, however, may be present without any symptoms whatsoever, particularly if the small intestine is affected. One or two small ulcers, on the other hand, in the lower portion of the colon, may set up continuous diarrhœa. The *stools* are composed of feces, mucus,

pus, shreds of tissue, and blood. If pus is discharged in large amount, an abscess has probably opened into the bowel. Moderate discharge of pus usually follows ulcers in the colon. Pus may be present in cancer. *Hemorrhage* is of frequent occurrence, and is an important diagnostic symptom, especially if profuse and occurring without symptoms of obstruction, of gastric ulcer, or of hemorrhoids. Fragments of tissue found in the stools may point to the nature of the process; they may be composed of the mucosa, connective tissue, or muscular coat. In dysentery the stools contain large quantities of disintegrating tissue. *Pain* occurs in many of the cases. It may be general and colicky, or circumscribed in cases of ulcer of the colon. Perforation of the intestine is followed by localized or general peritonitis. The occurrence of the latter depends largely upon the situation and the rapidity of the ulceration. When the perforation is in the posterior wall of the colon, a circumscribed abscess may develop. When it is situated in the upper zone, the pus may accumulate underneath the diaphragm or in the lesser peritoneal cavity. The signs of *pyopneumothorax subphrenicus* occur when the latter accident takes place, as both pus and air accumulate in the abscess-cavity. In such instances the ulceration usually takes place at the splenic flexure. Perforation of an ulcer of the cæcum may simulate appendicitis.

Tuberculosis of the Intestine.

The disease is usually secondary to chronic tuberculosis, but may be primary, especially in children. The symptoms are usually those of *diarrhœa*, and in the primary form this is associated with general emaciation, which advances rapidly, and with anæmia. Fever of the intermittent or remittent type is present. There is meteorism; the abdomen is much distended, but eventually becomes contracted. The mesenteric glands can be made out along the spinal column, and the intestines may become bunched into a mass, yielding a dull tympany on percussion in the centre of the abdomen. The diarrhœa is attended with colicky pains. The *diagnosis* is based upon the rapid emaciation, irregular fever, enlargement of the mesenteric glands in a patient, usually a child, who had probably been exposed to tuberculous infection. In one of my cases, the child, aged four years, ate of the same food, using the same utensils, as a brother, a man of twenty-two years, who was dying of pulmonary tuberculosis. The child was constantly with the brother. The remainder of the family, eight in number, remained in perfect health, and were all of good physique. The elder brother had become infected by association with tuberculous subjects in improper quarters away from home.

Intestinal Obstruction.

Ætiology. Intestinal obstruction may be acute or chronic. The causes in general may be arranged under the three following heads: diseases outside of the intestines, diseases of the intestinal walls, and accumulations within the intestines.

A. Diseases Outside of the Intestines. 1. Pressure of *tumors*, chiefly

ovarian, uterine, and omental tumors, and pelvic abscesses, or abscess about the cæcum. The obstruction may be *acute* or *chronic*. The symptoms of obstruction develop gradually, although in some instances they may take place suddenly, especially if aided by the accidental occurrence of fecal impaction.

2. *Constricting bands*, hernial openings, the remains of fetal structures, cause constriction of the intestine. In this class of cases there is usually pain, and the history preceding the obstruction is that of peritonitis, general or local, of old hernia, of appendicitis, of pyosalpinx, or of inflammation about the gall-bladder and gall-ducts. The onset may be *acute* or *chronic*. If the constriction is due to protrusion into hernial openings, the onset is usually sudden and without previous symptoms.

3. *Peritonitis* is a common cause of *acute* intestinal obstruction. It may be due to over-distention by gas and paresis of the bowel, or to pressure by external exudation.

4. Knots and twists of the intestines, usually seated about the sigmoid flexure, causing *volvulus*, are a common cause of *acute* constriction.

B. Diseases of the Intestinal Walls. 1. Invagination, or intussusception. The attack is *acute*, although the affection may continue over a long period of time.

2. Cancer and other tumors of the intestine generally lead to stricture and *chronic* obstruction.

3. The healing of ulcers, which are syphilitic in the larger number of cases, rarely tuberculous, will lead to stricture. The obstruction belongs to the *chronic* variety. It is seated, in the larger number of instances, in the rectum or sigmoid flexure of the colon.

C. Accumulations within the Intestines. 1. Feces. The obstruction takes place gradually, occurs in weak and debilitated people in the course of constipation, especially the constipation of acute disease.

2. Accumulations of improper food or foreign materials. The seeds of fruits or the husks of grain accumulate and cause obstruction. Magnesia, iron, and other articles taken as medicines, from their accumulation, may lead to obstruction of the intestine. In both of the above-mentioned varieties obstruction is *chronic*.

3. Impaction of gallstone within the intestine is followed by acute obstruction.

Causes of Acute Intestinal Obstruction. Various factors must be considered in order to estimate the cause of the obstruction.

The Age. Obstruction from intussusception occurs early in life; from bands or through apertures, in adult life, usually prior to forty years of age; from volvulus, between forty and sixty years. Obstruction due to a gallstone occurs during the middle or later period of life—always after the fortieth year.

The Previous History. In obstruction by bands of adhesion there is a history of peritonitis, or, as Treves points out, previous attacks of obstruction more or less marked. In volvulus the patient has been subject to constipation prior to the attack, and in intussusception there has been no previous history, unless polypus was present, causing dragging, colicky pains, and occasional discharge of blood.

The three principal causes of acute intestinal obstruction, when it does not develop in the course of a chronic obstruction, are: constriction or strangulation of the bowel by peritoneal bands or the contraction of apertures through which the bowel passes; volvulus of the colon; and acute intussusception.

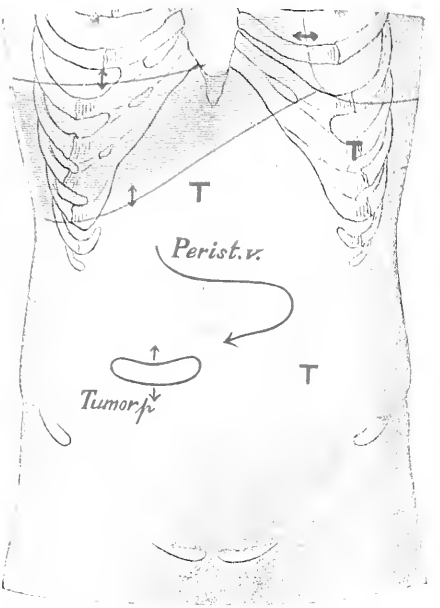
Strangulation. The type of the first variety is seen in strangulated hernia; but similar strangulations occur in apertures within the peritoneal cavity. Thus a loop of intestine may be caught and constricted in the duodenojejunal fossa, forming a so-called *Treitz retroperitoneal hernia;* or in the foramen of Winslow—*intersigmoid hernia;* and, finally, there is *diaphragmatic hernia,* or protrusion through the diaphragm of the intestine along with other abdominal viscera. These irregular forms of hernia may exist without symptoms, or they may lead to constriction or twisting of the loop of intestine with the development of acute obstruction. The bowel may become caught and constricted in an omental laceration, so-called internal constriction. The commonest situations for external constrictions are the hernial regions, where the gut is apt to be constricted by the dense fibrous adhesions, and in the vicinity of the uterus and the Fallopian tubes when these structures have previously been the seat of inflammation. The constricting bands that remain after the subsidence of the local peritonitis may gradually compress the gut to the point of complete obliteration of its lumen; or the bowel may become twisted about the adhesions with a similar result. Disease about the vermiform appendix, with secondary adhesions, has been observed to cause constriction. A common form of intestinal obstruction is caused by tangling of the intestine in the remains of the omphalomesenteric duct or Meckel's diverticulum, which is situated a short distance above the ileocæcal valve.

Volvulus. Volvulus is a rare form of acute intestinal obstruction due to twisting of the intestine, usually at the sigmoid flexure of the colon. When there is congential elongation of this portion of the bowel, the weight of the intestinal contents, dragging upon the gut, causes it to double on itself and form a loop which then becomes twisted; other portions of the intestine, under the influence of peristalsis, wind about the pedicle of the loop, and the obstruction becomes complete. In extreme cases true knotting of the bowel has been known to take place. Abnormal peristalsis induced by diarrhœa often precedes the appearance of the obstruction. *External injury* is said also to be occasionally responsible for intestinal obstruction.

Intussusception. (Plate XLI., Fig. 1.) Intussusception as a cause of intestinal obstruction occurs most frequently in children, and is due to invagination of the intestine, the upper portion being pushed into the lumen of the part immediately below by the active peristaltic movements, which persist in the proximal portion, while the distal portion becomes paralyzed. Intussusception is frequently found in the bodies of children who have died from exhaustion, the accident in these cases having occurred just before death. Intestinal polypi dragging one portion of the bowel down into the part immediately below are sometimes the cause of intussusception; large portions of the bowel may thus be involved.

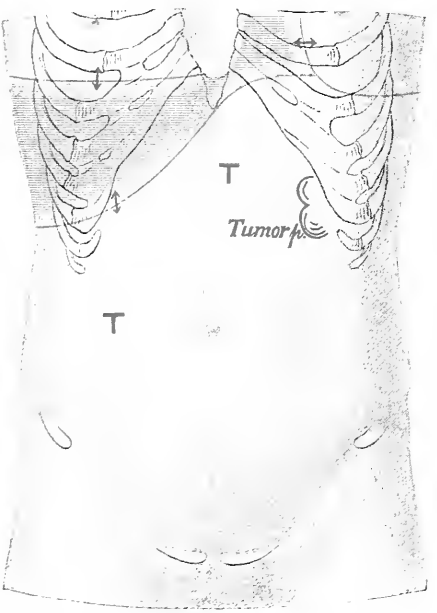
PLATE XLI.

FIG. 1.



Invagination of the Ileum.

FIG. 2.



Carcinoma of the Colon.

The invagination usually takes place in the lower portion of the ileum or in the cæcum; sometimes the invaginated loop extends to the rectum and presents at the anus. Intense inflammation, with the formation of adhesions, marks the course of the disease; the inner knuckle becomes gangrenous from the constriction of the afferent vessels, and may slough off and be discharged with feces through the anus, thus effecting a spontaneous cure.

Symptoms. When symptoms of intestinal obstruction occur, it is important to ascertain, in addition, first, the duration of the obstruction and its mode of onset; second, the possible cause of the obstruction; third, the seat of the obstruction.

Symptoms Common to all Forms of Acute Obstruction. The *local symptoms* depend upon the nature and the seat of obstruction. *Constipation:* The major symptom is stoppage of the intestinal contents. When this takes place suddenly, and there is a local injury to the bowel, the symptoms, both local and general, are severe and alarming. When constipation is complete, not even flatus escapes. *Pain:* The pain is at the seat of obstruction or about the umbilicus. It occurs suddenly, and is intense and colicky or lancinating in character, radiating from the point of obstruction. There is tenderness over the painful part. The pain is due to the injury by the constricting agent or to violent peristalsis. It may be relieved by pressure. If the pain is intermittent, the obstruction is incomplete; if constant, it is absolute. *Tumor:* In many instances a tumor can be outlined, due to single loops of intestine, thickened walls, or abnormal contents. This is particularly the case in the obstruction of invagination and the obstruction due to volvulus. *Peristalsis:* The obstruction further causes *increased peristalsis.* This takes place above the point of constriction. Sometimes the movements of the intestine can be seen through the abdominal walls. The extent of the peristalsis is an indication of the site of the obstruction. The higher the obstruction the less the peristalsis. *Meteorism:* Gas accumulates above the point of obstruction, giving rise to meteorism. If the obstruction is low down, the distention and meteorism are general. If high up, in the small intestine, from constriction by Meckel's diverticulum or internal hernia, the meteorism is in the upper part of the abdomen and may be limited in extent, or dilatation of the stomach alone may be present. *Vomiting:* Vomiting soon occurs in acute intestinal obstruction from decomposition of intestinal contents, irritation of the stomach by the intestinal contents, injury to the peritoneum at the seat of the obstruction, or, finally, to the occurrence of peritonitis. At first the contents of the stomach are ejected, then watery fluid, bile tinged or largely made up of bile, and later fecal matter. Although of fecal odor, this does not constitute true stercoraceous vomiting; the latter occurs later in the course of the disease. It must not be forgotten that any obstruction of the intestine may develop with extreme rapidity, so that *fecal vomiting* may occur within two hours of the commencement of an obstruction. It is recognized by the odor of the matter vomited and by the color. It is a grave symptom, indicating complete obstruction of the intestine. If the obstruction is high up, as in the jejunum, fecal vomiting does not

occur. Simple vomiting, however, is more persistent in high obstruction. *Eructations of gas* are frequent.

The **general symptoms** are those of *extreme prostration* or *shock* in its most pronounced form. The *abdominal facies* previously described develops very rapidly. The *tongue* is not changed at first, but soon becomes dry and brown. In a few instances, as in invagination, there may be *fever*, but in other cases usually at once, or very soon in its course, the temperature falls to or below normal, or remains at this point if it has not risen. The extremities are cold, the features pinched, the eyes sunken, the expression anxious. The pain causes the patient to double up in bed. The pulse becomes rapid, weak, and thready. The respirations are proportionately hurried, but are also made more rapid and shallow by the tympany. The mind remains clear until the supervention of peritonitis and septicæmia.

Symptoms of Various Forms of Acute Obstruction. It is essential in order to distinguish the form of acute obstruction to ascertain the *nature* of the obstruction, and to determine, if possible, its *site*. The symptoms of the various forms of acute obstruction vary somewhat. *Pain* in strangulation, from bands or hernia, is severe and paroxysmal in character and attended by collapse. It occurs early in volvulus, though it is not so severe as in the former variety, and occurs at long intervals, becoming constant with exacerbations. In acute intussusception the pain occurs early, and is steady. It increases, and then may suddenly subside. At first it is paroxysmal, attending discharge of blood and mucus from the bowels. *Local tenderness* in strangulation occurs late. In volvulus it occurs early, and may be noted over distended coils. In intussusception it usually corresponds to a sausage-shaped tumor. *Vomiting* is marked in strangulation, soon becomes feculent, and increases the severity of the paroxysms of pain. In jejunal obstruction it is excessive and non-feculent. In volvulus it does not come on so quickly, but is severe and constant when it takes place. The relaxation that attends vomiting often affords relief to the obstruction. In intussusception it does not occur as early as in the other forms, and is not so severe. It becomes feculent in only a small number of cases. *Constipation* is continuous in all cases except intussusception. In the latter there is some constipation, but it is not absolute; diarrhœa is not uncommon, and discharge of blood in the stools, according to Treves, occurs in 80 per cent. of the cases. *Prostration* is severe in all cases, although probably not so marked in volvulus. Because of its close proximity to the rectum *tenesmus* occurs in volvulus. The symptom is of frequent occurrence in intussusception, often beginning early in the attack.

Physical Signs. (Plate XLI., Figs. 1 and 2.) On palpation of the abdominal wall it is noted to be soft and flaccid in most of the cases, unless peritonitis has ensued. This occurs early in volvulus, hence rigidity is marked. A *tumor* can be made out as a rule only in intussusception. It is seated in the lower right quadrant of the abdomen. Early in the attack it is oblong and sausage-shaped. When peritonitis ensues, it disappears on account of the tympany. A portion of the gut may protrude at the anus or be felt on rectal examination. *Meteorism*

occurs about the third day in a strangulation; it occurs early, is very rapid and pronounced in volvulus; and is absent in intussusception, unless constipation or peritonitis takes place. It is not marked in high obstruction.

The Site of the Obstruction. The seat of obstruction is in a measure indicated by (1) the location of the pain or abnormal sensations, (2) the character of the swelling, (3) the character of the stools, (4) the character of the vomiting, (5) the degree of meteorism, (6) the results of a rectal examination, (7) the change in the urine, (8) the general condition. The patient is often able to indicate the location of the obstruction fairly well by the sensations of obstruction or fulness and by the great relief experienced when a free evacuation of the bowels takes place naturally or artificially. On auscultation, when the bowel is irrigated, a *murmur*, like the deglutition-murmur, may be heard at the point of constriction of the gut. In obstruction high up there is but little *meteorism*, the tumor is usually not detected, and pain is seated about the umbilicus or the upper quadrants of the abdomen. Obstruction at the ileocæcal valve may be indicated by a tumor in the lower right quadrant over the region of the valve or just above it. It is usually at this point that invagination takes place, and hence we may look for a tumor in this situation. (Plate XLI., Fig. 1.) On the other hand, in volvulus of the colon, or stricture of the rectum, the obstruction, being low down, is attended by much meteorism and by pain in the left lower quadrant of the abdomen. A tumor may be detected in this position. The position of the obstruction is sometimes indicated by the seat of *peristalsis*. This may be seen to stop at a given point, which usually indicates the position of the obstruction. The seat of obstruction may be indicated by the number of coils of intestine that are engaged in the peristaltic movement. The coils of intestine in front of the tumor are dilated and hypertrophied. In active movement they cause prominences which follow the course of the bowel. Wylie has called them "patterns of abdominal tumidity." If the obstruction is in the jejunum, peristalsis may not be observed. If the lower end of the large intestine is obstructed, the colon is prominent; if the gut about the ileocæcal valve, the region about and below the umbilicus is prominent. *The urine:* The position of the tumor, it is said, may be to some extent indicated by changes in the urine. When the obstruction is in the small intestine, indican is much increased from the decomposition of albuminous substances and products of putrefaction. In this location the urine may be suppressed. In stenosis of the large intestine indican is often not increased unless there is cancer. The value of the information derived from the character of the stools and the results of rectal examination is obvious. Obstruction in the duodenum or jejunum is followed by rapid collapse and anuria. In general, it may be said the more severe and rapid the symptoms the more likelihood that the obstruction is in the small intestine.

Strangulation. Obstruction from hernia or constricting bands occurs in adults of both sexes after the fortieth year. In stricture from pressure of bands there has usually been a history of previous attacks of peritonitis

or of inflammation of the structures in relation to the peritoneum. Hence, a cholecystitis or appendicitis is often found to precede the obstruction. The attacks begin suddenly, and the symptoms may from the start be most pronounced. They are the typical symptoms of intestinal obstruction. The local tenderness, however, may not be present as early as in other forms of obstruction. It is quite characteristic not to find a tumor or positive local cause for the obstruction, and also not to have meteorismus. This is due to the fact that the obstruction is usually high up in the intestinal tract.

Volvulus. Volvulus occurs most frequently in males. It occurs late in life, and is usually preceded by a history of constipation. Premonitory symptoms may have been present for a few days, but the symptoms of obstruction develop suddenly. They are the symptoms of acute obstruction, but as the lesion is in the lower portion of the bowel, meteorism is present to a marked degree, and rectal symptoms are found. Tenesmus is present in a small proportion of cases. Peritonitis is likely to set in early, with increase in the temperature, increased tenderness of the abdomen, and more pronounced symptoms of collapse.

Intussusception.

Intussusception (Plate XLI., Fig. 1), or invagination, occurs most frequently in children prior to the tenth year. It is characterized by severe colic and pain in the abdomen, first complained of about the navel. The severity increases in paroxysms, and only lessens if complete strangulation has taken place. With the onset of the pain there are one or two movements of the bowels, which contain mucus and blood. After this there may be constipation, or the stools continue to be loose, and are as frequent as fifteen or twenty in a day. Sometimes they are quite bloody, and almost always there is some tenesmus. In a short time after the attack vomiting commences; it may be constant or occur only after taking food. At first the abdomen is soft, but tender on pressure. A sausage-like tumor can be felt on the right side below the transverse umbilical line. On inspection of the rectum a portion of the intestine may be seen, dark and gangrenous in appearance, or it may be felt by palpation. If there is much tenesmus, the anus often remains open. In rare cases the bowel may slip back and the symptoms subside spontaneously. On the other hand, peritonitis may rapidly ensue with high fever, followed by collapse and death.

Differential Diagnosis. Intussusception must be distinguished from *enterocolitis* of childhood or the proctitis due to a polypus. In enterocolitis there is no tumor, and the collapse and prostration do not occur so early and are not so rapid. There is greater likelihood of a number of the stools being greenish like spinach. In a polypus of the rectum the symptoms are local. The child is worn out and restless; but great abdominal tenderness, and the tumor, meteorism, vomiting, and collapse are absent. The rectum must be examined.

In *peritonitis* symptoms of stenosis of the bowel from *ileus paralyticus* may be present. The history and sequence of events must be watched

carefully. Often the commencement of the affection about hollow viscera which have previously been the seat of disease, or its onset with sudden perforation will point to the nature of the affection. In peritonitis there is no active peristalsis; there is general distention of the abdomen with general tenderness; the urine is diminished, but does not contain indican in excess, except in purulent peritonitis when the amount is large. Collapse ensues rapidly. Signs of effusion within the abdomen may appear.

In *embolism* or *thrombosis* of the *mesenteric artery* and *infarction* of the bowel the symptoms take place suddenly. The patients have reached middle or late life, and have atheroma of the general arterial system. Sudden pain in the abdomen, with vomiting and symptoms of collapse, takes place. There is moderate obstruction with distention of the abdomen. After the pain diarrhœa with the passage of blood develops. The age and the absence of tumor are distinguishing features from intussusception, the only intestinal condition for which it may be mistaken.

Diagnosis of Acute Intestinal Obstruction from Other Conditions. Acute intestinal obstruction must be distinguished from peritonitis and appendicitis. This is sometimes very difficult. Careful attention must be paid to the evolution of the case and the history of previous abdominal disease, or of lesions on account of which, on the one hand, peritonitis may occur, or on the other, obstruction of the bowel.

In *peritonitis* the attack follows disease in the uterine appendages, the vermiform appendix, or the gall-bladder, or perforation in some portion of the gastro-intestinal tract. *Fever* usually attends the inflammation, with or without chill. *Vomiting* will probably occur at the onset, and then subside until the peritonitis becomes general. The first paroxysms of vomiting are apparently due to shock. The vomiting that occurs rarely becomes feculent. As the peritonitis advances the vomiting becomes passive; a simple constant regurgitation of a large amount of fluid, greenish or grayish yellow, or watery, takes place. It pours into the mouth, and is simply discharged without the occurrence of retching. The abdomen is swollen and *tympanitic*. The symptoms due to excessive tympany are more marked than in intestinal obstruction. As the movement of the diaphragm is interfered with, breathing is hurried. The abdomen is tender on pressure and is the seat of general *pain*. The general pain and tenderness, however, can usually be found to be more marked at the possible primary focus of the disease. Further, on local examination, fulness or undue prominence or *swelling* may be observed in these situations. *On palpation* over the point of origin there may be localized *œdema*. The symptoms of collapse do not differ materially from those of intestinal obstruction, although the peculiar appearance of the face and other nervous features occur more rapidly in peritonitis than in obstruction. It must be remembered that peritonitis in a large majority of cases attends obstruction.

In *appendicitis* the symptoms are somewhat like those of intestinal obstruction. There may be constipation and vomiting. The former is not pronounced, and can usually be relieved. Vomiting subsides after the first twenty-four hours, unless peritonitis supervenes; it is never stercoraceous. The local physical signs are characteristic. In appendicitis there is fixed tenderness on pressure at McBurney's point. Some

swelling can almost always be observed. On light or deep percussion there is a change in the note as compared with the other side. Fluctuation can often be detected in from two to four or five days. Both the tumor and fluctuation can be detected by bimanual examination of the abdomen and flank. Examination by the rectum may reveal a tumor at the brim of the pelvis in the right side. Fever attends the attack throughout. When peritonitis supervenes, there is rigidity of the entire abdomen, which at first was localized to the right lower quadrant.

Intestinal obstruction must not be confounded with *enteritis*. In all forms there is diarrhœa, in many vomiting. Pain of a colicky nature, spreading from the neighborhood of the umbilicus, is marked whenever obstruction to the passage of feces or gas takes place. Vomiting is not stercoraceous, and the general symptoms, collapse, etc., do not occur.

Acute hemorrhagic pancreatitis is also attended by symptoms similar to those of intestinal obstruction. There is sudden severe pain in the upper half of the abdomen, with vomiting and the rapid development of collapse; there may be constipation; the situation of the pain is of some significance. Vomiting never becomes stercoraceous; flatus can usually be passed and the bowels opened by an enema. Meteorism does not take place, although the epigastrium is tympanitic. If the symptoms are not so severe, there may be increased dulness, and possibly a tumor on deep palpation in the left upper quadrant of the abdomen along the margins of the ribs, which should be dull on percussion, or, on account of its relation to the stomach, give a dull tympanitic note. The symptoms of internal hemorrhage are present: pallor of the face and extremities, syncope, and, in addition, prostration and other symptoms of collapse.

Symptoms of Chronic Obstruction. The general symptoms are those *of chronic constipation*, with local symptoms due to the cause of the obstruction. The bowels are moved infrequently, and the stools are small. In obstruction due to stricture from cancer or cicatricial closure, the feces are ribbon-shaped. Reference must again be made to the occurrence of so-called spurious diarrhœa, with or without the passage of small scybalous masses, on account of impaction of feces. Some credence can be given to the oft-repeated expression of the patients that they have a sense of obstruction in the bowel and that they experience great relief when there is a free evacuation. In chronic obstruction the general symptoms are those of inanition, with the nervous train of symptoms that have been described in constipation; while the local symptoms depend upon the cause. When the local symptoms are due to the pressure of a tumor, or accumulation of pus or fluid within the abdomen, there is a history of local disease, on account of which the tumor developed; such history is obtained in fibroids or ovarian tumor, or in previous inflammation, which was followed by the occurrence of a tumor about the locality of the inflammation, as the pelvis or the appendix.

Chronic intestinal obstruction always occurs in *adults*. The *onset* is gradual. The *pain* that attends obstruction of this form is intermittent, and if there is fecal accumulation, it is not very prominent. *Vomiting* occurs late in the disease, is small in amount, and generally is not a

prominent feature. Obstruction to the passage of feces may be constant, or alternate with *diarrhœa*. In fecal accumulation it becomes complete, although spurious diarrhœa may attend it. The discharges may be bloody, suggesting cancer. *Tenesmus* is present in stricture low down in the large bowel. *Meteorism* is not marked when the obstruction is high up, as in acute obstruction. When the obstruction is in the large intestine, it may be extreme, and in fecal obstruction gradually increases as the obstruction becomes more marked. Coils of intestine in peristaltic movement are seen only in cases in which there is marked emaciation.

If the obstruction is due to *stricture* from *cancer* of the intestine, the symptoms of that affection are present. A tumor can be made out at some situation in the course of the bowel, usually about the cæcum or at one of the flexures. The symptoms are (1) the cachexia, emaciation, and anæmia; (2) pain; (3) tumor; (4) constipation with scybalous discharges; (5) bloody discharge; (6) mucous discharge. It is a striking fact that in many cases the symptoms of moderate obstruction come on suddenly in the midst of fair or good health. If the cancer is seated in the rectum, we find tormina and tenesmus, and the discharge of blood and scybalous masses. Local examination reveals the presence of a malignant mass. Obstruction due to *stricture* from the healing of an *ulcer* is seated in the rectum or sigmoid flexure of the colon. Pain and a sense of obstruction are referred to that locality. A history of syphilis may be obtained, and frequently the rectal tube or the finger will detect the stricture. In both there is a history of imperfect, irregular action of the bowels from time to time, with intervals of comparative comfort. These symptoms precede the constipation. When feces accumulate in the colon, the larger accumulations take place in the sigmoid flexure and in the cæcum. Fecal tumors, described under Constipation, are felt through the abdominal walls. Obstruction from *fecal accumulation* is preceded by a history of constipation (*q. v.*). The accumulations can be easily discerned as a rule. It must not be forgotten that chronic intestinal obstruction may at any time become acute.

Cancer of the Intestines.

Obstruction must not be confounded with carcinoma of the intestines. (Plate XLI., Fig. 2.) The disease usually occurs late in life, and is associated with progressive *emaciation* and *cachexia*. There may not be any symptoms save general failure of health until the sudden occurrence of obstruction of the bowel. The symptoms vary with the position of the carcinoma and the direction of its growth. The most common situations of cancer of the bowel are the cæcum, the flexures of the colon, and the rectum. In some instances with the general symptoms, there may be irregular *pain* in the abdomen, with irregularity of stools. The *tumor* may be detected if the small intestine is involved. Its detection is facilitated by having the patient get on the hands and knees, and palpating the abdomen in this position, and by clearing out the colon with a large enema. On auscultation the water may be heard to enter the dilated colon beyond the tumor, the sound resembling the deglutition-murmur

heard over the cardiac end of the stomach. If the tumor is situated in the lower colon, pain in the sacral region, resembling sciatica, may be complained of; if the cæcum or the sigmoid flexure is the seat of disease, a tumor is usually detected. Wherever the situation, the tumor found is tender, usually lying in the axis of the intestine—movable if in the small intestine, fixed if in the cæcum or the sigmoid flexure. In the latter location the tumor may be felt per rectum. One notable characteristic is that it may be palpable some days and not be present at other times. The position and size may vary from day to day, although the tumor is always hard and knotty, not doughy. By means of the proctoscope, with the patient in the knee-chest position, as described by Kelly, the presence of tumors of the descending colon will be revealed. *Constipation* is characteristic of most of the cases. It may alternate with diarrhœa. Paralysis of the sphincter ani with incontinence may take place. The stools are frequently ribbon-shaped, or they may be passed in scybalous masses; and large or oftener small amounts of blood, chiefly the latter, are passed with pus or mucus; sometimes masses resembling cancer can be found in the stools. If the tumor is in the rectum, there is great difficulty in defecation; the act is attended by pain. Later the pain becomes constant, and may radiate to the hip or the genitalia. Sometimes this pain is the only symptom complained of.

The diagnostic symptoms are: (1) the general symptoms of cancer; (2) the tumor; (3) the occurrence of constipation, which leads to complete obstruction, or obstipation, alternating with diarrhœa. Blood in the stools, with alteration in the shape of the feces, is significant.

Boas insists upon the importance of sudden occurrence of moderate obstruction in fair health in persons beyond thirty or thirty-five, and considers the observation of peristaltic waves, particularly sudden, lightning-like contractions, very important.

Enteroptosis.

It is by inspection, palpation, percussion, and auscultation that we discover the anatomical cause for the symptom-group about to be described. Attention to this affection may, however, be called only by the subjective symptoms.

This disease or physical condition, called sometimes Glénard's disease, after the physician who first called attention to its existence in 1885, is characterized by the falling down or descent of a number of the abdominal organs. This occurs on account of relaxation of the supporting ligaments, the number of which Glénard puts at six. This relaxation is largely due to a flabbiness and hence lack of support of the abdominal wall; or to strain from undue physical exertion; or to the abuse of cathartics; or possibly to injury. It is far more common in females who have borne children. It may be the result of feeble muscle-tone, following prolonged illness, and in many cases seems to be due to a congenital abnormality of the tissues, whereby the ligaments readily become relaxed. There seem to be two fairly distinct forms: one, the result chiefly of mechanical strains, etc., occurs mostly in multiparous women; the other,

depending upon the body-construction, is seen often in young unmarried women and not infrequently in men. The degree of descent, and hence the severity of the symptoms, may vary from slight displacement of one or two organs to that of the large intestine, the stomach, the liver, the spleen, and the right kidney (sometimes both). In moderate cases but two of the ligaments are relaxed—the *ligamentum colicohepaticum* and the *ligamentum gastrocolicum;* in the more severe all are affected.

Symptoms. The *objective* symptoms are due to the slight displacement, and are either purely physical or arise from the alteration of the function of the stomach and the intestines.

The earliest objective symptoms are: (1) pulsation of the abdominal aorta; (2) a linear tumor or band about midway between the xiphoid cartilage and the umbilicus, extending transversely from four to six inches in length; (3) gastroptosis, or descent of the stomach; (4) movable right kidney. Later, the liver may fall from one to four inches, the spleen become palpable, and the left kidney movable. The transverse tumor above mentioned was held by Glénard to be the thickened transverse colon. Ewald, however, seems to have demonstrated that it is the pancreas. The displacement of the viscera is recognized by the methods previously detailed for physical examination of the various organs. The patient must always be examined in the erect as well as in the recumbent position. Care must be taken to distinguish gastric dilatation from gastric descent. This can be done by careful percussion after inflation with air, by gastric diaphany, by measurement with a sound, and with fluids. Glénard laid much stress upon the *splashing sound.* This may or may not be present; it may be of gastric or intestinal origin, usually the former. It does not depend upon the displacement so much as upon the occurrence of gastric dilatation. It occurs in other affections. Stiller has shown that the tip of the tenth rib is usually freely movable in the congenital type of cases.

An objective sign of diagnostic value, to which attention has been called by Treves, is the relief the patient experiences when the lower half of the abdomen is supported by a belt or by the hands of the patient or surgeon. (Glénard's belt test.)

The objective signs of gastric origin depend upon functional or organic disease of that organ. We may have, on the one hand, only the perverted gastric secretion and digestion that go with gastric neuroses; on the other hand, we may have the perverted gastric secretion of gastritis, gastric atrophy, or dilatation, and the evidences of diminished digestive, motor, and absorptive power of these affections.

The *subjective* symptoms are due to the same cause. The displacement gives rise to local symptoms of *weight, heaviness,* and abdominal distress, amounting in some instances to *pain,* especially when in the upright position, and to protracted and pronounced *neurasthenia.* Later, we have the subjective symptoms of *dyspepsia, gastritis, gastric dilatation,* and *intestinal atony,* while the neurasthenic symptoms grow more aggravated.

The subjective symptoms also depend upon the functional or organic changes in the stomach and intestines, upon the displacement of the

organs, with or without the above, or upon the associated physical muscular condition of the individual and the state of the nervous system.

Glénard divided the progress of the subjective symptoms into three periods:

In the *first* there is gastric atony, when the patient experiences weight and burning after eating; a short period of wakefulness about 2 o'clock in the morning; a loose stool on rising; loss of strength.

In the *second* period the patient can not digest fats and starches, and the subjective symptoms arise late in the period of digestion. A dragging sensation or a feeling of emptiness occurs about three hours after meals. The patient awakens at 2 o'clock in the morning, and remains awake for two or three hours. Constipation, at times alternating with diarrhœa, is present. There is continued loss of strength, and a tired feeling is complained of on rising.

In the *third* period the symptoms of neurasthenia are most pronounced. The patient is emaciated, and complains of a constant weight and of cramps in the stomach. Constipation is obstinate, and the stools are scybalous and mucous. The patient is much prostrated and suffers from sleeplessness. The constipation and the intestinal distress are aggravated by aperients. Enemata must be resorted to for the relief of symptoms. Intestinal catarrhal or membranous enteritis is very likely to follow.

Pain throughout the abdomen, especially when walking about or in the erect posture, is frequently complained of. Some authorities speak of tenderness on pressure over the solar plexus and of tender points along the vertebræ.

The disease is sometimes overlooked and the symptoms are attributed to neurasthenia. It is often difficult to estimate which of the two preponderates.

DISEASES OF THE RECTUM.

The consideration of rectal lesions belongs to the surgeon. It is proper, however, to insist upon the very frequent deleterious effect of such lesions in neurasthenic subjects. Indeed, the bleeding which attends hemorrhoids may be sufficient to lead to profound anæmia upon which neurasthenia may readily develop. The local suffering due to rectal fissure, or prolapse, may aggravate any tendency to the state of neurasthenia, or aid materially, with other conditions, to fasten it more firmly upon the system. In cases of anæmia, of neurasthenia, of the gastric neuroses, of debility, or prostration, the cause of which can not be ascertained, the rectum should be examined. The appearance of hemorrhoids and other rectal affections is described in works on surgery. Hemorrhoids, ulcers, fistula, and carcinoma are to be sought for in abdominal affections.

Inspection and palpation are necessary. The symptoms are those of local pain, tenesmus, and frequently hemorrhage. The pain follows a movement of the bowels. There may be a feeling as of a foreign body in the rectum, with some itching and burning about the anus. The pain may be so severe as to inhibit defecation. Timid subjects will not endure the act; in consequence they suffer from vertigo, headache, tym-

panites, and symptoms of gastro-intestinal disorder. In some instances there is chronic catarrh of the rectum, with discharge of small stools containing mucus or pus streaked with blood. Cases occur in which hemorrhage is the only symptom, the constant recurrence of which leads to grave constitutional results. Hemorrhoids are the lesions for which the rectum is most frequently examined. They, as well as other lesions, are of diagnostic significance in affections beyond the rectum. Thus in all forms of portal congestion internal hemorrhoids are of constant occurrence, and when found in a toper may be one of the first indications of cirrhosis of the liver. Rectal fissure is not of much diagnostic significance. The finding of a small cancer, the symptoms of which may be those of hemorrhoids, may explain emaciation and the development of cachexia. Ulcer of the rectum may be due to syphilis, cancer, or tuberculosis. A fistula is often tuberculous. The rectum must be examined in cases of pyæmia, particularly of the portal variety, when jaundice, enlargement of the liver, and hectic fever are present, for local rectal disease may cause pylephlebitis.

CHAPTER X.

DISEASES OF THE LIVER, SPLEEN, AND PANCREAS.

THE LIVER.

THE symptoms of disease of the liver are due to the morbid processes, to disturbance of the functions of the hepatic cells, or to obstruction of the channels for the flow of blood and bile. As these latter extend beyond the glandular structure of the liver, obstruction of channels and physical alteration in the size and shape of the organ may be caused by disease outside of the organ. Hepatic symptoms may, therefore, be due to diseases other than those of the liver.

The *morbid processes* are the congestions, the inflammations, the degenerations, the morbid growths, and gross parasites.

Symptoms due to the Morbid Process. In *congestion* of the liver the symptoms are (1) the symptoms of the basal lesion, (2) enlargement of the organ from the increased amount of blood, (3) functional disturbance from the same cause. The congestion is not limited to the vessels in relation with the liver-cells, but involves the vessels of the mucous membrane also, hence the latter swell, obstruct the ducts, and produce a moderate degree of jaundice. The *inflammations* are toxic and infectious. The symptoms are due to the primary intoxication or infection, to the degree of obstruction of the vessels and ducts, and to the alteration of the shape and size of the liver and of its function. When the inflammation is diffuse, as in the cirrhoses, the hepatic symptoms are more marked; when localized, as in abscess, the infectious symptoms predominate. When the ducts are the seat of infection, the bile-channels are obstructed and jaundice ensues, while obstruction of the vessels produces ascites. In *morbid growths* of the liver the symptoms are those of malignant disease in general, to which are added symptoms due to the change in the size of the liver, and, more frequently than in inflammation, symptoms due to obstruction of the channels. The *degenerations* are so frequently secondary to and masked by the primary cause that, save in regard to change of size, there are no hepatic symptoms worth mentioning.

Symptoms due to Functional Disturbance of the Liver. The functions of the liver are to secrete bile; to destroy the hæmoglobin of the blood; to destroy, modify, or neutralize poisons, and to modify and render available for nutrition the peptones absorbed by the portal circulation; and the elaboration of glycogen. When the liver-cells are destroyed, as in acute yellow atrophy, the secretion of bile is abolished, and the liver does not destroy the usual amount of hæmoglobin. On the other hand, hæmoglobin may be so much in excess that the liver is not equal to the task of destroying it, and *non-obstructive jaundice* results. Functional

disturbance of the liver manifests itself clinically by symptoms due to the entrance into the circulation of imperfect products of digestion, or poisons which the liver has failed to destroy.

Symptoms due to Obstruction of the Channels. (1) Obstruction of the *bile-ducts*, either from disease or from external pressure, causes jaundice, pain, and fever. The three symptoms may occur singly or combined. In obstruction by *gallstones* jaundice may occur alone or be accompanied by pain; or jaundice, pain, and fever may occur together; rarely, pain or fever is present alone. Each symptom will be described later. (2) Obstruction of the *blood-channels* causes *congestion* of the liver, which may be active or passive, *portal obstruction;* here again the symptoms are modified by the process. Thus in portal obstruction from pressure the symptoms are quite different from those in portal obstruction due to suppurative inflammation of the vein.

Historical Diagnosis. Primary liver disease is comparatively rare; secondary liver disease, on the other hand, is of common occurrence. For this reason a knowledge of ætiological factors is of aid in the diagnosis of hepatic affections. There are but few general organic diseases or states of the system that do not in some way influence the liver. Functional disorders of the liver, as previously remarked, are so difficult to separate from functional disorders of the stomach and intestines, that from an ætiological and clinical standpoint they practically go hand-in-hand.

Social History. *Age.* Diseases of the liver usually occur late in life because the causes upon which they depend are operative only at that period. In a case, therefore, of ill health in a young subject, when the cause can not well be determined, the liver is not so likely to be the seat of disease as in older subjects. Late in life we have gallstones with their multiple consequences, inflammation, cirrhosis, and cancer; nevertheless, we may, although not so frequently, have the congestions and the degenerations in early life.

Sex. The sex is not of much significance from a diagnostic standpoint. Cancer may be more frequent in the female sex because cancer of the uterus and other organs is more common. Cancer of the biliary passages is more frequent in females, because in that sex gallstones, which are ætiological factors in cancer, are more common. Cirrhosis is the result of alcoholism and consequently more frequent in men.

Habits. It is always necessary to inquire into the habits. Alcoholism points to cirrhosis; the excessive use of stimulating foods to hyperæmia; sedentary habits and the use of starches and fats to gallstones. The occupation has but little influence on the development of hepatic disease. With regard to the *climate*, it may be said that in tropical countries hyperæmia and abscess of the liver are more frequent.

Family History. But little is gained for the diagnosis by a study of the family history, as most of the morbid processes are secondary to disease elsewhere, except as regards biliary calculi, the formation of which appears to be very commonly a disease of special families.

Previous Disease. It is absolutely essential, in order to establish a diagnosis, to inquire into the previous medical history, as liver disease is

usually secondary. The occurrence of heart disease or obstructive lung disease points to congestion; infectious diseases to cirrhosis when that condition is not otherwise accounted for; dysentery to abscess; ulceration or suppuration in the portal area to multiple abscess; syphilis to syphilitic disease; tuberculosis, suppurations, bone disease, and syphilis to amyloid disease; pyæmia to multiple abscess; tuberculosis to fatty liver.

Subjective Diagnosis. **Pain.** Pain in biliary affections, including diseases of the passages, may be constant as in perihepatitis and carcinoma, or paroxysmal, recurring at long intervals, as in cholelithiasis. In congestion and in carcinoma it may be dull and aching, with some sense of distention. It is sharp, lancinating, increased by breathing in perihepatitis; sharp, colicky, or spasmodic in cholelithiasis. In diseases of the gall-bladder and gall-ducts it is associated with spasm of the rectus muscle, with tenderness on pressure and on special movements, as in so-called "hooking under the liver," the value of which Murphy points out in cholelithiasis. The pain of gall-bladder disease is due to inflammation, to distention, to colic or spasm induced by a foreign body (gallstones).

Pain due to inflammation and spasm must be distinguished from:

1. Dietl's crises, in floating kidneys, which may be accompanied by jaundice.
2. Crises occurring in dilated and displaced stomachs, first described by Kussmaul, due to kinking and stenosis of the first and second portions of the duodenum, especially if adhesions have taken place.
3. Crises due to downward displacement of the liver (36 per cent. of cases—Steele).
4. Renal calculi.
5. Pancreatic pain: (*a*) hemorrhagic pancreatitis; (*b*) acute pancreatitis, occasional jaundice; (*c*) pancreatic colic, crisis, and sometimes jaundice.
6. Gastric ulcer with local peritonitis.
7. Duodenal ulcer; jaundice is present in a small proportion of cases.
8. Gastric neuroses.
9. Abdominal pain of thoracic disease, as pneumonia, diaphragmatic pleurisy.
10. Disease of the vertebræ—tuberculous and rhizomelic.
11. Appendicitis.
12. Intestinal pain; (*a*) acute ileocolitis; (*b*) lead colic; (*c*) epigastric hernia.
13. Gastric crises of locomotor ataxia.
14. Peritoneal pains of toxic origin often occurring in uræmia—pain in upper half of abdomen, more or less collapse with nausea and vomiting.
15. Pain resulting from a forgotten fracture of a rib with callus pinching the nerve.
16. Aneurism of the aorta.
17. Obscure alleged neuralgias of the liver.

Objective Diagnosis. **Jaundice.** Jaundice presents two distinct forms —*obstructive* and *non-obstructive*, sometimes also called hepatic and extra-hepatic. The old terms hepatogenous and hæmatogenous owe their origin to the erroneous belief that bile-pigments, which are usually produced in the liver, may sometimes result directly from blood-destruction without the intervention of the liver. There is no doubt, however, that bile-pigments are always formed in the liver; hence the term hæmatogenous jaundice is incorrect.

General Symptoms. The color of the skin and of the mucous membranes has been described elsewhere. (See page 206.) In addition to the yellow discoloration we find: 1. *Cutaneous irritation.* An attack of jaundice may be preceded by general itching. The pruritus, which may be sufficiently severe to cause great distress, occurs in all forms of icterus, but is most marked in obstructive jaundice of long duration. Scratch-marks are seen on the surface of the skin, and boils and erythematous eruptions frequently occur. *Xanthelasma* is a peculiar affection occurring on the tongue, the skin of the eyelids, and about the ears. (See page 173.) 2. *Discoloration of the secretions.* All the secretions of the body are changed in color, as previously described. 3. *Absence of bile from the feces.* The stools are ashy or gray in color. 4. *Retardation of the pulse.* The heart's action falls to 40 or 30 beats in the minute, or even lower. 5. *Hemorrhages.* In the later stages of all forms of jaundice hemorrhages are of common occurrence. In acute malignant jaundice they are seen underneath the skin and in mucous membranes. 6. *Cerebral symptoms.* Irritability and depression of spirits are marked. As the disease advances the mind grows sluggish, the patient is dull and sleeps most of the time; gradually the symptoms of the typhoid state develop. In the acute febrile forms coma and convulsions are of common occurrence. In the affection known as *acute yellow atrophy* the cerebral symptoms are marked and occur soon after the onset of the disease. Within the first twenty-four hours there may be convulsions, with delirium in the intervals, and subsequently coma.

Obstructive Jaundice. The obstruction may take place in the large ducts or in the smaller terminal ducts; it may be due to disease outside of the ducts, to disease of the ducts themselves, or to obstruction within the ducts.

1. Jaundice from Disease outside of the Ducts. *External pressure* by tumors of the stomach, kidney, pancreas, or omentum; by tumors of the liver itself or enlarged glands in the fissure of the liver; by accumulated feces in the colon; by an abdominal aneurism; and finally by the pregnant uterus, may cause obstructive jaundice. In the large majority of cases this form of jaundice is due to *disease of the pancreas*, particularly carcinoma. The condition increases gradually in severity, varying with the degree of pressure, and becomes chronic, except when due to pregnancy or fecal accumulation; it may cause death or persist until such termination results from the primary disease. It is recognized by the absence of pain; the presence of disease in other localities, indicated by its peculiar symptoms and signs; the absence of a history of gallstones; and, finally, by the patient's age. Its

nature must be inferred from the symptoms and physical signs of disease in neighboring structures. Thus, enlargement of the lymphatic glands may be inferred from the presence of primary carcinoma in other organs of the body, or from the condition of the lymphatic glands in other parts. If they are the seat of malignant disease, it can usually be recognized. Cancer of the liver must be excluded by the absence of the characteristic symptoms—enlargement with jaundice, moderate fever, rapid emaciation, and short duration of the disease.

2. JAUNDICE FROM DISEASE OF THE DUCTS. Catarrhal, suppurative, and adhesive inflammation, and cancer or other tumors of the ducts cause jaundice.

The most common form is that due to *catarrhal inflammation* of the ducts. The jaundice comes on suddenly, at least within forty-eight hours after the onset of the symptoms; there is no pain, but the attack is attended by vomiting and other symptoms of mild gastritis, and is usually accompanied by itching. It follows indiscretions in diet, and occurs in young subjects. A definite cause for the gastritis can usually be found. The *diagnosis* is based upon the age; the association of the jaundice with gastritis, for which a definite cause can often be assigned; the absence of organic heart disease, or any lesion within the body that might be responsible for the jaundice; the moderate degree of jaundice; the absence of emaciation and symptoms of portal obstruction; and the presence of moderate enlargement without pain. It must not be forgotten that jaundice due to obstruction from gallstones, or to pressure from tumors outside of the duct, is characterized in its onset by symptoms similar to those just mentioned. It is often necessary to wait before giving an opinion; when there is a history of previous attacks of jaundice in a patient more than forty years of age, great caution should be exercised in giving a diagnosis.

When the jaundice is due to *suppurative inflammation of the ducts*—suppurative cholangitis—the infection is usually associated with a previous history of gallstones. It must not be forgotten, however, that other causes of jaundice, such as obstruction by external pressure, may invite an infectious inflammation of the ducts also. The jaundice runs a chronic course and is attended with fever and other symptoms of an infection. In *adhesive inflammation* there is a history of trauma from gallstones, and the infection is chronic. In *cancer of the gall-ducts* the advent of jaundice is slow, the course protracted; the symptoms are the symptoms of carcinoma, to which are often added the physical signs of an enlarged gall-bladder. (See Diseases of the Gall-ducts.)

3. JAUNDICE FROM OBSTRUCTION WITHIN THE DUCTS. Foreign bodies within the ducts—inspissated mucus, gallstones, and parasites, such as round worms or hydatid cysts—are the common causes of occlusion of the ducts with resulting jaundice.

Foreign bodies within the ducts cause jaundice either by direct obstruction or by the catarrhal inflammation which their presence excites. The symptoms occur suddenly in the former instance, gradually in the latter. The characteristic symptoms of *gallstones* precede the jaundice. The patient is usually a woman past forty years, with habits of life that pre-

dispose to the formation of calculi. Colicky pains occurring in paroxysms, intermittent jaundice varying in intensity, and an intermittent fever point to this form of obstruction.

Jaundice due to *lowering of the blood-pressure* in the liver, so that the tension between the bile-ducts and the blood-passages is altered, occurs suddenly, is light in degree, and is not attended by marked symptoms; it is due usually to shock or emotion.

Non-obstructive or Extrahepatic Jaundice. Jaundice is *non-obstructive* when (1) the function of the liver-cells has been suppressed, as in acute yellow atrophy of the liver; (2) when blood-destruction is in excess of the capacity of the liver to remove the product of destruction—the urobilin, as in certain forms of malaria, in pernicious anæmia, in certain fevers, and in other toxæmias. The onset of the jaundice is rapid, the general symptoms are more pronounced, particularly the cerebral symptoms. They occur simultaneously with the yellow discoloration. They are *infectious*, as in acute yellow atrophy of the liver and in Weil's disease. The *toxic* forms of non-obstructive jaundice are not severe; the skin is light yellow, and the discoloration may not even be observed by the patient, or cause pronounced symptoms. The blood is destroyed rapidly in these cases; and as it can not be disposed of by the liver, spleen, or kidneys, the transformed hæmoglobin is deposited in the tissues. In this class of cases the urine contains but little bile-pigment, but there is a large amount of urobilin and indican. The stools are not clay-colored.

Malignant or Infectious Jaundice—Acute Yellow Atrophy of the Liver. Acute diffuse inflammation of the liver with necrosis of the cells, characterized by jaundice and cholæmia, is most common prior to the thirtieth year and is said sometimes to follow fright. Many of the cases occur during pregnancy. The symptoms are local and general. The discoloration is first noticed after an attack of gastroduodenal catarrh, it occasionally extends over the entire body, and, as a rule, there is only moderate itching. After a continuance of these mild symptoms for from two days to two weeks the patient complains of headache, and delirium sets in with stupor and convulsions. The headache is attended with vomiting. Fever of moderate degree begins at the same time, although in some cases it is absent.

Although the jaundice is not intense, the effects upon the blood are early seen; hemorrhages underneath the skin and from the mucous membrane take place. In pregnant women abortion results, the hemorrhage following which may be excessive. The stupor and delirium are followed by coma, and death takes place in the first week; or coma may be preceded by the typhoid state, and the disease last longer than a week. The urine is bile-stained, and contains albumin and casts. It is diminished in amount, and is soon passed involuntarily. Leucin and tyrosin are always present. The latter may be seen in the sediment, although it is more marked when a few drops of urine are evaporated on a cover-glass. The bowels are loose and the clay-colored feces are passed involuntarily.

On examination the liver is found diminished in size; this may not be

appreciated by percussion in the anterior region, but in the axillary region the width is reduced by 1 to 2 inches. There may be some tenderness over the liver and over the ducts.

DIAGNOSIS. The data upon which a diagnosis is based are the age, sex, pregnancy, the rapidity of onset of cerebral symptoms following jaundice, diminution in the size of the liver, and the finding of leucin and tyrosin in the urine. Acute yellow atrophy must be distinguished from the jaundice of hypertrophic cirrhosis of the liver, which at times becomes malignant. Some observers have thought that acute yellow atrophy may supervene upon this form of cirrhosis, thereby causing malignant jaundice; but there is more fever than in atrophy, while leucin and tyrosin are not found in the urine. It must not be forgotten that all cases of jaundice may terminate suddenly with delirium, followed by coma, or in the development of the typhoid state.

In *phosphorus-poisoning* the hemorrhages, the jaundice, and diminution in the size of the liver are the same as in acute yellow atrophy. Gastric symptoms are more marked, and there is more distinct swelling of the liver in the early stages, which is also more persistent.

Weil's Disease. This infection, in which *jaundice* is the chief symptom, has been considered in the chapter on Infectious Diseases.

Yellow Fever. The account of the jaundice attending this infection is found in the chapter on Infectious Diseases.

Infantile Jaundice. Jaundice in infants is due to two causes: *congenital obliteration* of the ducts and *catarrhal inflammation.* It must not be confounded with the yellow discoloration of the skin due to an excess of coloring-matter in the blood which is not disposed of by the liver; or perhaps to patency of the ductus venosus, as suggested by Quincke.

In *congenital obliteration* of the gall-ducts jaundice rapidly ensues and deepens to an intense degree; hemorrhages occur, the child becomes stupid or comatose, and may have convulsions; death takes place in coma. There is rapid emaciation, and the liver and spleen are enlarged. In some cases life is prolonged for many months.

Simple *catarrhal jaundice* in infants is associated with moderate gastric disturbance. The jaundice is light; the conjunctivæ alone may be discolored. In infants malignant or infectious jaundice may be due to inflammation of the portal veins secondary to umbilical phlebitis. The jaundice develops after suppurative inflammation about the umbilicus, and is attended by fever. There may be some tenderness over the liver; frequently peritonitis develops at the same time. Pyæmic symptoms may set in, and pus may be found in other situations. If death does not ensue early, the jaundice becomes more pronounced and causes cutaneous and mucous hemorrhages. Convulsions and coma are apt to supervene before death. Jaundice in infants also occurs in *interstitial hepatitis of syphilitic origin.* The evidences of hereditary syphilis are seen in the skin and mucous membranes. The liver is enlarged, and there may be tenderness from perihepatitis.

Hepatic Fever. The occurrence of fever may be of importance in distinguishing the various forms of obstructive jaundice. Fever occurs

frequently in jaundice, but is significant in certain forms only. In *catarrhal jaundice* it is present for three or four days only, disappearing as the severe gastric symptoms subside. It is probably toxic. In *hepatic colic* with jaundice it is transitory and associated with chills and sweats. In jaundice from *obstruction* it occurs with the onset of primary or secondary infectious cholangitis. A peculiar type known as *intermittent hepatic fever* (see page 366) is often seen. The intermittent fever is associated with gallstones in the following groups: First, with each paroxysm of hepatic colic moderate fever and jaundice are present. The latter becomes more intense after each paroxysm, but disappears in a short time. The paroxysmal attacks may recur at intervals for years. Second, the hepatic colic is attended by distinct ague-like paroxysms of chill, fever, and sweat, after each of which the jaundice, which continues to the end, is more intense. Third, hepatic colic and gastric disturbance occur with fever, but without jaundice. The symptoms occur in distinct paroxysms. *Gallstones* are probably the cause in all these conditions, leading in some cases to chronic obstruction of the duct without infection.

When *infectious cholangitis* with or without gallstones is present, the symptoms are somewhat different, although the fever is of the same type. Thus (1) there is more tenderness in the hepatic region, with enlargement of the gall-bladder; (2) the paroxysms are more frequent; (3) jaundice is not so intense and not paroxysmal; (4) the patient is ill in the intervals, and there is wasting. There are no periods of either local or general improvement. The most important diagnostic point in cases of gallstone is the subsidence of all symptoms between the paroxysms of fever.

Intermittent fever of this character must be distinguished from *malaria*. The history of gallstones, with pain in the region of the liver, and the negative findings in the blood, are sufficient to establish the diagnosis. This type, usually associated with ball-valve calculus, may be confounded with septicæmia (blood-cultures, streptococcus and gonococcus, or examinations of pus). Endocarditis (same means of diagnosis), with recurring fever (Ebstein's disease) due to lymphatic tuberculosis (tuberculin test). It is this type of fever that is seen in syphilis, and such a cause must be excluded. Murphy calls attention to the rapid rise and fall of the temperature in such cases.

Hepatic fever also occurs in cancer when the neoplasm grows rapidly, in certain forms of cirrhosis, and in obstruction from other causes than gallstones. It is particularly common in suppurative inflammation of hydatid cysts, or after they rupture and discharge into the biliary vessels. Without previous knowledge of the hydatid cyst the diagnosis is almost impossible, save that the pain is less when the obstruction is due to this cause than in obstruction from the passage of gallstones.

Physical Diagnosis. The diagnosis by physical means of exploration is considered in the chapter on Physical Diagnosis. It is necessary to differentiate enlargement of the liver from a false enlargement of the organ due to diseases above the diaphragm as pleural effusions from accumulations of fluid between the diaphragm and the liver; and from enlargement below the diaphragm due to fecal accumulation in the colon, renal enlargement, or tumors of adrenal origin.

Physical diagnosis of diseases of the gall-bladder and gall-ducts yield the following results by the special methods of investigation.

1. *Inspection.*—Fixation or restriction of side.

2. *Palpation.*—The presence of pain, tenderness, swelling, or spasm.

(1) Simple palpation. (*a*) Tenderness at Mayo-Robson's point, at junction of outer one-third with inner two-thirds of a line drawn from the tip of the ninth right cartilage to the umbilicus. (*b*) Outline tumor. If inflamed gall-bladder, continuous with liver; if distended, movable, pear-shaped, neck toward liver, having a tendency to disappear and bob up again.

(2) Bimanual. Fluctuation—gallstone rubbing.

(3) Method of Glénard.

(4) Eight fingers. Pressure upon four placed over part by four of other hand, the fingers of hand not pressing relaxed. (Pottalschek.)

(5) Hooking fingers under rib in region of gall-bladder or pressing deeply in this region while patient takes full breath. (Murphy.)

(6) Deep prod with closed fist (Jordan-Lloyd) over gall-bladder excites pain in cholelithiasis.

3. *Percussion.*—Outline tumor; note if (*a*) continuous with liver, (*b*) an interval of resonance which may exist between the liver and gall-bladder tumors. Auscultatory percussion not conclusive. Note, however, that percussion must be employed in both upright and recumbent postures to detect displacements of liver.

4. *Auscultation.*—Friction of perihepatitis; gallstone crepitus (rare).

X-Rays. Of some value in the hands of Beck. Results usually considered doubtful.

Laboratory Diagnosis. *The Blood—Leucocytosis.* It is remarkable how few reports are made. More precise information is required on this important point. Leucocytosis is present in acute cholecystitis and cholangitis other than typhoidal. Its presence points to streptococcus, pneumococcus, or *Bacillus coli* infection, and excludes typhoid, malarial, and tuberculous infections. It is often absent in amœbic abscess, but does not exclude multiple abscess of the liver. If lymphocytes are in excess, syphilis is suggested.

Iodophilia. Locke and Cabot found positive reaction in 5 of 7 cases of gall-ducts. Their studies, however, showed positive results in a very large percentage of cases in which suppuration was present. One can place about the same value upon iodophilia as upon leucocytosis.

Red-blood Count. This should be made repeatedly. In jaundice the number of red cells falls rapidly, and the degree of reduction should guide one in the indications for operation.

Coagulation-time. A close watch of the blood should be made with Wright's tubes. The normal coagulation-time is two to four minutes. In jaundice the blood may coagulate so slowly that eight to ten minutes elapse before clotting is completed.

Tuberculin Test. Jaundice may be due to enlarged lymphatic glands pressing on the ducts. Recurrent fever (Ebstein's) attends it. It may simulate gall-bladder infections. A tumor of the liver so situated as to resemble an enlarged gall-bladder may be tuberculous.

Congestion of the Liver.

Active Congestion. The liver is enlarged and may be tender. Weight and fulness, and often a painful sense of distention are complained of. Active hyperæmia may follow a chill or suppression of the menses; but the commonest exciting causes are indiscretions in diet and the free use of alcohol or highly seasoned food, followed by an attack of acute gastro-intestinal catarrh. The disease is quite common in the tropics from suppression of perspiration. It is recognized by the occurrence of symptoms of acute gastritis, with enlargement, pain and tenderness of the liver, and sometimes a moderate degree of jaundice.

Passive Congestion. Passive hyperæmia also leads to enlargement of the liver which may occasion a sense of weight and fulness, but pain is not present. The organ is often not tender, although sometimes distinctly so; the edges are smooth and indurated. Pulsation of the liver, which is sometimes present, is detected by placing the hand over the surface of the liver, when with each impulse of the heart the organ can be felt to expand. The symptoms of the cause of the passive congestion combine with those due to the enlargement, and in addition we have symptoms of obstruction to the flow of blood in the portal circuit. Pick has described what he calls "pericarditic pseudocirrhosis of the liver" in which, owing to passive congestion from pericarditis, the liver becomes cirrhotic, and ascites and other symptoms of cirrhosis predominate in the clinical picture. Since adhesive pericarditis is difficult to diagnosticate, the cases are likely to be mistaken for true cirrhosis. The condition is not very uncommon in children.

Passive congestion occurs in organic heart disease after compensation has failed and the right heart is dilated, the organ rapidly becoming congested because of its proximity to the overfilled ventricle. Mechanical congestion also takes place in emphysema of the lungs, in fibroid phthisis, and in the presence of intrathoracic tumors pressing upon the vena cava. The recognition of passive congestion is not difficult. The symptoms due to enlargement (see Objective Symptoms) and the symptoms due to portal obstruction point to the true nature of the morbid process.

Portal Congestion. Occlusion and overfilling of the branches in the liver occur in portal congestion, and most typically in cirrhosis of the liver. The circulation in the liver is interfered with; the blood is thrown back into the portal vein and overfills the vessels of the portal area. As a result we have (1) congestion of the mucous membrane of the stomach and bowels, with the symptoms of gastro-intestinal catarrh. (2) Dilatation of the veins, chiefly the hemorrhoidal, giving rise to hemorrhoids. (3) Ascites. (4) Hemorrhages. The *hemorrhages* may occur in any part of the gastro-intestinal tract. Hæmatemesis and intestinal hemorrhage are seen separately or combined. The vomited blood may be small in amount; often it is mixed with mucus. In some cases large and even fatal hemorrhages take place either from the mucous membrane of the stomach or from the veins about the œsophagus, which often become varicosed in cirrhosis. Intestinal hemorrhages may originate in enlarged

hemorrhoidal veins, an intestinal ulcer, or the intact mucous membrane. (5) Enlargement of the spleen. (6) Changes due to the collateral circulation. If complete collateral circulation is established, the above symptoms may not ensue. The *collateral circulation* may be through deep-seated or through superficial veins. In the latter case the *external veins* of the abdomen are enlarged; the epigastric and mammary veins become prominent, and the veins about the umbilicus may become so enlarged and prominent as to form a radiating tumor to which the term *caput Medusæ* has been applied. The venules along the line of attachment of the diaphragm in the lower thoracic zone are over-distended and may be the seat of pulsation.

In consequence of the portal overfilling, the enlarged terminal branches of the vein press upon contiguous structures, interfere with the circulation of the blood in the major vascular system of the liver, and invite catarrh of the terminal ducts, with obstruction, and hence *jaundice*. This is seen quite frequently in passive congestion of the liver, rarely in cirrhosis.

Abscess of the Liver.

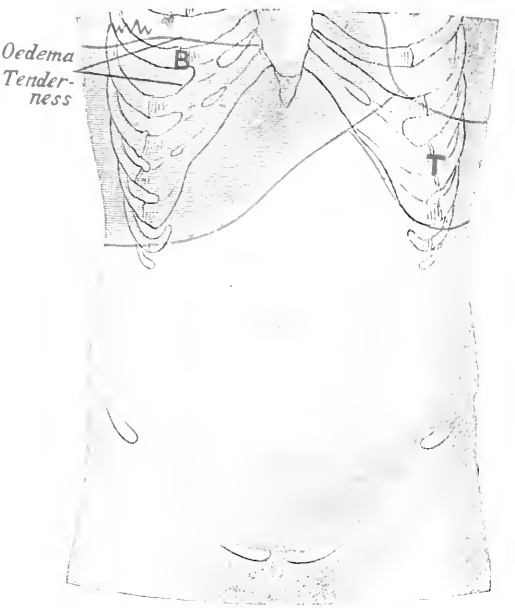
Two forms are seen: so-called *single* or *tropical abscess*, in which one or two abscesses are found; and *multiple abscesses*, found throughout the liver structure. The single or solitary abscess usually occurs in the course of dysentery, and, in all probability, in the amœbic form only; it may also be due to traumatism, particularly in children. Multiple abscesses occur secondarily to inflammation somewhere in the portal area. Inflammation and abscess about the rectum, inflammation of the appendix, ulceration anywhere in the gastro-intestinal tract may be followed by multiple hepatic abscesses. The abscesses, however, are not directly produced by emboli, as in the case of amœbic abscess, but after inflammation of the portal vein or *suppurative pylephlebitis*. Multiple abscesses of the liver also follow obstruction and infectious inflammation of the biliary passages (*suppurative cholangitis*).

Tropical or amœbic abscess varies in its clinical course. In a typical case the clinical picture is that of general suppuration developing in the course of, or soon after, an exacerbation of amœbic dysentery with local symptoms referred to the liver.

Symptoms. The *general symptoms* are those of intermittent fever, paroxysms of which may occur daily or only every second day, attended by chill, fever, and sweat. The fever may be remittent or continuous. The complexion in tropical abscess of the liver is peculiar, as all writers upon tropical diseases agree. The skin is sallow, the complexion muddy, the face pale. Through this a slightly icteroid tint may be seen, and the conjunctivæ are bile-tinged. Distinct jaundice is rare. On account of the enlargement the patient complains of weight and fulness in the region of the liver and suffers from cough and dyspnœa; vomiting, although not severe or even constant, is sometimes present. The appetite is lost and nausea at the sight of food is pronounced. The condition of the bowels varies with the state of the intestinal tract at the time of the hepatic complication. The dysenteric symptoms may subside entirely or

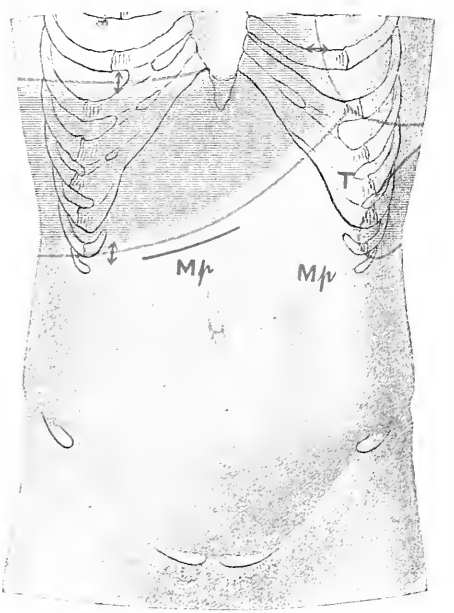
PLATE XLII.

FIG. 1.



Abscess of the Liver.

FIG. 2.



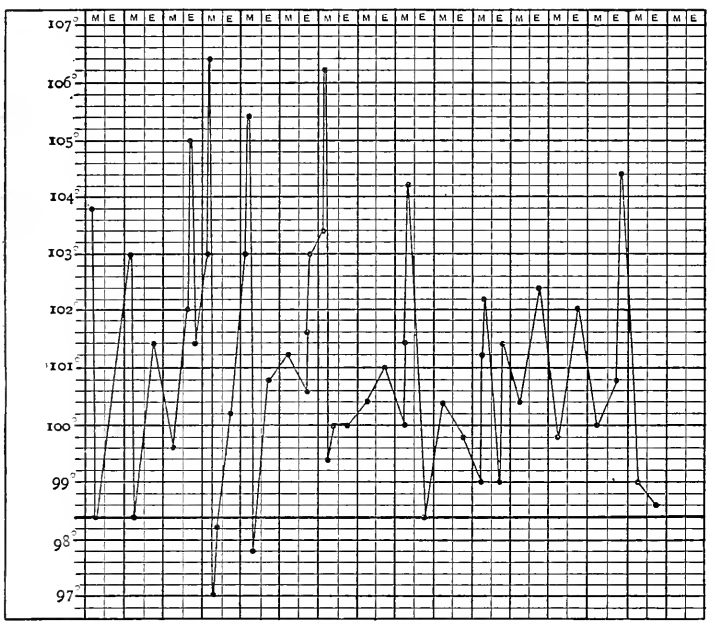
Hypertrophic Cirrhosis of the Liver with Enlargement of the Spleen.

they may continue; often there is only constipation with the passage of mucus and hardened feces. In an obscure case a study of the stools should be made, as the detection of amœbæ in the mucus or in the feces may point to the true conclusion.

Locally there is pain over the liver, which may be referred to the region of the right or the left lobe. It may be seated in the fifth or sixth interspaces anteriorly, or at the ninth and tenth ribs posteriorly. There may be pain in the right shoulder mechanism. The pain may be paroxysmal, or it may be intense and persistent.

Physical Signs. (Plate XLII., Fig. 1.) The enlargement may be uniform if the abscess is central; on the other hand, the enlargement may extend chiefly upward in the anterior, the axillary, or the posterior region. When the convex surface of the right lobe of the liver is affected, the enlargement is usually upward. An abscess in the lower portion of the right lobe produces a downward enlargement, and the lobe of the liver

FIG. 379.



Intermittent fever in abscess of the liver.

can readily be detected on palpation. When the abscess is limited to the left lobe of the liver, and is situated about the suspensory ligament, the enlargement may be seen below the xiphoid cartilage. The mass may extend to the umbilicus and project forward; sometimes it is sufficiently large to cause eversion of the ribs of each side and render the entire epigastrium unusually prominent. The surface may become reddened and œdematous. Over the tumor, which is hard at first but later becomes soft, there is tenderness on palpation, and there may be, as in other situations, fluctuation.

The irregular enlargement is made out by percussion. The outline may be difficult to map out on account of secondary pleural effusion, or secondary pleural inflammation with the development of hepatopulmonary fistula, causing dulness posteriorly. If the case has been seen from the first, a friction-sound may be heard, followed by the physical signs of effusion. The *x*-rays have been successfully used to determine the presence of an irregular enlargement of the liver.

Atypical cases are characterized by the absence of general symptoms or local signs. Fever may be absent entirely, exhaustion alone being present, which might be accounted for by the previous dysentery. Pronounced anæmia due to the dysentery may be the most marked symptom; inflammation of the joints, or neuritis, may be present. In a case under my care the only symptom for a long time, with the exception of anæmia and loss of appetite, was severe pain in the sixth interspace. In other instances there are no liver-symptoms whatsoever. General symptoms of infection, or an irregular, or even a continued fever, the cause of which can not be ascertained, may alone be present. In one of my cases there was moderate continued fever, with loss of appetite and dyspeptic symptoms; there was no diarrhœa; no cause could be found for the fever, although a slight enlargement of the liver was noted. The patient slipped out of the ward and went down into the yard to smoke; on his return he was seized with an intestinal hemorrhage which could not be checked and resulted fatally. At the autopsy a large abscess of the liver was found and there was ulceration of the rectum, from which the intestinal hemorrhage had taken place.

Perforation of Hepatic Abscess. The first intimation of the existence of an abscess is sometimes obtained from the occurrence of rupture. If perforation takes place into the peritoneum, it is not likely that the cause can be determined during life. The abscess frequently ruptures through the diaphragm into the pleura, and thence into the lung. An empyema may be set up, the true source of which may not be ascertained unless the pus is examined. The physical signs are those of empyema—dulness or diminished resonance, absence of fremitus and vocal resonance, diminished breath-sounds, impaired movement, cough, and dyspnœa. When the lung is infected, the physical signs may resemble those of consolidation. We find dulness, bronchial breathing, and increased tactile fremitus. A harassing, convulsive cough occurs, and sooner or later expectoration of a reddish-brown, brickdust-colored material resembling anchovy sauce. This characteristic expectoration is decisive. It contains amœbæ, and, in addition to blood-pigment and corpuscles, orange-red crystals of hæmatoidin, cholesterin-plates, and leucin and tyrosin. When the abscess perforates into the stomach or bowel, the discharge from either may be of the above-mentioned nature. Perforation into the pericardium is usually followed by immediate death.

Diagnosis. The diagnosis is usually not difficult in typical cases. Under all circumstances attention must be paid to the facts bearing upon the ætiology, and to the association of general and local symptoms. In the presence of general symptoms of suppuration hepatic abscess may be mistaken for *malaria*. Examination of the blood and the result of treat-

ment by quinine would establish a diagnosis of malarial fever. It is difficult sometimes to determine whether the abscess is in the abdominal wall or in the liver proper, or whether it is situated beneath the diaphragm. If the liver is movable with respiration, the two former conditions may be excluded. A *mural abscess* is not influenced by respiration, while in subdiaphragmatic abscess the movement is impaired. *Suppuration of a hydatid cyst* can not be distinguished unless it has been known beforehand that a simple hydatid was present in the liver. Under such circumstances, if suppuration occurs, it is likely to be confined to the cyst. Abscess of the liver must be distinguished from *gallstones* attended by intermittent fever without suppuration. While the distinction is difficult in many cases, yet the history of the case, the association of jaundice becoming intensified after each paroxysm, and the good general nutrition of the patient point to gallstones. Abscess of the liver is of shorter duration than cholelithiasis, and its primary cause can usually be ascertained by examination of the rectum, or the discovery of suppuration in other parts of the body.

Aspiration. Exploratory puncture must be employed in many cases, and it can usually be done with safety. The region in which the enlargement is greatest, or at which the swelling is most prominent is the point of election. In abscess secondary to dysentery a brownish-colored pus resembling anchovy sauce will be withdrawn. It may be of a peculiar odor, and, on examination, amœbæ may be found. If there is no point of election, the needle may be introduced in the lowest interspace in the anterior axillary, or the seventh interspace in the midaxillary line. A fairly large-sized needle should be used. Pus may be present, and yet not be reached by aspiration.

Suppurative Pylephlebitis.

Abscess of the liver may be due to *pyæmia*. It may be a part of general or of portal pyæmia. Parasites and foreign bodies, as well as gallstones, may excite an abscess. The echinococcus cyst may suppurate, or round worms may penetrate to the liver and cause suppuration.

The symptoms of *suppurative pylephlebitis* and of *pyæmic abscess* are general and local. Jaundice is more common than in solitary abscess; pain and tenderness are more pronounced over the liver, which is uniformly enlarged. With the enlargement of the liver and jaundice we have the usual symptoms of pyæmia. Sometimes the fever is distinctly intermittent; at other times it is irregular and septic in character.

Cirrhosis of the Liver.

A diffuse interstitial inflammation of the liver, frequently with atrophy of the organ, which in the large majority of cases is caused by irritants entering the portal circulation through the stomach. Among these irritants alcohol is the most common, particularly the stronger liquors, as gin and whiskey. Other irritants, as spices used to excess, may likewise cause the diffuse inflammation. Cirrhosis of the liver may also

be a sequel to the infectious diseases, notably scarlatina, and may be excited by malaria. The most frequent result of alcoholism upon the liver is the *atrophic* form of cirrhosis. *Hypertrophic* cirrhosis is a common disease, however, and is usually due to alcoholism, though it bears a closer relation to infections than does the atrophic form. There is good evidence that toxic matters of various kinds other than alcohol, when absorbed from the digestive tract, lead to cirrhosis of the liver, and it is uncertain whether the substances contained in the alcohol itself or toxic substances produced by the gastro-intestinal disturbance which the alcohol sets up cause the interstitial over-growth.

Hypertrophic biliary cirrhosis is due to obstruction of the bile-ducts, followed secondarily by over-growth of the connective tissue. Cirrhosis of the liver may arise in the course of *syphilis;* and finally a secondary cirrhosis of the liver which develops in the course of passive congestion of that organ is described under the name of *nutmeg-liver.*

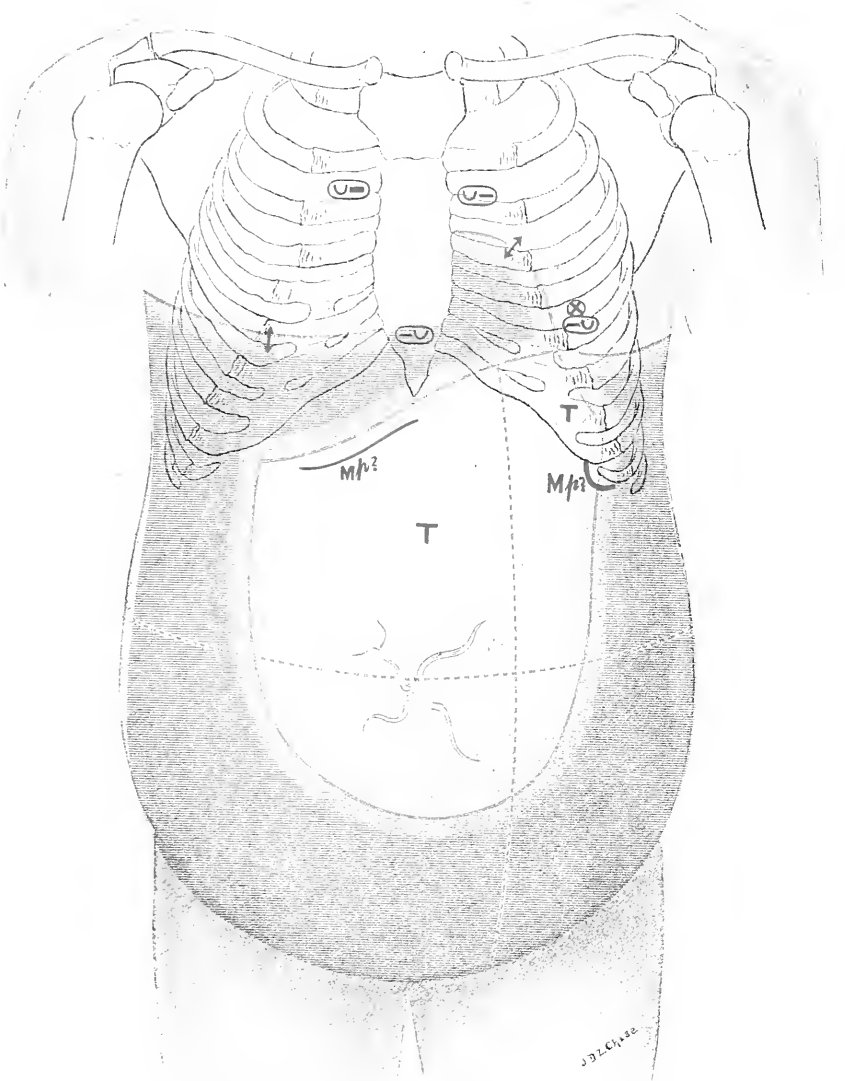
Atrophic Cirrhosis. Cirrhosis of the liver of the atrophic form, due to alcohol, presents various clinical features. In the first place, it may exist without causing any symptoms whatever during life, and be discovered after death from other causes ; or it may present no symptoms until an accident occurs in the course of the disease, as hemorrhage from some portion of the collateral circulation. In both cases the symptoms are absent because the collateral circulation is complete. If this is incomplete, however, grave local and general symptoms ensue. Before detailing these symptoms it may be well to state that the occurrence of one symptom, which we have termed accidental, may lead to the inference that cirrhosis of the liver is present, particularly if the patient has been an alcoholic. This symptom is *hemorrhage.* It may be from the stomach, causing death at once or after repeated loss of blood ; it may also take place from the intestine.

Symptoms. The symptoms are *general,* due to interference with the nutrition ; and *local,* their extent depending upon the degree of obstruction to the portal circulation. They have been divided into those of the *first stage,* or stage of enlargement, and those of the *second stage,* or stage of contraction. This division is, however, not usually recognized at present. There is no real stage of hypertrophy ; and while congestive enlargement does sometimes occur, it is by no means constant. Cheadle's experience is most convincing on this point. There are, however, two sets of symptoms.

During the *first stage* the symptoms are those of gastritis with enlargement of the liver. The gastric symptoms are morning retching or vomiting, with discharge of mucus, associated with other symptoms of gastric catarrh, as loss of appetite, nausea, tenderness in the epigastrium, eructations, constipation, and loss of flesh and strength. The liver may be found a little swollen.

The symptoms of the *second stage* are more severe and are due to obstruction of the portal capillaries. On examination *ascites* is detected. The transudate may be enormous, causing monstrous distention with pouting of the umbilicus. The *spleen* is found to be enlarged, extending over twice or three times the normal area of percussion. If ascites does

PLATE XLIII.



Cirrhosis of the Liver with Ascites.

not interfere, the edge of the spleen can be readily made out. The portal obstruction causes secondary gastro-intestinal catarrh, if it was not already present on account of the alcoholism. Although constipation is usually present, there may be persistent diarrhœa, usually lienteric and occurring in the morning only. Hemorrhages may take place at any time from the gastro-intestinal tract, either from the stomach or from the intestine. Not infrequently they occur from the œsophagus on account of varicosity of the veins at the junction of the œsophagus and cardiac end of the stomach. Hemorrhoids are always present and may bleed at each stool. Jaundice is not the rule, and, if present, is usually slight and due to the duodenal catarrh.

The **general symptoms** of cirrhosis, and particularly the symptoms of the later stages, are striking and diagnostic. The nutrition is much impaired. The patient who, in the large majority of cases, had been corpulent, becomes emaciated. The skin changes in color and becomes of an earthy-gray or dirty sallow hue. The capillary venules of the face are dilated; the distended capillaries on the nose are distinct. Later, ecchymoses may occur in the skin, and hemorrhages take place from the mucous membranes and into the retina. Debility ensues; œdema of the ankles is almost sure to occur, and sometimes general anasarca may take place. It is extremely rare to have fever unless complications occur. The pulse is small and becomes more rapid than normal; the heart-sounds grow weaker. The skin may be the seat of eruptions, and chronic skin diseases of various kinds develop.

The *urine* throughout the disease presents nothing characteristic; as ascites develops, it becomes scanty and dark, and loaded with urates and uric acid. In rare instances it may contain sugar, and if the uric acid is in excess, albumin.

The *collateral circulation*, which develops in order that the portal blood may reach the right heart, takes place in various ways. First, communication may be formed between the veins of the mesentery and those of the posterior abdominal walls; second, between the coronary veins of the stomach and the veins of Glisson's capsule and the phrenic veins; third, between the hemorrhoidal and the inferior mesenteric veins; fourth, between enlarged veins occupying the position of the obliterated umbilical vein in the ligamentum teres, and the epigastric and mammary veins.

Physical Signs. (Plate XLII., Fig. 2, and Plate XLIII.) It may be difficult to elicit the physical signs before paracentesis is performed on account of the extensive ascites. The liver will be found to have undergone contraction; although diminution in the area of dulness is not by any means so absolutely confirmative of contraction as the opposite condition is of hypertrophy. Percussion should be performed several times, because the distended intestinal coils may obscure the findings. A well-defined caput Medusæ is often present.

Diagnosis. The diagnosis is usually not difficult if the complete picture of the case is presented. It can not be established positively without definite knowledge of the cause. If the patient comes under observation after ascites has developed, the diagnosis is more difficult. It must, in the majority of cases, be based upon exclusion of heart, lung, and kidney

disease. A history of alcoholism and the presence of other symptoms of liver disease point to the hepatic origin of ascites. Ascites may be due to other causes within the abdomen, notably *chronic peritonitis*, exclusion of which is sometimes difficult. General tenderness, absence of distention, and splenic enlargement point to peritonitis. The *fatty cirrhotic liver* may present symptoms similar to those of the atrophic form, except that the organ is enlarged.

In a study of a case of cirrhosis of the liver a judgment as to its nature may be, in a measure, confirmed by the presence of other phenomena due to the same cause. Very frequently we have, at the same time, cirrhosis of the kidneys and sclerosis of the arteries, with secondary atheroma, both of which have led to hypertrophy of the heart. Strümpell refers to the association of cirrhosis and chronic tuberculous peritonitis, and thinks the former is the primary lesion.

The *duration* can not be determined accurately, as the onset is usually insidious. After the ascites appears the duration may vary from six to eighteen months, depending largely upon the completeness of the compensatory circulation. Death usually occurs from intercurrent disease or progressive exhaustion. In not a few cases cerebral symptoms occur. In addition to the cirrhotic cachexia, the sudden occurrence of coma and convulsions, preceded by delirium, may ensue; the cause of this is not fully known. It must be borne in mind that the occurrence of these symptoms in an alcoholic subject may be due to a cirrhosis, the presence of which had not been suspected during life.

Hypertrophic cirrhosis, or so-called biliary cirrhosis, presents a somewhat different picture. In the first place, there often is a history of gallstones, or obstruction of the duct from other causes. The patients are frequently young adults, and in general the age of the subjects is less than in the atrophic form. The liver is uniformly enlarged, and the surface is smooth and strikingly indurated. There are weakness and loss of appetite. Jaundice ensues very early, or may be the first symptom. It increases and persists throughout the course of the disease. Ascites is somewhat less common than in the atrophic form and is often very slight or absent altogether; only very rarely, indeed, does it become a pronounced symptom. The enlargement and jaundice may continue for months or even years without the development of grave symptoms.

Fever may, however, set in at any time, being in all probability due to the biliary obstruction. It comes on in distinct paroxysms associated with increase of jaundice. Sometimes distinct increase in the enlargement of the liver and exacerbation of the general symptoms grow temporarily quite marked; the temperature rises to from 102° to 104° F. In some cases the tongue becomes dry and brown, the pulse rapid, and all the symptoms of grave febrile jaundice ensue, the case ending fatally, with severe nervous symptoms. At any time during the course of the disease the patient may be seized with convulsions, followed by coma and death. It is now generally considered that in this form of cirrhosis the liver remains large; in other words, that there is no tendency to ultimate contraction. There is, however, not uncommonly more or less ascites, though the amount of fluid is often so slight as to escape clinical obser-

vation. Enlargement of the spleen is very common, and is probably due rather to a participation of the spleen in the cirrhotic process than to passive congestion from portal obstruction.

The **diagnosis** is often difficult. Gradual and persistent jaundice continuing for a long time without cause, and associated with persistent enlargement of the liver without symptoms of portal obstruction in a non-alcoholic subject, points pretty certainly to hypertrophic cirrhosis of the liver.

Syphilitic Disease of the Liver.

Syphilitic disease of the liver may result either in cirrhosis or in the development of gummata. *Syphilitic cirrhosis* presents the same symptoms as the alcoholic form. The history, the marked irregularity on the surface of the liver, and the existence of syphilis elsewhere may lead to a diagnosis of the true condition.

In *congenital syphilitic disease* of the liver the inflammation is diffuse; the liver is enlarged and hard; the surface is smooth; there are usually syphilitic lesions in other organs; the patient presents syphilitic eruptions, and has the well-known wizened appearance that belongs to the disease.

Syphilitic gummata in the liver may exist without presenting any symptoms whatsoever, or they may reveal their presence by pain and a localized swelling and discomfort which call the patient's attention to the region, particularly if his general health is reduced at the same time. Tumors are situated in the left lobe, in the median line, or along the margin of the ribs. The pain is usually localized in this region, but may extend more or less over the entire liver, particularly if there is general perihepatitis along with other evidences of syphilis; the latter are not always present, however. If the temperature is taken frequently, a moderate febrile range will be observed. It may not rise above 100.5 ° F.; but in the absence of other causes this is a valuable diagnostic symptom.[1] In other instances the gummata may grow in such a situation as to interfere with the portal circulation or press upon the gall-ducts. The latter is very rare. Gummata that are accessible to palpation appear as enlarged bosses, giving the sensation of flattened hemispheres. Sometimes several separate elevations can be made out on the surface of the enlarged organ. To determine the exact nature of the lesion is often very difficult. The symptoms may point conclusively to hepatic disease. Knowledge of the presence of syphilis aids in the diagnosis. If without a syphilitic history there are scars in the throat, nodes on the bones, or other signs of syphilis, the diagnosis will be tolerably certain. Severe pain is more prominent in syphilis than in cirrhosis, and the nodules of syphilis are very different from the granular surface of cirrhosis. Amyloid disease is often associated with the actual syphilitic lesions, and much of the enlargement may be due to this and persist after treatment has caused absorption of gummata.

[1] "The Diagnostic Importance of Fever in Late Syphilis." Musser, University Medical Magazine, October, 1892.

Fatty Liver.

The symptoms of fatty liver are not marked. The physical sign is a uniform enlargement extending in all directions. On palpation the edges can be felt; they are rounded and smooth. They are soft at first, but later become indurated. Fatty liver may be followed by cirrhosis after a period of alcoholism. The general symptoms are those of the primary disease. Fatty liver occurs in gouty subjects, but is notably present in wasting diseases, in tuberculosis, in chronic hip-joint disease, and in amyloid disease of the liver.

Fatty liver sometimes follows the congestion of the liver which is present in the course of organic heart disease. It is not a true fatty liver, but a fatty cirrhosis. There is increased fatty degeneration with an over-growth of connective tissue. This form is associated with heart and kidney disease. On palpation the edges of the liver are indurated. The liver may undergo diminution in size later, and the symptoms of cirrhosis ensue.

Amyloid Disease of the Liver.

Enlargement of the liver without pain is often due to amyloid disease. Similar disease is found in other organs, and there is present, to point to the nature of the enlargement, syphilis, bone disease, prolonged suppuration, or tuberculosis. In amyloid disease the pallor of the patient is great; the face may be swollen, and the ankles slightly œdematous. The spleen is enlarged, the urine albuminous and abundant, but of moderate specific gravity. A history of syphilis is an important point in establishing the diagnosis. Fatty liver can readily be distinguished from amyloid disease by palpation. In the latter the surface is smooth, but very much indurated.

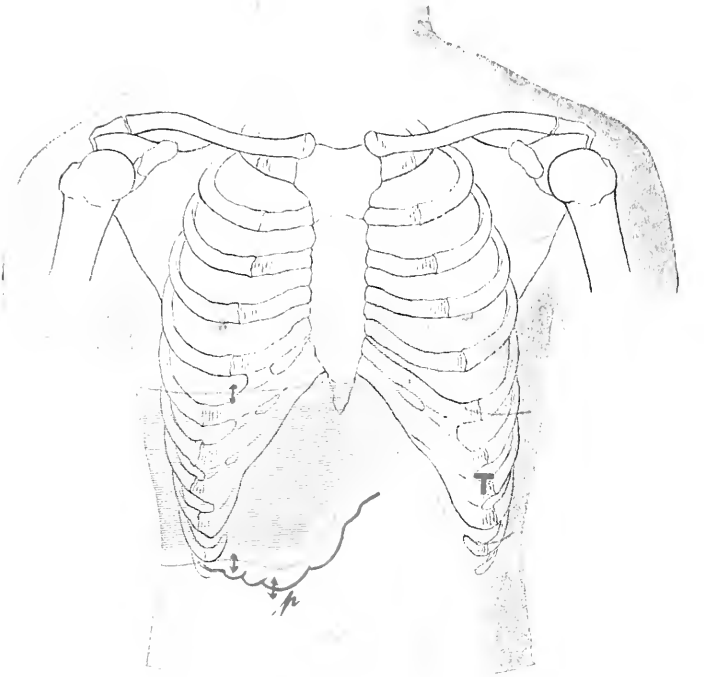
Cancer of the Liver.

The ætiological factors upon which the diagnosis of cancer is based are: the age of the patient—most frequently between the fortieth and sixtieth year; the sex, women being more frequently attacked; and heredity. The disease is nearly always secondary to cancer in some other situation; consequently, in cases in which symptoms point to cancer of the liver, search must be made for the primary lesion elsewhere. The most frequent seats are the rectum, the uterus, the stomach, and the remainder of the gastro-intestinal tract. Sarcoma of the liver, chiefly of the melanotic variety, is not uncommonly secondary to melanotic sarcoma of the eye. Further ætiological influences that may bear upon the diagnosis are: (1) the occurrence of gallstones, which act as the exciting cause in the development of primary cancer of the ducts, thence spreading to the liver; (2) the occurrence of trauma.

Symptoms. The symptoms of cancer of the liver may be due to increase in the size of the liver; to pressure of the growths upon the ducts or terminal portal vessels; and to the general effects of carcinoma upon the system—the *cachexia.* They are, *jaundice,* which is not very deep unless the common duct is affected; *ascites,* which is always present

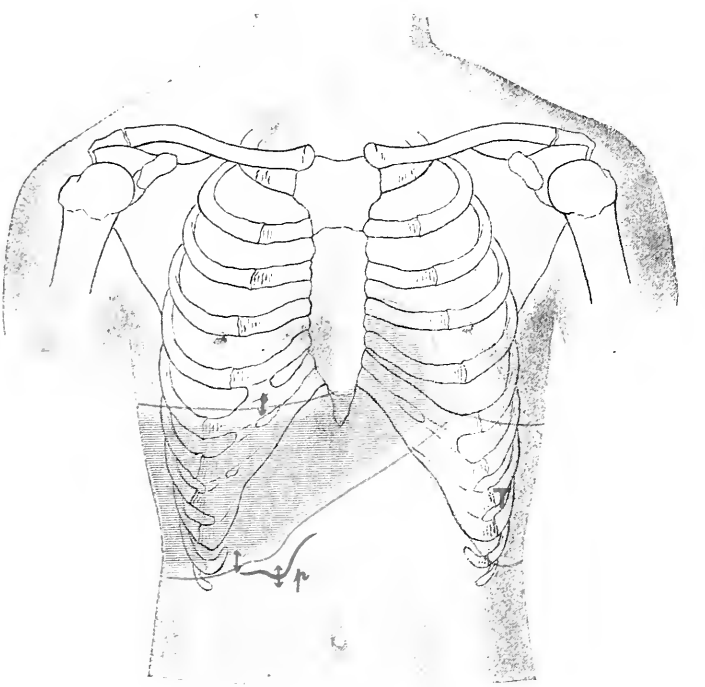
PLATE XLIV.

FIG. 1.



Carcinoma of the Gall Bladder with Involvement of the Liver.

FIG. 2.



Enlargement of the Gall Bladder.

in the atrophic forms, but may be absent when the liver is enlarged; the general symptoms of rapid *emaciation* and prostration; and in some instances *moderate fever*. Fever attends the rapidly growing cases; it is usually continuous, but may be intermittent, especially if there is suppuration or suppurative inflammation of the ducts. It is a well-known fact that *gallstones* are of common occurrence in patients suffering from cancer in any location whatever. The symptoms of biliary calculus or of obstruction may attend those of secondary cancer of the liver, and the stone has an ætiological significance.

In many instances *secondary cancer* of the liver may be present without symptoms to attract attention to this organ during life. If cancer in certain other regions has continued for the usual period of time, it is almost certain that at the autopsy cancer of the liver will be found.

Physical Signs. (Plate XL., Fig. 2; Plate XLII., Fig. 2; and Plate XLIV., Fig. 1.) The liver is enlarged and its surface irregular. By palpation the organ can be made out extending below the margin of the ribs. The edges are irregular, and bosses can be distinctly felt on the surface. In rare cases one or two masses only may be present, growing out of the substance of the left lobe of the liver and causing a large tumor below the sternum. The nodules are usually hard, but sometimes may be soft and even fluctuate. After emaciation becomes marked, the nodules can be seen as well as felt near the surface of the skin, and their number distinctly made out. The abdomen is distended.

The liver moves with respiration. Progressive enlargement can be noted while under observation. The enlargement can be well defined by percussion; and while the surface is irregular, the general shape of the dulness corresponds to that of the liver. The increased size and inflammation of the capsule cause a sensation of weight in the hepatic region and pain which may be intermitting in character. The nodules may be tender on palpation. The superficial veins are enlarged.

In not every instance do we find enlargement. In some cases the cancer is associated with cirrhosis of the liver, or may itself be of nodular type, and in the course of the disease undergo shrinkage. The liver is then normal or diminished in size.

Diagnosis. The diagnosis of cancer of the liver is not difficult when the changes in the liver can be made out on palpation and percussion. In rare instances, in which the liver is smooth, it may be mistaken for fatty or amyloid liver. A definite cause can usually be assigned for the latter; while the occurrence of jaundice, the rapid increase in the size of the liver, and the general symptoms of the cancerous cachexia indicate cancer of the liver. A *syphilitic liver* with irregular gummata may cause serious doubt; the history of the case and other signs of syphilis aid in the diagnosis, although locally the condition may exactly simulate carcinoma. The jaundice, however, is neither so frequent nor so deep in syphilitic gummata, and the cachexia does not ensue; but the therapeutic test may be necessary to make a diagnosis.

In *hypertrophic cirrhosis* of the liver the jaundice is deep and the liver is enlarged; but there is little wasting or anæmia. The surface of the liver is smooth; there are certainly no bosses, and the organ is painless.

Ascites is more common in cirrhosis; the patient is usually affected earlier in life than in cancer.

In a large growing cancer one or two of the nodules may suppurate and simulate *abscess of the liver.* Abscess follows a definite cause usually, and occurs in middle life; cancer is secondary to disease in other organs and occurs usually in late life. The results of aspiration differ in each. Moreover, a history of dysentery, the occurrence of pain, of profound anæmia, of pronounced hectic fever with irregular enlargement of the liver, but without jaundice or cachexia, point to abscess.

Cancer of the liver may be simulated by cancer of organs in close proximity to the liver, as the *pancreas*, the *pyloric end of the stomach*, or the *colon*. In addition to the fact that the usual symptoms of pyloric cancer are present, it will be found that jaundice occurs late. Cancer of the pyloric end is less freely movable with respiration unless it becomes adherent to the liver. *Cancer of the pancreas* also presents difficulties; a tumor in the midcostal region, however, with vomiting and the early development of jaundice, *before* the liver has become enlarged or nodular, and associated with other characteristic symptoms, such as intestinal dyspepsia and fatty stools, points to the pancreas as the primary seat of the disease. *Cancer of the omentum and colon* are not modified by respiration. The percussion-note over them is different; they frequently extend beyond the liver-confines and are associated with symptoms of obstruction of the bowels. *Fecal accumulation* in the transverse colon must not be mistaken for cancer of the liver. The large masses adjacent to the liver may closely simulate cancerous nodules. In doubtful cases the colon should be emptied.

Cancer of the liver and hydatid disease must not be confounded. The tumor in *hydatid disease* is usually single; it is large, and may fluctuate or yield the hydatid fremitus. It causes irregular enlargement of the liver when the tumor presents in the epigastrium or along the margin of the ribs. It is painless. Aspiration yields the characteristic hydatid fluid.

Cancer of the bile-ducts can not always be distinguished from cancer of the liver. Jaundice early in the course of the disease, in a person who has had gallstones, followed by enlargement of the liver and gall-bladder, in the absence of primary disease elsewhere, suggests cancer of the gall-bladder or ducts. This is more or less confirmed if the smooth and painless gall-bladder becomes hard, irregular, and tender on pressure.

Hydatid Disease of the Liver.

Hydatid disease is comparatively rare in this country, but, in my experience at least, it is undoubtedly increasing in frequency. Without any increase in the opportunities for observation, I have seen seven cases within the last two years, compared with the same number during the five preceding years. The disease occurs in people who live with dogs. It may occur at any age, but is most common in adult life. It is very rare before the fifth year.

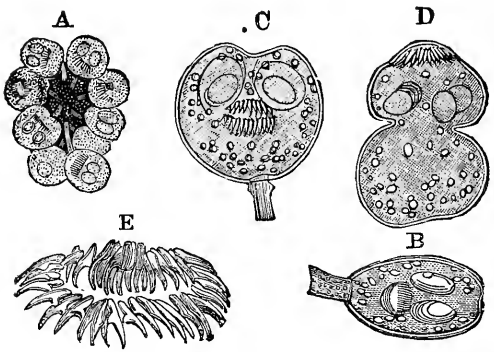
Symptoms. The *general symptoms* are negative; the nutrition does

not suffer unless the enlarged mass interferes, by its pressure, with the physiological acts of digestion and assimilation. If suppuration sets in, the general symptoms of abscess of the liver arise. Jaundice is more common than in tropical abscess. The abscess may perforate into one of the adjacent hollow viscera, or into the pleura and bronchi; it may perforate externally; and finally it may perforate into the pericardium or vena cava, and cause death. If perforation takes place in the biliary passages, obstructive jaundice arises, with secondary suppurative cholangitis. Urticaria sometimes follows rupture or aspiration of the cysts. This is not of diagnostic significance, except that it may point to rupture of the cyst. The *local symptoms* depend upon the size of the *tumor*. Small cysts may be present without causing any disturbance. Large and growing cysts cause signs of tumor, with great increase in the size of the liver. The *physical signs* depend upon the situation of the tumor. It may be found in the median line above the umbilicus, causing a distinct prominence, tense and firm, which sometimes yields fluctuation. Quite often the tumor grows at the suspensory ligament, pushing the diaphragm upward, dislocating the heart and causing an increased area of dulness in the left upper quadrant. In this position it may simulate a pancreatic cyst or effusion into the lesser peritoneal cavity. If the tumor is in the right lobe, the enlargement of the liver may be upward or downward. The upper border of liver-dulness may begin two or three interspaces higher than normal posteriorly or in the axillary region. If the cysts are superficial, when palpated with the fingers of the left hand and percussed with the right, a vibration or trembling movement is felt, which may continue for a certain time. It is known as the *hydatid fremitus*. It is not always present. The enlargement is painless. Local sensations of weight and dragging may be complained of. If suppuration sets in, there may be a good deal of pain.

Diagnosis. The diagnosis is not difficult. The occurrence of irregular painless enlargement of the liver without general symptoms is significant. If fluctuation or the characteristic fremitus is detected, a more positive conclusion can be reached. When suppuration takes place, the symptoms are like those of abscess of the liver. Hydatid disease is to be distinguished from *syphilitic hepatitis*, in which the enlargement is hard and irregular, and does not fluctuate. Sometimes the symptoms resemble cancer; but the age of the patient, the presence of jaundice, and the extreme emaciation and cachexia indicate that affection rather than hydatid disease. *Enlargement of the gall-bladder* containing a mucoid fluid, in which fluctuation can be detected, may simulate hydatid disease. The enlargement, however, may be preceded by conditions causing obstruction of the cystic duct. The gall-bladder is movable. In some instances there may be resonance between it and the liver. It is usually of a pyriform or oblong shape. In *hydronephrosis* the symptoms of a localized cyst are present, which does not, however, move with respiration, as in hydatid disease. Exploratory puncture is sometimes necessary to establish a diagnosis. A hydatid cyst is frequently confounded with *pleural effusion* of the right side, for there may be all the physical signs of effusion at the right base. The distinction can be made by the char-

acter of the line of dulness. In hydatid cysts, as Frerichs points out, the line corresponds to a curve having its greatest height in the scapular region. It is not difficult usually to distinguish hydatid cyst from other

FIG. 380.



Human echinococci. (From FINLAYSON, after DAVAINE.)

A, a group of echinococci, still adhering to the germinal membrane by their pedicles. × 40.
B, an echinococcus with head invaginated in the body. × 107.
C, the same compressed, showing the suckers and hooks of the retracted head.
D, echinococcus with head protruded.
E, crown of hooks, showing the two circles. × 350.

forms of painless enlargement. In *fatty* and *amyloid* disease the enlargement is uniform. Both occur more commonly in individuals who have been in ill health, whereas hydatid disease occurs in healthy individuals.

FIG. 381.

Hooks from *Tænia echinococcus*. × 350. (CORTI.)

An absolute diagnosis of hydatid disease is based upon the results of *exploratory puncture*. When this is made over a tumor, or the centre of dulness, if it is due to hydatid disease, a clear, slightly opalescent fluid is withdrawn. The fluid has a specific gravity of 1005 to 1009, is of neutral reaction, and does not contain albumin but does contain chlorides and sometimes traces of sugar. Hooklets may be found in the clear fluid.

Displacements of the Liver.

The literature contains about 80 cases, in many of which disease of the gall-bladder or gall-ducts was simulated because of (1) attacks resembling gallstone colic caused by obstruction of the ducts, (2) obstructive jaundice from kinking of the bile-ducts, and (3) the presence of a tumor simulating an enlarged gall-bladder.

Diagnosis. The diagnosis is based on (1) the association with other displacements, and the patient's physique—long-chested subjects are predisposed to displacement of organs; (2) the clinical course; and (3) the physical signs.

Clinical Course. Most of the cases occur in women, and there is usually a history of trauma or abdominal disease. The patients usually suffer from neurasthenia and gastro-enteroptosis. The condition is one of long duration marked by recurrent attacks of pain, transient jaundice, bilious vomiting, and symptoms due to pressure upon other organs; fever is absent except in a few cases.

Physical Examination. Diastasis of the recti muscles is frequently present. On palpation a tumor of the size, shape, and consistence of the liver is discovered in the abdomen; it moves with respiration like the liver, and can be replaced by palpation, or returns to the normal position when the patient assumes the recumbent posture. The signs are readily brought out with Glénard's *procédé du pouce* (*q. v.*). On percussion, which must be performed in the erect as well as in the recumbent posture, a tympanitic note is obtained over the normal area of liver-dulness, except in anteversion when the convex surface is in contact with the diaphragm and anterior abdominal wall, and the note is therefore dull.

Deformities of the Liver.

Floating Lobes, Riedel's Lobe. The abnormally elongated lobe forms a tongue-like process which sometimes simulates tumor of the gall-bladder.

Clinical Course. The clinical course is uneventful, or there may be occasional attacks of pain and tenderness coincident with the presence of the tumor, which disappears at times. Women are chiefly affected, especially such as have been addicted to the practice of abdominal constriction. Attacks of gallstone colic and cholangitis may accompany, or in rare cases follow the condition.

Physical Examination. The tongue-like enlargement is smooth and solid to the touch, of variable size, and movable by palpation and with respiration; in rare instances it behaves like a floating kidney. The tumor disappears at times, and it may or may not be possible to demonstrate by percussion its connection with the liver. The percussion-note is dull when the tumor is thick; but it may be so thin that the resonance of the underlying hollow structures is not obscured.

Diagnosis. *Cholecystitis* may be simulated, as the tumor is at times the seat of pain and tenderness. *Cholelithiasis* is a frequent accompaniment, and the coincidence may give rise to confusion.

Corset Liver. This deformity may also simulate tumor of the gall-bladder.

Clinical Course. The clinical course resembles that of floating lobe and is marked by hepatic incidents. Corset liver may be associated with cholecystitis, cholangitis, cholelithiasis, gastro-enteroptosis, and carcinoma.

Physical Examination. The tumor is not unlike that caused by a floating lobe. When separated from the liver it is variable in size and shape, of the consistence of the liver, and movable to an extreme degree. The abdomen is tender and swollen. The tumor is always superficial, is never found in the loin, and is sometimes the seat of spontaneous pain.

DISEASES OF THE GALL-BLADDER AND GALL-DUCTS.

Inflammation of the Gall-ducts. Catarrhal Cholangitis.

Causes: secondary to gastroduodenal catarrh, to pressure, to local spreading infections. Other cases of catarrhal jaundice are found to be primarily infectious.

Symptoms. The symptoms and signs are well known. The age of the patient, the presence of a cause, and the clinical course make up the picture. When long continued, catarrhal inflammation may resemble obstruction due to other primary or secondary processes.

Diagnosis. The diagnosis in cases of so-called catarrhal jaundice continuing more than six weeks should be revised if the number of erythrocytes suddenly diminishes, the hæmogoblin falls, the spleen enlarges, and there is loss of weight. The true nature can be determined by the antecedents or by accompaniments, which by this time may be more prominent, as the enlarged glands of syphilis or tuberculosis. Organic diseases, cirrhosis, cancer of the liver, and infections, as Weil's disease, must be excluded. Many so-called catarrhs are the result of typhoid, pneumococcus, or other infections.

Suppurative Cholangitis.

The conditions for which this disease may be mistaken are pyelophlebitis, abscess and syphilis of the liver, rare cases of cancer, and Weil's disease.

The diagnosis is based on the:

1. **Clinical Course.** Gradual onset; infection of ducts or gall-bladder; presence of foreign bodies (gallstones); history of a previous general infection.

2. **Objective Diagnosis.** Icterus may or may not be present. The fever is characteristic (hectic type). The sepsis due to the jaundice gives rise to toxic symptoms.

3. **Physical Diagnosis.** The liver is moderately enlarged and tender. There is also a tender area in the region of the twelfth dorsal vertebra, 2 to 3 centimetres from the median line. (Boas.)

4. **Laboratory Diagnosis.** Leucocytosis is present. The serum reaction should be taken to determine the cause, and the blood examined to exclude malaria.

Differential Diagnosis. From *pyelophlebitis* the distinction is made by the absence of any cause for infection in the portal area; from *amœbic abscess* by the clinical antecedents, the physical signs, and the abscence

of leucocytosis; from *malaria* by an examination of the blood; and from *simple cholelithiasis* by the absence of leucocytosis. (See Infections and Ball-valve Calculus.)

Cholecystitis.

Acute phlegmonous inflammation of the gall-bladder is attended by localized pain and tenderness, by high temperature, extreme prostration, and the rapid development of the typhoid state. Peritonitis rapidly ensues. It can be distinguished with difficulty from other forms of acute inflammation in the same region, unless there is a previous history of gallstones and tumor of the gall-bladder was present before the attack developed.

Suppurative inflammation of the gall-bladder may occur from gallstones and in infectious diseases. The colon bacillus, the diplococcus of pneumonia, and particularly the typhoid bacillus, give rise to infectious inflammation of the gall-bladder. The enlargement takes place suddenly and may increase, the tumor becoming tender and painful on palpation, while rigidity of the right rectus is marked. The direction of growth is toward the umbilicus. The general symptoms are those of suppuration. Hectic fever or markedly remittent fever occurs, and, unless surgical relief is given, peritonitis ensues from infection or from rupture. This complication may be suspected from the occurrence of collapse and increase of the local symptoms.

Either of these forms of cholecystitis is attended by pain in the region of the gall-bladder or in the epigastrium, or even as low down as the appendicial region. The pain is severe and paroxysmal. The symptoms of bacterial infection, of which vomiting and fever are the most prominent, rapidly follow. The general symptoms simulate those of appendicitis, intestinal obstruction, and pancreatitis.

The diagnosis is based on the :

1. **Clinical Course.** Primary cholecystitis follows acute gastroduodenitis and general infections; the secondary variety follows infections induced by foreign bodies and obstruction. The disease may occur at any age and in either sex. The mode of life appears to have little to do with its causation. The onset is variable; it may be fulminating, severe, or mild and gradual.

2. **Subjective Diagnosis.** The symptoms are pain from spasm or tension, and tenderness; nausea and vomiting.

3. **Objective Diagnosis.** The fever may be mild or severe, remittent or intermittent; chills occur in the fulminating type of the disease. Jaundice is not present.

4. **Physical Diagnosis.** Tumor at the end of the ninth rib exhibiting the characteristics of a swollen gall-bladder and movable with respiration unless inhibited by pain. There is spasm of the rectus muscle.

5. **Laboratory Diagnosis.** Leucocytosis is present in all infectious cases except those due to typhoid fever, and in 50 per cent. of the latter. It is important to take the serum reaction to determine the nature of the infection.

Differential Diagnosis. Cholecystitis simulates hyperæmia and infections of the liver, subdiaphragmatic abscess, diaphragmatic pleurisy,

pneumonia, pancreatic affections, perforation of a gastric duodenal ulcer, obstruction, uræmia, and endocarditis.

Cholecystitis in Typhoid Fever. The local infection may develop in the course of the disease—during the third or fourth week; or in the period of convalescence. All grades are seen. It may or may not be accompanied by cholangitis. If it occurs in the course of the disease, its onset may be marked by an increase in the temperature, varying with the intensity of the local infection. In mild forms the fever may become a little higher than the continued range that had preceded. In severe forms it may become remittent or intermittent, and chills are not uncommon.

Nausea and vomiting are common in the beginning and may continue for several days. Pain in the region of the gall-bladder, and localized tenderness and rigidity of the right rectus muscle develop with the fever.

Fulminating cases may occur during the course of the disease, simulating perforation of the bowel.

The symptoms of onset are very characteristic. Pain is often so extreme as to simulate gallstones or lead to collapse. It is usually in the situation described, but may be referred to the epigastrium. The pain may seem to be general at first and even be marked at some distance from the gall-bladder, as in the right iliac fossa. Often the pain is diffuse during the first twenty-four hours and attended by general rigidity and distention. Frequently the local symptoms are overlooked, and I am convinced that many cases of so-called relapse in typhoid fever are due to mild cholecystitis. Leucocytosis is not necessarily present; the data concerning this point are quite insufficient. In one of my cases it was absent, although, following the law of leucocyte increase, such absence might have been due to the extreme infection.

Cholecystitis during convalescence from typhoid fever is not unusual. Contrary to Da Costa's experience, I have seen it quite as frequently at this period as at any other. Here, too, the cases may be mild, or fulminating with perforation. The onset in the post-febrile period takes place in the first or second week, when we are about to place the patient on solid food. In consequence, the change of diet is held responsible for the pain, tympany, and fever.

Confusion regarding secondary cholecystitis. Cholecystitis occurring in a subject who has gallstones or a displaced liver differs from primary cholecystitis in the local rather than in the general symptoms. The altered position of the gall-bladder changes the location of the pain and tumor. Thus, the tumor may be in the transverse umbilical, or in the median line. The tumor may be continuous with liver-dulness. An antecedent history of cholelithiasis aids in the diagnosis.

Differential Diagnosis. Cholecystitis can easily be recognized, the difficulty arising in distinguishing some forms of it from *appendicitis* being greatest, and yet these are cases of secondary cholecystitis, and therefore more obscure because due to displacement of the gall-bladder. Primary cholecystitis can be recognized by the clinical course, the physical signs, and the associate symptoms, together with the results of laboratory diagnosis.

In the distinction between the *catarrhal* and *suppurative* varieties we are deciding in part between mild and severe forms of infection. The degree of the infection is estimated by the severity of the local symptoms and the intensity of the toxæmia as indicated by the fever, the cardio-vascular and cerebral symptoms, and the leucocytosis. An intense inflammation may arise without pus formation.

Cholecystitis must be distinguished from :

1. *Congestion of the Liver.* In cases of acute infectious endocarditis with cardiac dilatation, the left lobe of the liver often enlarges and is the seat of pain and great tenderness. This is accompanied by jaundice. An enlarged gall-bladder may be simulated, and as the general symptoms of infection prevail, that organ may be considered the site of primary infection. It is not unusual to have endocarditis without physical signs in the heart. Again, in a case of purulent pericarditis with congestion of the liver, the distention of the liver capsule was extreme, pain was excessive, and the picture was not unlike that of suppuration of the biliary passages.

2. *Syphilis of the Liver.* The history may help, but is often not to be relied on. Antecedent and associate conditions aid us here. The fever is commonly intermittent in syphilis. The paroxysms occur at fixed periods in the day however. Leucocytosis is absent. Justin's sign is not satisfactory. A localized tumor with the characteristics of an enlarged gall-bladder is usually not present. It may, however, exist, although it is not so tender. A rapidly growing localized gumma attended by fever and sweats may simulate gall-bladder infection. It has been the writer's experience to find a lymphocytosis more common in syphilis than in other infections.

3. *Multiple Abscess of the Liver or Pyelophlebitis. Portal Pyæmia.* The differential diagnosis is sometimes difficult or impossible. The antecedent history of abdominal infection is necessary to establish the diagnosis. The liver is large in multiple abscess; in cholecystitis, the gall-bladder. Pain is not marked in multiple abscess. Ascites, enlarged spleen, hæmatemesis, and diarrhœa are more common in portal obstruction.

4. *Abscess of the liver* due to amœba is recognized by the history, by physical signs posteriorly rather than anteriorly, and often thoracic rather than abdominal, by the frequent absence of leucocytosis, the rarity of fever whose type is more or less slow, and by the presence of uniform enlargement or enlargement in one direction of the liver. The spleen is more likely to be enlarged in abscess than in non-typhoid cholecystitis. Abscess of the liver is neither so painful nor so acute an infection, and hence not so intense an infection as cholecystitis.

5. *Subdiaphragmatic Abscess.* The history, physical signs, and results of exploratory puncture are helpful in arriving at a diagnosis (see papers by Mason, Osler, and others). The condition manifests itself posteriorly, and the signs are abdominal as well as thoracic. The distinction from primary cholecystitis is not difficult.

6. *Diaphragmatic Pleurisy.* Diagnosis at times difficult. Exposure to cold is a feature in pleurisy.

7. *Pneumonia.* It is only necessary to call attention to the likelihood of confusing the infections in some cases. In pneumococcus infection with gastric and abdominal symptoms dominant, the pulse-respiration

ratio and the expiratory grunt may alone suggest the lesion, especially in children. When jaundice occurs early, the confusion may be increased.

8. *Pancreatic Diseases.* (See pages 1107–1113, Diseases of Pancreas.)

9. *Perforation of Gastric and Duodenal Ulcer.* The diagnosis is sometimes difficult, but the marked difference in the previous history is most helpful. Without such carefully worked out history the diagnosis may be impossible.

10. *Intestinal Obstruction.* This condition does not simulate hepatic and biliary disorders as frequently as it does pancreatic lesions.

11. *Uræmia.* Like hysteria, uræmia may simulate almost any other condition. In that form in which abdominal pain and vomiting are most prominent, the surgeon may easily be led astray, particularly as albumin and casts are almost always present in gall-bladder infections.

Gallstones.

Gallstones form in the biliary passages and may remain there without creating symptoms; or the efforts to pass them may cause attacks of pain called *hepatic* or *biliary colic*, after which the stone may pass into the intestinal tract without causing further hepatic symptoms. It may become

FIG. 382.

Gallstones.

impacted in the biliary canal and set up catarrhal or suppurative inflammation, which in turn may be followed by stricture. Gallstones usually form, or at least show signs of their presence after the age of forty years, most frequently in women and in people who have led a sedentary life and partaken of rich and indigestible food. Individuals in different generations of the same family may be predisposed.

It is becoming increasingly well evidenced that gallstones are often the result of infection. This is particularly true of *typhoidal infection*. A history of typhoid fever preceding the development of gallstones is not uncommon, and typhoid bacilli have been known to furnish the nucleus about which they have developed. Typhoidal infection of the bile-passages acts by exciting catarrh of the mucous membrane; and in addition it is probable that the bacilli are agglutinated by the bile and thus afford a nucleus for the formation of calculi.

Hepatic Colic. The passage of gallstones may be attended by a slight amount of pain only, which, but for its location in the right upper quadrant, would pass for an attack of simple indigestion. In the large majority of cases the pain is severe. The attack may be preceded by biliousness or indigestion lasting twenty-four hours, and moderate pain or a sense of weight and fulness in the liver. It frequently follows the taking of food. Ringing in the ears, disturbance of vision, or undue flushings are said to precede it in some instances.

The attacks may be sudden. The patient is seized with *pain* along the margin of the ribs on the right side, or there may be pain above the ribs, over the liver, and in the right shoulder at the same time. From the hepatic region it extends to the median line. Very frequently the pain begins and continues in the epigastrium, or it may be most pronounced in this locality from the first. The pain is intense and paroxysmal. The patient is doubled up in agony. It causes more or less *collapse*. The pulse increases. *Vomiting* usually occurs at the same time, first of the contents of the stomach, and then of a yellowish, bile-stained fluid. The vomiting may be extreme so that the patient is tormented by the pain, the retching, and vomiting. The attack sometimes subsides as suddenly as it occurred, or wears off gradually. In very severe cases it is followed by symptoms of shock. The bowels are not disturbed during the attack. The urine may be suppressed; it is usually high-colored, and after the attack may contain bile.

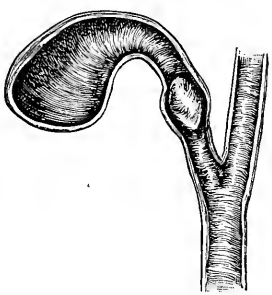
At the time of the attack there is considerable tenderness below the xiphoid cartilage and in the hepatic region. The *tenderness* is more marked on deep pressure in the gall-bladder region and to the right of the midclavicular line, at the margin of the ribs. The epigastrium may be slightly swollen. The tenderness persists after the attack, and the stomach may be weak or irritable for some time; pain, however, usually disappears at once. The attack may recur frequently until the stone has been passed, so that in twenty-four hours the patient may have a dozen or more paroxysms. After the attacks have subsided, light *jaundice* may supervene, but usually does not continue more than a week at most, during which period there are also symptoms of mild gastritis. (See Intestinal Colic.)

In some instances a *chill* precedes or immediately follows the pain, after which the temperature rises. After the paroxysm subsides, the fever disappears rapidly and is followed by profuse perspiration. If the gallstones have set up catarrhal inflammation, moderate fever may continue for a few days. (See Fever in Obstruction.)

During any paroxysm of hepatic colic it is desirable to determine

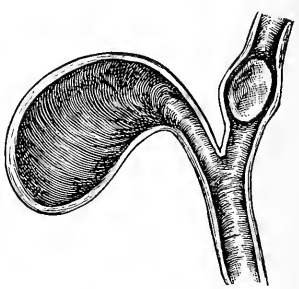
whether or not a gallstone has been passed. This can only be done by placing the feces in a sieve and pouring water upon them until they dissolve. Instead of gallstones, dark-colored granular bile, which has become inspissated, is sometimes seen in the stools. Bile in this form, according to Harley, gives rise to as much pain as true biliary concretions. If the stone is not passed, it may fall back into the gall-bladder and cause no further symptoms for a time, or become impacted in the ducts. The impaction may be such that no obstruction is caused by its position, the bile being forced through or around it; or complete obstruction may take place. (See Jaundice.)

FIG. 383.



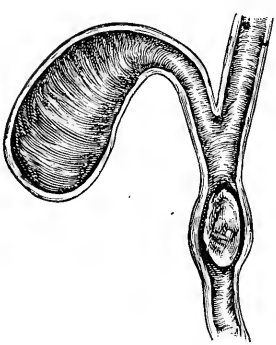
Gallstone in cystic duct.

FIG. 384.



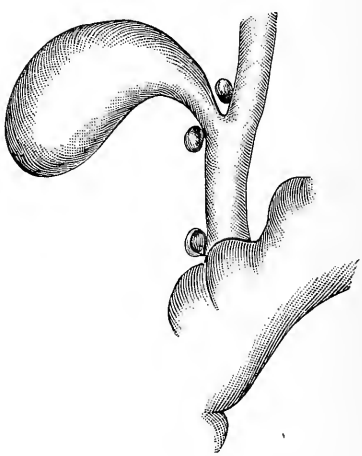
Gallstone in hepatic duct.

FIG. 385.



Gallstones in common duct.

FIG. 386.



Enlarged glands which cause jaundice.

Obstruction of the Common Duct by Gallstones. (*a*) In addition to jaundice paroxysms of chill, fever, and sweat occur, with catarrhal inflammation of the biliary passages. (1) The paroxysms resemble intermittent fever; (2) the jaundice may continue for years and deepen after each paroxysm; (3) hepatic colic may occur with the paroxysm; (4) the health fails but slightly. The paroxysms may occur daily or only once a

week, or they may be tertian and quartan in type. The pain is referred to other situations than the gall-bladder or the epigastrium. It is often relieved by vomiting or by certain positions of the body. The jaundice may be intermittent or remittent. On account of the obstruction in this situation the liver becomes enlarged. It is firm and smooth on palpation. The enlargement, as determined by percussion, is uniform. The gall-bladder is not enlarged. Fenger's thorough studies show that the *intermittent phenomena* are due to ball-valve action of a single stone. He also points out that emaciation is of common occurrence. (*b*) Gallstones may cause *suppurative inflammation of the ducts* or *of the gall-bladder*. The symptoms, both general and local, are severe. The fever may be intermittent, but is more likely to be remittent; jaundice is present, and is constant in degree. The local signs of enlargement and tenderness are made out. The patient dies of exhaustion or septicæmia. Sometimes the gall-bladder ruptures into the stomach or colon, and temporary abeyance of the symptoms results.

The Accidents of Gallstones. While these effects of the presence of stones in the biliary passages may rightly be considered as accidents, nevertheless their occurrence is so common as to be part and parcel of the history of gallstones. As accidents, we have most commonly the occurrence of localized peritonitis, which leads to dislocation of the gall-bladder, with constriction of the duodenum and secondary dilatation of the stomach; we also have the formation of biliary fistula, with passage of the gallstone into the contiguous organs or channels. The stone may ulcerate into the gall-bladder from one of the ducts, into the portal vein, or into the abdominal cavity—the most frequent accident. Perforation also takes place into the duodenum, into the colon, and, rarely, into the stomach. Such perforation can only be inferred from its secondary effects: (1) an attack of hepatic colic; (2) local inflammation with fever; (3) the occurrence of peritonitis, or the discharge of pus by the bowels or by vomiting. That it is due to gallstones is proved in those rare instances in which the stone is passed per rectum. Often the stone may be impacted in the intestinal canal, causing symptoms of acute obstruction; or in the rectum, causing local tormina and tenesmus. The perforation, however, occurs also in other directions. Sometimes a fistulous connection is formed between the gall-bladder and the urinary passages, calculi and pus being discharged in the urine. In other instances fistulæ between the bile-passages and the lungs are formed. The bile is coughed up and expectorated, sometimes with small calculi. In the most common form ulceration proceeds toward the surface, with formation of a cutaneous fistula. After the fistula has opened externally, gallstones in large numbers may be passed. If not, the cause of the fistula must be determined by the history and the results of probing, due attention being given to the condition of other organs.

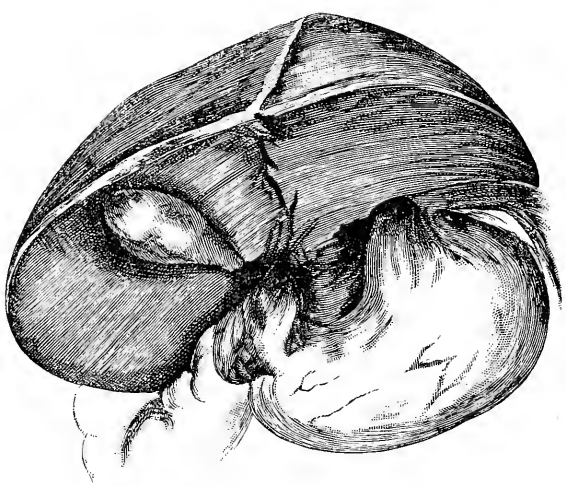
Enlargement of the Gall-bladder.

Enlargement of the gall-bladder may be due to obstruction in the *cystic duct*. (Plate XLIV., Fig. 2.) The liver is not secondarily affected.

The enlargement is noted at the edge of the liver in the usual situation, and may gradually increase to an enormous extent, so that it has been mistaken for an ovarian cyst. The gall-bladder is often quite movable, and on account of its location and movability, as well as its elongated shape, has been mistaken for a floating or movable kidney. If the gall-bladder is not too large, it can be felt as a rounded or pyriform mass when the hand is placed along the margin of the liver, becoming more marked when the patient takes a full breath. The enlargement is not attended by any other symptoms except mechanical ones, unless the contents of the gall-bladder are purulent. In obstruction with simple enlargement the fluid obtained by aspiration is thin, of a mucoid nature, and alkaline in reaction. It may contain cholesterin-plates, and sometimes blood. It must be distinguished from the fluid of a hydatid cyst.

Diagnosis. *Simple* enlargement of the gall-bladder must be distinguished from enlargements due to inflammation.

FIG. 387.



Cancer of pylorus pressing on the cystic duct.

Enlargement of the gall-bladder, usually due to cystic obstruction as previously mentioned, may be mistaken for *floating kidney*, for *tumor of pylorus*, and for *ovarian cyst*. Tumors of the gall-bladder from any of these causes are recognized by their position and shape, and by the character of the tumor. The *position* varies. The tumor is usually situated in the gall-bladder region, but may extend as low as the groin, or may be so large as to distend the ribs and fill almost the entire abdominal cavity. If, however, the case has been under observation from the beginning, the tumor must have been found originally in the gall-bladder region. This region corresponds to the point where a line drawn from the acromion process of the right shoulder to the umbilicus intersects the costal margin. The tumor grows from this point toward the umbilicus in nearly all the cases. It can be recognized by its *shape*, which is pyri-

form, globular, or conical. The *character* of the tumor varies. It is usually tender and firm, but elastic on pressure, and movable. Fluctuation may often be detected. The septic gall-bladder is symmetrical and resistant to the touch. If the enlarged gall-bladder contains calculi, they may be felt as small hard masses, which transmit a grating sensation to the finger. On aspiration, if the cystic duct is obstructed, the mucoid fluid previously mentioned, or pus, is withdrawn. If the common duct is obstructed, bile will pass through the trocar.

The enlargement must be distinguished from *tumors of the liver, stomach, duodenum, pancreas,* or *lymphatic glands.* Tumors of the liver are usually due to *carcinoma.* They are multiple, associated with enlargement of the liver, with jaundice, ascites, enlargement of the spleen, and emaciation. Tumors of the *stomach, duodenum,* and *pancreas* are in a different position, and are attended by functional disturbance of the respective organs from which they spring. An *abscess of the liver,* if purulent, may simulate enlargement of the gall-bladder. If the abscess can be palpated, an area of induration is first felt, followed later by softening and fluctuation of the swelling. In judging of the nature of the tumor we must bear in mind the causes of abscess. In *hydatid disease* the tumor develops slowly; it is painless; it may yield fremitus, and, if movable, the course is slow and not attended by general symptoms. *Multilocular hydatid disease* can rarely be distinguished save by the difference in position of the tumor, which is nodulated, hard, and tender, and associated with jaundice, ascites, œdema of the legs, enlarged spleen, and great emaciation and prostration, with rapid decline. A *gumma* in the liver may occupy the region of the gall-bladder. It can usually be made out as continuous with the liver structure. It is tender and painful, but irregular; other signs of syphilis, or a history of the infection and of symptoms of a primary and secondary period will aid in the distinction of the disease.

Floating Kidney. The gall-bladder is larger and fixed at one end, whereas the entire kidney is movable. The gall-bladder may fluctuate, and there are symptoms of hepatic disease. On the other hand, the well-known symptoms of floating kidney, the shape of the tumor, the sensation of nausea induced by palpation, and association with gastroptosis and enteroptosis, point to the renal origin of the mass. *Tumors of the kidney,* such as sarcoma, hydronephrosis, and pyonephrosis, must be distinguished. 1. There may be changes in the urine. 2. In renal tumors the intestine is in front of some portion of them, or a zone of resonance is found between the liver-dulness and the tumor. 3. Renal tumors are fixed. They may, as in hydronephrosis, come and go, preceded by attacks of renal colic and attended by anuria. From *ovarian* or *uterine tumors* the diagnosis must be made by examination of the genital organs, although with the former there is often difficulty.

Enlargement on account of calculous obstruction must be distinguished from enlargement due to *cancer of the gall-bladder.* This is often difficult and can not be done without having the patient under observation for a long period of time. Cancer of the gall-bladder is usually *primary.* It may begin in the gall-ducts. In the larger number of cases it occurs in patients who have had gallstones. It is found

most frequently in females, and after the fiftieth year. Tight-lacing or pressure around the abdomen may predispose to it. The *symptoms* are pain, jaundice, emaciation, cachexia, and the presence of a tumor. The pain is localized and lancinating in character. Jaundice occurs in 70 per cent. of the cases and gradually increases in intensity. The tumor is situated in the gall-bladder region, to the right of the umbilicus. It is hard or firm, painful, and the seat of tenderness. The tumor is fixed. Sometimes the disease is found in the cystic duct, in which case the gall-bladder is enlarged. As history of gallstones is frequently obtained in both instances, it is impossible to distinguish the two forms of obstruction, save that in carcinoma the emaciation and cachexia may point to the true nature of the case. In tumor of the gall-bladder due to cancer the secondary effects on the liver are usually more marked than in tumor from other causes. The liver enlarges and its surface becomes irregular or nodular.

Diseases of the Portal Vein.

Disease of the portal vein, or occlusion of its branches in the liver, obstructs the flow of blood. The diseases of the portal vein are thrombosis, and adhesive and suppurative inflammation. Obstruction of the terminal venous radicles in the liver occurs in cirrhosis.

Thrombosis of the portal vein may attend cirrhosis of the liver, or occur secondarily to pressure upon the vein by a tumor. Disease of the pancreas was the cause of the pressure in a patient under my observation. As the result of thrombosis *adhesive inflammation* of the vein takes place, with or without the establishment of a collateral circulation to replace its function. The symptoms of disease of the trunk of the portal vein are the same as those of obstruction of the terminal branches, and are known as the symptoms of portal congestion. In one respect only do they differ. While we may have *ascites* in both; in thrombosis of the portal vein it occurs suddenly and the fluid rapidly accumulates again after tapping.

Suppurative inflammation of the portal vein is attended by symptoms resembling pyæmia, and is also called *portal pyæmia.* The inflammation is secondary, and depends upon inflammation somewhere in the portal area. It may follow appendicitis, infectious inflammation of the hemorrhoidal veins, or of the veins anywhere in the gastro-intestinal tract. Pus is carried into the liver by the portal current, and multiple hepatic abscesses are formed. Three pathological affections are therefore seen: (1) suppuration in the portal area; (2) inflammation of the portal vein; and (3) multiple abscesses of the liver (for the symptoms of which see Abscess).

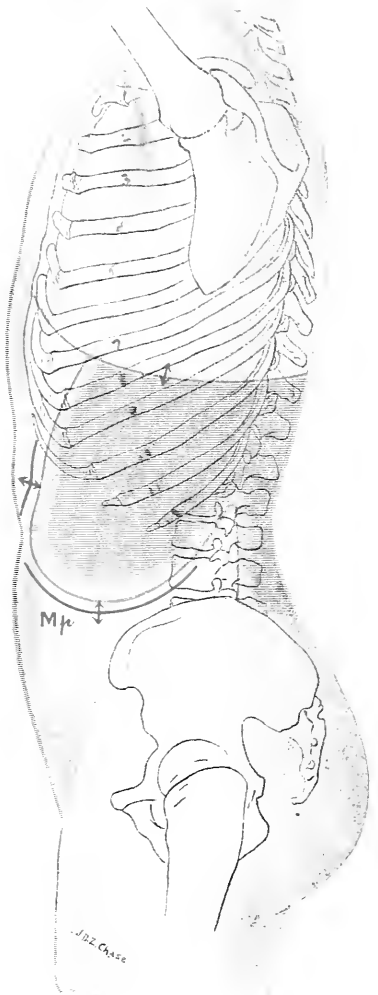
THE SPLEEN.

Enlargement of the Spleen.

Acute Enlargement. Enlargement of the spleen may be acute or chronic. Acute enlargement occurs in certain infectious diseases, particularly typhoid fever, typhus, smallpox, relapsing fever, scarlet fever, diphtheria, epidemic cerebrospinal meningitis, the malarial fevers and

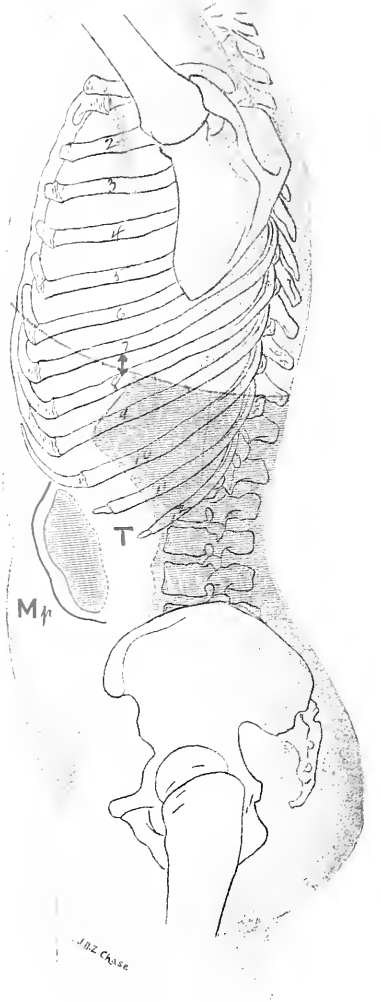
PLATE XLV.

FIG. 1.



Enlargement of the Spleen.

FIG. 2.



Tumor of the Left Kidney.

meningitis, and in diseases with blood-poisoning, as septicæmia, puerperal fever, and erysipelas.

A rare cause of enlargement is *acute splenitis*. Generally, it is the result of emboli lodged in the spleen and starting from an endocarditis. The area of splenic dulness extends rapidly, and there are local pain and tenderness on pressure, increased by coughing and deep inspiration; other symptoms are fever, nausea and vomiting, and occasionally delirium. If, as frequently happens in splenitis, emboli lodge in the kidneys also, the urine will be albuminous and bloody. If suppuration ensues, the fever becomes hectic, and the spleen continues to increase in size. Splenic abscess may, however, remain latent until rupture occurs.

Chronic Enlargement. Chronic enlargement of the spleen occurs as hypertrophy and as the result of amyloid disease, leukæmia and pseudo-leukæmia, chronic malarial poisoning (ague-cake), syphilis, hydatid tumor, and cancer. Enlargement is greatest in leukæmia, pseudoleukæmia, and chronic malaria. The spleen in well-marked cases of these affections may reach to the umbilicus and even beyond, filling up the hypogastrium and extending to the right iliac region; it may measure 13 or 14 inches in length and half as much in width, and be proportionately increased in thickness.

Primary splenic enlargement may occur without local or general symptoms; or anæmia, profuse hemorrhages, and brown pigmentation of the skin may be present at the same time; the hemorrhages are usually limited to the gastro-intestinal tract, and the anæmia is of a chlorotic type, with no change in the leucocytes. Splenic enlargement may also be associated with *cirrhosis of the liver* and *jaundice*, with gastro-intestinal hemorrhages and ascites. The affection is commonly known as *Banti's disease*. The blood-changes resemble those found in progressive pernicious anæmia, but differ in that nucleated red cells are absent or present only in small numbers, and the hæmoglobin shows relatively greater reduction. It may be confounded with chronic inflammation of the peritoneum, giving rise to ascites and associated with mediastinal pericarditis.

Diagnosis of Splenic Enlargement. (Plate XLV., Figs. 1 and 2.) Enlargement of the spleen can be distinguished from enlargement of the left *kidney* by the greater movability of the spleen. 1. The spleen does not extend so far back toward the spine as the kidney, so that the fingers can be thrust behind its posterior border, and if the other hand grasps the anterior edge, the organ can be moved backward and forward. Splenic dulness extends to the ninth rib or higher, while kidney-dulness has no thoracic area, but extends backward to the spine. 2. Again, the spleen is more movable with respiration than the kidney. 3. When the patient is in the knee-chest position, the spleen falls further toward the median line than the kidney. 4. An enlarged kidney has the colon in front of it, and hence its dulness is obscured by the tympany of the bowel. 5. The *shape* of an enlarged kidney is more globular than that of the spleen. The anterior surface of the latter is smooth and rounded, but at its junction with the flat posterior surface there is a sharp edge. 6. *Pain* in renal disease often shoots down the ureters and

into the testicles; in diseases of the spleen the pain is generally localized to the splenic region, and may shoot into the left shoulder. 7. An examination of the urine will often make clear that the disease is renal, or a negative result will point to the splenic origin of the tumor.

Splenic enlargement is sometimes difficult to distinguish from *enlargement of the liver*, particularly of the left lobe. Careful palpation reveals the edge of the spleen, which descends further than the liver in full inspiration. After the anterior edge has been found, pressure with the other hand posteriorly will bring the spleen forward, which would not occur if the suspected enlargement were the left lobe of the liver.

Regarding the diagnosis of *splenic hypertrophy* (ague-cake) in *chronic malarial affections*, Osler says: "The history of malarial cachexia, the absence of lymphatic enlargement, and the blood-condition will usually be sufficient for the purpose of a diagnosis. Great increase in the white blood-corpuscles is not often seen in the chronic splenic tumor of malaria; indeed, they may be much diminished in number. Toward the end in very chronic cases the clinical picture may be very similar; the large abdomen, possibly ascites, dropsy of the feet, and irregular fever may resemble closely splenic leukæmia, and the absence of an increase in the colorless corpuscles may be the only marked difference."

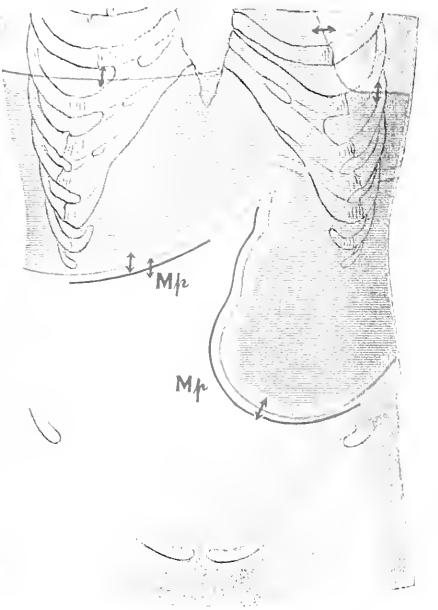
Splenic Leukæmia. The diagnosis of splenic leukæmia (Plate XLVI., Fig. 1) rests principally upon the blood-condition, particularly upon the existence of a marked increase of white blood-cells, a large proportion of which are myelocytes. Red cells are decreased, and altered forms are present. In addition to characteristic blood-changes there is a great disposition to hemorrhages; dropsies and priapism are common; and, in later stages, fever, diarrhœa, great weakness, and grave complications such as pneumonia. Hemorrhage in splenic leukæmia occurs from the nose, bowel, stomach, gums, or kidney; it may also be subcutaneous, intermuscular, cerebral, or retinal.

Amyloid Spleen. Amyloid disease with enlargement of the organ, occurs in conditions of prolonged suppuration, especially when the bones are involved, and in chronic phthisis and syphilis. The spleen is enlarged, hard, and painless. The enlargement is rarely sufficiently great to produce distress on that account, and it is so commonly associated with a similar condition of the liver and kidneys, if not of other organs, that any constitutional symptoms produced by the spleen are apt to be masked by those produced by other organs.

Hydatid Disease. Hydatid tumor of the spleen rarely causes symptoms except when it becomes very large; then it may give rise to discomfort and a dragging pain in the left hypochondrium. But hydatid tumors of the spleen are only exceptionally very large; when large enough to admit of palpation, and when the tumor is situated anteriorly or projects from the lower border or from beneath the organ, the detection of fluctuation, the withdrawal of the characteristic fluid by aspiration, and possibly the presence of hydatid fremitus, will establish the diagnosis, when taken in connection with the gradual development of the tumor and exposure to possible infection. In the absence of physical signs of a cyst, the nature of the tumor can only be suspected from the habits of

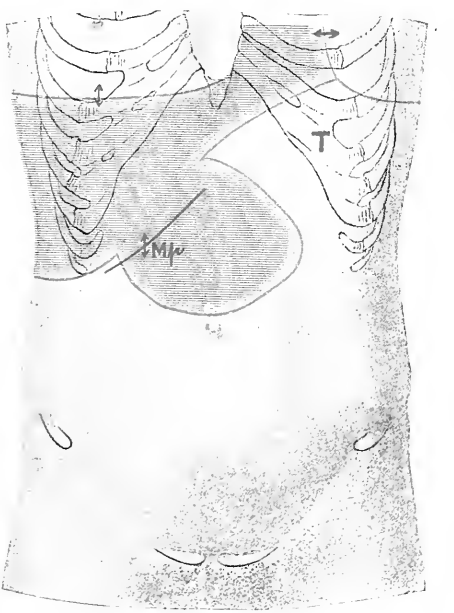
PLATE XLVI.

FIG. 1.



Leukæmia—Enlarged Liver and Spleen.

FIG. 2.



Cyst of the Pancreas.

the patient or his place of residence. Suppuration of the sac may be brought about by injury or rupture into the adjacent cavities, with grave if not fatal results.

Syphilis. *Inherited syphilis* and *chronic syphilis* are accompanied by enlargement of the spleen. They cause a chronic interstitial inflammation. The enlargement is usually not very great, and does not present characteristic features.

Malignant tumors of the spleen are rarely primary. The diagnosis must be made by noting malignant disease elsewhere, the very rapid enlargement of the spleen, with possibly nodules scattered over its surface, and the presence of cachexia and the usual constitutional signs of a malignant disease.

In *young children* enlargement of the spleen, with decided anæmia, is not uncommon. It is found associated most frequently with gastro-intestinal disease, rickets, syphilis, and malarial poisoning, and has been attributed to each of these diseases. In some cases none of these causes can be found, and the origin remains obscure. There is every reason for believing, however, that in most, if not all, instances the splenic enlargement is secondary to some other disease, usually an infection. Severe anæmia with splenic enlargement is uncommon in children over three years of age.

THE PANCREAS.

Just as the functional activity of the pancreas is separated with difficulty from that of other functionally related organs, so the aberration of such activity is discerned with the greatest difficulty. As the physiology and pathology are blended, so the symptoms are intermingled.

The pancreatic secretion aids in intestinal digestion, particularly in emulsifying fats; hence symptoms due to disturbance of this function are looked for, and it is, in a measure, true of all cases of pancreatic disease that there is some *intestinal indigestion.* For the purpose of determining whether the function of digestion of fats has been modified, the patient with suspected pancreatic disease is given fats in definite quantity, and the amount of fatty acids, soaps, and neutral fats in the stools is determined—a somewhat elaborate chemical procedure. If there is but little evidence of digestion of the fat taken—*i. e.*, if more than 75 per cent. of the fat in the stools is neutral fat—this is strong evidence of pancreatic disease, though the same conditions have in rare instances been seen in icterus without involvement of the pancreas or its duct. The mere observation of excess of fat in the stools, while it may arouse a suspicion of pancreatic disease, is of no real diagnostic value, as deficient fat absorption is most commonly due to absence of bile. The demonstration of good fat digestion, too, does not always show that the pancreas is normal; for it may be severely diseased without notable disturbance of fat-splitting ferment action. Sugar has been observed in the urine in many cases in which the pancreas was the principal seat of disease, and conversely, in many cases of diabetes morbid changes have been found in the pancreas. Both experiments upon animals and observation of

human beings show that the pancreas is closely connected with the origin of diabetes in some cases. Glycosuria, however, is not constant in pancreatic lesions. The three symptoms—*intestinal indigestion*, *fatty stools*, and *glycosuria*—are therefore not diagnostic of pancreatic disease, but only afford presumptive evidence of its presence.

Tumor of the Pancreas.

The most striking symptoms of disease of the pancreas, apart from those due to the morbid process, suppuration, or cancer, are those due to the pressure of the *tumor* upon surrounding structures. It may press upon the gall-duct and cause jaundice. From its situation in the epigastric region it may resemble an aneurism, or a tumor of the pylorus or of the transverse colon. Tumors of the pancreas are usually due to *cancer*. This is usually of the scirrhous variety, and generally primary. The enlargement cannot be distinctly made out unless the patient is very much emaciated. When it has advanced considerably, it may simulate aneurism, but is distinguished by the difference in the character of the pulsation. In an aneurism the pulsation is distensile; in disease of the pancreas it is an up-and-down movement—the hand is lifted with each pulsation of the aorta. Tumor of the pylorus is excluded largely by the more superficial position of the mass, its association with pyloric obstruction, and the absence of jaundice, which occurs less frequently than in disease of the pancreas. A pyloric tumor is more movable and may change its position after the stomach has been inflated with gas or distended with fluid. Examination with the patient on the hands and knees may aid in the distinction between the two. A tumor of the transverse colon is recognized by its nearness to the surface, its movability, its association with more or less constipation, and the occurrence of intestinal hemorrhage.

The general symptoms of the cancerous cachexia; the occurrence of intestinal indigestion, or loss of fat digestion; the gradual onset of jaundice; the occurrence of deep-seated epigastric pain; an immovable tumor, with glycosuria, make a symptom-group very characteristic of cancer of the pancreas. When the patient is on a milk diet, an examination of the feces will show that there is deficient pancreatic digestion with loss or reduction of the fat-splitting action.

Hemorrhage into the Pancreas.

We owe to F. W. Draper and Prince the greater part of our knowledge of hemorrhage into the pancreas. Since they have published the results of their labors the affection has been frequently recognized. The attack comes on suddenly in perfect health, and usually terminates life in a short period. It is associated with arteriosclerosis, particularly of the splenic artery and its branches, and the form associated with inflammation is probably frequently an infection. Nothing in the occupation or conduct of the patient at the time appears to favor the development of hemorrhage, except the occurrence of trauma. He is seized with *severe pain*, localized in the upper part of the abdomen. It increases in severity, and

may be intermittent like colic. *Nausea* and *vomiting* take place almost at the same time. The vomiting becomes obstinate. Extreme depression rapidly sets in, and the patient becomes anxious and restless. Collapse ensues in a short time. The extremities become cold and the forehead is covered with sweat. The pulse increases in frequency, and rapidly diminishes in strength. It soon becomes imperceptible. The pain and vomiting call attention to the upper abdomen. It is tender on pressure; the tenderness may extend throughout the entire upper half of the abdomen. Tympanites may develop. There is constipation in many cases. The temperature remains *normal*, or becomes *subnormal*. The pain, the vomiting, the anxious and restless state continue without relief.

From the above group of symptoms it can readily be seen that the *diagnosis* is obscure. The disease may be mistaken for perforation of the stomach by ulcer, although the vomiting is not, as a rule, so persistent and frequent. Intestinal obstruction in the upper portion of the tract presents allied symptoms. The hemorrhage symptoms, however, are more pronounced in pancreatic hemorrhage. Pallor of the face is sure to ensue. The vomiting is not fecal in character. Constipation can be relieved. It is, however, difficult and in many cases impossible to establish a diagnosis of hemorrhage into the pancreas. The rapidity of development of the symptoms is of importance. The pain and collapse of the rupture of an aneurism of the aorta simulate *pancreatic hemorrhage*.

Acute Hemorrhagic Pancreatitis.

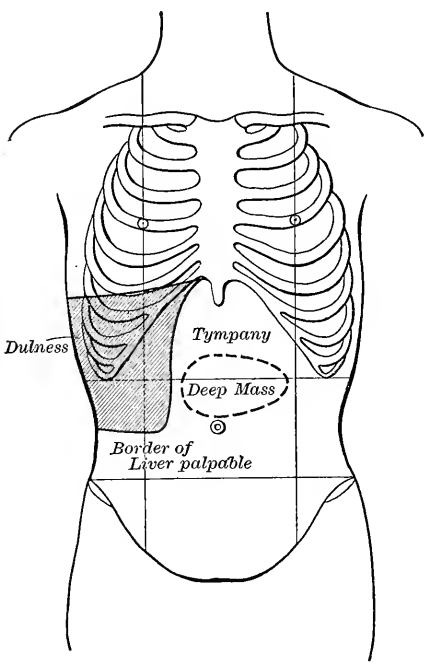
For our knowledge of this disease we are largely indebted to Fitz. He collated the facts from the literature, and, adding the results of his own valuable observations, has made the symptomatology sufficiently clear to allow of the occasional recognition of the affection during life. It usually occurs after the middle period of life, although it may occur in early childhood, the youngest patient known to the writer being eight months of age. It is more common in males, in those addicted to alcohol, and in fat subjects. The patient has often been the subject of attacks of indigestion, or of epigastric pain, or biliary colic. Indeed, cholelithiasis and infectious cholecystitis are well-known causes. It followed typhoid cholecystitis in one of my cases, injury in another, and the cause was unknown in another. A blow on the abdomen or injury in the lumbar region appears to have been the exciting cause in a number of cases.

The *attack* develops suddenly, not unlike pancreatic hemorrhage—indeed, the two are very closely related, the inflammation being often the result of hemorrhage into the organ. There is *violent pain*, which is at first complained of in the upper abdomen, although it is sometimes general. *Nausea* and *vomiting* are present in all the cases, *constipation* in most of them. The abdomen is frequently the seat of tympanitic distention. In many instances an obscure *tumor* can be made out in the lower epigastric region. *Collapse-symptoms* supervene; although *fever* may occur, the temperature rising to 102° F. The cases terminate by the fourth day, sometimes even earlier. The pain and collapse are probably due to pressure of the effused blood upon the cœliac plexus; while

the fever and the appearances of intoxication are probably the result of infection. Violent delirium resembling acute mania and not unlike that seen in atropine-poisoning, occurs in some instances. Symptoms of localized peritonitis arise, and if the patient lives, the tumor increases to a considerable size.

Diagnosis. The symptoms resemble those of acute *intestinal obstruction*, *intoxication with irritant poison*, or *perforation* of the gastro-intestinal or biliary tract, or an attack of hepatic colic. In several instances laparotomy has been performed for the relief of supposed obstruction. The intense pain in the epigastrium, with violent vomiting and distention of the upper abdomen, without a possible cause for obstruction, are favorable to acute pancreatitis. The difficulty of diagnosis, however, is so great that resort

FIG. 388.



Tumor of the pancreas.

to laparotomy is justifiable in order to determine the exact nature of the condition. In a most interesting case reported by W. S. Thayer, the diagnosis of acute pancreatitis (confirmed by laparotomy) was based upon the history of previous attacks of pancreatic pain, with fever, vomiting, and collapse, occurring in an adult who was over-fat and an alcoholic; the exclusion of disease in other organs and the absence of a history of gall-stones, gastric ulcer, or abscess from other causes; the occurrence of pain; the presence of a deep-seated tumor with indistinct signs of fluctuation, which was not movable with respiration, and the dulness of which was

not continuous with, or of the same character as that of adjacent solid organs. Epigastric tympany was also a point in favor of pancreatic disease. The accompanying figure indicates the site of the tumor in Thayer's case.

Suppurative Pancreatitis.

Suppurative pancreatitis occurs in adults under forty years, more frequently in males. Symptoms continue during several weeks, and many persist for a year. *Pain in the epigastrium* is complained of, associated with *irregular vomiting*, the latter persisting in spite of care as to feeding. *Fever* is irregular in type, and exhaustion ensues. Jaundice, fatty diarrhœa, and glycosuria have been observed in some cases. In a case under my observation obstruction of the portal vein took place and was followed by ascites, which was large and recurred rapidly after tapping. In this patient pain and gastric disturbance were absent. There was no fever. Emaciation, constipation, and a tumor above the umbilicus were present; the emaciation was extreme. The tumor was ill-defined, painless, apparently superficial. Many other symptoms of pancreatic disease pointed out by Roberts were present, such as apathy and despondency and bronzing of the face. The man was forty-two years old and addicted to the use of alcohol. He was thought to have cirrhosis of the liver. As happened in this case, the pus may accumulate in the duodenojejunal fossa and fill up the cavity of the lesser peritoneum, with more pronounced symptoms of tumor than occur in similar fluid accumulations in the above-mentioned cavity.

Gangrenous Pancreatitis.

This may follow later upon hemorrhage into the pancreas. The symptoms are extremely obscure. *Collapse* may occur, following *pain*, which is of longer duration than in the acute form, or *vomiting*, which is not so persistent. A patient of mine, upward of sixty years old, suffering from dyspepsia, vomited blood in the course of an illness which was characterized by loss of flesh and weakness. The anæmia became very profound after the gastric hemorrhage, and exhaustion was extreme. There was no marked tumor, but only resistance in the region below the xiphoid. There were dulness and tubular breathing at the base of the left lung. Fever was absent. Death ensued from exhaustion. A small flat carcinoma was found at the pyloric end of the stomach, but there was no perforation. Gangrenous pancreatitis, with signs of an ante-mortem hemorrhage, were found. The accumulation had taken place behind the stomach and colon, but in front of the kidney; its outer wall was bounded by the spleen. It was limited above by the diaphragm. Pleuritis and small pulmonary abscesses at the base of the left lung were found.

In some instances the pancreas has sloughed into the bowel; and in two such cases recovery took place after its discharge from the rectum.

Chronic pancreatitis is not recognized during life, although its possible

presence must be considered in all cases of diabetes, and in jaundice not otherwise explained.

Cyst of the Pancreas.

The cysts are not really true cysts, but accumulations of pancreatic fluid in the lesser peritoneal cavity. (Plate XLVI., Fig. 2.)

Causes. A pancreatic cyst may result from obstruction of the pancreatic duct with impacted biliary calculi; the obstruction is sometimes at the orifice. In some instances there is a history of a previous attack of so-called biliary colic or colicky pains in the upper abdomen, with vomiting but without jaundice—a condition characteristic of the presence of calculi in the pancreatic ducts. The disease occurs in adults, and often follows a trauma, a blow in the epigastrium being a frequent exciting cause.

Symptoms. The symptoms are those of a tumor occupying the upper abdomen in the median line or a little to the left, in the left upper quadrant. There is a sense of weight and fulness in the epigastrium. The tumor may be sufficiently large to fill the entire abdominal cavity and simulate ovarian tumor. As a rule, the cyst *grows slowly*, but in some cases the growth is quite rapid. *Fatty diarrhœa* is not present. In contrast to cancer, *pain is absent.* In this, as in other diseases of the pancreas, *diabetes* may be present. Senn has called attention to the peculiar complexion of patients suffering from pancreatic cyst; he describes it as of an unhealthy yellow, dirty, or earthy hue. The writer also considers that the history of the case, the location of the tumor and its relation to other organs are factors to be reckoned with in the diagnosis of pancreatic cyst.

Physical Signs. The objective signs are those of a *tumor* to the left of the median line, encroaching upon the left lobe of the liver above and extending almost to the transverse umbilical line. The cyst may *move with respiration.* At first it is always found in the region occupied by the pancreas, its exact position depending somewhat upon the portion of the pancreas from which it originated; it may be below the right lobe of the liver, below the xiphoid, or in the *left upper quadrant;* in the great majority of cases it occupies the last situation. Körte in a series of sixteen cases observed that the greatest prominence of the mass was *below the navel.* The tumor is smooth and may fluctuate; it is not hard and lobulated. On account of its presence the diaphragm may be arched so that the heart is dislocated upward and to the left; the apex is found in the third interspace. It displaces the stomach forward and to the right, the transverse colon downward, the diaphragm and the contents of the chest upward. The cyst may be movable in respiration. It also causes increased dulness behind on the left side, the upper border approaching the angle of the scapula. *Exploratory puncture* in either instance determines the nature of the fluid, and may establish the diagnosis. Boas does not think the chemical character of the fluid is sufficiently typical to establish a diagnosis. (See Examination of Cystic Fluid, page 696.)

Diagnosis. Pancreatic cyst must be distinguished from cancer of the

pancreas or adjacent organs, aneurism, hydatid cyst of the liver, the spleen, or the peritoneum, affections of the retroperitoneal glands, hydronephrosis, cystic disease of the suprarenal capsule, circumscribed peritonitis with exudation, ascites, and cystic disease of the ovary. *Pain* is an important symptom of disease of the pancreas in its more acute manifestations; it must be distinguished from the pain of intestinal obstruction and the pain of perforative peritonitis. The pain is always localized in the region below the xiphoid, or, in general, is confined to the upper half of the abdomen. It exactly simulates the pain of the affections just described. This resemblance is more pronounced because of the association of vomiting and collapse in obstruction and perforative peritonitis. Pain, although not so intense, but of a colicky nature, attended by diarrhœa or constipation, in some instances with intestinal hemorrhage, may be due to *calculous disease of the pancreas.* Frequently this form of pain can be recognized if other symptoms of pancreatic disease, such as glycosuria, steatorrhœa, and intestinal obstruction are present.

CHAPTER XI.

DISEASES OF THE KIDNEYS.

THE kidneys are affected by disease from several sources: (1) the great vascular supply is subject to the alteration which takes place in any large arterial area, either from direct *hyperæmia*, through the influence of the vasomotor nerves (see Hyperæmia), or from passive hyperæmia or *congestion* through the central organ of the circulation. (2) The blood-vessels may be the seat of *thrombosis* or *embolism*, particularly the latter, causing renal infarction. (3) *Infectious* material, as micro-organisms or toxins, is carried to the kidney, and in passing through the structure gives rise to *inflammation* either of an infective or of an irritative character. Similarly, *poisons* that are ingested, and products of metabolism excite irritation and lead to inflammatory changes.

But the kidney is open to attack from sources lower down in the urinary tract. Through the bladder and ureter *infection* may extend upward, causing the consecutive inflammatory processes which are often seen after disease of the urethra, bladder, or ureter. The kidney is at the apex of a system of tubes or channels. Any alteration of them, whether mechanical or functional, has a secondary effect upon the kidney. Obstruction of the ureter or obstruction of the conduits beyond leads to consecutive hypertrophy, inflammation, and atrophy. (See Morbid Processes.) If the urine is abnormal, one of these three causal conditions may be present.

Symptoms due to the Morbid Processes.

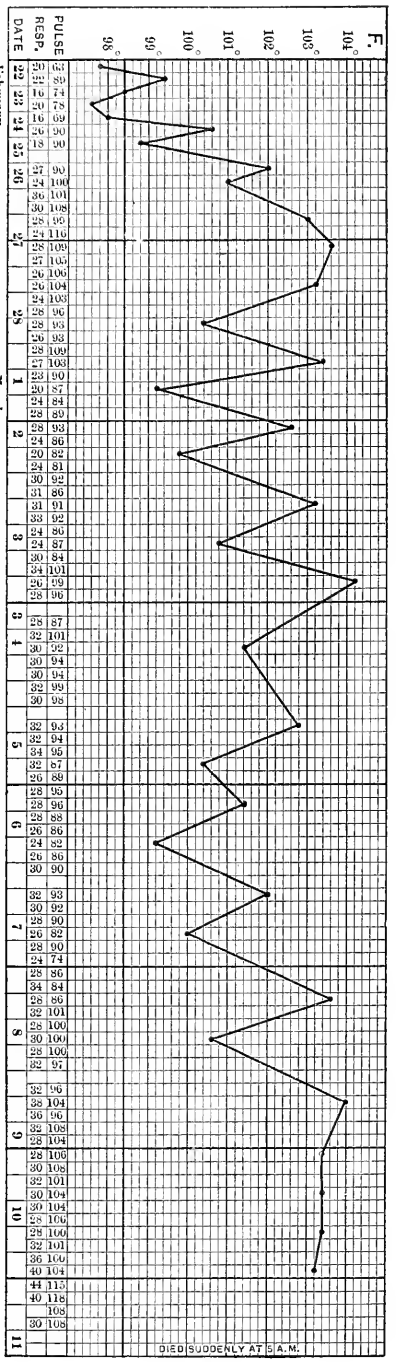
The morbid processes that may take place in the kidney are such as are common to all organs—congestion, inflammation, degeneration, morbid growths, and hydatid disease, and the symptoms are such as accompany similar processes elsewhere. The *general* symptoms of the morbid processes, chiefly fever and emaciation, are not marked except in the case of infectious inflammation or of morbid growths, as carcinoma. *Fever* occurs in acute nephritis, perinephric abscess, suppurative and tuberculous nephritis, pyelitis, and with twists of the ureter, in floating kidney. *Emaciation* occurs in chronic, suppurative, and tuberculous nephritis and carcinoma. Other general symptoms in renal disease are due to the interference with the function of the organs which usually results. *Pain* is the only *local* symptom due to the morbid process; *swelling* is the only physical sign.

Anatomical and Physiological Symptoms.

The symptoms of renal disease are also due to the functional or anatomical alteration of the kidney. But the structure is so closely inter-

woven with the function that morbid changes in one imply morbid changes in the other. As the anatomical alterations are usually beyond the pale of physical investigation, we find that functional symptoms alone are apparent. Hence, we look for *changes in the urine* and for symptoms resulting from abeyance or cessation of the renal function. Rarely we have *enlargement* due to obstruction of the channels causing hydronephrosis.

The symptoms due to alteration of function are: 1. *Uræmia.* 2. *Cardiovascular symptoms,* including *dropsy* and *anæmia.* 3. *Alterations of the urine.* 4. *Disturbances of micturition.* The symptoms of renal disease are therefore both subjective and objective.



Temperature in uræmia simulating typhoid fever.

Fig. 389.

Diagnosis of Renal Diseases.

The recognition of renal diseases is based upon evidence of the history, the subjective diagnosis, the objective diagnosis, the physical examination, and the laboratory diagnosis.

Historical Diagnosis. Age, sex, and habits are important factors. Acute nephritis attends any age, but chronic nephritis occurs late in life and with the onset of cardiovascular degenerations. The habits and hygienic conditions causal in the latter are likewise induced in the former. A previous history of infections, notably scarlet fever, is usual in acute and chronic renal processes. Chronic intoxications, as with metals, are also followed by renal inflammations.

Subjective Diagnosis. The chief symptoms which demand inquiry are pain and disturbed micturition.

Pain. (See Chapter VIII., Part I.)

Disturbances of Micturition. There are four causes of frequent micturition : (1) disease of the kidneys, the ureters, or the bladder ; (2) an increase in the amount of urine, as in diabetes ; (3) concentration of the urine, as in fevers, gout, or acute nephritis ; (4) reflex irritation or pure neurosis.

Increased frequency of micturition occurs in almost all organic affections of the genito-urinary system. It is seen in all forms of congestion and inflammation of the kidneys. In some forms of nephritis the increased frequency may be due to increase in the amount of urine as well as to increased sensitiveness of the organs. In chronic nephritis it may not be noticed save that the patient is called upon to pass urine at night, being aroused from sleep for the purpose. Disease of the ureter and disease of the bladder are also associated with this troublesome symptom. It occurs in its most aggravated and characteristic form in renal colic due to calculus, or when any foreign substance is located in the ureter or bladder. The frequency amounts to six, eight, or even a dozen times in an hour. It is often associated with tenesmus, the patient having a constant desire to urinate, but passing only a small quantity at a time. This form of tenesmus is more frequent in renal calculus and when the bladder or urethra is the seat of disease.

Objective Diagnosis. Uræmia. Under symptoms due to impairment of the functions of the kidney belong the various manifestations of uræmia. Diseased kidneys do not eliminate the products of tissue-waste, which are poisonous materials. The toxic matter is retained within the blood and produces toxæmia which may be acute or chronic. In *acute uræmia* the manifestations develop suddenly and continue but a short period of time, with alarmingly active symptoms, until death or recovery. In *chronic uræmia* the onset is gradual. The manifestations may be limited to one or two conditions, as headache or morning nausea, or they may include the more pronounced symptoms of uræmia.

Latent uræmia was first recognized by Sir William Roberts. It is seen in its most characteristic form in calculous suppression. The patient for several days will have subnormal temperature, myosis, occasional vomiting, and toward the end twitching of the voluntary muscles, and slight drowsiness. At the end of five or ten days coma, convulsions, or dyspnœa ensues.

Nervous Symptoms. *Headache.* The pain is situated in the occipital region and may extend down the neck. It is severe and of a bursting character, and may be associated with giddiness. In both acute and chronic nephritis headache is often the first manifestation ; it may be associated with eye symptoms ; it may be present on waking, and continue only through the morning hours. In acute uræmia it persists throughout the attack. Numbness and tingling of the fingers are often complained of at the same time.

Delirium. The delirium may be mild. This is usually the case in the typhoid state or if a subnormal temperature prevails. It is sometimes attended by delusions. There are often subsultus and picking at

the bedclothes. The delirium may amount to true mania, and the patient may exhibit other maniacal symptoms. On the other hand, the patient may be noisy, restless, and sleepless. Melancholia and delusional insanity may develop after the violent nervous symptoms of uræmia have passed off.

Convulsions. A convulsion may be the first indication of disease of the kidneys, or it may succeed a few days of persistent headache, or follow an attack of uræmic vomiting. The convulsion resembles epilepsy, and hence is known as an epileptiform convulsion. If the spasms recur in rapid succession, the interval is occupied by delirium or coma. If they are infrequent, the patient's mind may be clear in the intervals. Sometimes a focal or Jacksonian epilepsy occurs instead of the true epileptiform convulsion. The *temperature* is usually elevated. In worn-out subjects, or in those who are debilitated from exhaustive diarrhœa or some other cause, the temperature may be subnormal. A temporary blindness often follows the convulsion (*uræmic amaurosis*). Uræmic deafness may occur.

Coma. After the convulsion consciousness may return, or the patient may lapse into stupor, followed by complete coma. Coma may develop without convulsions, or immediately succeed a general convulsion. Headache or eye symptoms may precede the coma. In some instances the patient lapses into a typhoid state, in which the tongue is heavily furred and the breath very offensive. Unless the coma is profound, there is usually some twitching of the muscles of the hands and face.

Local Palsies. Dercum was among the first to call attention to the occurrence of *uræmic monoplegia* or *hemiplegia*. The cases resemble central cerebral disease. The nature of the palsy is inferred from the results of the examination of the urine and the condition of the heart and arteries. Palsy develops suddenly and may occur after a convulsion.

Cramps in the muscles of the calves, particularly at night, are of common occurrence, and should always lead to an examination of the urine.

Pruritus, local or general, is another nervous symptom that may be of uræmic origin.

Pain in the upper abdomen, particularly in the median line, is a frequent precursor of more severe uræmic symptoms. It is of uræmic origin itself. It may be seated in either of the upper quadrants, and thence extend to the lower portion of the abdomen.

Respiratory Symptoms. *Uræmic Dyspnœa.* Modifications of the breathing often accompany symptoms of uræmia. The dyspnœa may be constant or it may occur in paroxysms, or the two types may alternate. A common type in the uræmia of chronic nephritis is the Cheyne-Stokes breathing. Paroxysmal dyspnœa usually occurs at night, and resembles asthma in every respect. Cheyne-Stokes breathing continues even through the period of coma, although it is not necessarily associated with it. (See page 99.)

In addition to dyspnœa, the occurrence of *inflammatory pulmonary complications* may be the first indication that the condition of the urine should be inquired into. Bronchitis, pneumonia, and pleurisy are common complications of uræmia.

Gastro-intestinal Symptoms. *Loss of appetite* is common. It is attended with absolute distaste for food after a small portion has been taken. *Nausea* may be continuous, or more frequently limited to the early morning. *Vomiting* may be paroxysmal, occurring chiefly in the early morning; or it may be sudden in onset, uncontrollable, and continue until nervous symptoms of uræmia develop. Urea is found in the vomitus. The matter ejected is profuse, of low specific gravity, and at first acid in reaction. In cases of chronic vomiting the vomitus may be alkaline. The odor is often sufficient to cause its recognition. *Constipation* is the rule in the course of chronic Bright's disease. One of the manifestations of uræmia is *diarrhœa*, often in the form of sudden profuse serous purging. This may be so extreme as to cause collapse, or may usher in coma and convulsions. *Hiccough*, although a muscular affection, is usually associated with gastric disturbances. Fermentative dyspepsia, gastralgia, chronic gastritis, enteritis, and ulcerative colitis are of common occurrence.

Cardiovascular Symptoms. These symptoms are due to the retention of morbid products. The poison which is not excreted circulates through the system. One of its effects is irritation of the vasomotor nerves of the bloodvessels. Excitation of these nerves causes peripheral contraction of the smaller vessels. At once the flow of blood is obstructed, so that, on account of the contraction, hypertrophy of the heart rapidly ensues. The first prominent symptom, therefore, is due to changes in the heart muscle.

Hypertrophy of the Heart. The most pronounced change is *hypertrophy.* The persistent spasm of the peripheral vessels causes increased arterial tension. The blood-pressure is raised and causes increased accentuation of the aortic second sound. *High tension* in the artery is recognized by the peculiar character of the pulse and by means of the sphygmograph.

Dilatation of the Heart. Unfortunately, hypertrophy of the heart can not always be kept up. If it fails, we then have a second condition of the heart which is frequently found in renal inflammations; it is dilatation. The state of the coronary arteries predisposes to this condition of the heart muscle. The previously mentioned arterial tension favors the development of chronic endarteritis with general atheroma, and the coronary arteries take part in this process. The endarteritis hinders cardiac nutrition, dilatation of the heart muscle follows, and later comes the development of two other conditions, atrophy and myocarditis.

Here may be mentioned other relations of the heart and kidneys: (*a*) We have renal disease following forms of cardiac disease. In dilatation of the heart passive congestion of the particular organ takes place. The kidney quickly becomes the seat of such congestion. In the course of simple dilatation or of valvular heart disease, the secondary dilatation, passive congestion, and chronic inflammation develop slowly. Embolism may also occur. (*b*) Renal disease and cardiac disease may develop at the same time from a common cause, as alcoholism, gout, or endarteritis.

In addition to high arterial tension and accentuation of the aortic second sound, the objective symptoms of atheroma of the aorta and arteries are associated with chronic inflammation of the kidney. (See Endarteritis.) It is important to bear in mind that the two conditions frequently occur together, and also that in all instances of arterial disease

the condition of the urine must be looked into. It is needless to say that symptoms due to rupture of a bloodvessel, particularly in the brain, or to aneurism may supervene in the course of renal inflammation.

Hemorrhages. The arteries are very liable to rupture, causing epistaxis, retinal hemorrhage, hemorrhages from the bowels and lungs, and hemorrhages underneath the skin (petechiæ). Frequent, copious hemorrhages from any portion of the body should call attention to the condition of the urine.

Ophthalmoscopic Changes. The eye-ground should always be examined; indeed, the patient himself by his complaints often directs attention only to the eye, the examination of which discloses the presence of an albuminuric retinitis. The changes may occur in the acute or chronic forms of nephritis, although they are more common in the latter. 1. A diffuse, slight opacity and swelling of the retina, due to œdema. 2. White spots or patches of various sizes, for the most part the result of degenerative processes. 3. Hemorrhages. 4. Inflammation of the intra-ocular end of the optic nerve. 5. Atrophy of the retina and nerve may sometimes result from and succeed the inflammatory changes. These changes may affect one eye only (Gowers). It must not be forgotten that temporary blindness may occur independently of retinitis.

Dropsy. Dropsy may occur in all forms of nephritis. It is most common in the acute varieties, but it is also present in chronic diffuse nephritis. Renal dropsy usually begins in the face. It may develop suddenly in acute forms, and in grave diseases of the kidneys œdema of the eyelids may continue for a long time. All varieties of dropsy, from local œdema to extreme anasarca, are observed. The serous cavities are also filled. The œdema is usually associated with a diminution of the quantity of urine, and its subsidence is attended by increased diuresis. In chronic disease dropsy is usually due to dilatation of the heart. (See page 950.)

Anæmia. Anæmia is a frequent symptom in all forms of nephritis; it is usually marked. It is associated with the peculiar pallor of kidney disease, and attended by all the other usual symptoms of an impoverished state of the blood.

General Symptoms. The cause of renal disease, so far as symptoms pointing to the kidneys are concerned, is often latent. Instead of renal symptoms a generally depraved state of the system with *emaciation* and *weakness* may be the only indication of disease. Lassitude without cause demands an examination of the urine.

Appearance of the Face and Cutaneous Symptoms. In inflammatory affections of the kidney the appearance of the face and the condition of the skin are often characteristic and point at once to an examination of the urine. The face is pallid and of an ivory whiteness; in chronic renal disease the pallor gives way to an ashen-gray or sallow tint, and the skin becomes dry and harsh. In rare instances the skin is covered with a powdery substance resembling hoar-frost, which is derived from urea and is analogous to the chalky deposits of urates known as tophi.

Physical Diagnosis of Enlargement of the Kidney. Renal Tumor.

Enlargements of the kidney may be detected by percussion; the width of the kidney is increased, and the percussion-dulness therefore extends further to the right or left, according as the right or left kidney is affected. As the causes which produce enlargements of the kidney sufficiently great to be detected by percussion do not, with rare exceptions, involve both kidneys at the same time, comparison of the two sides is of great value in the diagnosis.

Renal tumors give rise to swelling in the loin with muscle resistance if there is inflammation. They may extend forward, and are then palpable between the ribs and the transverse umbilical line. The mass is never notched, is usually smooth, and often takes the shape of the kidney if that organ is involved in its entirety. Otherwise the outline is not reniform. The bowel is usually in front of the mass, although in tumors of the right kidney the cæcum and colon may be pushed to the inner side, and in tumors of the left kidney the colon may be pressed outward.

The diseases of the kidney attended by enlargement are: *malignant tumors, cystic kidney, hydronephrosis* and *pyonephrosis, abscess,* and *perinephric abscess.*

Laboratory Diagnosis. **Alterations of the Urine.** (See Chapter XLIII.)

Congestions of the Kidney.

Acute Congestion of the Kidneys. Any diseased kidney may become the seat of acute (or active) congestion. The insult of a prolonged surgical operation and extirpation of one kidney may be followed by acute congestion; but the commonest cause is some irritant poison. Death may take place from uræmia. *Urine:* The quantity may be diminished or there may be total suppression; catheterization is often required. The urine contains albumin in considerable quantities, blood, and numerous epithelial casts.

Chronic congestion of the kidneys is usually a part of general venous stasis—hence also spoken of as passive congestion; valvular heart disease with secondary dilatation, and pulmonary emphysema often give rise to the condition, which is not at all uncommon. *Urine:* the quantity is diminished, the color dark, the specific gravity high—1020 to 1030. Uric acid and urates are present in excess. Albumin is not present at first, but soon makes its appearance at intervals and in small quantities, later becoming constant and more abundant. The sediment contains hyaline casts and a few red blood-cells.

The condition is recognized by its association with congestion in other organs; by the diminution in the amount of urine, its high specific gravity, and excess of uric acid and urates. This form of congestion is serious because it leads to chronic nephritis.

Degenerations of the Kidneys.

Degeneration is always a secondary process and may be either acute or chronic. The primary causes are: inorganic poisons, as arsenic or phos-

phorus; the toxins of the infectious diseases; chronic visceral disease; and disturbances of the circulation.

In *acute degeneration* of the kidneys the quantity of urine may be normal or diminished; albumin is present in varying quantities, and the sediment contains casts and red blood-cells. The urinary changes may be the only symptoms, or signs of uræmia may develop at once. Dropsy and cardiac hypertrophy do not occur.

Chronic degeneration of the kidneys may be a sequel of chronic congestion, or may be produced by alcoholism or syphilis. It may develop during the course of gout or malarial cachexia, and is a not infrequent event in pulmonary phthisis and when there is a chronic suppurative process going on anywhere in the body. *Symptoms:* The simpler forms do not produce clinical symptoms. Loss of flesh and strength, anæmia, and in rare cases the development of the typhoid state mark the course of the severer forms of degeneration. In uncomplicated cases of degeneration there is no hypertrophy of the left ventricle, and albuminuric retinitis is rare. *Urine:* The findings are variable. The urine may be abundant, scanty, or suppressed; the specific gravity is normal, but albumin and casts are present.

Amyloid degeneration of the kidneys is associated with the same process in other organs, and the *symptoms*, when any are present, are due to the anæmia and cachexia of the primary disease and to the involvement of the other viscera. According to Purdy, dyspepsia and diarrhœal attacks are common. The liver and spleen are usually enlarged; œdema is not present as a rule, and uræmia is a rare occurrence. *Urine:* The quantity may be normal, diminished, or increased; it varies from time to time in the same case, being influenced by complicating conditions such as diarrhœa; as a rule the quantity is more abundant than in health. The color is very pale; the specific gravity ranges between 1008 and 1014. Albuminuria of considerable degree is a constant symptom, and hyaline casts and white blood-cells are always found; the presence of other casts points to a complicating nephritis. The distinctive features of the casts found in amyloid disease are their large size and waxy appearance. The *diagnosis* of amyloid disease is based upon the presence of a cause, the urinary changes, and signs of similar disease in other organs.

Inflammations.

The best classification from the pathological point of view of the inflammations of the kidney is that given below; but this classification does not always fit the clinical picture.

Acute Nephritis. *a.* **Acute Tubal or Parenchymatous Nephritis.** This is essentially an acute degeneration of the tubular epithelium, with little or no involvement of the glomeruli. The cells of the tubules are cloudy, swollen, granular, often fatty and vacuolated. Congestion is a marked feature in this variety, which is the type of mild kidney-lesion occurring in the course of typhoid and scarlet fever, diphtheria, pneumonia, and the other infectious febrile diseases, and is produced by the

action of the specific toxins. It is often classified, and perhaps more properly, as *cloudy swelling* or *parenchymatous degeneration.*

b. **Acute Exudative Nephritis.** In addition to the degenerative changes of the preceding form (*a*), there is an exudation of serum, fibrin, or red blood-corpuscles into the capsule of Bowman and into the tubules. When the exudation of red blood-corpuscles is very great, the term *acute hemorrhagic nephritis* is employed.

c. **Acute Glomerular Nephritis.** This form is characterized by a proliferation either of the cells of Bowman's capsule—*acute capsular glomerulonephritis*—with pressure-changes in the glomerular tufts; or of the cells of the glomerular capillaries—*acute intracapillary glomerulonephritis.* The glomerular lesion explains the oliguria or anuria frequently observed in this type of the disease. The lesions characteristic of (*a*) and (*b*) may and usually do accompany the glomerular changes. This is the type of nephritis commonly seen during convalescence from scarlet fever, and often spoken of as *scarlatinal nephritis.*

d. **Acute Interstitial (non-suppurative) Nephritis.** This unusual form occurs practically only in connection with diphtheria and scarlet fever. The characteristic pathological alteration consists in an infiltration of lymphoid and plasma cells between the tubules, in severe cases obliterating entire groups of tubules. Extensive degeneration of those structures results, with practically no change in the glomeruli. No distinctive urinary changes have been described.

e. **Acute Interstitial (suppurative) Nephritis or Abscess of the Kidney.** For the sake of completeness this form should be added. (See page 1120.)

The ætiology of all the forms of acute nephritis is practically the same. The form (*d*), which is practically always a complication of diphtheria or scarlet fever, differs from the others in that the urine is scanty, high-colored, and of high specific gravity; but the number of observations on the urine in this type of nephritis is small.

Chronic Diffuse Nephritis. *a.* **Large White Kidney.** This is often described as *chronic parenchymatous nephritis* in contradistinction to the chronic interstitial or contracted kidney; the term *chronic tubular nephritis* has also been employed. The kidney substance shows a slight, uniformly distributed increase of connective tissue; fatty or hyaline degeneration of the tubular epithelium; and, occasionally, a small accumulation of lymphoid cells. The glomerular tufts are lobulated and smaller than in normal specimens; sometimes there is a slight accumulation of serum in the capsule of Bowman. The condition may lead to the small white kidney.

b. **Amyloid Kidney.** This form is usually included under the degenerations (*q. v.*).

c. **Small Red Kidney.** Clinically, this form is known as the kidney of *chronic interstitial nephritis* or as the contracted, granular, gouty, or arteriosclerotic kidney; the last-named variety differs from the others only by its association with arteriosclerosis. The histological changes are: over-growth of connective tissue, atrophy and fibroid changes in the glomeruli, atrophy of some of the tubules with dilatation of others, and

an infiltration of lymphoid cells. In the arteriosclerotic form the characteristic lesions are found in the vessels.

Acute Tubal or Parenchymatous Nephritis (Cloudy Swelling). This form of nephritis is characterized by congestion, exudation of plasma, transudation of red and white blood-cells, and changes in the epithelium.

Causes. There may be a history of exposure to cold, but the disease occurs at times without definite cause. It is a frequent complication or sequel of the infectious diseases, notably scarlet fever, and is the expression of a peculiar type of typhoid fever. The kidney of pregnancy is one of the manifestations of this form of nephritis; the course of diphtheria, erysipelas, pneumonia, and septicæmia is frequently marked by its development; it may complicate dysentery and acute tuberculosis; and it is one of the modes of termination of diabetes.

Symptoms. The course of the disease may be mild, presenting only the urinary changes, or there may be, in addition, local and general symptoms.

In mild cases the *urine* is diminished in amount; micturition is frequent; the color is dark, the specific gravity usually high. There is a small amount of albumin, with a few epithelial and blood-casts, and sometimes blood. At the termination of the disease hyaline casts are found.

In severe cases the disease is ushered in by *chill*, attended and followed by *pain* in the loins, with *fever*, *headache*, and restlessness. There are a large amount of albumin and an abundance of hyaline, granular, epithelial, and blood-casts. Free red and white blood-cells and epithelium from the pelvis and tubules are found. The fever continues; the pain in the loins is sometimes very severe, and, unless the urine is examined, may be mistaken for lumbago. Within the first forty-eight hours the characteristic symptoms that follow the chill and accompany the urinary changes are headache, sleeplessness, more or less profound stupor, muscular twitchings, and general convulsions. Eye symptoms may be present. Instead of cerebral symptoms there may be marked dyspnœa; and with both, nausea and vomiting are of common occurrence. The heart's action is increased in force and frequency. The left ventricle rapidly becomes hypertrophied. The aortic second sound is accentuated. The *pulse* is hard and exhibits the characteristic features of high tension. From the onset of the first symptom, or within the first week, two other striking phenomena arise, *dropsy* and *anæmia*.

Dropsy or œdema is one of the most constant symptoms. It appears first in the face, especially the eyelids, and may be limited to this region, being most marked in the morning. From the face, in severe cases, it extends to the lower extremities and to the scrotum, and thence over all the body. *Anasarca* is the name applied to general dropsy in which the connective tissue is infiltrated with serum; it is recognized by the pallor of the swollen surface, the pitting on pressure, and the absence of heat and pain. (See page 228.)

Effusion may take place into the serous cavities, the pleura, pericardium, or peritoneum. In some instances there is œdema of the mucous membranes, as the conjunctiva, the soft palate, and the glottis.

Dyspnœa may be a pronounced symptom, being due to uræmia (uræmic asthma), œdema of the glottis, effusion into the pleura, or bronchitis. If dilatation of the heart occurs, the ensuing dyspnœa may be a direct result or due to the secondary œdema of the lungs.

With or without the occurrence of nausea or vomiting there is always *loss of appetite*, and the bowels are usually *constipated*.

The *fever* is usually moderate and irregular in type. *Prostration* is common; often there is emaciation. Symptoms of *uræmia* may occur at any time.

Exudative nephritis with excessive *pus* formation is of sudden onset, characterized by high fever and extreme prostration. There are rapid *emaciation* and the early development of the *typhoid state*, which is preceded by delirium, headache, and stupor, with great restlessness. In this form there is but little, if any, dropsy. Large numbers of red and white blood-cells and the usual casts are found in the urine. There is not so much diminution in the quantity of the urine as is usually seen. The disease may arise without apparent cause, or complicate scarlet fever or diphtheria. This form is very fatal, and resembles *acute meningitis*, from which it is diagnosticated by the changes in the urine.

Acute Glomerular Nephritis. In this form there are an over-growth of connective tissue and an excessive growth of the capsule-cells in the glomeruli, in addition to the lesions of the first form. The whole kidney is not necessarily affected, but only portions at a time. *Symptoms:* The onset is sudden. The subjective symptoms previously described are present in a marked degree. *Nervous* symptoms (uræmia) are most pronounced. *Dropsy* develops rapidly and to an extreme degree. There is rapid *loss of flesh with anæmia.* The remaining symptoms tally with those of the first affection.

The *urine* is scanty, bloody, and of high specific gravity. The microscopical appearances are like those of acute exudative nephritis. If convalescence is established, the urine becomes more abundant, with a corresponding fall in the specific gravity. The albumin and casts may persist for a time, but eventually disappear.

The *diagnosis* of the various forms of acute nephritis is based upon the examination of the urine. Ætiological associations are of value. The more pronounced cases follow scarlet fever and pregnancy. In the latter condition the course is usually slow. There may be no symptoms until the onset of uræmia. In some instances the disease resembles typhoid fever. In cases in which the onset is sudden, with early uræmic symptoms, it must be distinguished from epilepsy, delirium, and mania.

Chronic Productive or Diffuse Nephritis (Large White Kidney). In chronic inflammations the formation of new tissue always takes place. *Exudation* practically does not occur. The essential difference in the various forms lies in the amount of connective tissue. In this form the amount is slight and the degenerative changes in the tubules very prominent. In the contracted form there is great increase of the connective tissue; sclerosis of glomeruli is its predominating characteristic.

Causes. The diffuse form usually follows acute productive nephritis and chronic congestion or degenerations of the kidney. It develops in the

course of syphilis, tuberculosis, endocarditis, disease of the bone, and prolonged suppuration. Frequent exposure to cold and wet, residence in damp dwellings, and the alcoholic habit are causal conditions. It usually occurs in middle life, more frequently in men; but as a primary disease usually attacks young adults.

Symptoms. The disease develops slowly. General symptoms may be first observed. *Dropsy* may develop at first and continue throughout the disease, or recur at long intervals. The appearance of the patient is striking. The skin is of a peculiar *pallor* and pasty in appearance. The scleræ are very white. The *anæmia* which gives rise to the pallor is profound, and often closely resembles that of pernicious anæmia. The anæmia is due to diminution in the hæmoglobin and reduction in the number of red blood-cells.

Headache and *sleeplessness* are common symptoms. Pronounced acute uræmia does not often occur. *Chronic uræmia* may prove fatal by the patient lapsing into a typhoid state in which delirium alternates with stupor.

Urine. It must not be forgotten that the course of the disease and the urinary symptoms are often quite variable in chronic nephritis. The urine may be normal in amount, but during the exacerbations it is scanty or suppressed. The specific gravity and the amount of urea lessen. In the most rapid cases it varies between 1012 and 1020. In chronic cases it falls as low as 1005 and even 1001. In the later stages the amount of the urine and the specific gravity may both be increased. Albumin is present in large amounts. When the disease is most active and the dropsy at its height, the quantity of albumin is very large. In the quiescent period of the disease the amount is lessened. Epithelial, fatty, and granular casts are abundant; red blood-cells are often found.

Albuminuric retinitis frequently develops in the course of the disease.

Dyspnœa is often observed, and may be due to any one of the many causes previously described as producing this symptom in the course of nephritis. It is frequently limited to sudden attacks which develop in the night or early morning. There is often some bronchial catarrh.

Nausea and *vomiting* are common symptoms. The appetite is lost.

Hypertrophy of the left ventricle takes place in all cases, except in those persons who had been previously weakened by other disease. The right ventricle is often hypertrophied also. The second aortic sound is accentuated, and the pulse is of high tension. Symptoms such as *headache* and *vertigo* arise on account of the profound *anæmia*.

Course. Delafield has well outlined the course. The constant symptoms are anæmia, dropsy, and albuminuria. 1. The symptoms may be continuous and progressive in severity, death taking place at the end of one or two years on account of dropsy or uræmia. 2. The symptoms may continue for several months and the patient finally improve. Recurrent attacks take place, the symptoms being more severe with each attack. In the intervals of the attacks there is a small amount of albumin in the urine. 3. The patient may apparently recover, but the urine continues to be of low specific gravity, and contains some albumin. A fatal attack of uræmia, or an apoplexy, or the onset of an acute disease may cause an

exacerbation of the renal symptoms. 4. The symptoms may persist in a mild degree for years, the patient at the same time feeling comparatively well. 5. Spasmodic dyspnœa may be the first and only symptom for a long time.

The disease is characterized by remissions and exacerbations. During the exacerbations any one of the prominent symptoms that occur in renal inflammations may be present. Œdema is the one symptom which occurs most frequently, and is likely to continue the longest. The disease lasts from three months to three years, and may pass into the second variety of chronic inflammation.

Chronic Interstitial Nephritis. This form of nephritis is called also *chronic, non-productive, diffuse nephritis, granular kidney,* or *cirrhosis of the kidney.*

The kidneys are diminished in size, the capsules are adherent, and the surface is roughened. There is an over-growth of connective tissue with atrophy of the epithelium and of the tubules, and dilatation of some of the tubes, forming cysts.

Causes. This form of nephritis follows chronic congestion of the kidney, and is also caused by alcohol, lead, gout, syphilis, malaria, and by chronic endarteritis. The last condition, as well as cirrhosis of the liver and pulmonary emphysema, frequently develops hand-in-hand with the nephritis. This form of nephritis is notably prevalent in several generations of different families, so that a hereditary history is often readily obtained.

Symptoms. The onset of the disease usually occurs late in life, although well-defined cases may occur as early as the twenty-fifth year. The progress at first is very insidious, and the disease may have advanced to an extreme stage without the occurrence of a single symptom. Death, indeed, may be due to other causes; or a person in apparently perfect health may suddenly manifest symptoms of uræmia, or may develop apoplexy or some other usual accompaniment of interstitial nephritis. With the exception of the state of the urine, the only symptom present may be the loss of flesh and strength. At the same time the skin becomes dry and harsh. Œdema, however, is not usually present unless there is dilatation of the heart. Special symptoms are due to uræmia, to changes in the heart and arteries, and to neuroretinitis.

Uræmic Symptoms. These symptoms may occur at any time in the course of the disease. Headache is most common and constant. It may occur early in the morning only, or continue throughout the day. It may be continuous and cause sleeplessness. General neuralgic pains may be present instead of severe headache. Muscular twitchings or general convulsions may be other pronounced symptoms, or, instead, delirium, mild or violent, stupor, and coma may come on. These symptoms occur suddenly or develop very gradually. In acute uræmia with the above-mentioned cerebral symptoms there is peripheral spasm of the arteries, causing high arterial tension, and there is elevation of the temperature. The fever may rise to 103° or 104° F., but is usually about 102°, and is irregularly continuous. After the patient lapses into deep coma, if the attack is fatal, the tension of the pulse is lost, and it is increased in

frequency and diminished in strength. In chronic uræmia the cerebral symptoms develop gradually. The temperature is likely to be subnormal, particularly if diarrhœa or other debilitating influence is coincident. The pulse is rapid and feeble.

Pulmonary symptoms due to uræmia are quite common. They may be the first expression of uræmia. This is seen in all forms of nephritis. The most marked symptom is dyspnœa, which is spasmodic and of short duration. The attacks may occur frequently, and are usually increased by exertion and aggravated by a recumbent posture. The shortness of breath may occur in the early morning hours, or may continue throughout the day.

Pulmonary symptoms other than those due to uræmia may be caused by an intercurrent bronchitis, pneumonia, or pleurisy. Chronic bronchitis or œdema of the lungs may be present on account of dilatation of the right heart. The chief pulmonary symptoms that point to these conditions are dyspnœa and cough.

Spasmodic dyspnœa is the first and sometimes the only symptom for a long time. Later the renal symptoms become pronounced, pointing to the true nature of the disease.

Gastro-intestinal Symptoms. Catarrhal gastritis almost always complicates nephritis. In addition, gastric symptoms due to uræmia, and hence to deficient action of the kidney, ensue. The most common is the occurrence of morning nausea or of morning vomiting; the occurrence of spasmodic vomiting at irregular periods, or the occurrence of violent, acute vomiting, which is followed in two or three days by other symptoms of uræmia. The bowels are usually constipated. When the disease is complicated with cirrhosis of the liver, intestinal catarrh is common, and intestinal ulceration with consequent diarrhœa is frequently found. The onset of uræmia may be characterized by violent and profuse serous purging, which of itself may cause collapse and death.

Neuroretinitis is a frequent complication of nephritis, and may advance more rapidly than other complications, so that dimness of vision, blindness, or other eye symptoms may cause the patient to consult an oculist before attention is called to the condition of the kidneys. The occurrence of this complication points at once to the necessity for an examination of the urine.

Vascular Symptoms. It is common in the course of an interstitial nephritis to have accidents due to the condition of the arteries that accompanies this disease.

On account of the atheroma, and aided by the hypertrophied heart, rupture of the vessels frequently takes place. Apoplexy is, therefore, of common occurrence, and hemorrhage into other organs sometimes occurs.

The renal disease is often not suspected until after the patient has had an attack of apoplexy. The course of this form of nephritis is varied very much by the occurrence of complications, notably emphysema, endocarditis, or cirrhosis of the liver.

Catarrhs. There is always a tendency to chronic inflammations of the mucous membranes and to acute inflammations of serous membranes in the course of chronic diffuse nephritis. It is necessary, therefore,

when local inflammations of this character are present, to make thorough and repeated examinations of the urine, especially in a patient over forty years of age, with a history of one of the causal factors previously mentioned.

URINE. The quantity is increased, the color clear, and the specific gravity low. The albumin is small in amount, or may be absent. Repeated examinations extending over a considerable period of time may disclose its presence. Hyaline casts are present in small numbers. In some cases it may be necessary to examine a dozen or fifteen slides before they are found. Sometimes there are a few red blood-cells. Rarely the urine is bloody at irregular periods in the course of the disease, or actual hæmaturia may take place.

HEART. The left ventricle hypertrophies. The aortic second sound is accentuated. The pulse is of high tension. The arteries become more prominent and present all the signs of endarteritis. In the later stages, as nutrition fails, dilatation of the heart takes place, with regurgitation at the mitral valve and the development of a train of symptoms due to these changes. Among others we find general malaise, palpitation of the heart, dyspnœa, œdema, and visceral congestions.

Course. Several clinical forms of interstitial nephritis are observed. In the latent form the disease may have advanced to an extreme degree without any symptoms of renal disease during life, death taking place from an intercurrent disease or accident. On the other hand, palpitation of the heart may be the only symptom complained of, and the observer finds a hard pulse, general atheroma, and hypertrophy of the left ventricle with accentuation of the second sound. Apart from this the patient may enjoy very good health. The danger lies in the occurrence of pneumonia or inflammation of a serous membrane. Often the local inflammatory symptoms are slight or masked by the symptoms of renal disease, which develop rapidly.

In another group of cases some special symptom only may be complained of. In some instances it may be gastric catarrh, in some eye symptoms alone may be present, while in others hemicrania or other forms of headache are observed. With the headache there is usually vomiting. Again, we may have constant neuralgia or persistent muscular rheumatism as the only symptom. Nose-bleed is a symptom which may be the only indication of chronic nephritis, particularly if the epistaxis occurs frequently.

In other cases the course is not latent, but characterized by a series of attacks at varying intervals.

During the attacks the symptoms resemble those of the acute form of nephritis, with acute uræmia, the occurrence of dyspnœa, loss of appetite, nausea, and vomiting. The tension of the arteries is higher at the time of the attacks. The urine contains albumin, and is of low specific gravity during the time of the attack; during the interval albumin is found at irregular times.

Suppurative Nephritis (Abscess of the Kidney). Infectious matter is conveyed to the kidney either through the *blood*, as in pyæmia and ulcerative endocarditis (rarely dysentery and actinomycosis), or by the *ureters*,

as when it follows pyelitis or cystitis. A wound may infect the kidney directly.

Symptoms. The symptoms are those of the primary disease, and the affection is usually recognized only post mortem. Or the symptoms are merely those of suppuration. Pus is seen in the urine only on rupture of the abscess into the pelvis of the kidney.

Tuberculous Nephritis. Fever, emaciation, anæmia, and prostration characterize the course of the disease. Tuberculosis is usually found elsewhere. There may be no other symptoms. Sometimes hydronephrosis is present. A tumor is often present. It may be in the loins, or may be in front, above, and a few inches to the right or left of the umbilicus. The *urine* is normal or contains pus and detritus, or even bacilli. The finding of the latter is often necessary to establish a diagnosis. In all instances of pyuria, renal tuberculosis should be suspected. Catheterization of the ureters may show what organ is affected. The urine should then be centrifugated, and the sediment examined for bacilli, and, as Reynolds advises, a portion inoculated into guinea-pigs. The tuberculin test may be employed. The testicles and bladder should be carefully examined for primary tuberculosis.

Tuberculosis of the kidney presents symptoms like those of *pyelitis*, *renal calculus*, or a *new growth*. It is almost impossible to distinguish any one of the four until an interval has elapsed. In all cases the patient suffers from dull pain, sometimes with a bearing-down sensation. Hæmaturia occurs, and the patient is liable to attacks of renal colic. These symptoms may continue until a tumor can be made out. Even before this pain, which may extend all along the urinary tract, will be elicited on palpation. With the occurrence of the tumor the general symptoms of tuberculosis arise. Further diagnosis is based upon the results of the urinary examination.

Sarcoma and Carcinoma of the Kidney.

Either disease may be primary or secondary. Sarcoma may be congenital. The tumor may occur at any age, but is relatively common in young children. Twenty-five out of sixty-seven cases collected by William Roberts occurred in children under ten years of age. In older persons it is often preceded by calculus.

Symptoms. In some instances there are no symptoms during life. In others the disease may advance considerably before it presents any signs. If symptoms are complained of, they are usually limited to pain, the occurrence of hæmaturia, or the development of a tumor. The pain is dull and seated in the lumbar region. It may be neuralgic in character; and, indeed, there may be a true *sciatica* with paresis of the leg from pressure of the tumor. The *tumor* (Plate XLVII., Fig. 2) is firm; its surface is smooth or nodulated. It may be felt in the loins, and in front above the umbilicus, a few inches to the right or left of the median line; the descending colon lies in front of the tumor. The latter may grow with great rapidity and attain an enormous size, filling the abdominal cavity and giving rise to pressure-symptoms in surrounding organs. The growth

occurs more often anteriorly and downward toward the pubis because there is less resistance in these directions. As rapidly growing cancers are soft, the tumor frequently exhibits a certain degree of elasticity which may be mistaken for fluctuation. It is movable either by the hands or with respiration. On percussion the resistance is increased and the note is dull, except in front, where the colon, which has been pushed forward, gives a tympanitic note. If the colon should be flattened out between the tumor and the abdominal wall, it may be felt as a band stretching across the tumor, with dulness on percussion. In such a case inflation of the colon will be of great assistance in the diagnosis. Rare physical signs are pulsation and a blowing murmur. The *hæmaturia* may be constant or intermittent. The clots of blood may cause renal colic.

The *general symptoms* are those of carcinoma. A marked rapidity of the pulse has been noted in several cases. In girls a premature development of hair on the pubes and in the axillæ and pigmentation of the skin have been observed.

Hemorrhage is an early symptom, and in the absence of nephritis or cystitis should always suggest tumor. It may occur early and may be intermittent or persistent. In some instances it occurs but once; usually it is repeated. When excessive, the growth is never innocent. Pain is not of much diagnostic value, and may be absent until perinephritis occurs. Symptomatic *varicocele* may occur. The examination of the urine, save that it discloses the presence of blood, is negative. In this sense it is of value. Pus occurs if there is secondary infection or if calculi precede the growth. Rarely fragments of carcinoma are said to be detected. In order to determine which kidney is affected, the urine should be obtained separately from each organ.

The tumor must be distinguished from tumors of the lymphatic glands, of the liver, of the spleen, and of the ovary. It must not be confounded with psoas abscess or perinephric abscess, which causes a tumor in the lumbar region.

Cystic Kidneys.

1. Congenital. The kidney consists of a small mass of cysts filled with clear fluid. It may interfere with the birth of the child on account of its large size.

2. Acquired. The causes are trauma and obstruction of the ureter, the presence of which is determined by catheterization. The symptoms are those of a fluctuating renal tumor. The urine may be normal, or hæmaturia may be present.

Hydronephrosis.

Hydronephrosis may be congenital; it may be caused by obstruction of a ureter by a stone; the pressure of a tumor; twisting of the ureter, as in movable kidney; or represent an exudation.

Symptoms. In addition to the symptoms of the causal condition we have, upon the development of hydronephrosis, the presence of a *tumor* arising in the region of the kidney and extending toward the middle line.

Sometimes fluctuation can be detected; often it can not. Variations in the size of the tumor may occur with changes in the amount of urine passed. Exploratory puncture and the finding of a fluid with elements of urine in it are valuable means of diagnosis; but if the hydronephrosis is old, this fails, as the fluid loses its urinary character, and can not be distinguished from that of an ovarian cyst, for instance. When on one side, the urine may be normal; when on both sides, it is diminished; anuria and uræmia may occur. If pyelitis is present, pyuria is observed.

Pain may or may not be present. Gastric symptoms are very common. Either constipation or diarrhœa is seen. Hypertrophy of the left ventricle may occur, as in *chronic* nephritis.

Hydronephrosis consists in a dilatation of the kidney pelvis with urine, which is prevented from escaping by obstruction of the ureter, either by the pressure of a tumor or by disease of the bladder or ureter itself. In time the kidney atrophies from the pressure and a large cyst forms. The tumor has the physical characteristics of pyonephrosis, but the history is different, and if there is any discharge, it is free from pus. As in pyonephrosis, the tumor may become small after a copious discharge—in this case of urine; or may even wholly disappear if the obstruction is removed. This sign is pathognomonic.

Intermittent hydronephrosis is associated with *movable kidney*, hence it is more frequent in women. It is characterized by the development of a renal tumor which disappears at varying intervals, pain, nausea, and vomiting; during this period the urine is scanty. In a few hours or days there is an increase in the amount of urine and the tumor subsides.

Diagnosis. If absolute obstruction continues, the diagnosis must be made by the detection of a fluctuating renal tumor, the absence of fever and signs of suppuration, and by the result of exploratory puncture. The urine is usually free from pathological changes.

Hydronephrosis when very large may be confounded with ascites, but hydronephrosis is rarely bilateral, and the fluid does not change its level upon change of position of the patient, as is the case with ascites. The history of the two conditions is different.

An *ovarian cyst* can usually be traced into the pelvis; it does not carry the colon in front of it, and hence is dull, even on superficial percussion, and it leaves the loins resonant.

Pyelitis. Pyonephrosis.

Pyelitis. The antecedent causal factors of pyelitis, which is rarely primary, are: severe infectious diseases (typhus, variola, diphtheria, pyæmia); toxic substances ingested (cantharides, etc.); chronic nephritis; inflammation of the bladder or ureter; strictures of the ureter or urethra; hypertrophy of the prostate; spinal palsies of the bladder; calculus; parasites; and blood-clots; infection being the active cause.

Symptoms. *Urine.* Pus in the urine with pelvic epithelium—although it is not safe to base a diagnosis on the presence of the latter; casts of the canals opening into the pelvis, which are more characteristic; epithelial casts and casts containing micro-organisms. The urine is often increased,

acid, and contains pus and albumin, rarely blood. *Pyuria* may be the only renal sign. In all forms of pyuria above the bladder Kelly withdraws the pus by catheterization and suction. He allows the catheters to remain for from ten minutes to four or five hours, in order to estimate the functional power of each kidney. Of course, the pus is studied microscopically and bacteriologically. *Pain* in the region of the kidney, often severe, is complained of, although it may be absent. When present, it is often of a tearing character. A *tumor* is often present, most prominent in the loin or in the abdomen. In the latter the mass can be felt two inches to either side of the umbilicus, usually above the transverse line.

Abscess of the Kidney. Pyelitis differs from abscess of the kidney. The latter may be the result of a local infection from the pelvis of the kidney or may be pyæmic. In abscess of the kidney there is some fulness in the loin of the affected side. The kidney is felt to be enlarged, and is tender and painful. A tumor may be detected anteriorly. The diagnosis is based on a study of the cause (acute nephritis, pyæmia, impacted calculus in the ureter, erysipelas) or the detection of blood and pus in the urine, which is scanty, and on the constitutional symptoms. The progress of the case is usually acute. If the abscess is *tuberculous*, tubercle bacilli can be detected in the purulent sediment of the urine, and there will be other foci of tuberculosis with a corresponding clinical history.

Pyonephrosis. When the pus is confined by an occluded ureter, the pelvis is over-distended. In pyonephrosis the *tumor* is tense, smooth, and globular. Fluctuation may be detected. Tenderness is usually absent; the course is slow and does not affect the general health so much as abscess. The pus may be discharged copiously from time to time, and the tumor be therefore diminished in size. The urine may be occasionally almost clear. *Fever* is irregular, remitting, or septic. The fever and pyuria may be the only symptoms. If the *bladder* is healthy, vesical symptoms fail to aid in diagnosis. Pyonephrosis arises secondarily to pyelitis, and often after the latter has lasted some time.

Perinephric Abscess.

This may occur as a primary disease in apparently healthy individuals, or follow one of the infectious diseases.

Perinephritis arises usually from extension of inflammation and suppuration from the kidney, but may be the result of strain, exposure to cold, or injury. Perinephritis may also be pyæmic, and occur after infectious fevers and in actinomycosis.

Symptoms. The **secondary forms** have symptoms of the primary disease, and, later, swelling and pain in the renal region.

Primary Form. Chills and fever, pain, difficulty in defecation. The general condition suffers. Finally, in all cases, a swelling, hard at first, develops in the lumbar region; then œdema of the skin follows, and fluctuation is detected. The abscess may descend and point above Poupart's ligament. It may press upward and cause dyspnœa. Great tenderness and pain in the region of the swelling may arise, and the pain

may radiate to the leg. Irregular septic fever and chills appear. The *urine* is not generally changed unless there is some communication with the pelvis or ureter. The patient lies on his back, turned toward the affected side. The knee and hip of this side are flexed, and the thigh is rotated outward. The affection may simulate *coxitis* and *appendicitis*.

The *swelling* of a perinephric abscess appears in the lumbar region of the side affected. It is rounded in form and doughy. (Da Costa.) Like other kidney tumors, it is not affected by respiration. The usual signs of confined suppuration exist, and pulmonary or pleural complications may occur. As the abscess progresses, the local signs of suppuration become more marked, the skin reddens, and pus may be discharged externally.

The most marked subjective symptom is *pain*, which may amount to agony, and is paroxysmal; soreness from restricted motion of the psoas muscle is apt to be complained of.

A *tumor* was present in the loins in 65 out of 71 cases analyzed by Fenwick, but did not generally manifest itself until the inflammation had made considerable progress. There is dulness on percussion even in the early stage, and later, fluctuation. The general symptoms are vomiting, constipation, fever, and sometimes rigors. The disease is more common in males than in females (61 males to 39 females in Fenwick's cases).

Hydatid Disease.

A *hydatid cyst* of the kidney presents the usual physical signs of such cysts. A fremitus may be detected, or small cysts may be found in the urine.

The disease is comparatively rare. Usually there are no symptoms until a tumor is felt. Then pain gradually develops. The cyst may open into the pelvis of the kidney, and cysts or scolices be discharged, with *colic*.

Pyelitis and cystitis may also develop.

Echinococcus cysts may become inflamed and lead to general pyæmia. Puncture of the discovered tumor, which should be performed in most cases, is otherwise the only means of diagnosis. It must be differentiated from hydronephrosis and ovarian tumors.

Nephrolithiasis (Renal Calculus).

Renal calculi vary in size from "sand" through "gravel" to "stones." The last may be from the size of a cherry to one large enough to fill the pelvis of the kidney. They consist usually of uric acid, and are hard, brownish-red or blackish, and crystalline; the larger ones are arranged in distinct layers. More rarely we have calculi of calcium oxalate, extremely hard and nodular. Some stones have alternate layers of the two salts; others consist of phosphates, but usually the centre is of uric acid or calcium oxalate, the phosphates having been deposited after the urine became alkaline. Very rare forms are composed of cystin, xanthin, indigo, etc.

Frequency. A consideration of the frequency of the affection and some ætiological data aid in the diagnosis. It is not a common affection. I have had 29 cases in private practice and 11 in hospital practice (1898). Thirteen cases only have been treated in the Presbyterian Hospital in twenty-five years, during which time over 8000 cases of all kinds were treated.[1] It is a disease of the middle and upper classes. This is particularly true of uric acid calculus. It is not, in my experience, a disease of the old or the very young. The youngest subject was twenty-five years of age; the oldest, sixty-nine. Twelve of my private patients were of the female sex, seventeen of the male sex. There does not seem to be much difference between the two sexes in regard to frequency. Most authorities, however, hold to the preponderance in women, the ratio being as 3 is to 1. Sedentary occupation and an indoor life are predisposing factors.

Symptoms. With the exception that some present no symptoms whatever, the cases may be divided into three groups:

a. Calculi may remain in the pelvis of the kidney, and not cause any renal symptoms. They may cause gastric disturbance or catarrh of the bladder or renal pelvis. There may be occasional pain in the lumbar region, the cause of which is unsuspected.

b. The calculus may excite pain, hæmaturia, and frequent micturition.

c. The stones may attempt to pass from the pelvis of the kidney into the ureter, causing renal colic, the symptoms of which have been described above. In the intervals between the attacks of colic the patient may be free from symptoms.

The symptoms ascribed to the presence of a calculus in the pelvis of the kidney are *pain, intermittent hæmaturia, pyuria, pyelitis, renal intermitting fever, acute orchitis, frequent micturition,* and *renal colic.*

Pain. Pain of the affected organ is the most constant symptom, and is increased by movement, by jolting, and by pressure. Indeed, pain induced by pressure is of as great significance as spontaneous pain. It frequently is persistent, and even continues in any position assumed by the patient. The pain is seated not only in the regions just indicated, but extends also along the ureter, from the loin to the front of the abdomen. It may persist for some time, at a point on either side of the umbilicus above or below it, or at a point on the surface of the abdomen opposite the brim of the pelvis. Thence the pain radiates into the bladder, either above the pubis (the hypogastric region) or into the testicle, or down the inside of the thigh. It may be in the loin and at the end of the penis at the same time, or cause lancinating irritation along the whole urinary tract. In rare cases the pain is in the kidney of the healthy side. The pain of renal colic is always associated with frequency of micturition, with or without pain during the passage of the urine. Unlike that of renal calculus, it comes and goes, and is more commonly intermittent and paroxysmal. Very frequently, however, it is constant and localized either in the region over the kidney or anteriorly in the region mentioned. In my experience it comes on during the day, particularly the after-part of the day, and not, as Jacobson would have us believe, at night. That it may occur spontaneously is not so much a

[1] "Renal Calculus," J. H. Musser, Philadelphia Medical Journal.

peculiarity of renal calculus as that it can be excited by pressure or movement.

Pain is of greater diagnostic significance in *renal calculus* than in any other renal affection. Every attribute that has been applied to pain belongs to the pain of renal calculus. Its very vagaries render its presence one of the most valuable signs. Often, however, it behaves like the flitting nerve-aches of hysteria, and we must see to it that this counterfeit is not passed upon us. Urinary phenomena do not serve for the distinction; other neurotic manifestations or the stigmata of hysteria aid in the diagnosis. The pain may be aggravated by the function of menstruation and even bear a close relationship to it.

Hæmaturia. Hemorrhage from the kidney is the classical symptom of stone. It is the most constant and positive symptom of renal calculus. Prior to the use of the centrifugal machine, blood, when in small amounts, no doubt escaped the eye of the observer, partly because it was destroyed as the urine advanced in decomposition during the period it was set aside for sedimentation and partly because the small number of the corpuscles rendered them difficult to find. Excluding all causes outside of the kidney—*i. e.*, of vesical and ureteral origin—renal hæmaturia may be due to congestion and inflammation, to infarct, to new growths, to tuberculosis, to renal calculus, or to parasites. The fevers and infections, and scurvy, purpura, leukæmia, and hæmophilia are responsible for a number of cases.

In six years 2923 samples of the urine of 1997 persons were critically examined in my laboratory. Blood was present in 364 cases, detected by microscopical examination alone. The hæmaturia resulted from congestions or hyperæmias (pregnancy, goitre, heart disease, the fevers, infections, and jaundice) in 56 cases. In 42 cases the hæmaturia occurred in the course of acute and chronic Bright's disease, and in 19 more in arteriocapillary fibrosis, either of renal or of cardiac origin. Gastric disorders, rheumatism in many forms, gout, neurasthenia, and anæmia account for 81 of the cases, conditions always associated with the copious discharge of urinary salts, which are irritating. Vesical disease accounts for 17 cases, renal calculus for 28, and in 20 the diagnosis was not noted at the time and is forgotten. All the cases of renal calculus had hæmaturia. It is not an intermittent phenomenon alone, but one that is constantly present.

It is necessary to eliminate all sources of urethral, vesical, and ureteral hemorrhage before coming to a conclusion that the hemorrhage is of renal origin. *Cystoscopy* must be resorted to, of course, and possibly *ureteral catheterization*, which demands expert skill. If the hemorrhage is free, the time of its passage in the act of urination must be determined. The reaction of the urine must be borne in mind. It is true that catheterization alone may suffice to determine which kidney the hemorrhage comes from. *Blood-cylinders* are rarely if ever present in renal calculus. They denote hemorrhage from the renal substance.

In a person of middle life that may be supposed to have, by virtue of heredity, occupation, and habits, uric acid or oxalic acid tendencies, and in whom no cause for the hemorrhage can be found in the urethra, blad-

der, or ureter, the chances are that it is of pelvic origin, due to the irritation of gravel or of urine heavily loaded with salts.

Klemperer[1] has recently called attention to hæmaturia from healthy kidneys as the result of over-exertion, in one case from horseback-riding, in another from bicycling. He also reports 4 cases of hæmophilia and a group due to an angioneurosis. Hyaline casts were not present, although blood-cylindroids were. General symptoms of neurasthenia supported the diagnosis in the angioneurotic cases.

Pyuria. Pus in the urine is looked upon by all authorities as almost essential to the diagnosis of renal calculus, but in my experience this product of inflammation is usually absent. Of the 28 cases which I have examined in 15 there was no pus; in 6 a few cells or a very small quantity was found (4 uterine, cause obvious); in 1 it was noted as considerable (old gonorrhœa and syphilis, 4 examinations); in 1 a small quantity (male, cause assignable); in 1 it was small in amount—twice only in some 50 examinations; in 1 it was abundant and due to genito-urinary infection as well as to pyelitis. Pyuria is not present unless an accidental infection has taken place from the lower tract.

Albumin. In 21 patients albumin was found. It was in large excess in 3, due to coexisting Bright's disease. As a trace it is of frequent occurrence and does not imply a coexisting nephritis.

Urinary Casts. Casts are present in the urine in nearly all cases of renal calculus. The specimen must be centrifugated. They are hyaline —not abundant—long and narrow. Their persistence without other kinds, with or without albumin, is diagnostic of renal irritation, and with other signs points unfailingly to calculus.

Specific gravity of the urine is an aid in the diagnosis. Its persistence above the normal is both a comfort and a sign. It enables one to exclude renal cirrhosis and eliminates hysteria or a renal neurosis.

Frequent micturition is not in my experience an indication of stone in the kidney, save when attempts are made to pass the stone (colic), although it is spoken of as a symptom of value by most authorities.

Paroxysmal renal fever, allied to hepatic fever in its expression, rarely occurs, but when present, may be due to calculus. It may also be due to absorption of retained products if the kidney is floating and becomes twisted. It may be due to pyelitis.

Duration of symptoms and family history are also valuable data.

Diagnosis. Middle life is a predisposing factor, and persistent hæmaturia is symptomatic, but pyuria rarely so, while albuminuria and hyaline casts in urine of high specific gravity are prominent elements of the symptom-complex upon which a diagnosis is based.

The diagnosis can be established by the symptom-complex of *pain*, local *tenderness*, persistent *hæmaturia*, *albuminuria*, and *casts* (the cardiac, vascular, or renal origin of which is excluded), by the phenomena of *renal colic*, and by the *passage* of *fragments* of stone.

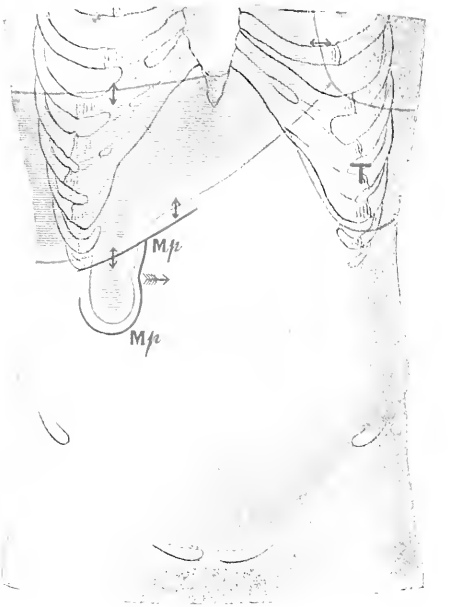
If the hemorrhage persists after prolonged rest, it is more likely of cancerous or tuberculous origin.

The *differential diagnosis* must be made from *appendicitis*, *movable* and

[1] Deutsche med. Wochenschrift, March 4, 1897.

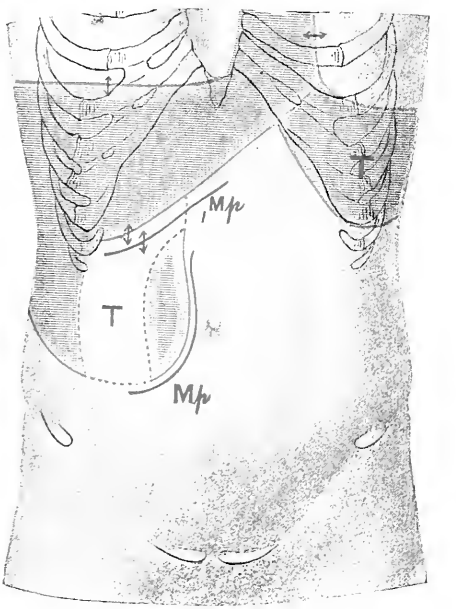
PLATE XLVII.

FIG. 1.



Movable Kidney.

FIG. 2.



Sarcoma of the Right Kidney.

twisted kidney, biliary colic, and other affections that simulate these conditions. Catheterization and exploration through the ureter are required in many cases. Hollander believes that in a large number of cases a diagnosis can be made without the aid of catheterization, and fears the danger of infection from below.

Kelly performs ureteral catheterization very skilfully and aspirates the ureters, thereby bringing down fragments of calculi. He also explores the ureters with hard-rubber bougies tipped with wax. He can determine the presence of calculi by the markings on the tip of the bougie.

Movable Kidney.

Movable kidney is usually seen in women after the age of forty years, who have done manual work or have borne many children.

It is frequently preceded by a history of injury, unusual lifting or strain, followed by tearing or dragging sensations in the abdomen. Pain may continue for several weeks after the injury and then subside, and the accident be forgotten, or subjective sensations may continue.

In other instances the movable kidney is part of a general visceral displacement seen in men, though more frequently in women. Gastroptosis and gastro-enteroptosis can usually be made out in such cases.

Symptoms. The symptoms are due to the local dragging or pulling of the kidney on its bloodvessels and nerves, or to reflex symptoms, or to pressure upon adjacent organs.

The *pain* of movable kidney is usually referred to the right or left of the median line; sometimes to the hypogastrium. It may be constant, dull, and aching in character, aggravated at intervals; or it may be paroxysmal. The paroxysms continue for three or four days, during which time other subjective symptoms arise. The attacks are known as *Dietl's crises*. Nausea may attend the paroxysms, or be more or less constant. Sometimes vomiting takes place. Swelling and tenderness of the kidney are found. The pain, inducing shock, vomiting, and local tenderness, may simulate peritonitis. Instead of pain, a dragging sensation is experienced. The patient may be aware of the presence of a tumor or lump in the abdomen as well as of its movability.

The *reflex* symptoms are chiefly referable to the nervous system. Emotional disturbance is observed when the organ is displaced. Hysteria may be present. Palpitation of the heart is a common reflex symptom. There are often depression of spirits and hypochondriasis. *Jaundice* may occur from pressure, and the intestine may be occluded from the same causes.

Urinary Symptoms. At the time of a crisis the urine is scanty and hydronephrosis may develop. As the kidney slips back into its bed, the twisting of the ureter is relieved, and copious discharges of urine take place. Polyuria and perhaps frequent micturition are not uncommon.

Objective Symptoms. (Plate XLVII., Fig. 1.) The abdominal walls are usually relaxed, and may or may not contain a large amount of fat. *Movable kidney* is best detected by palpation. The patient should stand with the body bent forward and the hands resting on a chair, as described

elsewhere. The organ is recognized by its rounded borders, its bean shape, its movability, the detection of the hilus and perhaps of the pulsation of vessels in it, and by the fact that it can be replaced. Palpation causes a sickening feeling analogous to that experienced when a testicle is compressed, but less in degree. Percussion will demonstrate that a body, supposed from palpation to be the kidney, is a solid organ. The tumor can be found to the right or left of the median line, freely movable and changing its position with that of the patient. If the tumor is situated on the right side, it may be in close proximity to the liver, or be felt opposite the umbilicus, or often in the iliac region. When near the liver, by careful palpation the fingers can be introduced between the border of the liver and the mass. Usually it does not move with respiration, but sometimes it is found to do so. On the left side it may be as high up as the margin of the ribs. It is generally felt in the midclavicular line, a little above the level of the umbilicus.

In a case recently under the writer's care the woman, aged fifty-five years, experienced pain in the abdomen about once a month, to the right of and above the umbilicus. At times nausea and vomiting accompanied the attacks; at other times marked depression or hysteria. Anuria always occurred and continued for a variable time—not longer than five days. With one of the paroxysms a tumor was found in the region of the gall-bladder, movable with respiration, but distinctly defined from the liver by placing the fingers between the lobe and the kidney. It moved with each change of position of the patient, and at first the hilus could be distinctly felt. As the pain continued, the anuria persisted and a marked change in the tumor was observable; it gradually increased in size, and a portion of it fluctuated; it was round and partook of the character of a cyst. The fluctuation was detected by placing the hand on the tumor in front and pressing firmly toward the other hand placed in the loin above the pelvis. After several days a copious discharge of urine took place and the swelling subsided.

Diagnosis. Movable kidney may be confounded with tumor of the *gall-bladder*, tumor of the *pylorus*, and with tumors in the *pelvis*. It is not likely to be confounded with an omental tumor, carcinoma, or tuberculosis, because the phenomena of these processes are not present and ascites does not occur, nor is there rise of temperature, as in many cases of tuberculosis. As pointed out by Henry Morris, tumor of the gall-bladder and movable kidney are frequently of conjoint occurrence. Movable kidney is distinguished by the absence of previous history or of symptoms or signs indicating disease of the gall-ducts. If jaundice is present, it is not so intense as in tumors of the gall-bladder. While the gall-bladder is movable, it is less distinctly so than movable kidney. The gall-bladder moves in an arc of a circle, the centre of which is at the edge of the right lobe of the liver. It can be pushed further to the left than to the right, but never downward like a movable kidney. Moreover, the gall-bladder is always palpable; the movable kidney can not always be felt. The gall-bladder, if it contain calculi, is very hard compared to the kidney. Anuria does not occur. The kidney tends to spring back to its place in the loin; the gall-bladder to the anterior part of

the abdomen. Even if the gall-bladder is enlarged, the kidney may be felt by bimanual palpation; while the opposite does not obtain. In cancer of the pylorus the emaciation and anæmia are more pronounced than in movable kidney. The vomiting usually present in that affection and the physical signs of dilated stomach can be made out. Tumors of the pelvic organs are determined by examination according to the usual methods.

CHAPTER XII.

DISEASES OF THE MUSCLES.

Myalgia.

MYALGIA is an inflammation of the muscles produced by a "cold," by trauma, or by rheumatism. There are pain on movement and spontaneous pain in the muscle; it is tender on pressure, and may be the seat of spasm.

Muscular Rheumatism.

In this variety of rheumatism there is pain in the affected muscles, which often comes on suddenly in the night or is first noticed when the patient attempts to rise in the morning. The pain when the patient is at rest may be inconsiderable, rarely amounting to more than a dull, aching, sore feeling; on attempting to move, bend, twist, or straighten himself, however, the patient catches himself suddenly on account of the agonizing, tearing, or burning pain. When the muscles are relaxed, the patient is fairly comfortable. Sudden movement is the most painful. The affected muscles are tender to the touch and to sharp blows. Muscular rheumatism may be acute or chronic. In the latter the symptoms are very much like those of chronic articular rheumatism, except that the muscles and not the joints are affected. There is the same proneness to recur in unfavorable weather and in cold, damp seasons.

The disease receives different names according to the muscle affected. The most common subvarieties are: *lumbago*, in which the muscles of the small of the back are affected; *pleurodynia*, in which the intercostal muscles suffer; and *torticollis*, in which the sternomastoid and trapezius are painfully contracted.

In *lumbago* the patient holds himself rigid and is unwilling to rotate the trunk upon the vertebræ. Often the most comfortable position is that in which he sits and bends slightly forward over another chair. Motion is painful, but pressure is not. Fever is absent. There is a history of repeated attacks or of exposure, such as lying upon damp ground. Lumbago needs to be distinguished from disease of the spinal membranes, from disease of the vertebræ, aneurism, abdominal abscess, and diseases of the uterus and ovaries. The diagnosis of rheumatism is arrived at by exclusion.

In *pleurodynia* there is usually tenderness upon pressure as well as upon motion and deep inspiration. The pain is of the same sore, burning character, aggravated by coughing and sneezing. The patient breathes as little as possible, and often bends over toward the affected side to lessen the motion. Pleurodynia is distinguished from pleurisy by the absence

of fever, cough, and, above all, of friction-sounds. In intercostal neuralgia there are painful points upon pressure, whereas in pleurodynia firm pressure is grateful, though tapping is painful.

In *torticollis* the head is drawn to one side and fixed in that position. The sternomastoid especially is rigid and tender on pinching. In spinal affections the head is retracted, and there are antecedent symptoms, as headache and darting pains with fever.

Thomsen's Disease (Myotonia Congenita).

This is an hereditary disease, and may occur in several generations of a family. Tonic cramps take place in the muscles when voluntary movements are attempted. The disease begins in childhood, rarely after puberty. The muscles become rigid and fixed when put in action. The lack of voluntary control of the muscles is shown by the slow contraction and relaxation when voluntary efforts are made. The rigidity may wear off and the limb can then be used. It is particularly noticeable when walking is attempted. As the leg is advanced slowly it may remain stiff for a second or two, but after it becomes limber the patient can walk for hours. If he stops walking, the same difficulty is experienced when he starts again. Both arms and legs are affected. Patients are usually well nourished, however. There are no atrophies. The muscles are irritable, so that mechanical stimulus or pressure causes tonic contraction. Movement and cold aggravate it. Sensation and the reflexes are not affected, and there is no evidence of disease of the cerebrospinal system, save the occurrence of hypochondriasis in some cases. The myotonic reaction described by Erb is induced. (See electrical diagnosis—Diseases of the Nerves.)

Paramyoclonus Multiplex.

In this affection there is clonic contraction of the muscles. It is usually confined to the extremities, and occurs in paroxysms. It may have been caused by sudden twitching or violent motion. The clonic spasms at first do not interfere with the patient's occupation, but gradually they increase. Both legs are affected, and the number of contractions varies from 50 to 150 a minute. The contractions may be rhythmical. In severe cases the muscles of the back and abdomen contract violently. Tremor of the muscles may be present in the intervals. (For *paralysis, spasm, tremor, contraction*, etc., see Diseases of the Nervous System.)

Myositis. Inflammation of the Muscles.[1]

In inflammation of the muscles there are pain, swelling, and loss of power. In universal myositis the inflammation begins in the lower extremities and gradually involves other muscles of the body. They are swollen, hard, and painful on pressure. Atrophy supervenes in groups of muscles. The muscles may become more or less rigid. Local œdema of the skin over the muscles occurs. The progress is gradual, and death ensues when the respiratory muscles are involved.

[1] See also Trichinosis.

The three cardinal symptoms that attend the disease as described by Löwenfeld are: (1) swelling of the extremities due to subcutaneous œdema and swelling of the muscle, causing functional disturbance; (2) extension to the muscles of respiration and deglutition; (3) a more or less extensive eruption. This last is erythematous, its distribution is usually general but irregular, and may be followed by pigmentation. The disease must not be confounded with *trichinosis*. In the latter, examination of a small portion of muscle reveals the trichinæ.

Progressive ossification of the muscles is rare. The muscle tissues undergo gradual ossification, either in localized spots or in widespread areas. Inflammation of the muscle precedes the ossification. As the inflammatory swelling subsides, the muscles become hard and are gradually converted into bony tissue. The disease lasts many years.

CHAPTER XIII.

DISEASES OF THE NERVOUS SYSTEM.

THE semeiological classification of nervous diseases presents many difficulties. Many forms that are closely analogous in their symptoms are widely different in their pathology or ætiology, and many diseases present such variations in their symptom-complex that at one period they could properly be placed in one group and at another period elsewhere. In general it may be said, however, that the diseases of the peripheral motor neurons differ so widely from those of the central motor neurons that they can be classified as two separate groups, and in a third group would come the diseases of the sensory neurons. Combinations of these three groups, producing on their part rather clearly marked complexes of symptoms, may then be described, and finally the general and local diseases of the brain and cord. An entirely separate group, characterized by peculiar symptoms, comprises the so-called functional nervous diseases, or the neuroses.

DISEASES OF THE PERIPHERAL MOTOR NEURONS AND THE MUSCLES.

DISEASES CHARACTERIZED BY PURE MOTOR DISTURBANCE.

Progressive Muscular Atrophy.

Two forms are recognized—the *scapulohumeral type of Erb* and the *facio-scapulo-humeral type of Déjérine-Landouzy*. The scapulohumeral type of the disease commences about the age of puberty in the majority of cases. It is frequently distinctly hereditary or familiar, cases having occurred either among the ancestors or in other members in the family in the same generation. The onset is very gradual; usually the muscles of the shoulder are first affected, especially the pectorals and the latissimus dorsi. Next the adjacent muscles are involved, followed by the muscles of the arms, thighs, and finally those of the calf. There is gradual loss of power corresponding to the atrophy of the muscles, but reactions of degeneration do not occur. As a result of the wasting, peculiar alterations appear in the configuration of the body—that is, the shoulder-blades become prominent, lordosis develops, and, as a result of the weakness of the glutei, it may be necessary for the patient to rise, as in the pseudohypertrophic form, by climbing up his legs. The gait, as a result of the atrophy of the quadriceps, is waddling in character. Sensory disturbances are absent. The reflexes are not altered in the early stages, but as the muscles gradually atrophy and become weaker, the ten-

don-reflexes undergo a corresponding diminution. The electrical reactions remain normal until late in the course of the disease, and even then show only a diminution of response proportional to the degree of muscular atrophy. In doubtful cases, or for the purpose of confirming a probable diagnosis, a small portion of a muscle may be excised and examined for the characteristic histological changes. This is most easily accomplished with the muscle-harpoon.

The *facio-scapulo-humeral type* (*infantile form*) commences earlier in life—about the third or fourth year. It is also hereditary, and the symptoms are essentially the same, excepting that the earliest sign of atrophy appears in the muscles of the mouth and the eyelids. As a result, the patient acquires a peculiar and somewhat characteristic expression. The eyes are partially open even during sleep, the lips remain apart, and saliva dribbles from them. Ultimately, the other muscles of the body are involved, and the disease assumes the characteristics of the preceding form.

Pseudohypertrophic Muscular Dystrophy.

This disease commences in early life—from the third to the sixth year. Ordinarily, the muscles of the calves are first involved. These become greatly enlarged, hard, and there is great loss of power. Other muscles of the legs are next involved; then those of the back, and perhaps the arms. Not all the muscles that undergo atrophy show a preliminary hypertrophy. The electrical reactions remain normal, and the loss of power is due merely to the atrophy of the true muscle-substance. The gait is waddling, and the patient is unable to arise from the ground except by getting upon the hands and knees and then gradually climbing up his legs. There is usually lordosis or scoliosis, and occasionally contractures occur, leading to formation of club-foot. Sensation remains unimpaired throughout the disease. The reflexes show diminution corresponding to the loss of muscular substance. The intelligence of the patient in this, as in the two preceding forms, remains intact. The course is slowly progressive.

DISEASES CHARACTERIZED BY MOTOR DISTURBANCE, WITH DEGENERATIVE CHANGES IN THE MUSCLES.

Progressive Neural Muscular Atrophy. (Charcot-Marie Hoffmann Type. Peroneal Type of Gowers.)

This is a disease of early life, the first symptoms appearing just before puberty. The muscles first affected are those of the feet and hands; usually in the former, the peronei, the extensors of the toes, and the small muscles of the foot; in the latter, the interossei and the muscles of the thenar and the hypothenar eminences. The affected muscles show distinct fibrillary twitchings and usually the characteristic reactions of degeneration to the electrical current. These reactions of degeneration are also present in the nerves. There is usually a coarse, irregular tremor, and the atrophy of some of the muscles with contractures of others gives

rise to various deformities, such as the *ape-hand*, the *main en griffe*, or, if the foot is first affected, to *foot-drop*. Later the foot assumes the position of equinovalgus or equinovarus. In this disease there is sometimes involvement of the sensory fibres, and the patients may complain of slight paræsthesia or even of pain, but the nerve-trunks are not sensitive. Hypæsthesia is also occasionally present. The process usually is restricted to the limbs, the muscles of the trunk and face escaping; bulbar symptoms are almost unknown, and the functions of the bladder and rectum are not disturbed. In a form of this disease described by Déjérine under the title of *Infantile Hypertrophic and Progressive Interstitial Neuritis* there are, in addition to the above changes, the symptoms of locomotor ataxia—that is, Romberg's symptom, lancinating pains, ataxic gait, and even disturbance of the pupillary reflexes. The nerve-trunks become enlarged and can be felt beneath the skin.

Progressive Spinal Muscular Atrophy. (Chronic Progressive Anterior Poliomyelitis. Type of Duchenne-Aran.)

The idiopathic form of this disease usually commences about middle life. The hereditary form appears, as a rule, somewhat earlier. The course varies somewhat, but in general is as follows: The first changes are observed in the muscles of the hand, particularly in those of the thenar eminences, giving rise to the formation of the *ape-hand*. The interossei and lumbrical muscles are next involved, and the interosseous spaces become deeper, the fingers become gradually weakened, and ultimately are fixed in a semiflexed condition—incomplete *main en griffe*. The muscles show fibrillary twitching and give the reactions of degeneration to the electrical current. The process affects first one hand, usually the right, and then the other. As the disease progresses it next involves the muscles of the shoulder, especially the deltoids, and later the muscles of the upper arm, and then those of the forearm. Finally, the muscles of the back, and even those of the lower extremities, become involved. Sensory disturbances are rarely present. Occasionally the patients complain of slight pains and paræsthesiæ. The reflexes are lost early. The course of the disease is exceedingly slow, and for a long time a considerable amount of compensation for the muscular disability is acquired. The emaciation is extreme, but total paralysis occurs only very late in the disease. Death ultimately results from the involvement of the intercostal muscles and the diaphragm, giving rise to respiratory failure. It is usually due to a terminal pneumonia. In addition to the typical course described, the disease may assume many other forms, and sometimes for long periods appears to be arrested.

Acute Anterior Poliomyelitis. (Infantile Spinal Paralysis.)

This ordinarily occurs in early life, between the first and twelfth years, but may appear considerably later. Frequently it is epidemic, and many cases may be observed in a limited district. It is really an infectious disease, commencing with chills, fever, headache, pains in the limbs and

back, and occasionally convulsions, and characterized by the rapid appearance of flaccid paralysis in one or more limbs. The onset is usually sudden, and the paralysis may occur before the development of the general symptoms. The legs are more frequently involved than the arms; the muscles are usually affected in functionally similar groups, such, for example, as the flexors of the upper arm. The paralyses are more extensive at first than later, many of the muscles recovering after subsidence of the acute symptoms. The affected muscles are flaccid, atrophy rapidly, and, during the second week, begin to show the reactions of degeneration; when the atrophy becomes complete, all electrical response ceases. Secondary contractures usually appear in one or two months, and may produce deformities, particularly the various forms of club-foot, scoliosis or lordosis, and contractures of the hand. The tendon- and skin-reflexes are lost. Sensation is not disturbed. Circulation in the paralyzed limb is usually poor. The disease almost invariably occurs in children, and subsequently the affected extremity does not grow so rapidly as the other. Occasionally adults are attacked.

Chronic Anterior Poliomyelitis.

Chronic anterior poliomyelitis is characterized by the slow development of paralysis in one or more groups of muscles of the extremities of the body. The flexors are more frequently involved than the extensors. The muscles show fibrillary twitchings and give the reactions of degeneration, and the paralysis is usually flaccid. The process is usually self-limited, but bulbar symptoms may appear and cause death. The disease closely resembles progressive spinal muscular atrophy.

Muscular atrophies secondary to joint-lesions also occur. These are purely functional in character, and ordinarily there are neither reactions of degeneration nor fibrillary twitchings. Sometimes, however, both phenomena are present.

Periodic Paralysis. (Goldflam's Disease.)

This is a disease characterized by the occurrence from time to time of paralysis of all four extremities. The paralysis is usually flaccid in type, occurs without pain, and is associated with extraordinary increase in the electrical resistance of the skin. The disease usually occurs in several members of the same family, the paroxysms lasting three or four days.

DISEASES CHARACTERIZED BY DISTURBANCE OF MOTION OCCURRING WITHOUT REFERENCE TO ANY DEFINITE PORTION OF THE CENTRAL NERVOUS SYSTEM.

Chorea.

Sydenham's chorea is a disease of early childhood. The attacks may recur for a number of years. They are nearly always more severe in winter than at any other season. Girls are affected slightly more frequently than boys. It is characterized by irregular twitching movements

affecting various groups of muscles in the body that are usually functionally associated, so that the movements appear to be the result of voluntary innervation. These movements may be generally distributed, or more pronounced on one side than on the other, or may even occur in only one part of the body. They may involve the muscles of the face, the arm, the leg, or the muscles of the trunk, particularly the diaphragm, giving rise to an irregular, jerking inspiration. They may vary in severity from slight, almost imperceptible contractions to severe, general convulsive movements in which the violence is so great that bruises or even fractures may occur. They cease during natural or artificial sleep. As a rule, the affected limbs are slightly weaker, and in some cases this paralysis is very pronounced (*paralytic chorea*). The mind is usually clear, but there may be loss of memory or irritability of temper. In a few cases with violent movements there are pronounced insomnia and violent delirium (*chorea insaniens*). Speech may be affected either as a result of choreic movements of the lips or on account of psychic disturbance. The reflexes, sensation, and electrical reactions of the muscles are normal. Chorea is often associated with articular rheumatism and endocarditis, and is therefore probably an infectious disease. Occasionally in the violent form fever occurs.

Huntingdon's Chorea.

Huntingdon's chorea is characterized by the development, between the ages of twenty and forty, of choreiform movements of moderate degree, associated with gradually progressive dementia. The disease is strictly hereditary, occurring only in the offspring of those who have suffered from it; in some families it has been traced through five generations. The twitchings resemble those of chorea, but are rarely violent, and are often associated with a slight rigidity. The earliest mental symptom is usually loss of memory; later, there may be delusions of grandeur or melancholia. Usually life is prolonged to an advanced age, the mental symptoms gradually passing into the type of severe senile dementia. A curious feature is the tendency of the patient to avoid society.

Chorea Electrica.

There are several varieties of this condition—one occurring in children, characterized by lightning-like contractions of groups of muscles, sometimes those of the trunk or those of the extremities; another, *Dubini's disease*, which appears to be an infectious process, commences with violent pains in the head, neck, and back, slight fever, and general convulsions. Muscular contractions occur, usually involving all the muscles of the body. These attacks are characterized by their frequent recurrence and brief duration. Death is the usual termination.

Paramyoclonus Multiplex.

This is a disease, possibly hysterical in nature, characterized by lightning-like contractions in groups of muscles, which do not, however, produce movements that in any way resemble co-ordinated actions. Often

the patient from time to time emits a peculiar sound resembling a grunt, probably the result of diaphragmatic involvement. The electrical reactions are normal, and the reflexes are sometimes slightly increased.

Habit Spasm.

Habit spasm is characterized by the repetition of some peculiar, unnecessary movement, such as shrugging the shoulders, winking the eye, rubbing the elbow against the side, etc. Emotional disturbances or the presence of bystanders always increase the symptoms.

Saltatoric Spasm. (Jumpers' Disease. Latah.)

Saltatoric spasm is a hysterical manifestation in which the patient, whenever he or she attempts to stand, is compelled to rise on the toes or even to spring from the ground. Often after such movement the patient falls. The spasm disappears if the patient lies down, but may be produced by pressure upon the soles of the feet. It often occurs as an epidemic manifestation, affecting whole communities at once.

General Tic. (Maladie de Gilles de la Tourette. Maladie des tics convulsifs.)

General tic is a psychical condition characterized by curious movements of the limbs, grimaces, and the utterance of words that have no relation to the environment, and are often profane or obscene (*coprolalia*), or the imitations of sounds heard (*echolalia*). It usually appears during early adult life, but may develop in childhood. The patient becomes more or less melancholy, and may even be violently insane.

Paralysis Agitans.

This is characterized by a peculiar, fine tremor of the extremities, rigidity of the muscles, a characteristic attitude, disturbance of gait, and gradually progressive paresis. The disease usually occurs late in life, although cases have developed between the ages of twenty and thirty. The first symptom noticed is slight rigidity or impairment in agility of the arms. Later this rigidity involves all the muscles of the body, including those of the face, and there is a peculiar rigidity of the facial muscles, causing loss of expression, which is perhaps the most characteristic symptom of the disease. The rigidity also causes the patient to assume a typical attitude: the body and head are thrust forward, and the arms are slightly flexed and swing forward; if the patient stands, the knees and hips are slightly flexed, and the trunk is carried back for the purpose of balancing; the whole attitude, as well as all the movements, indicates stiffness. It will now be found that the patient will have difficulty in rolling over, if lying down, and that there is difficulty in commencing to walk and afterward a tendency to take quick steps (*festination*). The patient, if watched, will be seen to have from time to time a

slight movement forward or backward, which, if he is standing or walking, may cause him to fall in one direction or the other (*propulsion, retropulsion*). The symptom can often be elicited by giving the patient a slight push. Speech is also involved, difficulty in articulation being characterized at first by slight halting and then by the rapid utterance of the words. The tremor usually commences in the hands; it is spoken of as pill-roller's tremor (*q. v.*). The tremor of the head is a to-and-fro nodding movement. There may also be irregular movements of the toes or legs. The tremor is diminished or abolished temporarily by voluntary movement, and disappears during sleep. It is increased by excitement. In a few cases the tremor is entirely absent or occurs only at intervals (*paralysis agitans sine tremore*). Objective sensory disturbances are rare; the patients usually suffer from a persistent subjective sensation of heat, which is occasionally associated with an actual increase of the surface-temperature. The patients are excitable, in spite of the lack of facial expression, and may complain of insomnia. Trophic changes are rare, thickening of the skin is often present, and has been supposed to be the cause of the rigidity; but the skin is sometimes very delicate.

Tetany.

Tetany is probably an infectious disease characterized by cramp of the muscles of the arms and the persistence of peculiar nervous and mental alterations. The attack usually commences with paræsthesia or pain in the limbs; then the muscles controlling the fingers become stiff. The flexors gradually contract and draw the fingers and thumb together—the so-called obstetrical hand. The spasm is tonic in character, and may last for several minutes or even for many hours. It is often associated with intense pain. Nearly always it is bilateral. During the interval it may be reproduced by prolonged, severe pressure upon the nerve-trunks, particularly the median nerve (Trousseau's sign). The muscles show marked irritability to mechanical stimuli, particularly those of the face, and twitching may be caused by tapping upon the trunk of the facial nerve, upon the malar bone, or over the infra-orbital foramen (Chvostek's sign). The muscles show extreme electrical irritability, contract to very weak currents, and in some cases AOTe and COTe have been obtained (Erb's sign). Finally, the patient is extremely sensitive to the induced current (Hoffmann's sign). During the attack, and even during the interval, there is sometimes slight œdema of the face, hands, and feet, and the latter have a tendency to assume a partial equinovarus position. Often there is slight fever. Very rarely trophic changes occur, such as disturbance of the growth of the hair and nails or eruptions upon the skin. The disease appears to be endemic in certain localities, particularly in Vienna. It occurs most frequently in the months of February and March, and, curiously enough, has a special predilection for shoemakers. Tetany is also a symptom of certain morbid processes. Occurring after thyroidectomy, it often runs a severe course to a fatal termination. It may be present in myxœdema. It may also be associated with various disorders

of the gastro-intestinal tract in children, or with dilatation of the stomach in adults, and in this condition renders the prognosis very grave.

Occupation Neuroses.

These are characterized by the development of pain in the limb employed when the attempt is made to perform some habitual movement. They ordinarily occur in early adult life, particularly in neurotic individuals. In a few cases distinct hereditary influence can be traced. Great stress is laid upon the fact that the subject has been in the habit of performing the motions that cause the pain, such as writing in a faulty manner, holding the pen clumsily, and writing with the fingers alone. He first notices that he becomes more readily fatigued than usual, and there may be dull pains in the joints or in the palm of the hand. The painful sensations may then extend up the arm, often as far as the shoulder. They are rarely severe, but by their persistent, dull character are extremely annoying. The motor symptoms are characterized by a tonic spasm of the muscles employed in grasping the pen, so that it is held too tightly, and often there is difficulty in holding it properly. From time to time the spasmodic condition may increase and cause inaccurate strokes. The writing is usually heavy and often quite illegible. The muscles apparently never degenerate. The electrical reactions are normal or only slightly altered. If the patient learns to write with the left hand, the symptoms of the disease usually develop in it after a short time. Similar symptoms occur in piano-players, violin-players, dairy-maids, telegraphers, and various other persons who are obliged to perform the same type of movement for long periods.

Thomsen's Disease. (Myotonia Congenita.)

This is characterized by the occurrence of tonic spasm as the result of voluntary innervation of the muscles. It is described in Chapter XII., Diseases of the Muscles.

DISEASES OF THE SENSORY NEURON, WITH DISTURBANCES OF SENSATION.

These are generally included under the term *neuralgia*. Neuralgia is a condition characterized by pain of a dull, burning, or shooting character that occurs in the distribution of some particular sensory nerve or nerves. The pain may be remittent or intermittent and is often regularly paroxysmal. It is exaggerated, as a rule, by external irritation or emotional disturbance, and persists for some time after the exciting cause has ceased to act. During the attack the pain may extend beyond the limits of the particular nerve involved, and be felt in the surrounding or even remote parts of the body; this is called irradiation. The nerve-trunk is often tender, not only during the attack, but also during the interval, and there are special *tender points* along its course, usually where it passes through or over a bone. Associated symptoms are often present. The most

common are the vasomotor disturbances, the area of distribution of the affected side showing persistent or paroxysmal flushing or occasionally pallor, especially at the beginning of a paroxysm. Secretion of sweat is sometimes increased, and there may be exaggeration of the activity of glands supplied by the nerve. Occasionally there is marked œdema of the skin, and sometimes a herpetic eruption. Atrophy or occasionally even permanent thickening of the skin may occur. Very rarely in neuralgia there is local graying or falling out of the hair. Motor symptoms may also occur. These consist of spasmodic twitching that may be associated with exacerbations of the pain or more or less transient paresis. Neuralgias due to various general conditions sometimes have a characteristic localization. Thus in diabetes, sciatica occurs; in malaria, supra-orbital neuralgia; in neurasthenia, occipital neuralgia.

Special Forms of Neuralgia.

Neuralgia of the Trigeminal Nerve. This usually occurs in only one branch of the nerve, and is commonly unilateral. The terms ophthalmic, supramaxillary, and inframaxillary neuralgia are used to designate the special forms. Only one branch may be involved, as in supra-orbital neuralgia. The pain is paroxysmal and very severe, and is often referred by the patient to some supposed source of peripheral irritation, as disease of the nose, carious teeth, etc. It is usually associated with increase in the secretion of various glands, such as the tear-glands, the salivary glands, the nasal mucous membrane, etc. Trophic changes are not uncommon. These may vary from herpetic eruptions and graying of the hair to atrophy of the soft parts and even of the bones of the face. Occasionally trophic lesions also appear in the cornea.

Tic douloureux is a particular form that results apparently from disease of the Gasserian ganglion. The pain is usually intense, the paroxysms come on suddenly, last only for a few seconds, but may be repeated at very short intervals. During the attacks the features are rigid and the expression is one of intense agony.

Occipital Neuralgia. This involves the occipitalis major nerve, but occasionally the auricularis magnus and the nerves of the neck are also affected. The pain is distributed over the occipital region of the head, and is usually bilateral. The point of greatest tenderness is over the second cervical vertebra, usually slightly to one side of the spinous process. It may indicate caries of the upper cervical vertebræ.

Neuralgia of the phrenic nerve has been described, but its existence is exceedingly doubtful. The symptoms are said to be pain in the epigastrium, shooting to the shoulder, increasing when pressure is made in the epigastrium, with various respiratory disturbances—sneezing, coughing, etc. It is needless to say that these symptoms, should they occur, would be more likely to suggest an inflammatory process.

Brachial neuralgia is characterized by pain distributed in the arm of the affected side. This may be either persistent or paroxysmal. If the latter, paræsthesiæ in the hand or arm are frequent during the intervals. The points of tenderness are found where the nerves pass over

the bones or just behind the clavicle. Occasionally trophic changes are observed.

Intercostal neuralgia is characterized by pain distributed along the course of the intercostal nerves. There are three characteristic tender points—one next to the spinal column, one in the axillary line, and one over the sternum or rectus abdominalis. There are usually trophic disturbances in the skin over the affected nerve, characterized by reddening or especially by a herpetic eruption (*herpes zoster*).

Mastodynia, or painful breast, is a special form, characterized by pain, usually paroxysmal, and sometimes by the occurrence of painful nodules in the breast.

Lumbar neuralgia is characterized by pain radiating from the lumbar to the gluteal region. Occasionally the anterior surfaces of the thighs are also involved. The sensitive points are found over the lumbar vertebræ, along the edge of the crest of the ilium, and over the linea alba.

Crural neuralgia is characterized by pains radiating from the front of the thigh into the feet. Paræsthesiæ are frequently present during the intervals of the attacks.

Sciatica is characterized by pain in the posterior part of the thigh, often radiating to the feet. The disease usually occurs late in life, is more likely to affect men than women, and is frequently associated with obesity and with various gouty manifestations. It is an exceedingly common form, usually paroxysmal in character, the attacks being preceded by paræsthesiæ. The pain is increased by any movement tending to stretch the nerve, and as a result the patient walks with a peculiar gait, the thigh of the affected side being held fixed and parallel with the body, and the leg flexed on the thigh. This sometimes results in a slight curvature of the spine. According to Minor, the patient exhibits a peculiar method of getting up from the ground. He usually lies upon his back with the diseased limb flexed, gradually thrusts the body up with the hands, then lifts it on the hands and feet, the back being downward, turns half over toward the sound side, thrusts the trunk forward with the arm on the sound side, and gradually rises to an erect posture. The nerve is often sensitive throughout its entire length. The special points of tenderness are found near the posterior spine of the ilium, at the lower edge of the gluteus maximus, just outside the tuber ischii, and in the cavity of the knee-joint. The reflexes are usually slightly exaggerated. There are sometimes slight weakness of the muscles and occasionally fibrillary twitchings.

Other forms of neuralgia are *coccygodynia*, and various neuralgia-like pains in the viscera.

Meralgia paræsthetica is a disease somewhat similar to neuralgia. It is characterized by tingling, burning, or tearing in the area of the distribution of the external cutaneous nerve of the thigh, usually unequally bilateral, and made worse by prolonged exercise, either walking or standing. Frequently there is a tender point just below the anterior superior spine of the ilium. Sensory disturbances in the form of hypæsthesia,

hypalgesia, and diminished electrocutaneous sensibility are common. Stout persons and physicians appear to be particularly susceptible.

Acroparæsthesia is a condition characterized by tingling or pain in the extremities. The affected members are usually tender, and there is hyperæsthesia. Occasionally vasomotor disturbances are present. An allied condition is the symptom known as *tender toes*, which occurs in the course of typhoid fever.

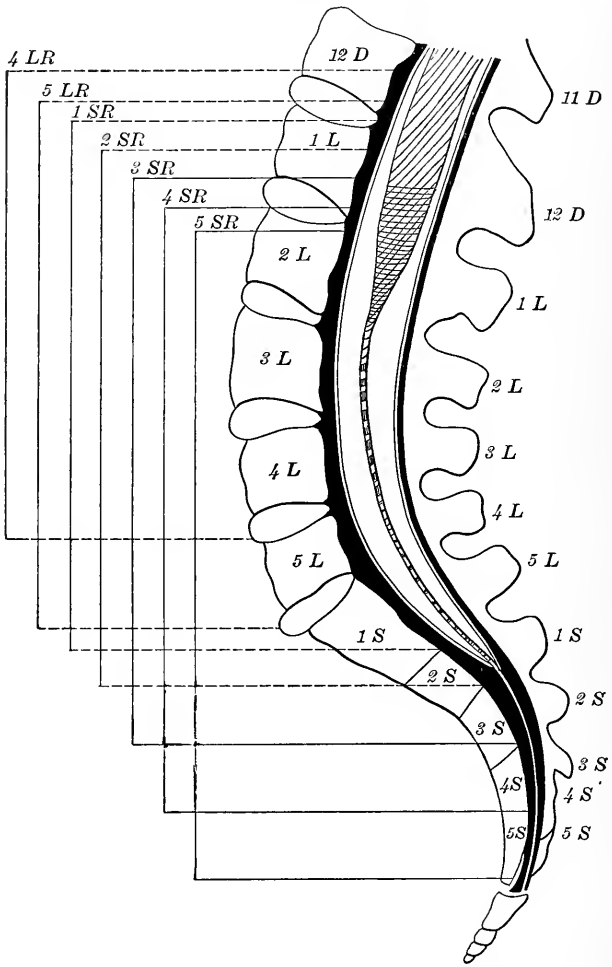
DISEASES OF THE SENSORY NEURON CHARACTERIZED BY DISTURBANCE OF MOTION AND SENSATION, AND BY TROPHIC DISORDERS.

Tabes Dorsalis. (Locomotor Ataxia, Posterior Spinal Sclerosis.)

This is characterized by ataxia, particularly of the lower extremities, lancinating pains in the legs, loss of the knee-jerk, and the presence of the Argyll-Robertson pupil. It usually occurs in the decennium from thirty to forty. A few cases have been reported that occurred before twenty, and some have occurred later in life. It affects men more commonly than women, and is exceedingly rare among negroes and the savage or semicivilized races in general; among the white civilized it is more common in the educated than in the ignorant. A history of syphilitic infection can often be obtained. It is divided into three stages: the pre-atactic, the atactic, and the paralytic. The symptoms of the *preatactic* stage frequently commence with disturbance in the nerves controlling the movements of the eyeball. There may be paresis of the abducens, giving rise to diplopia; of the levator palpebræ, giving rise to ptosis—both of which may be transient and may be discovered only by the history of diplopia or drooping of the eyelid. The reaction to light on the part of the pupil may be sluggish or absent, while the reaction to accommodation still persists (Argyll-Robertson pupil). The symptoms in the nerves of the lower extremities are particularly the lancinating pains that are felt in the posterior portion of the thigh. These are paroxysmal and the patient feels as if he had been stabbed. They are more frequent in damp weather, and are often confused with rheumatism. These *pains* may persist for years as an isolated symptom. The knee-jerk is absent (*Westphal's symptom*), and the patient may note that it is somewhat more difficult to walk in the dark. Often there is distinct weakness in the legs, manifested by fatigue after some moderate exertion and occasionally by giving way of the legs. The station in the early stage is usually only slightly affected. There is a sense of constriction about the body (girdle pain), and sometimes hypæsthesia of the lower extremities that may be associated with a slight hyperalgesia in the zone just above it. The patients may also remark that they have slight difficulty in urination and some diminution of sexual potency. The second stage, or the stage of *ataxia*, is characterized by the symptoms of the preceding stage, all of which are now pronounced. In addition the patient exhibits incoordination of movement, especially in the lower limbs. Station is so impaired that it is usually impossible for him to stand alone with the

eyes closed and the feet together (*Romberg's symptom*). Walking in the dark is difficult and results in frequent falls. In the daylight, with the aid of the eyes, the patient can usually walk quite well, but lifts the feet higher than usual from the ground, and separates them widely. (See

FIG. 390.



Schematic representation of the lower portion of the spinal cord and cauda equina, according to Raymond. The bodies and spines of the vertebræ are designated by the letters *D* (dorsal), *L* (lumbar), *S* (sacral), with the number of the vertebra prefixed. The roots are designated by the same symbols followed by *R*. The upper horizontal line indicates the level at which the root emerges from the cord, the lower horizontal line the level at which it pierces the dura. The vertical line joins the two horizontal lines belonging to the same root.

Ataxic Gait.) The incoordination is manifested by the difficulty with which the patients perform certain movements, such as touching some object with the tip of the finger—as, for example, the nose or ear—or in bringing the heel of one foot against the knee of the other. There is

diminished muscle-tone, so that it is possible to bring the foot to the shoulder without bending the knee; and, of course, absolute loss of the tendon-reflexes, even when reinforced. The nutrition of the muscles remains good, and the electrical reactions are not altered. There are paræsthesiæ, especially in the lower extremities; analgesia in the same situation, or sometimes delay in the conduction of pain. The nerve-trunks lose their sensitiveness to pressure; this is best tested with the ulnar nerve, which can be rolled under the finger against the lower end of the humerus without causing pain (*Biernacki's symptom*). Visceral anæsthesia is also present. The skin-reflexes are often preserved late in the course of the disease. Micturition is sometimes difficult; at other times there is incontinence, but insufficiency of the sphincter ani rarely occurs.

Fig. 391.

Testing the knee-jerk.

Impotence is complete. The Argyll-Robertson pupil is present; there are usually myosis, nyctalopia, and occasionally atrophy of the optic nerve, first manifested by disturbance of the color-perception (red and green blindness). In the latter condition it has been noted that when blindness has fully developed, the ataxia becomes less pronounced or may disappear completely (*amaurotic tabes*). The visceral crises are characterized by attacks of intense, cramp-like pain, involving usually the stomach, but sometimes affecting the larynx, heart, kidney, bladder, rectum, or other viscera. The laryngeal crises are often accompanied by distressing cough and dyspnœa that may simulate the symptoms of aneurism of the arch of the aorta. Trophic changes occur, of which the most common are the arthropathies. These involve particularly the knee, hip, and shoulder-joints. In addition, the patient may have painless falling out of the teeth or rapid softening of them. The bones often show a marked tendency to fracture, and an equally remarkable tendency to rapid knitting. Occasionally an osteo-arthropathy of the foot gives rise to the so-called tabetic foot, characterized by great thickening of the bones and a real or apparent shortening. In certain cases a chronic ulcer

develops on the sole of the foot, which usually progresses until it has produced perforation (*mal perforant*). In the *paralytic* stage of ataxia the loss of muscle-tone has reached so extreme a degree that locomotion is impossible. The patients by this time have usually developed cystitis, and death occurs as a result either of exhaustion or of general septicæmia.

The Cervical Type of Tabes Dorsalis.

This is characterized by the development of the symptoms chiefly in the arms. The lightning pains occur in the upper extremities, there is loss of the bicipital and tricipital reflexes, and the girdle sensation is usually felt in the upper part of the thorax. The ocular symptoms are the same as in the usual form. The visceral crises are likely to affect the larynx. In this form ataxia in the legs, Romberg's symptom, and the absence of the knee-jerk may not be present until late in the disease, but there are usually disturbance of micturition and loss of sexual power.

Friedreich's Ataxia.

This is a disease of early life, distinctly hereditary in character, ordinarily affecting several members of the same family. It first appears between the ages of seven and twelve years. It is characterized by incoordination, loss of knee-jerk, weakness, irregular speech, and slight deformities. The first symptom is incoördination of the lower limbs. This gradually becomes more severe, the muscles grow weaker, the flexors more so than the extensors, often giving rise in time to pes equinovarus with hyperextension of the great toe. The muscles of the back also grow weaker, giving rise to scoliosis; the knee-jerks are absent, the pupillary reflexes remain normal, and intelligence is unaffected. The speech is peculiar, some of the syllables being pronounced readily and others slowly, with a drawl. The gait becomes markedly ataxic, the patients keeping the legs widely separated. In time the paresis and incoördination become so severe that walking is impossible. There are frequently irregular choreiform movements in the muscles and the so-called static ataxia—that is, inability to hold the limb in one position for more than a moment. There is often a nystagmus similar to that observed in multiple sclerosis. Sensation remains normal throughout. The course is progressive.

The *cerebellar* type of hereditary ataxia differs from the foregoing by the fact that the knee-jerks are exaggerated, and there is occasionally absence of the pupillary reflex to light.

DISEASES OF THE PERIPHERAL MOTOR AND SENSORY NEURONS.

These are all characterized by disturbances of motion and sensation, usually associated with more or less severe trophic changes.

Acute Neuritis.

Acute inflammation of the nerves is characterized by pain localized in the course and distribution of the nerve affected, paræsthesia, tenderness, and perhaps paresis or paralysis of certain groups of muscles. The pain is made more severe if the limb is held in such a position that the nerve is stretched. The nerve may be distinctly enlarged to touch. As it is a true inflammatory condition, there are usually constitutional disturbances, such as fever, malaise, etc. Often the disease is progressive, extending from the peripheral to the more central nerve-trunks. This is spoken of as ascending neuritis. Along the course of the nerve there are often vasomotor and secretory disturbances, such as pallor, redness, œdema, or the lesions may be more severe, such as atrophy of the skin, with glossiness of the skin, or trophic changes in the hair and nails, or even a herpetic eruption. The symptoms vary according as the nerve affected is chiefly motor or chiefly sensory. *Inflammation of a motor nerve* causes flaccid paralysis in the group of muscles supplied by the nerve, and if the inflammatory process persists, these muscles rapidly atrophy and give the reactions of degeneration. The reflexes in the parts controlled by these muscles are lost. The diagnosis can usually be made by a careful study of the distribution of the paralysis. The paralysis may be permanent, or recovery may ensue if the process has not proceeded too far. *Inflammation of a sensory nerve,* in addition to pain, is usually characterized in the early stages by hyperæsthesia of the skin in the distribution

FIG. 392.

Alcoholic polyneuritis. Foot-drop and wrist-drop.

of the nerve, which later becomes anæsthetic, and is often associated with hyperalgesia (*anæsthesia dolorosa*). Vasomotor and trophic changes are common. The course is usually favorable—that is, in time, either as a result of reparative processes in the nerve or by compensatory action of the adjacent nerves, the sensory disturbances disappear.

Multiple neuritis is characterized by the appearance of the symptoms of the disease in a number of nerves at the same time. The nerves of the limbs are far more frequently affected than those of the trunk. The symptoms are modified by the cause. In alcoholic polyneuritis there are

usually slight paræsthesiæ of the limbs, with marked paresis of the muscles, particularly the extensors, giving rise to foot-drop and wrist-drop. (See Fig. 392.) The disease usually affects all four extremities. In lead-poisoning the disease is sometimes unilateral, is usually restricted to the arms, and the sensory disturbances are very slight or absent. There is paralysis of the extensor muscles of the arm, which, in severe cases, goes on to muscular degeneration. Neuritis may also be produced by arsenic. Diphtheritic polyneuritis is usually characterized by paralysis of the muscles of the palate, but occasionally the muscles of the limbs are also involved. In certain of the chronic forms of polyneuritis, instead of or with the loss of power, there is marked loss of coördination. This is spoken of as the ataxic variety.

Beri-beri, or **kakke,** is an infectious disease of the Pacific Islands and the western coast of Asia, characterized by the symptoms of a peripheral multiple and symmetrical neuritis. It is considered in the chapter devoted to Infectious Diseases.

Multiple neuromata sometimes occur very extensively upon the nerves of the skin, at times producing symptoms of multiple pressure upon the nerves—that is, paræsthesiæ, paralyses, or loss of sensation. At other times they produce no symptoms whatever, and can be recognized only by inspection.

DISEASES OF THE SPINAL CORD INVOLVING THE CENTRAL MOTOR NEURONS.

Primary Spastic Paraplegia.

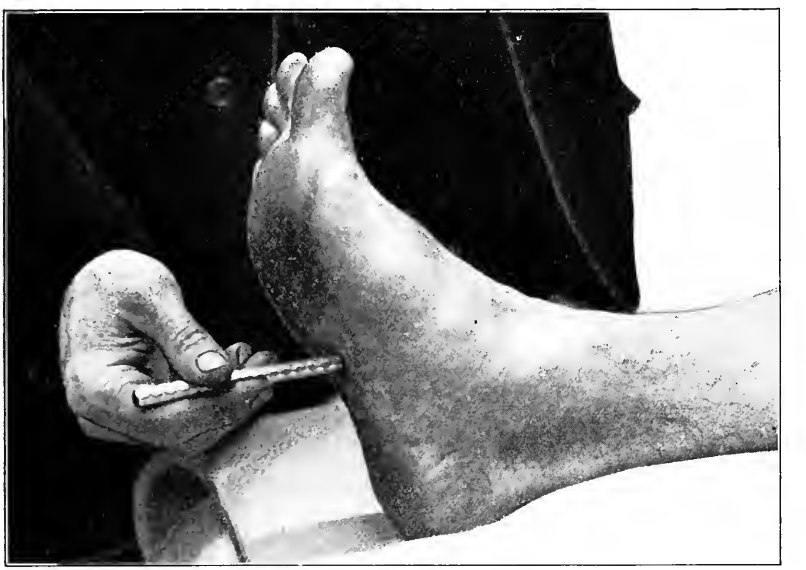
Primary spastic paraplegia is characterized by weakness of the legs without muscular degeneration and with increased reflexes. The disease was formerly supposed to be the result of the involvement of the lateral columns of the cord. The first symptom is weakness or a feeling of heaviness in the legs; then spontaneous cramps appear. The reflexes are greatly exaggerated, and the muscle-tone is so increased, particularly in the extensor muscles of the thigh and the muscles of the calf, that the patient walks with the leg partially extended, dragging the toe along the ground; the arms are rarely involved. The electrical reactions of the muscles are normal. The sphincters are rarely involved, and sensation usually is unimpaired. If cramps are frequent, however, the muscles may be sore. In children the adductors become stronger than the abductors, and a peculiar, cross-legged gait is thereby produced. It may occur in several members of the same family (*familiar form*), the symptoms usually appearing in early adult life. It may develop in early childhood, often after premature or difficult birth (*Little's disease*). In this form the gait is characteristic, the feet being lifted high from the ground to prevent dragging of the toes (stepper or chicken gait).

Amyotrophic Lateral Sclerosis.

The disease usually occurs in adults about middle life. In very rare cases it occurs in children. It is characterized by a spastic paraplegia,

with exaggeration of the reflexes and degeneration of the muscles. Spasms are common, and may give rise to muscular pains. The symptoms consist of weakness in the legs, which at the same time become stiff; the muscles rapidly atrophy; there are fibrillary twitchings and reactions of degeneration. The arms are usually involved first, the degeneration commencing in the muscles of the hands and giving rise ultimately to the production of various deformities, such as the claw-hand. The tendon-reflexes are greatly exaggerated; there are patellar and ankle clonus. The Babinski phenomenon is present. The muscles are greatly weakened, but remain rigid until late in the course of the disease. The sphincters are rarely involved, the pupillary reflexes are normal, and there are no sensory disturbances. Bulbar symptoms—that is, paralysis of the larynx, pharynx, and palate—occur, giving rise to dysphagia,

FIG. 393.



Testing the Babinski reflex.

dysarthria, and occasionally to entire loss of speech. In the early stages of this complication there may be distinct jaw clonus, elicited by tapping upon the lower jaw or the chin. When the bulbar symptoms become advanced, the lower part of the face is absolutely expressionless. It has a mask-like appearance, and often saliva dribbles from the mouth. In this stage inspiration pneumonia, due to involvement of the laryngeal branches of the vagus, is quite common and usually causes death.

Multiple Sclerosis.

This is a condition that involves the spinal cord and brain. The situation of the lesions is not constant, and therefore the symptomatology is variable. It usually commences at about the age of thirty years, and

is more common in men than in women. The characteristic symptoms are intention tremor, nystagmus, and scanning speech. The *intention tremor* involves chiefly the limbs, and causes disturbance of writing, walking, etc. It is most easily elicited by having the patient grasp some small object. There may also be rhythmical oscillations of the head. Ataxia, apart from the intention tremor, has also been described. There is usually persistent lateral nystagmus. The tendon-reflexes are greatly exaggerated, and there are ankle-clonus and a spastic gait. The pupillary reactions are normal. Occasionally diplopia occurs as a result of abducens paralysis; it may be transient. In a large proportion of the cases there is more or less complete atrophy of the optic nerve. The speech is slow, drawling, and often tremulous. The voice lacks expression, and, on account of a slight nasal tendency, is usually disagreeable. Not infrequently there may be stuttering. Sensory changes consist of paræsthesiæ, transient hyperæsthesiæ and anæsthesiæ, and occasionally disturbances of the function of the bladder. Less frequent symptoms are vertigo, occurring in paroxysmal attacks, diminution of intelligence, alternate states of depression and exaltation, and a tendency to laugh or weep without provocation. Apoplectiform and epileptiform attacks may also occur. The disease is usually chronic, but from time to time there are exacerbations. It appears to be frequently associated with hysterical manifestations. In some cases bulbar symptoms appear early and rapidly lead to death.

Hypertrophic Cervical Pachymeningitis.

This is characterized by pain in both arms, followed by muscular degeneration commencing in the hands. Later, there may be spastic paraplegia of the legs, with anæsthesia of the body below the affected segment. Occasionally this disease, which is usually due to tuberculous meningitis, may occur in other portions of the spinal cord, giving rise to various symptoms.

Acute Spinal Meningitis.

Acute spinal meningitis is characterized by intense pain in the back, radiating into the legs; rigidity of the spinal column, with opisthotonos; intense hyperæsthesia of the skin of the body, and, if the disease lasts long enough, paralyses. Kernig's symptom—that is, the inability to extend the flexed leg as a result of flexor cramp—is present. The *tâche spinale* occurs also in other conditions. It is always due to an infection, and therefore fever, chills, and prostration are present. The diagnosis is greatly assisted by examination of the fluid withdrawn by spinal puncture (*q. v.*).

Syphilitic Spinal Meningitis.

Syphilitic spinal meningitis produces a great variety of symptoms. There are, however, pains due to pressure upon the posterior roots, girdle pains of the body, and occasionally paralysis of the muscles of the extremities, with atrophy and degeneration. Often, also, the spinal cord is in-

volved, giving rise to the symptoms of pressure or transverse myelitis (*q. v.*) or Brown-Séquard's syndrome (*q. v.*). The sensory symptoms, aside from the pains, consist of hyperæsthesia, hypæsthesia, or anæsthesia. The tendon-reflexes of the lower extremities may be lost and reappear, and by some this is supposed to be pathognomonic of the disease.

DISEASES CHARACTERIZED BY THE SYNDROME OF TRANSVERSE INTERRUPTION OF THE SPINAL CORD.

Pott's Disease.

Pott's disease (*caries of the vertebræ*) in the majority of cases commences in childhood. Occasionally the first symptoms may be noticed in adult life. It is characterized by an angular deformity of the spine, spastic paraplegia, and various disturbances of sensation in the body below the level of the lesion. In the earlier stage the only symptom may be pain in the back, usually radiating around toward the ventral surface. There may be no deformity, but sudden pressure upon the head, jarring of the spine by coming down heavily upon the heels, and pressure over the tender point in the back may elicit sharp pains. In this stage there are usually slight exaggeration of the reflexes and perhaps a slight weakness of the legs. Later, the angular deformity becomes apparent, usually in the form of a sharp projection in the dorsal portion of the spinal column, but it may appear also in the cervical and lumbar region. The weakness of the lower extremities becomes more pronounced, and may give rise to an actual paraplegia. The pains are usually severe, radiate around the trunk, and sometimes affect other portions of the body. Sensation may be slightly impaired. There may be distinct dissociation below the lesion —that is, loss of temperature- and pain-senses, with preservation of tactile-sense—or there may be total anæsthesia. The bladder is usually involved, there is difficulty in micturition, and in the later stages, incontinence of urine or overflow. As in myelitis, bed-sores or other trophic changes of the skin are very likely to develop, and patients suffer severely in general nutrition. In the earlier stages, and more particularly in the stage of recovery, after the deformity has become stationary, ataxia may exist. The reflexes are sometimes greatly exaggerated, and there are often ankle-clonus and Babinski's phenomenon. When the paraplegia has become complete, all the reflexes are usually abolished. Girdle sensation is also very common. The tuberculin reaction, if positive, is not of much value in this disease, as other tuberculous foci may be present, although not suspected, and cause a rise of temperature after the injection. It is especially untrustworthy in adults. The course is very variable. At times the destruction of the body of the vertebra is rapid, and the symptoms develop acutely. At others, the condition occurs very slowly, and the symptoms, even after years' duration, may be exceedingly slight. Often after progressing to a certain extent the spinal deformity becomes stationary, and the nervous symptoms may gradually disappear. Caries of the upper cervical vertebræ produces pains that involve the neck and the occipital region of the head. The position of the head is peculiar: it is drawn slightly forward

and carried very rigidly, and the chin is elevated. These patients may sometimes die suddenly as a result of pressure by the odontoid process on the medulla.

Tumors of the Cord.

The symptoms of this condition are extremely variable, and depend upon the location, nature, and extent of the growth. Occasionally deformities occur as a result of pressure upon the arches of the vertebræ. Paraplegia usually develops, sometimes very suddenly, sometimes gradually. There are usually exaggeration of the reflexes and ankle-clonus; but these in time may disappear, or may never occur if the tumor is situated in the lumbar region. When the posterior roots are pressed upon, there are root-pains and the girdle sensation. Sensory disturbances are more or less complete according to the degree of destruction that has occurred in the spinal cord. At first there is ordinarily only hypæsthesia; later there may be dissociation of sensation, and finally, when the transverse lesion has become complete, total anæsthesia. After complete destruction of the spinal cord at any point trophic changes occur.

The diagnosis of the position of a tumor is more important than the recognition of its nature. The position may be at any level of the cord, and in the vertebræ, the meninges, or the substance of the cord. The segment or segments involved may be recognized by the determination of the upper level of the sensory and motor changes. If the anterior cornua or roots are destroyed, the muscles supplied by them will give the reactions of degeneration. If the posterior roots are irritated, neuralgic pains may be felt along the course of the nerves arising from them. If the tumor has produced a total transverse lesion, the symptoms at the different levels will correspond to those given in the table on pages 335 and 336. The situation of the tumor with reference to the spinal cord or the walls of the canal is more difficult to determine, and errors are not uncommon. Deformity of the spinal column is a valuable sign of vertebral tumor. Tenderness is of more doubtful significance, but is certainly more frequent when the bones are involved than when they are not. Root symptoms, preceding slowly developing compression, indicate a dural tumor, the predominance of motor or sensory changes, or the existence of a more or less well-developed Brown-Séquard syndrome (see page 337), suggesting that the tumor is on the anterior, posterior, or lateral aspect of the cord. Intramedullary tumors may give rise to symptoms resembling those of syringomyelia—that is, dissociation of sensation and the symptoms of transverse lesion. Naturally, the size and location of the tumor have considerable influence upon the early symptoms. If the tumor enlarges sufficiently to produce pressure, all the conduction-paths are interrupted. The nature of the tumor may sometimes be suspected by the course; if very slow, it is probably benign. Primary malignant tumors in other parts of the body strongly suggest metastatic growths.

Chronic Internal Meningitis.

This is usually characterized by pain that radiates into various portions of the body, particularly the limbs, and by more or less hyperæsthesia.

The motor symptoms consist of tremors, spasms, and occasionally, when the anterior roots are involved, paralyses, with muscular degeneration. In the milder forms the only motor symptoms may be incoördination of movement. Herpetic eruptions along the course of the nerves arising from the involved posterior roots are quite common. There is usually more or less rigidity of the spinal column, with pain on movement. The disease is usually syphilitic, and is often associated with tract degeneration in the spinal cord. Usually the posterior columns and the lateral columns are involved, and there may be the syndrome of combined sclerosis.

Acute Myelitis.

There are a number of varieties of this condition, the most common and typical being transverse myelitis. It is an acute inflammatory disease associated with constitutional disturbance—that is, chills, fever, and malaise—and is occasionally ushered in with a convulsion. The symptoms are those of transverse lesion of the spinal cord. Ordinarily the dorsal part is affected ; and there are therefore in the earlier stages weakness and paræsthesia of the legs, and perhaps a girdle sensation and hyperæsthesia over the spine, in the zone supplied by the involved segment. Not infrequently painless spasms occur in the legs. In the course of a few days or hours the weakness of the legs increases until there is complete paraplegia. The tone of the muscles is enormously exaggerated, the knee-jerks are increased, and there are patellar and ankle-clonus, Babinski's sign, and often Sinkler's toe-jerk. The limbs are usually spastic and kept in a position of extension. From time to time the muscles give violent twitches. At first there is hypæsthesia, soon passing into complete anæsthesia up to the horizontal line surrounding the trunk, at which point there is girdle sensation, and above it there is a zone of hyperæsthesia. The muscles supplied by the affected segment atrophy and give reactions of degeneration. Those in the region below maintain their nutrition for a considerable time. There are difficulty in micturition, usually paralysis of the bladder, and finally overflow from retention. The urine becomes alkaline, cystitis develops very rapidly, and is often followed by extensive sloughing of the surrounding parts. Bed-sores occur early and extend deeply. Trophic lesions also occur in the legs, the skin becomes thin and glazed, and the toe-nails are brittle. Even arthropathies have occasionally been observed. After the acute stage has passed more or less improvement may occur, characterized by gradual return of power in the legs and partial recovery of sensation. The localization depends upon the same principles as in any other transverse lesion of the cord.

Acute Focal Myelitis.

This gives rise to only part of the symptoms described above, depending upon the tracts involved by the process and the various nuclei that have been destroyed. There is therefore usually a monoplegia, associated with exaggeration of the reflexes and irregular areas of anæsthesia, or,

if the focus be in the arm- or the leg-centre, diminution or loss of the reflexes and degeneration of the muscles.

Disseminated Myelitis.

Disseminated myelitis gives rise to a complicated group of symptoms, according to the number, situation, and extent of the lesions. It resembles perhaps most closely transverse myelitis (*q. v.*).

Chronic Myelitis.

Chronic myelitis is distinguished from the acute form by the more gradual development of the symptoms. The patient first notices weakness of the legs, perhaps characterized from time to time by complete transient loss of power (*giving way of the legs*). If the reflexes are examined at this time, they will be found slightly exaggerated; later they become very markedly increased, and ankle-clonus develops. The patient also complains, in the early stages, of paræsthesiæ in the limbs that may involve the arms as well as the legs, and sometimes the trunk. A girdle sensation is also frequently present. The sensory disturbances, however, are much less prominent than in the acute form. Total anæsthesia rarely occurs; usually there is only hypæsthesia, but as the different forms of sensation are unequally involved, there may be partial dissociation of sensation or anæsthesia dolorosa. Finally, muscular atrophies, and even severe trophic disturbances, occur, the picture ultimately resembling that of the late stage of acute myelitis.

Landry's Paralysis.

This is characterized by progressive paralysis of the legs, arms, and muscles of the throat, leading ultimately to death. The prodromes consist of slight fever, malaise, and paræsthesia in the legs. These are followed by weakness of the legs, which may involve both, or at first only one. This gradually ascends, and at the same time the paræsthesia becomes more distressing. There are, however, few or no objective sensory disturbances excepting occasionally a slight hypæsthesia. The reflexes are lost, the muscles are without tone, and the paralysis is therefore flaccid. Electrical changes do not occur, or only in very chronic cases. The paralysis gradually ascends, involving the muscles of the abdomen, the thorax, and the arms. When the thorax is involved, the patient usually has rapid respiration and complains of dyspnœa. Later there are symptoms of bulbar involvement, difficulty in deglutition, and interference with speech. The diaphragm becomes paralyzed, and the patient dies as a result of exhaustion. The intelligence remains normal throughout the disease; there is never loss of consciousness, and there is no disturbance of the function of the bladder or rectum.

Hemorrhage into the Cord. (Spinal Apoplexy.)

This is characterized by the sudden interruption of the function of the cord at a certain level. There is usually, at the time the hemor-

rhage occurs, severe pain, then rapidly developing paralysis of the legs, which may be flaccid if the lumbar region is involved, or spastic if the lesion is higher up. Hæmatomyelia into the cervical region may cause paralysis of the arms, but death usually occurs suddenly. The sensory disturbances are irregular in character. At times there is dissociation of sensation, more frequently complete anæsthesia up to the level of the hemorrhage. The patient has no fever, consciousness is not disturbed, but there is interference with the functions of the bladder and rectum. Occasionally the hemorrhage involves particularly one side of the cord or only one-half of the gray matter, producing the syndrome of Brown-Séquard (*q. v.*). The diagnosis can frequently be made from the subsequent course of the case. If death does not occur, rapid improvement is usually the rule. The sphincters regain their functions, power returns to the limbs, and ultimately the patient may recover completely. In some cases, however, recovery, although pronounced, is only partial.

Syringomyelia. (Cavity in the Spinal Cord.)

Although by some this is considered a congenital disease, the first obvious symptoms rarely occur before early adult life. These are of the segmental type, and in many of the cases the areas of sensory disturbances extend in the form of ribbons along the arms. Sometimes, however, they differ from this, and approximate more closely the cerebral type of anæsthesia. The anæsthetic areas on the trunk are always bounded by a horizontal line. Syringomyelia is characterized by a group of symptoms whose occurrence together is almost pathognomonic. First, dissociation of sensation: pain and temperature senses are lost, tactile and muscular senses are retained. Second, degenerative atrophy of the muscles, associated with fibrillary twitchings and alteration of the electrical reactions. Third, trophic lesions which may involve the skin, particularly that of the fingers or the joints. The size and position of the cavity and the extent of the destruction of the substance of the cord by the gliomatosis vary greatly, and as a result the symptoms, which are due entirely to the interference with the structures of the spinal cord, differ considerably in different cases. This difference, however, is quantitative rather than qualitative, and the three groups of symptoms are practically always present. The disease appears to develop with extreme slowness. The earliest symptom may be the occurrence of painless whitlows—that is, inflammation around the fingernail, with perhaps the ultimate destruction of the nail itself. These may recur in one finger after another for several years and without the presence of any other symptoms excepting perhaps a slight disturbance of sensation in the fingers. Later, muscular atrophies appear. These involve particularly the muscles of the shoulder or the hand. In the latter situation they may give rise to the appearance that occurs in progressive spinal muscular atrophy. At the same time the sensory disturbances become more pronounced, gradually ascending the arm and perhaps involving the trunk. The upper border forms a horizontal line about the body—that is, the alterations are segmental in type. The trophic changes may then

assume a more severe form, giving rise to deep, painless ulcerations in the fingers, and perhaps loss of the terminal phalanges. For a long time the symptoms may remain almost exclusively unilateral, and it is rare for the two sides to be affected equally. The motor symptoms, aside from the weakness resulting from the muscular atrophy, consist of weakness of the legs with exaggeration of the reflexes—that is, spastic paraparesis. At times the lower portion of the cord is particularly affected, and then sensory and trophic changes are found in the legs. Station may be slightly altered in the later stages of the disease, but this is by no means a characteristic symptom. Ultimately the patient develops scoliosis, trophic changes affect parts other than the hands, giving rise to arthropathies or to a form of dry arthritis with absorption of the bone. There may be vasomotor disturbances, and in some cases inequality of the pupils. The intellect is undisturbed. The patients ordinarily die as a result of exhaustion or pulmonary involvement, but occasionally in the later stages of the disease bulbar symptoms occur.

Morvan's Disease.

This is characterized by the appearance of painless whitlows in the fingers, sometimes associated with deep ulcerations of the soft parts. There are usually sensory disturbances similar to those found in syringomyelia, with the addition of tactile anæsthesia, but muscular atrophy rarely exists. The disease is exceedingly chronic. It is possibly only a variety of syringomyelia.

Traumatism of the Spinal Cord.

This may either produce destruction, partial or complete, of the tissue of the cord itself, giving rise to the syndrome of transverse interruption, or else give rise to a group of indefinite motor, sensory, and mental disturbances that have been grouped under the term traumatic neuroses. (See Hysteria.) The symptoms which result from an organic lesion may come on gradually or immediately. They are similar to those produced by pressure upon the cord or by hemorrhage into it.

DISEASES OF THE BRAIN CHARACTERIZED BY GENERAL SYMPTOMS AND SENSORY AND MOTOR DISTURBANCES.

DISEASES CHARACTERIZED BY MENTAL, MOTOR, SENSORY, AND SOMETIMES TROPHIC DISORDERS.

External or Pachymeningitis.

External or pachymeningitis is a rare condition, usually secondary to traumatism or abscess, characterized by fever, headache, often sharply localized, and convulsions. Frequently the symptoms are masked by the existence of some graver condition, such as status epilepticus, coma, or uræmia. Occasionally no symptoms are produced. If there is much thickening of the membrane, evidence of focal disease in the form of

paralysis or convulsions of the Jacksonian type may be present. *Hæmatoma* of the dura mater is a condition usually occurring in cases of chronic disease. There may be slight fever and headache without other symptoms. In some cases, however, the onset is sudden and apoplectiform in type. The patients develop hemiplegia, unconsciousness, and occasionally unilateral convulsions.

Internal or Leptomeningitis.

There are many varieties of this; thus, according to the location, there may be meningitis of the convexity, of the base, or of the brain and cord; according to the cause there may be suppurative, epidemic, or tuberculous meningitis; other varieties not so easily classified are serous meningitis and meningismus.

Suppurative meningitis. The symptoms vary according to the nature of the process, its localization, and extent. The patient may complain of malaise and headache for a few days preceding an attack; then there is often a chill, followed by fever, convulsions, and delirium. The headache becomes more and more intense, and frequently there is vomiting, sometimes with, sometimes without, nausea. The headache is often localized to the frontal or occipital regions; occasionally, however, it is more general. From time to time there are acute exacerbations, causing the patient, especially if a child, to cry out—the meningitic cry. The skin is hyperæsthetic; all the sensory nerves have their functions increased; there are photophobia and inability to tolerate noises. Frequently there is paresis of the vasomotors of the skin, so that localized cutaneous irritation, such as may be produced by drawing the end of a blunt object across the surface, gives rise to a persistent red mark (*tache cérébrale*). Herpes labialis is very common; less so is a disseminated erythematous eruption or irregular bluish patches; the latter occur most frequently when there is violent septic infection. The patient usually lies with the head drawn far back and the muscles of the neck tense and rigid. This, however, occurs only when the cervical portion of the spinal cord is also involved. It is an exceedingly important and an almost pathognomonic symptom. Any attempt to straighten the head causes intense pain. Examination of the eye-grounds usually shows intense congestion and more or less perineuritis. Sometimes there is very distinct choked disk. The pupils are often unequal, and strabismus and even nystagmus frequently occur. Paralysis of any of the cranial nerves may occur, but is not so common as in tuberculous meningitis. The oculomotor or some of its branches is most frequently involved, although slight paresis of the facial nerve is not uncommon, and occasionally there is distinct monoplegia or hemiplegia. The tendon-reflexes are usually somewhat exaggerated, the muscular tone is increased, and Kernig's sign is usually present, although it can not be regarded as pathognomonic. Fever, headache, and delirium usually persist throughout the course of the disease; the fever is often very high. In the early stages the pulse is usually relatively slow; later it may become rapid. The blood-pressure is always increased. By means of Quincke's

lumbar puncture it is sometimes possible to make a bacteriological diagnosis from the fluid withdrawn. Meningitis due to pyogenic micro-organisms, such as the pneumococcus, staphylococcus, etc., may be suspected: when the fever is high; when there is marked retraction of the head, indicating spinal involvement; when there is general leucocytosis; when there is an excess of albumin (more than 1 per cent.) and of polymorphonuclear cells in the spinal fluid; and especially when there is or recently has been some other focus of pyogenic infection in the body, such as pneumonia, sepsis, or middle-ear disease. The course is progressive and almost invariably terminates in death. But remissions may occur or there may be considerable temporary improvement after spinal puncture.

Epidemic cerebrospinal meningitis may exactly simulate the symptoms of purulent meningitis. In some cases, however, the course is more prolonged, and even when the termination is fatal, there is apt to be a remission of longer or shorter duration. The diagnosis can be made only by the discovery of the meningococcus in the spinal fluid or the nasal secretion. If an epidemic exists, it may be readily suspected, but it must be remembered that other forms of meningitis are not less frequent.

Tuberculous meningitis is the most common form. It affects chiefly the base of the brain. There is usually recognizable tuberculosis of some other part of the body, and often a history of injury to the head. The course is slow, although in rare instances it may be very rapid; the prodromal stage is prolonged and the symptoms are slight, consisting of headache, insomnia, malaise, loss of appetite, progressive emaciation, and occasionally distinct psychical disturbance, such as extreme irritability, light coma, a confusional state, or loss of interest in the surroundings. As the disease progresses, more distinct lesions occur, the cranial nerves are involved, and there may be ptosis, diplopia, or paralysis of the pupil. Paresis or even paralysis of the facial nerve is common, and lesions of the other nerves may develop. Occasionally monoplegia or hemiplegia occurs. The fever is moderate, the course deliberate, and extreme intracerebral pressure develops rather late, so that the blood-pressure may not be greatly elevated. The fluid obtained by spinal puncture is usually clear, but tubercle bacilli may sometimes be obtained by centrifugation. It is said to contain an excess of mononuclear cells, but it is dangerous to base a diagnosis upon this sign alone. There is moderate leucocytosis (10,000 to 15,000), Kernig's sign is more frequently absent than present, the knee-jerks are usually increased, sometimes unequally, and ankle-clonus may occur. Spinal symptoms, such as rigidity of the spine and retraction of the head, are comparatively rare. Aside from the discovery of tubercle bacilli in the spinal fluid the only pathognomonic sign is the presence of tubercles upon the choroid. In very rare instances a secondary infection with some other form of micro-organism occurs in tuberculous meningitis. This appears to render the course shorter and more violent.

Serous meningitis is characterized by a marked increase of the intracerebral pressure, and the symptoms that result from it—headache, coma, or convulsions. There are usually slight fever, moderate leucocytosis,

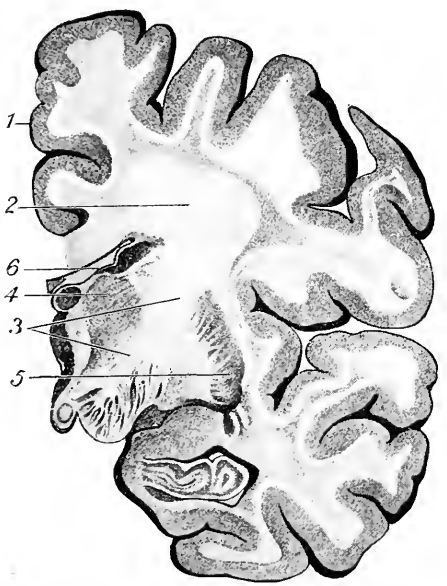
increase in the reflexes, and slight rigidity of the back of the neck. The face is flushed, but eruptions are rare. The differential diagnosis from suppurative meningitis can be certainly made only by an examination of the spinal fluid. This contains more albumin and cells, chiefly ependymal, than normal, but is sterile. This condition usually occurs in the course of some infectious process.

Meningismus is a term invented by French clinicians to describe a condition that may occur in the course of severe infections; it is characterized by symptoms practically identical with those of serous meningitis, but more transient. Ordinarily it is entirely relieved by spinal puncture. As Kernig's sign may occur in acute infections, it is of little value in the diagnosis of this condition.

Cerebral Hemorrhage. (Apoplexy.)

This usually occurs in advanced life in patients who have pronounced arteriosclerosis. Occasionally, however, it occurs in young adults and in children. It is characterized by a great variety of symptoms, depending largely upon the location of the lesion. They may be divided into those

FIG. 394.

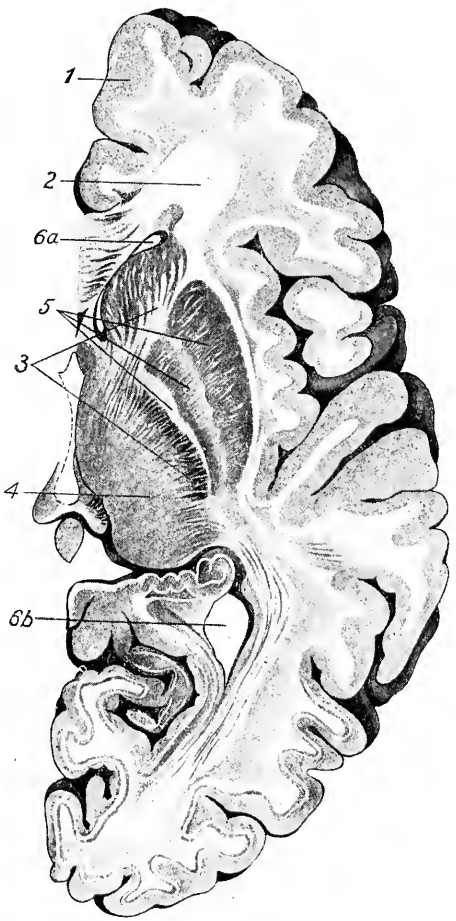


Vertical transverse section through the right hemisphere: 1, cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6, part of the lateral sinus. (After DÉJÉRINE.)

of the attack and those of the post-apoplectic stage. The symptoms of the attack consist of prodromata—that is, headache, tendency to vertigo, a sense of fulness in the head, roaring in the ears, and perhaps some thickness of speech. These may pass off without further disturbance or may lead directly to an attack. The latter is usually characterized by

the sudden occurrence of complete unconsciousness. The patient falls to the ground, and there is at first a temporary pallor. This is succeeded by flushing of the face, which may become almost purple. The pulse is full, bounding, and compressed with difficulty. The blood-pressure is greatly increased. The breathing is stertorous, slow, and occasionally of the Cheyne-Stokes type; the pupils are usually contracted and often

FIG. 395.



Horizontal section through the right hemisphere; 1, cortex; 2, white substance; 3, internal capsule; 4, optic thalamus; 5, lenticular nucleus; 6*a*, anterior horn, and 6*b*, posterior horn of the lateral ventricle. (After DÉJÉRINE.)

unequal. Often there is vomiting or involuntary micturition or defecation. The limbs remain completely paralyzed, or in some cases there are unilateral convulsions. If, as is commonly the case, the hemorrhage has involved the motor tract, there is complete flaccid paralysis of one side, with lost reflexes. If death does not occur in the course of the first twenty-four hours, the patient usually begins to show signs of

consciousness, and may be aroused from his comatose condition by sharp questioning. He then may pass into a still more deeply comatose condition, with rise of temperature, followed by death, or there may be no further indications of hemorrhage, and recovery may set in. As a rule, in those cases in which the prognosis is favorable no rise of temperature occurs. It may now be found that the patient has hemianopsia, usually the visual fields on the same side of the lesion being blinded. Conjugate deviation may or may not have existed from the first, the patient ordinarily looking toward the sound side. If the speech-centre has been involved, there is absolute aphasia; but even when it is not directly affected, partial aphasia is very common. The hemiplegic limbs remain paralyzed; the others regain their power. It is now necessary to determine the extent of the damage and to locate as nearly as possible the position of the lesion. Complete hemiplegia may involve the lower branch of the facial, the arm, and the leg. The upper branch of the facial and the muscles of the trunk commonly escape, although the former may show slight paresis. Often there is considerable inequality in the paralysis of the different parts. The arm may be more involved than the leg, or the reverse, or the face may escape almost entirely. Sensory disturbances may or may not be present. There is sometimes loss of all forms of sensation and sometimes disturbance of only the tactile or the muscular sense. Occasionally, when tactile sense is preserved, there may be loss of the stereognostic sense. Complete hemiplegia with disturbance of sensation almost invariably indicates destruction of the internal capsule upon the opposite side. Motor disturbances in the form of clonic convulsions may also occur in the paralyzed limbs, and occasionally, probably as the result of a double lesion, in the limbs of the sound side. They are commonly the result of cortical lesion, apparently irritative in character—either infarction or else some growth pressing upon and involving the cortex. As the case progresses there are usually more or less return of motor power and almost complete return of sensation. This may, however, be exceedingly gradual, several weeks elapsing before the sensory disturbances have entirely disappeared. The muscles that remain permanently paralyzed gradually atrophy, but nearly always give normal qualitative electrical reactions until the muscular substance disappears, leaving contracted fibrous tissue. The muscles themselves may contract early, the flexors ordinarily overcoming the extensors. Repeated attacks of apoplexy are by no means uncommon, and the double lesions thus produced may give rise to very complex symptom-groups. Hemorrhage into the pons or medulla gives rise to various forms of crossed paralysis. (See also Cerebral Localization and Aphasia.)

Cerebral Embolism and Thrombosis. (Softening of the Brain; Encephalomalacia.)

This condition may occur at any period of life, and is nearly always associated with some valvular disease of the heart or an extreme cachexia or anæmia predisposing to thrombosis. It is characterized by symptoms very similar to those of cerebral hemorrhage. Prodromal symptoms, in

the form of headache, vertigo, weakness, and malaise, are often present. At times there may be also slight impairment of speech, or the patient may be dull and apathetic. The attack usually comes on more gradually than hemorrhage, although this is not invariably the case. In some instances consciousness is not entirely lost, and as a result the hemiplegia may develop before the coma. When unconsciousness does occur, there are usually less congestion of the face and not such marked evidence of increased arterial tension as we find in hemorrhage. Among the other general symptoms may be mentioned convulsions, often epileptiform in type, vomiting, and occasionally delirium. The permanent symptoms resemble exactly those produced by hemorrhage, but recovery is usually more rapid and more complete than in the former condition. As the Sylvian artery is most frequently affected, the symptoms resemble the cortical type of apoplexy.

Apoplexy occurring in children differs from that occurring in adults only by the fact that the initial symptoms are more severe, and the convulsions are frequent and may be repeated. The permanent symptoms differ slightly, inasmuch as aphasia rarely persists. The paralysis may be partial, and may in some instances be replaced by or associated with athetoid movements. Sensation is rarely impaired.

Bulbar Paralysis.

Bulbar paralysis is a disease of the peripheral motor neurons arising in the medulla. It is characterized by the degeneration of the muscles of the lips, tongue, and pharynx, which grow smaller, exhibit fibrillary twitchings, and give the electrical reactions of degeneration. Sensation is not affected. The course is slowly progressive. The earliest symptom is dysarthria, then difficulty in swallowing, chewing, and phonation. The face becomes expressionless, the mouth remains open, saliva dribbles from it, and occasionally the eyelids are involved and the eye remains open (lagophthalmos). The cardiac action and respiration may be rapid. Death usually occurs as a result of inspiration pneumonia or exhaustion. In the variety known as *asthenic bulbar paralysis* there may be long remissions or even permanent recovery. The muscles become readily fatigued and exhibit the myasthenic reactions.

Pseudobulbar palsy is characterized by the same symptoms as bulbar palsy, but no lesions are found in the medulla; symmetrical lesions are sometimes found in the cerebral hemispheres.

Acute Encephalitis.

This is a condition that rarely can be diagnosticated during life. It may be suspected, however, if, in the course of some other acute infectious disease, especially acute endocarditis, the patient develops intense headache, severe delirium, and perhaps local palsies. There may be general exaggeration of all the reflexes, with ankle-clonus, and usually hyperæsthesia of the skin and exaltation of the special senses. Examination of the eye-grounds usually fails to reveal optic neuritis. There is leucocytosis, and cultures from the blood and spinal fluid are often positive.

Abscess of the Brain.

This is usually secondary to some local focus of suppuration or to pyæmia. There is often a history of mastoid disease. It may be acute or chronic. Acute abscesses are characterized by violent general disturbances, chiefly fever, chills, leucocytosis, headache, delirium, and coma. Chronic abscesses give rise to symptoms of local disease, which depend, of course, upon the location of the abscess. The course is usually intermittently progressive; thus there may be first a monoplegia, then hemiplegia, and finally aphasia; or the earliest symptom may be hemianopsia, followed by paralysis. The commonest seat is in the temporosphenoidal lobe, as a result of infection following ear disease. This often gives rise to mind-blindness or amnesia. Sometimes there are no general symptoms if the abscess is located in the so-called blind regions of the brain. The focal symptoms may not be manifest until rupture has occurred. This often gives rise to an epileptiform attack. Chronic abscess of the brain is occasionally observed in cases of congenital heart disease.

Tumors of the Brain.

Like the preceding lesion, these give rise to two groups of symptoms: general, which are merely those of increased intracranial pressure; or local, which are due to involvement of centres and tracts. The general symptoms of brain tumor are: (1) Headache; this is usually very severe, of a boring character, and subject to exacerbations. (2) Vomiting; this is paroxysmal, and often occurs without nausea. (3) Papillitis; it usually occurs early, is intense, and often leads rapidly to blindness. (4) Increased blood-pressure. The local symptoms are, of course, numerous. Tumors in the *frontal lobe* may give rise to none, or only to slight disturbance of intelligence and perhaps to a tendency to make puns. The headache is usually frontal, and occasionally in subcortical tumors there may be nystagmus. Tumors in the *motor region* may cause irritative or destructive changes in the tissue. Irritation is manifested by local spasms, which may or may not be succeeded by general convulsions (Jacksonian epilepsy). Paralytic lesions belong to the monoplegic or hemiplegic types. Tumors in the *parietal lobe* may cause interference with the muscle-sense or some disturbance of vision- or speech-centre, according to their situation. The loss of the stereognostic sense is a common symptom. Tumors in the *occipital lobe* usually cause mind-blindness—that is, inability to recognize objects, and preservation of the pupillary reflexes. If unilateral, they often cause hemianopsia. Tumors in the *cerebellum* usually produce marked disturbance in coördination, and the patient exhibits the peculiar staggering gait, not increased by closing the eyes, that has been described. Optic neuritis is an exceedingly common complication, but there may be descending atrophy of the optic nerve without preceding choked disk. The headache is severe; it is situated in the posterior portion of the head; there is often tenderness over the external occipital protuberance. The patient may suffer from vertigo, which at times is almost constant. The general symptoms are usually pronounced. There

are convulsions, vomiting, disturbances of respiration, and sometimes marked slowing of the pulse. Tumors of the *basal ganglia* produce very variable symptoms, the majority apparently being the result of pressure upon the internal capsule. Tumors of the *thalamus* often give rise to hemianæsthesia with loss of mimicry, and in some cases apparently to athetoid movements. In all forms of cerebral tumors, but particularly in slowly growing tumors of the cerebellum, the early symptoms may be those of neurasthenia or even hysteria, and the diagnosis for a long time is exceedingly difficult. Tumors in the different fossæ of the skull often give rise to symptoms dependent upon pressure upon the cranial nerves. In the *anterior fossa* there may be loss of the power to smell upon one side. In the *middle fossa* the nerves chiefly affected are the optic, giving rise to unilateral blindness; or, if the tumor involve the chiasm, to bitemporal hemianopsia; if it passes upon the oculomotor nerves, the abducens, and the pathetic, it may give rise to more or less complete ophthalmoplegia. Tumors in the *posterior fossa* commonly involve the facial and auditory nerves; and facial paralysis with nerve-deafness on the same side is characteristic of tumor in this situation. The hypoglossal nerve may also be involved. Tumors may grow slowly, rapidly, or cease to increase in size, and the symptoms show a corresponding rate of development. In rapidly growing tumors apoplectiform attacks are frequent, but a certain amount of compensation occurs, and remissions are not uncommon. In slowly growing tumors the symptoms may remain apparently stationary for long periods. Tumors are sometimes entirely latent, and are only discovered accidentally at the autopsy.

Sclerosis of the Brain.

This is usually a diffuse or a multiple lesion that gives rise to a great variety of symptoms, more or less indefinite in character. Ordinarily the lesion is congenital or develops shortly after birth. The patient remains an imbecile or an idiot, and soon develops epileptic convulsions. If the sclerosis is more pronounced on one side than on the other, there is usually a tendency to fall toward the opposite side. There may be arrest in development in these limbs, and more or less muscular paralysis. Occasionally, apparently as a result of fœtal thrombosis or embolism, the sclerosis may be limited to one portion of the brain or even to one hemisphere. In this case there is always arrest in the growth of the opposite side of the body.

Hydrocephalus.

Hydrocephalus (*chronic infantile form*). This is characterized by an extraordinary alteration in the contour of the head, which becomes greatly enlarged and globular in shape, while the face remains small and infantile in appearance. The fontanelles and sutures remain widely open, or are closed only very late by Wormian bones. The veins of the scalp are very distinct, and occasionally fluctuation may be detected in the head. The symptoms are sometimes exceedingly pronounced; at other times entirely absent. Persons with a moderate degree of hydrocephalus have

displayed through life a normal intelligence. In pronounced cases the head is heavy and the muscles of the neck are unable to support it. The child develops slowly in intelligence or may even be an imbecile or an idiot, and epileptic convulsions are very common. Occasionally ocular symptoms may be present. These consist of ptosis, strabismus, or nystagmus, and sometimes of atrophy of the optic nerve and blindness.

Acute Delirium.

This is a disease characterized by prodromata and a stage of excitation, and usually terminates in death. The prodromata consist of disturbances of the general health, loss of appetite, and insomnia. The patient is restless, anxious, and may show diminution of intelligence, and become more or less violent. He then rapidly passes into the stage of excitation, is restless, noisy, and frequently homicidal, shouting disconnected words or sentences, singing or shrieking. Sometimes there are delusions of persecution, and he attempts to escape. In addition, there are the symptoms of the so-called typhoid state—high fever, profound prostration, dry tongue, and rapid and weak pulse. The patient refuses all food, is continually active, and emaciates very rapidly. Among the objective symptoms are increase of the reflexes, narrowing of the pupils, and hyperæsthesia, with more or less hypalgia. From this stage the patient passes into a state of collapse, lies in a condition of muttering delirium, with carphology, and usually dies from exhaustion.

General Paralysis of the Insane.

General paralysis of the insane is a form of progressive dementia characterized by delusions of grandeur or states of depression associated with exacerbations of maniacal character. There are, in addition, weakness and tremors of the muscles of the face, paresis of the extremities, Argyll-Robertson pupil, and peculiar disturbances of speech. It is a disease of middle adult life, the first symptoms occurring between the thirtieth and fortieth years. There is often a history of syphilis. It is usual to recognize three stages: The prodromal stage, characterized by irritability or sometimes by depression; diminution or loss of the moral sense; impaired judgment, particularly in business affairs; and a tendency to extravagance and dissipation. Frequently symptoms associated with degeneration, such as intolerance for alcohol, intense egotism, etc., appear. The sexual appetite in this stage is often greatly increased. Memory fails, and the intellectual capacity is considerably diminished. There are often slight disturbances of speech, and sometimes paralytic pupils. Frequently there are insomnia and occasional attacks of migraine. In the second stage, which usually develops gradually, the attacks of migraine are replaced by apoplectic or epileptic attacks or by distinct maniacal conditions; memory is greatly impaired, the intellect is considerably disturbed, the patient becoming unable to do easy mathematical problems, to comprehend his environment, or to sustain a simple conversation. Usually there are delusions of grandeur, the patient believing himself rich, beautiful,

successful, intelligent, and reiterating constantly his advantages, although from time to time there are states of depression and partial recognition of the failure of power. In other cases, however, particularly among chronic alcoholics, there is distinct melancholia; the patient is hypochondriacal, or may have delusions of persecution or a sense of misfortune. The disturbances of speech are characteristic; the most common is the omission of syllables. This may best be tested by asking the patient to repeat certain words, particularly those containing a number of r's and l's, as "third artillery brigade," "truly rural," etc. The intonation is often monotonous and often there is an unpleasant nasal drawl. There is marked tremor of the lips and of the tongue, producing a sort of ataxia in the speech, with the disturbance of the formation of nearly all the sounds. The pupillary changes are similar to those described in the prodromal stage, but usually are more pronounced. Piltz and Westphal have described a peculiar reflex contracture of the pupil upon forcible closure of the lids that is more common in general paresis than in any other disease. The extremities are weak, and often exhibit distinct tremors. Trophic lesions are not so common as in tabes, but do occur. They are of the same character and possibly due to tabetic changes in the cord. Finally, the patient becomes completely demented, usually lies quietly and placidly in bed, or occasionally mutters unintelligible sounds. Sensation, either as a result of impaired perception or because of degenerative changes in the peripheral nervous system or the spinal cord, becomes greatly impaired, particularly the pain sense. The patient is unable to stand, and has involuntary or rather unperceived micturition and defecation, and frequently develops bed-sores or cystitis; even rupture of the bladder may occur. A curious and quite common symptom is the gnashing of the teeth, which in some cases is almost persistent. Death usually occurs from exhaustion. Among the less frequent symptoms are a curious unsteadiness of gait, exaggeration of the reflexes, and rapid diminution in weight, particularly in the last two stages.

Epilepsy.

This is a condition characterized by attacks of clonic convulsions associated with loss of consciousness and usually some impairment of intelligence. In the characteristic epileptic fit we can usually distinguish three stages: the prodromal stage, the attack, and the post-epileptic stage. In the prodromal stage auræ are frequently present. These may be of the most varying character. A patient may have either a curious sensation in the epigastrium, paræsthesiæ in a limb, and the subjective sensation of movement, or disturbance of the special senses, particularly an unpleasant odor or a whirring sound. Sometimes the sensations are painful or distressing, as a sense of constriction about the throat. At other times there is giddiness, vertigo, or nausea, or the recurrence of some particular idea. Occasionally the auræ consist of some imperative movement, such as whirling about, running, or jumping. At the commencement of the attack there is usually a cry—the epileptic cry. Ordinarily this is a curious sort of gasping, due to the forcible contraction of the thorax and partial

closure of the glottis. In some cases, however, it may be a loud shriek. The patient then falls to the ground, and the convulsive movements commence. These are rarely of equal vigor on both sides; the head and the eyes show conjugate deviation; the face is bluish and pallid; the mouth is filled with frothy fluid, which is often blood-stained because the tongue has been bitten; the limbs may be extended or flexed in tonic contraction. This is soon replaced by a violent to-and-fro tremor. The patient is completely unconscious, and may, in falling, cause himself serious injury. There is no conjunctival reflex; the pupils are widely dilated; frequently the urine is passed during the attack, and there is occasionally profuse sweating. Toward the end the convulsive movements become less frequent. Respiration is re-established: at first it is irregular, but gradually becomes more and more steady. The cyanosis disappears, and the patient usually passes into a profound sleep. This may last several hours, and he then awakes, feeling dull and fatigued, but otherwise normal. At other times, immediately after the attack, there is vomiting or nausea, and sometimes a feeling of excessive hunger. He may become maniacal, usually with homicidal tendencies, or the post-epileptic stage may be manifested by nothing more serious than some imperative movement, such as running or shouting. The convulsive stage may be replaced by purely sensory phenomena, without complete loss of consciousness, or there may be merely a fine tremor, or the patient may simply run or be otherwise violent while wholly unconscious. Often there is loss of memory for a longer or shorter interval after the attack (post-epileptic amnesia). The attacks may occur with very varying frequency. In some cases the interval may be years, in others months, weeks, or days; or several attacks may occur in the same day. The severity of the case is to be determined rather by the frequency of the attacks than by their individual violence. If they recur so frequently that one is not completed before the other commences, and the patient remains unconscious for some time, the condition is termed *status epilepticus*. The prognosis is grave.

Petit Mal. In this condition the loss of consciousness is so transitory and the motor symptoms are so slight that its nature often escapes detection. The patient, if talking, will suddenly stop for a moment; there are a peculiar rigidity of the expression and perhaps slight swaying. These will disappear almost immediately, and the patient will resume the conversation. Sometimes after these attacks there will be a feeling of drowsiness for a short period. Auræ may be present in the form of giddiness or twitching of a limb. The attack may also occasionally be ushered in with a scream or a peculiar gasping expiration. Immediately after the attack automatic movements may be performed. Attacks of *petit mal* often occur during sleep, and the only symptoms then that point to the existence of the disease are a feeling of heaviness in the morning, perhaps a sore and bitten tongue, and nocturnal enuresis.

Focal Epilepsy (Jacksonian epilepsy). This form resembles general epilepsy, with the difference that the motor or the sensory disturbances always commence in the same part of the body, and from this part gradually extend until they become general. Thus the thumb may be first affected, showing a tonic and then a clonic spasm; then the hand, the

arm, the whole of that side, or both sides; or the disturbance may commence in the foot. The disease almost invariably indicates the existence of a focal lesion in the brain.

General Symptoms in Epilepsy. Epileptics are usually dull, apathetic, having a tendency to excess in eating. An excess of indican is often present in the urine. In many cases there is a distinct mental impairment, or, when the disease occurs early in life, there may be congenital imbecility or idiocy. The temper of epileptics is usually irritable, and they are likely to commit acts of violence. They are peculiarly intolerant to alcohol.

Migraine. (Hemicrania.)

This is a disease characterized by paroxysmal attacks of headache associated with nausea and vomiting, and frequently with disturbances of the special senses. The attacks are usually followed by prolonged sleep. The headache is peculiar, in that it commences slowly as a dull but severe pain that gradually increases in intensity, with occasional exacerbations or throbbing, and is limited to one side of the head. Occasionally, however, it is bilateral, but is then usually unequal. At the same time the patient experiences a sensation of intense nausea that may be followed by vomiting. The special senses are affected in various ways. There may be photophobia, hyperacusis, and occasionally the appearance of a peculiar scotoma, which commences as a bright spot that spreads, the outer edge being of an irregular, jagged character, and finally disappears at the periphery of the field of vision. New lines constantly form at the centre, and follow those first appearing. Sometimes the patient complains of dimness of vision, and this may affect only part of the visual field. Occasionally there is temporary aphasia, particularly if the pain occurs in the left side of the head. In addition, the patients may observe vasomotor symptoms, paræsthesia, or occasionally stiffness or spasms in a limb. The paroxysm usually terminates in sleep, which may be prolonged, and when the patient awakens all symptoms have disappeared. Sometimes there is a severe attack of polyuria. The intelligence is not impaired.

Ménière's Disease. (Aural Vertigo.)

This is characterized by attacks of vertigo associated with nausea. The attack usually begins with tinnitus, then intense vertigo, which may come on so suddenly that the patient falls to the ground or is obliged to lie down and remain in this position until the attack is over.

Hysteria.

Hysteria is a disease due to disturbance of the self-control, producing a curious complex of symptoms that appear to be the result of imitation or of a desire to attract attention or sympathy, associated with certain disturbances of the special senses and of sensation. It usually occurs in young adults, especially in women, although males are frequently

affected. There is often neuropathic heredity, and frequently the stigmata of degeneration are present. The psychical symptoms are a certain tendency to self-consciousness, so that the patient is anxious to describe his or her sufferings to surrounding persons; is in the habit of performing ludicrous or startling acts for the purpose of attracting attention; is emotional, weeping or laughing readily, and is often irritable and suspicious. Among the *sensory symptoms* are areas of tactile anæsthesia or analgesia. These may involve exactly one-half of the body, including the accessible mucous membranes, or they may be symmetrical in distribution on both sides of the median line, and often form geometrical figures. They are not the result apparently of simulation on the part of the patient, because they remain unchanged for a number of days. Tenderness—that is, hyperalgesia—may be present over the ovaries and the spine. The areas of anæsthesia may be transferred from one part of the body to the other, either spontaneously or as a result of suggestion. The latter is most effectual when the transfer is made by means of a magnet or metals. The special senses may have their function exalted, so that the patients have an extraordinary acuteness of smell or hearing, or find it difficult to endure strong lights. Depression of the function of the special senses is perhaps more common, particularly loss of the sense of smell and of taste. Hysterical deafness is exceedingly rare. Hysterical blindness not infrequently occurs, and is characterized by widely dilated pupils which usually react to light, and by normal eye-grounds. The hysterical stigmata associated with the eye are of great importance, partly on account of their peculiarities, partly on account of their persistence. The most frequent is simple contraction of the formed field. This, however, occurs in other conditions, and is therefore not so characteristic as contraction of the formed field with inversion of the color field—that is to say, a red object will be seen further from the central visual point than a blue one. Monocular diplopia, in the absence of structural defect in the eyeball, is pathognomonic of hysteria. In rare cases three images may be perceived.

The *motor symptoms* are paresis, or occasionally complete paralysis. The commonest form of this is *hysterical aphonia*, in which the patients are unable to contract the vocal cords for the purpose of producing sound, but may be perfectly able to cough or perform any other function with them. In these cases speech usually returns suddenly under the influence of a strong emotion or suggestion. The paralysis in other parts of the body occurs in imitation of some form of organic disease. Thus there may be paraplegia, hemiplegia, or monoplegia. Loss of power is rarely complete, and occasionally patients move the limbs when they believe themselves unobserved. The electrical reactions remain normal, although the degree of resistance in the skin may be greatly increased. The reflexes are exaggerated, especially those due to cutaneous irritation, such as the plantar reflex, but ankle-clonus does not occur. The gait may be staggering, imitating cerebellar ataxia or the ataxia due to intoxication; sometimes there are tremors, coarse and irregular, and rarely constant. In some cases of hysteria actual contractures of the muscles occur, indicating the existence of trophic disorders. Spasmodic contractions

sometimes occur in the muscles of the abdomen, giving rise to an apparent or *hysterical abdominal tumor*. Actual trophic changes may also occur in hysterical patients, but these are rare in this country. There may be hemorrhages into or from the skin, particularly from the forehead, palms of the hands, and soles of the feet (*stigmata of the Passion*), or there may be localized areas of gangrene of the skin.

The *attack* (*crise hystérique*) may be divided into the prodromal period and the convulsive. The auræ consist of a variety of sensory disturbances, of which the most common is the sensation of a ball rising in the throat (*globus hystericus*). The patient may also have a sensation of heat or cold, or moisture of the skin, or various painful impressions. Occasionally the tenderness over the ovary is greatly increased (*ovaria*), and the attack may be precipitated by pressure in this region. It is impossible to describe all the movements that occur in the *grande crise*. The convulsion may be tonic or clonic. The patient may assume the most extraordinary positions. Among the most characteristic is opisthotonos, in which the heels and back of the head rest upon the floor or bed, while the body forms an arch; or the patient may assume attitudes that suggest or are characteristic of mirth, sorrow, fear, passion, etc. *Catalepsy*, a condition in which the limbs are plastic and remain in any position in which they are placed, may supervene. Consciousness is rarely entirely lost, although there may be subsequent total amnesia for the period of the attack, and, no matter how violent the movements of the patient, injury to any part never occurs. Gradually the movements become less violent, the patient becomes quiet, and consciousness returns. During the attack the pupils are usually dilated, the reflexes may be increased, and respirations are commonly extremely rapid—in one case that I observed they reached 100 a minute. Occasionally the attack may be cut short by pressure upon one of the hysterogenic zones. After the attack the patient may be perfectly normal. At times there may be persistent, perverse tendencies, such as unwillingness to eat, or, at least, a simulation of fasting.

Neurasthenia.

Neurasthenia is a disease characterized by an exceedingly complex symptomatology. The most common general symptom is a subjective sense of fatigue, both mental and physical. The symptoms may be divided into the general and special groups: the former including those common to all forms of neurasthenia, the latter those associated particularly with subjective and objective functional disturbance of the various organs. The mental symptoms are various. The patients are usually querulous, depressed, and hypochondriacal. They are very irritable, but incapable of prolonged emotional exaltation. They find difficulty in concentrating their attention, particularly upon those subjects with which they have previously been familiar. Memory is impaired, and the intellectual capacities are apparently diminished. It must be remembered, however, that careful testing of the memory or judgment rarely shows that it is seriously affected. An important symptom is the insomnia. This may

be of all varieties, but ordinarily the patient, after sleeping in the early part of the night, will awaken and be unable to sleep again for some hours. The statements by the patients in regard to this symptom are very unreliable. Frequently they complain of unpleasant or frightful dreams. Among the sensory symptoms the most important is headache. This is of a peculiar but almost typical form. The patient complains of a heavy, dull feeling, as if wearing some heavy object, the usual simile being a leaden helmet. Occasionally the pain is localized—sometimes to the occipital region and sometimes to a circumscribed area, the latter usually the result of suggestion. Another symptom that is very common is pain in the back. This is usually felt in the neck or in the lumbar and sacral regions; it is of a dull, persistent character, and may be associated with points of tenderness over the spine. Occasionally there are disturbances of the special senses. The patient may complain of inability to see sharply, or there may be muscæ volitantes. At other times he will fail to hear distinctly, or may complain of roaring or tinnitus. Actual diminution of the visual power or of the sense of hearing does not occur. The patients may complain, however, of paræsthesiæ in the limbs and of various symptoms usually the result of suggestion. Sensation is otherwise normal. There is usually a general decrease in muscular power. Sometimes this may be preserved for short periods of activity, but fatigue, as a rule, comes on very rapidly. At other times it is impossible for the patient to exert the amount of force that would be normal for his muscular development. Occasionally this weakness is localized to one limb or side of the body. When the patient is directed to hold a limb rigid or to extend the fingers forcibly, a fine tremor of the extremities is visible. This may be persistent or readily exhausted; in addition, fibrillary twitchings of the muscles not infrequently occur. The tendon-reflexes are generally exaggerated. Ankle-clonus, however, excepting the form spoken of as pseudoclonus, is exceedingly rare. Absence of the knee-jerk does not occur in neurasthenia. The cutaneous reflexes are sometimes greatly exaggerated, sometimes decreased. Vasomotor symptoms are very common. The patient flushes easily, and there is often dermographia; he complains of palpitation and occasionally of irregularity of the heart's action. Tachycardia is not uncommon. Often perspiration is produced by slight exertion.

In addition to these symptoms, the neurasthenic may complain of various local disorders of the nervous system; he usually suspects that he has locomotor ataxia, and he will probably have learned the symptoms of this condition sufficiently well to imitate them more or less accurately, or he may believe himself suffering from general paresis or brain tumor, or any other condition with which he may be familiar. From general paresis the diagnosis is sometimes quite difficult unless the Argyll-Robertson pupil, which never occurs in neurasthenia, is present. Another common form is gastro-intestinal neurasthenia. The patient may complain of excessive acidity, and, in fact, vomit from time to time masses of acid material, or there may be difficulty in digestion and hypochlorhydria or anacidity. Constipation is an exceedingly frequent symptom. From time to time the patient may also evacuate large quantities of mucus, and

sometimes there may be persistent mucous diarrhœa. This is one of the most intractable forms of the disease. Finally, the patient may be a sexual neurasthenic and believe himself suffering from organic or functional disease of the genital organs. In this variety the various types of sexual perversion are usually, but I believe incorrectly, included. The degree of neurasthenia is spoken of as mild or severe, according as the symptoms are slight or pronounced.

Traumatic Neurosis (Railway Spine).

This can hardly be described as a clinical entity. Some of the cases are unquestionably hysteria; others partake of the nature of neurasthenia; others are instances of pure simulation. Not infrequently all these factors are united in one patient. The usual course of these cases is as follows: After some severe accident, with or without demonstrable injury, usually of such a nature that a claim for damages can be made, the patient begins to complain of various paræsthesias in the supposedly injured part, usually the back; these gradually increase in severity, may be associated with motor disturbances, such as tremor, spasms or paresis, and occasionally with objective sensory disturbances. At the same time there is insuperable dislike for any form of sustained work, irritability of temper, more or less severe insomnia, and occasionally impairment of the general nutrition. Tremors often of the most severe form are present, usually elicited by voluntary movement or the presence of observers. They may be associated with the simulation of intense suffering. The tendon-reflexes are usually increased, the skin-reflexes sometimes diminished, the sexual power impaired, and the visual fields contracted. The patient may complain of palpitation, and the pulse may be high. He devotes himself to the study of his symptoms, to the exclusion of other forms of mental occupation. The course is variable, but in the great majority of cases more or less improvement occurs as soon as damages have been obtained. Nevertheless, some cases do not improve under these circumstances, and others occur under circumstances that preclude any possibility of recompense. The diagnosis should never be made until the patient has been under skilled observation for a considerable period.

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