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Fig A.

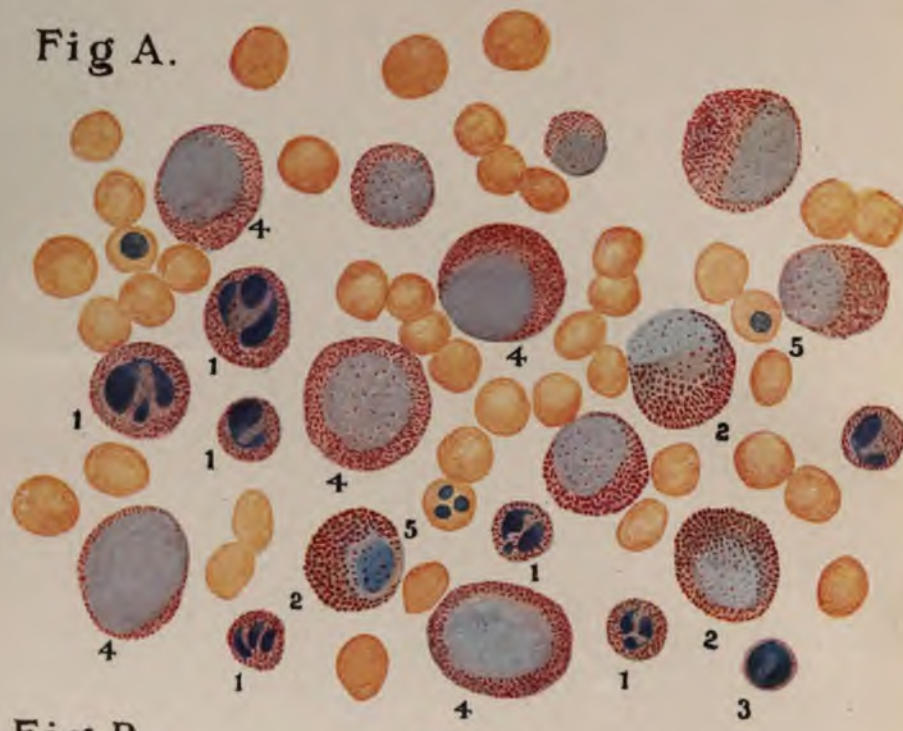
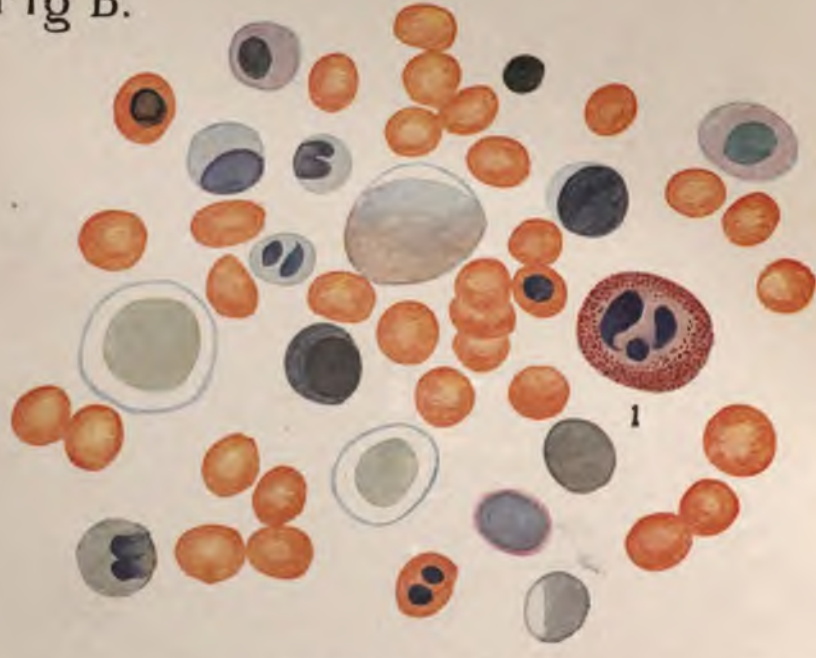


Fig B.



THE BLOOD IN LEUKEMIA.

FIG. A. SPLENOMYELOGENOUS LEUKEMIA: YELLOW CELLS, ERYTHROCYTES; 1, POLYMORPHONUCLEAR NEUTROPHILES; 2, EOSINOPHILES; 3, LYMPHOCYTE; 4, MYELOCYTES; 5, NUCLEATED ERYTHROCYTES.

FIG. B. LYMPHATIC LEUKEMIA: YELLOW CELLS, ERYTHROCYTES; 1, POLYMORPHONUCLEAR NEUTROPHILE. ALL OTHER CELLS ARE LARGE OR SMALL LYMPHOCYTES.

A TEXT-BOOK

ON THE

PRACTICE OF MEDICINE

Designed for the Use of Students

BY

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ILLUSTRATED BY TEN FULL-PAGE PLATES AND FIFTY WOOD-ENGRAVINGS

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PREFACE.

THERE are many excellent textbooks on the Practice of Medicine. Nevertheless, an experience of nearly twenty-five years as an instructor has convinced me that a book of the character of this one cannot fail to meet the requirements of medical students. It has been my aim to state only the facts of medicine accepted by the best authorities, and to express them in simple, concise language, and, as far as possible, in logical sequence. Personal experience has been kept in the background, and the pleasant diversion of case reports has been entirely omitted. The names of authorities have been given only when it seemed improper to omit them. While I am indebted to all the recent textbooks and larger treatises, and although I have scanned the current periodicals, I have endeavored to make use of only the established facts, omitting discoveries that rest solely upon theory or undemonstrated observations.

Both the English and metric systems of weights, measures, and temperatures are given throughout the text, the metric system in parentheses. When the exact equivalent is unimportant, the nearest round number is given, as an aid to memory.

Part I, entitled Principles of Medicine, is inserted for the purpose of affording the student a convenient means of refreshing his memory in the pathological processes constantly recurring in the study of diseases. And it is hoped that Part III, on Clinical Methods, will serve the same end and prove of continued, if not of greater, value, after the erstwhile student has attained to the higher station of a practicing physician.

JAMES M. FRENCH.

CINCINNATI, September 1, 1903.

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PART I.

THE PRINCIPLES OF MEDICINE.

A TEXT-BOOK
ON
THE PRACTICE OF MEDICINE.

DISEASE is an abnormal state of the body, a perversion or interruption of the function of any organ or tissue, with or without corresponding structural change.

The disturbance must be more or less continuous. A temporary alteration of function, due to a transient cause, may be strictly physiological and it may result in the removal of some harmful agent or substance and thus prevent more permanent disturbance. The rapid respiration and quickened heart's action which follow active exercise, for example, do not denote disease, for experience has shown that they are normal and necessary to the vitality of the tissues; but if a similar acceleration of these functions habitually follow slight exertion, it is to be regarded as pathological, an indication of disease.

In many instances disease is first manifested by either an increase or a diminution of functional activity, on the order of that just referred to, or there may be an evident loss of harmony between two or more physiological processes. As a rule abnormal function denotes an impairment of structural integrity, although we may not be able to discover it. But an interruption or perversion of function may undoubtedly occur in an organ free from structural change. Our knowledge of the pathological changes which underlie the manifestations of disease is becoming daily more exact, but it does not yet enable us to exclude from our classification all those affections which have been regarded as functional.

In a systematic study of medicine, the diseases are studied as individuals, as entities. The first aim of the student is to become able to recognize each disease by its cause, the structural changes and clinical manifestations in which it differs from all others. In some affections it is one of these features, in some another, that is most important to its recognition. But Medicine is not yet an exact science, and it is probable that many affections which we now regard as distinct diseases will, in the course of time, be found capable of more accurate subdivision. The acute exanthemata as we know them were at one time regarded as varieties of a single disease, and at the time this is written it is a matter of dispute whether or not there is yet a "Fourth disease" in the measles and scarlatina group. In the study of individual diseases, again, we must often take into consideration two or more subvarieties of the same affection, owing to differences in the symptomatology of different cases, as in malaria and rheumatism. And it is not improbable

that many of these will, in the future, be resolved into independent affections when their causes become more definitely known.

CLASSIFICATION OF DISEASES (NOSOLOGY).

It is customary to classify diseases according to their origin, clinical course, duration, and other features. No entirely satisfactory classification has ever been devised, however, and the chief object in presenting the following brief classification is to familiarize the student with the terms that are generally employed:

I. Every disease may be classed as general or local in character. (1) A *general* disease involves the entire system. The class embraces (*a*) most of the acute infections, as typhoid fever, measles, smallpox, (*b*) the so-called constitutional or blood diseases, as pernicious anemia, gout, and scurvy, and (*c*) the intoxications by lead, arsenic, opium, and other poisons.

(2) *Local* diseases are those which affect particular organs or tissues. They may be subdivided into (*a*) *organic*, or structural, embracing affections of the brain, heart, lungs, skin, or other organs or tissues, and (*b*) *functional* disorders, in which the action of an organ is impaired without discoverable structural lesion. The number of functional disorders has been greatly reduced in recent years by the discovery that many which were formerly so regarded depend upon lesions more or less remote from the part in which the clinical manifestations appear. And, since the disturbing influence often originates in the nervous system, or is at least conveyed through the nervous system to the point of its expression, these affections have been grouped under the head of reflex *neuroses*. It should be remembered also that many of the general diseases have their local expressions in some organ or tissue, and that a primarily local disease may lead to general disturbance of the system.

II. A disease may be acute, subacute, or chronic. (1) It is *acute* when it is severe in character, of short duration, and runs a rapid course, a feature of the acute exanthemata, (2) *subacute* when these features are less marked as in some cases of rheumatism, and (3) *chronic* when of slow progress and long duration, as in tuberculosis and syphilis. The distinction between acute and subacute is entirely one of degree and not always clearly defined.

III. The course of a disease may be paroxysmal, periodic, continuous, or recurrent. (1) A *paroxysmal* disease is characterized by sudden exacerbations of severity, or it manifests itself in sudden, explosive seizures, as in epilepsy and some forms of neuralgia. (2) A *periodic* disease is one which occurs with regularity at definite intervals of time, as tertian and quartan malaria. (3) The term *continuous* is applied to some fevers to describe their uninterrupted course, and (4) *recurrent*, to designate a disease that returns during or soon after apparent convalescence, as relapsing fever. The term *recrudescence* is applied when the symptoms unexpectedly return after a remission and when their return is due to a revivifying of the original infection or to a reinfection by the same organism, as occasionally occurs in typhoid fever.

IV. Diseases are further classified as sporadic, endemic, epidemic, and pandemic in the extent of their prevalence. (1) A *sporadic* disease is one that may occur in any place at any time; the term is employed

chiefly to distinguish such affections as sporadic cholera from similar affections of an epidemic character. (2) An *endemic* disease is one which, owing to some local influence, is more prevalent in a certain locality than elsewhere, as is usual with malaria and yellow fever. (3) An *epidemic* disease attacks simultaneously or in quick succession a large number of individuals in the same locality, or spreads rapidly over a large territory, as is frequently the case with smallpox, scarlatina, and measles, and (4) a *pandemic* is one that attacks almost without exception the entire population of a city or country, as occurs in influenza, cholera, and bubonic plague.

V. With reference to their mode of origin, diseases are hereditary, congenital, or acquired. They are (1) *hereditary* when communicated to an individual by his progenitors. In most instances it is only a susceptibility to the disease that is thus handed down. (2) A *congenital* disease either exists or originates at the time of birth, and (3) an *acquired* disease is one that develops in after-life, independently of either hereditary or congenital influences.

VI. In their causation, diseases may be infectious, parasitic, or toxic. (1) The term *infectious* is now generally limited to diseases that are more or less definitely known to owe their origin to the presence of bacteria. Their number is continually being added to as new discoveries are made in bacteriology. The class is sometimes subdivided into (*a*) *contagious* and (*b*) *non-contagious*, to denote that the disease can or cannot be communicated to a healthy person who comes into contact with one who is infected. The contagion is said to be *mediate* when it can be carried through the medium of fomites, such articles as clothing, furniture, draperies; or *immediate* when actual contact is necessary. (2) The term *parasitic* is generally restricted in its application to the diseases due to the presence of animal parasites. (3) A *toxic* disease, or intoxication, is caused by a chemical poison. The poison may be (*a*) organic, including ptomains and leucomains, or (*b*) inorganic, phosphorus, arsenic, lead, etc.

VII. Such terms as primary, secondary, and specific are sometimes employed. An affection is *primary*, or essential, when it develops spontaneously or independently of any other affection. A *specific* disease is one that is due to a definitely recognized virus or microbe and runs a definite course.

THE CAUSES OF DISEASE (ETIOLOGY).

Any influence that is capable of impairing the integrity of an organ or tissue or of disturbing its function may become a cause of disease. In the production of most affections a succession or combination of such influences is operative.

The causes of disease may be divided into two classes, predisposing or remote, and exciting or determinate.

1. A *predisposing* cause is one which prepares the individual for the action of the *exciting* cause by rendering him susceptible to its action. It is owing to some predisposing influence that one individual falls victim to a disease from which another individual similarly exposed escapes. Predisposition may be either *inherited* or *acquired*. Among the influences that are recognized as the most common predisposing causes are (*a*)

age. (*b*) sex. (*c*) race. (*d*) occupation, (*e*) diet, (*f*) habits of life, (*g*) climate, and (*h*) previous illness.

2. The most prominent exciting causes are (*a*) infection by bacteria, (*b*) autointoxication, (*c*) invasion by animal parasites, and (*d*) poisons. Injury, exposure to heat or cold, improper food or drink, and many other influences may act as either predisposing or exciting causes. All these influences will be considered in connection with the individual diseases.

Infection is the condition produced in the body by the entrance and propagation of pathogenic bacteria. It is considered under the head of Bacteriology, on page 37.

Autointoxication, or self-poisoning, is a term applied to a class of diseases not yet fully identified, which result from the accumulation in the system of the products of metabolism or those of bacteriological decomposition. It results in some instances from processes which are in themselves normal, the poisonous effect arising from a disturbance of the relation between production and elimination. There may be (*a*) overproduction alone, or (*b*) only deficient elimination; or these conditions may be combined. We are indebted chiefly to Bouchard for the knowledge we possess of the conditions.

1. ***Leucomains.***—The products of metabolism have been named leucomains. They are believed to be derived from the nuclein in the nuclei of the cells. The best known members of the class are: adenin, creatin, creatinin, guanin, xanthin, hypoxanthin, and paraxanthin. Vaughan and Novy have found them nontoxic, except paraxanthin, which has been found only in the urine. To their presence are, nevertheless, attributed many disturbances, especially on the part of the nervous system. Urea is closely related to these bodies in its origin and supposed action.

2. ***Ptomains.***—The decomposition of proteids by the action of bacteria gives rise to another class of poisons, alkaline bases, known as ptomains. Among those which have been isolated are cadaverin, putrescin, neuridin, saprin, and the aromatic group, indol, phenol, and cresol; there are many others which have been produced for the most part through experimentation. They may enter the body preformed, in food that has previously undergone putrefaction, as when partially decomposed meat or fish is ingested, or they may be formed by the decomposition of proteids in the intestine, through the action of bacteria.

3. ***Acid Intoxication.***—Another form of intoxication is due to the metabolic production of such acid bodies as uric, lactic, sarcolactic, sulphuric, phosphoric, and fatty acids, especially as a result of fever, inanition, anemia, acute yellow atrophy of the liver, diabetes, and cancer. The production of these bodies is attributed both to decomposition of proteids and to defective oxidation. Their presence in abnormal quantity is supposed to be indicated (*a*) by disturbances of the nervous system, mental dulness, and especially the coma which so often announces approaching dissolution; (*b*) by the production of a cachexia; (*c*) but especially by excessive elimination through the kidneys.

4. ***The retention of bile salts*** and their entrance into the circulation produce a form of autointoxication, *cholemia,* which is mani-

fested by the characteristic discoloration of the skin and other tissues, accompanied with various systemic disturbances. The remains of disorganized blood and broken-down tissues, the results of injury, may be absorbed and cause intoxication, often manifested in the so-called *aseptic fever.*

Many of the substances which are capable of producing autointoxication are always present in the body, and we are not fully acquainted with the influences that cause them to be absorbed into the blood only at certain times. It has recently been attributed to a change in the osmotic pressure of the blood.

Our knowledge of the autointoxications is not yet so complete as to enable us in all cases to refer a group of symptoms to its specific cause. The most prominent manifestations are generally seen in the derangement of nervous functions, frequently accompanied with disturbances of the gastrointestinal tract and changes in the composition of the urine. Headache, drowsiness, anorexia or vomiting, hebetude or coma, sometimes convulsions, characterize most of the cases. The symptoms are often erroneously assumed to be due to uremia. Bouchard regards autointoxication as a frequent cause of trophic disturbances in the muscles, joints, and other tissues.

Animal Parasites.—A large group of diseases arises from the presence of animal parasites within the body. The lowest class of these parasites embraces the *protozoa,* to which belong the parasites of malaria and dysentery; the *psorosperms,* known also as cytozoa on account of their being found within cells, and the *coccidia,* sometimes classed with the psorosperms. The best example for study is the coccidium oviforme, found in small saccular dilatations of the bile-ducts of rabbits. Different parasites of this class have been described by various investigators, but not fully demonstrated, as the causes of carcinoma, epithelioma, sarcoma, measles, scarlet fever, pernicious anemia, and other diseases; and others are occasionally associated with skin diseases or intestinal disturbances.

Of the more highly organized animal parasites there are two classes, namely, the *epizoa* and the *entozoa.* The former exist only in the skin or upon its surface; the latter, about fifty in number, penetrate to the deeper parts of the body.

The entozoa are more familiar to us as worms. They may be divided into three classes: cestodes, nematodes, and trematodes. (*a*) The *cestodes* are the tapeworms. (*b*) The *nematodes* are round or threadlike worms, including the lumbricoids, filiariæ, and trichina. (*c*) The *trematodes* constitute a class to which belong the liver-flukes. Some of the entozoa enter the body as mature worms, some in a larval state, while others develop within the body from eggs that have been taken in with food or drink. Their relation to the production of diseases is further considered under the diseases attributed to them.

PATHOLOGY.

Disturbances of Nutrition and Metabolism.—The term metabolism is applied to the processes constantly going on in the body through which (1) the tissues appropriate the nutriment that is brought to them in

the blood and to prepare the protoplasm of the cells for special uses or for excretion. It is in the first instance a constructive process (anabolism) and in the second a destructive one (katabolism). So long as these two processes maintain a proper balance, the body remains in a normal state of nutrition. The source of supply is the food. In order to fully replace the losses of heat and energy occasioned by all the vital activities, the food ingested must be not only sufficient in quantity and suitable in kind, but its essential elements, proteids, fats, and carbohydrates, must be appropriated in sufficient amount.

Excessive Nutrition.—Oversupply of food does not necessarily produce excessive nutrition. The appropriation depends in part upon the character of the food, in part upon such extrinsic influence as exercise, but to a greater extent upon individual peculiarities of the metabolic processes. In many cases oversupply leads to only an excessive retention or discharge of such end-products of metabolism as urea. The overappropriation of nourishment seen in obesity is derived in part from the fat ingested with the food, but more particularly from the carbohydrates. It is very often not in proportion to the quantity of these substances ingested. Obesity is therefore regarded, in most instances, as a result of abnormal metabolism the nature of which has not been fully determined. By some investigators it is regarded as a result of deficient oxidation, especially when it is associated with anemia.

Diminished Nutrition.—A deficient supply of food, or an inability of the system to prepare and appropriate that received, results in a condition of inanition which may be slight or so extreme as to result in death. The first indications of inanition are generally a loss of body weight and a diminution of the energy of the various organs. The loss of weight results from the consumption by the organism itself, first of the fat and later of other tissues. The body appropriates its own tissues for food. The tissues are said to undergo atrophy. Impairment of the nutrition of a single organ or tissue from any cause leads to local atrophy, as in the wasting of a paralyzed member. General lack of nutrition is seen in many pathological processes, notably in fevers and in infectious diseases. When the result of chronic disease or of the growth of a tumor, the wasting is called *marasmus* or *cachexia*; when the result of toxic matter carried in the blood, it is sometimes spoken of as a *dyscrasia*.

Generalization of Disease.—An organ often becomes diseased as a result of a morbid process in another organ. Some diseases are definitely local in character and produce little or no disturbance in other parts of the body; while others begin as local processes and rapidly become generalized. The generalization of the infectious diseases depends in part upon the action of the toxins upon the nervous system and in part upon their affinity for particular cells. A similar generalization occurs in many noninfectious diseases, especially in the autointoxications, producing, as prominent manifestations, elevation of temperature, loss of strength, and emaciation.

An impairment of the function of one organ sometimes exercises an important influence upon other organs. At first functional, such disturbances may become organic. The original impairment of function in

some instances throws toxic matter into the circulation which acts injuriously upon the parenchyma cells of other organs, causing more or less pronounced degenerative changes in them. Or a similar result may follow the loss of an agent normally secreted by the organ which becomes the seat of a morbid process. The heart, voluntary muscles, glands, and kidneys are especially liable to become the seat of such degenerations, the kidneys more than other organs, perhaps, because they are called upon to remove from the system a greater part of the poisonous materials resulting from the disease processes.

The blood serves as the carrier of the toxic matters resulting from disease, as well as of those producing it, and consequently undergoes important changes in composition and quantity. These changes diminish the supply of nutrition to the organs and tissues and therefore constitute another factor in the generalization of the morbid processes.

Changes in the Blood and Circulation.—***The Blood.***—The quantity and composition of the blood remain remarkably constant during health, despite the many influences to which it is exposed. Slight changes take place from hour to hour, it is true, with the ingestion of food and drink, and the circulation is made the avenue of the effete products of metabolism on their way to elimination.

Plethora, or overabundance of blood, formerly regarded as of much importance, is believed to be unusual and of short duration. In the *oligemia*, or reduction of quantity, which results from hemorrhage, the loss is quickly compensated for by the contraction of the blood-vessels, the appropriation of fluids from the tissues, and generally by an increased supply of water that is drunk in order to quench the imperative thirst.

An abnormal increase of the water in the blood gives rise to *hydremia*, a condition which is overcome by a rapid formation of new blood-cells and the elimination of the water through the secretions. Hydremia is believed to occur in some conditions of anemia. The opposite condition, *anhydremia*, in which the water of the blood is deficient, is produced by a profuse discharge of water through the bowels in cholera, through the kidneys in diabetes, or by excessive sweating.

The diseases involving changes in the erythrocytes are considered under the Diseases of the Blood.

Leucocytosis (hyperleucocytosis) is a recognized feature of an increasingly large number of diseased conditions. When moderate, it is sometimes regarded as physiological. The number of leucocytes in the cubic millimeter of normal blood varies from 4,000 to 10,000. Increase or decrease beyond these limits indicates a pathological condition. Diminution of the number is termed hypoleucocytosis. Leucocytosis may be active or passive. The best example of the former is seen in phagocytosis (p. 40). The principal causes of leucocytosis are: (*a*) Infection and the resultant toxemia, (*b*) disease of the blood-forming organs, (*c*) malignant disease, (*d*) hemorrhage, and (*e*) the action of certain drugs. It sometimes develops also immediately before death, although absent during the course of the disease.

As a result of toxic influences, leucocytosis occurs in probably all the acute infectious diseases, except typhoid fever, uncomplicated tuberculosis, measles, and perhaps influenza. As an autointoxication it is seen

in acute disorders of digestion, gout, cirrhosis and acute yellow atrophy of the liver, acute and chronic nephritis, and hydronephrosis. In both these classes of cases it is an active process having for its object the protection of the system.

Hypoleucocytosis is always pathological and met with: (*a*) In the infections, typhoid fever, measles, tuberculosis, and influenza; (*b*) under certain conditions, in infections ordinarily attended with leucocytosis; (*c*) in some cases of leukemia and pseudoleukemia; and (*d*) as a result of the action of certain drugs. The development of a mixed infection in these cases immediately induces a hyperleucocytosis.

The Circulation.—The circulation of the blood is maintained almost entirely by the rhythmical contractions of the heart; the uniformity of blood pressure, by the elasticity of the blood-vessels. The pressure in the pulmonary artery is never so strong as that in the aorta. The normal relation between the heart's action and the blood pressure is under the supervision of the nervous system, to a great extent under that of the ganglia situated in the heart itself.

Deficient Blood Pressure.—The systemic blood pressure is diminished by every impairment of the integrity of the heart, whether involving its muscle, its valves, or its ganglia, unless the defect is fully compensated for by increased force of action. The heart muscle may be impaired by fatty and other degenerations resulting from (*a*) long-continued fevers, (*b*) the presence of poisonous matter in the blood, or (*c*) such impairment of nutrition as that caused by sclerosis of the coronary arteries. The action of the heart is interfered with also by (*a*) adhesions which bind the organ to adjacent structures, (*b*) an accumulation of fat or fluid in the pericardium, (*c*) the pressure of tumors above or below the diaphragm, as well as by (*d*) hydrothorax or ascites.

Deficient blood pressure in the arterial system, due to defective cardiac action, is generally attended with an increased accumulation of blood in the veins; a venous stasis, passive hyperemia, or engorgement. Incompetency of the mitral valve, for example, permitting the regurgitation of the blood into the left auricle, produces engorgement of the pulmonary circulation. The increased action of the right ventricle prevents for a time a further retardation of the circulation. But when, as sooner or later happens, the right heart loses its ability to compensate, the engorgement becomes general. When the right side of the heart is primarily affected, passive hyperemia rapidly develops in all parts of the body.

Weakness of the circulation, aided by gravitation of the blood, especially after long confinement to bed in chronic febrile diseases, often leads to such local disturbances as *hypostatic congestion* of the lungs or an accumulation of blood in the vessels of the more dependent portions of the body. *Extravasations* occur in the same manner and are seen as ecchymoses in the skin. They are often followed by edema and sloughing, as in the formation of bedsores.

Increased blood pressure is generally transitory, like that which results from overaction of the heart during violent muscular exercise or nervous excitement. It may result also from the presence of toxic substances in the blood, but it then gives place, as a rule, to weakness.

Overaction from violent effort may lead to hemorrhage, especially when there is a defect in the blood-vessel walls. The increased action of the heart occasioned by an effort to compensate for abnormal conditions within itself, or by changes in the blood-vessels, as in general arteriosclerosis, leads first to hypertrophy of the ventricles, but later to degeneration of its muscles, with permanent dilatation of its chambers. Many disturbances of the heart's action and of the circulation are to be attributed to the influence of the nervous system expressed through the vasomotor nerves.

Local Anemia.—When from any cause the blood supply of a part is diminished or completely cut off, a local anemia, or ischemia, is produced. This varies from a very slight deficiency to complete absence of blood. When an artery is suddenly obstructed, as by an embolus, this anemic condition is immediately produced. This is true especially of those organs whose circulation is supplied through terminal or end arteries, where an immediate relief of the anemia through anastomotic circulation is impossible. Here the anemia becomes the first step in the development of an infarction. The term *ischemia* is often restricted in its application to anemia caused by arrest of the arterial blood entering the part. A *collateral anemia* is the condition produced when the blood is withdrawn from a region to meet the demand for it in an adjacent area which is in a state of congestion.

Causes.—Local anemia is caused for the most part by (*a*) disease of the walls of the vessels supplying the area, (*b*) compression of the vessel by cicatricial tissue, tumors, or accumulated fluids, (*c*) inflammatory processes around it, or (*d*) by influences operating upon it through the nervous system. The most important of the diseases of the vessel walls capable of operating in this manner are acute inflammation, sclerosis, atheroma, syphilis, and amyloid disease. Thrombosis and embolism produce anemia of the part supplied by the obstructed vessel. The application of cold to a part renders it relatively anemic by constricting its vessels; freezing produces absolute anemia. Anemia resulting from hemorrhage is most profound in the extremities. Among the instances of local anemia produced through the action of the nervous system may be mentioned the early symptom of Raynaud's disease, the blanching often seen in a part affected with neuralgia, and the pallor of the face accompanying nausea or fright. A more or less profound anemia of the brain and of the skin accompanies inflammatory diseases of the abdominal viscera.

An anemic part becomes pale, softer, and cooler than normal; its nutrition and function are impaired. Long-continued partial anemia of a part or organ leads to fatty and other degenerations, with atrophy; complete anemia leads to necrosis.

Local hyperemia is an increase in the quantity of blood in a circumscribed region of the body. It may be active, when the blood is arterial, or passive, when there is an accumulation of venous blood.

1. *Active hyperemia*, or congestion, may be due to an increased demand for nutrition in the part, such as occurs in reparative processes. Pathologically it may be due to a dilatation of the vessels through vasomotor influences, central or peripheral in character. This occurs when the vasoconstrictor influence of the sympathetic nerves is inter-

rupted, as by the pressure of a tumor, or when the vasodilators in the spinal cord are stimulated, as sometimes occurs in neuritis. An interesting example of it is seen also in the unilateral flushing of the face in pneumonia. Active hyperemia occurs also when a tissue is injured mechanically or chemically; it may occur as a reaction from local anemia. A *collateral hyperemia* sometimes develops in consequence of profound anemia in another part. Hyperemia is always present in inflammation. Increased heat, redness, and slight swelling of the part are its usual manifestations. Its results are, at first, an increase of functional activity; later, inflammation or degenerative changes.

2. *Passive Hyperemia.*—When the presence of an increased amount of blood is due to a retardation or arrest of the flow of venous blood from the region, the hyperemia is passive. This occurs when a vein is compressed or closed in any manner. It varies in degree from a slight retardation to a complete stoppage (venous stasis). It is caused by inflammation of the vein or of the parts around it, by thickening of its walls through sclerosis, calcification, or syphilitic induration, or by compression of a new growth. A more general passive hyperemia is seen in the various organs, especially in the lungs and liver, as a result of valvular disease of the heart. The affected tissues at first become swollen and intensely red, then a transudation of serum occurs, and edema is produced; later, if the condition continues, fatty degeneration and ultimately necrosis may take place. The destroyed area is sometimes replaced by new fibrous tissue which is often deeply pigmented. The condition is then known as *brown atrophy.* The best example of it is found in the myocardium as a result of arteriosclerosis of the coronary arteries.

Dropsy and Edema.—Dropsy is a generic term and embraces all abnormal accumulations of fluid within the connective-tissue spaces and serous cavities of the body. Although the condition is practically the same in all cases, usage has given us several names for the designation of dropsical accumulations in different regions. When only the connective-tissue spaces of organs are involved, it is spoken of as an *edema;* an accumulation in the subcutaneous-tissue spaces, especially those of the lower extremities, is an *anasarca;* that in the peritoneal cavity, *ascites;* in the pleural cavity, a *hydrothorax*, or pleuritic effusion. When in the arachnoid space and ventricles of the brain, it is a *hydrocephalus*, and when in the pericardium, a *hydropericardium.* In general dropsy there is a progressive involvement of the connective tissue spaces and cavities.

The serous and connective tissue spaces normally contain a small quantity of plasma, which is fairly constant in each part. It is derived for the most part from the capillary blood-vessels, but in part, perhaps, from the lymph-vessels. Under normal conditions the plasma is taken back into the circulation after it has remained in the tissues for a time, performing its function of supplying nutrition. Some authors refer this return of the fluid entirely to the action of the lymph-vessels, while others believe that the blood-vessels are even more active in picking it up. A normal condition of the blood-vessel walls with reference to permeability and normal blood pressure in the capillaries is regarded as essential to the maintenance of a normal quantity of this fluid in the spaces,

and it has been suggested that osmosis through the capillary walls is responsible for its ebb and flow. It is no longer regarded as a product of secretion.

Causes of Dropsy.—1. A dropsical accumulation of fluid is generally the result of a disturbance of the relation between transudation and absorption. This in turn may be due to (*a*) increased blood pressure, (*b*) changes in the capillary walls which render them more permeable to the plasma, (*c*) changes in the composition of the blood which render it more diffusible, or (*d*) influences which otherwise retard the return of plasma to the circulation. The first of these causes, an increased blood pressure, is generally due to a retardation of the capillary and venous circulation and is therefore on the order of a hyperemia. It is a passive hyperemia, an increase of venous rather than of arterial pressure. Weakness of the circulation favors the transudation of serum, and the best examples of dropsical effusion are seen in cases of heart disease after compensation has failed and the blood has become stagnated in the veins.

2. *Arterial Edema.*—The existence of a strictly arterial edema has been questioned. The term is generally limited to the edematous condition in an inflamed area, always a transient condition. The permeability of the vessel walls is increased by thermal or chemical injury as well as by degenerative changes consequent upon disease.

3. Alterations in the character of the blood are looked upon as a most important factor in the production of dropsies of renal origin. A hydremia has been described in these cases in which the blood becomes watery through a reduction of its solid constituents, but the condition cannot always be demonstrated even in advanced stages of nephritis. The hydremia has been referred also to the retention of toxic substances in the blood owing to an inability of the kidneys to remove them. A third explanation refers it to deficient oxidation.

4. Interference with the flow of lymph through the larger channels may sometimes be a cause of dropsical accumulations, especially in the thoracic and peritoneal cavities. Probably nothing short of an obstruction of the thoracic duct is capable of acting in this way, and such obstruction is known to increase an already existing ascites.

5. The nervous system is not regarded as operative in the production of dropsical accumulations, except so far as the vasomotor nerves may sometimes be involved in it.

6. In some instances an accumulation of fluid replaces tissue that has been lost, as when a portion of the brain or spinal cord has been removed. This is called edema ex vacuo.

The fluid of a pure edema corresponds in its saline and aqueous composition to the serum of the blood, but it is deficient in albumin. The fluid of ascites is richer in albumin than is that of edema. When the effusion is due to disease of the blood-vessel walls, it contains more albumin and as a rule a larger number of blood-cells.

The swelling which accompanies an edema varies from a slight tumefaction to the most extreme distention. In extreme anasarca the skin becomes tense and glazed, and it is often rent to permit the escape of the fluid. The swollen part usually appears anemic; it may be cyanotic; it feels doughy or sodden, pits on pressure, and degeneration or necrosis is

apt to result. Edematous organs are lighter in color and "juicy" when incised. Their function is impaired. The gravity of the condition depends largely upon the part affected. Edema of the brain, glottis, or lungs is always attended with danger to life, and a general dropsy with serious failure of the circulation.

Hemorrhage.—Hemorrhage signifies the escape of blood from a vessel—the escape of all the elements of the blood in contradistinction to the escape of only the plasma, as in edema. It may be external or internal. In the latter form, the blood is retained within the tissues or one of the inclosed cavities of the body. In its origin, the hemorrhage may be arterial, venous, or capillary. Arterial hemorrhage occurs only through a lesion of the vessel-wall (rhexis); venous and capillary bleeding may take place either through a lesion of the vessel-wall, or more gradually by diapedesis, a stepping out of the elements of the blood through the normal spaces in the vessel-walls. A migration of the leucocytes from the vessels into the adjacent tissues is normal; but when the red corpuscles thus escape, it constitutes a diapedesis and is pathological. The principal causes of diapedesis are degenerative changes in the vessels due to the action of toxic substances, mechanical or thermal injury, or arrest of the circulation. Hemorrhage of this character is not usually great in quantity.

Causes.—The causes of arterial hemorrhage are: (*a*) Laceration or rupture of the wall of the vessel while in a state of health, a surgical condition, (*b*) disease of the blood-vessel, (*c*) increased blood pressure, and (*d*) nervous influence. Disease may affect the blood-vessel either internally or externally. The most important internal affections are fatty and other degenerations and sclerosis, often leading to miliary or larger aneurisms. Degeneration of the tunics of the vessel results chiefly from the toxemia of the infections or from malnutrition incident to fever, anemia, or the cachectic states. Among the causes of external disease of the vessels are the pressure of tumors, suppuration, and tuberculosis, thermal and chemical irritation.

Increased blood pressure is sometimes spoken of as absolute when it follows violent muscular effort, paroxysms of intense joy or grief, the coughing of acute bronchitis or pertussis, and convulsions. It is relative when due to the withdrawal of normal external pressure, as in asphyxia, or when induced by ascent to high altitudes.

Hemorrhage of nervous origin is due to paralysis of the vasomotor nerves or to a reflex mechanism which is not fully understood. Bleeding from the nose, lungs, stomach, or bladder, or into the substance of such organs as the kidneys and suprarenal bodies is sometimes of this character. Another interesting class of hemorrhages is exemplified in the stigmatization of hysterical ecstasy, in which blood infiltrates the skin of different regions, most frequently those wounded in the crucifixion.

Some persons have a natural tendency to hemorrhage; they are called bleeders, and the condition is known as the hemorrhagic diathesis. When inherited, it constitutes hemophilia. The hemorrhagic diathesis is sometimes acquired by persons not previously predisposed to hemorrhage, in the course of typhus, yellow fever, cholera, the plague, scurvy, hypertrophic cirrhosis of the liver, septicemia, pernicious anemia, purpura hemorrhagica, and certain other affections.

Varieties.—External hemorrhages are generally classified according to their source. Hemorrhage of the nose is designated epistaxis, hemorrhage of the lungs hemoptysis, that of the stomach hematemesis, that of the intestine enterrhagia. Uterine hemorrhage is subdivided into menorrhagia and metrorrhagia, occurring during or between the menstrual periods.

Internal hemorrhages receive their nomenclature in part from their location and in part from their character. Hemorrhage into the pericardium is called *hemopericardium*, that into the pleural cavity *hemothorax*. A hemorrhage into or beneath the skin or a mucous membrane is an *ecchymosis;* if this is confined to small areas and it produces small reddish or dark brown spots these are called *petechiæ*. A larger accumulation of blood in a tissue is a *suffusion*, but if the accumulation be large enough to form a tumor it is call a *hematoma*. (See also Infarction.)

Results.—Sudden profuse hemorrhage of any kind produces profound prostration, syncope, or shock. The individual is rendered unconscious and death may result. If not fatal, the unconsciousness is recovered from as soon as the blood-vessels, by contraction and by appropriation of fluid from the tissues, have in a measure compensated for the loss. Hemorrhages are spontaneously arrested by the decrease of blood pressure, by the retraction of the injured vessel, by the pressure of overlying structures, or by coagulation of the blood at the point of hemorrhage. A more or less profound secondary anemia may result from repeated small losses of blood.

A hemorrhagic accumulation of blood is reduced in size by absorption of the serum after coagulation has taken place. The remaining coagulum may also be more or less completely taken up by absorption, especially when it is in a serous cavity. It is sometimes replaced by the formation of new, deeply pigmented fibrous tissue. The coagulum, on the other hand, may undergo decomposition, through the action of microorganisms, and serious toxemia may follow. In other instances the clot becomes encapsulated and remains indefinitely as a harmless cyst.

Thrombosis.—Coagulation of blood within the heart or blood-vessels during life is termed thrombosis. It may occur anywhere within the chambers of the heart, in the arteries, capillaries, or veins. The coagulum is called a *thrombus*.

Causes.—The recognized causes of thrombosis are damage or removal of the endothelium lining the vessel, slowing of the blood-current, and changes in the blood which favor its coagulation. 1. Injury of the vessel-wall is the most important. It is doubtful, indeed, whether thrombosis ever occurs independently of such defect. The most common causes of such damage, aside from trauma, are fatty or other degeneration of the intima, inflammation of the vessel or of surrounding tissues, arteriosclerosis, and such dilatation as occurs in aneurism, enlargement of the heart cavities, and varicosity. Inflammation is a more frequent cause in the veins than in the arteries. The intima of the vessel may be impaired also by deficiency of nutrition, producing fatty degeneration; by foreign bodies, parasites, or neoplasms. The endocardium may be injured by inflammation, and both these membranes by the toxic agents developed in infectious diseases.

2. Slowing of the blood-stream probably does not produce thrombosis so long as the walls of the vessel are intact. It is believed that coagulation is often prevented in cases of advanced atheromatous disease, for example, by the rapidity of the current. A slowing of the current aids coagulation by favoring the preliminary accumulation of the blood-cells along the sides of the vessel and their attachment at any defective point. This tendency to stagnation of the blood may result from weakness of the heart, diminished elasticity, with either dilatation or contraction of the vessel. The circulation is normally slow in the cerebral veins and sinuses, and in the veins of the pelvis and lower extremities, especially when varicose dilatation is present. These are therefore frequent seats of coagulation. When the heart is dilated the apices of the ventricles, the space between the trabeculæ and the auricular appendages are frequent sites of thrombosis. In the veins the thrombus generally forms just back of the valves.

The thrombus is composed of superimposed layers. The first layer is known as the *primary thrombus*, subsequent layers as *secondary*. A thrombus remaining attached to the side of the vessel is a *lateral thrombus;* when it completely obstructs the vessel, it is an *obliterating thrombus*. Peculiar *ball thrombi* have been described as occurring in the heart. They are polyp-like masses attached by only a slender pedicle or lying free within the auricle.

A thrombus which has formed in blood that has almost stagnated in the vessel is dark and soft and resembles a post-mortem clot. When it forms in circulating blood it is yellow or white in color. These facts depend upon the changed circulation that results from retardation. In the normal circulation the red corpuscles and blood-plates travel through the center of the current, leaving a zone along the vessel-wall consisting of plasma and leucocytes. When the circulation becomes slow, the blood-plates leave the central zone and cling in little clumps to the vessel-wall. Leucocytes soon join the accumulation and fibrin is then formed. The *white thrombus* is, therefore, composed of blood-plates, leucocytes, and fibrin. The importance of the blood-plates in coagulation of extravasated blood, as well as that within the vessels, has been studied by Arnold and others, who regard them as the most important factor.

Results.—Collateral circulation may be established so quickly after the formation of a thrombus as to prevent serious consequences; but in organs provided with terminal arteries, and when the anastomotic circulation is poor, infarction results. Thrombi are sometimes removed. The exact process by which this is accomplished is not known, but the softening which is known to occur in them is doubtless one step in it. They may be organized. In other words, the thrombus may be replaced by new vascular connective tissue which is nourished from the vessel-wall as though it were a normal part of the body.

The other results of thrombosis are (*a*) changes in the vessel-wall, (*b*) obstruction of the circulation, and (*c*) embolism.

a. When the thrombus becomes organized, the vessel-wall is at first thickened, but it later undergoes atrophy. It may undergo suppurative softening through the action of micrococci; suppurative arteritis or phlebitis is then produced and septic infection of the system may follow.

b. Obstruction of the circulation produces results which vary with the size of the vessel, its location, the character of its anastomotic connections, and the suddenness of the obstruction. It is usually of less gravity than embolism. When an artery is obstructed, the result is a local anemia, which, if continued, leads to degenerative changes in the parts supplied. When a vein is closed, congestion and edema follow. A not unusual example of venous thrombosis is seen in the plugging of the iliac or femoral vein after typhoid fever or in the late stages of tuberculosis and other chronic diseases. In the latter conditions it is known as marasmic thrombosis.

FIG. 1.—Greatly retarded blood-stream. *a*, Axial stream. *b*, Peripheral zone with blood-plates. *c*, A collection of blood-plates. (After Eberth and Schimmelbusch.)

Embolism.—Embolism is the lodgment of any solid substance, carried by the blood, in a vessel whose lumen is too small to permit its further passage. The solid substance while passing through the vessel is called an embolus. The most frequent forms of embolus are: (*a*) A fragment of clotted blood that becomes detached from a thrombus; (*b*) vegetations from the heart cavities in endocarditis; (*c*) fragments of calcareous matter separated from atheromatous plates; (*d*) pieces of neoplasms, especially sarcoma, which have penetrated the vessel; (*e*) pigment masses, as in malaria; (*f*) air; (*g*) fluid fat; (*h*) hyalin masses produced by burns; (*i*) clumps of bacteria; and (*j*) the scolices of echinococcus or other parasites. A retrograde embolism is described in which the embolus travels in a direction opposite to the usual course of the blood, especially in the hepatic veins. It is said to sometimes occur in conditions producing increasing intrathoracic pressure, as in the paroxysms of whooping-cough. The location of the embolism depends for the most part upon the source of the embolus. An embolus from the veins and right side of the heart lodges in the lungs; rarely, it is broken up in the lung into small fragments, which pass on into the general circulation; this is true especially of fat-emboli. An embolus from the left heart and arteries passes into the general circulation and most frequently finds lodgment in the brain, kidneys, or spleen. It may lodge in other peripheral vessels where it is less productive of injury.

Results.—The immediate results depend upon the character of the embolus and the size and location of the vessel obstructed. (1) Instant death may result from the occlusion of a large vessel in the brain, a main branch of the pulmonary artery, or one of the coronary arteries of the heart. Less rapidly fatal results may follow the development of a thrombus beyond an incomplete embolism. (2) An embolism may undergo softening and be removed; it may become organized, or the development of collateral circulation may prevent the more serious consequences.

(3) Profound anemia is always produced in the area dependent upon the obstructed vessel for its blood supply, and, when collateral circulation is not promptly established, this may be followed by an infarction.

Infarction.—By infarction is meant the occlusion, by an embolus or thrombus, of a small artery having poor anastomotic connections, and the changes thus produced. It is limited, therefore, chiefly to parts supplied with terminal or end arteries, as the heart, kidney, spleen, base of the brain, and the retina. Infarcts are usually classified as hemorrhagic or red, and anemic or white. If the anastomotic circulation of the affected region is sufficiently abundant to permit a return flow of blood into the area after arrest of the circulation, the infarct appears hemorrhagic or red; otherwise the area remains anemic, has at most a yellowish or gray color, and is called a white infarction. The external zone of an infarct is always red, however, on account of hemorrhage and congestion of the surrounding tissues. White infarction occurs almost exclusively in the terminal arteries.

FIG. 2.—Edge of recent hemorrhagic infarct of lung. *a*, Interalveolar septa with engorged capillaries. *b*, Septa showing nuclei. *c*, Vein with red thrombus. *d*, Alveoli filled with coagulum. (Ziegler.)

An infarction is almost always situated in the peripheral zone of an organ. In form it resembles a cone or wedge, with the base outward. Irregular shapes are encountered when several small vessels in the same territory are simultaneously obstructed.

Changes in the Infarct.—An infarct generally undergoes degenerative changes. In the hemorrhagic form these consist in a breaking down of the blood, a removal of the fluid portion, and the growth of a deeply pigmented cicatrix to replace the destroyed tissue. In the white infarct, coagulation necrosis takes place, and this may be followed by caseation or absorption and the formation of a nonpigmented cicatrix. If microorganisms gain entrance, suppuration may be set up, and the infarction sometimes becomes the nidus of more extensive infection and the development of metastatic abscesses in more or less remote parts.

The term infarction has recently been employed, rather unfortunately, to designate the infiltration of the renal tubules or tissue spaces with blood, pigment, or the salts of the urine or bile. The most important

of these are the uric acid, calcareous, hematoidin, melanin, and bilirubin infarctions. The *uric acid* infarct is found in the kidneys of the fetus or newborn infant and in the tubules of the kidneys in adult gouty subjects. *Sodium urate* infarcts occur in the same class of adults. *Calcium* infarcts occur in the kidneys of aged persons, in destructive diseases of bone, and after poisoning with such mineral substances as mercuric chlorid, phosphorus, and bismuth. *Hematoidin* infarcts are associated with hemoglobinuria, hemoglobinemia, and methemoglobinemia following infection, poisoning, or extensive burns. The *melanin* infarct is rare, occurring in the form of casts of the renal tubules in metastasis from melanotic sarcoma. *Bilirubin* infarction is seen as casts of the collecting tubules in some cases of obstructive jaundice.

FEVER.

Elevation of the temperature of the body amounting to more than 1° C. (1.8° F.) above the normal, 37° C. (98.6° F.), is the most prominent feature of the condition known as fever. In a majority of the febrile diseases the temperature ranges between 38° C. (100.4° F.) and 40° C. (104° F.). The condition is known also as pyrexia. When the temperature rises above 40.5° C. (105° F.), the condition is termed hyperpyrexia. But elevation of the temperature is only one of many disturbances characteristic of fever. The action of the heart is accelerated, the secretion of nearly all the glands is impaired, and the processes of metabolism are deranged. The destructive processes (katabolism) are for the most part increased, while nutrition is to a great extent suspended.

The principal source of normal animal heat is the combustion of food substances involving the absorption of oxygen and the liberation of carbon dioxid; a small part of the heat is derived from the secreting glands and probably other processes. The temperature of the body in health remains almost constant, owing to the balance which is maintained by the nervous system between the production of heat and its dispersion. The chief avenues of heat dissipation are: radiation from the surface of the body, the expired air, and the excreta. It is generally believed that at least two centers are engaged in the control of temperature, one governing heat production, the other governing its dissipation.

Febrile elevation of temperature suggests overproduction of heat, underelimination, or both. The overproduction of heat represents an increased combustion, or oxidation, especially of the albuminous substances of the body, with increased discharge of carbon dioxid and nitrogenous waste, especially urea, creatin, and creatinin. The increase in elimination of nitrogen compounds usually amounts to from 70 to 100 per cent. of the normal, but may reach 300 per cent.

Causes of Fever.—1. Since the temperature is regulated by the nervous system, the simplest form of its derangement is observed in purely nervous conditions. In childhood and during convalescence from disease, owing to the instability of the nervous system, the most trifling influences are capable of causing elevation of temperature. The fever of hysteria is probably due to an interruption of nervous control, but in many cases it is difficult to exclude from its production such influences as violent muscular action or the withdrawal of peripheral circu-

lation. Either muscular or nervous exertion is capable of increasing the elevation of temperature in the course of a febrile disease.

2. Exposure to high temperature causes fever in such conditions as sunstroke or heat-prostration, after a derangement of the nervous control, or, as some believe, after toxic substances have been developed in the system as a result of the exposure.

3. The fever of the infectious diseases is believed to be caused by the action of the toxins upon the nervous system. Albumoses, peptons, various unformed ferments, as pepsin, fibrin-ferment, diastase, and many others, are capable of causing elevation of temperature when introduced into the blood, and it is probable that we have an example of their action in the fever of the autointoxications. The toxic bodies resulting from mechanical injuries of tissues sometimes produce fever.

The temperature range of the febrile diseases is generally divided into three stages: (1) the invasion; (2) the fastigium, or stage of greatest intensity, and (3) the decline. The duration of the different stages and the peculiarities in the course of each are important in giving individuality to the disease, and these features of the invasion are particularly valuable in diagnosis.

The invasion is generally announced in the acute diseases by a rigor or by chilly sensations. In children a convulsion or vomiting frequently takes the place of the chill. Benedetto de Luca looks upon the chill as a result of the altered biochemical processes for the production of heat. When the bacteria or toxins have incited these to the production of a temperature of 40° C., for example, and the body is still at 38° C., a chill occurs, just as it would if the temperature of the body were reduced to 35° C. while the mechanism was acting for the production of 38° C. During the invasion of an infection the temperature generally shows a progressive daily rise; sometimes it attains its height within the first 24 hours. In the fastigium, it pursues a more or less uniform daily fluctuation. The decline is by crisis or lysis. In crisis, the temperature drops to the normal within from 24 to 36 hours; in lysis, several days are occupied by the decline.

Results of Fever.—Fever is always attended with changes in the blood, disturbance of various functions, and wasting, but it is difficult to determine to what extent these phenomena are a result of the high temperature and how far they are due to the influences which give rise to the elevation of temperature. A moderate fever of long duration is more serious in its consequences than a much higher temperature elevation of short duration. Hyperpyrexia is always regarded as dangerous. The chief dangers are the degenerations of the heart, voluntary muscles, and nervous system.

The elevation of the temperature in fever is not looked upon as necessarily and invariably evil in its effects. It is probably in many instances an effort of nature to destroy the infection, since the vitality of many micro-organisms is impaired by increased temperature.

RETROGRADE PROCESSES.

Atrophy.—In a restricted sense, atrophy signifies a diminution of the size of an organ or tissue, without structural or chemical change. As

a rule, however, it is accompanied with degeneration; in other words, the atrophy is degenerative in character. The term aplasia is applied to local absence of development from birth or from an early period of life, and to the lack of development of the entire body which is seen in dwarfs. Hypoplasia signifies a partial development. It is frequently seen in the central nervous system, as in microcephalus and in undeveloped genitourinary organs.

A physiological atrophy occurs in the thymus gland, the Wolffian bodies, and in the fetal blood-vessels.

Causes of Atrophy.—The most frequent causes of atrophy are malnutrition, loss of function, perversion of trophic nervous influence, and excessive waste.

Malnutrition may be general or local. Defective metabolism, a failure to assimilate nutriment, is probably the most frequent form of malnutrition. We see the results of a general lack of nutrition in the atrophy of old age, when the tissues begin to lose their vigor. It is sometimes associated with anemia, marasmus, and cachectic conditions.

Local atrophy is generally the result of partial arrest of the circulation of a part, as through compression by cicatricial tissue, neoplasms, or aneurisms. It is also exemplified in the corset liver and in the wasting of bone in deformities.

Loss of function produces general atrophy, in conditions which prevent the individual from taking sufficient exercise; or local atrophy, when it is confined to only a part. The atrophy of a gland whose function has ceased, that of a muscle that is paralyzed, or of a nerve that is isolated from its center, are examples of local atrophy. We do not know, however, to what extent these atrophies are the result of a loss of trophic nervous influence and how far the result merely of interruption of function. Neuropathic atrophy is seen also in such central disturbances as progressive bulbar paralysis, in paresis, and in diseases of the anterior horns and gray matter of the cord, as polyomyelitis anterior, and progressive hemiatrophies.

The most frequent examples of atrophy from excessive waste are seen after profuse or repeated hemorrhages, suppuration, and excessive discharges of albumin or sugar. The progress of the atrophy is often slow in the latter diseases.

All tissues are not equally subject to atrophic change. In general atrophy, the adipose tissue of the body is first consumed; next the muscles; the heart and central nervous system are usually the last to be reduced.

Cloudy swelling is a minutely granular parenchymatous degeneration of the cell, through which it is enlarged and rendered opaque. Its most frequent seats are the parenchyma cells of the liver, kidneys, and other organs, and the muscle fibers. The mode of its production is not understood; by some investigators it is regarded as a precipitation of the protoplasm of the cell. In some instances, particularly in febrile diseases, it is probably due to disturbance of metabolic processes. It is always present in a tissue that is inflamed.

Causes.—Cloudy swelling is often attributed to either general or local malnutrition, but a more frequent cause, no doubt, is intoxication, either by the products of infectious organisms or through the presence

of other organic or inorganic substances. Increased cellular activity incited by abnormal nervous stimulus may produce it.

The effects upon the cell are enlargement and loss of contour; and the normal features of the cell are replaced by exceedingly fine granules which give it a cloudy appearance. Later, vacuolization may occur. The function of the affected part is impaired, but complete recovery is possible. If, however, the cause of the degeneration remains operative, the process is readily converted into fatty degeneration.

Fatty degeneration is believed to consist in a conversion of the protoplasm of the cell into fat. The fatty degeneration which occurs in the mammary and sebaceous glands in the elaboration of their secretions is physiological. Whether or not this is identical with what occurs in pathological degeneration is not known, for we know little of either process. It is even doubtful whether the fat may not be derived from a metamorphosis of such substances as sugar, glycogen, or mucin, which are also constituents of many cells, as well as from proteid matter. The fat appears at first in the form of minute granules; later, by the union of granules, larger or smaller droplets are formed. The droplets are not so large as those generally seen in fatty infiltration, a fact which is regarded as of importance in the differentiation of the two conditions, though it is not always sufficiently marked to distinguish them.

Causes.—Fatty degeneration may be a primary change, but it is more frequently secondary. It often follows cloudy swelling, less frequently coagulation necrosis, amyloid or other degenerations. In many cases it results from intoxication with the toxins of disease; less frequently that of such metallic poisons as phosphorus, arsenic, and lead. It may result also from the action of carbon dioxid, the chlorates, some of the coal-tar products, chloroform, ether, and iodoform. The other important causes are either general or local disturbances of nutrition, disturbed metabolism, alterations in then utritive supply, or partial arrest of circulation. It may be exceedingly rapid in its course, sometimes developing within a few hours in phosphorus-poisoning, or it may progress very slowly.

Fatty degeneration occurs under the most varied conditions. It is always present in leukemia, frequently in chlorosis, pernicious anemia, and tuberculosis; in the vicinity of inflammation, in tumors, exudations, thromboses, embolisms, and it may appear in many other relations. The parts most frequently affected are the parenchyma of the liver, renal tubules, heart, blood-vessels, diaphragm, and various other tissues. The affected cells become large and may rupture. When this occurs, the fat becomes disseminated and may be absorbed, liquefaction may occur, or caseation, often with the production of crystals of the fatty acids and cholesterin.

FIG. 3.—Fatty liver-cells. (Ziegler.)

Results.—Fatty degeneration is of more serious consequence than fatty infiltration, for it implies the death of the affected cell. A moderate degree of degeneration does not necessarily cause an arrest of function and possibly may be recovered from; but a cell which is in an advanced state of fatty degeneration can never be restored.

Fatty infiltration consists of a deposit of fat within the cells. It differs from fatty degeneration in that the fat is formed outside of the cell and merely replaces the protoplasm of the cell. It cannot always be distinguished from fatty degeneration. It is a physiological process in the growth and development of adipose tissue, as well as in the intestinal epithelium and liver parenchyma, after the ingestion of fatty food.

As a pathological process it is more apt to occur in regions normally containing fat, as in the subcutaneous and subserous tissues, in the bone marrow, the liver, the mesentery and omentum, under the pericardium, about the kidneys, and between the muscles. Fat may be deposited in a tissue as a substitute for a part that has been destroyed or that has undergone atrophy.

Causes.—Many persons inherit a predisposition to the accumulation of fat, and these are probably more liable than others to the pathological infiltration. The common exciting causes of it are the excessive formation of fat or a diminished oxidation. Both these influences are doubtless operative in many wasting diseases, tuberculosis, chlorosis, diabetes, and cachectic conditions. Lack of exercise is probably an important factor in its production in tuberculosis and other chronic cachexias.

Results.—The effect of fatty infiltration is an increase of the size, diminution of color, and more or less complete loss of function of the cell. But the fat may be removed and the cell may be fully restored to its normal state and function. On the other hand, continued fatty infiltration may, chiefly by compression, incite a fatty degeneration of the protoplasm of the cell.

ALBUMINOID DEGENERATIONS.

Amyloid Degeneration.—Amyloid degeneration is a process as a result of which there is found in various tissues, especially in the walls of the smaller blood-vessels, a firm, colorless, translucent substance. Whether this substance is formed by a degeneration of the tissues themselves or is merely deposited in them has not yet been determined. The amyloid substance is composed of carbon, hydrogen, nitrogen, and sulphur, and some investigators have referred its formation to a union of chondratin-sulphuric acid and a proteid, a combination which is rendered possible by the normal presence of chondratinic acid in bone, cartilage, and elastic tissue. The liver, spleen, kidneys, intestines, and lymph-glands are the most frequent locations of the degeneration, which begins generally in the middle coat of the smaller arteries, but sometimes in the trabecula of the lymph-nodes. It rarely involves connective tissue elsewhere. It may occur in the larger blood-vessels, in the heart, or in the mucous membranes of the respiratory passages. Few or many parts are simultaneously involved in different cases.

The organs affected by it are markedly enlarged and much increased in firmness. Their color is usually pale, though this may be altered by the presence of other degenerations or by pigmentation.

Causes.—It results from wasting diseases, particularly from suppuration and ulceration, more especially when these involve bone, and

yet more certainly when the disease is of tuberculous or syphilitic origin. It is rarely encountered in the absence of any of these influences, as a result of mixed infection, severe malarial infection, dysentery, leukemia, and in cachectic conditions.

Results.—A moderate degree of amyloid degeneration does not immediately jeopardize life, but recovery never occurs. The individual is always anemic and has a peculiar waxy, cachectic appearance. The local impairment of function corresponds to the degree of degeneration.

Mucoid degeneration affects the cells or intercellular tissue and produces a semifluid, translucent substance containing mucin.

Its occurrence may denote only an increased functional activity of the cells, as in catarrh; but in many instances, more truly pathological, the mucin formation appears to be entirely in the intercellular substance. It is generally found in the subcutaneous tissue in myxedema and scleroderma, and it is a common degeneration in neoplasms. It may affect also cartilage, bone, and other tissues. The cells and tissues affected may be entirely destroyed. When only the intercellular substance is involved, the cells may be destroyed as a result of compression.

Colloid degeneration is closely related to mucoid, but the substance produced is not the same. It resembles rather the colloid matter of the thyroid gland. It is usually confined to the cells and affects the intercellular substance only by inducing atrophic changes. It may involve the cell but partially, or it may entirely replace the protoplasm and cause its rupture. It occurs in goiter and neoplasms of the thyroid gland, in the kidneys and adrenals, the prostate and seminal vesicles; but very seldom in tumors elsewhere than in the thyroid. Colloid matter may be transformed into mucoid or hyalin, and mucoid matter may be converted into colloid. The colloid substance may undergo solution in the products of serous transudation, leaving cysts filled with a brownish fluid often containing blood, pus, and cholesterin crystals.

Hyalin degeneration (waxy or vitreous degeneration) resembles amyloid except in the character of the substance produced. The hyalin deposits are generally in the form of sharply defined, round or oval, rarely bottle-shaped bodies. The nature of the process is unknown. Some investigators refer to mesoblastic, epithelial, or blood hyalin, referring the origin of the hyalin matter to one or other of these tissues. It is generally found in the smaller blood-vessels in old age, following prolonged fever, or as a result of arteriosclerosis; in the brain, lymph-glands, ovaries, renal tubules, and voluntary muscles (Zenker's degeneration); in the walls of aneurisms, in the lesions of tuberculosis and syphilis, and in the retina and choroid coats of the eye. It has been observed also in leucocytes, blood-plates, and fibrin. It is probably a feature in coagulation necrosis. It is caused by infection and intoxication, especially by lead-poisoning.

Glycogenic degeneration is a condition in which clear, globular masses of glycogen are formed in cells where this substance is not present, or in abnormal quantity where its presence is normal, as in the liver, cartilage, and muscle. The glycogen bodies are closely allied to the amylaceous bodies of the prostate, and resemble amyloid bodies in appearance except that they may be concentrically striated. The degenera-

tion occurs especially in diabetes. It sometimes involves leucocytes, pus-cells, and various tumor structures of mesoblastic origin.

Dropsical infiltration is a term applied to a condition in which the cells become edematous. In ordinary dropsy the fluid accumulates in the intercellular spaces, and the cells are affected only by compression. It occurs for the most part as a result of cloudy swelling or in such conditions as burns, pemphigus, and other vesicular diseases of the skin, and probably in erythema nodosum, urticaria, herpes, and other nervous affections. The cells are much enlarged and may rupture. The protoplasm is compressed and is consequently liable to undergo fatty degeneration.

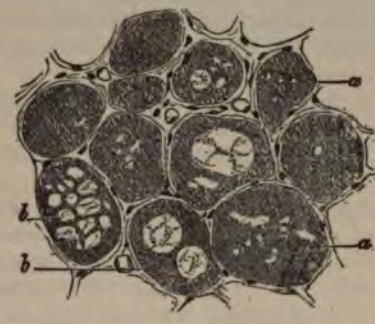

FIG. 4.—Transverse section of a bundle of muscular fibers in a state of hydropic degeneration. *a*, Muscular fibers with small drops of fluid; *b*, Fibers with large drops. (The preparation was hardened in Muller's fluid, then stained with hematoxylin, and finally mounted in Canada balsam. Magnified 66 diameters). (Ziegler.)

Calcification consists of a deposit of the earthy salts in the tissues, especially the carbonate and phosphate of lime. It is purely a passive process, an infiltration as distinguished from a degeneration. It usually occurs in tissues that are dead or in a state of advanced fatty, hyalin, or other degeneration. Deficient nutrition is an important factor in predisposing to the infiltration. In some conditions, as in old age and after extensive necrosis of bone, it is attributed by some writers to an abnormal accumulation of lime within the system. It is believed that the salts, as a rule, are simply deposited in the tissues, although it has been suggested that they, perhaps, form combinations with proteids and fatty acids. And another theory holds that soluble salts become insoluble within the tissues. In psammomata the salts are probably deposited in hyalin matter previously formed.

The infiltration usually occurs first in the form of fine granules scattered throughout the intercellular substance, but it may later invade the cells. It generally forms irregular spherical bodies showing concentric striations, but in the blood-vessels it often assumes the form of plates. It also forms incrustations around foreign bodies and in the walls of cysts. In neoplasms so great an extent of tissue may be involved as to produce large masses of mortar-like matter or large, irregular, solid concretions.

The most serious consequences of calcification are met with in the heart and blood-vessels, especially in sclerotic endocarditis of the valves and in arteriosclerosis. The aorta, coronary artery, and the vessels of the brain are common seats of calcification. As a rule it affects the middle and internal coats of the vessels. In the pericardium it follows inflammations, and, after the obliterative form, it sometimes incloses the heart in a calcareous sheath. Calcification sometimes assumes the magnitude of an almost universal invasion of the body. The nonvascular neoplasms are especially liable to calcareous infiltration,

but it is found also in sarcomata. The gall-bladder and urinary bladder, the walls of cysts, old abscesses, hematomata, thrombi, cicatrices, dead ganglion cells, dead epithelium like that of the kidney tubes, especially after mercurial poisoning, and dead parasites are often infiltrated. The occurrence of the infiltration in the form of brain-sand and as a senile change in the vessels and cartilages is not regarded as pathological.

Results.—The deposit of a small amount of calcareous matter in a region does not necessarily destroy its vitality, but complete calcification denotes the death of the tissue, and no restoration is possible.

Pigmentation consists in the formation or deposit within the tissues of substances which give them an abnormal color. There are four varieties, based upon the origin of the pigment. These are termed metabolic, hematogenous, hepatogenous, and extraneous.

1. *Metabolic* pigmentation is due to cellular activity. It is illustrated in freckles and some of the skin diseases, possibly also in the melanotic sarcoma. It occurs in Addison's disease, in some cases of diabetes, and in the anemias and cachexias. The pigment is doubtless derived from the hemoglobin of the blood, but the mode of its formation is not known. It may be deposited within the cells or between them, in the form of granules, rarely as crystals.

2. *Hematogenous* pigmentation, in which the pigment is derived from the hemoglobin, is divided into two classes: (*a*) Siderous, from iron-containing pigment, and (*b*) nonsiderous, free from iron. The chief siderous pigments are hemosiderin and its modifications; the nonsiderous pigments are various derivatives of hematin. It is possible that the latter modifications are the result of cellular activity. There are two groups of siderous pigmentation, one in which the pigments are set free in the blood, the other in which they are deposited in the tissues. In malaria, pernicious anemia, and certain infectious and septic processes, we see examples of the former; and in bruises and the diffusion of pigment from thrombi and interstitial hemorrhage, examples of the latter form.

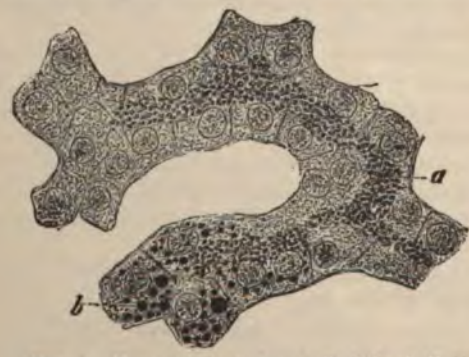

FIG. 5.—Hemosiderin in liver-cells (*a*). *b*, Fatty degeneration of cells (osmic-acid stain). (Ziegler.)

3. *Hepatogenous* pigment is derived from bilirubin or biliverdin. These are deposited in solution, granules, or crystals in almost every tissue except the brain, and especially in the liver, skin, mucous membranes, and glands.

4. *Extraneous* pigmentation occurs principally in the respiratory passages and is generally called pneumonokoniosis. It results from the inhalation of minute particles of stone (calcicosis), iron (siderosis), or coal (anthracosis). The pigments are deposited in the submucosa of the bronchi and in the fibrous tissue of

the lungs, or they may be carried to the tracheobronchial and mediastinal glands. They rarely pass into the circulation and are carried to the liver, spleen, kidneys, and elsewhere.

Argyria is a form of pigmentation which results from the ingestion of soluble silver salts and affects especially the skin, gastric and intestinal mucous membranes, the liver and kidneys.

NECROSIS.

By necrosis is meant the death of a tissue. Death of cells is termed necrobiosis; that of an entire part, gangrene. The latter term is applied also to a putrefactive change in necrotic tissues of any kind.

Causes.—The different forms of necrosis are caused: (1) By insufficient nutrition, especially by complete interruption of the blood supply; (2) by the toxic products of bacteria or chemical agents; (3) by mechanical injury; or (4) by trophic disturbances.

1. Profound local anemia, however it may have been produced, is capable of causing necrosis; venous stasis, especially that produced by mechanical obstruction or chemical agents, may be its cause. Senility, general anemia, cachexia, abnormal metabolism and its products, are among the special predisposing causes.

2. The toxic products of the bacteria are often the immediate cause of necrosis; heat, cold, the alkaloids, metallic salts, acids, alkalis, and many other substances act in the same manner, producing it either directly or indirectly by first causing various degenerations. Inflammation may lead to necrosis, and on the other hand necrosis almost invariably incites inflammation in the surrounding tissues.

3. Chief among mechanical injuries is pressure which acts directly upon the tissue, or indirectly by causing circulatory disturbance, as for example, in the pressure of neoplasms, calculi, and other concretions or exudations.

4. Trophic disturbances are not regarded by all authorities as operative in the production of necrosis. Many attribute to disturbance of nutrition or pressure a class which Stengel and others maintain are due to a disturbance of the biological mechanism of the cells. Among the examples of this character are bedsores, the skin lesions sometimes accompanying trigeminal neuritis, and various arthropathies.

Varieties.—Several distinct forms of necrosis are recognized, chief among them: (1) Coagulation necrosis, (2) liquefaction necrosis, and (3) fat necrosis.

1. **Coagulation necrosis** is a peculiar form of tissue death in which, through a process resembling coagulation, the cell contents are replaced by a hyalin-like substance. The process is supposed to be a species of fibrin-formation.

Causes.—Among the causes that are especially likely to produce this form of necrosis are the toxic effects of the pus-forming bacteria, and the bacilli of tuberculosis and diphtheria. Coagulation necrosis often occurs in the products of exudation or transudation.

Results.—A tissue that has undergone coagulation necrosis loses its function. The necrotic mass may be separated by ulceration and cast

off; it may undergo caseation, liquefaction, or suppuration, or it may be removed by absorption and its place filled with cicatricial tissue.

2. **Caseation** is a term applied to coagulation necrosis when the result is a mass resembling cheese, but less homogeneous and more granular. It occurs especially as a result of tuberculosis, but may occur in other granulomata, especially those of syphilitic origin or as a result of other processes, as in the liquefaction necrosis of the central nervous system. The results are the same as those of coagulation necrosis.

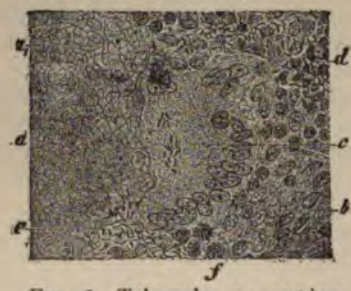

FIG. 6.—Tubercular caseation. *a*, Granular, cheesy material. *b*, Fibrocellular tissue. *c*, Degenerated giant-cells with bacilli. (Ziegler.)

3. **Liquefaction Necrosis.**—In this form of tissue death, the product is liquefied. It may occur as a primary process, or it may be secondary to inflammation, the other forms of necrosis, the degenerations, or gangrene. The necrotic mass varies in consistency with the duration of the process, and with the character of the tissue affected, and in color from white to a dark brown.

4. **Fat necrosis** is a form which affects the fatty tissues. Its occurrence is limited almost exclusively to the subperitoneal cellular tissues and the fat of the abdominal walls, and it usually results from disease of the pancreas. The necrotic areas are small, white, and soft, but frequently become gritty from deposit of lime salts. It is attributed to the action of steapsin, the pancreatic fat-splitting ferment.

5. **Gangrene** is a putrefactive necrosis. The term has generally been used to designate the death of an entire tissue or member or of extensive areas. It may be primary, but is more frequently secondary to other forms of necrosis.

Causes.—The predisposing cause of gangrene may be an injury of any kind—mechanical, chemical, electric—or an arrest of circulation in the area, as by a thrombus or embolus. The immediate cause is an invasion of the tissues by micro-organisms, for the most part by saprophytic bacteria. Several different microbes have been found capable of producing primary gangrene.

Forms.—There are two principal forms of gangrene, designated dry and moist, from the character of the necrotic tissue produced.

(*a*) *Dry gangrene* occurs most frequently as a result of arterial obstruction in regions having insufficient collateral circulation to maintain their vitality. It occurs in senility, Raynaud's disease, ergotism, thrombosis or embolism, or as a subsequent change in moist gangrene. The tissue becomes opaque and finally black; it is generally completely mummified, and may be very slow to separate.

(*b*) *Moist gangrene* is more frequently a result of the closure of a large vein as by the pressure of tumors, or cicatricial bands, or by torsion or swelling, as in intussusception and other strangulations, floating kidney, etc. In the lung it may develop after thrombosis or embolism of the pulmonary arteries or veins, bronchiectasis, abscess, or

pneumonia. It sometimes affects the extremities in diabetes, and occurs as a primary affection, probably as a result of the action of a specific bacillus, in noma. The tissue affected becomes dark brown and soft, and in most instances it is ultimately liquefied. The tissues often become emphysematous, owing to the liberation of gas by the bacteria. The necrotic mass may be separated from the surrounding healthy tissue by an area of inflammation (the line of demarcation); it is always surrounded by an area of coagulation necrosis of variable extent. The gangrenous mass is often cast off as a slough, or sphacelus; it may become encysted, or converted into a dry gangrene and undergo very slow separation. Hemorrhage sometimes follows the rupture of vessels in the surrounding tissues. A fatal toxemia is not infrequently induced.

INFLAMMATION.

Inflammation is a complex process of a degenerative, proliferative, and regenerative character, affecting the blood-vessels and tissues as a result of injury. The causes may be mechanical, bacterial, or thermal—any agent, in fact, which is capable of producing strong irritation without occasioning the complete necrosis of the tissues affected. It has been defined as the response of living tissue to injury.

The process is probably nearly or quite the same in all instances. The phenomena generally described as occurring in experimentally induced inflammation are: (1) A transitory contraction of the arteries which may be of so short duration as to escape observation. This is still referred by some investigators to the action of the vasomotor nerves, while by others it is looked upon as a result of degenerative changes in the vessel-walls. (2) A dilatation of the arteries, then of the capillaries and veins. (3) Following this, an exudation or transudation of the corpuscles and plasma with other changes to be more fully described.

The blood at first flows more rapidly, then more slowly, and finally may stop, especially in the capillaries of the central zone of the inflamed area. As the blood-current becomes slower the leucocytes in the plasmic zone of the vessels become more numerous and adhere to the sides in a row. In a capillary, a cluster of leucocytes frequently alternates in passage with clusters of red corpuscles or of red and white in normal ratio.

The migration of the leucocytes is abnormal only in the excessive numbers passing into the tissues. Large numbers of red blood-cells soon follow, accompanied by highly coagulable plasma rich in albumin. The activity of the leucocytes has been attributed to irritant substances possessing an attraction for the leucocytes (chemosis). These irritant substances are supposed to be set free by the destruction of cells in traumatic inflammations, or to be derived from the toxic substance which induces the inflammation. The escape of the red corpuscles and plasma is regarded as purely mechanical, a result of the blood pressure within the vessels.

In the connective tissue about the vessels a proliferation is set up leading to karyokinesis and the formation of small round cells (round-cell infiltration). The new cells are formed, in part at least, from the

cells of original connective tissue. The changes in the tissues are at first degenerative in character; later they become proliferative. The degenerations, cloudy swelling, fatty, mucoid, even necrosis, affect primarily the walls of the blood-vessels or the connective tissues about them. The proliferation of connective tissue produces small round formative cells, larger than leucocytes and having large, round or oval, pale nuclei, which often show karyokinesis. The same form of proliferation is sometimes seen in parenchymatous cells.

Ziegler describes, among others, the following forms of inflammation, basing the distinctions between them upon the character of the exudates belonging to each, and the changes which they subsequently undergo, rather than upon any essential difference in the process:

1. **Serous** inflammation is characterized by a fluid exudate containing comparatively few cellular elements. When affecting the skin or subcutaneous cellular tissue, it is called inflammatory œdema. It affects also the mucous membranes, serous sacs, parenchyma of the kidney and other organs. The fluid is rich in albumin and fibrin factors.

2. **Fibrinous** inflammation is characterized by immediate coagulation with the production of fibrin. It occurs especially upon serous and mucous surfaces, after desquamation of the epithelium, and forms whitish, more or less adherent membranes. It sometimes forms under the epithelium, or spreads over the epithelial covering, of adjacent areas.

3. **Hemorrhagic** inflammation is usually associated with the fibrinous form, as in fibrinous pneumonia. It differs from that form of inflammation only in the greater number of red blood-cells that are present. Hemorrhagic inflammation occurs also in the central nervous system, kidneys, lymph-glands, and skin. Hemorrhage into an inflamed area is to be excluded from this class.

FIG. 7.—Section of uvula after diphtheritic destruction of its epithelial covering. *a*, Micrococci. *b*, Mucous membrane, infiltrated and broken down. *c*, Small-celled infiltration. *d*, Fibrinous exudate.

4. **Purulent** or suppurative inflammation is generally a result of infection by pyogenic micro-organisms, but may be produced by a number of chemical irritants in the absence of bacteria, or by bacteria which are not ordinarily regarded as pyogenic, as the typhoid and colon bacilli. It is often a result of bacterial infection of an area involved in another form of inflammation. When the suppuration occurs in the midst of a tissue or organ, it constitutes an abscess; when it causes destruction of the surface of the skin or of a mucous membrane, it is an ulcer; when confined to the substance of the skin, it is a furuncle. When the micro-organisms enter the blood they produce

pyemia; when only the toxins are absorbed into the circulation, a condition of septicemia, or sepsis, is produced.

5. **Diphtheritic** inflammation occurs, not only in diphtheria infection, but as a result of other infections, or of the action of a chemical irritant for the most part upon a mucous membrane. It is characterized by a coagulation of the exudate and coagulation necrosis of the cells of the inflamed area. The exudate consists, as a rule, of coagulated fibrin inclosing degenerated cells and bacteria. The exudate of true diphtheria is distinguished by the predominance of the Klebs-Löffler bacillus, that of croupous pneumonia by the fibrin and pneumococci.

6. **Necrotic** inflammation is a form in which infection has been induced by gas-forming bacteria, producing putrid necrosis of the tissues.

Termination of Inflammation.—Inflammatory processes terminate: (*a*) By delitescence, a sudden, early cessation, with rapid restoration of integrity; (*b*) by resolution, a slower return to the normal condition; or (*c*) by the development of degenerations or necrosis.

7. **Chronic Inflammation.**—This term is used to describe, not only inflammations of long duration, due either to slow progress or a continuation of the causal irritation, but more particularly with reference to their results. When the process involves the proliferation and reproduction of connective tissues, it is sometimes spoken of as interstitial inflammation. It generally results in induration and contraction of the tissues, as in the scleroses of the liver, kidneys, and other organs. When on or near the surface, it forms bands of adhesion between adjacent structures.

REGENERATION.

Regeneration is a reparative process by which new cells and tissues are formed to replace those that have been destroyed. It may be normal or pathological in character. Normal regeneration is constantly going on in the body for the restoration of cells that have been consumed in the vital processes. So far as it is understood, normal or physiological regeneration consists in a proliferation of cells without other changes.

Pathological regeneration produces cells and tissues to replace those lost as a result of disease or injury, but the new-formed cells and tissues are not always of the same type as those which they replace. The cause and limitation of the process probably lie in the inherent tendency of cells to proliferate, but we do not know why it is developed or by what influence it is ordinarily arrested when the proper limit has been reached. Some authors regard the process as a part of inflammation, but in most instances the tissue changes are quite distinct from those of inflammation.

The new tissue consists at first of new blood-vessels, loops formed by a process of budding from the old vessels, surrounded by embryonic tissue formed in part from leucocytes and in part, by proliferation, from the connective tissue cells. Cell-proliferation alone is capable of restoring lost surface epithelium. In the regeneration of connective tissue there occur an enlargement and elongation of the original round formative cells into fibroblasts, which, together with the homogeneous

intercellular substance, undergo fibrillation, through a process of cleavage. In the regeneration of muscular tissue the original formative cells are designated sarcoblasts; in that of cartilage, chondroblasts; in that of bone, osteoblasts. The subsequent changes in the cells are not, however, identical with those in the regeneration of connective tissue. In some instances, too, the regeneration of these tissues occurs by a direct growth from the pre-existing cells of the same type; less frequently from those of another type (metaplasia).

In the repair of lesions in glandular organs, the normal tissues of the organ are to a greater or less extent reproduced, generally, however, in an atypical form, the new tissue remaining imperfect; but the greater part of the destroyed tissue, as a rule, is replaced by new fibrous connective tissue. In some instances the new, atypical formation is exuberant and results in the formation of adenomatous tissue.

THE BACTERIA OF DISEASE.

General Bacteriology.—Bacteria are the smallest, and in structure the simplest, members of the vegetable kingdom. From their resemblance to the fungi, and from the fact that they are reproduced for the most part by transverse division, they are sometimes referred to as fission-fungi, or schizomycetes. Their size is measured in micromillimeters (designated μ). They receive their nutriment by direct absorption of soluble living or dead matter; but, being devoid of chlorophyll, they are unable to decompose substances into their simpler elements suitable for absorption. From the character of the nourishment they are able to appropriate, bacteria are divided into two major classes, the saprophytes and the parasites. The former are the more numerous and live upon dead organic matter; the latter live upon or within some other living organism and receive from it their nutrition. But there are some members of each class that are able to adapt themselves to the conditions of the other; parasites that can for a time live upon dead matter, and saprophytes that can exist as parasites. These are called facultative parasites and saprophytes. The saprophytes are for the most part harmless to man; they are often, indeed, beneficial, in so far as they consume dead animal and vegetable matter.

Structure.—A bacterium consists of a cell believed to have a cell membrane, not always clearly defined, within which is a protoplasmic layer and a central fluid. No nucleus has yet been discovered. The interior of the cell is generally homogeneous, but occasionally it appears granular, as is the case with the diphtheria bacillus under the action of suitable stains. Babés named these granules metachromatic bodies, but Ernst regards them as sporagenous granules.

Morphology.—All micro-organisms fall under one of three classes when compared with reference to their form. The first class consists of the micrococci, spherical in form; the second, bacilli, rod-shaped; the third, spirilla, shaped like a spiral: and the members of each group are capable of reproducing bacteria only after their kind. The cocci multiply chiefly by transverse division; sometimes by division in two or more planes; sometimes they divide irregularly. The bacilli and spirilla multiply almost entirely by transverse division, but occasionally, perhaps,

by the formation of spores. The form of the micro-organism undergoes considerable change in the process of division. The spherical coccus, as a rule, becomes enlarged and oval, and division into two cells gives each half a more or less perfect semilunar shape. When more than one division occurs, the young cocci have the appearance of imperfect spheres; they are sometimes lanceolate or biscuit-shaped. A short bacillus produces two nearly round or square cells; sometimes, indeed, the short diameter may be distinguished with difficulty from the long.

The mature micrococci vary in diameter from 0.3μ to 3μ; those of the same species are generally of uniform size. They occur singly, in pairs (diplococci), in chains (streptococci), in groups of four (tetrads), in cubes (sarcinæ), and in irregular grape-like clusters (staphylococci).

Bacilli may be compared, when mature, to minute cylinders whose longitudinal and transverse diameters are never equal. They vary in length from 0.2μ to 30μ and in width from 0.1μ to 4μ. The largest pathogenic bacilli do not average more than 3μ in diameter. It is customary to speak of a bacillus as being slender when the ratio of its length to its width is from 4:1 to 10:1, and as thick when the ratio is about 2:1. The typical bacillus is straight, uniform in diameter, with flat ends, but many of the more slender forms are bent, as is occasionally seen in the tubercle bacillus. Others, as the bacillus of diphtheria, are not of uniform thickness, often appearing nodular or thicker at one end. The formation of spores also gives the rod an irregular outline. A beaded appearance is often seen, also, which is not due to spore-formation. Some forms, especially those endowed with the power of motion, have rounded ends. Bacilli occur singly or in chains of greater or less length; sometimes only two or three remain united.

Spirilla may be compared to segments of a spiral. They occur singly, in pairs, or as a continuous chain and have the appearance, according to their length, of a comma, an S, or a complete spiral. They may be slender or thick; dichotomously branching forms are also seen.

Sporulation.—Reproduction by the formation of spores has not been determined to the satisfaction of all investigators; but there seems to be little doubt that it is the mode of propagation in some species. Two methods of sporulation have been described. In one the spores develop within the cell (endospores); in the other they produce a sprout-like separation of the end of the cell. Spores are much more tenacious of life than is the parent cell, being more resistant to the action of many harmful agents. The young cell grows from one or other surface of the spore.

Chemical Composition.—Bacteria consist largely of an albuminous matter which has been called mycoprotein, fats, salts, and water. They contain also small quantities of extractives. Cellulose is found in some species, and a gelatinous carbohydrate, similar to hemicellulose, in others. The presence of grape-sugar in any species is denied by Cramer. Nuclein has been separated in very minute quantity, but the nuclein bases, xanthin, guanin, and adenin, are more abundant. Sulphur is found in one group. The quantities of the various substances vary so widely with the character of the culture medium upon which the bacteria are grown that estimates are of little practical value.

Vital Phenomena.—The vital phenomena of bacteria are of little im-

portance to us here, in comparison to their chemical activities. The power of motion possessed by some, the ability to produce light, heat, or coloring matter, acids, etc., interest us only as means of distinguishing different species.

Motility.—A peculiar, trembling motion may be observed with the microscope in all minute particles, whether living or dead. This is known as the Brownian movement and is in no way attributable to vitality when seen in a micro-organism. Many living bacteria, however, have a power of independent motion which can be readily seen when they are examined suspended in a drop of fluid. The movement varies from a slow, undulating, or wormlike creeping to a darting progression, so quick that it will not permit a close examination of the germ. This movement is produced by means of flagella, fine hairlike processes projecting from the sides or ends of the cell and not unlike the cilia of epithelial cells. The character and rapidity of the motion are to some extent characteristic of the species, but it depends to a great degree upon the culture medium and the temperature of the fluid in which the bacteria are suspended. Nearly all motile bacteria are attracted by certain substances, especially by pepton and urea. This attraction is known as positive chemotaxis. They are nearly all repelled by such substances as alcohol and by some of the metallic salts—negative chemotaxis. Many substances possess a variable degree of chemotaxis, positive or negative in character, for one or more species, which they do not have for others.

Chemical Action.—In chemical activity the bacteria are truly remarkable. 1. Hueppe gives us four methods by which they are able to build the chemical substances required for their own nutrition: (*a*) Polymerization, by which a simple compound appears to be doubled; (*b*) synthesis, a union of simple compounds into one or more complex substances; (*c*) the formation of anhydrids, by which new substances are formed through the abstraction of water from old ones; and (*d*) reduction or the removal of oxygen, which is accomplished by the entrance of hydrogen into the molecule. They are able also, through oxidation, hydration, or the overcoming of polymerization, to convert bodies of complex organic structure into simpler ones.

2. One of the most interesting features in the vital phenomena of micro-organisms is their behavior in the presence or absence of oxygen. To some the presence of oxygen is essential; to others it is harmful or destructive. The former group are called aërobes, the latter anaërobes. Most bacteria are facultative in this respect, but their products are not the same under the two conditions. They produce, in the presence of oxygen, profound molecular changes in the substances upon which they act, which they do not produce in its absence, and the quantity of material disintegrated is much less. The products of anaërobes in the presence of oxygen are frequently further decomposed by the aërobes and thus rendered inert.

3. *Fermentation* is a process of decomposition of organic matter: (1) By the direct action of bacteria, (2) by the substances contained in the bacteria (organized ferments), or (3) by chemical substances (chemical ferments or enzymes) produced by the bacteria and capable of acting independently of them and without loss of their own identity. Several

processes are recognized as fermentations and named by some observers which are not so regarded by others. Some authorities do not recognize as fermentation any process which is not attended with a liberation of gas, while others apply the name to all forms of decomposition through the action of bacteria, or to any process developed by a ferment.

The principal kinds of ferments are: (*a*) Proteolytic, transforming albumins into simpler, more soluble substances (liquefying gelatin); (*b*) diastatic, transforming starches into sugars; (*c*) inverting, changing nonfermentable sugars into fermentable; (*d*) emulsifying; (*e*) coagulating; (*f*) liquefying; (*g*) hydrolytic, converting urea into ammonium carbonate; (*h*) fat-splitting; (*i*) oxidizing; and (*j*) nitrifying.

4. *Ptomains.*—Many bacteria are known to produce poisonous crystalline substances known as ptomains, or putrefactive alkaloids. These bodies are a product of the action of bacteria upon dead organic matter, and although many of them are poisonous, they are to be distinguished from the toxins upon which the manifestations of most of the infectious diseases depend. The poisoning caused by eating decomposed meat, fish, cheese, or decayed vegetables, for example, is due to the presence of ptomains in the food eaten. Quite a large number of ptomains have been isolated and their chemical compositions have been determined with accuracy, especially by Vaughan and Novy. Some of them are harmless, others are extremely poisonous.

5. *Proteins.*—Buchner isolated poisonous substances which he called proteins. Koch's old tuberculin belongs to this class of bacterial products. A protein is a substance free from sulphur which is obtained by the action of potassium hydroxid upon a proteid (an albuminous constituent of an organism). Buchner found these substances were not affected by heat, but capable of producing fever and inflammation.

6. *Toxins and Toxalbumins.*—The term toxin is now generally used to designate the albuminoid products of bacteria, although there is chemically a difference between toxins and toxalbumins. A toxalbumin is an albuminoid body precipitated from bouillon cultures of the bacteria by the agents which ordinarily precipitate albumin. They are similar, both in origin and in toxicity, to ricin, the toxalbumin of the castor bean, and abrin, that of the jequirity bean. Some authors compare them also to the venom of snakes or to the enzymes; while others classify them with the albumoses or peptons. Toxin is regarded by some investigators—among them, Roux and Yersin—as a ferment of the same group as the diastatic and hydrolytic, and the toxalbumin is looked upon by them as simply an impure form of the toxin resulting from its combination with various albuminous substances in the culture medium. Brieger and Cohn have succeeded in obtaining toxins free from albumin reaction. Uschinsky claims to have obtained the albuminoid poisons of tetanus and diphtheria in culture media free from albumin, and more recently Brieger and Cohn have found that the cholera vibrio produces a nonalbuminous toxin in Uschinsky's culture media. At present the diphtheria toxin is regarded as non-albuminous.

It is believed that each species of micro-organism produces a toxin peculiar to itself, hence different in virulence, as in other attributes, from those produced by other bacteria. Outside of the body, the virulence

of any species may be increased or diminished in many ways, as by variations of temperature and culture media, which are not of interest here, except in so far as they suggest that similar changes may occur under other conditions of growth. Among the most virulent toxins are those of tetanus, of which 0.00005 milligram is capable of killing a mouse weighing 15 grams, and that of diphtheria, whose highest virulence is almost as great.

The toxins are believed to enter directly into chemical union with the body-cells. They do not merely enter the cells in the form of a solution, as is the case with mineral poisons, neither do they destroy the cell, as do some of the latter.

Many bacteria are capable of producing more than one toxic agent; some, in fact, produce a whole series of toxins. The diphtheria bacillus, for example, produces not only the diphtheria toxin, but a second, which is known as a toxon. Charrin has found that the bacillus pyocyaneus produces two classes of products, one soluble in alcohol, and affecting the nervous system; the other insoluble, slower in action, and producing varied and severe phenomena and immunity. The tetanus bacillus produces, in addition to tetanospasmin, another, which is known as tetanolysin; and the cholera spirillum produces penta- and tri-methyl endyamin, methylguanidin, and other toxins. The great diversity of the manifestations sometimes seen in disease may be accounted for in part by variations in the proportions of these toxic substances.

7. *Other Products.*—Many bacteria produce other chemical substances through their action upon various bodies. In the presence of nascent hydrogen, hydrogen sulphid is formed from albuminous matter, powdered sulphur, thiosulphates and sulphites. In the same manner, blue litmus, methylene blue, and indigo are decolorized, and nitrates are converted into nitrites and ammonia. Aromatic substances are formed by another class of bacteria. The most familiar of these are indol, skatol, phenol, and tyrosin. Under suitable conditions, fats are converted into fatty acids, and these fatty acids may, with their salts, be again converted into other fatty acids. Putrefaction, as we have seen, is due to the action of bacteria and is usually, although not always, attended with the production of malodorous gases.

For the other chemical phenomena of bacterial activity, the student is referred to works on bacteriology.

THE PATHOGENIC BACTERIA.

The micro-organisms which are capable of producing disease are designated pathogenic, in order to distinguish them from the larger class, which are harmless or nonpathogenic. The former class is constantly growing, as one after another of the bacteria supposed to be innocuous is found, under favorable conditions, to cause infection. But the specific relations of bacteria to many of the infectious diseases remain unknown.

Diseases not infrequently pursue an unusual course on account of the development of a mixed infection by the entrance and growth of more than one kind of bacteria at the same time.

Contagion.—An infected person may always become directly or indirectly a source of infection to others. Of such diseases as measles,

scarlet fever, and smallpox we know that the infectious agent may be transmitted through close contact, and we say that these diseases are contagious. There are, nevertheless, other means of transmission which are less subject to demonstration. We assume that the micro-organisms can be carried through the air to a greater or less distance. Welch has shown, however, that the importance of the air as a carrier of infection has probably been overestimated. It is only when the bacteria have been reduced to the form of dry dust, or when they adhere to such small particles, that they can be transferred by atmospheric currents. And this possibility can be assumed only of those bacteria that are capable of retaining their vitality against ordinary drying.

The importance of flies and other insects as carriers of germs, although suspected for a great many years, was not demonstrated until quite recently, and it is a remarkable fact that malaria, the disease which has been regarded as the type of air-borne disease, is now known to be transmitted nearly or quite exclusively by the mosquito. The possibilities of the transmission of contagion are so nearly unlimited, especially in view of the remarkable tenacity of life displayed by many micro-organisms, that we should not be too ready to limit the number of affections in which such transmission is possible. It is remarkable also to what extent the most virulent bacteria can be handled with impunity in the laboratory so long as actual contact is avoided.

Susceptibility.—Each infection owes its origin and most of its features to the action of a definite, specific micro-organism, but it does not follow that the entrance of a specific germ will under all circumstances produce precisely the same result. The variation in the effect is due in part to different degrees of virulence in the organism, but in great measure to difference in the susceptibility of different individuals or in the same individual at different times.

All the predisposing causes of disease that have been referred to may operate to render an individual susceptible to infection, but the most important, perhaps, are age, race, previous illness, or injury and fatigue. Some diseases are peculiar to childhood, others to adult life. Some races are immune to certain infections; the negro is much less susceptible to malaria and yellow fever than is the white man. Some diseases, notably erysipelas, rheumatism, and influenza, render a person more liable to reinfection of the same character, and others increase susceptibility to infection by other organisms. The influence of heredity has no doubt been exaggerated, but it is still believed that a tendency to such diseases as tuberculosis, gout, and some nervous affections can be transmitted. It is more difficult to explain the greater prevalence of certain diseases at one season of the year than at others, except as the conditions favor the growth of bacteria. Almquist, who has made a study of the subject, concludes that the outdoor temperature, the moisture of the air, the quality of the dwellings, and the mode of life all have a bearing upon it.

INFECTION.

Definition.—Infection is the condition produced in the body by the entrance and propagation of pathogenic bacteria.

In order to produce disease a micro-organism must be capable, not only of gaining entrance to the body, but of growing and multiplying in it. Although the virulence of different bacteria varies within wide range, a greater or less growth is required of any species to produce enough toxic matter to induce the manifestations of disease. Other influences must also be taken into consideration in the study of this subject. Park aptly says, in his work on bacteriology: "To understand at all the production of disease through bacteria we must recognize that both the body invaded and the bacteria which invade are living organisms. They are in bulk wide apart, but both have life. Just as there are different races and species of animals, there are different races and species among bacteria, and just as the descendants of one animal species under changing conditions gradually become diverse, so do the descendants of one bacterial species. Considering these facts, we can readily understand how all of the bacteria do not grow equally well in every variety of animal, nor even find the body of the same animal always equally suitable."

Micro-organisms do not normally exist within the tissues of the body. They must invariably enter from without. The usual avenue of entrance is one of the mucous membranes, especially the respiratory, the alimentary, or the genitourinary. Infection may occur, however, through the external auditory canal or, in the presence of a lesion, through the integument of any part of the body.

The body is protected in many ways from the entrance of bacteria, and it is capable, under ordinary circumstances, of destroying a certain number of those that gain entrance. It is protected: (1) By the epithelial covering of the exposed surfaces and by the presence of cilia and mucus on these surfaces; (2) by the power of the gastric juice and mucus to destroy bacteria; and (3) by the presence in the normal fluids of the body of substances antagonistic to the bacteria or neutralizing to their chemical products; (4) certain cells in the blood and tissues have the power of destroying bacteria; and (5) many germs are filtered out by the lymph-glands. Manfredi has found microbes in nearly all the lymph-glands, especially in the subcutaneous, bronchial, and mesenteric. It is therefore apparent that, in order to produce infection, a greater number of bacteria must gain entrance to the system than the system is able to destroy.

It is believed by most investigators that there must be a point of impaired integrity or of reduced vitality (locus minoris resistentiæ) in order to render infection possible; but this is disputed by others, especially with reference to the respiratory passages and alimentary canal. Even if a break of continuity or an impairment of vitality is essential, however, this may be so slight as to escape the most careful search for it, since the scratch of a pin, a puncture, or the bite of an insect is quite sufficient. The term cryptogenic infection has been applied to those instances in which the point of entrance cannot be determined.

The production of infection depends more upon the multiplication of the bacteria within the body than upon the number entering. With most species the multiplication is rapid, but only after they have found a suitable medium for their subsistence. With the exception of the

anthrax bacillus, they do not, as a rule, propagate while being conveyed through the blood.

The pathogenic bacteria show many peculiarities in their method of attacking the body. One group grows only upon a circumscribed area. The diphtheria bacillus, for example, grows upon the mucous membrane of the respiratory passages, but not in the blood or beneath the skin. The cholera vibrio grows upon the intestinal mucous membrane, but not in the blood or tissues, and the tetanus bacillus develops in wounds of the subcutaneous tissues, but not upon the surface of the skin. Another group grows most readily in certain tissues, but is capable under favorable conditions of more extensive invasion. The typhoid bacillus, for example, finds its most suitable soil in the lymph-follicles of the intestine and in the mesenteric glands, but invades also the spleen, the blood, and other tissues. Although the tubercle bacillus is capable of almost unlimited invasion of the body, it generally remains for a long time confined to a single region, as the cervical lymph-glands or the apex of one lung; and the pneumococcus, although developing most readily in the lung, sometimes invades the blood, connective tissues, and serous membranes. Some bacteria invade the system generally, while others produce isolated foci.

The local lesions produced by the bacteria depend, on the one hand, upon the character and individuality of the bacteria, and, on the other hand, upon the condition of the tissues, the soil. In many instances, as in the intestinal lesions of typhoid fever, all the several processes of degeneration, inflammation, necrosis, and regeneration occur in succession. But the same type of inflammation or degeneration is not always produced by the same organism under different conditions. The degeneration of cells and probably also the inflammation may be in some instances the direct result of the action of the bacteria upon the tissues, but it is generally due to a direct or indirect action of their chemical products and the withdrawal of nutrition. Local disturbances are sometimes an expression of disturbed innervation resulting from the action of the toxins upon the nervous system.

The general manifestations of disease depend for the most part upon the action of the toxins upon various organs and tissues, upon the nervous system, perhaps, more than upon any other. Some investigators regard the toxalbumins more important in this connection than the toxins. Their action, when artificially introduced into the body, is slower than that of the toxins, often being delayed for hours or even days. The individuality of the different infections, the peculiarities of their clinical manifestations and course, through observation of which we are able to differentiate one from another, are due chiefly to the varying action of different toxins. All under favorable conditions produce fever, but in other respects their manifestations are very different.

ANTAGONISM OF INFECTION.

The presence of bacteria or of their toxins in the body arouses an antagonism on the part of the system, one element of which is manifested in the development of antagonistic bodies called antitoxins. The result is the production of a chemical body, often in many times greater

quantity than is actually required for the neutralization of the toxin that occasioned its formation. With regard to this antagonistic action of the body there are many theories, no one of which fully accounts for all the phenomena. The more important of them may be grouped under the two heads of cellular activity and chemical activity.

Phagocytosis.—One of the most important theories of cellular action is that of phagocytosis. It is based upon the behavior of certain cells toward inanimate particles of matter, first observed by Virchow in 1840, and toward living bacteria as described by Koch, Sternberg, and Roser from 1878 to 1881, but given greater importance by the investigations of Metchnikoff, published in 1884. It was observed that these cells had the power of seizing or swallowing, and dissolving or digesting, certain bacteria. The cells were therefore named phagocytes. Metchnikoff observed further that there are two kinds of cells thus engaged—one motile, the other stationary. The former class consists of large uninuclear leucocytes known as macrophages, having much protoplasm and a prominent nucleus, sometimes lobate in form (the polymorphonuclear macrophagocyte, or eosinophile leucocyte of Ehrlich), and the smaller microphagocyte, having either several nuclei or a single nucleus in the act of splitting. The stationary or fixed phagocytes are regarded as derivatives of connective tissue, endothelium, and other tissues. The motile phagocytes, through their ameboid movements, possess not only the power of seizing the bacteria, but they are able to move for some distance toward their prey and, having seized it, to carry it through the circulation to a place of deposit, especially to the spleen. The attraction apparently exerted by a microbe for the leucocyte has been called chemotaxis. These cells have also a power of selection through which they seize by preference bacteria that are dead, or, if more than one species is present, that which will prove the less harmful to themselves. Metchnikoff demonstrated, however, that they can envelop living germs, by successfully cultivating bacteria which he had found thus enveloped.

Phagocytosis is almost universally present in the infections; and the more immune the subject of the disease, the greater is the affinity shown by the phagocytes for the bacteria.

Chemical Theory.—This theory does not replace that of phagocytosis, but rather throws additional light upon the phenomena of infection and immunity. It deals on the one hand with the production of toxins, and on the other hand with the cellular activity developed as a result of their production. As has been previously stated, the presence of microorganisms or of their toxins in the blood calls forth the development, chiefly, perhaps, by the leucocytes, of antagonistic anti-bodies to which Buchner gave the name of alexins. These are chemical in character. They have either a bactericidal action, destroying the bacteria, modifying their vital activity, or neutralizing and counteracting the toxins (cellulohumoral theory).

Ehrlich's Theory.—Ehrlich has probably offered the most plausible theory to explain the affinity that exists between the toxins and antitoxins. The theory is based upon the supposed stereochemical configuration of atomic groupings. Each living cell is assumed to possess, in addition to its nucleus, numerous side chains or arms, groups of atoms which have an affinity for some assimilable substance and act as

receptors. The normal function of the receptors is to appropriate nourishment to the cell. They combine with substances introduced as food. It is assumed that the food-stuffs for which they have the strongest affinity contain groupings similar to their own. In like manner the toxin molecule is regarded as having at least two sets of atomic groups or side chains. One of these corresponds to the food-stuffs in that it is able to unite with the receptors of definite cells. It is therefore designated a haptophore group. The other arm of the toxin contains the poisonous matter and is designated the toxophore group. It has the power of injuring or destroying protoplasm, but it can reach it only through the haptophore groups of the cells.

It is then assumed that when a toxin enters the organism or is produced within it, the haptophore groups of the molecules immediately become active and combine with the receptors of cells which have corresponding side arms. In this way the toxin replaces the food-stuff in the cell, and one of the first results is an interruption of nutrition. The toxophore group is slower to develop, hence a period of incubation usually intervenes before the toxic effects of an infection become manifest. This toxophore group is also less stable than the haptophore and may be destroyed by heat or by the action of certain chemical agents, thus converting a toxin into a harmless toxoid. The great variation in the manifestations which arise under the influence of different infections is in a measure thus explained, for all cells do not possess groupings adapted to combine with all toxins. The tetanospasmin and the diphtheria toxin find their strongest affinity in the cells of the nervous system, while tetanolysin combines most readily with the receptors of the red blood-corpuscle. Other toxins find suitable receptors in the leucocytes or in the endothelium of the blood-vessels.

The same theory has been applied to explain the formation of antitoxins by the cells of the body. When the receptors of the cells which ordinarily appropriate food have combined with the haptophores of a toxin, the function of the cell is interfered with, a process of regeneration is set up, and new receptors are formed. Assuming that Weigert's theory of regeneration is correct, the building of new receptors readily goes beyond the needs of the cell, and there is an "overcompensation." The superabundance of receptors results in their being thrown off into the circulation, and these detached or discarded receptors are believed to constitute the antitoxins. Each molecule of antitoxin possesses a side chain which is capable of uniting with a molecule of toxin and neutralizing it.

Under the names bacteriolysins, cytolysins, hemolysins, coagulins, agglutinins, etc., a great many substances have been isolated with more or less precision from the animal juices by different investigators. They exist in part normally, and are in part formed as a result of the entrance of infectious organisms or their toxins.

With reference to the place of origin of the numerous anti- bodies a great many investigations have been made. The results seem to show that what are not produced by the blood-cells, chiefly the leucocytes, are formed in those organs to which the blood-cells owe their origin, particularly in the spleen, lymphatic glands, and bone marrow. Koudriascheff has expressed the belief that the enlargement of the spleen in

infection is a result of the increased demand upon it for the manufacture of white blood-corpuscles.

IMMUNITY.

Immunity is a state of the system in which it has the power to resist the entrance of pathogenic bacteria, to prevent their propagation after entrance has been secured, or to overcome the results of their vital activity. The term is a relative one. Absolute immunity is rare, while instances of partial or temporary immunity are comparatively common.

Immunity, whether absolute or partial, may be inherited (natural) or acquired. It is possible that every individual possesses some degree of natural immunity against almost every infection. He has at least the power of preventing to some extent the entrance of infection, and it is a well-established fact in experimental infection that a certain number of bacteria are destroyed after their introduction into the body. It is only after this power of resistance has been overcome that infection occurs.

Natural immunity is sometimes a racial property, or it may be peculiar only to the individual. Racial immunity is seen in the almost complete resistance of the negro to yellow fever and malaria, and in the freedom of the Japanese from scarlatina. Barlow has expressively defined natural immunity as "the inheritance of acquired characteristics."

Individual immunity is closely allied to personal idiosyncrasy or to that unaccountable quality which renders some persons immune to the venom of snakes.

Acquired immunity is often the result of accident, as that which is sometimes conferred by a previous attack of the same infection. The first attack of many diseases leaves the individual immune from further attacks. This is always true of yellow fever and generally true of typhoid fever and the exanthemata. In typhoid fever, however, the immunity is probably not developed until some time after convalescence, for a full relapse sometimes occurs several weeks after convalescence. Another class of diseases confers immunity from a slightly different infection.

Immunity may be artificially induced (*a*) by the introduction of another infection, as in vaccination, (*b*) by the injection of cultures of a specific microbe which have been reduced in virulence or of cultures from which the living organisms have been removed or destroyed by heat or other means, or (*c*) by the injection of the serum of animals that have been rendered immune. Artificial immunity is not as a rule so lasting as that which is inherited or as that acquired from a previous attack of the same disease.

The mechanism of immunity has been variously explained. It is probable that the process is not the same in all instances and that no single theory will ever account for its many features. The fact often seems to be overlooked that an individual may be immune to the action of a microbe, but not to the toxin which it produces, and vice versa. Buchner, as already stated, attributes it to the presence of alexins in the body. Another explanation attributes the inhibitory influence of the serum to its power to produce a change in the vital properties of the

bacteria, but this has been shown not to be universally true. The theory of phagocytosis attributes immunity to the power of the cells to destroy the bacteria. This has been somewhat modified in the cellulohumeral theory, which attributes to the cells the power of producing chemical substances destructive of the bacteria or capable of overcoming or counteracting their action. The phagocyte is believed not only to envelop the microbe, but to produce a chemical substance for its destruction or to neutralize the toxin. Metchnikoff speaks of the "complicated interplay of the biological and chemical functions of living cells." Manfredi has shown as a result of experiments that the lymph-glands act in the capacity of filters for the removal and retention of bacteria, and that a specific, latent microbism is developed in these glands by virtue of which the body is rendered immune to the diseases. In other words, the existence of a latent infection in these glands so affects the general system as to prevent reinfection by the entrance of organisms of the same species. F. A. Packard, in support of this theory, directs attention to the fact that evidence of former tuberculous involvement of the superficial lymphatic glands is rarely found in cases of pulmonary tuberculosis.

The early theory of Chauveau, that the cells of the body become accustomed to the poison, is still recognized by many as an accessory feature in general immunity.

Ehrlich has applied his theory of chemical affinity to the explanation of both natural and acquired immunity. In natural immunity the cells of the animal organism do not furnish side chains capable of forming a chemical union with the toxin. Acquired immunity is due to the overproduction of receptors by the cells of the body. Those which are produced in excess of the requirements of meeting and counteracting the toxins present are thrown off into the circulation and remain ready to combine with any toxin having the same affinity that may thereafter be formed or gain entrance to the body.

As a result of the studies of immunity, artificial antitoxins have been produced with more or less success. The best known examples of these are the antitoxins of diphtheria, tetanus, bubonic plague, and streptococcus infection.

It has been shown further that the antitoxin may disappear from the blood without necessary loss of immunity, and, on the other hand, that death may occur, doubtless as a result of extreme susceptibility, while the blood is saturated with antitoxin. It should be borne in mind also in this connection that an antitoxin is merely an antagonistic or neutralizing agent, and in no sense one possessing powers of regeneration. While it assists the body to resist and to counteract infection, it does not directly assist in the processes of repair that are necessary for restoration to health.

PART II.

PRACTICAL MEDICINE.

SECTION I.

The Infectious Diseases.

TYPHOID FEVER.

ENTERIC FEVER, ABDOMINAL TYPHUS, EXANTHEMATIC TYPHUS, AUTUMNAL FEVER.

Typhoid fever occurs endemically or epidemically in all parts of the world. No climate, no locality, is exempt from it, but in some regions its virulence is greater than in others. In the United States it prevails with greater severity along the Atlantic and Gulf coasts and on the shores of the Great Lakes. In Canada and on the Rocky Mountain plateaux it usually assumes a mild form. It was first introduced into Puerto Rico and Hawaii by our armies in 1898. The name Typhoid is derived from τῦφος, cloud or smoke, and refers to the obscuring of the mind incident to the disease.

Definition.—An infectious disease of about four weeks' duration, caused by the bacillus typhosus, characterized pathologically by lesions in the lymph-follicles of the intestine, the mesenteric glands, spleen, and other organs, and clinically by a continuous fever, usually attended with asthenia, great mental depression, diarrhea, a rose-colored rash, and a tendency to intestinal hemorrhage.

Etiology.—The specific cause of the disease is the bacillus of Eberth. The avenue of invasion is believed to be, in most cases, the alimentary canal. The bacilli enter with the food or drink, escape the destructive action of the gastric juice, and pass through the intestine until they reach the lymph-follicles, where they find conditions suitable for their propagation. From the follicles they enter the lymph-vessels and thence pass into the blood, to be disseminated throughout the system. Their poison, typhotoxin, is regarded as an ingredient rather than a product of the bacilli, since filtered cultures are almost free from it.

Age.—Youth and early adult life are the periods of greatest susceptibility. More than half the cases occur between the fifteenth and thirtieth years. The disease may be encountered, however, from early infancy to extreme old age. It may be congenital when the mother becomes infected during the last months of pregnancy and communicates it to the fetus. The intestinal lesions have been found in premature infants and in those dying in the first weeks of life. A few cases have been reported in persons over 80 years old. The greatest mortality has been between 20 and 30.

Sex.—Hospital records, as a rule, include a much greater number of men than women, but under equal exposure the sexes are about equally attacked. Pregnancy and lactation are thought to afford partial protection to women.

Susceptibility.—Some persons and some families appear to be less susceptible than others, but absolute immunity is at least rare. The disease affects almost equally people in all walks of life. If robust workingmen are oftener attacked, as has been suggested, it is probably on account of greater exposure and less regard of prophylaxis. Early settlers and the armies of invasion are especially prone to the infection, but this is doubtless due to the greater liability of their uncertain water supply to contamination. One attack generally protects from future infection. Such chronic affections as phthisis and heart disease are believed by some authors to partially protect. Bad ventilation, overcrowding, intemperance, and mental and physical fatigue predispose, if at all, by increasing susceptibility. Gross errors in diet, and filth, favor infection. Intestinal catarrh involving desquamation of epithelium is regarded by some authors as essential to infection by establishing a nidus for the growth of the bacilli, but others regard this as of little importance.

Season.—Autumn is the season of its greatest prevalence, hence the name autumnal fever; but in many localities, especially in the great cities, it occurs in sporadic form the year round. That it is most prevalent after a hot, dry summer, a belief that has been handed down for many years, is not confirmed by all authorities. Drought probably has its influence in favoring the concentration of the infectious agent in the depleted streams and springs and in favoring the draining of contaminated foci.

Water and Food Contamination.—Typhoid fever is typically a water-borne disease. Many of the most notable epidemics have arisen from the pollution of the water-supply of a town with the dejections of a single case, and it is in most instances owing to similar contamination of the drinking-water that it is so prevalent in armies and new settlements. The disease is carried with the invaders, and the contagium reaches the water-supply through careless disposal of the stools from the infected persons of the party. A notable illustration usually referred to was the epidemic of Maidstone, England, in 1897. Over 1,900 cases occurred among 35,000 inhabitants, and in over 95 per cent of the cases the infection was traced to a definite contamination of the water-supply. Defective drainage is ordinarily the most prolific source of water contamination, but the germs are probably never carried in the vapor arising from sewers, however contaminated it may be in other respects. The bacilli have been carried in ice. Milk becomes a source of infection through the addition of polluted water, or by the use of such water to cleanse the vessels in which it is stored. Shellfish, especially oysters, have in several instances been the source of widespread infection, and the bacilli have been repeatedly found in them.

Contagion.—The danger of contagion by direct contact is very slight, except to the nurse and the laundress who wait upon the patient and wash his linen. Germs are probably seldom or never conveyed by the air, although they resist ordinary drying. It is evident that the hands of the nurse, the thermometer, and utensils used by the patient if by any possible means contaminated with the feces or urine, may convey the bacilli, unless thoroughly sterilized; and it has been sufficiently demonstrated that flies may serve as carriers of the virus.

Bacteriology.—The bacillus is usually a rather short rod, measuring about 2.5μ in length, or one-third the diameter of the red blood-corpuscle, and about a third as wide as it is long. It is sometimes longer and more slender, and frequently forms chains of considerable length. It resists freezing and a temperature of 156° F. (69.0° C.), but is killed by complete drying, exposure to bright sunlight for two hours, or to the air for a longer period. In form it is not unlike the colon bacillus, but it is actively motile, having long flagella. It is not known to form spores, but sometimes shows granules at the poles. Its growth within the body is remarkably rapid. Great numbers of bacilli are found between the cells of the lymph-follicles in the intestine and in smaller foci within the mesenteric lymphatic glands, in the spleen, liver, kidneys, pleuræ, and meninges. They are found also in blood drawn from the rose-spots of the skin, and in a few instances have been obtained in that drawn from the general circulation; sometimes in the bone marrow and other tissues. Bacilli have even been found in the bile, sweat, sputum, and tears; in the milk of pregnant women, and for months after recovery in abscesses and diseased joints. The stools, and in some cases the urine, contain large numbers of them after the tenth or twelfth day of the disease. They disappear from the stools, as a rule, about two weeks after the fever ceases, but they have been found in the urine several months later. From the frequent discovery of the typhoid bacillus alone in foci of suppuration, Golgi and others attribute pyogenic properties to them.

Many unsuccessful attempts have been made to produce the disease by artificial inoculation; but most of the lower animals have proved to be resistant. The symptoms produced by injection of bacilli into their blood or their peritoneal cavities are attributed to the toxin accompanying the injection. The bacilli are generally quickly destroyed.

The bacilli probably do not have a permanent independent existence outside of the human body, but they are capable of retaining vitality for weeks and months in the outer world, under favorable conditions, especially in moist earth and in water. It has been demonstrated that they can resist repeated freezing and thawing and retain life for 25 days in fresh water or in sterile sea-water, and for a much longer time in unsterilized fecal matter. A few authorities, led by Pettenkofer, regard the ground-soil as the normal place of development, but the vast majority of investigators maintain that the original source of infection is always a previous case of the disease.

Types of Infection.—Chiari and Kraus, Hodenpyl, Flexner, and others, by a series of investigations, have been led to the belief that the disease may be divided into four groups corresponding to as many forms of infection. These are: (1) Ordinary typhoid fever with marked intestinal lesions, a group to which a great majority of the cases belong. (2) Typhoid septicemia, a general infection without special local manifestations. (3) Typhoid fever with lesions elsewhere than in the intestine, a form in which the disease is characterized by typical clinical manifestations, and the diagnosis may generally be confirmed by the demonstration of the bacilli in the blood or other fluids of the body and by the Widal test. The intestinal lesions are very slight or entirely absent, the principal pathological changes being found in the lungs,

spleen, kidneys, or the cerebrospinal meninges. (4) Mixed infections. This term is restricted in its application to cases of secondary infection of the typhoid patient with such organisms as the colon bacillus, the streptococcus, staphylococcus, or pneumococcus. Cases of double infection, as with the organisms of tuberculosis, diphtheria, or malaria, are to be excluded from the group, since, in such cases, two diseases coexist.

Morbid Anatomy.—The specific lesions of typhoid fever are found in the lymphatic system, especially in the solitary and agminated follicles of the intestine, the mesenteric glands, and the spleen.

The Intestines.—The changes may involve the follicles in any part from the jejunum to the lower end of the ileum, or in the cecum and colon, but they are generally confined to the lower part of the ileum. They may be divided into four kinds, which in a measure correspond in the time of their occurrence to the clinical division into four weeks. It should be borne in mind, however, that the weeks represent somewhat indefinite periods of time, often slightly more or less than seven days. The first week is characterized in the follicles by congestion and proliferation, the second by degeneration and necrosis, the third by ulceration, and the fourth by cicatrization.

1. The *congestion* consists in an accumulation of multinuclear leucocytes, blood-plasma, and occasionally some red blood-cells. The proliferation occurs chiefly in the endothelium of the follicles and small lymph channels. The young cells are abnormally large and speedily undergo degeneration. The entire follicle becomes swollen and unduly prominent. Any part or all of the agminated follicles may be involved. The infiltration may involve also the submucosa and muscular coat.

2. The processes of *degeneration* are induced by the irritant action of the bacilli, present in large numbers, and are favored by anemia due to occlusion of blood-vessels by pressure or thrombosis. A slough is formed, which may be so superficial as to scarcely destroy the epithelium or so deep as to include the muscular layer.

3. The *ulceration* corresponds in location and extent to the follicular degeneration, but sometimes attains great size by the coalescence of the process in two or more Peyer's patches. It does not ordinarily penetrate deeper than to the submucosa, but it may extend through to the peritoneal covering and lead to perforation of the bowel under the force of gaseous distention. The edges of the ulcer are generally much elevated, soft, and overhanging; the floor is usually smooth.

4. Cicatrization is accomplished by the formation of new fibrous connective tissue.

The spleen is much enlarged and engorged with blood; it is at first firm, but later soft, almost diffluent. It contains clumps of bacilli, and not infrequently infarctions. Spontaneous rupture has rarely occurred.

Other Organs.—Catarrhal enteritis of variable extent is usually present, the liver is generally enlarged, the cells granular from degenerative changes and crowded with fat. It frequently contains clusters of bacilli and lymphoid and necrotic nodules which are sometimes converted into abscesses. Acute yellow atrophy has been noted. The mucous membrane of the gall-bladder is so uniformly affected as to virtually constitute a culture soil for the bacillus. The epithelium of the renal tubules shows cloudy swelling, and there are generally clumps of bacilli or of

round-cell infiltration which sometimes break down and form miliary abscesses. The latter lesions were described by E. Wagner as lymphomatous formations. Acute nephritis rarely occurs. Pyelitis and cystitis are occasionally found. The pancreas sometimes shows proliferation of cells, with cloudy swelling and fatty degeneration. Bacilli have been found in it. Hypostatic congestion and edema of the lungs, pharyngitis and laryngitis, sometimes of an ulcerative form, may be found. Myocarditis occasionally develops, less frequently pericarditis or endocarditis. In the latter condition, bacilli are found in the valvular vegetations. Peritonitis seldom occurs in the absence of perforation. It very rarely proves to be a conservative process, causing closure of the opening without more extensive involvement of the peritoneum. Parenchymatous degeneration of the muscles, thrombosis of the femoral veins, meningitis, and peripheral neuritis are sometimes met with. Among the less frequent lesions are suppurative parotitis, otitis, infarction or gangrene of the lungs, edema of the glottis, and gastric or esophageal ulceration.

The Blood.—The bacilli have been repeatedly discovered in the blood, reaching it from the lymph-vessels of the mesentery, in all probability through the thoracic duct, where they have been found in much greater numbers than in the blood. Pyogenic organisms may also be discovered. In other respects the blood-changes correspond to those of secondary anemia; the red corpuscles are reduced in number and their hemoglobin is diminished in a still greater proportion, giving the disks a pale appearance. The leucocytes are not increased until convalescence begins, except in rare, complicated cases. The absence of leucocytosis is a distinctive feature of the disease. The osmotic tension of the blood is remarkably constant; the freezing-point remaining within the narrow limit of —0.55° and —0.57° C.

Symptoms.—*General Course.*—In a typical case the period of incubation is generally marked by a feeling of lassitude, a disinclination to work, or undue fatigue after slight exertion, with headache, vertigo, possibly a slight nose-bleed, a furred tongue, loss of appetite, nausea, aching pains in the back and legs, sometimes colicky pains in the abdomen. Chilly sensations are usually felt; a distinct rigor is very exceptional. The feeling of illness is more pronounced toward evening and increases with each succeeding day. The bowels are generally constipated. The incubation lasts from 7 to 14 days as a rule, but may be much shorter or it may continue as long as 21 days. Some cases are characterized by a rather severe prodromal stage of only three or four days' duration. The manifestations of illness depend largely upon the temperament of the patient. Some persons are greatly distressed, while others only realize that they have not been altogether well for two or three weeks, after the more severe symptoms have developed.

First Week.—The period of invasion, or first week of the disease, begins with the elevation of temperature; but as the patient frequently does not consult a physician at this early period, the exact time of the onset is often unknown. It that case the beginning of the disease is generally reckoned from the day on which the patient first remained in bed. The chilly sensations now give place to feverishness; the weakness increases,

but the aching pains subside after a few days' rest. The headache becomes more severe and the abdominal pains may perhaps continue. Slight deafness is often noticeable. The patient becomes nervous and irritable. His sleep is restless and broken by dreams. The temperature gradually rises, each evening showing from 1° to 1.5° F. greater elevation than the preceding, until 103° or 104° F. (39.5°—40° C.) is reached. The pulse is rapid, generally from 100 to 110; it is full, but its tension is low; sometimes it becomes dicrotic. The tongue is coated with a white or yellowish fur, except at the tip, which may be unnaturally red. It is often tremulous. The papillæ may be prominent. Nausea sometimes continues, vomiting is occasionally troublesome, and there is no appetite. The lips become dry, and thirst is complained of. Sore-throat, slight pharyngitis or laryngitis, and bronchial catarrh are not infrequent accompaniments. Nose-bleed sometimes first makes its appearance in this stage; it is generally slight, only a few drops of blood being lost. The face is pale, with tinges of red upon the cheeks; the pupils are generally dilated. Diarrhea may develop spontaneously, but is often induced even by an unusually mild purgative. In some cases, however, diarrhea is absent throughout the entire course of the disease, and there may be an obstinate constipation. The urine is diminished in quantity, high in color and specific gravity; the urea is increased, chlorides diminished, and traces of albumin are sometimes found, as in other febrile diseases. The abdomen generally becomes distended and somewhat tender, especially in the right iliac fossa. Gurgling is frequently detected by palpation in this region, especially if diarrhea be present. Enlargement of the spleen usually becomes recognizable by the close of the first week.

Second Week.—The symptoms become aggravated. The temperature continues high and the range is uniform throughout the week, with but slight morning remission. The pulse, still rapid, even reaching 120, is not generally dicrotic, although it may have been so in the first week; but its volume is diminished. The heart's action is weak and the first sound indistinct. The headache generally subsides, but the deafness becomes more marked and the eyes are sensitive to light. The nervous restlessness of the first week gives place to sopor, but although the patient sleeps much of the time, his rest is not refreshing. He lies on his back with his eyes half open, his arms at his sides, a picture of extreme weakness. He often slips down toward the foot of the bed. When aroused, he is apathetic, utterly indifferent to his surroundings, and rarely expresses a desire. Delirium of a wandering or muttering character, accompanied with muscular twitchings, often supervenes, especially at night. The face has a dull, listless expression and sometimes a dusky hue; the cheeks become more flushed, but the pallor and emaciation are more pronounced. The lips are dry, the tongue brown and parched, and tremulous when protruded. The abdominal distention (meteorism) increases and the diarrhea continues; there are usually three or four stools a day, generally of a yellowish brown color and of "pea-soup" consistence. Sometimes they are lumpy and of a pale buff color. Rose-colored spots usually appear by the eighth day of the disease, first on the abdomen or on the sides of the thorax and in the lumbar region. There may be only three or four, or

a dozen, round, slightly elevated, bright red maculæ about an eighth of an inch (3 mm.) in diameter. At first they disappear on pressure, but immediately return. Successive groups appear for about ten days, each group remaining for two or three days. They are sometimes so numerous, however, as to resemble the eruption of measles, appearing on the arms and legs as well as on the trunk. The disease may terminate fatally toward the end of the week, with profound nervous manifestations or as a result of hemorrhage or perforation.

Third Week.—In the very mild cases the temperature begins to show greater remissions during the night, but in most cases the symptoms are the same as in the second week or more severe. In the more aggravated cases the temperature remains high, the pulse is still rapid and weak, sometimes running, and the emaciation becomes profound. The nervous manifestations are also prominent. The patient is more apathetic, often delirious or semicomatose, especially at night. During the day he spends much of his time in muttering and picking at imaginary objects on the bed or in the air. Subsultus tendinum (a jerking of the tendons of the wrist and fingers) is often pronounced. Owing to the extreme weakness and emaciation, or to dryness of the nostrils, the mouth often remains open, the tongue becomes hard and fissured and sordes appear on the teeth. Bedsores are apt to form. Diarrhea and meteorism may make their appearance during the third week. Retention of urine and incontinence of feces are at times troublesome symptoms. The stools may be tinged with blood, or profuse hemorrhages may occur. The hemorrhage is sometimes first announced by a sudden gush of blood from the rectum, which leaves the patient in a state of collapse, pulseless, and bathed in cold, clammy sweat, with temperature normal or subnormal. Albuminuria frequently appears in this week of the disease, or, if present in the preceding week, it becomes more abundant. By this time, also, hypostatic congestion of the lungs or even pneumonia may develop.

Fourth Week.—This is the period of beginning convalescence in most cases. The improvement may be delayed, however, into the fifth or even the sixth week, rarely longer, when complications arise. From the middle to the end of the week the temperature generally reaches the normal in the morning and does not exceed 101° or 102° F. (38.8° C.) in the evening, and by the end of the week the evening temperature is generally normal. Slight evening elevations sometimes persist into the fifth week. The pulse, as the fever subsides, becomes stronger and there is general evidence of improvement. The symptoms all begin to abate; the abdominal tension disappears and the diarrhea ceases; the tongue becomes moist and the sordes disappear from the teeth; the appetite returns, the sleep becomes normal, and the patient awakes refreshed, with a clear mind. But relapse or reinfection may occur after several days or a week of normal temperature and apparent convalescence.

Convalescence usually begins in the fourth week, occasionally earlier in mild cases. It may be delayed, however, until the fifth or sixth week. The chief danger in the latter cases lies in the weakness of the heart. This is a period, too, in which complications and sequelæ are most likely to arise. The patient first recognizes his weakness after the fever has subsided. But although he is for a time unable to raise himself

in bed, an effort which is fraught with danger on account of the liability to heart failure and the possibility of rupturing a degenerated muscle, his strength soon begins to return. By the end of a week his desire to leave the bed often becomes so strong as to require a careful watch to be kept, and his appetite demands a supply of food that is altogether unsafe. A relapse is often brought on by yielding to either of these inclinations. Other prominent features of the convalescent period are the anemia, which persists for several weeks; the falling of the hair, which sometimes amounts to alopecia; and in some cases profuse sweating, especially during sleep. From four to six weeks, sometimes several months, and in the worst cases a year may be required for full restoration of strength. The mental faculties are generally early restored, but in other cases weeks or months elapse before the patient becomes fully normal in this respect.

Special Types.—No other disease presents so great diversity of clinical manifestations, particularly with reference to severity.

In *mild cases*, the general features are the same, but all the symptoms are less severe. The patient is frequently so little indisposed that he yields to confinement with reluctance. In this, however, lies the greatest danger, for the intestinal lesions and the danger of hemorrhage or perforation are often just as great as in the more characteristic cases. Under supervision, however, these cases are rarely attended with hemorrhage, delirium, or other serious symptoms.

In the so-called *abortive form*, the onset of the disease may be unusually severe. The onset is sometimes announced by a chill and early rise of temperature to 103° F. (39.5° C.) or higher. The rose-spots may be found as early as the second to the fifth day, and they may be numerous. The fever pursues a uniform course, but begins to decline by the end of the first week or beginning of the second. In fact, the case has the appearance of having passed unrecognized during the first week. The abortive form appears to occur more frequently in Europe than in this country.

The Severe or Fulminant Form.—The disease pursues a course of unusual severity from the beginning, especially in those cases in which the manifestations on the part of the nervous system are most pronounced. The headache is extreme; delirium may develop early, and in it the patient may for days wander about the streets. In some cases the predominance of such symptoms as headache, photophobia, and retraction of the head leads to a suspicion of cerebrospinal meningitis, or the drowsiness and stupor, especially in children, suggest a basilar meningitis; and in yet another class of cases such special features as neuralgia, acute bronchitis, pleuritic pain, persistent vomiting and diarrhea, or acute nephritis prevent the recognition of the true character of the disease.

Ambulatory Form.—Another class of cases belongs to the ambulatory or walking type of the disease. The patient often persists in his usual occupation day after day until he is suddenly stricken down, it may be late in the second week, with delirium, a temperature of 104° or 105° F. (40—40.5° C.) hemorrhage, or the pain occasioned by perforation.

An *afebrile* type has been described, but extremely few cases have

been seen in this country. The course of the disease is the same as in the ordinary form, except that the temperature remains normal. The characteristic lesions have been found after death.

Other forms of the disease have been named and described, but the deviation of type in most of them has been due to the development of complications.

In children the disease generally pursues a mild course, but the rise of temperature may be more abrupt, and it is often high in the beginning. Epistaxis seldom occurs and diarrhea is often absent. An initial bronchitis sometimes occurs. The nervous manifestations, especially wakefulness and delirium, may be severe. The rash is usually slight, but it may be so profuse as to resemble measles. Hemorrhage and perforation rarely occur, but their possibility should not be disregarded. The mortality has been greatest in those rare cases that occur in the first year of life.

In the Aged.—After the fortieth year the disease generally runs a mild course, but the mortality is greatly increased by the common occurrence of complications on the part of the heart and lungs.

In Pregnant Women.—The disease is rare in pregnancy. It usually occurs during the first half of the period. The mortality is but little above the normal. Abortion or premature delivery generally occurs during the second week.

Special Symptoms and Complications.—*The Facial Aspect.*—In the beginning of the disease the cheeks are flushed and the eyes often have an increased luster, but by the end of the first week the countenance loses its animation and the eyes become expressionless. In the height of the fever the expression becomes dull and heavy, and the face has sometimes a dusky color. The flush does not usually disappear until the fever has subsided, and then for the first time anemia becomes apparent. The emaciation is progressive throughout the disease.

Temperature.—The typical temperature curve of typhoid fever is one in which, during the period of invasion, or first week of the disease, the elevation is from 1° to 1.8° F. (0.5—1.0° C.) higher than at the same time on the day previous, until an evening temperature of 103° or 104° F. (39.5—40° C.) is reached, about the fifth or sixth day. It then pursues a uniform course during the second week or fastigium, the fluctuation from morning to night being little more than occurs in health. During the third week it becomes remittent; there is often a difference of 3° F. or more between the morning and evening records, and toward the end of the week the evening temperature usually becomes normal; that of the morning may be slightly subnormal for a few days. But a perfectly typical chart is unusual. It is modified in many ways; as a result of rest, treatment, and complications, or unrecognizable influences.

The height of the temperature at the end of the first week is generally an index to the severity of the attack, but it may prove a fallacious guide. The principal variations from the usual range are: (1) An abrupt rise at the beginning of the first week, especially in children and in cases beginning with a chill or convulsion. In children the fever often pursues a strictly remittent course. (2) In a mild or abortive form the temperature begins to decline early in the second week, and often

by a lysis which is unusually rapid. Very rarely a crisis occurs, the temperature reaching the normal within from 12 to 24 hours. (3) Hyperpyrexia may occur in which the temperature reaches 106° to 108°

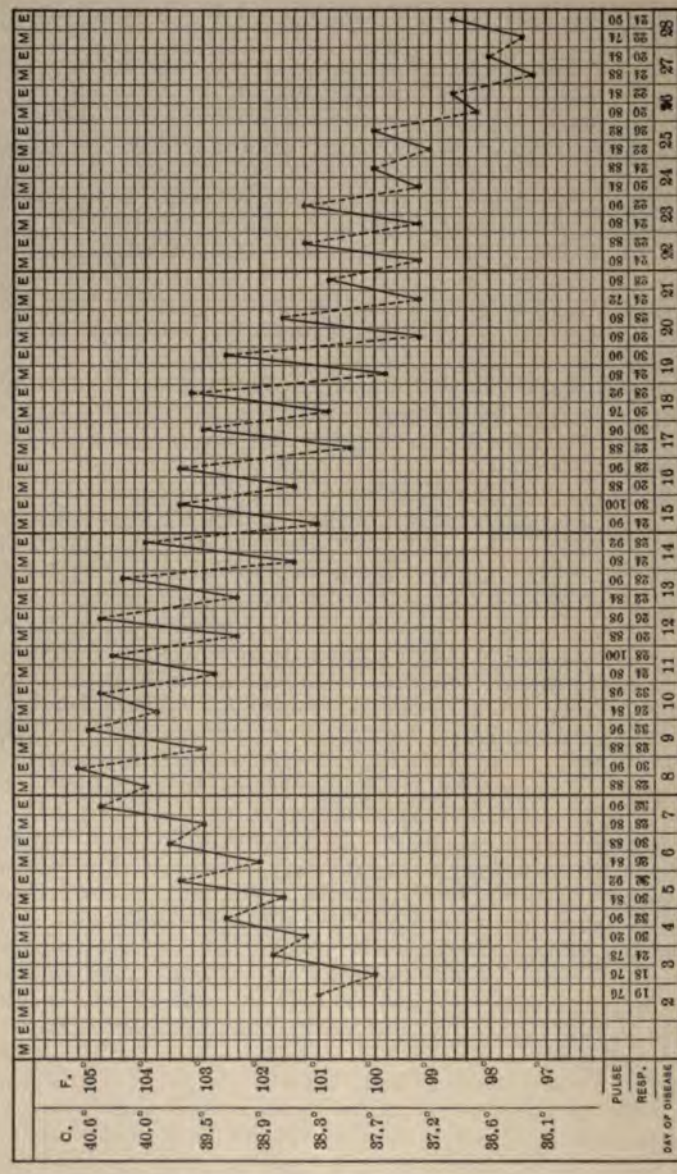

FIG. 8.—Temperature chart of a case of typhoid fever, showing the daily fluctuations of temperature. The dotted lines represent the decline during the hours of the night.

F. (41.1°—42.2° C.) in the evenings. Such cases are usually fatal. (4) A chill sometimes occurs during the second or third week, and the temperature is elevated two or three degrees above its former course,

especially when malarial infection complicates the case. (5) Recrudescence sometimes occurs; the temperature, after running a mild course for a few days, if not from the beginning, suddenly becomes elevated. This may occur during convalescence, when it is usually a result of errors in diet, constipation, or nervous excitement. (6) Hemorrhage of the bowel causes a sudden drop of temperature to the normal or below it, although the blood is sometimes retained within the bowel and the hemorrhage is said to be concealed. Perforation is followed by a similar decline. (7) Sometimes the fever continues at its former range or pursues a more or less irregular course for six or eight weeks. Such cases arouse suspicion of sepsis or tuberculosis, unless the diagnosis has been confirmed by bacteriological and blood tests. (8) Occasionally the temperature curve is reversed, the morning record being higher than the evening.

With the exception of hyperpyrexia and a sudden fall of temperature, these deviations do not necessarily indicate danger in the absence of other grave symptoms. The persistence of fever after all symptoms on the part of the alimentary canal have disappeared is often a nervous manifestation. The fever of relapse frequently pursues a course similar to that of the original disease, showing a gradual rise, a few days of fluctuation, and a decline. It is usually shorter than the original pyrexia.

Chills occurring during the course of the disease are often indicative of a complication, notably hemorrhage, sepsis, malaria, pneumonia, or thrombosis. They may be produced also by the administration of antipyretics or by constipation, but they cannot always be accounted for.

Circulatory System.—*The Blood.*—Typhoid bacilli can be found in the blood by cultivation on suitable media. At the end of the disease other pathogenic germs may also be found in such cultures. The coagulability of the blood and the fibrin-formation are normal until the approach of defervescence, and the specific gravity varies only with the hemoglobin. During the first two weeks of the disease, the blood shows little change except concentration, with apparent increase of the cellular elements, due, as a rule, to diarrhea. In the third week the red cells begin to decrease, and reach the minimum about the time of defervescence. They seldom sink below 2,000,000. The hemoglobin undergoes a relatively greater diminution, and it is restored more slowly after recovery. The number of leucocytes seldom departs from the normal (8,000 per c.mm.), except in the beginning, when the count is relatively higher on account of concentration. The larger forms predominate. A slight leucocytosis may develop, however, at the beginning of convalescence. Cabot does not find it a constant condition at this time, but observes that leucocytosis may fail of development through inability of the system to react, even in the presence of pneumonia or suppuration, in a very exhausted patient. The polymorphonuclear cells diminish as the other forms increase, often falling below 60 per cent. toward the end of the disease, thus contrasting strongly with their behavior in other infections. They begin to increase again in from three to ten days after defervescence, and become normal in number in the sixth or seventh week. Eosinophiles are present in small numbers. When acute inflammatory processes develop, as after perforation, leu-

cocytosis immediately develops, the polymorphonuclear forms predominating. During convalescence the coagulability of the blood is sometimes greatly above normal.

The pulse is increased in rapidity, as in all fevers, but not usually to so great a degree. In the first week it is generally above 100, but full and of low tension, often dicrotic. With the progress of the disease, it becomes small, feeble, and more rapid, sometimes reaching 150. It is rendered suddenly rapid, small, or even imperceptible by hemorrhage or perforation and by cardiac dilatation. It becomes rapid and irregular, during convalescence, upon the slightest exertion or excitement. Bradycardia (slow heart) sometimes occurs, especially during convalescence, and may persist for two or three weeks, the rate in some cases being as low as 40 or 30.

The Heart.—Myocarditis sometimes occurs. In protracted cases the heart muscles become soft and flabby to a variable extent, but the fibers show little or no change microscopically, except a granular degeneration in some instances. Fatty or hyalin degeneration is found only in the most protracted cases and in association with profound anemia. Other degenerations are rare.

Endocarditis is one of the rarest complications. It is attributable to the direct action of the toxin upon the endocardium.

Pericarditis is infrequent and is found almost exclusively in children or associated with pneumonia. Two cases of suppurative pericarditis are recorded.

The heart-sounds often remain normal, but in asthenic cases the first sound becomes weak and may become inaudible. In extreme weakness the first and second sounds become much alike and the long interval becomes shortened (embryocardia). A soft blowing murmur may replace the first sound. The systolic impulse becomes feeble and sometimes imperceptible.

The Arteries.—Thrombosis and embolism are not common, but sometimes cause obliteration of one or more arterial trunks. They generally form during the second or third week, sometimes during convalescence. When the femoral artery is closed, the circulation of the leg and foot is arrested, and gangrene of the foot and leg may result, as in the remarkable case recently recorded by Nammack. In a few instances both femorals have been obliterated. Embolism is thought to be a result of fragmentation and detachment of clots formed in the dilated cavities of the weakened heart, favored by the increased coagulability of the blood. Infarcts are sometimes found in the kidneys, spleen, and lungs, in association with arterial thrombosis.

The Veins.—Venous thrombosis is a complication in about one per cent of cases, occurring most frequently in the veins of the lower extremities, especially in the left femoral, rarely in the popliteal. It generally develops during convalescence. Its more frequent occurrence on the left side has been attributed to the slight pressure of the left common iliac artery upon the vein at the point of crossing. The clots are sometimes remarkable in size, extending from the deep femoral, through the iliac, into the vena cava. The fact that the bacilli have been found in the walls of the affected veins and in the clot suggests the probability that the coagulation is due to local infection of the

wall of the vein. As a result of the thrombosis, the leg becomes much swollen, painful, and tender; convalescence is prolonged, and permanent disability may remain. Gangrene of the leg has been observed in a few instances, but perhaps not solely as a result of the venous obstruction. Thrombotic obliteration of the left iliac vein has been followed by sudden death. Thrombosis may involve also the cerebral sinuses and very rarely the veins of the upper extremities. Symmetrical gangrene suggestive of Raynaud's disease has been seen. Thyroiditis has been observed during convalescence.

The spleen is enlarged to such an extent that, by the end of the first week, it can generally be felt below the margin of the ribs, unless, as is frequently the case, it is pushed back by the distended colon. In this condition the dullness may be unrecognizable on percussion. By the end of the third week it usually becomes reduced in size. Sometimes in elderly patients and generally after profuse hemorrhage, it is of small size. Owing to its friability, it may be ruptured by a blow, or, possibly, by too forcible palpation, as noted by Bartholow. Spontaneous rupture sometimes occurs. Hemorrhagic infarction may be found at autopsy.

Digestive System.—Complete loss of appetite (anorexia) is generally one of the earliest symptoms and the appetite does not return until several days after the fever has subsided. During convalescence it often becomes ravenous. Thirst is constant. In the height of the fever, when the demand for liquids is greatest, the patient rarely asks for drink, owing to his mental dullness, although he drinks with avidity the liquids that are put to his lips.

The tongue is generally moist, but coated with a thin white fur during the incubation. It is often tremulous when protruded. During the first week it becomes slightly swollen, the coat heavier and usually of a yellowish color, except at the tip, which is, as a rule, clean and of a bright red color. This appearance may persist throughout the course of the disease, but in most cases by the end of the second week the mouth and tongue become excessively dry, partly owing to a deficiency of saliva, and the coating often becomes dark brown. Catarrhal stomatitis may develop. If the patient breathes through the mouth, the dryness becomes extreme, the coating almost black, and deep fissures are formed. A similar coating (sordes) often forms on the teeth and lips. The condition of the lips is often aggravated by the patient's picking. As convalescence becomes established, the moisture returns to the mouth and the tongue loses its coating. In some cases, especially in children, the tongue remains clean throughout the disease.

The fauces and pharynx often become dry and red, as in scarlatina, or coated with a tenacious mucus that interferes with swallowing. Occasionally ulcers form, and a fatal membranous pharyngitis has been observed in the third week. Thrush sometimes arises as a complication and may extend from the mouth into the esophagus. Noma has also been observed affecting either the cheek or the genitalia.

Otitis media may arise from extension of inflammation through the Eustachian tube.

The Parotid Gland.—Complications on the part of the parotid gland occur in some cases. The inflammation is usually suppurative and confined to one side. It is more liable to develop during convalescence.

The infection may be either by extension through Steno's duct or by metastasis. Some authors regard the condition as highly fatal. The submaxillary gland may be similarly affected.

Vomiting is not a frequent symptom. It may occur early, however, especially in children, and sometimes persists throughout the disease. Repeated vomiting during the second and third weeks may indicate the development of peritonitis, nephritis, gastric ulcer, or a cerebral lesion. It has proved fatal in a few instances.

Chronic gastritis and dilatation of the stomach have been attributed to a preceding attack of typhoid fever. Keen reports two cases of esophageal stricture apparently due to cicatrization of typhoid ulcers in this unusual location.

Diarrhea.—There is probably no one symptom of typhoid fever which shows so much variance as this, or in regard to which so much diversity of opinion has existed. Probably, as Thompson remarks, it is less common and less severe than formerly. It is absent throughout the disease in fully half the cases and is rarely severe. It is probably a more prominent feature when the follicles of the large intestine are involved; but neither its presence nor its absence can be regarded as a reliable indication of the extent of intestinal involvement. Diarrhea usually develops by the end of the first week, but it may not begin until the third or fourth. The number of discharges seldom exceeds three or four a day, but may reach ten or more. The stools are alkaline, usually of a grayish yellow color, often not unlike the normal contents of the small intestine, though more offensive. They separate, on standing, into a thin supernatant layer containing albumin and salts, and a lower, flocculent, layer containing epithelial débris, numerous triple phosphate crystals, and remnants of food. Sloughs from the follicles may be found in them about the end of the second week. Microscopic examination reveals also numerous micro-organisms of different kinds and sometimes blood-cells. The typhoid bacilli are found in great numbers, as a rule, after the middle of the first week or beginning of the second.

Constipation not infrequently prevails throughout the entire course of the disease. In the experience of not a few writers it is the more usual condition. It occasionally becomes extreme, and fecal impaction with more or less complete obstruction of the bowel has resulted in a few instances.

Meteorism or abdominal distention of moderate degree is present in most cases, especially during the second week, the weakened, edematous, and probably paretic condition of the intestinal walls favoring the accumulation of gas. When extreme it is one of the most alarming and dangerous complications, preventing the healing of ulcers, favoring the production of perforation, and interfering with the action of the heart and lungs. The intestinal distention is thought also to favor the absorption of the products of food-decomposition and thus to increase the fever. It sometimes develops during convalescence as a result of a too liberal diet.

Abdominal tenderness and gurgling in the right iliac fossa are present in most cases, and although they are by no means pathognomonic, they are of some diagnostic importance, indicating the presence of fluid

feces and gas in the cecum and colon. Spontaneous pain does not usually occur, and it is generally confined to the iliac fossa. It may be so severe as to cause a suspicion of appendicitis, especially when associated with tenderness. In children the pain and tenderness are often referred to the umbilicus.

Intestinal hemorrhage occurs in from 3 to 10 per cent of all cases, and it proves fatal in from 30 to 50 per cent of the cases in which it occurs. It generally develops at the time the sloughs separate from the ulcers, toward the end of the second week or in the third; more frequently in cases that have run a severe course, but sometimes in the mildest. The hemorrhage generally comes on without warning. The patient suddenly sinks into a state of collapse, with pallor, restlessness, a sense of suffocation, cold extremities, and profuse cold sweat. The temperature declines rapidly, sometimes dropping six or eight degrees in a few hours and becoming subnormal. The pulse becomes extremely weak, often entirely fading away. Death may supervene before the blood has left the bowel. When recovery occurs, the discharges may continue to contain offensive dark clots for several days. Intestinal hemorrhage, like excessive epistaxis, may result from a natural tendency to bleed (hemophilia). It is important, in cases of supposed moderate intestinal hemorrhage, to exclude the bleeding of internal hemorrhoids.

Perforation of the bowel is an exceedingly dangerous complication and is responsible for from 2 to 3 per cent of the mortality. The perforation is most frequently located in the ileum, next in the cecum or colon, but it has been found in the jejunum. The time of its greatest liability is from the end of the second week to the beginning of the fourth, but it has been observed as early as the eighth day or late in the convalescence, even in the sixteenth week. It is more frequent in men and rare in childhood or after the fortieth year. It is more apt to occur in cases that have run a violent course, marked by profuse diarrhea and great abdominal distention, but it is not infrequently seen in mild, ambulatory cases. The cause of the perforation cannot usually be determined. Too active movements, as getting out of bed; improper food, meteorism, and vomiting may induce it. A lumbricoid worm has been found in the peritoneal cavity after perforation and the accident has been attributed to the parasite. Cold bathing has been, perhaps unjustly, censured as a possible cause.

The immediate result of perforation is the escape of the intestinal contents into the peritoneal cavity. This gives rise to an acute septic peritonitis that speedily becomes generalized. In a few instances the inflammation has remained circumscribed and, by forming adhesions, has shut off the portion nearest the opening and thus prevented an involvement of the general peritoneum. Intestinal hemorrhage is not infrequently associated with perforation.

The first indication that perforation has occurred is, in most cases, a sudden, severe pain in the abdomen, immediately followed by collapse, great weakness, pinched features, and generally a small rapid pulse. The abdomen becomes greatly distended and sensitive, the breathing is impeded. The legs are generally drawn up to relieve the tension of the abdominal walls. Nausea and vomiting generally ensue upon the beginning of inflammation, and may persist until the patient becomes

exhausted. In another group of cases the accident is followed by no distinctive symptoms, or the onset is so gradual as to escape observation. This is especially the case when the perforation occurs late in a case that has been characterized by delirium or coma. Percussion reveals an obliteration of the splenic and hepatic dullness, a sign belonging also to intestinal distention. The most valuable diagnostic feature in most cases is the development of leucocytosis. It is of value, however, only in the absence of suppuration or other complications. The abdomen may remain flat and hard, the patient soon passes into a moribund state, and the lesion is found after death. In some instances the first shock terminates fatally; but when death is due to pertonitis, it generally occurs on the third or fourth day after perforation.

A circumscribed peritonitis not infrequently occurs independently of perforation, and occasionally it becomes generalized. It is a result of the extension to the overlying peritoneum of the inflammation from the ulcers; from a suppurating infarct of the spleen or kidney, or rupture of the gall-bladder. Localized peritonitis of this character is rarely diagnosticated; it is recognized post-mortem by the adhesions that have resulted. A fatal case of chronic follicular colitis with villous outgrowth was observed, a few weeks after the fever, by Thompson. Abscesses sometimes form in the mesenteric glands, producing sepsis, perforation of the colon, rectum, or vagina, or fatal erosion of a blood-vessel.

The Liver.—Complications on the part of the liver are not frequent. Particularly is this true of single abscess-formation. Pylephlebitis is more common, and suppurative colangitis has been seen. Necrotic foci frequently occur, but, although they are sometimes numerous, they are ordinarily of little importance and are replaced by new connective tissue. They are attributed to the action of the typhoid toxin.

Jaundice sometimes occurs and may be a result of toxemia, catarrh or ulceration of the bile passages, the presence of calculi, or an extensive necrosis of hepatic cells. As previously noted, the mucous membrane of the gall-bladder is a favorite nidus for the growth of the typhoid bacillus. In some cases they cause no disturbance, while in others they give rise to suppuration, perforation, and resultant peritonitis. These disturbances are sometimes delayed until weeks or months after apparent recovery. The rather frequent occurrence of gall-stones after typhoid fever has been attributed to this persistence of the bacilli in the gall-bladder.

Respiratory System.—Epistaxis is of so common occurrence during the incubation or invasion as to be a symptom of diagnostic value. It is sometimes profuse, especially in the presence of the hemorrhagic diathesis, and it has proved a serious, even fatal, complication. The entire respiratory mucous membrane may become inflamed.

The larynx is not often the seat of complications, but simple and ulcerative laryngitis, edema often associated with ulcer, and perichondritis have been observed. Stenosis may follow. Paralysis of the laryngeal muscles, due to neuritis, has been noted.

Bronchitis is present in nearly all cases. Although the cough is often so slight as to cause little annoyance, auscultation reveals sibilant râles. It is seldom a serious complication except in children and the aged, in whom it may lead to lobular pneumonia. This affection is rendered

somewhat more dangerous in this connection by a tendency to suppuration or gangrene. Perforation of the pleura with resultant pyopneumothorax may follow the formation of a pus cavity.

Lobar pneumonia is a more frequent complication. It sometimes develops before the typhoid infection has manifested itself and may have nearly run its course before the latter disease has been recognized. The crisis is usually prevented by the typhoid infection. In other instances, the pneumonic consolidation occurs, sometimes after chill, and usually with some increase of temperature, during the second or third week of the fever or later. It then constitutes a most dangerous complication. The symptoms of pneumonia are sometimes so light or so obscured that the condition is probably often overlooked. Whether the pneumonia is induced by the typhoid bacillus or is due to a specific organism has not been determined.

Hypostatic congestion and *edema* of the lungs are generally associated. They occur to a variable extent in the posterior portions of both lungs, in a large proportion of cases, as a result of enfeebled circulation and the dorsal decubitus which the patient is probably too often permitted to occupy. Hemoptysis has occurred during the course of the disease in a few instances.

The Nervous System.—The effect of the typhotoxin upon the nervous system is a pronounced feature of the fever in most cases. *Headache,* usually temporal or occipital, sometimes general, predominates in the first week. It is occasionally accompanied by vertigo and in aggravated cases by pain in the back of the neck and in the dorsal region. These symptoms may be associated with retraction of the head, photophobia, and muscular twitching or rigidity, suggestive of meningitis. Such cases are classified by some authors as belonging to a cerebrospinal form of the disease. The symptoms probably depend for the most part upon the action of the toxin, although the bacilli have repeatedly been found in the meninges after death. The anatomical lesions are generally limited to a hyperemia of the pia mater of the brain and cord.

Meningitis is, however, one of the least frequent complications and is manifested by convulsions, opisthotonos, photophobia strabismus, disturbance of the cranial nerves and usually by an increase of fever with diminution or obliteration of the morning remission. *Wakefulness* and restlessness, especially at night, are prominent during the first week, but somnolence and apathy soon develop, as a rule, and the patient passes most of his time in sleep. But his rest is not refreshing. The lethargy is generally so marked that the patient must be aroused for the administration of nutriment and drink, and immediately after partaking of them he falls into his former state of indifference. *Convulsions* rarely occur except at the beginning of the attack, and then only in children, or at the onset of complications or intercurrent diseases. They are somewhat more frequent, however, in alcoholic subjects.

Delirium is not so often seen as it was before the adoption of present methods of treatment. Delirium of a few moments' duration is not infrequent, especially at night or just after awaking from sleep. It may assume several forms. When it is mild the patient often [illegible] especially when he is alone, or he may be [illegible] A worse form is accompanied with [illegible]

of the face, jerking of the tendons of the fingers and wrists (subsultus tendinum), picking at the bedclothes or at imaginary objects in the air (carphology), and attempts to get out of bed. These patients have repeatedly escaped at an unguarded moment and the result has frequently been disastrous. The tremulousness and delusions are much like those of delirium tremens, and they are more apt to occur in alcoholic subjects. They sometimes assume a hysterical aspect. In the worst cases, the patient sinks into a state of unconsciousness, is oblivious to his surroundings, and cannot be fully aroused. The urine and feces may pass involuntarily. Or the condition may be one of coma-vigil, in which the patient lies with open eyes, apparently seeing, often following the movements of his attendant, although he is entirely unconscious. As Niemeyer expressed it, he lives in an excited dream. These are the most fatal cases, and no doubt represent the highest degree of toxemia.

Theodore Diller reports a case of meningomyelitis following the disease in a girl of 15½ years, and affecting the cord from about the eleventh thoracic segment down. The paralysis of the legs, bladder, and bowel was complete for two months, then gradually improved.

Neuritis is not uncommon and may develop during the height of the disease or not until after convalescence. It may be local or generalized. It sometimes sets in with excruciating pain and great hyperesthesia of the nerve trunks or muscles of one arm or leg, affecting especially the extensors, in which form it leaves more or less permanent wrist-drop or foot-drop. Another form of neuritis has ensued upon a week or two of the cold-bath treatment. It produces the "tender toes" first described by Hanford, in which the pads of the toes become extremely sensitive to pressure.

Multiple neuritis, especially of a paraplegic type, sometimes develops during convalescence, affecting any one of the extremities or all four. The symptoms resemble those of multiple polyomyelitis, and paraplegia or an atrophic paralysis of a single extremity may remain.

Acute anterior polyomyelitis has been met with as a complication, particularly in children, and associated with a gradually ascending paralysis, often fatal in a few days. *Hemiplegia*, from hemorrhage or encephalitis, is rare, coming on during convalescence. It may be accompanied with choreic movements, or aphasia when the right side is affected. Recovery is the rule in children. True tetanic attacks are to be classed with the rarest complications.

Following typhoid fever, *mental disturbances* may remain for a time; the most common are loss of memory and melancholia. Insanity, usually of the confusional type, may develop in persons predisposed to it. Acute mania is occasionally encountered. Epilepsy also seems to be brought out in some cases. Brain abscess due to the bacillus typhosus has been observed.

The typhoid spine, which was first described by Gibney, as probably due to perispondylitis, an acute inflammation of the periosteum and of the fibrous structures which bind the vertebræ together, is regarded by Osler as a neurosis. It usually develops several weeks after the fever has subsided and may follow a jar or shock. It is not attended with fever or other signs of inflammation. The symptoms are chiefly of a hysterical nature.

Organs of Special Sense.—Conjunctivitis and keratitis, sometimes with phlyctenulæ, and iritis and choroiditis are occasional complications on the part of the eye. Panophthalmitis is exceedingly rare. Dilatation of the pupil is common; temporary paralysis of accommodation is not infrequent. Paralysis of the external muscles of the eye is sometimes met with during convalescence, probably as a result of neuritis. Retinal hemorrhage, alone or accompanying other hemorrhages, is more frequent, according to De Schweinitz, than is generally realized. Profuse retinal hemorrhage is rare, but amaurosis sometimes develops after severe intestinal hemorrhage, and the blindness is sometimes permanent.

Single or double optic neuritis may develop independently of meningitis, and is generally followed by atrophy of the optic nerve. Cataract has been noted as a sequel of typhoid fever. Trélat records the occurrence of double cataract in two cases. Orbital hemorrhage and thrombosis of the veins of the orbit are possible complications.

Otitis media occurs in about 2.5 per cent. of cases, but recovery is generally prompt without involvement of the mastoid cells.

Renal System.—There is the usual febrile concentration of the urine, with increase of specific gravity and color. As the fever declines, the quantity is increased and the density diminished. The chlorids, on the other hand, are reduced during the febrile stage and increased when the elimination becomes more abundant. Retention of the urine is often an early symptom and may recur periodically. During the somnolent state, the patient seldom expresses a desire to urinate, and a careless nurse may permit the bladder to become distended. Pain and restlessness are produced. Incontinence may occur during delirium.

Febrile albuminuria is observed in nearly a third of all cases. The urine contains also, in nearly all cases, an undetermined substance which yields a peculiar yellow color in the diazo reaction (see p. 734). Acute nephritis occasionally develops. 1. It may occur at the onset of the disease, producing a type to which German authors give the name nephrotyphoid. The passage of scanty, bloody urine at this time sometimes masks typhoid infection, which, developing later, may be for a time regarded as uremic in character. 2. Developing during the height of the fever, especially during the beginning of the second week, nephritis often escapes recognition, but should be recognized by the presence of albumin, casts, blood, and epithelium. 3. As a sequel, nephritis is generally promptly recognized, on account of the production of edema. It is not usually serious in its results. The lymphomatous nephritis of Wagner is not attended by symptoms. Pyuria has been noted, sometimes early, sometimes as late as the twenty-eighth day. The origin of the pus is obscure. The colon bacillus was found in the pus of seven, the typhoid bacillus in two, and the staphylococcus albus in one of Osler's cases. Suppurative pyelitis is rare. It may be membranous at first and associated with membranous inflammation of the bladder. Later the membrane gives place to an erosion and ulceration. Hematuria is usually associated with acute nephritis, but may occur independently; the blood is of renal origin. In some cases hemoglobin alone is found. The toxicity of the urine is increased throughout the entire course of the disease and convalescence.

Cystitis may occur later in the disease or during convalescence, especially as a result of retention or from infection by unclean catheters.

Urethritis is reported to have originated spontaneously during convalescence in a few cases in which other organisms than the gonococcus were present. Orchitis and epididymitis, separately or together, have also been observed. They are attributed by Keen to a pure typhoid infection. Abscess of the ovary, of the same character, has been noted. Gangrene of the genitalia (noma) has occurred in a few cases, chiefly in women.

The Typhoid Eruption.—The characteristic eruption is a rose-colored rash which ordinarily makes its appearance on the seventh or eighth day, but sometimes as late as the tenth or twelfth. It generally consists of not more than a dozen distinct, round or lenticular, slightly elevated papules flattened on top, from 2 to 4 mm. in diameter, occasionally showing small vesicles in the center, sometimes petechial or dark in color. It appears first on the upper part of the abdomen and lower part of the thorax, sometimes on the back and thighs. When the spots are numerous they may be found also on the extremities, rarely on the face. Successive crops develop, each persisting two or three days, and they sometimes leave brownish pigmentation. They can often be felt with the finger, and, when touched, vanish, but quickly reappear after the pressure is removed. They are rarely to be found after the middle of the third week. They are occasionally absent, particularly in children and old people.

Erythema, a scarlet-colored rash, sometimes appears early, especially on the thorax and abdomen, rarely extending to the extremities. Urticaria, papular eczema, and purpura hemorrhagica have been encountered. Herpes may appear on the lips, but is less frequent than in other fevers. Peliomata, peculiar, pale blue, subcuticular spots of irregular outline and from 4 to 10 mm. in diameter, are sometimes seen. They are believed to be due to the presence of pediculi and, therefore, bear no relation to the disease.

Tache cérébrale is the name given to the red line with white margins which appears, especially in nervous subjects, after the finger-nail has been drawn over the skin. It is not peculiar to this disease. Osler calls attention to a pinkish, sometimes mottled appearance of the skin of the abdomen and arms that is seen in some cases when exposed to the air.

Ecchymoses rarely appear except in connection with the hemorrhagic diathesis or on the dependent portions of the body in a moribund case. The palms and soles often become dry and harsh, apparently thickened. More or less general edema may occur as a result of anemia, or locally after obstruction of the circulation, as by thrombosis. It is also an important symptom of nephritis in a later stage of the disease.

Bedsores occur for the most part in emaciated subjects or after protracted fever. They are not so frequently seen since cleanly methods of nursing have been adopted. They may occur, however, under the most careful supervision, as a result of profound trophic disturbance, or when the vitality of the skin has been impaired by one of the eruptive disorders. They occur, as a rule, over the sacrum or buttocks, but sometimes over other prominences, the shoulders, spinous processes, elbows,

heels, and occiput. Rarely they appear in places not subjected to pressure. Gangrene, as previously stated, is generally a result of thrombosis or embolism.

Boils and abscesses are not infrequent sequelæ, resulting from pyogenic infection of the skin, especially of the axillæ, back, buttocks, arms, or legs. Curschmann thinks that they are more frequent after the cold-bath treatment. They are sometimes so numerous as to greatly weaken the patient and prolong convalescence. They may continue to appear for a month or more after recovery. As a result of these suppurative affections, the pus-forming cocci sometimes gain access to the blood and produce pyemia. It is quite probable, indeed, that in many cases the multiple abscesses are themselves the result of pyemic infection.

Atrophic lines are sometimes left in the skin of the abdomen and sides of the thighs similar to those produced by pregnancy. They are probably due to neuritis.

Sweats.—During the height of the fever, the skin is generally dry and hot; sweating is rare. The chest and abdomen may, however, become moist for a while after a bath. A sudoral form of the disease, characterized by profuse sweating, is described by French writers. Chills occurring during the course of the disease are sometimes followed by sweating similar to that at the close of a malarial paroxysm, although no malarial infection is present. A suspicion of sepsis often is aroused. During defervescence, however, sweating is more common. Sudaminal and miliarial eruptions are occasionally seen in cases characterized by free sweating. They are generally limited to the axillæ, abdomen, and inner sides of the thighs. They sometimes terminate in a desquamation in the form of flakes or large pieces, especially in children.

A peculiar odor is frequently noticeable; possibly it is a cutaneous exhalation. It sometimes appears to be an exaggeration of the individual odor, but in some cases it is better described by Nathan Smith as a "semi-cadaveric" smell.

Alopecia.—The loss of hair, which is almost universal during recovery from typhoid fever, may be slight or it may reach the degree of baldness. It is generally confined to the head, rarely affecting the beard or other parts of the body. The hair is generally fully restored, but in some cases the growth is lighter and other characteristics may be altered.

Muscles, Bones and Joints.—The muscles are generally atrophied. Granular, fatty, or hyalin degeneration sometimes occurs. As a result, especially of the last form of degeneration, the muscles become friable and may be easily ruptured. Abscesses and hemorrhages sometimes occur in the substance of the muscles.

Bone-lesions are exceedingly common and troublesome sequelæ. Of Keen's 237 collected cases, periostitis occurred in 110, necrosis in 85, caries in 13, osteitis (bone abscess) in 12, osteomyelitis in 10. The tibia was affected in 91 cases, the ribs in 40. In 51 cases examined, pyogenic cocci were found in 13 and typhoid bacilli in 38. The disease is generally chronic and liable to recur. It is favored by traumatism received shortly before or after the fever. Witzell thinks that injury on the side of the bath-tub may cause it. Keen believes that muscular strain is sufficient, and that this fact accounts for the comparative fre-

quency of periostitis of the crest of the ilium, the anterior superior spinous process, and promontory of the ischium, where independent affections are rare.

Arthritis.—Keen recognized rheumatic, septic, and typhoid forms of this comparatively rare complication in 84 collected cases. Spontaneous dislocation, especially of the hip, is liable to occur.

Associated Acute Infections.—Malaria sometimes occurs in conjunction with typhoid fever, giving rise to the double infection, typhomalarial fever. This is not a distinct, hybrid disease, as was at one time believed. Some cases of typhoid fever show more or less distinctly remittent or intermittent features, even when the plasmodium is absent. Measles, smallpox, chicken-pox, scarlet fever, diphtheria, whooping-cough, and noma sometimes develop during the course of the fever. Erysipelas has rarely been observed. Typhus fever has been encountered in association with typhoid, in a few instances. Miliary tuberculosis is sometimes associated with it. It is an interesting fact that choreic movements and epileptic seizures generally cease during typhoid fever, and that sugar may for a time disappear from the urine of the diabetic patient.

Septic infection may happen during the later weeks of the disease or as late as two weeks after the fall of the temperature. It may take the form of septicemia, indicated by chilliness with moderate fever, sweating, and weakness; or the infection may be pyemic in character, usually manifested by frequent chills, high but irregular temperature, and the development of thromboses, abscesses or boils about the buttocks, axillæ, or joints. Abscess of the breast has been observed.

Relapses.—A relapse is due to reinfection. It usually occurs after complete defervescence, sometimes several weeks after the temperature has been normal. It may occur, however, before complete defervescence. Of the cause we know comparatively little. It has been suggested that the reinfection is due to the inoculation of healthy intestinal follicles by the sloughs cast off from the original ulcers; but in some instances the more recent lesions are found higher up in the bowel than those of the original infection. As Chiari suggests, the reinfection no doubt often arises from the escape of bacilli from the gall-bladder. From one to five relapses have been observed in the same patient.

The onset of a relapse is sometimes abrupt, often with a chill, and the temperature may rise suddenly, but in most cases it shows the typical daily ascent. There is no prodromal stage. All the symptoms return, including the rash and splenic enlargement. The eruption may appear as early as the third or fifth day. The course of the disease is not usually so long or so severe as in the original attack, but in some instances, especially when the original attack was mild, it has been much more protracted and even fatal. Its course is often more severe than that of the original disease, especially when it develops early. The differential diagnosis of a relapse is often difficult.

Recrudescence is simply a return of the fever without aggravation of the other symptoms after the temperature has been normal for a few days. Its cause cannot always be determined, but in some cases it is due to too free nourishment, nervous excitement; occasionally, perhaps, to malaria.

Diagnosis.—*General.*—The fact that typhoid fever is the most frequent of the continued fevers warrants its consideration in every case of protracted elevation of temperature. When a person between 15 and 30 years of age has a slight fever, with a rapid, soft pulse, a furred and slightly tremulous tongue, and gives a history of lassitude, headache, pains in the back and limbs, anorexia, chilly sensations, restless sleep, gradually becoming more pronounced for a week or two, typhoid fever is highly probable. The diagnosis should rarely be made, however, at the first examination. If nose-bleed, constipation, or diarrhea is added to the symptoms, and a cathartic has been found unusually brisk in its action, a tentative diagnosis may be made, particularly if the disease is prevalent in the vicinity at the time or a probable source of infection can be traced. If, after a few days' observation of the case, it is found that the temperature has followed the regular course of elevation; that the pulse has become more compressible, rapid, and dicrotic, the tongue more heavily coated and red at the tip and edges, the abdomen distended and tympanitic, with tenderness and slight gurgling in the ileo-cecal region, and enlargement of the spleen—then the diagnosis is all but positive. Add to these symptoms, after seven or eight days of fever, the characteristic rash, and the diagnosis becomes positive. Unfortunately such cases are the exception rather than the rule. Some symptoms are usually wanting, and in some cases the patient is not seen until the condition of hebetude obscures the history and subjective symptoms. In the absence of the eruption in such cases the diagnosis becomes exceedingly difficulty, and often impossible, for a time at least. Rarely the diagnosis is revealed by such an accident as a profuse intestinal hemorrhage, or possibly only by the discovery of pathognomonic lesions after death. In many instances, particularly in the rural districts, small towns, or summer resorts, it is possible to clear up an obscure case by tracing the infection to a previous case of the disease. To more effectually establish the diagnosis, several tests may be made.

Specific Diagnosis.—*Bacteriological Test.*—The bacilli may be found, by plate culture, in the urine and feces of most cases at a variable period from the beginning of the first week to the end of the second and thereafter. (For method, see p. 747.) They may be found also in the blood obtained by puncture of the rose-spots; puncture of the spleen is rarely justifiable.

Diazo Test.—For Ehrlich's diazo reaction, see p. 734.

Blood Test.—The most valuable feature of the blood-count is the absence of leucocytosis at all stages of the disease. Anemia is also absent until the decline of the temperature.

Serum Test.—Widal's serum test is described on page 717.

Differential Diagnosis.—The chief obstacle to diagnosis is the great diversity of manifestations in different cases, many of which assume a resemblance to other diseases. The most distinctive features are: the peculiar temperature curve, the eruption, the absence of leucocytosis, and the reactions to the diazo and Widal tests. The following summary will assist in its differentiation from the diseases which it most frequently resembles:

Malaria.—Typhoid fever may assume an intermittent course, to the extent that the fever is higher on every second or third day, and chilly

sensations may be felt; but the periodical chills, profuse sweats, and complete intermission of temperature are rarely seen. Quinin has little effect on the temperature. It may resemble intermittent fever, especially in children, in whom the eruption does not always appear. The estivo-autumnal type of malaria is excluded with most difficulty, however, especially in malarious districts. In it there is often a history of malaise preceding the fever, the chill may be absent, vomiting and diarrhea are often present, the temperature range may be almost uniform, the cheeks are flushed, the tongue dry and coated, possibly with a yellowish or brownish fur, and the spleen is enlarged. But the rash does not appear, the serum test is negative, and the plasmodium or an abundance of pigment is found in the blood.

Cerebro-Spinal Meningitis.—Cases that show unusual irritation of the meninges bear a strong resemblance to this disease, but meningitis is a much less frequent disease. The nervous disturbances, rigidity of the neck or opisthotonos, convulsions, photophobia, and strabismus are usually more pronounced than in any case of typhoid fever. The cutaneous and tendon reflexes are very irregular in their responses, the rose-spots are absent, and the abdominal symptoms are less pronounced or absent. For a few days, however, a diagnosis may not be possible in some cases.

Lobar Pneumonia.—When a double infection occurs and both diseases are present, the greater prominence of the initial symptoms of pneumonia, as compared to those of typhoid fever, may prevent the early recognition of the latter disease. Again, typhoid sometimes begins with intense pleuritic pain which leads to a suspicion of pneumonia. In the absence of double infection, however, there is little cause for confusion, since there is less cough, no dullness on percussion, the râles are mostly sibilant and not confined to one region, no rusty sputum, and a different temperature curve. The blood-count and serum test assist in the diagnosis.

Septicemia.—When the location of the suppuration has not been recognized, this condition may resemble typhoid fever, but the temperature is not so uniform; leucocytosis is present and careful search will generally reveal suppuration. In pyemia, severe chills are generally present, and the temperature range is wide, with decided hyperpyrexia in many cases. The Widal test fails.

Uremia.—A uremic condition may obscure the diagnosis in the beginning of some cases, rarely at any other time. But the tense pulse, rapid respiration, contracted pupils, and absence of abdominal symptoms would serve to distinguish the condition. Uremia may, however, occur as a complication, and prolonged uremic coma sometimes resembles typhoid fever so closely as to be differentiated only by careful analysis of the urine.

Acute Miliary Tuberculosis.—In many cases there is a history of previous tubercular disease; the pulse and respiration are rapid, the mind is usually clear, the cough is more annoying, the sputum is often bloody, the abdominal symptoms are not so well marked, tubercle bacilli may be found in the blood or sputum, leucocytosis and anemia are present, and tubercles may be discovered in the choroid on ophthalmoscopic examination.

Tubercular meningitis usually occurs in children; there is a history of irritability preceding the stupor, constipation is persistent, the abdomen is usually flat, leucocytosis is present, and tubercles may be found in the choroid.

In *tubercular peritonitis*, the temperature is irregular, sometimes subnormal, the abdominal tenderness greater and more general; ascites may be present, and the leucocytosis excludes typhoid fever.

Appendicitis is usually more sudden in development, with greater pain and ileo-cecal tenderness; or, if of slow development, the constitutional symptoms are less prominent. Vomiting is often present. Percussion of the region elicits tympanitic dullness, as distinguished from the resonance of distention, and tumefaction may be felt. Tenderness at McBurney's point and, more particularly, leucocytosis and the failure of the Widal test establish the diagnosis.

Influenza of the abdominal type is sometimes excluded with difficulty for a time on account of the headache, abdominal tenderness, pain, and diarrhea. But the onset is generally more sudden, the prostration earlier developed; the symptoms more numerous and painful. The tongue in influenza is bright red with prominent papillæ.

Malignant endocarditis sometimes resembles typhoid fever, but is an infrequent disease. Abdominal tenderness, diarrhea, splenic enlargement, and stupor are sometimes present. There is generally, although not always, great cardiac pain and distress. The onset is sudden, the temperature irregular, and leucocytosis is present.

Relapsing fever is very infrequent in America. The invasion is sudden, with a chill; the pain is epigastric in location; there is no rose-eruption; the nervous phenomena are less pronounced and the spirilla are readily found in the blood.

Typhus fever is seldom encountered except in epidemics confined to a single institution or ship. The onset is sudden, the stupor profound, the face is dusky, the eyelids swollen, the pupils contracted; there is a macular eruption usually appearing on the fourth or fifth day, changing into petechiæ; the duration of the disease is short and its termination is by crisis.

The *Para-Infections*.—There remains a not well defined group of diseases, clinically almost identical with typhoid fever, but due to infection by the so-called paratyphoid or paracolon bacilli. In many instances a differentiation can be made only by the bacteriological or serum tests. The Widal reaction is almost always absent.

Ptomain-poisoning and autointoxication with leucomains are generally to be excluded by the abruptness of onset, often with vomiting, diarrhea, and prostration, and by the absence of serum reaction.

Prognosis.—An unconditionally favorable prognosis should never be made in this disease, for the most distressing complications often arise in the mildest and most hopeful cases; and in the absence of complications a sudden, even fatal, collapse may occur. The mortality in private practice is usually from 5 to 10 per cent, and in hospital practice from 7 to 15 per cent. The death-rate has been reduced about one-half since the introduction of the cold-bath treatment. Some epidemics are characterized by a low mortality, others by an excessively high rate. Regional differences may also be observed in some instances. The mor-

tality in women is higher than in men and in fat persons than in lean. From puberty on, the disease becomes more fatal with the advance of years, yet old people often make excellent recoveries. The disease is usually mild in children. An impoverished state of nutrition, particularly that due to chronic alcoholism, diminishes the chance of recovery. The severity of the type of infection has a great influence on the mortality and probably accounts for the difference in different epidemics. The extent to which the nervous system is involved and the degree of pyrexia are important factors. Hyperpyrexia is indicative of danger only when it is continuous for a number of days with but slight remissions. No definite degree of fever is necessarily fatal, but recovery seldom occurs after the temperature has reached 106° F. (41.1° C.) four or five days in succession; the danger lies in the profound intoxication of the nervous system revealed by the high temperature. Delirium is an unfavorable symptom, especially when it develops early and assumes the low muttering form. Excessive meteorism and hemorrhage are also dangerous symptoms, but not necessarily fatal. The prognosis is especially bad in the ambulatory form of the disease, owing to the greater frequency of complications. Sudden death sometimes occurs without premonitory symptoms and without discoverable cause even after convalescence has been well established. It is probably due in most cases to a failure on the part of the weakened heart, a "delirium cordis." But, on the other hand, it should be remembered that recovery often occurs in the most hopeless cases, and a positively unfavorable prognosis should not be too soon pronounced.

Treatment.—*Prophylaxis.*—The measures to be adopted for the prevention of typhoid fever are in part municipal and in part personal. It is the duty of physicians to enlighten the people in the possibility of preventing the disease, and the measures to be adopted. The importance of a pure water-supply, free from possible contamination, and the necessity of a proper system of sewers should be promulgated. Dairymen should be taught the importance of using only boiling water for the cleansing of cans and utensils.

Individual protection theoretically requires the abstaining from everything that has not been disinfected by heat. Drinking-water and milk must be thoroughly boiled and cooled in bottles, for the addition of ice is not safe. All utensils must be cleansed in water that has been boiled. Green vegetables, to be eaten raw, must be thoroughly washed in sterilized water or refrained from. Oysters fed in the mouths of streams contaminated with sewage must not be eaten raw. In times of danger, these requirements are imperative, and there are few cities in which the requirements in regard to water must not be carried out at all times. In villages and in the country, however, the purity of the water is often a matter of certainty, and boiling may be neglected. Filtration has done much to reduce the prevalence of the disease in some places, notably in London, but it is absolutely untrustworthy for the purification of contaminated water, since the bacilli are capable of passing through many of the house filters. Visitors and recent residents should be particularly careful to obtain their food and drink from uncontaminated sources, or to thoroughly disinfect them.

Disinfection.—Methods of antisepsis must be applied to the patient, to

his excreta, to the bed-linen, and more or less generally to all articles coming in contact with the patient. The best disinfectants for feces and urine are: a 1:500 acidulated solution of mercuric chlorid, a 1:20 solution of commercial carbolic acid, and fresh chlorinated lime. Recently a 1:20 solution of formaldehyd has been much employed. For solutions and methods, see p. 727.

Preventive inoculation has been practiced with apparently some degree of success. In Maidstone, 95 persons escaped infection after inoculation. In the recent Boer war, 200 English troops were inoculated, of whom 3, or 1.5 per cent., took the disease; none died. The inoculation produces local reaction, a rise of temperature and the serum of the person acquires agglutinative properties, reacting to the Widal test. The results are not conclusive, but justify a hope for future success.

General Management.—Good nursing is more important than drugs. The patient should be put to bed immediately when the disease is suspected, and kept there until convalescence is fully established. This is usually from ten days to two weeks after the temperature has ceased to rise above the normal. The sick-room should be large and airy, if possible on the sunny side of the house. An abundance of fresh air should be admitted without too much draft. The temperature of the room should be 68° F. (20° C.) in daytime and 65° F. (18° C.) at night. Perfect quiet must be maintained. The attendants should be limited to a nurse and one member of the family; visitors should be excluded. Unnecessary talking, but, above all things, whispering, must be prohibited. The bed should be narrow and low enough to permit easy handling of the patient. A woven-wire spring, with a soft hair mattress covered with a double blanket, affords the greatest comfort. A rubber cloth should be placed under the sheet. The bed must be kept clean and smooth at all times to prevent bedsores, and an air-cushion should be used if their formation is threatened. The head should not be too high.

A competent nurse should be employed when circumstances permit. The physician should write out daily his instructions as to diet, nursing, disinfection, and medication, and in return should receive from the nurse a daily record of the temperature, taken every three hours, with a report of the number of dejections, baths, hours of sleep, amount of nourishment and drink administered, and any unusual symptoms—in short, a history of the case for the last 24 hours. The temperature chart should be kept from the patient's view. He should not be informed of any unfavorable symptoms or complications; he need not be made aware of intestinal hemorrhages. He should not be permitted to lie constantly on the back, but should be carefully turned upon either side from time to time. He must never be allowed to rise for any purpose. It is better to have him naked in bed, for a gown becomes wrinkled and rough. Food and drink must be given through a tube. The bedpan must always be used. The patient generally finds difficulty at first in its use, but rarely fails to become accustomed to it in a few days. It is only in the rarest cases, when the nervous excitement occasioned by unsuccessful attempts to use the bedpan is a positive menace to the patient, that the physician should consent to his being carefully

lifted upon a commode. The patient must not be allowed to make the slightest exertion.

The mouth, tongue, and lips should be cleansed once or twice daily. A solution of borax, or equal parts of hydrogen peroxid and glycerin, or listerin, on a soft rag may be employed. It may be necessary to scrape the tongue. The system requires an abundance of water for the maintenance of the secretions, especially that of the kidneys, which are burdened with the elimination of the toxic matters. At least a quart a day of pure cold water should be given with the regularity of medicine, for the patient seldom asks for it. This is one of the most important items in the treatment. The addition of a few drops of dilute hydrochloric or phosphoric acid or a little lemon-juice is beneficial and generally agreeable. The aromatic sulphuric acid may be substituted when the diarrhea is excessive or the sweating profuse.

Hydrotherapy.—The Brand method, or cold-bath treatment, is the most esteemed of the methods of hydrotherapy. The benefits obtained from it are: (1) The reduction of temperature. This is, however, one of the minor considerations. (2) The stimulating effect upon the nervous system. There is no other method that will so promptly and so effectually clear the intellect and arrest the tremor. After the bath the patient generally falls into a tranquil sleep of several hours' duration. (3) Stimulation of the heart's action. The danger of sudden failure of the circulation is removed; the pulmonary circulation is rendered stronger, and thus the tendency to hypostatic congestion and thrombosis is diminished. The increased renal circulation results in a more abundant elimination of the toxic matter. (4) An increase of respiratory movements, especially deep respiration, through which the tendency to bronchitis is lessened.

The full Brand method is contraindicated: (1) When pneumonia or pleurisy is present; (2) when alarming paroxysms of dyspnea, coughing, or cyanosis are induced by the bath; (3) after perforation or peritonitis has developed; (4) when there are extensive bedsores which would be injured by the cold water. A modified bath must sometimes be administered to very elderly persons and those who cannot become accustomed to the full bath. As in all therapeutic methods, however, it is, as a rule, better to omit the treatment altogether than to give it in a half-way or careless manner. The first sensation of a dip into cold water when the temperature of the body is abnormally high is by no means pleasant, but there are few persons who will persistently object to it after they have experienced the after-effects and have been made to understand the benefits they will derive from the treatment.

The Brand treatment should be begun early, always before the fifth day if possible. The baths should be repeated every three hours as long as the rectal temperature exceeds 103° F. (39° C.) or as long as the sensorium continues depressed, even with a lower temperature.

The Method.—A portable bath-tub of sufficient length and width to accommodate the patient should stand in readiness at his bedside, protected from his view by a screen. It should be filled to three-fourths of its depth with water of 90° F. (32° C.) for the first bath. The temperature of the water should be reduced 5° F. (2.5° C.) at each successive bath until it has reached 65° F. (18.5° C.) and no

lower. The patient is given a stimulant, brandy or black coffee; he is then uncovered, a napkin placed over the genitals, and the face bathed with cold water. He is now transferred to the bath by two attendants, one grasping him under the shoulders, the other just below the knees. This should be done as gently as possible. Brisk friction is performed during the bath. Every part of the body except the lower abdomen should be gently but firmly rubbed in order to prevent chilliness and to stimulate the cutaneous reaction. Nothing short of chattering teeth or a cyanotic appearance of the face should be regarded as an indication to discontinue the bath in less than twenty minutes. While the patient is in the water, the bed should be prepared for his return. This is done by laying over it a double blanket, and over this a sheet. He is laid upon this and the sheet is folded over him, a fold being passed between the arms and sides and between the legs, and the blanket is wrapped around this. Here he is permitted to lie undisturbed for five or ten minutes. If, however, the temperature was but moderate before the bath he may be dried at once, first with the sheet, and afterward with soft towels. Hot bottles should be placed at his feet. Reaction is generally prompt. Prolonged shivering after the bath, Baruch tells us, points to some defect, either in duration or temperature, or the friction may not have been properly performed.

It is often a difficult question to determine whether the cold-bath treatment should be administered to a patient first seen in the second or third week of the disease. It must be left to the judgment of the physician, based upon the condition of the patient. A young physician would perhaps better select a mild case in the beginning of its course for his first application of the treatment, particularly if he be located in a community where the method has not been practiced.

A great many intelligent physicians oppose the cold-bath treatment upon various grounds, others employ it in a modified form. Probably the best modification, especially for the treatment of children, the aged, and persons who persistently refuse to become accustomed to the Brand bath, is found in the use of water at 90° F. (32° C.) at the beginning of every bath, subsequently reducing the temperature of it by the gradual addition of ice-water until 70° F. (21° C.) or even 65° F. (18.5° C.) is reached (Ziemmsen's method). As substitutes for the cold bath, sponging and the cold pack are the most popular.

The cold pack is applied by wrapping the patient in a sheet wrung out of water at 60° or 65° F. (15.5°—18.5° C.), and then sprinkling him with water of the same temperature from a watering-can. It does not compare favorably in its results with either the cold bath or sponging.

Cold sponging should be practiced in all cases that are not subjected to the cold-bath method. The entire body is sponged with likewarm, cold, or iced water for 15 or 20 minutes at a time. The effect of the ice-cold sponging is an almost as pronounced stimulation of the nerve centers as is obtained from the bath, and it is secured with much less labor.

Dietetic Treatment.—Nourishment must be administered entirely in fluid form. Milk is the best food. Four pints should be given to an adult in twenty-four hours, which is about equivalent to a tumblerful every two hours in daytime and a little less often at night. It may

be given cold or warm. If objectionable to the patient, its taste may be modified by the addition of a little salt, Vichy, carbonated water, a little coffee, tea, or cocoa. In some persons it produces constipation, flatulence, tympanites, or diarrhea, with undigested curd or fat in the stools. In such cases, if the addition of lime-water or Vichy does not relieve the difficulty, the milk may be partially predigested, or the quantity must be reduced and other nourishment given. Beef, mutton, or chicken broth and bouillon answer this purpose. A little thoroughly cooked rice or the white of an egg may be added to the broths. If, however, the diarrhea be severe, broths and beef-juice must be omitted on account of their tendency to increase it. The whites of five or six eggs may be given during a day, beaten with milk or sherry, or in the form of albumen-water, flavored with lemon or orange. Panopepton or other predigested food may be tried with caution. But it should be borne in mind that every unavoidable departure from a strict milk diet adds risk and an occasion for regret in case of a fatal issue.

Diet of Convalescence.—It is a safe rule not to allow solid food for eight or ten days after the fever has remained normal, and to exercise especial caution in the addition of meat to the dietary. After a few days of normal temperature, a soft egg, milk toast, custard, and junket may be added, one at a time and at only one meal each day. Then blanc-mange, bread and milk, boiled rice, bread pudding, and other articles made of eggs and milk may be given in small quantity, the effect of each article added being carefully watched. If elevation of temperature, diarrhea, or other disturbance be produced, the milk diet must be resumed for a few days.

Medicinal Treatment.—No routine course of medication is required. Symptomatic indications should be met, but these are generally few. In some localities, the administration of quinin for a few days is judicious, in order to exclude the possible presence of malaria. Small doses of calomel, gr. 1-10 (0.006), may be given for a few days, for its antiseptic effect, especially if constipation exist. Salol, β-naphthol, creosot, guaiacol, and other intestinal antiseptics are in favor with some authorities. They probably have little effect upon the infection.

Inoculation Treatment.—Several attempts have been made to treat the disease with sterilized liquid obtained from cultures of the bacillus typhosus or the bacillus pyocyaneus; with blood-serum obtained from convalescent patients, or with the serum of dogs that have been inoculated with typhoid cultures. In most instances the only apparent result was a reduction of temperature; that the course of the disease was modified could hardly be asserted. The recent experiments of Richardson, in which normal blood-serum was added to the curative serum, seem to promise better results.

Treatment of Special Symptoms.—*Fever.*—The best means of reducing the temperature when the cold-bath treatment is not employed is by repeated sponging with ice-water. Antipyretics of the coal-tar series, as phenacetin and acetanilid, should be carefully used, if at all. A mixture of equal parts of guaiacol and glycerin or guaiacol carbonate may be applied with friction to the outer side of the thigh. Its action should be watched, however.

Diarrhea.—Four or five stools a day cannot be regarded as harmful. If moderately excessive, they may be checked by the administration of bismuth, grs. x to xv (0.65—1.0), Dover's powder, grs. iij to v (0.2—0.35), or camphorated tincture of opium in teaspoonful doses repeated every three hours. If the diarrhea be profuse, the lead and opium pill or morphin, gr. ⅛ (0.008), may be required. If the stools are offensive, salol, grs. v (0.35), may be given with the opiate. The cause of the diarrhea should be sought by examination of the stools, especially for undigested milk.

Constipation is best overcome by enemata of soapsuds, oil, or glycerin. Small doses of calomel may be employed in the first days of the attack.

Tympanites.—Turpentine stupes or hot fomentations should be applied. Ten drops of turpentine internally, in emulsion or on a lump of sugar, are often effective. When the colon is greatly distended, the introduction of a rectal tube often gives vent to the gas.

Delirium.—Such causes as meteorism or deficient action of the kidneys should be looked for. Quiet restraint must be exercised. The bromids act well in some cases; morphin must be employed in others. Hydrotherapy is probably the most certain means for its prevention.

Mania requires the most careful attention. The patient should not be left alone for an instant. Hyoscin hydrobromate in 1-100 grain (0.0006) doses may be given, and, this failing, morphin in full doses must be resorted to.

Hemorrhage of the Bowels.—Absolute rest and quiet must be secured. The patient should be kept in a quiet doze for several days by the administration of lead and opium pills, laudanum, or morphin in sufficient doses. The foot of the bed should be raised and a Leiter coil or other cold application should be applied to the ileo-cecal region. Alcoholic stimulants should not be given, except in case of extreme collapse. Even in this condition, strychnin given hypodermically is safer. Transfusion of 0.7 per cent saline solution into a vein or the subcutaneous tissue may assist in tiding the patient over.

Perforation.—Surgical aid should be at once called, unless the condition of the patient is so extreme as to clearly preclude the possibility of an operation. In the mean time the patient should be kept under morphin.

Cardiac Weakness.—Strychnin should be given every three or four hours in increasing doses, up to 1-20 grain (0.003) if necessary. Whisky or brandy may be given in the absence of contraindications, from a tablespoonful every three hours to an ounce every hour until reaction is obtained.

Bedsores can be generally prevented by thorough cleanliness and keeping the bed and skin dry. The prominences should be bathed twice daily with alcohol, brandy, or spirits of camphor, dried, and dusted with an antiseptic flesh-powder or the stearate of zinc.

Treatment of Convalescence.—As soon as the patient has begun to take solid food, he may begin to sit up in bed. He may be partially raised on pillows for fifteen to thirty minutes at a time. This should be practiced for several days, gradually lengthening the time, before an attempt is made to sit in a chair. As strength returns, the amount of exercise permitted may be increased. As soon as the patient is able,

he should spend much of his time in the open air, getting as much sunshine as he can bear. A trip to the country, or, better, to the mountains, is a pleasant and profitable mode of recuperation. He should be fully instructed before departure, however, as to the danger of overexertion and, more particularly, as to that of overeating.

A bitter tonic should be prescribed, and iron or arsenic is often required for the anemia of convalescence. The elixir of iron, quinin, and strychnin is one of the most serviceable preparations at this time.

TYPHUS FEVER.

HOSPITAL FEVER, JAIL FEVER, CAMP FEVER, SHIP FEVER, SPOTTED FEVER.

Typhus fever is endemic in England, notably in London, in Ireland, Scotland, Russia, southern Europe, and Mexico. It is seldom met with in the United States. Cases have, however, occurred in Boston, New York, Philadelphia, and Baltimore, for the most part among Irish immigrants.

Definition.—A highly contagious acute infectious disease having a sudden onset and definite course, with macular eruption, terminating with crisis in about fourteen days.

Etiology.—Overcrowding, uncleanliness, poor food, poverty, and intemperance are the principal predisposing factors, hence the comparative frequency of the disease in jails and camps. The disease occurs at all seasons, but especially in winter and spring, probably because this is the season of overcrowding among the poor. The sexes are attacked about equally and no period of life is exempt. A majority of its victims are between 20 and 40, and those between 10 and 30 are about equal to those between 30 and 50.

The specific infectious agent is not known. A streptobacillus, a diplococcus, and a peculiar spirocheta have been found in the blood or other fluids by different investigators. It is one of the most contagious of the infectious diseases; in some epidemics the physicians and nurses have been almost universally attacked. This feature is much less frequently noted, however, in cases that occur in private residences, where careful attention can be given to methods of prophylaxis, including the removal of all unnecessary furniture, carpets, and draperies from the room, and to the disinfection of the patient, especially his mouth and nostrils. The infection is believed to be transported by the desquamated epidermal scales, by the sputum of the patient, and by fomites. The poison may be retained in articles of clothing for several months.

Morbid Anatomy.—No characteristic lesions are found in any of the organs. The eruption remains upon the skin after death, and ecchymoses are found upon dependent surfaces. The blood is dark and abnormally fluid. It has been compared to a mixture of serum and snuff. The muscles have a dark red color, frequently show granular degeneration, particularly in the heart, and sometimes extravasations of blood. Myocarditis is often found, and the endocardium may be reddened. There are no constant lesions in the intestines, but the lymph-follicles are frequently enlarged without ulceration. The liver is enlarged and soft, and the spleen is usually large. The kidneys are hyperemic and may show the changes of nephritis. The bronchial mucous membrane is con-

gested and coated with mucus; sometimes lobular pneumonia, less frequently lobar pneumonia or pleuritis, is found. Hypostatic congestion of the lungs is quite common. The discoverable lesions of the nervous system are confined to slight congestion of the cerebral and spinal meninges, sometimes with effusion of serum into the subarachnoid spaces and ventricles.

Symptoms.—The incubation lasts from nine to twelve days, during the last two or three of which there is a feeling of languor or a loss of appetite, and headache. The *invasion* is usually sudden, with a distinct rigor or a succession of chills for several days. Severe headache, pain in the loins and limbs, with profound prostration, early confine the patient to his bed. The face is flushed, the eyes expressionless, the conjunctivæ reddened, the pupils contracted. The tongue has a white fur, soon becomes dry, and sordes form upon the teeth. Nasal catarrh and bronchitis are generally present. Persistent vomiting may be a troublesome symptom. The patient complains of ringing in the ears, of black spots before the eyes, and the nervous manifestations increase in severity until delirium supervenes.

The temperature rapidly rises, often reaching 104° F. (40° C.) on the first evening. It may attain its maximum, 105°, 106° F. (40.5°—41.0° C.) or even higher, on the second or third day. Its course is almost uniform, showing but slight remissions. The pulse is rapid and full; less frequently dicrotic than in typhoid fever. The heart sounds become indistinct, and a systolic murmur is often heard. The respiration is moderate. The urine shows the usual febrile changes—diminution of quantity, with increase of solids, especially of urea and coloring matter, decrease of chlorids, and often a trace of albumin.

The eruption appears on the third to the fifth day, first on the abdomen and upper part of the chest, then on the extremities and face. It has the form of distinct dark red papules under the cuticle, which soon become hemorrhagic and petechial in character. The skin between the papules is often reddened. Herpes is rarely present. The general integument is dry. A furfuraceous desquamation during convalescence has been described. In mild cases the eruption occasionally fades away without passing into the petechial state, and the symptoms begin to abate with the decline of the fever, about the seventh day. Some epidemics have been characterized by the great number of mild cases, and others by the remarkable severity of all. In severe cases the temperature remains high, delirium develops and often becomes violent or it may deepen into coma. Coma-vigil is often seen. The bronchitis may pass into lobular pneumonia, hypostatic congestion becomes marked, and death may ensue from exhaustion. If the case is to terminate favorably, a crisis occurs usually on the 17th day, occasionally a little earlier or later. The temperature sinks almost uniformly to the normal within 24 to 48 hours, and all the symptoms rapidly abate. The crisis is accompanied with profuse sweating or free micturition. An extreme elevation of temperature, sometimes reaching 109° F. (42.7° C.), generally precedes a fatal termination and is sometimes observed before the crisis. Relapses are extremely rare.

Complications.—The complications are those ordinarily seen in a severe febrile disease. In some epidemics gangrene of the toes, fingers, or

nose, and, in children, noma, have been observed. The sequelæ are few. Sometimes anemia persists, and neuralgia or paralysis, probably due to neuritis, has been seen.

Diagnosis.—Owing to the resemblance of mild cases to typhoid fever the diagnosis may for a time be difficult, particularly in the absence of an epidemic. It can generally be determined by the following points: (1) The onset is more abrupt, often with a pronounced rigor. (2) The nervous manifestations appear earlier and are usually more severe. (3) The eruption comes out earlier, is more abundant and of a petechial character, a type rarely seen in typhoid fever. (4) The headache and pains in the limbs are more severe. (5) The temperature is higher, more uniform, terminates earlier and by crisis.

Prognosis.—This is determined chiefly by the character of the attack, the severity of the epidemic, and the general condition of the patient. The mortality is as low as 6 or 7 per cent in mild cases, but may exceed 20 per cent in others.

Treatment.—Owing to the contagiousness of the disease, the patient should be immediately isolated, and the most rigid antisepsis should be practiced. The general indications are the same as those for typhoid fever. Hydrotherapy affords the best means of combating the fever and the depression of the nervous system. In addition to this, the treatment should be supportive and stimulating. Alcohol should be freely given, with an abundance of milk and beef-juice, and the heart's action should be supported with strychnin. Every effort must be made to maintain the strength of the patient until the infection has expended its force.

RELAPSING FEVER.

RECURRENT FEVER, RELAPSING TYPHUS, SEVEN-DAY FEVER, FAMINE FEVER.

Relapsing fever has prevailed more or less epidemically in various parts of the Old World at different periods during the last two hundred years, possibly from antiquity. It was brought to America by Irish immigrants in 1844, but has not been encountered here since 1869.

Definition.—An acute infectious disease caused by the spirillum of Obermeier, characterized by from two to five febrile paroxysms, each lasting about six days and separated by a febrile interval of the same duration.

Etiology.—The specific cause of the disease is a spirocheta, a delicate filamentous spirillum about 30μ in length, or five times the diameter of a red blood-corpuscle. These are found in the blood during the febrile paroxysms and are then actively motile. Shortly before the crisis they disappear and remain absent during the afebrile period. They have not been detected in any of the other fluids of the body, but are found in the spleen and very rarely in the blood after death. Inoculation with the blood of a patient produces the disease, even in one who has previously passed through an attack. The blood-serum of a person having the disease contains, at certain periods, a substance which is exceedingly toxic to the spirillum. The disease is contagious to a slightly less degree than typhus fever. In some epidemics, however, this feature is more prominent than in others.

A Russian physician has recently investigated the possible transmission of the disease by insects, and found numerous spirilla in the bodies of bedbugs that had feasted upon patients with relapsing fever. From this it is inferred that the disease may be transmitted by the bug to other individuals.

Morbid Anatomy.—There are no typical lesions. Ecchymoses are generally found. After death during a paroxysm, the spleen is large and soft, and cloudy swelling may be found in the parenchyma of various organs. Infarcts are sometimes seen in the spleen and kidneys.

Symptoms.—The incubation period is generally from five to eight days. Prodromal symptoms are generally absent. The invasion is abrupt, usually beginning in the morning with one or more chills; the temperature rapidly rises to 102° or 104° F. (38.9°—40° C.) by the first evening, with headache, violent pains in the limbs and back, and extreme prostration. Vertigo, nausea, and vomiting are sometimes present. The breath is fetid. The pulse ranges from 110 to 130. The liver and spleen rapidly enlarge and become sensitive to pressure. Profuse sweating is usual. There is no eruption, as a rule, but occasionally herpes, petechiæ, or miliary vesicles are observed. The prostration, restlessness, and fever reach their greatest severity from the fourth to the sixth day. There is often a sense of oppression in the right hypochondrium, and great dyspnea. The crisis occurs while the disease is at its height, usually on the fifth or sixth day, rarely as early as the third or late as the tenth day. A profuse sweat comes on and the temperature drops from 7° to 10° F. during a single night. The pulse becomes slow and all the symptoms subside. Old persons often sink into collapse. Convalescence is rapid, but recovery becomes more delayed with the repetition of the paroxysms. In some instances the disease terminates after the first paroxysm. A fatal termination may follow collapse or result from prostration or heart-weakness, but it is generally due to some complication.

Bilious Typhoid.—A special form of the disease, in which pernicious jaundice greatly adds to its gravity, has been described under this name. A mild icterus is sometimes observed, and other deviations from the usual course are not uncommon.

Complications are not frequent. The most important are pneumonia, nephritis, hematuria, hematemesis, rupture of the spleen, and paralyses. Iritis and other ocular affections are encountered. Pregnant women frequently abort.

Diagnosis.—The disease often passes unrecognized until the first relapse occurs. Examination of the blood during a paroxysm reveals the spirilla.

The prognosis is favorable except in the bilious form or after repeated relapses with complications.

Treatment.—Beneficial action is claimed for quinin, calomel, arsenic, methylene blue, potassium iodid, and other remedies, but there is no unanimity of opinion in regard to any of them. Hydrotherapy is beneficial, although the spirilla are capable of prolonged life in blood of normal temperature. Injection of serum from immunized animals has been resorted to during the first afebrile period, with arrest of the disease in about half the cases treated. In other respects the treatment is symp-

tomatic. The strength of the patient must be supported and the special symptoms and complications must be combated as they arise, by the methods employed in typhoid and other fevers.

INFLUENZA.

LA GRIPPE—THE GRIP, CATARRHAL INFLUENZA, EPIDEMIC INFLUENZA.

Definition.—An acute endemic or epidemic, often pandemic, infection caused by the bacillus of Pfeiffer and characterized by a strong tendency to attack the respiratory mucous membranes.

Etiology.—The bacillus of Pfeiffer is found in the blood and nasal mucus, but more abundantly in the bronchial secretion, during the attack and sometimes for a long time after. It is a small, short, non-motile rod with bulbous ends, staining freely with a dilute aqueous solution of carbol-fuchsin. The disease is highly contagious and spreads with rapidity. Some investigators think that it can be carried by the air, and there is some evidence of its transmission by a third person or by fomites. Epidemics are more frequent during the winter, but have occurred in the warmer months. Adults are more susceptible than children, but no age is exempt. In pandemics few are spared. One attack renders the individual more liable to future infection. Sporadic cases usually occur for several years after an epidemic. The common sporadic influenza is a separate infection (influenza nostras), but its etiology is not known. The influenza poison is thought to be antagonistic to that of malaria, since a marked decrease in the prevalence of the latter disease has been observed during an epidemic of the former.

Symptoms.—The disease appears in so many forms that it is customary to consider them separately. Clinically, however, the distinction is not always clear, for one type may blend with another or merge into it. The incubation varies from one to four days. Prodromal lassitude, headache, and dullness are sometimes observed.

General Symptoms.—A train of symptoms is more or less common to all cases. The invasion is generally abrupt, with a chill or chilly sensations and a rapid rise of temperature, often to 104° or 105° F. (40°—40.5° C.), intense headache, tenderness and aching of the muscles and joints, which are independent of motion. Mental and physical depression, restlessness, and insomnia are often extreme. Catarrhal symptoms are generally, though not invariably, present. Franke has recently called attention to a peculiar redness of the mucous membrane of the mouth, particularly the gums and tongue, with swelling of the papillæ at the extremity of the tongue often equal to that seen in scarlatina. He regards it as pathognomonic of the disease. The temperature pursues an irregular course and not infrequently terminates by crisis. The pulse is usually rapid and feeble, sometimes intermittent; in other cases it is extremely slow (bradycardia).

Respiratory Type.—This is the most frequent form. It may affect the entire respiratory system from the nose to the air-cells. The symptoms are much the same as those of an ordinary catarrhal fever, but the fever is more intense and the prostration greater. There is frequent sneezing, suffusion of the eyes and lachrymation, often accompanied with pharyngeal irritation, hoarseness, and cough. The cough is at first

dry and excessively irritating. In a day or two, however, there is abundant bronchial secretion, which rapidly becomes purulent. The sputum is generally of a pale green color and is ejected in firm lumps. Dyspnea is often a prominent symptom. Cyanosis may result from the extension of the inflammation to the finer tubes or from edema of the lungs.

Nervous Type.—In this form the patient is often suddenly seized with severe headache, muscular pains or neuralgia, and profound mental and physical prostration. There are frequently cutaneous hyperesthesia and sensitiveness to light and sound, rigidity and tenderness of the neck muscles. Convulsions are not uncommon, and delirium with hallucinations appears in some cases. Some cases sink into a typhoid stupor; the resemblance to meningitis is often striking. The analogy to typhoid fever was most pronounced in cases observed by Pelon and by Feindel and Froussard, in which lenticular rose-spots appeared. The Widal test was negative, however, in all the cases. The recovery is generally slow, and melancholia and great mental inactivity often persist for some time.

Gastrointestinal Type.—This is characterized by persistent nausea and vomiting, or by intense abdominal pains and diarrhea. The fever may be high. The symptoms are often suggestive of appendicitis, if, indeed, the appendix is not sometimes implicated. Jaundice sometimes develops.

Complications and Sequelæ.—Lobar and bronchopneumonia are the most frequent and serious complications on the part of the respiratory system. Both are peculiar in the great diversity of the pathological lesions found in fatal cases. Pleurisy and empyema are not infrequent. Bronchitis characterized by a preponderance of streptococci in the sputum has been noted, especially by Forchheimer. The complications on the part of the circulatory system are of special importance and are attributed to the direct action of the influenza toxin. Tachycardia and bradycardia are alike frequent. Pericarditis, myocarditis, phlebitis, and thrombosis are occasionally developed. Endocarditis is rare, and Sansom attributes the systolic murmur that is sometimes heard at the apex during or after the attack to a progressive degeneration of the myocardium which cannot be compensated for by hypertrophy of the ventricles. Neuritis, hemiplegia, monoplegias, myelitis, otitis, and various affections of the eye have been observed. A pre-existent tuberculosis and cardiac or renal disease are always intensified by an attack of influenza; even the encroachment of old age sometimes seems to be hastened by it.

Prognosis.—Uncomplicated cases usually terminate in recovery. The mortality is due chiefly to the complications or to the aggravation of pre-existent disease. On this account the general mortality list of a locality shows an increase during the prevalence of an epidemic which is in but small part accounted for by the deaths directly due to the grip. The prognosis is especially unfavorable in alcoholic subjects and in very aged persons.

Diagnosis.—The disease is differentiated from other catarrhal affections chiefly by the severity of the pain and the profound prostration. In the presence of a pandemic, the diagnosis is seldom difficult. The bacteriological examination removes all doubt.

Typhoid fever, as compared with influenza, shows a longer prodromal stage and slower elevation of temperature; epistaxis is frequent, the rose-rash much more constant. The Widal test establishes the diagnosis.

Cerebrospinal meningitis rarely suggests this disease. The catarrhal symptoms are usually absent; the character of the epidemic is different.

Dengue is a disease of warm climates and is generally distinguishable by its peculiar febrile paroxysms, early exanthem, and enlargement of lymph-glands.

Treatment.—Prophylaxis is difficult, but may be attempted by general regard for the health and avoidance of fatigue and loss of rest. Old persons should be as much as possible protected from exposure to the infection.

The patient should be confined to bed during the entire attack, in order to avoid complications. An attempt may be made to abort the disease. With this end in view, the patient should take a hot bath the first night and follow it with a glass of hot lemonade. He should then be warmly covered in bed in order to induce free sweating. A dose of calomel, grs. ij to v (0.13—0.32), may be given, and followed with a Seidlitz powder in the morning. Quinin and Dover's powder are excellent remedies, to which belladonna may be added to reduce the coryza. The fever and pain are relieved by 5 to 10-grain (0.32—0.64) doses of phenacetin, to which 2 grains (0.13) of citrated caffein should be added, particularly if the heart is weak. The prostration calls for stimulation, champagne if the stomach is irritable, or brandy and strychnin, gr. 1-60 to 1-40 (0.001—0.0016), three times daily. One-twelfth-grain (0.005) doses of heroin are best for the cough. A menthol and camphor spray relieves the nasal and pharyngeal irritation. A solution of the extract of suprarenal gland or of adrenalin (1 :1000) has yielded good results. In the abdominal form of the disease the diet must be carefully regulated and in some cases should be restricted to milk. Broths, poached or soft-boiled eggs, milk-toast, and custard may generally be allowed, and eggnog and milk when stimulation is required.

DENGUE.

BREAKBONE FEVER, DANDY FEVER.

Dengue is a disease of tropical and subtropical regions and is most prevalent in the East and West Indies, India, and Egypt. It is usually confined to the coast and valleys, but in 1870-73 it spread over the whole of India. During the warmest weather it sometimes invades the more northern countries. In 1880 it reached Charleston, S. C., and Augusta, Ga., and in 1897 and 1898 it prevailed to some extent in Georgia and Florida.

Definition.—An acute epidemic or pandemic exanthematous infection occurring in two febrile paroxysms with excruciating pains in the head muscles and joints.

Etiology.—A specific germ has not been demonstrated. The infectious agent is generally believed to be conveyed by the air, by fomites, or by direct contact. It is exceedingly virulent, often attacking fully 75 per cent of the inhabitants of a district within a few weeks. Heat and

humidity favor its transmission. Susceptibility is almost universal and uninfluenced by age or sex. The recent investigations of Graham, of Beyrouth, indicate that the mosquito (culex) may inoculate the disease. He carried mosquitoes that had bitten dengue patients up into the mountains, far from any recognized source of infection, and saw the disease develop in two healthy young men who voluntarily submitted to their bites.

Symptoms.—The incubation lasts three to five days. The attack consists of three periods, two febrile paroxysms and the interval. The invasion is generally abrupt, with chilliness, sometimes a rigor, rise of temperature to 103° F. (39.5° C.) or higher, even to 107° F. (41.5° C.), prostration, and excruciating pains in the eyeballs or back of the head, the loins, and limbs. A deep flushing of the face may be the first symptom. The tongue is furred; gastric oppression and vomiting may occur. The initial rash often invades the entire body and the visible mucous membranes. An area around the eyes often becomes a deep purple. The throat may be congested and sore. This stage lasts from one to four days; it often terminates by crisis with profuse sweating, diarrhea, diuresis, or epistaxis. The pain subsides, and the patient, although weak, may feel well.

The interval lasts but three or four days, then there is a return of fever and pain, with a roseolar eruption (the terminal stage). The relapse is usually milder than the first paroxysm; either the fever or the eruption may escape notice. Reddish brown spots appear on the palms and backs of the hands, with prickling and tingling, and quickly spread. On the body the spots coalesce into areas of variable size or into an unbroken mantle. The attack lasts only a few hours or several days. A furfuraceous desquamation follows, which may last three weeks. Recovery is usually rapid. Relapses are common. Convalescence may be delayed by continuance of the pains, anorexia, and weakness; and boils, urticaria, or other eruptions, with intense pruritus, may occur. Other complications and sequelæ are rare.

Diagnosis.—Influenza, malaria, rheumatism, and yellow fever are to be excluded. *Influenza* is a disease of colder climates and winter. It is characterized by catarrhal symptoms without eruption, adenitis, or intermission. The bacillus may be found in the nasal and bronchial secretions. *Malaria* is excluded by the chills or continued fever, the absence of coryza, and the presence of the plasmodium in the blood. Both articular and muscular *rheumatism* are more frequent in the cold months and in regions exempt from dengue. They have no eruptions or intermissions. Sweating is profuse in the articular form, seldom in dengue. The continued fever, albuminuria, jaundice, black vomit, and grave nervous manifestations of *yellow fever* are distinctive.

The prognosis is good. Fatal convulsions may occur, however, in young children. A malignant type of the disease with fatal edema of the lungs has been seen in Calcutta.

Treatment.—This is directed to the relief of pain and the support of strength. Light liquid diet, rest, the avoidance of chill, the administration of a saline diaphoretic with aconite, and cold applications to the head at the outset are recommended by Manson. Phenacetin and belladonna relieve the pain; morphin may be required. Stimulants should be

avoided. During convalescence small doses of potassium iodid and quinin, with the application of electricity, are beneficial.

CHOLERA.

ASIATIC CHOLERA, EPIDEMIC CHOLERA.

Cholera belongs to India. It has prevailed there almost annually for centuries, repeatedly becoming pandemic and spreading over Asia and a great part of Europe. It did not reach America until 1832, when it was brought by emigrant ships to Quebec and New York. Cases occurred also in 1835-36. Entering at New Orleans in 1848, it extended up the Mississippi and westward to California, and recurrences appeared in 1849. In 1854 it again entered at New York and spread over a greater part of the country. Mild epidemics occurred in 1866, 1867, and 1873. A remarkable reduction in its prevalence in the Philippine Islands has been noted since the advent of United States control.

Definition.—An acute infectious epidemic disease caused by the comma bacillus of Koch, and characterized clinically by severe purging and vomiting, with collapse that often proves fatal.

Etiology.—The comma bacillus, or spirillum, is conveyed to the human being by drinking-water or food which it has contaminated. It is capable of living on meat, milk, butter, or other raw food for a week, in water for perhaps a longer time. The ultimate source of infection is always a previous case. The germs are found in great numbers in the dejections and the vomit of patients. They flourish outside of the body, however, under the influence of heat and moisture, particularly in cesspools and decomposing animal matter. Infection is favored, therefore, by hot weather, bad sanitation, and uncleanly habits. The poison is carried in such articles as clothing or baled rags, but not by the air. Caravans and ships have been the principal carriers of it. The disease is not highly contagious so long as contamination is avoided. Washerwomen are more exposed than nurses. Students have become infected while working in the laboratory with the cultures. The disease prevails best at or near the sea-level and rarely reaches the higher altitudes.

Age and sex are of little importance in etiology. Fear, and physical debility from age, intemperance, or illness, favor infection and retard recovery.

Bacteriology.—The cholera vibrio is an actively motile organism resembling a comma or an S. It is about half as long and thicker than the tubercle bacillus. It grows rapidly, but is not extremely tenacious of life. Drying or exposure to the air for three hours kills it. It will live in water rich in organic matter for a month or more. It grows best at a temperature between 86° and 104° F. (30°—40° C.). A temperature of 104° F. (40° C.) destroys it, but freezing does not. In the body it is anaërobic; without, it is aërobic. There is probably no natural immunity further than that afforded by the power of the healthy gastric juice to destroy the bacilli. But living bacilli have been found in the stools of healthy men during an epidemic. It is noteworthy also that lower animals are not infected by cultures introduced into the stomach unless the gastric juice has first been neutralized.

Morbid Anatomy.—The body appears emaciated and shrunken, the face drawn, and the skin of dependent portions mottled. A post-mor-

tem elevation of temperature is often noted. Rigor mortis comes on early and is extremely developed, and muscular contractions frequently occur after death which cause the eyes, jaws, or limbs to move or the whole body to change its position. In fulminant cases, dying within the first three or four hours, there are no internal lesions, except frequently a distention of the bowels with a flocculent fluid. In ordinary cases the tissues appear dry, the serous membranes feel sticky. The blood is dark and concentrated. The intestines are shrunken and thin, often congested throughout. Within them is found a thin flocculent serum, the same as constitutes the "rice-water" dejecta so characteristic of the disease. The flocculi consist of desquamated epithelium and large numbers of the spirilla and other micro-organisms. The spirilla are found also in the ntestinal walls and lymph-vessels in cases that have not been rapidly fatal. Parenchymatous degenerations and sometimes areas of coagulation necrosis and desquamation of tubular epithelium are found in both the liver and kidneys. The spleen is usually small. The heart is dark, and the left ventricle, as a rule, is contracted. The lungs are congested, particularly at the bases.

Symptoms.—The period of incubation usually lasts from two to five days; it is perhaps shorter in some cases. It may pass without symptoms, or it may be marked by abdominal distress, slight pain and tenderness, visible peristaltic movement, or moderate diarrhea and depression. This stage is sometimes called the prodromus. The course of the disease is generally divided into three stages, that of serous diarrhea, that of collapse (or the algid stage), and the stage of reaction.

1. *The Stage of Serous Diarrhea.*—Following the prodromal symptoms the discharges become more frequent and more profuse, or, if no prodromes have been present, the onset is indicated by a rapid succession of thin, copious dejections, with or without severe griping pain and tenesmus. Extreme prostration and collapse, with violent cramps in the legs and feet, sometimes precede the diarrhea. Urgent thirst develops, and persistent vomiting follows within a few hours, first of the undigested contents of the stomach, later of transuded fluids. Hiccoughing often accompanies it. After the normal fecal matter has been discharged, the stools become thin and almost clear, often having a specific gravity below 1.010 and an alkaline reaction. They contain flocculi which give them the "rice-water" appearance. Blood is rarely present. The collapse rapidly becomes extreme. The face appears shrunken, and often has a bluish, livid color; the lips are almost black; the conjunctivæ dry and congested, the pupils small, and the eyes have a vacant stare. The cheeks alone are flushed. The mouth is drawn and the nose is pinched, the alæ vibrate. The abdomen becomes flat and flabby. The patient appears to dry up. All the secretions become diminished and are finally arrested, with the exception, it is stated, of the lacteal secretion in nursing women. Total suppression of the urine may last for days. Violent cramps develop in the abdomen as well as in the legs. Emaciation becomes extreme. The skin is "icy" cold and hangs in folds. A young person appears old and wrinkled within a day.. The rectal temperature may, however, register 102° F. (39° C.). In fulminant cases death often occurs before the diarrhea has commenced (cholera sicca), and, owing, perhaps, to paralysis, the intestine is found distended with

the fluid. The stage of diarrhea may last from 2 to 24 hours and then merges into the algid stage.

2. *The Algid Stage, or Stage of Collapse or Asphyxia.*—This results from the extreme concentration of the blood, and the feebleness of the heart which permits it to stagnate in the capillaries. Respiration becomes feeble and cyanosis develops. Tonic spasms seize the muscles of the abdomen, arms, and legs, and the suffering is intense. The voice is lost. The pulse, if discernible, is moderately rapid, 100 to 120, but soon fades away, as the patient sinks into a coma. The alvine discharges become converted into a constant dribbling from the relaxed anus. Death may occur from asphyxia or from heart-failure. Not infrequently the patient lies for hours in a state so close to death that it is difficult to determine at what moment life becomes extinct. In other cases a typhoid condition develops, with rise of temperature, full or flickering pulse, and delirium. This is sometimes accompanied by a "cholera eruption," a roseola, erythema, or urticaria. It may finally end in recovery, but usually passes again into fatal collapse, which has been attributed to uremia.

3. *Stage of Reaction.*—When the patient passes the stage of collapse, his condition gradually improves, with a cessation of the diarrhea, vomiting, and cramps; the skin becomes warm and the secretions are restored. The convalescence lasts two or three weeks. Diarrhea may persist. Relapse often occurs.

Cholerine.—During an epidemic not a few cases of so-called cholerine are encountered, cases with diarrhea, cramps, and prostration of a milder character than belong to true cholera and without complete collapse or anuria.

Complications and Sequelæ.—Albuminuria often follows the anuria, and the fatal issue is often attributed to uremia or sepsis. Bedsores, superficial ulcers, furuncles, occasionally gangrene and diphtheritic inflammation of the mucous membrane of the throat, colon, or genitals, have been observed. Abscesses sometimes form, especially in the parotid gland. Broncho-pneumonia develops in some cases.

Diagnosis.—It is only the first cases in an epidemic that occasion difficulty. Cases of cholera nostras are sometimes almost identical with true cholera. A serum reaction similar to the Widal test has been found almost universally present in a considerable number of cases, with serum diluted from 1:10 to 1:100, and as early as the first or second day. But the bacteriological examination is more conclusive. A gelatin culture from the dejections will produce a characteristic growth in a few hours.

Prognosis.—The mortality in different epidemics varies from 40 to 90 per cent. Much depends upon the physical condition of the patient and no less upon the promptness of the treatment.

Prophylaxis.—The patient should be isolated and a strict quarantine established. It is only as a result of our national quarantine methods that the disease has been kept out of this country for the last 30 years. The most rigid antiseptic measures must be practiced. All discharges from the patient should be immediately treated with a strong disinfectant solution and, if possible, destroyed by fire. The same solutions and methods of disinfection may be employed as in typhoid fever. (See

p. 72.) Articles that have come into contact with the patient should be burned or thoroughly disinfected and exposed for days to the sun's rays. The dead should be disinfected and immediately placed in a hermetically sealed coffin or cremated. Water used for drink or for bathing should be boiled, and market vegetables and fruit should not be eaten raw.

Treatment.—A full hypodermic dose of morphin, gr. ⅓ to ½ (0.02—0.03), should be administered immediately, and repeated upon return of the pain. This should be followed with the deodorized tincture of opium in doses of 20 drops (0.75) at intervals sufficiently short to hold the case under control and to keep the patient quiet. All food must be withheld during the diarrheal and algid stages. The thirst may be relieved with chipped ice and ice-water acidulated with dilute hydrochloric acid, gtt. xxx every two hours, since it is antagonistic to the bacilli. Strength should be maintained with brandy and black coffee by the rectum, and strychnin nitrate hypodermically in doses of gr. 1-20 (0.003). After vomiting has been checked, milk may be given in small quantities at regular intervals. The surface temperature must be maintained by applications of dry heat, and the abdominal pains relieved by hot fomentations.

Saline injections afford a most valuable means of counteracting the depletion of the blood. The normal salt solution is injected into the subcutaneous tissue of the abdomen at a temperature of 104° F. (40° C.), allowing from one to two quarts (liters) to flow by gravity through the hypodermic needle. *Enteroclysis* often produces excellent results. From two to four quarts of a 2-per-cent solution of tannic acid at a temperature of 110° F. (43° C.) should be injected, preferably through the long rectal tube. Both these measures are useful in overcoming the anuria.

Antitoxin Treatment.—Better results have been reported from the use of this method as a prophylactic measure than in treatment. The inoculation is made first with a weak culture, and five days later with a stronger one. Immunity develops in five days after the second inoculation.

YELLOW FEVER.

BLACK JACK, THE BLACK VOMIT.

Yellow fever is endemic in the tropics. Guiteras has given us the following useful classification of the areas of infection: 1. The focal zone, from which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. 2. Perifocal zones, or regions of periodic epidemics, including the ports of the tropical Atlantic in America and Africa. 3. The zone of accidental epidemics, between the parallels of 45° N. and 35° S. latitude. It is noteworthy, however, that under the supervision of the United States authorities, Havana has been removed from the first to the second or third of these classes.

Definition.—An acute infectious disease probably caused by the bacillus icteroides, and having as its most distinguishing features a peculiar jaundiced, congested facies, fever with slow pulse, hematemesis (black vomit), and albuminuria or total suppression of the urine.

Etiology.—The bacillus icteroides, of Sanarelli, is now generally accepted as the cause. It is a slender, motile, facultative, anaërobic ba-

cillus from 2 to 4μ in length. Its etiological relation is supported: (1) by its frequent presence in the blood and viscera of the dead, (2) by a serum test in which the bacilli become agglutinated and motionless after the manner of typhoid bacilli in the Widal test, and (3) by the production of the disease in man through inoculation experiments conducted by Sanarelli and others. The disease is not directly contagious, and recent investigations apparently disprove the old belief that the poison is carried by fomites. If the investigations made by the Committee of the Pan-American Medical Congress be correct, the disease is produced only by inoculation, and the only known mode of its transmission from one individual to another is by the mosquito (*Stegomyia fasciata*). This discovery supports the old theories that the disease is most infectious at night and in low-lying districts, and that it may be carried to some distance by air-currents. The same investigations seem to show that the bacillus remains in the body of the mosquito from 12 to 18 days, the length of time depending upon the temperature of the air, before it can be inoculated into a human being. The recent investigation in regard to the transportation of mosquitoes on shipboard, taken in connection with these probable facts, seems to explain the sudden outbreak of the disease in places remote from a known center of infection, as in Galveston, in 1897, after an absence of 30 years, an occurrence which occasioned much discussion and no little criticism of the physicians who recognized the character of the disease.

Season.—The disease is favored by high temperature, from 72° F. (22° C.) and upward, and by a high degree of humidity. It is quickly arrested by frost, which kills the mosquito, but freezing does not entirely devitalize the bacillus. In tropical countries it may prevail the year round. It usually reaches our shores in the autumn.

Age and Sex.—The disease attacks people of all ages, but is less frequent toward either extreme of life. Children appear more susceptible than adults. Infants have the disease, if at all, in a mild form. The negro is to a certain extent immune and usually recovers.

During an epidemic such influences as fatigue, heat prostration, worry, fear of the disease, alcoholism, and debauchery increase susceptibility.

Immunity.—One attack generally confers immunity, but second attacks have been observed. Protracted residence in the North and long absence of the disease from a locality are thought to overcome immunity. The question arises, may not the immunity be sustained by repeated inoculation of the disease by the mosquito?

Morbid Anatomy.—The skin is jaundiced, and ecchymoses are frequently seen in it. The icteric hue is said to deepen after death. The urine and other fluids are yellow. The blood shows little change in cases of moderate severity. The bacillus is occasionally found in it. In severe cases free hemoglobin is found in the blood-serum as a result of destruction of the red corpuscles, and these corpuscles usually show degenerative changes. Fatty or other degeneration is more or less constantly found in the heart, liver, and kidneys. The liver in early cases is congested, but in cases of longer duration it is pale. The hepatic cells show fatty degeneration and necrosis, and the small bile-ducts are gorged with desquamated, degenerated epithelium. The bile in the gall-bladder is thick and dark. The brain and meninges are congested.

Hemorrhagic infarcts are sometimes found in the lungs and elsewhere. The mucous membranes, particularly of the stomach, show ecchymoses and erosions, and similar changes are found in the serous membranes. The black, tarry matter which constitutes the black vomit is found in the stomach and intestines of hemorrhagic cases.

Symptoms.—The incubation period is from 18 hours to a week, usually three or four days, being shorter in proportion to the severity of the disease. Prodromes are usually absent. The invasion is sudden and generally occurs in the morning hours. The clinical history is divided into three stages:

First Stage.—Chilly sensations, sometimes a rigor, or in children a convulsion, with headache, severe pains in the back and calves of the legs, are almost constant symptoms. The temperature rises during the chill to 103° or 105° F. (39.5°—40.5° C.). The cheeks and conjunctivæ immediately become bright red with capillary dilatation. The eyes are watery and staring. The lips and eyelids are generally puffy, and a slightly icteric tinge may be detected in the conjunctivæ and skin of the face on careful examination, often on the first day. These appearances are characteristic of the disease. Vomiting begins on the first day, as a rule; the vomitus consists first of the contents of the stomach, then of a thin, grayish mucus, and finally, in severe cases, of blood. The pulse is seldom over 100, full and soft, in the beginning, and becomes slower and more feeble as the disease progresses, until it is perhaps 50 or less. The epigastrium is so sensitive that slight pressure causes vomiting. The tongue is dry and pointed, the gums swollen and red, often bleeding. The skin is at first dry; it sometimes becomes moist and then has a peculiarly offensive odor. The urine is scant, highly acid, and albuminous, particularly in the evening. By the second or third day the jaundice deepens to a saffron color. The temperature, as a rule, remains high until the third or fourth day, then subsides and often becomes subnormal.

Second Stage.—This is known also as the "calm" or "remission." With the decline of the temperature the other symptoms abate and the patient experiences a relief. In children this may occur as early as the second day. In mild cases it is permanent and marks the beginning of convalescence. In another group of cases destined to recover, the convalescence is delayed by recrudescence of two or three days, with irregular reactionary fever. In severe cases, however, the calm lasts but a few hours, possibly 36, and the patient rapidly declines into a collapse.

Third Stage, or Collapse.—This is marked by suppression of urine and hemorrhages from the mucous membranes, particularly of the stomach. Fever may be present, but the temperature sometimes remains normal. In fatal cases it often rises to 108° or 110° F. (43° C.) before death. The disease is of short duration, not usually exceeding a week. Relapses occasionally occur.

Special Symptoms.—*Facies.*—Even on the morning of the first day the face is flushed more than in any other acute infection, and the eyelids and lips are swollen. The superficial capillaries of the face and those of the conjunctivæ are dilated, and on close examination slight icterus can be recognized. The eyes have a peculiar stare and a distinctive "alertness."

Fever.—The temperature may be only moderately high in an ordinary case. After the initial rise it often subsides on the second day to 102° or 103° F. (39.0°—39.5° C.). It is then irregular in its course and may terminate by lysis. A rapid fall below normal often precedes collapse, and a rapid rise a fatal termination.

The pulse is slow and out of normal ratio (4:1) to the temperature from the beginning, and, although the temperature may be still rising, it becomes progressively slower. It sometimes reaches a rate of only 35 to 50 during defervescence. Occasionally it becomes rapid and irregular, reaching 120 or more. At first full, it becomes extremely feeble in fatal cases. Respiration is usually accelerated, sometimes irregular; dyspnea may be extreme in the later stages.

Black Vomit.—Extreme irritability of the stomach is a constant symptom from the beginning. Black vomit occurs in about one-third of the cases. When the blood is copious there is usually severe pain in the stomach and esophagus. Hemorrhages from the gums, nose, eyes, kidneys, and uterus frequently occur, and petechiæ may appear in the skin. The bowels are usually constipated. The stools have not the clay color of jaundice; they are frequently black, from the presence of blood.

Albuminuria generally appears not later than the evening of the third day even in the mildest cases. An intense nephritis, with much albumin and casts, develops in severe cases; complete suppression may occur and lead to fatal uremia.

Mental Condition.—In mild cases the mind remains clear and the patient watches all that transpires. Delirium and coma develop when the disease is severe.

Clinical Varieties.—With regard to the severity of the manifestations, different types of the disease have been recognized. There are mild cases with moderate fever, slight or no jaundice, and early recovery. A transitory albuminuria may be present. "Walking" cases are not uncommon. A comatose type is recognized in which, without fever, the patient passes into a stupor on the first or second day, with great prostration, feeble pulse, and albuminuria. Death often occurs on the third day. Another class of cases is distinguished by violent delirium from the beginning.

Complications and Sequelæ.—These are not common and are generally of the same character as are encountered in other acute infections, as phlebitis and thrombosis of the femoral veins, acute nephritis, and suppurative parotitis. Fatal hematemesis has followed an error in diet several weeks after recovery. Pregnant women generally abort.

Diagnosis.—The three distinguishing features of the disease, as emphasized by Guiteras, are: the facies, albuminuria, and the slowing pulse, with maintenance or elevation of temperature. The urine should be examined in the evening. The headache, pain in the calves, gastric irritability, epigastric tenderness, and the black vomit are valuable factors in diagnosis. The agglutination test may be applied as early as the second day.

Dengue.—This is probably the most difficult disease to distinguish, since it so frequently occurs in the same localities and at the same seasons and is similar in onset and symptoms. It is not, however,

accompanied by so great weakness, gastric irritability, jaundice, albuminuria on the first or second evening, or hemorrhages. The pulse is rapid and the temperature rises more slowly. An eruption often appears. The blood would probably not agglutinate the bacillus icteroides.

Malaria, especially the irregular remittent, estivo-autumnal type, when accompanied by vomiting and slight jaundice, is often difficult of distinction. But the icterus does not appear so early, the face is usually dull, not alert; the tongue is broad, flat, and pale, not dry and pointed. The discovery of the plasmodium in the blood is distinctive.

Relapsing fever is readily recognized by the discovery of the spirilla in the blood, as well as by the slower onset, more rapid pulse, enlargement of the spleen, and the absence of black vomit and extreme gastric irritability.

Acute yellow atrophy of the liver is accompanied by gradual elevation of temperature, without pain or so great gastric irritability. The urine contains large quantities of bile pigments, leucin, and tyrosin.

Acute febrile jaundice (Weil's disease) is characterized by less severity of onset and less prostration; black vomit, albuminuria, and suppression are absent.

Prognosis.—The death-rate in epidemics is very different. It may be as low as 10 to 20 per cent or as high as 80 to 90 per cent. The prognosis is rendered less favorable by previous debility, anxiety or fear, alcoholism, pregnancy, or the puerperal state. Suppression of urine is an unfavorable symptom, and when this is accompanied by black vomit recovery rarely follows. Black vomit alone is not, however, extremely dangerous. The virulence of an epidemic appears to be greater in proportion to the length of time that has elapsed since the last preceding outbreak. Much depends upon the promptness with which treatment is instituted.

Prophylaxis consists less in the inspection and quarantine of ships from infected ports than in the isolation of the sick, with especial reference to the exclusion of mosquitoes. By the systematic warfare that has been waged upon these pests and their larvæ during the last two or three years, Havana has been freed from yellow fever for the first time in 150 years. Stress has always been laid upon the importance of burning all fomites, but if recent investigations prove to be correct this is not necessary. Susceptible persons have slept in the midst of infected linen for 28 days without infection so long as mosquitoes were excluded. Sanarelli has used horse serum, Freire and others that of immune persons, with some success.

Treatment.—Good ventilation and absolute rest are important. The patient should not be disturbed, the bedpan must be used, and nourishment and drink must be given through a tube. Removal of the patient to other quarters is harmful. Food should be administered by the rectum during the period of gastric irritability. This irritability calls for the administration of cracked ice, or, better, iced champagne. Dilute hydrocyanic acid may be given in 3-drop doses. Sternberg highly recommends the following mixture: Sodium bicarbonate, grs. cl (10.0); hydrargyri bichlorid, gr. ½ (0.02); pure water, Oij (1000), to be given in doses of three tablespoonfuls every hour. It has been found to reduce the gastric irritability, to maintain the urinary secretion, and in other

ways to reduce the mortality. Nunez has had good results from potassium bitartrate and salol. Quinin in 20-grain (1.30) doses was formerly much employed. The cardiac weakness, particularly in the second stage, should be met by stimulation with brandy and strychnin hypodermically or by rectum. The pains may be in a measure relieved by applications of heat and sinapisms; some patients prefer the ice-bag. Morphin should be used cautiously, if at all, since it has proved a dangerous remedy (Sternberg). For cerebral congestion ice-bags should be applied to the head and sinapisms to the feet. The tendency to hemorrhage often resists treatment; the acetate of lead and opium may be employed with caution. Good results have been obtained from hypodermic injections of ergotin.

Sanarelli's serum treatment is reported to have proved successful in a considerable number of cases.

During convalescence the greatest care must be exercised in order not to overtax the stomach. Alimentation must be begun cautiously; the food should be of the most delicate character and administered in small quantity. Such tonics as iron, quinin, and strychnin, separately or combined, hasten recovery.

THE PLAGUE.

BUBONIC PLAGUE, MALIGNANT ADENITIS, BLACK DEATH.

The plague is a disease of the Orient, where it has prevailed from antiquity. Its frequent prevalence in the Philippine Islands, its outbreak in Hawaii in 1899, importation to San Francisco in 1900, and continued presence in Mexico as late as the spring of 1903 have given it an importance to American physicians which it did not before possess.

Definition.—A virulent acute infectious disease caused by the bacillus pestis of Kitasato, running a rapid febrile course with bubonic swellings in different parts of the body, and often accompanied by hemorrhages from the mucous membranes. Three clinical forms are usually recognized, the glandular, the pneumonic, and the septicemic.

Etiology.—The bacillus pestis has been proved the cause of the disease by inoculation experiments in animals, as well as by its uniform presence in the body after death, particularly in the blood and enlarged glands. The bacillus is a short, thick rod with rounded ends. It is obtained with least difficulty from the bloody sputum of pneumonic cases, and may be cultivated on an alkaline agar medium. It is believed to have an independent existence outside of the body, in the ground. The avenues of its entrance into the body, it is generally believed, are the mucous membranes of the respiratory passages and the cutaneous surface after injury. The tonsil is also believed to be a possible portal of entrance. The poison is thought to be carried on clothing, bedding, and other articles, and to cling tenaciously to houses and localities. Epidemics have followed the opening of the graves of plague victims. Rats, cats, dogs, and other animals become infected. Rats especially are regarded as carriers of infection, even to distant lands, by gaining entrance into ships. They often die in great numbers before and during an epidemic. Flies, bugs, lice, and especially fleas are also capable of

conveying infection to man. The chief predisposing causes are overcrowding, filth, and deficient ventilation.

Season.—The disease is favored by warm weather and humidity, but outbreaks sometimes occur in winter. It is somewhat more frequent between the ages of 20 and 30; persons over 50 are seldom attacked. Both sexes are about equally susceptible. It is believed to be only mildly contagious.

Morbid Anatomy.—The lymph-glands of the inguinal or the femoral region, less frequently those of the axillæ or neck, are enlarged and firm, or in a state of suppuration. The overlying skin is edematous and much thickened. After death in the most rapidly fatal cases, the primary bubo, or lymph-gland nearest the site of inoculation, may be so small as to be found with difficulty. In it the parenchyma is destroyed. Necrotic or hemorrhagic areas appear, or suppuration may have occurred. Pyogenic cocci are frequently found in addition to the bacillus pestis. Ecchymoses and petechiæ are found on the surface of various parts of the body. The secondary buboes are intensely hyperemic and occasionally contain hemorrhages. The various hemorrhagic lesions are believed to be the direct result of the bacteria, and not due to the toxins. Parenchymatous and fatty degenerations of the heart, liver, and kidneys are common. The spleen is much enlarged and soft, being distended with blood rich in polymorphonuclear cells. In the pyemic form of the disease, metastatic foci of suppuration are found in the lungs, liver, spleen, and muscles, often surrounded by extravasated blood. The lung lesions in the pneumonic form are primarily lobular, but they may be so extensive as to cause the solidification of an entire lobe. Bronchitis is always found and the bronchial glands have the appearance of primary buboes.

Clinical Forms.—With reference to its severity, three forms of the disease are recognized. These are: (1) Pestis siderans, a rapidly fatal septicemic form; (2) pestis major, the usual form; and (3) pestis minor, a mild form characterized by glandular enlargements without pronounced constitutional disturbances. The last form is seen particularly in the beginning of an epidemic. A more useful classification, perhaps, is based upon the character of the pathological lesions: (1) a glandular type, (2) a pneumonic type, and (3) a septicemic type.

Symptoms.—*Glandular Type.*—The incubation period is usually from two to five days. Headache, pain in the back and limbs, vertigo with a staggering gait, languor, nausea, vomiting, and epistaxis are sometimes complained of during the last day or two of this period. A more or less distinct chill follows, with rapid rise of temperature, usually to 103° or 105° F. (39.5°—40.5° C.), sometimes even above 108° F. (42° C.). The pulse ranges from 120 to 150 and the breathing is quickened. As the disease progresses, the headache, nausea, and vomiting become more severe, extreme thirst develops, and the lymph-glands rapidly enlarge. The face is intensely flushed, the conjunctivæ are congested. The patient may fall into a stupor, but in some cases delirium develops. After three or four days the glandular swelling becomes extreme. By the involvement of a group of glands large buboes are formed, the apparent size being increased by the edema of the overlying tissues. These are found especially in the groin or along the femoral groove of one or both legs, less frequently in the axillæ or neck. The tonsils are

sometimes similarly involved. The primary bubo is usually larger and in a later stage of development than the secondary buboes. The skin over the affected glands becomes stretched and glossy. The swellings are generally extremely painful and exquisitely sensitive to touch. Suppuration frequently occurs and is considered a favorable change. When the cervical glands are involved, dyspnea and venous obstruction are produced. Carbuncles often form on the back of the neck. The frequent appearance of petechiæ over the body has given the name "black death" to the disease. Leucocytosis is usually present. The urine shows the usual febrile changes. The acute, febrile, period of the disease lasts from 3 or 4 to 10 days. Convalescence is generally rapid, unless it be retarded by the suppuration of the glands.

2. *Pneumonic Type.*—This begins with a chill, pain in the side, severe headache, high fever, and rapid breathing, with signs of pulmonary consolidation. Extreme dyspnea and cyanosis are often present. The sputum is bloody, not "rusty" as in lobar pneumonia. This form of the disease is nearly always fatal in from one to five days. Broncho-pneumonia sometimes occurs as a complication of the glandular type of the disease, causing it to resemble the pneumonic.

3. *Septicemic Type.*—Cases of a septicemic character (pestis siderans) are not infrequently encountered during an epidemic. The indications all point to a severe and rapidly fatal sepsis. They usually terminate within three days, often within a few hours, and before glandular enlargement has become recognizable. Even in these cases, however, there is marked sensitiveness over the regions of the lymph-glands, possibly over the entire body, and frequently there are hemorrhages into the skin and from the various mucous membranes.

Diagnosis.—The frequent occurrence of mild cases in the beginning of an epidemic often prevents the immediate recognition of the disease. Septic cases at this time are seldom recognized. In ordinary cases the mode of invasion, with the early tumefaction, pain, and tenderness of the lymph-glands, is distinctive after the prevalence of the disease has been recognized. Tubercular and venereal buboes, when accompanied by fever, may cause temporary uncertainty during an epidemic, but not otherwise. Other diseases are sometimes difficult of exclusion.

Typhus fever is accompanied by an eruption, usually petechial in character, but the glandular swellings or pneumonic symptoms are absent.

Malaria and *relapsing fever* are usually distinguished, if not by the absence of glandular involvement, by the recognition of the specific micro-organism of each in the blood. Cases are occasionally observed, however, in which one or the other of these affections has been coincident with the plague.

Prophylaxis.—This consists in the most rigid measures of sanitation in the infected districts, absolute quarantine, and a general cleaning up of adjacent territory. The extermination of rats is essential. The houses that have been occupied by the patients and all articles that have come into contact with them must be thoroughly disinfected or destroyed. No measure was ever more appropriate or more effective than the total destruction of the infected district by fire practiced by our Government at Honolulu a few years ago. For individual protection, Haffkine's serum may be used. It is a sterilized, attenuated bouil-

lon culture of the bacillus pestis. Although it does not always afford complete immunity, the disease has proved less virulent after its use.

Treatment.—The general treatment is wholly supportive and symptomatic. The strength should be supported by nourishing liquid food and brandy. The heart's action may be maintained by frequent full doses of strychnin, gr. 1-60 to 1-20 (0.001—0.003), and ammonium carbonate. Since suppuration is considered favorable, it may be encouraged by the application of hot poultices to the buboes. The Yersin-Roux serum, obtained from immunized horses, has been used with reported benefit in some cases.

CLIMATIC BUBO.

Under the names climatic bubo and malarial bubo, various authors have described a nonvenereal enlargement of the inguinal glands which attacks by preference young adult Europeans after a residence of three months or more in tropical countries. The disease occurs chiefly at the end of the rainy season and in persons who are suffering from fatigue or those run down and anemic. It is apparently independent of any relation to malaria. The only recognizable cause is the entrance of micrococci through slight wounds of the integument of the lower extremities or the bites of such insects as fleas and mosquitoes. The affection has followed the dhobi itch and other skin lesions. The blood-count shows anemia and leucocytosis. Fever is usually present, but it rarely exceeds 101° F. (38° C.). On account of the adenitis and fever the condition has been mistaken for the pestis minor, or mild form of bubonic plague. The treatment consists in the removal of the glands, which promptly arrests all symptoms.

MALTA FEVER.

MEDITERRANEAN FEVER, GIBRALTAR FEVER, NEAPOLITAN FEVER, UNDULANT FEVER.

The disease is endemic at Malta and occasionally spreads in epidemic form along the shore of the Mediterranean. It is also met with in the East and West Indies.

Definition.—An infectious disease caused by the micrococcus Melitensis of Bruce and characterized by a series of febrile attacks with profuse sweating and painful swelling of the joints.

Etiology.—The disease is not contagious. The infectious agent probably originates in small foci, often, apparently, in the rooms occupied by a previous patient, but the means of its transmission is not known. June, July, and August are the months of greatest prevalence at Malta. Epidemics sometimes occur. The most susceptible age is from 6 to 30 years. Infants and the aged generally escape. Immunity is thought to be conferred by one attack.

Symptoms.—The incubation varies from 6 to 17 days. The onset is often much like that of typhoid fever, with anorexia, thirst, pain in the head, back, and extremities, and a gradual rise of temperature. The tongue becomes coated and the pharynx congested; the epigastrium is

tender. Constipation is the rule. Delirium sometimes occurs at night. The fever may reach 103° or 104° F. (39.5°—40° C.) and is usually of a remittent type, occasionally distinctly intermittent. The characteristic curve is a gradual ascent for a week or ten days, followed by a decline of about the same duration. A profuse sweat occurs toward morning, when the temperature is low. During the fever the joints swell in rapid succession, and become painful, as in acute rheumatism. After from one to three weeks, the symptoms subside for three or four days. A relapse then occurs; the former symptoms return, often with increased severity. Another interval occurs after three or four weeks, and thus the disease progresses, sometimes for several months. In mild cases, recovery may follow the first relapse, but another relapse may occur after several months. A malignant form of the disease is recognized which is usually fatal in about 10 days.

Complications and Sequelæ.—The chief of these are pneumonia, neuralgia, and anemia. Orchitis sometimes occurs without other infection.

Diagnosis.—*Typhoid Fever.*—During the first rise of temperature, the differentiation may be difficult. The absence of roseola after the eighth day and failure of the Widal test are important. A similar agglutination reaction may be obtained with a culture of the micrococcus Melitensis and the serum of the patient.

Malaria may be distinguished by the latter test and by the finding of the plasmodium in the blood.

Prognosis.—The mortality is about 2 per cent. Death is usually the result of sudden hyperpyrexia, exhaustion, or complications.

Treatment.—No specific treatment has been discovered. The indications are in all respects the same as those of typhoid fever.

BERIBERI.

KAKKI, ENDEMIC NEURITIS.

This disease prevails endemically in many isolated regions of the tropics, especially in China, Japan, the Philippine Islands, Hawaii, South America, and the West Indies. Cases are occasionally carried by ship to the United States. In 1895 a disease believed to be beriberi broke out among the inmates of the State insane asylums of Alabama and Arkansas. It has appeared also among the fishermen of Newfoundland and Cape Cod.

Definition.—An acute or chronic disease of tropical and subtropical countries characterized by multiple neuritis with motor and sensory disturbances, edema, and visceral lesions of greater or less severity.

Etiology.—Two theories are maintained in regard to the character of the disease: First, that it is an infection due to an unrecognized micro-organism; second, that it is due to a toxemia from food.

1. The principal argument in favor of the theory of infection is that a micrococcus has been found which, by inoculation, produces peripheral neuritis. Ogata, however, attributes it to a bacillus. The disease occurs at a definite season and attacks young, robust individuals. It is a place disease, clinging to houses and more particularly to isolated localities, as does malaria. There is some evidence that it is contagious.

2. The theory of food toxemia is held especially in Japan and Java,

where the disease is attributed to the excessive consumption of white (hulled) rice. It is said to have been repeatedly checked by the adoption of European food. Visitors to Japan do not become affected so long as they do not adopt the rice diet.

The fermentation of rice is regarded by several writers as the more direct cause. Capt. E. R. Rost, I.M.S., asserts that in Rangoon, where the disease is epidemic, it is caused chiefly by drinking rice-water liquor made by the Chinese from damaged rice. The disease is not seen in children there, seldom in women, and it is not infectious or contagious.

Males from 16 to 25 are most frequently attacked, but it may affect either sex at any age. Hot, moist atmosphere and overcrowding favor its development.

Morbid Anatomy.—Peripheral neuritis is the essential lesion. The vagus and phrenic are sometimes involved. Hypertrophy of the right ventricle, with degeneration of the myocardium, is usually present. The skeletal muscles may be also degenerated.

Symptoms.—The incubation probably lasts a month. The initial symptoms are generally catarrhal. These are followed by pain and weakness in the legs, and paresthesia and edema gradually invading the entire body. The muscles become soft and sensitive. The heart's action is weak and irregular; palpitation may be felt and dyspnea is produced. The urine is scant, but not albuminous. Recovery may occur after a few weeks or after several months. Relapses often occur at the same season for many successive years. Three forms of the disease are described:

1. *Dry, Atrophic, or Paretic Form.*—This is characterized by a painful atrophy, with more or less complete paralysis of the muscles of the arms and legs, sometimes involving also those of the face. The tendon reflexes are abolished. Edema is not usually present.

2. *Wet, or Dropsical Form.*—Edema is the most marked feature. The subcutaneous tissue and serous cavities of the entire body are often invaded. The degenerative changes in the muscles may not be prominent. Cardiac weakness and dyspnea are seldom absent.

3. *Acute, Cardiac or Pernicious Form.*—Cardiac weakness is the predominant symptom. Death from heart-failure may take place within the first few days, before the development of other symptoms. When the vagus is involved, the larynx may be paralyzed, and vomiting is a prominent symptom. When the phrenic nerve is affected, death may result through paralysis of the diaphragm.

Diagnosis.—This is seldom difficult in tropical regions. The ordinary form of peripheral neuritis does not involve the vasomotor and visceral nerves, and it is not attended with so great dyspnea or edema.

Prognosis.—The mortality ranges from 2 to 50 per cent. Much depends upon the character of the epidemic, the strength of the patient, and the hygienic conditions. The greatest mortality has been among coolies. Vomiting is regarded by the Japanese as of fatal import.

Prophylaxis.—This consists in proper diet and hygienic measures, particularly the prevention of overcrowding. Visitors to localities where the disease prevails should not adopt the exclusively farinaceous diet.

Treatment.—The patient should be immediately removed to a high

and dry locality, when his condition will permit. This is regarded by Manson as important in order to avoid reinfection. The treatment should be begun with free purgation. Following this the salicylates should be given in 20-grain (1.30) doses four or five times a day. Stimulation must be practiced when the heart becomes weak. Strychnin should be given in doses of gr. 1-40 (0.0016). If the arterial tension is high, glonoin, gr. 1-100 (0.0006) every half-hour, is indicated until the heart's action becomes normal. Blood-letting has been practiced with benefit in some cases, and inhalations of amyl nitrite have been recommended when there is danger of cardiac failure. Nitrogenous food should constitute the principal part of the diet; and if rice is eaten, it should be the unhulled or red variety.

SCARLET FEVER.

SCARLATINA, SCARLET RASH.

Definition.—An acute infectious disease manifested by severe angina and an erythematous exanthem, with constitutional symptoms of variable severity.

Etiology.—The disease may be either sporadic or epidemic. It is extremely contagious and frequently spreads with great rapidity among the inmates of schools and asylums. Forchheimer has shown that it is probably not capable of transmission to any great distance through the atmosphere. Mediate contagion is the rule. No specific organism has been discovered. Streptococci and other germs have been found in the blood, urine, skin, and in various organs in fatal cases, but their relation to the disease has not been proved. Inoculation has been successfully performed with the blood, serum, nasal and pharyngeal secretions. Contagion is generally believed to take place directly from the patient, through the exhalations, but by far the most dangerous source of infection is found in the desquamated epithelium. Clothing, books, toys, anything that has been handled by the patient, furniture, carpets, even the dust from the sick-chamber, retains the poison and may convey it to others. The contagium has been retained for several years in articles of clothing protected from the air, longer than is known to be possible in any other disease. Physicians and nurses have carried the infection, and it is not infrequent for those coming into contact with the patient to contract a severe angina, although they may have had the disease in childhood. Pets, birds, cats, and dogs, probably become carriers of the infection in some instances. Food, especially milk, is readily contaminated. Defective house-drainage has been held responsible for the disease in some instances. The mucous membrane of the throat is probably the usual avenue of infection.

Age.—Scarlatina is typically a disease of infancy and early childhood. Half the cases appear before the fifth year, and 90 per cent before the tenth. Nurslings are seldom attacked. Infants, although born during the illness of the mother, may escape, but sometimes they are born with the disease. Adults are occasionally affected. Sex does not modify the susceptibility to it.

Season.—Sporadic cases are seen at all seasons; epidemics generally prevail during the autumn and winter.

The immunity conferred by one attack is generally permanent, but second and third attacks are not extremely rare. So far as known, the Japanese alone possess natural immunity. The susceptibility of all individuals is not equal, for it is not uncommon to see different degrees of severity manifested by the disease among the children of the same family, or for one or two members of a family to escape. Some families are much more susceptible than others. The virulence of the disease is much greater in some epidemics than in others.

Morbid Anatomy.—The cutaneous and pharyngeal lesions are alike hyperemic in character and promptly disappear after death, except in the hemorrhagic form of the disease. A section of the skin shows only capillary dilatation, without the changes of inflammation. There are no characteristic lesions; those found in the organs after death are the result of high temperature or of pyogenic infection. The serous membranes are more generally involved than the mucous. The most important complication is on the part of the kidneys, an acute nephritis being found in a large proportion of fatal cases. As a result of the throat lesions, sometimes pseudomembranous in character, the cervical lymph-glands are often greatly enlarged and in a state of suppuration. Gangrenous sloughs are sometimes found. Lobular pneumonia may be the immediate cause of death in such cases. The lesions of endocarditis, pericarditis, pleurisy, and peritonitis are sometimes found. Gastro-intestinal congestion may be present. The spleen shows the usual febrile enlargement, and interstitial changes have been seen in the liver.

Symptoms.—*The incubation* is from one to ten days. There are generally no prodromes, but slight indisposition may be noticed during the last day or two.

The invasion is usually sudden and may be severe. Vomiting, sore throat, and chilliness, rarely a distinct chill, are commonly present. One or more convulsions may announce the onset in young children. The patient becomes restless and delirium may develop within the first 24 hours. Thirst and dryness of the throat are complained of. The tongue is at first white with red edges, and the papillæ often protrude through the coating, producing the characteristic "strawberry tongue." A few days later, the fur is cast off, and with it the surface epithelium, leaving the tongue intensely red and the denuded papillæ prominent, an appearance which has been called the "raspberry tongue." Leucocytosis generally develops early in the disease and may be extreme in severe cases.

The Eruption.—A scarlet erythema invades the skin, generally on the second day, sometimes within the first 24 hours. It is seen first on the sides of the neck, upper part of the chest and back, in the form of minute pale red papules, which rapidly coalesce to form an intensely scarlet flush, that spreads within a few hours to the surface of the entire body. A punctate eruption sometimes appears first in the mucous membrane of the mouth and throat. Petechiæ are rarely seen, except in hemorrhagic cases. Papillary elevations are occasionally noticeable, and minute yellowish vesicles, probably sudamina, sometimes appear (scarlatina miliaris). The face is livid, except around the mouth, where the skin remains normal, but appears excessively white by contrast. The eyelids become edematous in severe cases, particularly when nephritis develops.

The eruption is occasionally limited to regions or appears in isolated patches. Burning and itching are often complained of, and the skin may be hyperesthetic. In malignant cases extreme cellulitis of the neck is frequently encountered, and a false membrane may develop on the tonsils and spread rapidly to adjacent surfaces. True Klebs-Loeffler diphtheria is sometimes present as a complication. The eruption begins to subside, as a rule, by the third day. *The temperature* remains high, often 104° or 105° F. (40°—40.5° C.), with slight morning remissions, until the fading of the eruption. Delirium not infrequently persists throughout the febrile stage, particularly at night. In mild cases the temperature may not reach 103° F. (39.5° C.), but in the malignant type it often exceeds 108° F. (42.2° C.) shortly before death. *The urine* shows the ordinary febrile changes, diminution of quantity with increase of solids, particularly the urates. The frequency of renal complications renders daily examination of the urine imperative.

Desquamation usually begins within two or three days after the subsidence of the eruption, but may be delayed for nearly a week. It never fails to occur. It follows the same course of progression as the eruption, beginning on the neck and chest. It may be furfuraceous or membranous in character, the epidermis separating in scales or in sheets. More or less complete molds are sometimes obtained from the hands and feet. The denuded skin is for a time red and tender. Desquamation usually lasts from three to five weeks. Several coats are sometimes shed, and the progress may be protracted to 7 or 8 weeks. The danger of communicating the infection does not end until the skin has become quite normal. In rare cases the hair and nails are also cast off. The itching accompanying desquamation is often intense. The character of the desquamation does not always conform to the severity of the disease, but, as a rule, it is less extensive in the milder cases.

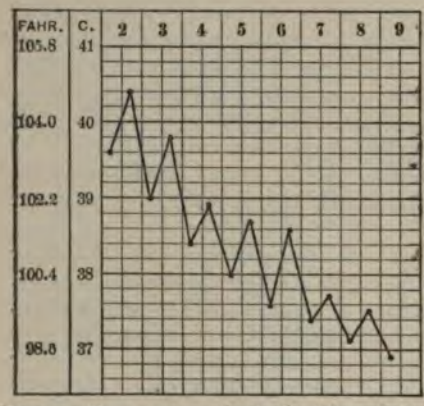

FIG. 9.—Temperature chart of mild scarlatina.

Forms of Scarlatina.—Great difference is manifested in the severity of symptoms. The disease may be so mild as to readily escape notice or to render the diagnosis difficult. The fever may not exceed 100° F. (38° C.), and may last but a few hours. The throat symptoms are mild and only a trace of albumin may appear in the urine. Desquamation may, however, be abundant. Cases have been reported in which the eruption was absent (scarlatina sine eruptione). Occasionally the rash does not appear until the fourth or fifth day.

Malignant Scarlatina.—1. Cases occur in which the symptoms of toxemia predominate. The temperature reaches 106° F. (41° C.) or higher on the first evening, and profound prostration, delirium, and gastrointestinal disturbances are predominant. In some cases the fever continues for 10 to 14 days and subsides by a slow lysis.

2. *Foudroyant cases* are encountered. The invasion is extremely severe, with repeated convulsions, immediate rise of temperature to 107° or 108° F. (41.5°—42.0° C.), intense delirium, profound stupor or coma, and projectile vomiting. The pulse is feeble and dyspnea urgent. Death may occur within the first 24 hours, before the appearance of the eruption.

3. *A hemorrhagic type* is rarely met with. It usually terminates fatally in the first few days. Blood is extravasated into the skin and mucous membranes, and there is bleeding from the nose, stomach, and bowels. The temperature may be moderate.

4. *Anginose Type.*—In this form the throat symptoms predominate. A false membrane usually develops upon the intensely swollen tonsils and pharynx and quickly spreads into the nose and larynx, often through the trachea into the bronchi. Gangrenous sloughs form in the throat, and suppurative otitis media results from extension of the inflammation along the Eustachian tube. A suppurative cellulitis of the neck follows the adenitis, and a general septic infection usually leads to a fatal issue, if, as is generally the rule, death has not occurred earlier in the disease.

Puerperal and Surgical Scarlatina.—It is now generally believed that most of the cases which were formerly regarded as of this character are in reality cases of septicemia. The view is well supported by the remarkable decrease in the number of these cases since the adoption of methods for the prevention of sepsis. Scarlet fever may, however, attack the surgical patient or puerperal woman.

Complications and Sequelæ.—(1) *Nephritis* is the most serious of the complications. Three forms occur. They probably result from toxemia, although micro-organisms have been repeatedly found in the kidneys. They usually develop during desquamation, in the second or third week of the disease, and may occur in either mild or severe cases:

(*a*) *Acute Degenerative Nephritis.*—This is a mild form in which the lesions are not inflammatory in character and are, in most cases, confined to the parenchyma of the tubules. It is indicated by a reduction in the quantity of urine, a moderate quantity of albumin, a few hyalin, epithelial, or granular casts. The constitutional disturbances are slight, the edema moderate. Recovery is the rule.

(*b*) *Glomerulonephritis, or Exudative Nephritis.*—In this form the glomerulus, to which the involvement is chiefly limited, is compressed by an abundant exudation of serum, red and white blood-cells, and epithelium within the capsule. The condition is announced by an almost complete suppression of urine. That voided contains blood, a large quantity of albumin, and increased urates. The microscope reveals different kinds of casts, blood-cells, and pigment. The constitutional symptoms are severe and may appear early. They are: edema of the face, hands, and feet, headache, nausea, vomiting, dyspnea, muscular twitchings, and sometimes delirium. The fever and rapid pulse continue with high arterial tension and irregular action of the heart. The dropsy may become extreme and it may involve the lungs. Under careful management, recovery generally occurs in from four to six weeks. The condition may become chronic, however, or a fatal uremia may supervene.

(*c*) *Acute Diffuse Nephritis.*—This is the most severe form of the

disease, affecting both parenchyma and interstitial substance of the glomeruli and tubules. It usually arises in the third week, either suddenly or gradually. Vomiting, marked anemia, and more or less complete suppression of the urine are the constant symptoms. Convulsions often occur. Blood and albumin are abundant in the urine. Death usually occurs early from uremia.

(2) *The Heart.*—Acute endocarditis is not uncommon, and frequently leaves permanent lesions of the valves, often to be recognized in after-life. The malignant form of endocarditis is rare. Pericarditis with serofibrinous or purulent exudation may occur, and myocarditis sometimes develops.

(3) *Serous Membranes.*—Pleurisy is frequent and may lead to empyema. Peritonitis may also be encountered.

(4) *Nervous System.*—Chorea and hemiplegia develop, especially in cases complicated with arthritis and endocarditis, and are probably a result of embolism. Mania sometimes occurs. Progressive paralyses have been noted. Thrombosis may affect the lateral sinus or the cerebral veins. Meningitis and abscess of the brain have been observed.

(5) *The Ear.*—Suppurative otitis media, due to extension of the throat inflammation, is so frequent and so severe as to render scarlatina one of the most common causes of deafness. The suppuration generally extends to the labyrinth and may involve the mastoid cells.

(6) *Suppurative cellulitis* of the neck, with gangrenous sloughing, is an occasional result of the throat inflammation.

(7) *The Glands.*—The adenitis, although extreme, usually subsides in a few weeks, but in some cases it persists indefinitely. Suppuration may develop and it may extensively involve the surrounding tissues.

(8) *The Joints.*—Painful swelling of the joints sometimes occurs during the height of the fever, but more frequently during its decline. It is regarded by some writers as a form of rheumatism, by others as a septic infection analogous to gonorrheal rheumatism. Suppuration sometimes develops in the affected joints.

(9) *More Complications.*—Among these may be mentioned blindness from iritis or neuroretinitis, symmetrical grangrene, noma, furunculosis, and purpura hemorrhagica. The association of scarlatina with other diseases, notably measles, variola, varicella, and pertussis, is occasionally observed.

Diagnosis.—The sudden onset with vomiting, rapid rise of temperature, the angina with enlargement of the cervical glands, the early appearance of the eruption, and the strawberry tongue seldom leave the diagnosis long in doubt. Cases arise, however, in which much difficulty is experienced.

1. *Acute Exfoliative Dermatitis.*—This affection closely simulates scarlatina in its sudden febrile onset and uniform red rash. The throat symptoms are usually absent, the tongue is not typical, the eruption appears first on the trunk and has not faded away until desquamation sets in again. The hair and nails are usually involved in the exfoliation. Repeated attacks are common, even within short intervals of time, a fact which doubtless explains many instances of supposed recurrent attacks of scarlatina.

2. *Measles.*—In this disease we have prodromal catarrh, a less violent

invasion, subsidence of temperature before the appearance of the eruption on the third or fourth day. The eruption is papular, more abundant on the face, and often shows crescentic arrangement. The throat symptoms are mild or absent and the leucocytes are not increased.

3. *Rotheln.*—This disease is usually excluded by the mild invasion, slight febrile disturbance, and the paleness and mottled character of the rash, which appears first on the face.

4. *Diphtheria.*—In most cases the absence of the Klebs-Löffler bacillus is sufficient to distinguish scalatina with membranous throat formation from true diphtheria. When, however, this bacillus is present, it is often difficult to decide whether the case is one of diphtheria with erythematous eruption or a double infection. The diphtheria rash is usually dark red and confined to the trunk.

5. *Septicemia.*—The more uniform and prolonged febrile course of this condition may suffice for differentiation. It is not always possible to distinguish the two affections in the puerperal period.

6. *Drug Rashes.*—Belladonna and quinin, less frequently potassium bromid and iodid, chloral, acetanilid, and other drugs produce rashes resembling that of scarlet fever. The other symptoms are lacking.

Prognosis.—The disease is most fatal in young children and among the poor. Some epidemics are much more fatal than others. High fever, delirium, membranous angina, and hemorrhages are exceedingly unfavorable symptoms. Even in the mildest cases a serious nephritis may develop at a time when recovery seems certain. Nephritis is not necessarily a fatal complication; most cases recover. The total mortality of the disease ranges from 5 to 10 per cent in the milder epidemics and from 20 to 30 in the more severe.

Prophylaxis.—The patient should be isolated and the house quarantined. The other children of the family should be kept from school and prevented from associating with their playmates long enough to determine that they have not also contracted the disease. The same precautions should be taken with reference to the apartments and the conduct of the physician and nurse as are recommended under the prophylaxis of smallpox. The patient need not be confined to bed longer than a week or ten days after the fever has subsided, in the absence of other contraindications; but care should be exercised to avoid exposure to cold for three or four weeks longer. The quarantine should last six, or, better, eight weeks, or in any case until the last indication of desquamation has disappeared.

Treatment.—The room should be well ventilated, and a uniform temperature of 68° F. (20° C.) should be maintained. The patient should wear a flannel gown, but the bedclothing should be light. The diet should be liquid during the febrile stage, preferably milk, in addition to which gruels, broths, and egg albumen may be allowed. Ice cream is nourishing and soothing to the throat. An abundance of water should be given. Solid food may be allowed after the fever has subsided in a mild case, but a continuance of the milk diet reduces the liability to nephritis. Medication is unnecessary in mild cases. An antiseptic, as sodium sulphocarbolate or salicylate, should be given with a view to reducing the liability to complications. For high temperature, restlessness, or delirium the bath of 90° F. (32° C.), gradually reduced, cool

sponging, or the wet pack should be employed, and the bromids may be administered. The ice-cap is often useful. The bowels should be regulated with magnesium citrate, compound licorice powder, or other aperient. The action of the kidneys must be favored by a plentiful supply of pure cold water, lemonade, or other drink. Irrigation of the bowel every six or eight hours with a pint or more of water at 110° F. (43° C.) is recommended for the restoration of the renal secretion and for the relief of convulsions.

The throat, nose, ears, heart, and urine should be examined daily, and the examinations of the urine should be continued periodically during convalescence. For weak heart, stimulants should be given. Pericarditis requires special treatment described under that disease. For the throat and nose, a spray of 5 per cent menthol and camphor in liquid albolin or 10 per cent hydrozon is beneficial. Otitis requires puncture of the drum membrane as soon as tension becomes prominent. Renal complications are to be treated according to the methods given under Nephritis.

As soon as desquamation has commenced, the patient should be given a warm bath morning and evening, followed by thorough inunction of the entire body with sweet oil or carbolated vaselin in order to limit the dissemination of the scales.

MEASLES.

RUBEOLA, MORBILLI.

Definition.—An acute infectious disease running a febrile course and exhibiting a papulomacular exanthem.

Etiology.—The bacterial cause of the disease is unknown. Measles is the most infectious of the exanthemata, and immunity is exceedingly rare. It is highly contagious during its entire course, including the last days of the incubation. It occurs endemically in cities at all seasons, and epidemically about every second winter. Age has probably little influence on susceptibility, but few persons escape the disease during their childhood. Infants under six months are seldom attacked. The disease is by no means infrequent in adults. Second, third, and even fourth attacks have occasionally been reported. The contagium is communicated by the breath and secretions of the patient, particularly by the nasal mucus, saliva, and tears. It may be carried by a third person, by fomites, or by the air, but the poison is not so virulent or so retentive of life as that of scarlatina. Inoculation has been performed.

Morbid Anatomy.—The lesions of the mucous membranes are the same as those of other catarrhal conditions. The mortality is due chiefly to complications, especially to bronchopneumonia. The bronchial glands are always enlarged. The gastrointestinal mucous membrane is frequently hyperemic and the solitary and agminated follicles are often greatly enlarged. Leucocytosis is absent. Demme found during the height of the fever a diminution of the red blood-corpuscles, numerous microcytes and free nuclei, and a diminution in the quantity of fibrin in the blood.

Symptoms.—The period of incubation generally lasts from 7 to 14 days. Fretfulness and slight fever may be noticed during this time. The invasion is often announced by chilly sensations, occasionally by vomiting; rigors and convulsions are rare. The first symptoms are usually those of coryza, generally accompanied by hyperemia of the pharynx and larynx and conjunctival congestion, with lachrymation and photophobia. Sneezing, cough, and hoarseness develop and the child becomes fretful and cross. The tongue is furred; the edges may remain red and the papillæ prominent.

The temperature may rise abruptly on the first day to 103° or 104° F. (39.5—40.0° C.), but it sometimes pursues a more gradual elevation until the appearance of the eruption. Nausea and vomiting are occasionally persistent. In mild cases the symptoms of invasion may be so trifling as to escape observation.

Koplik's Spots.—Peculiar spots, first described by Koplik, can be seen on the mucous membrane of the lips and cheeks with the aid of strong daylight, in most cases 24 to 48 hours, sometimes four or five days, before the appearance of the eruption. They are small, bright red spots, each of which shows in its center a minute, bluish white speck. The speck can be picked off with a forceps or removed by rubbing. The spots coalesce, and, when fully developed, the labial and buccal mucous membranes appear uniformly rose-red, with a studding of myriads of bluish white specks.

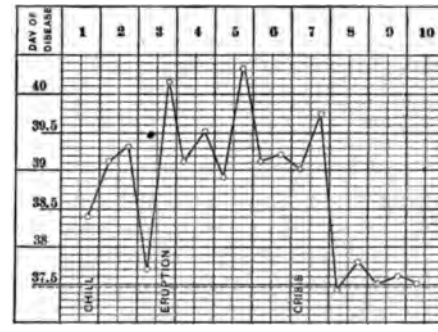

FIG. 10.—Temperature chart of measles.

Stage of Eruption.—On the evening of the third day the child generally appears more fretful and its sleep is more restless. The morning of the fourth day, the eruption can generally be detected upon the face, and it becomes distinctly visible during the day. It appears first on the forehead, chin, and sides of the neck, in the form of slightly elevated, round or lenticular papules of variable size. The papules enlarge and are often so numerous on the face as to cover its entire surface with dark red blotches which can often be distinctly felt with the finger. On the chest, arms, and back they usually coalesce into crescentic figures. Petechiæ are sometimes seen, especially in the more malignant cases, and miliary vesicles are sometimes observed. From the face and neck the eruption gradually invades the entire surface of the body, reaching the lower extremities by the evening of the fifth or sixth day of the disease. It remains two or three days in each locality, then rapidly fades. A fine, bran-like, furfuraceous desquamation, often scarcely noticeable, follows its disappearance. The mucous membranes of the mouth, throat, and larynx are often invaded by the eruption.

With the appearance of the exanthem, the catarrhal symptoms become aggravated and the bronchial mucous membrane becomes involved. The temperature often reaches 105° or 106° F. (40.5°—41.0° C.) on the

evening of the fourth day; the pulse is rapid, 120 to 150, and bounding. Epistaxis sometimes occurs. The cough is often distressing. The restlessness is greatly added to in some cases by persistent vomiting, thirst, irritability of the bladder, diarrhea, intense burning of the skin, and insomnia. Delirium develops in severe cases.

There is more or less general, though moderate, enlargement of the lymphatic glands. The urine frequently contains albumin, pepton, and aceton and gives the diazo reaction. All the symptoms promptly subside as the eruption fades, with the exception of those due to the bronchial catarrh, which frequently persists.

Atypical Cases.—(1) In epidemics, it is not unusual to meet with cases in which the eruption appears on the second or third day, and others in which it is delayed as long as the sixth day or later.

(2) Cases occur in which the catarrhal symptoms are prominent, but the eruption absent, and others in which the eruption appears without the usual catarrh.

(3) **Hemorrhagic or Black Measles.**—This form of the disease is characterized by hemorrhages into the skin and mucous membranes, great prostration, hyperpyrexia, and violent delirium or profound stupor. It is encountered especially in prisons and asylums or among the aborigines of a country in which the disease has not previously prevailed. The mortality may exceed 25 per cent.

(4) Malignant cases occur with the same symptoms of profound intoxication as in black measles, but without the hemorrhages. Death may occur before the appearance of the eruption.

Complications and Sequelæ.—Bronchopneumonia is a frequent and often fatal complication, particularly in debilitated children and amid bad hygienic surroundings. The enlarged bronchial glands often become tubercular and may thus become the nidus for the development of acute miliary tuberculosis. Although the laryngeal catarrh is often severe, edema seldom develops. A fatal pseudomembranous growth sometimes spreads over the pharynx and larynx. In some cases it is due to diphtheritic infection. Corneal ulcers and blepharitis not infrequently occur and optic neuritis may develop. Otitis is sometimes a sequel. Noma of the cheek or vulva often owes its origin to this disease. Enterocolitis, with profuse diarrhea, is an occasional complication. Diphtheria, whooping cough, and other affections are occasionally associated with the disease. Tuberculosis is the most serious of the possible sequelæ. Such affections as paralysis, generally due to neuritis or myelitis, pleurisy, pericarditis, nephritis, and arthritis are seldom seen.

Diagnosis.—Measles is generally differentiated by the character of the prodromal symptoms, particularly the cough, sneezing, congestion and suffusion of the eyes, and the presence of Koplik's spots. A febrile period of four days associated with these symptoms and followed by a papular eruption on the face serves to distinguish it from other affections.

Scarlatina is much more acute in its onset; the diffuse eruption appears on the second day, and the strawberry tongue is characteristic.

Rotheln is distinguished by the mildness of the invasion, brighter color of the efflorescence, the absence of crescentic figures, and the greater enlargement of the cervical lymph glands. Copaiba, quinin, and anti-

pyrin occasionally produce rashes resembling measles, but the fever and catarrhal symptoms are absent.

Treatment.—As prophylactic measures the patient should be isolated for two weeks from the onset. After recovery, he should receive an antiseptic bath; the clothing and bed-linen should be thoroughly disinfected by boiling, and the room with formaldehyd vapor.

In a mild case confinement to bed and daily sponging are often all that are necessary. The room should be well ventilated, moderately dark, and of uniform temperature. The diet should be liquid during the height of the fever. Medication is often necessary. The restlessness, insomnia, and delirium, when present, call for the administration of the bromids and cool baths. The cough should be kept under control by a mixture of camphorated tincture of opium and ipecacuanha, squill, or ammonium chlorid. The danger of the tubercular infection requires that the treatment be continued until recovery is complete and the cough has entirely ceased. The eyes should be cleansed several times a day with a 2 per cent boric acid solution, and a little pure vaselin should be applied to the edges of the lids. The throat and ear should be occasionally examined and antiseptic sprays used when indicated. If otitis develops the membrane must be promptly incised. During desquamation the skin should be anointed once a day after bathing, with oil or vaselin.

GERMAN MEASLES.

RUBELLA, ROTHELN, RUBEOLA NOTHA.

Definition.—An acute infectious disease of mild type, characterized by a macular cutaneous eruption and enlargement of the cervical lymph-glands.

Etiology.—Rötheln is a highly contagious disease, prevailing mostly in the winter and spring months, among children, and often assuming epidemic proportions. Old age is not exempt, and congenital cases have been observed. It is entirely distinct from measles and scarlet fever. One attack usually confers immunity. The specific cause is unknown.

Symptoms.—Incubation lasts from 10 to 20 days. The invasion is like that of a very mild case of measles, with slight headache, coryza, sore throat, pain in the back and extremities, chilliness in some cases, and an elevation of temperature seldom reaching 102° F. (39.0° C.). The cervical lymph-glands are distinctly enlarged, sometimes for several days before the development of other symptoms. The eruption appears on the first or second day, sometimes, it is stated, as late as the third. It comes out first on the face and palate, then on the chest, and extends within 24 hours to the entire surface of the body, including the palms and soles. Cases have been reported in which the rash was confined to limited areas. In character it may be macular or papular, slightly elevated, round, rose-colored spots, distinct except on the buttocks and inner sides of the thighs, where the maculæ frequently coalesce. The spots vary in size as do those of measles, but are often larger. The efflorescence is brighter than that of measles and does not usually

form crescents. The intervening skin is often hyperemic, without the punctate appearance of scarlatina. A few small vesicles or pustules have been observed in connection with the rash. Itching is sometimes present. The eruption begins to fade in two or three days. The disease runs its course in about five or six days. It is followed by a fine, flaky desquamation. Slightly pigmented spots are frequently left. Sometimes it is more severe, resembling measles except in the character of the eruption. Albuminuria has been noted in some instances.

Diagnosis.—The disease is to be differentiated chiefly from measles and scarlet fever; erythema and urticaria may enter into the consideration.

Measles.—The eruption is paler, less elevated, and appears earlier than that of measles and does not form crescentic figures. The symptoms are in every respect milder.

Scarlatina.—The spotty character of the eruption, appearing first on the face, the less intensity of the throat symptoms, the slower, milder invasion without vomiting, and the absence of the strawberry tongue generally distinguish it from scarlatina. In the absence of an epidemic it may be extremely difficult to distinguish a severe case from a mild attack of either measles or scarlatina.

Erythema appears for the most part on the hands and feet, is generally accompanied by burning pain, and is not attended with coryza.

Urticaria is characterized by the appearance of "wheels," with intense itching, chiefly on the extremities. There is no coryza.

Treatment.—The treatment is that of a mild case of measles. In most cases medication is superfluous; it is generally difficult to confine the patient to the house.

RUBELLA SCARLATINOSA.

The provisional name of "Fourth Disease" was given by Dukes, of England, in 1900, to a train of symptoms which he regards as belonging to a distinct infection not heretofore differentiated from scarlatina and rubella. He studied it in 19 cases in the school at Rugby. A more extensive study is that of Curtis and Shaw, of Albany, in 1902, comprising 147 cases, of whom 81 were adults.

Symptoms.—Incubation lasts about 19 days. During this stage the malaise is so slight as to readily escape observation. Vomiting does not occur. The eruption is generally the first symptom to attract attention. It envelops the entire body in a diffuse erythema without the punctate features of scarlatina. Pressure causes only the most transient blanching. The throat is red and swollen, and an exudate sometimes forms on the tonsils, but in many cases it occasions little discomfort. The Klebs-Löffler bacillus is not found. The tongue is furred throughout, but "cleans as all furred tongues do." Desquamation sometimes lasts six or seven weeks. In some cases the skin becomes merely rough, while in others the epidermis comes off in strips or lamellæ as extensive as any seen in scarlet fever. It bears no relation to the intensity of the eruption. The lymph-glands are uniformly enlarged, hard, and tender, but less so than in rötheln. The temperature is moderate, averaging 101° F. (38.3° C.), and usually subsides on the third or fourth day.

The pulse ranges from 100 to 120. Albuminuria is absent. Treatment is not usually required. There is much doubt as to the propriety of admitting the disease as an entity and not merely as a form of rubella.

CEREBROSPINAL MENINGITIS.

CEREBROSPINAL FEVER, SPOTTED FEVER, EPIDEMIC LEPTOMENINGITIS.

Definition.—A severe infectious fever caused by the diplococcus intracellularis meningitidis, occurring epidemically or sporadically and characterized by an inflammation of the cerebrospinal meninges and a great diversity of clinical manifestations.

Etiology.—The diplococcus intracellularis meningitidis is recognized as the specific cause of the disease. It resembles the gonococcus in form, but not in its behavior on culture media. It is found chiefly in the polynuclear leucocytes, both in the tissues and in the cerebro-spinal fluid, sometimes in the fluids of the joints. From the fact that it is found in the secretions of the nose, Strümpell and Weigert believe that infection takes place through this channel, and it has been suggested that the meninges are reached by way of the Eustachian tube and ear. Weichselbaum calls attention to the possibility of its occurring through the auditory canal. Its transmission from place to place is not understood. The frequent occurrence of the disease among soldiers in crowded barracks and among prisoners suggests contagion, but is probably due rather to unhygienic surroundings, for it often occurs sporadically in populous tenements, and epidemics have been more frequent in rural districts than in the cities. It occurs most frequently in children and young adults, but no age is exempt. Overexertion, lack of ventilation, uncleanliness, and crowding are important predisposing influences. A second attack has been reported in five instances (Councilman).

Morbid Anatomy.—In the early fatal cases death is probably due to the intense action of the toxin. No lesions are usually found beyond intense hyperemia of the meninges. In less rapidly fatal cases, a fibrinoplastic exudation is found, especially at the base of the brain and along the fissures and sulci of the cortex; the pia is opaque. The membranes at the base may be much thickened. In more protracted cases there is still more marked thickening of the meninges, the ventricles are distended with a fibrinopurulent fluid, a fluid consisting of serum, fibrin, pus-cells, and diplococci. The posterior cornua often contain pure pus. The brain substance is softened and has a pinkish tinge; areas of encephalitis and hemorrhagic foci are frequently found. The cranial nerves, especially the second, fifth, seventh, and eighth, are frequently involved, and the spinal nerve roots are embedded in the exudate and their axis cylinders are swollen. A chronic hydrocephalus is sometimes developed, particularly in children. Congestion, with granular and fatty degeneration of the heart, liver, or kidneys and other organs, is often found. The spleen is enlarged and soft. Hemorrhages may be found in the skin, serous membranes, particularly the pleura and pericardium, or in the viscera. Congestion or edema of the lungs and bronchopneumonia occur, and a lobar pneumonia, due either to the pneumococcus or the diplococcus

intracellularis, is by no means infrequent. The larger joints often become distended with a seropurulent exudate, and the muscles show granular or fatty degeneration.

Symptoms.—The incubation period, probably lasting a week or ten days, is not usually accompanied by prodromal symptoms. Headache, pain in the back, loss of appetite, and slight nasal catarrh are sometimes observed. The onset is generally abrupt, with intense headache, a chill or convulsions, rise of temperature to 102° or 103° F. (39.0°—39.5° C.), and projectile vomiting. The muscles of the neck and spine soon become sensitive, painful, and rigid, the pain and rigidity often extending also to the muscles of the extremities. In extreme cases the head is drawn far back and in some cases the entire spine is bowed (opisthotonos). Motion of the head is painful or may be impossible, on account of the rigidity. Unconsciousness or delirium early supervenes. Photophobia, sluggish reaction or unequal dilatation of the pupils, are commonly observed, and strabismus, nystagmus, or ptosis, with conjunctivitis, is not infrequently present. Hypersensitiveness to sound is observed in almost all cases, and swallowing is often painful. The face has a drawn appearance expressive of pain. The temperature range is exceedingly variable, sometimes rising suddenly to 104° or 105° F. (40° —40.5° C.), and then declining nearly or quite to the normal, only to rise again, without apparent cause for the fluctuation. A fatal termination is generally preceded by a sudden rise, perhaps to 110° F. (43.3° C.), or by a decline to a subnormal degree. The pulse may be rapid or slow. Respiration is usually accelerated. Slow respiration, with dyspnea, due to pressure on the respiratory centers, is sometimes noted in a late stage of the disease. A sighing or a Cheyne-Stokes respiration is sometimes observed.

The nervous manifestations are usually prominent features of the case. After the first delirium the patient may arouse with apparent promise of improvement, but the delirium soon returns and often becomes maniacal. It usually gives place in a few days to a stupor, which may deepen into coma. The face is usually dull and expressionless, except at irregular intervals, when it is drawn into a distressing grimace, and the patient, if a child, utters a shrill, piercing, characteristic cry (the hydrocephalic cry). A spasm of the muscles is not infrequent, when the features remain constantly drawn into a peculiarly ghastly grin (the risus sardonicus). Twitching is frequently observed in the muscles of the extremities; the forearms are flexed upon the chest, and the thighs and legs are flexed in many cases and often rotated to one side. Later the limbs may become fixed in these positions.

Herpes of the lips is seen in about half the cases; petechiæ or purpuric spots sometimes occur, especially in malignant cases, general purpura is seldom encountered, although it gave the name "spotted fever" to the disease. Dusky erythematous spots are occasionally seen over the course of the peripheral nerves. Rose-spots like those of typhoid fever have been observed, and urticaria, ecthyma, pemphigus, and very rarely gangrene of the skin have been recorded.

The vomiting generally subsides after the first day or two, but it is sometimes a distressing symptom throughout the disease. Diarrhea occurs in some cases; constipation is a more general condition. The

urine sometimes contains albumin, less frequently sugar; hematuria is a frequent feature of malignant cases.

Lumbar puncture shows an increase of pressure within the spinal canal, and the fluid obtained is turbid, often purulent, and when examined microscopically reveals polynuclear leucocytes and numerous diplococci. Leucocytosis is usually, but not invariably, observed in the blood-count.

Varieties of the Disease.—The types of the disease generally recognized are the following:

1. The ordinary form that has been described.

2. Unusually *mild* cases, in which headache, vertigo, vomiting, and moderate fever are observed, possibly hyperesthesia and stiffness of the extremities, but the disease pursues a moderate course and recovery is usually complete.

3. *Abortive cases* in which the onset is sudden and often severe, but the symptoms rapidly subside, sometimes with profuse sweating or epistaxis, as if by crisis. Convalescence is established often within the first week.

4. A *malignant form* in which the nervous manifestations are of extreme severity, although the temperature may not be high. The pulse is often below 60 and feeble. A purpuric eruption is usually observed. Death often occurs within 24 hours.

5. An *intermittent type* is recognized, in which the temperature pursues a course more characteristic of pyemia, rising more or less abruptly every day or every second day, falling nearly or quite to the normal in the interval of remission.

6. A *chronic form*, which often lasts five or six months, to be followed even then by incomplete recovery or a fatal issue. A condition of extreme emaciation (marasmus) and various sensory and psychical disturbances, with contractures of the limbs, is generally produced. The protracted course of the disease is marked by occasional recurrences of fever and other symptoms. Osler looks upon these protracted cases as probably due to chronic hydrocephalus or abscesses of the brain.

Complications and Sequelæ.—Lobar and bronchopneumonia, pleurisy, endocarditis, and pericarditis are not infrequent complications. Persistent headache, and various affections of the eye or ear resulting in blindness or deafness, are only too commonly encountered. Deaf-mutism, aphasia, chronic hydrocephalus, imbecility, and various paralyses frequently remain. Arthritis of variable severity is almost always present. In the worst cases the exudation into the joint becomes purulent, and permanent contractures and deformities result. Even after convalescence has progressed favorably for weeks there is no assurance of ultimate complete recovery. Emaciation, anemia, feeble digestion, and general debility often remain for months.

Diagnosis.—The sudden invasion with chill, the intense headache, pains in the neck, back, and extremities, but more particularly the cervical rigidity, explosive vomiting, constipation, photophobia, sensitiveness to sound, and hyperesthesia are always suggestive of the disease, especially if other cases have occurred in the vicinity. Later, the irregular temperature, rapid or abnormally slow and weak pulse, peculiar facial expression, emaciation, muscular tremor, soreness and rigidity, the cry, and other phenomena already reviewed, establish the diagnosis. Kernig's sign is a valuable aid to diagnosis in many cases in which it is

present, but unfortunately it is occasionally absent. It consists in a peculiar flexion of the knees when the patient sits up in bed. When the patient lies upon his back the legs can be flexed or extended by the hand of the examiner, but, if the patient is raised into a sitting posture, his knees become partially flexed and cannot be fully extended on account of contraction of the flexor muscles. The extreme limit of flexure is usually under 135° it may be as low as 90°. The sign indicates simply that the meninges are involved and is not peculiar to this form of meningitis. Unfortunately, cases of pneumonia, typhoid fever, and other affections occur in which the symptoms are suggestive of meningeal involvement, and other forms of meningitis must be excluded.

Simple meningitis is often difficult to differentiate in the absence of an epidemic. As a rule, the onset is less severe, the tremor, contractures, and joint-involvement are less prominent features; the sardonic grin and hydrocephalic cry are not typical, if present.

Tubercular meningitis is generally more insidious in its invasion and appears, as a rule, in persons already suffering from tubercular infection. If this be located in the lung, the bacillus may be found in the sputum. Lumbar puncture proves a valuable aid in the differentiation, since Pfaundler has shown that the pressure of the fluid is often greater in tubercular meningitis than in any other condition, while the fluid may remain nearly or quite clear, a condition never found in the acute disease.

Pneumonia may be complicated by meningeal irritation or inflammation and is then with some difficulty distinguished from a complication of cerebrospinal meningitis, especially if the latter disease be prevalent at the time. In pneumonia the symptoms point more distinctively to an involvement of the cerebral meninges alone. It may, however, require the lumbar puncture and examination of the spinal fluid to determine the real condition.

Typhoid Fever.—In this disease symptoms referable to irritation of the meninges do not usually appear until the second week. The history of the invasion is different. The rose-spots, greater enlargement and firmness of the spleen, the absence of leucocytosis, and positive reaction to the Widal test are usually sufficient to determine the condition.

Typhus fever can usually be excluded by the character of the epidemic prevailing. Both diseases are, however, peculiarly prone to occur in barracks and jails and may be differentiated with difficulty in the start. The extremely high temperature, dusky hue of the face, and less pronounced manifestations on the part of the spinal muscles are often the most valuable symptoms.

Prognosis.—The course of the disease is so variable that the prognosis is made with difficulty. The mortality in different epidemics has ranged from 20 to 80 per cent. In mild cases convalescence begins within the first week; malignant cases generally terminate fatally within the same period; in the ordinary form it begins in from two to three weeks. Park tells us that about 40 per cent of the cases in which the diplococci are found in the fluid removed by lumbar puncture recover, while nearly all those due to the pneumococcus and streptococcus die. The completeness of recovery can rarely be prognosticated.

Treatment.—The patient should be isolated in a quiet, moderately darkened room. All excitement should be avoided. Rest must be secured,

if by the administration of opium. The headache, delirium, restlessness and cervical pains may be moderated by the application of ice-bags to the head and spine, but morphin or other opiate must be resorted to in severe cases. The bromids in large doses may be sufficient in the case of a child, and when it is found necessary to administer opium it should be the camphorated tincture or deodorized tincture, beginning with a small dose. Urethane in 30-grain (2.0) doses is recommended for the relief of the muscular twitchings in an adult; warm baths and cannabis indica for the rigidity. The high temperature is best combated by cool sponging or the wet pack whenever the temperature reaches 103° F. (39.5° C.). The use of the coal-tar antipyretics in this case is generally condemned on account of the weakness of the heart and consequent danger of greater depression. The heart may be strengthened by the free administration of stimulants, which are usually well borne. If the respiration becomes irregular, atropin may be judiciously administered with the morphin. Ergot and belladonna are thought, by some writers, to exert a beneficial influence on the meningeal congestion, but are regarded as of doubtful service by others. Blisters and other irritants to the nape of the neck should be used, if at all, only in the early stage on account of the tendency to bedsores. This tendency should be further guarded against by proper bathing. Wet cups applied to the nape of the neck in the beginning of the disease are sometimes found of benefit.

The diet should receive careful attention. During the acute stage it should be restricted to milk, broths, and beef-juice. Water should be plentifully given. If swallowing becomes difficult, resort to rectal alimentation is necessary. The milk should then be predigested. The bowels should be kept freely open by the saline cathartics, perhaps with an occasional dose of calomel.

After the more acute manifestations have subsided, the iodids may be administered with a view to hastening the absorption of the exudations; the sirup of the iodid of iron is especially indicated on account of the anemia also present. During the convalescence, codliver oil, malt, strychnin, iron, and arsenic are indicated. Massage and electricity may hasten the restoration of tone to the muscles after motion has become established.

PNEUMONIA.

LOBAR PNEUMONIA, FIBRINOUS PNEUMONIA, CROUPOUS PNEUMONIA, PNEUMONITIS, LUNG FEVER.

Pneumonia occurs in all parts of the world, and ranks as one of the most fatal of the acute infections. In the United States the mortality attributed to it is second only to that of tuberculosis, and in many of the cities it outranks the latter disease.

Definition.—An acute infection caused by the micrococcus lanceolatus and characterized by inflammation of the lungs, with fever and other evidences of toxemia.

Etiology.—*Bacteriology.*—The micrococcus lanceolatus, the recognized cause of the disease, is known also as the pneumococcus. It is found in about 90 per cent of all cases in the pulmonary exudate, but has been repeatedly found in the mouths of healthy persons. It is seen in the rusty sputum, sometimes in the blood, and almost always in the

smaller blood-vessels after death. It can be demonstrated without difficulty, since it takes up the usual stains and is identified by its fairly lancet or elliptical shape and encapsulation in pairs, particularly when found in the sputum. The usual avenue of infection is doubtless the respiratory passages, probably the lung itself, although Menzer believes that it may occur through the tonsil. The viability, virulence, and other characteristics of the micrococcus are so different under different circumstances that the existence of more than one species has been suggested. Eyre and Washburn distinguish several types, notably a parasitic, the most virulent and including the pneumococcus, and a saprophytic, almost devoid of virulence and including the species so often found in the respiratory passages of healthy individuals.

Other Organisms.—The Friedlander bacillus pneumoniæ is also found in the lungs, but not so uniformly as the pneumococcus. This is a larger organism, a short rod inclosed in a capsule, and it exhibits very different vital phenomena from those of the diplococcus. The staphylococcus and streptococcus pyogenes are also found in some cases usually associated with the pneumococcus, but they may be found alone, particularly in the pneumonia of children. The bacilli of influenza, diphtheria, and typhoid fever have each been encountered in pneumonia.

The mode of transmission of the disease is not understood. The possibility of direct contagion has been too little regarded in the past, for recent investigations reveal a comparatively frequent occurrence of successive cases in the same locality, as in the house referred to by Schröder, which furnished 32 cases to the clinic of Kiel in 15 years, 6 of them in one year. Endemics including many cases in rapid succession have been repeatedly observed in prisons, camps, and on ships. Tyson refers to 410 cases among a ship's crew of 815.

Age.—The disease occurs at all ages. In childhood it is more frequent before the sixth year. Holt's table of 500 cases in children under 14 shows 15 per cent of cases in the first year, 62 per cent between the second and sixth, 21 per cent from the seventh to the eleventh, and only 2 per cent after the twelfth year. Netter observed a case in which the disease was transmitted from the mother to the fetus, and two instances in which the blood from the uterine vessels of the patient contained pneumococci. After puberty, from decade to decade, there is probably not much difference which cannot be better attributed to other influences than age.

Sex.—Men are oftener attacked than women, probably on account of greater exposure.

Race.—The negro in our country is highly susceptible.

Physical Condition.—The disease is so common among robust workingmen that it was once thought to have an affinity for persons in full vigor. In many, if not in a majority, of cases, however, there has been a previous impairment of health. This may have been induced by fatigue, alcoholism, a chronic disease, or a catarrhal condition of the respiratory passages. A previous attack affords only temporary immunity and it seems to render the individual more liable to the disease in the future.

Some of the other acute infections are more or less frequently followed by lobar pneumonia; among them, typhoid fever, erysipelas, dysentery, and cerebro-spinal meningitis. Tuberculosis does not notably increase

the susceptibility to it; asthma, emphysema, and valvular diseases of the heart are thought to lessen it. Excessive indulgence in alcohol is one of the most universally recognized predisposing causes of the disease. In the large proportion of the cases admitted to hospitals the disease follows a debauch. Many cases follow exposure to cold and wet.

Climate is not an important factor, since the disease prevails almost everywhere, in warm climates as well as in cold. In some countries, as in Switzerland, it is met with especially in the higher altitudes. Winter and spring are the seasons of greatest prevalence in our country.

Trauma in which the lung has been injured, with or without fracture of the ribs, has been followed by pneumonia in some instances, and it is possible that such injury favors the entrance of the pneumococcus.

Morbid Anatomy.—Three distinct stages are recognized in the inflammatory process in the lungs: Engorgement, red hepatization, and gray hepatization.

Engorgement.—The first stage is one of hyperemia or congestion. The vessels are distended with blood. The affected portion of the lung is dark red, heavier than normal, though it still contains air, crepitates on pressure, and floats in water. When it is incised, bloody serum flows from the cut surface. The alveolar spaces are diminished in size by the thickening of their walls, but they contain as yet no exudate. With the occurrence of exudation the condition passes into one of solidification or red hepatization.

Red Hepatization.—In this stage the exudation soon becomes completely coagulated within the alveoli. The walls show less evidence of congestion than in the first stage. The lung is as firm as liver, contains no air, and is increased in volume to such an extent that it usually shows the indentations of the ribs. It is also friable, so that it can easily be crushed between the fingers. The surface of a section is comparatively dry, has a reddish brown color, and appears granular, owing to the projection of minute fibrinous casts from the alveoli. The exudation consists principally of fibrin, in the meshes of which are numerous red blood-corpuscles, polynuclear leucocytes, and epithelial cells. The diplococci can also be seen in stained preparations.

Gray Hepatization.—The conditions found at this stage are the result of degenerative changes in the exudate of the previous stage. The lung is still firm and even more friable, but has a gray or yellowish gray or mottled color. The cut surface has the same color and is smooth and moist. The fibrin and erythrocytes have disappeared, and the exudate is composed chiefly of leucocytes, epithelial cells, and pus-corpuscles. This stage merges into the resolution, or perhaps it would be proper to say that it is a part of the latter process, having for its object the removal of the exudation. This is accomplished chiefly by absorption into the blood current; in part also by expectoration.

The pathological process of pneumonia probably begins by a destruction and desquamation of the epithelium of the finer bronchi and air-cells by the micrococci, and, as in other croupous inflammations, a coagulable exudate forms on the injured surfaces. (See p. 30). Later the white corpuscles migrate from the vessels into the exudate, and the red corpuscles and fibrin are dissolved by a chemical process sometimes regarded as a form of peptonization. After the absorption

of the exudate the epithelium is regenerated. The entire process usually runs its course within ten days. When this reaches the periphery of the lung, the pleura invariably becomes involved in a fibrinous inflammation. Sometimes the process of resolution passes into suppuration and abscess-formation, occasionally terminating in gangrene of the lung.

The extent to which the lungs are involved in the disease is very different in different cases. It may be confined to a single lobe, or it may be more extensive. The lower lobes are most frequently involved; the upper lobes rank next. The corresponding lobes of both lungs are sometimes affected (double pneumonia). The right middle lobe is seldom independently involved. Strümpell, in 244 cases, saw the right lung affected in 137, the left in 86, and both lungs in 21. Involvement of the lower lobe of one lung simultaneously with the upper lobe of the other lung is a condition rarely met with (crossed pneumonia). It is not unusual to find red hepatization in one lobe and gray in another in the same patient. Less often they are found side by side in the same lobe, and occasionally all three stages are simultaneously present. These phenomena result from the successive involvement of different areas.

Symptoms.—*Typical Case.*—The incubation is probably short, only a day or two, for the initial chill frequently occurs within a few hours after some unusual exposure. Prodromal symptoms are often absent; there may be a catarrh of the upper respiratory passages for a day or two; sometimes headache, pains in the limbs, and anorexia are complained of. The onset is almost always abrupt with a chill which often lasts from fifteen to thirty minutes or longer. The chill may, however, be slight, it is occasionally absent, seldom repeated. It may seize the individual in the midst of his work; it often comes on at night during sleep. While the patient still shivers, the temperature rises, and within a few hours it reaches 104° or 105° F. (40°—40.5° C.). Its course is high throughout the disease, the diurnal fluctuation often amounting to but 1° F. It almost always terminates by crisis. The pulse becomes rapid, generally 110 to 120, full and bounding, seldom becoming dicrotic at any time. Shortly after the chill, sometimes before it, the patient is seized with a sudden, often agonizing pain in the affected side of the chest. At the same time, and partly as a result of the pain, the respiratory movements increase in frequency, and the movements are shallow, particularly on the affected side. A dry, painful, suppressed cough is also present, but in a day or two this becomes moist, and the characteristic bloody, "rusty" sputum is expectorated. The dyspnea is sometimes distressing, and the expirations are frequently accompanied by moans.

The patient's position and appearance are often typical of the disease. He lies at first on his back; but as soon as solidification has occurred he turns upon the affected side and assumes an attitude of restraint, avoiding motion on account of the pain it occasions. The face is flushed, often dusky, and in the center of each cheek, or frequently only in that of the side corresponding to the affected lung, there is a bright red spot. The expression is that of anxiety and pain. The tongue is furred and often becomes dry and brown. Vomiting sometimes occurs, especially in children; the bowels are generally constipated. A herpetic eruption

appears in about a third of the cases, on the lips, about the angles of the mouth, on the chin or nose, occasionally about the eyes, or on the helix of the ear, rarely on the genital or anal region. Sleep is usually restless, or insomnia may prevail. Delirium not infrequently develops; it is especially frequent and severe in alcoholic subjects.

The disease runs its course in from three to ten days; then, as a rule, terminates by crisis. The temperature falls within a few hours to the normal or lower, the pulse and respiration become slow, pain and dyspnea vanish, and the patient falls into his first peaceful slumber. For a few hours he is usually bathed in a profuse sweat; diarrhea sometimes occurs, but soon ceases after the crisis is over. The cough may continue for a few days, but the blood disappears from the sputum and the quantity of the expectoration rapidly diminishes. Convalescence is usually rapid, and recovery may be complete within a week or ten days.

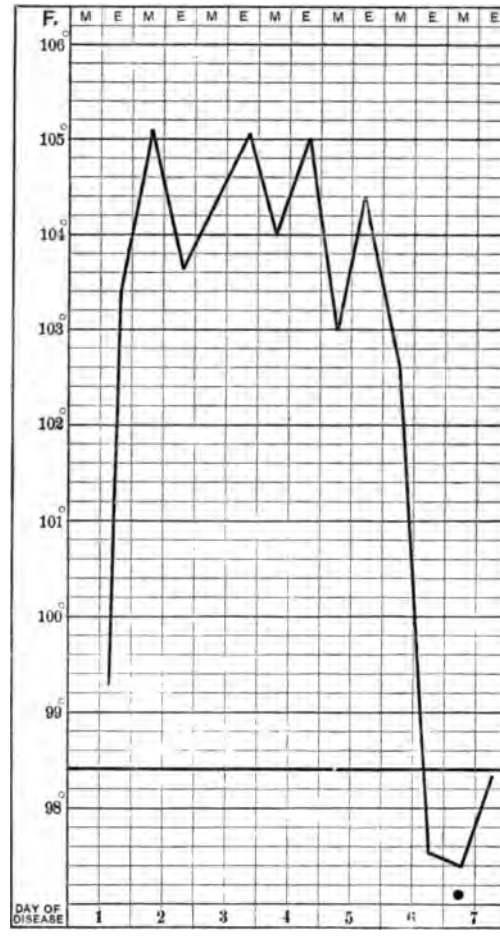

FIG. 11.—Temperature chart of pneumonia, terminating by crisis on the sixth day. (Ziegler.)

Special Symptoms. — *The Chill.*—There is no other disease in which the invasion is so constantly announced by chill or in which the chill is uniformly so severe. It is a pronounced rigor, often lasting more than a half-hour; it is seldom repeated. Chills may, however, occur during the course of the disease, and then generally signify an involvement of an additional area of the lung. The chill is absent, according to some observers, in about 20 to 30 per cent of the cases.

The Fever.—The distinctive features of the fever are the sudden rise of temperature, often to 104° or 105° F. (40°—40.5° C.) within 12 hours; its uniform course frequently not varying more than 1° or 1.5° F. during two or three days, and its termination by crisis. The surface temperature often shows comparatively little increase; hence the rectal temperature may be several degrees higher than that of the axilla.

Irregular types of fever are sometimes observed, as when the temperature, alone or along with the other symptoms, more or less completely subsides before the crisis. A drop to normal, with amelioration of the other symptoms, sometimes occurs, and is then followed by a hyperpyrexia which in turn ushers in the crisis. A convulsion or vomiting sometimes takes the place of the chill in children, in whom the rise of temperature is also more gradual and the daily fluctuation may be greater. The same conditions are sometimes observed in adult cases beginning without a chill. In very old persons the temperature may be but slightly elevated, and the termination is often by lysis. Hyperpyrexia is not uncommon. The temperature may reach 107° or 108° F. (41.6°—42.2° C.), even 109.5° F. (43.0° C.), as in a case reported by Ironside.

The crisis may occur at any time from the third to the tenth or twelfth day. A peculiar frequency of its occurrence on odd days, as on the seventh, ninth, or eleventh, has been repeatedly noted, but it not infrequently occurs as early as the end of the sixth day. The fall of temperature is often very abrupt, and it may amount to 5° or 6° F. (2.7°—3.3° C.) within from three to eight hours, sometimes becoming from 1° to 3° F. subnormal. The temperature sometimes rises one or two degrees just before the crisis, and it may rise to 102° or 103° F. (39.0°—39.5° C.) in 24 hours after the crisis and remain elevated for two or three days.

A pseudocrisis is sometimes observed in which, particularly on the fifth or sixth day of the disease, the temperature falls to 101° or 102° F. (38.5°—39.0° C.), but immediately rises again to its former height. It is usually indicative of the involvement of additional lung space. The crisis may still occur, but lysis is the usual termination in protracted cases.

Pain is constantly present at the beginning. It may subside to a great extent after complete solidification of the lung. It is often excruciating, and is greatly increased by coughing or efforts at full inspiration. In a double pneumonia the moans of the patient can often be heard at a great distance, notwithstanding the restricted breathing. The pain is confined to the affected side of the chest and is usually referred to the lower axillary space or to the region of the nipple. The pain is probably due almost solely to the accompanying pleurisy, for it is often slight in a central pneumonia.

The *dyspnea* varies in severity with the extent of lung involved, the degree of temperature, the amount of pain, and probably in a great measure with the extent to which the toxemia affects the respiratory centers. It is always a prominent symptom. The respiratory movements are usually increased to 30 or 40, often to 50 or 60 a minute. In children they may reach 80 or 100. They are shallow, but regular, as a rule. The dyspnea is accompanied by the usual signs, movement of the alæ nasi, and a sense of suffocation or of constriction beneath the sternum. But, as Grisolle remarks, the most rapid respiratory movements in some patients may be only a manifestation of nervous irritation at the pain and may not denote so great dyspnea as is often indicated by 24 or 28 respirations a minute in other cases. The disturbance of the ratio of respiration to pulse-rate is a striking and characteristic feature of pneumonia. Instead of the normal ratio of 1 :4, it is

often 1:2 or 1:1.5, for the pulse may be only 120 when the respirations are 60 or more.

Pulse and Heart.—The acceleration of the pulse is moderate in most cases, being strikingly out of proportion to the respiration as stated in the last paragraph. The pulse is usually full and bounding in the beginning; it may become weak after a few days and occasionally dicrotic. In alcoholic cases and old persons, it is frequently feeble and rapid from the beginning. The second sound of the heart is accentuated owing to the increased tension in the pulmonary circulation, and a temporary systolic murmur is often heard over the pulmonary valve; sometimes also over the mitral. A fatal cardiac distention or paralysis sometimes results from trifling exertion in the presence of profound toxemia.

The cough is an early symptom, rarely absent except in infants and very aged or feeble persons, but it may be suppressed on account of the agonizing pain which is occasioned by it. It is at first short, sharp, and dry, but by the second or third day it is attended with expectoration. The sputum is at first mucous in character, but after 24 to 48 hours it becomes tinged with bright arterial blood. The color soon changes to an orange-yellow, iron-rust color, which has given it the name of rusty sputum. Free hemoptysis occasionally occurs at the onset of the disease. The sputum is often so tenacious and viscid that it clings to the tongue and lips and is wiped away with difficulty; the cup can often be inverted without spilling it. In some cases, however, particularly in asthenic cases, the sputum is more fluid and has the dark brown color of prune-juice, a name often applied to it. The quantity of the expectoration is exceedingly variable. Occasionally there is no expectoration, or the sputum may pass into the esophagus and escape observation, particularly in women and children or in the presence of extreme prostration. Microscopic examination of the sputum reveals numerous degenerated bronchial, probably also alveolar, epithelial cells, normal and degenerated erythrocytes and leucocytes. The micrococcus lanceolatus and other bacteria are often present in great numbers.

Cerebral Disturbances.—Headache is a common symptom. Convulsions rarely occur except at the beginning of the disease in children. In some cases of so-called cerebral pneumonia in children, the rigidity and retraction of the neck, and the muscular twitching form a picture very suggestive of cerebrospinal meningitis. Delirium is a not unusual symptom, especially in alcoholic subjects, children, and the aged. It is often mild in character, but it may become maniacal, particularly in the drunkard. Typical delirium tremens not infrequently develops on the third or fourth day in these cases. Such patients often show a propensity for getting out of bed, and have jumped out of windows in the absence of the attendant. Delirium is frequently absent, however, even in fatal cases among alcoholic patients. Exceptionally, the delirium, instead of ceasing at the crisis, becomes more violent for a few hours, and in another class of cases it makes its first appearance at this time, but it is then usually of short duration. Other cerebral disturbances continuing after the crisis usually terminate favorably.

Toxemia.—Cases are now and then observed in which toxemia resembling uremia is present. Chill, pain, and cough may all be absent in the

beginning, and the fever slight, but in a few days the temperature rises and the patient passes into a low, muttering delirium, or a coma which proves rapidly fatal.

The Blood.—The most characteristic feature of the blood is simple leucocytosis, developing early, continuing to the crisis and sometimes reaching the extent of from 30,000 to 60,000 per c.mm., or from three to eleven times the normal limit. Polynuclear corpuscles are most abundant in the beginning, but eosinophiles become numerous later. Absence of leucocytosis is an unfavorable sign usually seen in protracted cases. Stockton, however, reports the case of a little girl with two relapses and ultimate recovery, in which there were only 4,000 leucocytes to the c.mm. during the first relapse. As is remarked by Thompson, the theory that the leucocytes are active agents in the production of immunity receives much support from this clinical fact. It may be applied thus: "The toxin formed by the pneumococci is active for a few days until sufficient leucocytes accumulate to manufacture antitoxin to destroy it, producing a crisis. Absence of leucocytosis gives free scope to the toxin in severe cases. Insufficient leucocytosis postpones the crisis and resolution." The micrococci are recognized in the blood with difficulty.

The urine is high in color, specific gravity, and solids, particularly in uric acid, and a trace of albumin is often present. As in other fevers the chlorids are generally, though not always, diminished, a phenomenon which is perhaps best explained by Koranyi's theory of molecular interchange in the kidney. F. Pick calls attention to the fact that in from 24 to 48 hours after the crisis the urine often becomes neutral or alkaline for a period of 24 to 36 hours, after which the acidity returns. Sternberg regards secondary infection of the uriniferous tubules by the pneumococcus as probably not infrequent. The toxicity of the urine has been found greatly reduced during the disease.

Physical Examination.—1. During the stage of congestion, inspection shows a diminution of the movement of the affected side, and in double pneumonia the breathing is chiefly abdominal. By palpation, an increased fremitus is generally noticed over the affected area. Percussion reveals dullness or a high-pitched tympanitic note over the region involved and a tympanitic note over the surrounding region of the lung. On auscultation, the breathing is found to be bronchovesicular in character and it is reduced in amplitude. An exaggerated respiratory murmur may be heard over the other regions of the chest. Subcrepitant râles are usually present, or the crepitant râle may be heard at the end of a forced inspiration.

2. *Stage of Hepatization.—Inspection.*—The respiratory movement of the affected side is much limited if an entire lobe is involved, and a corresponding increase in that of the opposite side is observed. The healthy side of the chest may appear larger than the affected side. The tactile fremitus is generally increased. A pleuritic friction fremitus may be felt. On percussion the note varies from a tympanitic dullness to flatness; occasionally a metallic quality can be detected. In central pneumonia the dullness may be almost unrecognizable, although the sense of resistance imparted to the finger may be increased. In children, careful, light percussion is always required to elicit dullness. Auscultation reveals typical tubular breathing and bronchophony in most cases;

rarely egophony, over the affected area. Subcrepitant râles are not infrequently heard. All sounds may cease when the bronchi become completely closed by the exudate.

3. *Resolution.*—During this period the respiratory movements become less restricted and percussion resonance gradually increases. The subcrepitant soon give place to coarser moist râles, which may continue throughout the week or more of absorption of the exudate. The respiration again becomes bronchovesicular and finally returns to its normal quality.

Varieties of Pneumonia.—This term is employed, not to designate different types of the disease, but rather the different features presented in different cases. The affection is, so far as is known, the same in all cases, and the variations probably result from differences in the age, vulnerability or susceptibility of the patient, or from accidental or unrecognizable influences. The terms *apical* and *basal* are sometimes applied to the disease to indicate that the apex or the base is involved.

Frank pneumonia is merely a synonym for the ordinary type of the disease.

Epidemic pneumonia is usually a virulent type, often occurring in individuals previously debilitated by influenza or other infection.

Migratory pneumonia is a frequently fatal form in which one area after another is involved until the vitality of the patient has been exhausted.

Massive pneumonia is that condition in which an unusually large area of the lung is solidified and the bronchi are completely filled with the exudate. Expectoration may be absent.

Central pneumonia is a rather frequent form, at least in the beginning of the attack. In it the exudation is often confined to the center of a lobe or to the base of the lung and does not reach the periphery during the first two or three days of the disease. Both the subjective and objective signs may be very indefinite during this time.

Secondary pneumonia is a term sometimes used when the disease has developed as a complication of other infectious diseases. The base of the lung is usually affected and the diagnosis may be difficult, particularly when other micro-organisms than the pneumococcus are present.

Bacelli describes a form of pneumonia which follows pleurisy. The signs of pleurisy are distinct on the first day. On the fourth or fifth, symptoms of intense pulmonary edema appear, dyspnea, serous sputum, blood-stained or hemorrhagic. The disease runs a rapid, often fatal course.

Terminal pneumonia signifies that the disease has developed in an aged person or one who is the subject of a chronic disease. In other words, it becomes the fatal termination of senile debility, tuberculosis, cancer, cardiac or renal disease. This part is more frequently played, however, by bronchopneumonia or hypostatic congestion.

Alcoholic pneumonia is often masked by the predominance of the acute symptoms of intoxication. It should be recognized, however, upon examination, the necessity of which is indicated by high temperature, rapid respiration, dyspnea, often with cyanosis.

Typhoid pneumonia is a term without much to recommend it. It is used to designate either pneumonia complicating typhoid fever, or a

condition of stupor like that of typhoid fever supervening upon a lobar pneumonia. Either condition is grave.

Pneumonia in infants usually begins with a convulsion and runs an irregular course, often affecting the apex, attended with delirium, and terminating by lysis, occasionally by crisis. (Fig. 12).

Pneumonia in the Aged.—The peculiarities of the disease in old people have already been stated. Bronchopneumonia is more common in them, as also in children.

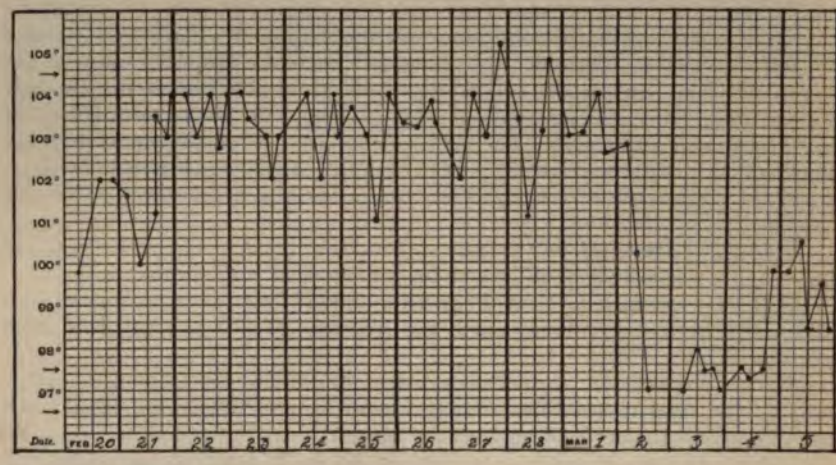

FIG. 12.—Temperature chart of a case of pneumonia, terminating by crisis, in an infant of nine months. Pseudocrises are shown also upon the sixth and ninth days.

Apyretic pneumonia has been described. Guider attributes the absence of fever to exhaustion of the economy, functional disturbance of the nervous system, and the action of infectious agents.

Pneumonia after Surgical Operations.—The occurrence of lobar pneumonia after surgical operations is probably purely accidental and is very infrequent. Ether pneumonia and respiration pneumonia are both bronchopneumonias and should not be confounded with this disease.

Delayed Resolution.—In a typical case of pneumonia, resolution does not occupy more than a week or ten days. The cough ceases, and physical examination reveals no abnormal condition further than slight dullness due to thickening of the pleura. This dullness sometimes persists for a month or more. The resolution is sometimes delayed beyond the usual limit, particularly in debilitated subjects, but sometimes also in robust individuals in whom the slow recovery is unaccountable. In some cases the consolidation of lung continues, with or without fever. In other cases, after the temperature has subsided by crisis or lysis, it returns again at intervals, or runs an irregular course for several weeks. There may be intense bronchial breathing, but there is usually no cough or expectoration. In other cases, again, the moist râles and expectoration continue. The temperature curve and frequent sweating in some cases suggest the possibility of sepsis. Tuberculosis

rarely follows lobar pneumonia, but a latent pulmonary tuberculosis may be awakened by it.

Relapse.—A true relapse of pneumonia is seldom seen. In some cases, however, there is an apparent recrudescence due to delayed resolution or to the development of complications. In other cases the apparent relapse is due to the invasion of additional lung space, with return of the symptoms, at a time when the crisis is being looked for, but the condition can hardly be regarded as a relapse.

Recurrence is not infrequent. There have been well-authenticated instances in which the same person passed through eight or ten attacks.

Complications and Sequelæ.—*Pleurisy* is so constantly an accompaniment of pneumonia that it may well be looked upon as a part of the disease, rather than as a complication. It is absent only in central pneumonia before the inflammation has reached the surface. In some cases, again, the pleural inflammation predominates to such a degree that the condition is frequently spoken of as a pleuro-pneumonia. The fibrinous exudation is always unusually abundant in these cases and interposes a thick cushion between the consolidated lung and the chest wall, greatly interfering with the transmission of the respiratory sounds. A serofibrinous form of pleurisy is not infrequently encountered in long-continued cases. Occasionally it becomes suppurative and an empyema may be developed. In such cases it is the rule to find both the pneumococcus and streptococcus in the fluid. The condition is recognized by a continuance of the fever, usually with a remittent or intermittent course, and persistence of the leucocytosis, frequently with sweating and other signs of pyothorax. The diagnosis is established by the withdrawal of pus through the aspirator needle.

Endocarditis is a comparatively frequent complication, especially in the presence of an old valvular lesion, and perhaps for that reason it is more frequent in the left heart. It may follow the crisis. Its development is often concealed, there being no murmur even in severe cases; and, as already stated, a murmur may be present for a time without lesion of the valves. In other cases, a rough diastolic murmur is heard. A malignant endocarditis is to be suspected, however, when chills, irregular fever, and sweating continue to occur, and particularly when embolic infarctions develop in any of the organs. The pneumococcus is sometimes found in the cardiac vegetations after death. Meningitis is not infrequently associated with a malignant endocarditis of this character.

Pericarditis is seldom met with in adults and is more frequent in the left-sided pneumonia of young children. It is a result of an extension of the inflammation from the left pleura to the pericardium. It may be recognized early, before effusion has taken place, by the friction sound, or if this be absent, by the precordial pain, increased heart dullness, intense dyspnea, indistinct heart sounds, and the feeble pulse.

Embolism has been found in the larger arteries; thrombi occasionally form in the veins. Aphasia, with or without hemiplegia, has been observed in a few instances, probably as a result of embolism.

Meningitis, attributed to the migration of the pneumococcus, is encountered during the height of the fever in some cases. It usually affects the cerebral cortex, but is much more easily recognized when it attacks the base. It is then indicated by severe headache, sluggish response or

unequal dilatation of the pupils, rigidity and retraction of the neck, with a tendency to delirium or stupor.

Croupous colitis occasionally develops late in the disease.

Jaundice appears early in some cases. It is seldom severe in character. The mode of its production is not understood. Some writers regard it as a true hematogenous icterus.

Acute articular *rheumatism* may precede, accompany, or follow pneumonia, and it is frequently accompanied by endocarditis or pleurisy. Among the rarest complications are acute nephritis, otitis media, croupous gastritis, parotitis, peritonitis, and peripheral nueritis. Abscess and gangrene of the lung sometimes follow imperfect resolution.

Diagnosis.—Pneumonia is seldom difficult of recognition except in children and very old or feeble patients, too weak to expand the chest and bring out the auscultatory signs. It may be readily overlooked when it develops insidiously during the course of another affection. The attitude in bed, the anxious expression, flushed face with bright red cheek, the chill, fever, pain, rapid pulse with disproportionately rapid breathing, the cough and expectoration of rusty sputum, leave little chance for error in a typical case. When there are added to this the physical signs, particularly the bronchial breathing and the crepitant râle, the picture is complete. In children the diagnosis is often difficult either on account of the predominance of meningeal irritation or the absence of characteristic features. Percussion must always be performed with delicacy in a child in order to elicit the dullness, for a resonant tone may be transmitted from the viscera beyond the area of consolidation, on forcible percussion. An agglutinative reaction has been obtained upon the pneumococcus with the serum of individuals suffering from pneumococcus infection, but its utility as a test for the disease has not yet been demonstrated.

Pleurisy and Empyema.—A pleuritic effusion, especially if purulent, may cause confusion unless the exploratory needle is used. In pneumonia, however, there is no displacement of the heart or other viscera, as in hydrothorax. The presence of vocal and tactile fremitus cannot be relied upon in the exclusion of pyothorax.

Acute pneumonic phthisis runs a slower course, as a rule. The sputum has the prune-juice appearance rather than the rusty color, and the sweating, rapid emaciation, and irregular temperature are distinctive. The delirium is often more constant and more severe than that of pneumonia.

Typhoid fever may begin with rapid respiration and other symptoms suggesting pneumonia, and, on the other hand, the pneumonia patient may sink into a typhoid stupor. The temperature curves are quite different, and the absence of the rusty sputum, the presence of rose-spots, pea-soup stools, a positive Widal reaction, and the absence of leucocytosis in the former disease render the distinction positive in most cases.

Prognosis.—Pneumonia is one of the most fatal of the acute infectious diseases. The prognosis is modified, however, by the age, sex, and vigor of the patient, and to a very marked extent by his habit as to indulgence in alcohol. In private practice the mortality is usually from 10 to 15 per cent, in hospitals from 20 to 30 per cent. In epi-

demics it often exceeds 50 per cent. The death-rate is very different in different localities, at different seasons, and in different classes of patients. It is more fatal in the extremes of life. Infants under one year and adults over 65 seldom recover. Alcoholic subjects and the victims of chronic disease are especially liable to succumb. Children almost invariably recover, and the prognosis is generally favorable in robust adults of middle age. Much depends in any case upon the degree of the toxemia, as manifested by high fever, delirium, prostration, and feeble circulation. Diffuse bronchitis adds gravity to the case. Absence or deficiency of leucocytosis indicates feeble resistance on the part of the economy; and increased heart action, cyanosis, or disappearance of the second sound over the pulmonary valve late in the disease are of evil import, indicating a failure on the part of the heart. Death is usually due to an intense action of the toxin upon the heart or central nervous system, or a sudden, fatal dilatation of the right heart, seldom solely from interference with the respiration, even in extreme cases of double pneumonia. A complicating endocarditis is not necessarily fatal, but a meningitis is almost always so. The pulse is not always a safe guide to the prognosis, for a slow, full, compressible pulse often accompanies the failure of the circulation which precedes death by only a few hours.

Prophylaxis.—Comparatively little is known on this subject. Probably too little attention has been given to the danger of communicating the disease through careless disposal of the sputum. The fact that in a few instances the nurse has become infected while waiting upon the pneumonic patient suggests the advisability of adopting measures of thorough antisepsis, including destruction of the sputum by fire or steam and thorough disinfection of the apartments after the recovery of the patient. N. S. Davis, Jr., recommends the use of antiseptic mouth-washes several times a day.

Treatment.—*General.*—The treatment of a case is chiefly symptomatic. We have no means of cutting short an attack, and probably none of greatly modifying its course. As Osler remarks, "Patients are more frequently damaged than helped by the promiscuous drugging which is still too prevalent." Good nursing and the amelioration of symptoms are, however, requisite. The patient should be placed in a quiet, sunlit, well-ventilated room kept at a temperature between 65° and 70° F. (18°—20° C.). Visitors should be excluded. Ordinary ventilation is not enough. The patient should be placed in the open air or directly at an open window when the weather will permit. The relief experienced in a severe case by this treatment is remarkable. Frequent sponging is beneficial for stimulation and for the reduction of temperature. The Brand method, however, has not given good results. The cold pack may be employed, and the warm plunge of 90° F. (32.0° C.) is beneficial to children. The diet should be liquid; milk, broths, gruels, and soft eggs may be given. Plenty of water, plain or carbonated, or lemonade should be given at regular intervals. The patient should under no circumstances be permitted to raise even his head. Food should be administered through a tube. The bowels should be kept open. A dose of calomel may be given in the beginning, and a saline laxative at intervals during the disease. Tympanites must be immediately relieved by the application

of turpentine stupes or the adminstration of 5 to 10 drops of turpentine every hour or two, on account of its interference with respiration. The rectal tube will sometimes remove the flatus.

Local applications of heat or cold, especially the application of the continuous ice-bag, to the affected side afford great relief. Counter-irritants are still employed by some, blisters are rarely used. The pneumonia jacket, consisting of a thick layer of cotton covered with oil silk, is employed by some physicians, but it is generally better to leave the chest accessible. Bleeding at the beginning of the disease has been found highly beneficial in robust individuals with full, bounding pulse, prominent arteries, and high fever. Dry cups, over the affected area, and wet cups afford marked relief when applied early in cases beginning with severe pleurisy and edema of the lungs.

Drugs.—The most important of these are the cardiac stimulants, especially strychnin, digitalis, nitroglycerin, and alcohol. Each should be given with reference to the effect obtained, and not by rote. Strychnin should be given hypodermically in doses of gr. 1-60 to 1-20 (0.001—0.003) every three hours unless muscular twitching is produced. Nitroglycerin, gr. 1-100 to 1-50 (0.0005—0.001), may be given when dyspnea or other symptoms indicate a disproportion between the heart's action and the arterial tension. Digitalis should be given in full doses of the infusion, an active tincture, or fluid extract. Ammonium carbonate, gr. v—x (0.3—0.6), and citrated caffein, gr. ij—iij (0.1—0.2), are often beneficial. Veratrum viride, once much in vogue, is employed by some physicians, but should be used with great caution, in 2 to 5 drop doses of the tincture only until the heart's action has become normal.

Diaphoretics should not, as a rule, be employed, although they have been thought to reduce sweating and prostration during the crisis if administered before it. Alcohol is highly regarded by some writers. As a rule, its use is unnecessary, if not injurious, and it is inferior to strychnin, even in alcoholic cases. Quinin is the safest antipyretic. The coal-tar antipyretics are unsafe, as a rule.

Creosot carbonate has recently been employed in many cases, with results which apparently justify the claim that it is a specific. It immediately reduces the temperature, quiets the cough, and often produces an early termination by lysis or by crisis free from weakness and prostration. It should be given in doses of gr. v (0.3), increasing rapidly to gr. xv (1.0), every two or three hours, from the beginning.

Treatment of Special Symptoms.—Pain must be relieved at the onset, often by morphin hypodermically, and followed with codein at frequent intervals. But the action of an opiate must be watched, lest it produce cardiac depression. Holt regards phenacetin as better than opium for restlessness and cough in children. A single dose in 24 hours is often sufficient.

Inhalations of oxygen often assist the patient to reach the crisis. It is administered pure for 15 minutes at a time, or, diluted with atmospheric air, for longer periods. It must be pure and fresh.

Edema of the lungs may be checked by cupping and the administration of atropin, gr. 1-100 (0.0005), with each dose of strychnin, to check secretion.

Delirium may be modified by the administration of the bromids in

full doses, but morphin, gr. ⅛ to ¼, may be required. Stimulation and cold sponging often have a beneficial influence upon it.

Saline injections have been employed in severe cases, and apparently with benefit, in carrying the patient over a critical period. From one to two pints of a 7 per cent solution are allowed to flow by gravity through a large hypodermic needle into the subcutaneous tissue.

Serum Treatment.—If the theory be true that the crisis announces the victory of the antitoxin produced by the system over the toxin of the disease, there is much reason to hope for a successful serumtherapy. The results obtained in this direction have been encouraging. The serum of Pane, obtained from the larger animals, is injected into the subcutaneous tissue in the quantity of from 40 to 120 c.c. in 24 hours. Intravenous injections have been made in a few instances. The serum of convalescents has also been employed.

DIPHTHERIA.

DIPHTHERITIS, ANGINA MALIGNA, PUTRID SORE-THROAT.

Definition.—An acute infectious disease occurring sporadically or epidemically, caused by the Klebs-Löffler bacillus, and attended with a fibrinous exudate on the mucous membrane of the pharynx and upper respiratory passages and symptoms of toxemia.

Etiology.—The Klebs-Löffler bacillus is constantly found in the pseudomembranous deposit on the throat, and is recognized as the specific cause of the disease. Diphtheria is endemic in most of the cities and towns of the United States and frequently becomes epidemic during the winter season. Sporadic cases occur and are often virulent in character. The disease is encountered in all climates and its spread is favored by the cold of winter and spring. It is highly contagious and may be contracted by direct contact. It has frequently been conveyed by kissing, and physicians have been repeatedly inoculated by injudicious attempts to clear by suction an obstructed trecheotomy tube, or by having the membrane coughed into their faces while examining or treating the throat. The infectious agent may be carried by clothing, bedding, handkerchiefs, drinking-cups, hair, shoes, pencils, and other articles from the sick-room, but probably not by the air or by sewer-gases. So far as known, it is carried by pet animals only in their fur. The so-called diphtheria of animals and birds is generally due to other bacteria than the diphtheria bacillus. The bacillus is so tenacious of life when protected from light and air that it has been known to retain its virulence for several months, and to remain alive in the throat for almost a year. It grows freely in milk, and cases have been traced to this source. It may be carried in the throat of a healthy individual without producing any disturbance for days or weeks. As W. H. Park remarks, "When we consider that it is only the severe cases of diphtheria that remain isolated during their actual illness, the wonder is not that so many, but that so few, persons contract the disease."

The bacilli are believed to enter the system only through inhalation, by being conveyed in some other manner to the mouth, or by the inoculation of an abraded surface in some other part of the body.

Age is an important factor in susceptibility. A large majority of cases occur between two and fifteen; the period of greatest susceptibility appears to be between the third and tenth years. The disease seldom occurs in infants or in adults after the thirtieth year, but A. Jacobi has seen it in the new-born and in a man of 86 years. Some individuals and some families are undoubtedly more susceptible to infection than others. Epidemics vary greatly in severity. Caillé emphasizes the importance of enlarged tonsils, chronic nasopharyngeal catarrh, carious teeth, and an unhealthy condition of the mucous membrane of the mouth and throat as predisposing causes.

Immunity.—Some degree of natural immunity is probably possessed by many persons, else the disease would be much more prevalent than it is. It probably depends upon the inability of the bacillus to penetrate into a healthy mucous membrane. An attack of the disease confers immunity for an indefinite, but probably short, space of time. Second attacks have frequently been seen after a few months. Golay reports a case in which the bacilli were constantly present in the throat of an individual for 362 days, during which time he had three acute attacks.

Bacteriology.—The diphtheria bacilli, as they are usually found in the false membrane of diphtheria, are straight or slightly curved, nonmotile rods from 1.5 to 6.0μ in length and from 0.5 to 0.8μ in thickness, with rounded or clubbed extremities. They are not usually uniformly cylindrical, but are thicker at one end, or swollen in the middle and more slender at the ends. They are sometimes found in pairs, rarely in chains. What appear to be branching forms are sometimes seen. The bacillus is an aërobe, but varies greatly in morphology and other qualities under different methods of cultivation. It is very resistant to extreme cold, but readily succumbs to a temperature of 58° C. The usual stain for it is Löffler's methylene-blue solution. Neisser's differential stain should be employed to distinguish the true bacillus from the pseudobacillus. It is almost the rule to find in the false membrane also streptococci, staphylococci, or pneumococci. The streptococcus pyogenes is usually found in suppurating glands when they occur in this disease.

False Diphtheria.—Other bacteria besides the Klebs-Löffler bacillus are capable of producing pseudomembranous inflammation. Some of these are almost constantly to be found in the secretions of the throat, and only under favorable circumstances produce lesions similar to those of diphtheria. The streptococcus and pneumococcus most frequently act in this capacity. In false diphtheria, these forms are sometimes found, but in many cases a pseudodiphtheria or diphtheroid bacillus occurs which is different in morphology and culture from the Klebs-Löffler bacillus.

Morbid Anatomy.—The distinctive lesion of the disease is the pseudomembrane, which consists of a fibrin reticulum inclosing in its meshes leucocytes in a state of hyalin degeneration, degenerated epithelial cells, and occasionally a few erythrocytes. The membrane dips deeply into the epithelial layers of the mucous membrane, but does not invade the submucosa. The mucosa undergoes rapid degenerative changes and necrosis. A change occurs in the deeper tissues, also, which is in part due to degeneration, in part to infiltration; necrosis may occur in them. The bacilli are usually found in the membrane, and they alone are charac-

teristic of the diphtheritic membrane. Other cocci are generally present. The false membrane is more frequently found upon the tonsils. It is rarely confined to them, but in more than half the cases it is limited to the tonsils and uvula. It often spreads from these to adjacent surfaces. It may invade, primarily or secondarily, the pharynx, nasal chambers, larynx, trachea, and bronchi, involving the entire surface, or pass forward over the soft palate and pillars to the mucous membrane of the entire mouth; occasionally even over the lips to the face. The membrane has rarely been found in the esophagus, stomach, rectum, bladder, vagina and puerperal uterus, and on the external genitals of both sexes. Its character varies. It is, as a rule, thick, tough, and elastic, usually laminated, rarely thin, but always firmly adherent except in the larynx. It may be an eighth-inch thick. After death it becomes soft and friable. The capillaries and smaller blood-vessels in the vicinity show hyalin degeneration.

The heart is usually flabby, the right ventricle or both dilated. The myocardium generally shows fatty or other degeneration. There may be also degeneration or necrosis of the endocardium, and thrombi may be found in the chambers.

The Lungs.—Bronchopneumonia is commonly found. There may be also marked congestion, edema, and atelectasis. Thrombi are sometimes found, and there may be gangrene of the lung. The bronchi are generally involved and may contain a membranous exudate. The bacilli are often found in greater numbers here than in any other region affected. Dilatation of the lymph-vessels is also common; they may be densely filled with lymph and plasma cells. The cervical glands are enlarged to some extent in almost all cases, but markedly in only about a third of the cases which involve the throat. The tissues about the glands are sometimes edematous, and the submaxillary gland may be swollen.

The *liver* and *spleen* show the usual changes arising from toxemia. Kidney changes varying from slight degeneration to intense acute nephritis are found in all fatal cases. The lesions are most pronounced in the rapidly fatal cases.

Nervous System.—It is in the nervous system that the most prominent results of toxemia are usually found, and more particularly in the nerve-trunks of the central system. The change begins in the myelin sheath at some point near the axis cylinder, and gradually extends around and along it. The lesions are essentially those of neuritis and may be found anywhere in the nervous system.

Symptoms.—*General.*—The period of incubation varies from two days to a week, but is seldom more than four days. The initial symptoms vary remarkably in severity. The onset is seldom marked by more than slight indisposition. Vomiting, rarely a convulsion, may occur in young children, and the prostration is often out of proportion to the other indications of illness. General muscular soreness and stiffness of the neck are occasionally observed. The symptoms are generally proportionate to the extent of surface invaded by the membrane, but the rule is not without many exceptions. In some cases the constitutional symptoms are intense without recognizable membrane-formation, and in some of the most extensively membranous cases the systemic disturbances are exceptionally slight. The patient is generally pale, the face

often has an ashen hue. If a child, it becomes languid or fretful and restless and it may complain of headache, loss of appetite, and nausea. It may later pass into a stupor or become delirious. In many cases, however, the mind remains clear throughout the disease, although severe. The tongue has a white coat and the breath is offensive. There is usually an urgent thirst. The constitutional symptoms are generally more pronounced in adults than in children, although the membrane-formation is usually less extensive. They become more prominent as the disease reaches its height, and are especially severe when necrosis and sloughing occur.

The *temperature* is not usually high. In the majority of uncomplicated cases it does not reach 102° F. (39.0° C.) at any time, very often it does not exceed 101° F. (38.5° C.) and the fever may subside in 48 to 72 hours. Instances of subnormal temperature are not infrequent. The pulse is accelerated out of proportion to the temperature and frequently reaches 150 or 160 in children. It may reach 180. In cases characterized by great cardiac weakness, however, it often becomes as slow as 40 or 50.

In some cases the disease begins with early elevation of temperature, marked nervous manifestations, and it may terminate fatally within a few days from the intensity of the toxemia. Dyspnea is present in many cases, particularly when the larynx is involved; it may, however, result from disintegration of the red blood-corpuscles by the toxins, from degenerative changes in the heart, or from a sudden spasm or paralysis of the vocal cords.

"The *heart* is probably affected in every case of diphtheria" (Jacobi). Either tachycardia or bradycardia is a feature of almost all severe cases. A systolic murmur is heard in about 10 per cent of cases, usually at the apex, and the pulmonic second sound is often accentuated. Since endocarditis is not common, this sound is probably due to relaxation. Cardiac dilatation frequently precedes a fatal issue.

The *nervous system* is profoundly affected by the toxins. This is shown, not only by the frequency of psychical depression, but also by the development of paralysis in about 10 per cent of cases. These are more fully considered under the head of Complications.

Moderate *albuminuria*, due to the irritation of the kidneys by the toxins, appears frequently on the second day of the disease, or later, in a majority of cases. Hyalin and granular casts are often present, usually appearing later as a result of the obstruction of respiration.

The cervical glands usually become enlarged, and the tissues about them are edematous. The voice becomes nasal.

The blood-count reveals moderate leucocytosis in all cases. The count of polynuclears and mononuclears is shown to be characteristic and important by Besredka. In all but the most fatal cases the former type of cells is much increased, while a decrease in the mononuclears is always observed. There is a deficiency of red cells amounting to from 500,000 to 2,000,000 in the c.mm. and the hemoglobin is reduced from 12 to 25 per cent.

The membrane usually remains attached for 5 or 6 days, and, gradually becoming detached around the edges, soon separates, leaving a denuded surface or superficial ulceration. The swelling and glandular en-

largement rapidly subside, but the convalescence is often slow and may be markedly interrupted by the most distressing complications.

In fatal cases the temperature may subside while the pulse becomes more rapid, irregular, and feeble or extremely slow, 50 or less. The action of the kidneys may fail; the urine becomes scant and highly albuminous, and uremia develops. Other cases terminate fatally on account of pulmonary edema, collapse, or bronchopneumonia. Sudden death from paralysis of the heart has been repeatedly observed, particularly as a result of excitement or exertion during convalescence.

Varieties of Diphtheria.—In addition to the symptoms just enumerated, others occur which depend, for the most part, upon the location of the disease.

Tonsillar or Pharyngeal Diphtheria.—The first complaint is usually of soreness or dryness of the throat upon swallowing or speaking. When the tonsils are first affected there is usually a slight rise of temperature, rarely exceeding 101° F. (38.5° C.). The tonsils at first appear slightly enlarged, intensely hyperemic, and on the surface of one or both there is one or more small patches of a grayish membrane. The uvula, the pillars, and the pharynx are usually congested. If an examination is made only a few hours later, the membrane may be found to have spread over the entire surface of the tonsils, and new patches may have developed on adjacent surfaces. Swallowing and speaking rapidly become more difficult and painful, and the symptoms of prostration more pronounced. The cervical glands are enlarged and sensitive, but less so than in tonsilitis. In other cases the exudate remains confined to the tonsils or spreads to but a limited area of the adjacent mucous membrane; little or no pain is complained of and the adenitis is slight.

Malignant cases not infrequently occur in which the extension of the membrane is extremely rapid and the constitutional disturbance most profound. Within the first 24 hours the entire surface of the tonsils, the sides of the pharynx, the uvula, and soft palate are covered with a heavy exudate, and the glands of the neck become enormously enlarged. The temperature may be but slightly elevated; it may even be subnormal, but the heart's action is rapid and feeble or slow and irregular. Stupor develops and the case terminates fatally within three or four days from toxemia. In other malignant cases the membrane, although not extensive in its invasion, has a foul, necrotic appearance, giving the breath a sweetish, fetid odor. The adjacent tissues for a considerable distance may be involved in the necrosis, and symptoms of sepsis often supervene. The tongue becomes dry, the temperature runs up to 104° or 105 °F. (40°—40.5° C.), the pulse is rapid and feeble, the extremities become cold. Death may result from exhaustion or from the supervention of bronchopneumonia.

Atypical cases are not infrequent, in which: (1) The mucous membranes of the throat are intensely hyperemic and edematous, but no pseudomembrane is formed. (2) The membrane may be punctate in form, remaining confined to small, isolated areas. In either of these forms the exudation may rapidly form or suddenly assume an active growth and spread with great rapidity into the nares or larynx. (3) The exudate may be soft and creamy or pultaceous in character.

Laryngeal Diphtheria.—When primarily affecting the larynx the dis-

ease begins with hoarseness and a harsh, croupy cough. By the second or third day there may be complete aphonia, stridulous respiration, a shrill, whistling cough, and the most alarming dyspnea, and cyanosis, with great restlessness. When the laryngeal involvement is a result of the extension of the disease from the pharynx, these symptoms are added to those already described. As the disease advances, the signs of stenosis become extreme. The accessory muscles of respiration are called into play. Rigidity of the sternomastoid muscle is an early indication of it (McCollom). The nostrils vibrate, the supraclavicular and intercostal spaces sink with each inspiration, cyanosis becomes extreme. The child sits up and gasps for breath until exhausted or overcome by the suffocation, then falls back, possibly to doze for a moment, but not to find relief. The detachment of a fragment of the membrane by coughing affords a short respite, but the membrane is soon replaced and the dyspnea returns. The constitutional symptoms soon become intensified. The temperature often rises to 103° F. (39.5° C.) and the action of the heart may suddenly cease. The patient may sink into a coma or die of exhaustion. Bronchopneumonia follows this type of the disease oftener than any other. Fortunately, under present methods of treatment these extreme cases are seldom encountered.

Nasal diphtheria may develop primarily or as an extension from the disease of the pharynx. It is much more frequent in children than in adults. It is usually characterized by mixed infection. The initial symptoms may be those of an acute nasal catarrh. The nostrils become obstructed and a thin, mucopurulent, sometimes sanguinolent, irritating fluid flows from them. Sneezing is caused by the irritation, and the lips become excoriated. Distinct enlargement of the glands at the angle of the jaw and of the submaxillary glands is developed early. In one group of cases the nostrils become completely filled with a thick membranous formation (fibrinous rhinitis), while in another class they are obstructed by the intense hyperemia and swelling, without an exudate. In the former class the constitutional disturbances are often slight and recovery occurs in the usual time, while in the other class the system may become charged with the toxins and the disease may assume a malignant character. The bacilli are usually numerous in the membrane or discharge, and great numbers of other micro-organisms are also found, particularly streptococci and staphylococci; sometimes the yellow sarcina, the bacilli subtilis and proteus are present. The inflammation frequently extends to the Eustachian tube and middle ear or to the antrum, occasionally through the lachrymal ducts to the conjunctivæ.

Diphtheria of Other Parts.—Primary diphtheria very rarely attacks the conjunctiva, producing a catarrhal or membranous inflammation. The globe is sometimes perforated in a single day. The presence of the Klebs-Löffler bacillus establishes the identity of the disease. This organism may alone be present, or the infection may be of a mixed character. The external auditory meatus is sometimes the seat of the disease, which is generally a secondary involvement from the middle ear.

Diphtheria of the skin is generally confined to the regions about the mouth, but it may be conveyed by the fingers of the child to more remote parts, particularly to the external genitalia and the anal region.

Wounds are occasionally infected, producing either a superficial inflammation, occasionally accompanied by necrosis, or the formation of a false membrane. Thec onstitutional symptoms of wound diphtheria are usually slight, but paralysis sometimes follows it.

Complications and Sequelæ.—Hemorrhage sometimes occurs as a result of ulceration in the nose or throat, especially in the nasal form of the disease. It is encountered also in malignant cases, probably as a result of profound blood changes, in the nature of toxemia.

Pneumonia.—Bronchitis frequently develops as early as the first or second day of the disease, especially in laryngeal cases. It may be delayed until after the beginning of convalescence. Pulmonary collapse or bronchopneumonia sometimes results from it. The diagnosis is often difficult, for all of the usual signs may be obscured by the great restriction of respiration, deficient expansion of the chest, and the loud noises produced in the larynx. An increase of temperature, accompanied by marked rapidity of breathing, usually indicates the condition. Lobar pneumonia is rare. An aspiration pneumonia, which may terminate in gangrene of the lung, may be induced in cases attended with extensive sloughing or a soft pultaceous membrane.

Heart-failure is one of the most dangerous complications. It is most likely to develop after the membrane has become detached, and the danger continues great from the third to the fifth week. Cases in which the appearance is most indicative of anemia are most liable to it. The asthenic condition of the heart may be recognized by the slowness, irregularity, and weakness of the pulse, as well as by cyanosis and occasional attacks of syncope. In some cases apparently progressing favorably such slight exertion as sitting up or vomiting or the excitement occasioned by the visit of a friend causes the heart to become extremely erratic in its action or to suddenly stop. Similar accidents may happen even during the first week. Hibbard found degenerative changes in the vagus in all fatal cases of heart-failure.

Paralyses (postdiphtheritic paralyses), due to toxic neuritis, occur in about 10 per cent of cases. They generally develop during the second or third week, but may occur as early as the seventh day or as late as the sixth week. They may be either local or multiple, unilateral or symmetrical. They follow the mildest attacks with apparently as much certainty as the most severe, but are more frequent in adults than in children. The symptoms produced in the multiple form are exceedingly variable in character, depending upon the nerves affected. One of the most common local paralyses is that of the uvula. This is attended with difficulty in swallowing, regurgitation of food through the nose, and a nasal tone of the voice. The palate is relaxed, and the uvula appears elongated and dependent. The constrictor muscles of the pharynx are sometimes involved in the paralysis. Either the extrinsic or the intrinsic muscles of the eye may be paralyzed, causing ptosis, strabismus, or a loss of the power of accommodation. Facial paralysis sometimes occurs and may prove to be persistent. In some of these cases a paraplegia or a paralysis of the arms is associated with that of the muscles of the eye or of the throat. The termination is usually in recovery, except when the heart or the muscles of respiration become involved. One or both of either the upper or lower extremities may

become partially or completely paralyzed, without the involvement of other parts. A solitary paralysis of the bladder has been recorded in one instance.

Nephritis.—Albuminuria is a constant feature of severe cases. The quantity of albumin present may be a mere trace and is probably due to the irritation of the kidneys by the toxins or to the fever. Actual nephritis is not a frequent complication. When present, it is indicated by a marked reduction in the quantity of urine voided, rarely by total suppression, a large percentage of albumin, and epithelial and blood casts. Edema is seldom a feature of diphtheritic nephritis. The prognosis is generally favorable.

Septic infection not infrequently occurs in the more malignant cases, as a result of extensive necrosis and sloughing of tissues. A septic infection of the joints and other parts is sometimes observed.

Cutaneous eruptions are sometimes observed. Erythema and urticaria are the more frequent types. Several cases of gangrenous stomatitis and vulvitis (noma) have been reported in which the diphtheria bacillus was found.

Diagnosis.—The discovery of the Klebs-Löffler bacillus is the only means of making an absolutely positive diagnosis of the disease. When this is found in a throat that is inflamed, whether or not a membranous formation is present, it indicates in almost every case that the individual is suffering from diphtheria, and even more positively that he may prove a source of infection to other persons. Diphtheria does not exist without the bacilli, but these may be overlooked for a time, and it should be borne in mind that the bacilli have been repeatedly found in the throats of healthy children and adults. There is no other disease in which bacteriological examination is so important. The health departments of all the larger cities and of many villages recognize this fact and provide for the examination free of charge of all specimens submitted. In most places an examination of the secretions from the throats of all suspicious cases, by an official bacteriologist, is required by law. The examination may be made directly from the throat, but it is ordinarily so difficult and requires so much experience and skill that it is customary to make it from a culture.

The throat appearances in diphtheria are more or less typical and are usually sufficient to establish a tentative diagnosis when accompanied by the usual symptoms, and more particularly when the disease is epidemic in the locality. The diphtheritic membrane is gray or yellowish gray in color. It does not rest lightly upon the surface, but has the appearance of having grown from the mucous membrane. It cannot be detached by ordinary rubbing with a swab, while the membranous formations produced by other bacteria, with the exception of Vincent's bacillus, are generally removed without much difficulty. (Forcible removal should not be attempted, on account of the danger of increasing the infection.) The extent to which the surfaces are invaded by the disease is also of value. As a rule, the more extensive its formation, particularly if it cover the entire surface of the tonsils and begin to invade other regions, the more positive is the diagnosis of diphtheria. The appearance of membrane in the nose is almost invariably due to this disease.

The hyperemia of the tissues surrounding a membrane-formation is seldom, if ever, so intense in any other condition as it is in diphtheria. An intense hyperemia of the throat without discoverable membrane should be regarded as diphtheritic until the presence or absence of the bacillus can be determined. The absence of such hyperemia strongly indicates the nondiphtheritic character of a membranous formation, but should not be too implicitly relied upon. Paralysis following an inflammatory condition of the throat, with or without membrane, is usually proof that the case was one of diphtheria, although cases have occurred in which the Klebs-Löffler bacillus had not been discovered.

The symptoms presented by the patient are also of value. In many cases the mild fever, rapid pulse, and great prostration, taken in connection with the throat symptoms, leave no justifiable doubt of the diagnosis. A history of exposure may be of great value in making an early diagnosis. It is very often expedient and sometimes imperative to make a positive diagnosis from the clinical manifestations, and not to delay the treatment until a bacteriological examination can be made.

Prognosis.—The physician is never justified in pronouncing an unqualifiedly favorable prognosis in this disease. The mortality has been reduced since the introduction of the serum treatment from over 40 per cent to less than 10 per cent, but epidemics differ greatly in severity, and there is no means of foretelling the course of an individual case. The disease is not always less severe in robust children, but convalescence is usually more rapid and more complete. The younger the child, as a rule, the more unfavorable is the outlook. The highest mortality is seen between the second and sixth years. Unusually extensive membrane-formation reduces the chances of recovery. Laryngeal diphtheria is always extremely dangerous. In the nasal form the cases accompanied by a sanguinolent discharge without membrane are the most unfavorable. Extreme adenitis is unfavorable. The presence of a mixed infection always increases the gravity of the case. Streptococci are particularly dangerous, by increasing the liability to sepsis. Absence of polynuclear leucocytosis was noted by Besredka only in fatal cases. Weakness and irregularity of the pulse are of evil import. A rapid pulse with low temperature is equally grave. The fatal termination of a case may be due to paralysis of the heart, pulmonary collapse, bronchopneumonia, general sepsis, or toxemia. The mortality independently of such complications depends almost as much on the promptness with which the serum treatment is instituted as upon the type of the disease. Malignant cases are now and then encountered, however, which run a rapidly fatal course under the most careful and skillful treatment. Serious, even fatal, accidents may occur as late as the fifth or sixth week. The danger of paralysis is not passed for at least two months, but paralysis usually ends in complete recovery.

Prophylaxis.—The patient should be isolated in a room from which all unnecessary furniture, carpets, and hangings have been removed. Thorough ventilation and a uniform temperature of about 70°F. (21° C.) should be maintained. The atmosphere should be kept moist by steam generated from a suitable vessel in the room. The vapor of turpentine, carbolic acid, or benzoin is thought to be beneficial. A special nurse should be in charge, and she should neither mingle with the other

members of the family nor permit them to enter the sick-chamber. The physician should exercise the same precautions in his visits as in other contagious diseases, wearing a gown and cap and disinfecting his hands and face after leaving the room. The child should expectorate into a vessel containing a strong disinfectant solution (corrosive sublimate 1:500) or upon patches of muslin that can be immediately burned. Infection from the secretions of the throat is possible for a month or longer in some cases, hence isolation should be maintained until bacteriological examination no longer shows the presence of bacilli. It is no less important to isolate mild cases and those in which the diagnosis is for the time uncertain. It is good practice to separate all cases of sore throat from the other members of the family, even when the case is not regarded as diphtheritic. The members of the family who have been exposed to the contagion should be kept at home, and, together with those who will come into contact with the case, may be still further protected by the administration of a prophylactic injection of from 200 to 1,000 units of antitoxin. This will protect for about two weeks, when it should be repeated. The nurse and attendants should gargle their throats several times a day with an antiseptic solution, preferably one containing 1:10,000 of corrosive sublimate. After the recovery of the patient the premises should be thoroughly disinfected, as after other infectious diseases.

Treatment.—*General Management.*—The diet of the patient should be liquid in character—milk, broths, soups, albumen-water, and ice-cream, with an abundance of cold water. Holt recommends that nursing infants be fed the milk obtained from the mother's breast by means of a breast-pump; they should not be put to the breast. Forced feeding by means of a soft tube passed through the mouth or nose must be practiced when, in the later stages of the disease, the patient refuses nourishment or when the muscles of deglutition have been paralyzed. Every measure for the support of the patient's strength should be adopted in the beginning. Brandy (from 1 to 6 ounces in 24 hours) and strychnin, gr. 1-100 to 1-40 (0.0006—0.0016), t. i. d. or oftener, should be administered as soon as asthenia becomes apparent. When the heart's action is rapid, and when it becomes weak or irregular, caffein or digitalis in small doses should be given in addition to the strychnin. The tincture of strophanthus (gtt. ij to v) probably acts more promptly than digitalis. When syncope develops, ammonium carbonate and camphor or musk are indicated. When heart-failure threatens, the patient should be kept absolutely quiet. It is safer in this condition to secure rest by the hypodermic administration of morphin every few hours, in sufficiently large doses to keep the patient drowsy. Paralysis of respiration may be so sudden in its onset that nothing can be done, but in a few instances several days of persistent treatment, consisting of the administration of strychnin in full doses for the age, at short intervals, the application of the galvanic and faradic currents, and artificial respiration when occasion required, have been rewarded with the recovery of the patient.

The internal administration of remedies for the purpose of antagonizing the disease is of doubtful utility. A. Jacobi and others highly recommend the administration of mercury, preferably the bichlorid in

small doses, gr. 1-60 (0.001), often repeated. It should be given in a small quantity of water. Some writers prefer calomel, gr. ⅛ (0.008), dropped upon the tongue without water. Pilocarpin is thought to hasten the elimination of the toxins, but is seldom employed. Large doses of the tincture of the chlorid of iron, quinin, and a host of other probably useless remedies are employed by many physicians. Emetics are sometimes given to strong, vigorous children to assist in the removal of the membrane, particularly in laryngeal cases, when a flapping sound indicates that it has become partially detached, or when suffocation threatens. Antipyretics are seldom required, and those of the coal-tar group should not, as a rule, be employed. The temperature may be more safely reduced by cold sponging.

The complications are treated in the same manner as if they were independent affections in a debilitated patient; special methods are therefore to be adopted for bronchitis, pneumonia, nephritis, otitis, and other affections.

Local Treatment.—Local measures are less relied upon than they were before the introduction of antitoxin; possibly they are too much neglected. They should be employed with a view to cleanliness and the prevention of such complications as aspiration pneumonia or involvement of the larynx, rather than with a view to overcoming the disease. In very young or nervous children, when it is apparent that more worry and exhaustion is produced by persistence in local applications than is compensated for by any possible benefit, it is often wise to omit all local applications.

Numerous antiseptic solutions have been recommended for use by means of a cotton swab, spray, or irrigation. The last method is usually preferred in nasal diphtheria. For swabbing, the solutions most used are: Mercuric chlorid (1:1000), hydrogen peroxid (1:5), and Löffler's solution (menthol, 10 grams, dissolved in sufficient toluol to make 36 c.c.; liquor ferri sesquichloratis, 4 c.c.; and absolute alcohol, 60 c.c.). For irrigation the solutions should be much diluted, or less irritating solutions, as Seiler's alkaline antiseptic solution or boric acid, may be employed in large quantity. Nasal hemorrhage sometimes prevents irrigation for a time, or astringent solutions of alum or of the chlorid or subsulphate of iron (Monsel's solution) may be employed. It is claimed for aqueous solutions of the various preparations of papaya and trypsin that they dissolve the diphtheritic membrane. A solution of lactic acid is still employed by some physicians, although it probably has no specific action.

Antitoxin Treatment.—The diphtheria antitoxin should be administered at the earliest possible moment, for the results are better in proportion to the promptness with which the treatment is instituted. Statistics show that the mortality is three times as great when the treatment is begun on the third day, and five times as great when it is begun on the fifth day, as when on the first. It should be given in all forms of the disease, and in sufficient quantity to subdue the action of the toxin. It is only in the mildest cases that the physician is justified in waiting for the result of the bacteriological examination. If the case appears at all severe, the injection should be given at once, for it is better to err on the safe side, and no harm can be done if the disease

proves not to be diphtheria. Unfortunately, a time is reached in every severe case after which the use of the serum is of comparatively little benefit. This time limit may be three or four days, or it may be only six or eight hours, and we have no means of determining which it will be in any case. The dose of antitoxin is measured in units. Each unit represents the quantity required to counteract ten times the minimum dose of diphtheria toxin necessary to kill a guinea-pig weighing 250 grams. It is not possible to determine the quantity of toxin that is to be antagonized in a case of diphtheria affecting a human being, hence the quantity to be administered must be determined by the effect that is produced. The syringe should be large enough to hold the entire quantity for one injection, and it should be so constructed that it can be thoroughly sterilized by boiling in a 5 per cent solution of carbolic acid before each injection. The serum is usually injected into the cellular tissue of the loin or gluteal region. From 1,500 to 2,000 units are generally given as the first dose in an ordinary case, except in an infant, when from 500 to 1,000 units may be sufficient. If the case is one of unusual severity, or if it be accompanied by signs of laryngeal stenosis, from 3,000 to 5,000 should be given at the outset. The beneficial effects—an amelioration of all the symptoms, including a reduction of temperature and pulse-rate—are usually apparent within a few hours. If after ten or twelve hours there is no distinct improvement, or if the membrane is found to have invaded new areas, the injection should be repeated, and as many subsequent doses may be given as the case demands. It is seldom that more than three injections are required in a case in which the treatment has been instituted early.

The serum treatment is never more imperatively demanded at the earliest moment than in laryngeal diphtheria. Here it should be employed at the first evidence of inspiratory or expiratory obstruction, even when no membrane can be seen.

The only outward effects of the antitoxin treatment are: an immediate rise of the temperature in a few cases, a cutaneous eruption, or the formation of an abscess at the point of injection. In from 5 to 20 per cent of cases there is an eruption of urticaria affecting the skin immediately around the needle wound, or rarely spreading more or less generally over the entire body. This disappears within a few days. Sometimes, when the eruption is general, it resembles that of scarlatina or measles. It may appear as early as the second day or as late as the fifteenth; it may be accompanied with fever and pain in the joints for two or three days, and is often followed by a profuse desquamation.

The antitoxin treatment is of benefit only in true diphtheria. Its action is limited to the neutralizing or antagonizing of the toxin produced by the Klebs-Löffler bacillus, and it is absolutely useless in streptococcous or other forms of infection. If after the first injection the bacteriological examination proves to be negative, the dose should not ordinarily be repeated. But in a severe case, and particularly if benefit has followed the first dose, it is better to repeat the dose on the supposition that some unavoidable accident has prevented the discovery of the bacillus, a supposition that not infrequently proves correct.

Treatment of Laryngeal Diphtheria.—In addition to the methods that have been described, some writers advocate the inhalation of the fumes

of subliming calomel, produced by dropping about 20 grains of the drug on hot coals if a special apparatus is not at hand. Inhalations of steam, alone or with the addition of such volatile substances as benzoin, turpentine, carbolic acid, or eucalyptol, are undoubtedly beneficial. They are administered by erecting an improvised tent, for which a sheet or blanket will answer, over the child. Steam may be generated by slowly pouring water on a heated brick or iron at the side of the tent. The calomel fumes are not thought to exert any action upon the bacillus, but only to favor the detachment of the membrane, and should not be used too freely.

Intubation.—If the methods that have been described do not afford relief, intubation of the larynx may become necessary. It should not be too long delayed, but, on the other hand, it should not be too readily resorted to, for the introduction of the tube in no way limits the extension of the disease, and if in any manner an abrasion is produced by the introduction of the tube, an additional point for infection is established. As a rule, frequent or persistent attacks of cyanosis indicate the necessity for a resort to intubation. The intubation outfit consists of a set of gold-plated or hard-rubber tubes of different sizes, a holder for their insertion, and an extractor. The mouth of the child is held open by a small gag and the tube, grasped with the holder, is guided back over the epiglottis by the finger of the operator and gently passed into the larynx. The instrument may sometimes be worn for several days. If no accident happen, it should be allowed to remain for five or seven days from the beginning of the stenosis; but if it becomes obstructed, it must be immediately withdrawn, cleansed and replaced with as little delay as possible. The tube is sometimes coughed out of place and may pass into the esophagus. It must then be withdrawn by means of a thread which is always attached to it. Some children are able to take fluid, or, better, semi-fluid nourishment, while the tube is in place. To attempt this, they should be held face downward, or with the body inverted. Occasionally a child can drink in the ordinary way, but it is generally safer to use a tube. This failing, the food must be introduced through a small, soft stomach-tube passed through the mouth or nose. McCollom objects to nasal feeding, on the ground that it favors infection of the middle ear. The atmosphere of the room should be heavily impregnated with moisture while the child wears the tube.

Tracheotomy is now seldom resorted to until intubation has failed to afford relief, or until the membrane has extended into the trachea and relief is no longer to be expected from intubation. In country practice, where the physician cannot respond promptly in the event of an obstructed tube, the operation may be deemed a safer procedure. Unfortunately, the benefit to be hoped for from tracheotomy in many of these cases is but temporary, on account of the extreme growth of membrane or the development of such complications as edema of the lungs or bronchopneumonia.

DIPHTHEROID.

PSEUDODIPHTHERIA, MEMBRANOUS CROUP, STREPTOCOCCOUS DIPHTHERIA.

Definition.—An acute infection, or group of infections, closely resembling diphtheria in local and general symptomatology, but distinguished from it by the absence of the Klebs-Löffler bacillus.

Etiology.—The affection is generally caused by the streptococcus pyogenes alone or in association with other bacteria. The pseudo-diphtheria bacillus may be present, but it is not of known etiological importance. One of the most important, perhaps, is the bacillus of sputum septicemia. The affection most frequently occurs as a complication of the acute infectious diseases, notably scarlet fever, measles, erysipelas, typhoid fever, or whooping-cough, but it is occasionally primary. It is sometimes incited by the inhalation of hot steam, the fumes of ammonia, arsenic, corrosive sublimate, or other irritating substances.

Morbid Anatomy.—A membrane is formed which is identical with that of diphtheria, except that the specific bacillus is absent. The exudate is most frequently found in the larynx, but it is sometimes confined to the tonsil, or it may affect all the surfaces usually involved in diphtheria. The membrane is, however, more loosely attached to the mucous membrane. In some cases, too, just as in diphtheria, membrane-formation is wanting, and there is only an intensely hyperemic surface; and in other cases, again, there is a soft, pultaceous formation. General streptococcous infection of a severe type sometimes follows the local phenomena.

The ***symptoms*** are those of diphtheria, but they are generally less severe in character. Severe attacks are occasionally met with, however, and death has repeatedly occurred in cases regarded as of this character. The affection usually lasts about a week. Albuminuria is seldom present, and other complications are infrequent.

The ***treatment*** is that of diphtheria affecting the same region, but without antitoxin. In laryngeal cases inhalations of steam are of the greatest value. Intubation may become necessary in severe cases. The antitoxin of diphtheria is entirely without beneficial influence, but should be employed in a severe case if there is the possibility of an error in diagnosis.

WHOOPING-COUGH.

PERTUSSIS, TUSSIS CONVULSIVA.

Definition.—An acute infection characterized by paroxysms of a convulsive cough followed by a long-drawn sonorous inspiration or whoop.

Etiology.—The disease is usually conveyed by contagion. It may be contracted by brief contact with a patient. It is seldom conveyed by a third person, but clothing and houses apparently become infected. The poison is much less virulent than that of measles or scarlet fever. The sputum and probably the breath convey the contagion. Bacteria have been found in the sputum by different investigators. Koplik found a short, slender, facultative, anaërobic, motile bacillus, probably first described by Afanassieff, in 13 of 16 cases, and Czaplewski and Hensel found one which they regarded as the same in all of 44 cases examined. Absolute immunity is usually conveyed by an attack; natural immunity is rare. Sporadic cases occur at all seasons, but epidemics are more frequent in winter and spring. They frequently precede an outbreak of measles, less frequently that of scarlatina. Epidemics last two or

three months. About 50 per cent of the cases occur during the first two years of life. It sometimes attacks infants of only a few weeks, or adults up to advanced age. Girls are slightly more susceptible than boys; and weak children, particularly those affected with catarrh, more readily contract the disease.

Morbid Anatomy.—The morbid changes are rather those of the complications, of which pulmonary collapse and bronchopneumonia are the most frequently found in fatal cases. Enlargement of the tracheal and bronchial glands is constant.

Symptoms.—The course of the disease is generally divided into a catarrhal and a paroxysmal stage. The incubation varies from a few days to two weeks. The catarrhal stage lasts from one to two weeks and is characterized by slight indisposition, fever at night, and evidences of laryngeal or bronchial catarrh, as in an ordinary cold. The cough is usually hoarse and becomes peculiarly sonorous, and finally paroxysmal. The face becomes swollen and the lower eyelids puffy.

The paroxysmal stage begins with the first appearance of the whoop. The cough becomes distinctly spasmodic. It is of a rapid staccato character and ceases only after the air in the lungs has been exhausted; and it is followed by a long audible inspiration, the air being drawn through the glottis while the vocal cords are approximated. Several paroxysms frequently occur in succession until a mass of tenacious mucus is expelled. From four or five to eighty spells occur in a day; they are often more frequent at night. They are brought on by any slight irritation of the throat, as by the inhalation of dust or cold air; and the child soon learns to refrain from laughing or talking, even from eating, particularly when it feels the aura-like inclination to cough which generally precedes the attack. As the spell comes on, the child generally runs to its mother or seizes any near object for support. During a severe paroxysm the face becomes cyanotic, the thorax is contracted, the eyeballs protrude, the conjunctivæ become injected, often remaining blood-shot, and hemorrhages frequently occur from the mouth and nose. Hemoptysis has been observed. Convulsions may occur in nervous children. Vomiting very generally follows a paroxysm, particularly just after a meal. The sphincters sometimes give way. A small ulcer generally forms under the tongue during the disease.

Examination of the chest during an attack reveals dullness from deficiency of air in the lungs; during the intervals, the signs are those of emphysema and bronchitis, mucous râles and absence of the vesicular murmur. The paroxysms begin to diminish in frequency and severity in three or four weeks, but the cough continues to be spasmodic in character until the last. The contraction of a cold renews the peculiar cough for months after recovery.

Complications and Sequelæ.—The repeated vomiting and subsequent inanition induce anemia. Hemiplegia has been induced by a severe paroxysm, and sudden death has occurred from subdural hemorrhage. The pulmonary complications are the most frequent and most important. The bronchitis, enlargement of the bronchial glands, and emphysema may persist. Bronchopneumonia sometimes proves a fatal complication, or tuberculosis may be engrafted upon it. Pleurisy is frequent, lobar pneumonia infrequent. Pericarditis and lesions of the valves have been

attributed to the great strain thrown upon the heart during the paroxysms. Hernia has been produced by the violent coughing. Nephritis sometimes occurs. Anders found it in 20 per cent of cases, but Blumenthal found only increase of the uric acid, with high specific gravity. Leucocytosis develops early.

Diagnosis.—The whoop is pathognomonic, but in young infants it is often absent. A dry cough with an occasional short whoop sometimes occurs in other catarrhal affections of the nose and throat.

Prognosis.—The frequency and severity of the complications always warrant the giving of a guarded prognosis. The bronchopneumonia is almost always fatal. The younger the child, the greater is the danger. The mortality is very high among the children of the poor, probably owing to neglect. Cases among all classes of people are too often allowed to run their course without treatment, owing to the popular fallacy that nothing can be done for the disease.

Treatment.—Isolation should always be insisted upon, and in severe cases the patient should be confined to bed in a well-ventilated room. Milder cases, in warm weather, will do better in the open air. The diet should be light, easily digested, consisting largely of milk, and it would best be given in small quantities at short intervals on account of the vomiting. Removal of the patient from the city to the country is immediately beneficial in most cases.

Local Treatment.—Various methods of local treatment have been recommended. These are applied by insufflation, with a brush, in the form of spray, or by inhalation. Quinin with bicarbonate of soda and powdered gum acacia, or a mixture of quinin and resorcin, has been extensively employed by insufflation three times a day. Boric acid, benzoin, salicylic acid, iodoform, and other powders have also been used. For application with the brush or swab, a 2 per cent solution of resorcin or carbolic acid has been recommended. In the form of spray the same solutions may be used, and equal parts of hydrogen peroxid and glycerin are highly recommended. For inhalation, the vapor of creosot, naphthalin, or bromoform and ozone has been recommended.

Internal Medication.—Quinin and belladonna are most relied upon. The former is given in doses of 1 to 2 grains for each year of age, up to 5, three times daily, and the belladonna should be pushed until the face flushes after each dose. They may be given together. Bromoform in 1 to 5 minim doses has been highly recommended, but should be used with caution, commencing with the minimum dose, owing to its tendency to depress the heart.

MUMPS.

EPIDEMIC PAROTITIS.

Definition.—An acute infectious disease whose chief symptom is inflammation of the parotid gland.

Etiology.—The specific cause of infection is not known. The disease prevails sporadically, endemically, and sometimes epidemically. It is highly infectious to persons coming into close contact with those affected, and the contagium may be carried on clothing. It frequently attacks

more than 90 per cent of the inmates of schools and barracks. It is most prevalent during springtime and autumn. Childhood and youth are the ages of greatest susceptibility. Infants under 2 years and adults over 25 are seldom attacked. Boys are more frequently affected than girls. One attack, including possible relapse, generally produces permanent immunity.

Morbid Anatomy.—A serous exudation into the glandular and periglandular tissues of the parotid, and catarrhal inflammation of the ducts, constitute the usual lesions of the disease. Suppuration rarely occurs. The affection is limited to one gland in some instances. The submaxillary glands may be affected, alone or in conjunction with the parotids. In boys the disease is not infrequently accompanied by inflammation of the testes, and in girls by inflammation of the ovaries, vulva, or mammary glands.

Symptoms.—The incubation is about 14 days, without symptoms. The invasion is usually announced by a slight elevation of temperature, seldom beyond 101° F. (38.3° C.). Nausea, vomiting, headache, and restlessness may be present. In about 24 hours after the onset, slight pain and a sense of fullness in the region of the parotid gland are experienced. Swelling is soon noticeable and by the third day forms a prominent protrusion which interferes with speech and deglutition by restricting the opening of the mouth. Slight deafness, tinnitus, earache, pharyngitis, and epistaxis occasionally add to the discomfort. After a day or two the other parotid gland usually becomes affected; it is seldom that both are affected in the beginning. When the submaxillary glands alone are involved, the swelling is confined to the region beneath the chin, but may extend far down the neck. The secretion of saliva may be either increased or diminished. The more severe symptoms generally abate in two or three days, and the swelling slowly subsides after a week or ten days, but one or more relapses not infrequently occur. In the most severe cases the temperature runs high, 103° or 104° F. (39.5°–40° C.), and nervous symptoms develop, notably delirium or stupor, occasionally mania; rarely the patient passes into a typhoid state.

Orchitis is not usually seen before puberty. It may occur during the height of the disease, seldom earlier, but more frequently develops during convalescence and is accompanied by a renewal of the former constitutional disturbances. It is generally unilateral, but it may involve both testes simultaneously or in succession, and sometimes lasts longer than the original parotitis. Effusion of serum into the tunica vaginalis, edema of the scrotum, and slight urethral discharge often accompany the condition. The epididymis is not usually involved. Atrophy of one or both glands may result. Vulvovaginitis sometimes occurs in girls; ovaritis is rare. The mammary glands are occasionally involved in boys, though not so frequently as in girls.

The ***complications*** and ***sequelæ*** are few. The most important are those on the part of the nervous system—meningitis, peripheral neuritis, and paralyses (hemiplegia and facial paralysis), or edema of the brain from compression of the jugular vein. Acute mania and insanity have followed the disease. Stomatitis, laryngitis, and otitis media are occasionally encountered, and deafness and atrophy of the optic nerve have been attributed to the disease.

Diagnosis.—The peculiar character of the swelling, free from redness of the skin or special tenderness, and passing around the lobule of the ear, with the more or less marked constitutional disturbance, serves, as a rule, to distinguish parotitis from other inflammatory affections of this region. Idiopathic parotitis is rare in childhood.

Treatment.—The patient should be isolated and confined to bed during the acute stage. Hot applications, preferably a wad of absorbent cotton wrung out of hot water and covered with oil silk, soothe the pain and probably reduce the inflammation. Cold applications are preferred by some patients. Medicine is seldom required except for the relief of nervous manifestations. Two to 5 grain (0.15—0.30) doses of phenacetin, according to age, relieve the headache and reduce the fever. The bowels should be kept open with saline laxatives. If orchitis develop, the testicles should be elevated and treated with hot or cold applications. Thompson recommends the application of equal parts of guaiacol and glycerin or 30 per cent ichthyol in lanolin.

SEPTICEMIA.

SEPSIS, BACTEREMIA, BLOOD-POISONING.

Definition.—A general disease, caused by the entrance of pyogenic micro-organisms or their toxins into the blood, and characterized by chills, irregular fever, sweating, and great prostration.

Etiology.—The phenomena of septicemia are commonly preceded by suppuration, but the location of the suppuration is not always recognized before the development of the septic condition. The pyogenic micro-organisms become localized at one or more points within the body or on an abraded cutaneous or mucous surface, and there set up a suppurative or putrefactive process. From this the bacteria or their toxins, or both bacteria and toxins, gain entrance to the general system through the blood or lymph circulation or through both these channels. It is an essential feature of septicemia that no secondary foci of suppuration are developed. In this regard it is distinguished from pyemia. Any disease or condition attended by suppuration may become a cause of septicemia. Among these are abscesses of the breast, lymph-glands, liver or other organs, empyema, suppurative peritonitis, chronic otitis, malignant endocarditis, and pyelitis. Sepsis is a common termination of chronic tuberculosis.

Bacteriology.—The bacteria most frequently causing the disease are: (1) The Streptococcus pyogenes, (2) Staphylococcus pyogenes aureus, (3) Gonococcus, (4) Micrococcus lanceolatus, (5) Bacillus pyocyaneus, (6) Bacillus proteus, (7) Bacillus influenzæ, (8) Bacillus typhosus, (9) Bacillus coli communis, and (10) the Bacillus aerogenes capsulatus. Of these the most important are the first and second. Klebs and Koch attribute the disease to a specific microbe which is smaller than the pus-forming organisms.

The term septicemia is sometimes applied to conditions in which the blood becomes highly charged with the toxins of other bacteria, but toxemia is a better designation for these. Conditions closely resembling septicemia have been induce by Bergmann, Angerer, and others through the injecting into the blood of pepsin, pancreatin, and trypsin.

Infection may occur through an incised wound, as in surgical septicemia, or through accidental wounds or abrasions. In puerperal sepsis it is taken up from the uterine canal. The serous membranes, especially the pleura and peritoneum, or the mucous membranes of the respiratory or alimentary canal, not infrequently admit the poison, and suppuration of bone is often the source of infection. Suppurating lymph-glands in any part of the body may act as hidden foci of infection in the so-called cryptogenic form of the disease.

Morbid Anatomy.—In sapremia and fermentation fever there are usually no lesions. In true septicemia death not infrequently occurs before recognizable lesions have been produced. In cases that have run a less rapid course the most striking feature is a condition in which the body undergoes rapid putrefaction. The blood is black and tar-like and the muscles are dark. Hyperemia and ecchymoses are often found in the pia, pleura, pericardium, and peritoneum, and punctiform hemorrhages may be found in the skin. The spleen and lymph-glands are usually enlarged, and the spleen may be soft. The liver and kidneys show cloudy swelling and sometimes other degenerations. Bacteria may be found in great numbers in the various tissues, especially in inflammatory foci, or exudations, and in the renal glomeruli. It has been suggested that the presence of numerous bacteria in the blood and tissues after death may be in part due to an agonal or post-mortem invasion or to the rapid growth after death of a few organisms present during life.

Varieties of Septicemia.—1. *True or Progressive Septicemia.*—Senn restricts the use of this term to cases which are caused by the entrance into the circulation of microbes from some local septic focus. It is caused not only by poisons which are produced at the primary seat of infection, but also by those produced in the blood by the bacteria which it contains. 2. *Sapremia* is the septic intoxication caused by the entrance into the blood of toxins or ptomains previously formed by putrefactive bacteria in dead tissues. It is not accompanied by the entrance of bacteria, and usually subsides as soon as the primary cause has been removed. 3. *Intestinal or Ptomain Poisoning.*—This form was first described by Vaughan as due to the absorption of tyrotoxicon, a poisonous chemical substance often found in cheese. The same investigator and others have since added to the list a large number of other poisonous ptomains. Strictly classified, poisoning by these substances is a form of sapremia. The term is sometimes employed, however, to designate conditions in which the bacteria have found their way into the circulation through the intestinal canal, particularly with reference to the bacillus coli communis.

Fermentation Fever.—Closely allied to these conditions is that known as fermentation, aseptic, or resorption fever. It is a general febrile disturbance caused by the absorption of the products of aseptic tissue necrosis. It appears as a temporary condition soon after injuries and operations in which bacterial invasion has not occurred, but more particularly when there has been extravasation of blood. It is supposed, in some cases at least, to be due to the entrance into the circulation of fibrin ferment. It resembles sepsis only in the febrile character of its symptoms.

Symptoms.—(1) In *septicemia*, the period of incubation lasts from a few hours to several days. The onset is usually gradual and is not announced by a chill. There is generally a distinct rise of temperature varying greatly in degree in different cases. Headache, nausea, vomiting, prostration, and mental dullness are generally present. Diarrhea may be an early symptom, but constipation is more common in the beginning. The fever may reach only 100° F. (38.0° C.) in mild cases, but may exceed 110° F. (43.5° C.) in the worst. It is usually a continuous fever, but the daily fluctuations may amount to 3° or 4°F. The pulse is rapid, often 130 or over, usually small and tense; sometimes it is soft and feeble. Leucocytosis is usually present. Cabot remarks in this connection that leucocytosis indicates a struggle between the system and the infection, and that it may be absent in the mildest and severest cases. The respiration is rapid and superficial. Cyanosis is sometimes present. The skin, at first hot and dry, becomes bathed with a profuse perspiration. In mild cases all symptoms frequently subside within from 24 to 72 hours; and in the severe, or fulminant, cases death may occur within the same limit of time. Other cases run a variable course of from four or five days to as many weeks, and chronic cases last for months. The symptoms in the chronic form are often severe, but run a remittent or intermittent course. The exacerbations are frequently accompanied by a rigor, and chilly sensations or distinct chills often occur independently of remissions or exacerbations. Diarrhea may develop and the patient become anemic and emaciated. Erythema, petechiæ, punctate hemorrhages or ecchymoses, and other cutaneous eruptions not infrequently develop.

(2) The symptoms of *sapremia* differ much in different cases. At times they are quite like those of septicemia, but are usually of shorter duration. Severe cases are initiated by a chill and continuous fever, reaching from 102° to 104° F. (39°—40° C.), with slight morning remissions. A soft, full, compressible pulse is quite characteristic. Anorexia, vomiting, and diarrhea are frequently present, particularly in the intestinal form. The tongue is furred, becomes dry, assuming a "dried-beef" appearance in severe cases. The urine is scant, rich in urates, and increased in toxicity. All symptoms quickly subside upon removal of the cause.

(3) The symptoms of *fermentation fever:* The temperature usually rises rapidly without a chill, reaching a maximum of from 100° to 104° F. (38°—40° C.). It then remains almost stationary for from one to three days and drops suddenly to normal. The pulse undergoes a corresponding acceleration. There is usually little or no disturbance of the nervous system, the sensorium remaining clear or even appearing stimulated to greater activity.

Diagnosis.—This depends, in many cases, upon the recognition of the source of infection. The frequent chills, irregular temperature, and sweating, with rapid production of anemia and emaciation, should always arouse suspicion of sepsis in an obscure case. Secondary abscesses belong to pyemia, not to septicemia. The appearance of leucocytosis is often a valuable feature in diagnosis.

Typhoid Fever.—This disease is usually recognized by the more gradual elevation and more uniform course of the fever, the absence of chills

during its course, the rose-spots and the absence of leucocytosis. But the disease is not infrequently complicated by septicemia or sapremia, causing a modification of its symptoms.

Malaria, especially of the estivo-autumnal or remittent type, resembles septicemia, but the course is generally more uniform; the greater splenic enlargement, the absence of suppurative foci, and, above all, the presence of the plasmodium in the blood establish the diagnosis.

Chronic Tuberculosis.—In this disease many of the symptoms are due to sepsis, hence the differentiation must often depend upon the discovery of pulmonary or other lesions and the isolation of the tubercle bacillus. In acute miliary tuberculosis, the cough, rapid respiration, and the discovery of the bacillus in most cases serve to differentiate the affection.

Prognosis.—In mild cases of sepsis, in most cases of sapremia, and in all cases of fermentation fever the prognosis is good. In acute septicemia it is always grave. In all septic cases, however, as much depends upon the physical condition of the patient as upon the virulence of the infection. There is much difference in individual power of resistance. Rapidly fatal cases sometimes follow the most trivial surgical, dissection, or post-mortem wounds; while cases arising from the most extensive suppuration often recover after months of severe illness. The early removal of the cause, when this is possible, has a decidedly favorable influence on the prognosis.

Prophylaxis consists in the strict observance of antiseptic precautions, the removal of extravasated blood, and the prompt evacuation of pus cavities. Attention to the condition of the hands, especially with reference to slight abrasions, before undertaking surgical or post-mortem work, is of the utmost importance. When an injury is received during an operation, bleeding should be encouraged or increased by sucking, and the wound should be immediately cauterized.

Treatment.—The cause should be promptly removed. In fermentation fever, the establishment of drainage or antiseptic irrigation is usually all that is required. In sapremia of intestinal origin, prompt purgation by calomel, castor oil, or a saline cathartic is often sufficient. Intestinal antiseptics, salol, or β-naphthol in 5-grain doses may be employed. In septicemia the treatment is largely symptomatic. The temperature may be reduced by frequent sponging or cold bathing. Antipyretics should be avoided on account of their depressing effect. The strength of patient must be supported by a nutritious, easily digested diet, consisting of milk, broths, eggs, egg-nog, and gruels. Codliver oil and malt preparations are beneficial in chronic cases. The heart's action should be supported by strychnin, gr. 1-30 (0.002), and alcohol. The action of the kidneys should be favored by the administration of large quantities of water and potassium acetate, gr. x (0.65), or liquor ammoniæ acetatis, ʒ ss (2.0). Quinin in large doses, gr. x to xx (0.65—1.3), proves of great benefit in some cases, but is useless in others. Diarrhea, when excessive, must be controlled by opiates. Marmorek's antistreptococcous serum may be injected, but it is not always of benefit. Inunctions of unguentum Credé, a preparation of metallic silver, have been recommended, but are often disappointing. About ʒ i (4.0) daily is rubbed into the sides of the thighs, abdomen, and other regions in succession

as in the administration of mercurial inunctions. In many cases surgical measures must be resorted to. Wernitz has strongly recommended slow, protracted irrigation of the intestine with a 0.5 to 1 per cent saline solution under low pressure. The intravenous injection of formalin solution (1:5000) has also been resorted to, with success in a few cases. From 500 to 750 c.c. of the solution were introduced at each injection. The measure is not free from danger.

PYEMIA.

SEPTICOPYEMIA.

Definition.—An acute febrile disease caused by the entrance of pus-forming micro-organisms into the blood, and characterized by high fever, frequent chills, and sweating, with the formation of metastatic abscesses, phlebitis, infarcts, and hemorrhages in various parts of the body. It is pathologically identical with septicemia, except in the production of secondary suppurative processes.

Etiology.—Streptococci and staphylococci are the most frequent causes of the infection, but the other bacteria named under the Etiology of Septicemia may be operative in its production, and the same suppurative conditions may be the sources of infection. The avenue of entrance is usually through the wall of a vein which is in a state of inflammation and degeneration. Entrance through the lymph-vessels is possible, but the germs must reach the general circulation before pyemia can develop.

Pathology.—There is first a point of suppuration adjacent to or involving a vein, rarely an artery. A suppurative phlebitis (or arteritis) is thus induced. Thrombi are formed, and the pyogenic bacteria find their way into the clot, soften and disintegrate it into numerous fragments which are carried off in the circulation as emboli. When a vein is involved, the septic emboli are generally arrested in the lungs, and the abscesses may be confined to them. If, however, a radicle of the portal vein is involved, the emboli are distributed to the liver. From a malignant endocarditis the pus is distributed to all parts of the body.

The lesions found after death depend to a great extent upon the duration of the disease. If death have occurred early, the original phlebitis and the suppurative infarctions may be found. At a little later period, numerous miliary abscesses are often discovered. In more protracted cases there may be abscesses from one to four inches in diameter, especially in the liver, lungs, spleen, or kidneys, sometimes in the brain, joints, or serous cavities. Subcutaneous abscesses may occur, especially in the vicinity of the joints. The heart muscles are usually soft, the spleen is enlarged, soft, and dark, and ecchymoses are often revealed upon the surface of the pleura or pericardium, or hemorrhages in the subcutaneous tissue.

Symptoms.—Wound septicemia is always a sequel to suppuration, hence it seldom develops earlier than the seventh to the fourteenth day after the receipt of the injury. The infection may be preceded by changes in the wound. The granulations become pale, and the pus thin and ichorous or saneous and scant. The edges of the wound become

puffy and edematous. A thrombosis in one or more veins leading from the focus of suppuration may be recognizable on close examination. Mild symptoms of intoxication may precede those of the infection, notably a slight rise of temperature and mental depression. The pyemic invasion is announced by a severe chill, and the temperature rises to 104°, 105° F. (40°—40.5° C.) or higher before the shivering ceases. Headache, vomiting, and extreme prostration are usual accompaniments.

The pulse becomes rapid, often 140 to 160, and feeble. Chills occur at short intervals, daily or every other day, or there may be two or three chills a day. They often increase in frequency and severity as the disease progresses. The temperature range in acute cases is irregular, intermittent or remittent, with fluctuations amounting to several degrees in some cases, but seldom declining to the normal. In severe cases intermissions occur in which the temperature becomes normal or subnormal. Profuse sweating, anemia, and rapid emaciation are characteristic of the disease. The tongue becomes dry, the breath has a sweetish odor, and sordes often form on the teeth. The skin becomes pale or of a dusky color, the features pinched, the expression anxious. Slight jaundice is occasionally seen. Nervous symptoms are usually absent until late in the disease. Delirium develops in severe cases and deepens into coma as the fatal issue approaches. If, however, metastatic meningitis develops, it is promptly announced by strabismus, ptosis, deafness, and hemiplegia. Other symptoms, particularly pain, tenderness, and swelling, occur as a result of thrombosis, infarction, or abscess formations in different parts of the body. When these form in the lungs, rapid respiration, cough, and dyspnea are produced, sometimes with purulent or bloody expectoration. Malignant endocarditis is attended with increased pulse-rate and temperature, dyspnea, and a harsh systolic bruit. Leucocytosis, reduction of erythrocytes, and moderate poikilocytosis are the usual blood-changes. Cutaneous eruptions, erythema, purpura, or pustules may occur, and hyperesthesia is commonly present. The urine is febrile in character, often contains albumin, casts, and sometimes pus and blood-corpuscles. Albumose has been found in it. Suppurative inflammation of the joints is not uncommon late in the disease, sometimes after the fever has subsided.

Diagnosis.—Pyemia may be distinguished from septicemia, as a rule, by the extreme fluctuation of the temperature, the frequency of chills, and yet more particularly by the thromboses, infarctions, and metastatic abscesses. In other respects the symptoms are practically the same. The features thus combined serve to differentiate pyemia from almost all other affections.

Treatment.—This is in all respects the same as that of septicemia.

ERYSIPELAS.

Definition.—An acute febrile disease caused by the streptococcus of Fehleisen, affecting primarily the skin or a mucous membrane and producing general symptoms of toxemia resembling those of sepsis.

Etiology.—The streptococcus described by Fehleisen is regarded as the specific cause. Attempts to produce the disease by inoculating with other cocci have not been entirely successful, although some of the

factors of erysipelas have been produced. The poison is evidently not extremely virulent, and it does not act at a great distance. The disease is contagious and communicable, however, and it may be communicated by a short period of exposure at times. It clings tenaciously to furniture and the walls of houses, especially in ill-ventilated hospitals and tenement-houses. An injury or break in the continuity of the epithelial surface is required as essential for the admission of infection, although this is frequently so slight as to escape careful search for it. The disease frequently attacks the subjects of surgical operations or women after confinement. It may follow vaccination. No injury is too trifling to admit the infection. Alcoholism, Bright's disease, inanition, debility, previous exhaustive illness, bad hygiene, and filthy habits all predispose to the disease. Certain individuals and families are particularly susceptible. Some persons are attacked regularly at about the same time every year, particularly if they continue to reside in the same dwelling. Of individual immunity little is known. One attack does not confer immunity.

Age is probably not of importance, since the disease occurs in all. It is less frequent toward the extremes of life, probably on account of less exposure. For the same reason, no doubt, men are oftener attacked than women. Springtime is the period of its greatest prevalence. The disease is endemic in most places. Epidemics are much less frequent since the introduction of antiseptic methods into surgery and midwifery and of better sanitation into hospitals and dwellings.

Etiology.—The streptococcus is known also by the names Streptococcus erysipelatis and S. pathogenes longus. It is peculiar in its forming long, slender chains in its growth on different media, and in the fact that it produces this disease when inoculated into man or susceptible animals. It is probably a facultative aërobe. It cannot be distinguished morphologically from the ordinary streptococci of suppuration.

Morbid Anatomy.—The lesions are found in the skin or mucous membranes. The local process is one of hyperemia which rapidly spreads from the point of original entrance for a variable distance in one or more directions. Fehleisen described three zones in the erysipelatous area: A central zone in which the process may be receding, a middle circle in which the disease is still advancing, and an outer in which it is only beginning. Section of the affected area reveals an infiltration of the skin and subcutaneous tissue, often including the fat, with granular leucocytes and serum. The leucocytes are most numerous in the capillaries and lymph-spaces of the peripheral zone; they are often particularly numerous also about the hair-follicles and sweat-glands. The edema is most pronounced in loose cellular tissue, as about the eyelids, prepuce, etc. A proliferation of fixed connective-tissue cells is described by some pathologists. Metchnikoff believes that the inflammation is arrested in the outer zone by an accumulation of phagocytes.

The visceral lesions are those usually accompanying sepsis and fever, and are not, therefore, peculiar to the disease. Abscesses and infarctions are occasionally found in the lungs, spleen, and kidneys. Malignant endocarditis, septic pericarditis, or pleuritis is sometimes found. Meningitis and edema of the brain are not infrequent, and pneumonia is occasionally developed. Nephritis is not common.

Symptoms.—The incubation varies from 48 hours to seven days. There are usually no symptoms, but malaise, anorexia, and slight fever may be present. An initial chill is almost invariably the mode of onset. It may be repeated, and is generally accompanied by headache, muscular pains, sometimes by vomiting. The severity of the rigor is generally regarded as an index to the severity of the disease. The temperature runs up, often to 104° F. (40° C.) or higher. In an ordinary case it remains high with but slight remissions for four or five days, then rapidly subsides. Headache and delirium are especially liable to occur when the scalp is affected. Nervous manifestations are especially frequent in alcoholic cases. Albuminuria is usually present. In severe cases the pulse becomes rapid and feeble, delirium deepens into coma, the tongue becomes dry, hyperpyrexia develops, and death may occur within a few hours from toxemia.

The local manifestations of the disease are to be seen at the point of original infection. This is often at the junction of the skin with a mucous membrane, at the inner canthus, or on any part of the face or ear. The part appears intensely red and swollen. The surface temperature is raised from 1° to 4° F. (0.5°—2° C.). Itching, burning, and tension are complained of. The area varies in form and is often irregular, but is readily distinguishable from the surrounding skin by its color and elevation. The process reaches its acme in three days, then begins to decline. The disease may, however, be prolonged by slow or rapid extension. The entire face is often involved and sometimes the entire body (erysipelas migrans). The skin is usually smooth and glossy, but vesicles sometimes form, and, by coalescing, produce large bullæ resembling blisters. In its progress the disease follows the lymph-channels, as is shown by red, edematous lines radiating from the periphery of the inflamed area. The neighboring lymph-glands become enlarged and occasionally suppurate. Abscesses are formed also in some cases by an invasion of the deeper tissues by the streptococci. The disease sometimes invades the mucous membrane of the mouth and pharynx; but it is not invariably severe in this location. The larynx sometimes becomes edematous through an extension of the inflammation from the skin directly through the intervening tissues. The cutaneous eruption is followed by profuse desquamation in most cases.

Complications and ***sequelæ*** are rare, with the exception of those of a septic character, to which reference has been made under the pathological anatomy. Acute rheumatism has been observed. Pulmonary erysipelas is described by Strauss, and Ivanowski saw the lesions of erysipelas in the large intestine as an extension from the perineum. Peripheral neuritis has been observed as a sequel.

Diagnosis.—There is rarely any difficulty in recognizing the disease when it affects the skin. When the oral or nasal mucous membrane is affected, the intense redness, swelling, burning pain, with high temperature and enlargement of lymph-nodes, should, in the absence of other specific cause, excite suspicion of this disease. Other forms of *adenitis* are not accompanied by so great fever. *Erythema* is not accompanied by enlargement of the glands, does not show the three zones or the elevation of erysipelas. *Glanders* at the beginning is accompanied with hard, edematous induration, but the edges are not so much elevated.

The ***prognosis*** is favorable except in infants, puerperal women, and alcoholic or debilitated subjects. When the mucous membrane of the respiratory tract is involved the prospect is less favorable. The general mortality, according to Anders, is 5.6 per cent.

Prophylaxis.—The patient should be strictly isolated, and the most rigid methods of antisepsis should be adopted, as in other acute infectious diseases. The physician while attending a case of erysipelas should not undertake an obstetric case or surgical operation of any kind.

Treatment.—Internal medication is usually unnecessary, except to meet symptoms as they arise. The diet should be light and nutritious, and stimulants and tonics should be administered in adynamic or alcoholic cases. The tincture of iron is not regarded with so much favor as formerly, but is useful in ordinary doses (15 drops in water after meals) during convalescence. The bromids, trional, or morphin may be required to induce sleep.

Local Treatment.—Many remedies have been recommended, but ichthyol is probably the most effective. It may be applied in aqueous solution, in oil, or in an ointment in the strength of from 15 to 25 per cent, covering the entire surface and a surrounding area. A mask of lint should be worn over the affected part to protect it from the air. Solutions of corrosive sublimate (1:1000), the lead and opium wash, or ointments containing carbolic acid or other antiseptic drugs, but particularly salicylic acid, are almost as effective. With a view to preventing the extension of the disease, collodion may be applied to the healthy skin just outside of the infiltrated area so as to completely surround it, or a 2 per cent solution of carbolic acid or 1:2500 solution of mercuric chlorid or biniodid may be injected into the skin.

ACUTE RHEUMATISM.

ACUTE ARTICULAR RHEUMATISM, ACUTE INFLAMMATORY RHEUMATISM, RHEUMATIC FEVER.

The term rheumatism has unfortunately been applied to a great number of very different affections, and it is probable that some of the conditions now thought to be rheumatic will in the future prove to be etiologically different. The word rheumatism has been handed down from the time of the ancient Greek writers, when the disease was attributed to a humor (*ῥεῦμα*) flowing through the system.

Definition.—An acute infectious disease manifesting high fever, inflammation of the joints, profuse sweating, and a tendency to involvement of the endocardium and other fibrous structures.

Etiology.—The specific cause is yet unknown. Bacteria have been found in the joints and other lesions, but their etiological relation has not been proved. It has been suggested that several different microorganisms may be capable of producing the disease. That it is due to infection there is little doubt, from its analogy to other infectious diseases. It occurs in epidemic form every few years in some localities. These outbreaks are usually followed by two or three milder epidemics following seasonal influences. The onset is sudden, often during good health, with chill, fever, and other symptoms all favoring the view that

it is due to a specific organism. It has been compared in many of its symptoms to pyemia, but the suppurative symptoms are absent. The avenue of entrance to the system is believed to be through the tonsils and possibly the adjacent mucous membranes, for tonsilitis and pharyngitis are often initial symptoms. The recent investigations of Walker and Beaton support the view of Poynton and Payne that the disease is due to a specific micrococcus, possibly that first described by Popoff.

Two other theories have had the support of eminent authorities, but are now maintained by few. They are the chemical theory and the nervous theory.

(1) The *chemical theory* refers the disease to an excess of either lactic or uric acid in the blood, which, it is claimed, can always be demonstrated with sufficiently delicate tests. But it is claimed, on the other hand, that these acids may result from bacterial activity, and an excess of uric acid is at least not constant.

(2) *The Nervous Theory.*—The advocates of this theory regard the joint lesions as trophic and a result either of the action of cold upon the nerve centers or as due to an accumulation of lactic acid in the system, on account of faulty metabolism, which in its turn is a result of disturbances of the nervous system.

Climate.—Rheumatism is most prevalent in the temperate zone and is favored by humidity of atmosphere. The largest number of cases occur during the winter and spring, especially in February, March, and April. In many localities the disease is endemic, and sporadic cases occur at all seasons. Epidemics are seldom observed in the United States.

The *age* of greatest susceptibility is from 15 to 30. The disease is rare before the tenth or after the fiftieth year, but young children are by no means exempt. Infants are occasionally attacked.

Sex.—Previous to puberty, rheumatism is more prevalent in girls than in boys; in after-life, perhaps on account of greater exposure, it attacks men more than women.

Heredity.—The influence of heredity is doubtful. Different members of the same family are not infrequently attacked, and some families appear to be more susceptible than others, even in successive generations. Some writers have inferred that a systemic or local type of vulnerability is transmitted. An arthritic diathesis is thought, especially by English writers, to be inherited, on account of which one individual will acquire rheumatism, another gout, and another, perhaps, arthritis deformans. But this diathesis does not manifest itself to any great degree in our country. Occupation and social position are more important, perhaps, since the disease often follows exposure to cold and wet or a sudden checking of the excretions by change of temperature. Poverty, with its attendant deprivations, and occupations necessitating a disregard of the weather are, therefore, influential in its production. Injury of a joint by direct violence, excessive activity, or strain may operate to localize the disease. The infection is believed also to be favored by anemia, inanition, nervous debility, shock, and chronic alcoholism. One attack does not confer immunity; on the contrary, it renders the individual more susceptible to future infection. Many individuals and some families appear to be immune.

Morbid Anatomy.—After death, changes are found in the joints, in the blood, and sometimes in the heart, but they are not distinctive of the disease.

The Blood.—The number of red blood-corpuscles is reduced more rapidly than in any other disease. They are often as few as 2,500,000 or less to the c.mm., and the leucocytes are more than doubled in number, rarely reaching 20,000 or over. The quantity of fibrin is also doubled, closely resembling the condition found in pneumonia, but the coagulability of the blood is diminished rather than increased.

The Joints.—The synovial membranes are swollen and hyperemic and frequently studded with fibrin flakes. The joint fluid is turbid with albumin, fibrin, and leucocytes. Pus is rarely present in uncomplicated cases. The inflammation sometimes extends along the tendon sheaths, especially in the hands, and may invade the bursæ. Capillary dilatations, ecchymoses, or extravasations are found on the cutaneous, mucous, and serous surfaces in severe cases.

The Heart.—The frequency of heart involvement is differently estimated. Some authors assert that it occurs in from a fourth to a third of all cases, probably a high estimate. The endocardium of the left side is most frequently affected. The mitral cusps, particularly along the line of contact, are swollen and covered with vegetations usually of the simple, verrucose kind. Ulcerative endocarditis is rare. Contraction and deformity of the valves remain after recovery from the rheumatism. Simple fibrinous or sero-fibrinous pericarditis is not uncommonly met with. Myocarditis sometimes occurs.

Symptoms.—Such prodromes as slight malaise, headache, pains in the joints or muscles, and slight tonsilitis or pharyngitis are sometimes observed. These are often absent, however, and the disease sets in with chilly sensations, less often with a distinct rigor and a rapid rise of temperature, often to 103° or 104° F. (39.5°—40.0° C.). At the same time one or more of the joints become swollen, hot, red, tender, and very painful. The pulse becomes moderately rapid, 100 or over, but it is usually full and compressible. Respiration is generally normal, or corresponds to the temperature. The disease reaches its acme, as a rule, within the first 24 hours. The temperature ordinarily pursues a very irregular course, fluctuating between 102° and 104° F. (39.0°—40.0° C.). The tongue is coated, the breath foul, and the patient complains of thirst. The bowels are usually constipated. A profuse acid sweat of a peculiarly sour odor is usually a striking symptom in the beginning. Later the perspiration becomes neutral or alkaline if persistent. Sudamina often form on the skin. The urine is usually scant, highly acid in reaction, and deposits much uric acid on cooling. The chlorids may be absent. Albuminuria may be present, as in other febrile conditions. The saliva is also highly acid, and an excess of potassium sulphocyanid has been found in it. The nervous phenomena are generally limited to insomnia and restlessness. Delirium sometimes develops in connection with hyperpyrexia. All these phenomena are in some cases aggravated by large doses of the salicylates. So-called cerebral rheumatism is probably nothing more than a congestion of the meninges due to toxic irritation. Cerebral embolism rarely occurs, and probably only as a result of endocarditis. As a rule, the patient lies on

his back, shrinking from the slightest motion on account of the extreme pain it occasions, and sensitive to the slightest jarring of his bed. The knees, ankles, elbows, and wrists are most frequently attacked, sometimes simultaneously, but usually in succession. If the disease begins in one of these joints, it may remain in it for a few hours or for several days, then suddenly invade another articulation. The patient may fall asleep with the disease confined to the ankles, and awake to find the elbows or wrists involved and the original joints in a state of comparative comfort. This migratory tendency, as it was regarded by the older writers, is one of the strongest characteristics of the disease. In extreme cases almost all the larger joints are simultaneously affected. The phalangeal articulations are not often involved in the first attack, and the sternoclavicular and maxillary even less frequently. Subsequent attacks frequently affect the smaller joints alone or in connection with the larger. The tendency to endocarditis should always be borne in mind, and a careful watch should be kept on the heart, particularly in young patients who have passed through a previous attack. A murmur is often heard at the apex as in other febrile diseases, which is not due to endocarditis.

The course of the disease is exceedingly variable. If not overcome by treatment, it usually lasts from 20 to 30 days, then gradually subsides. Relapses frequently occur, and the development of complications may prolong the illness.

Subacute Rheumatism.—This is a common form of the disease in every way similar to the acute form, but milder in all its features. It may follow an acute attack, or it may run a subacute course from the beginning. It is often more persistent and less amenable to treatment than the acute form and the danger of endocarditis or pericarditis is almost as great.

Acute Rheumatism in Children.—Children usually suffer from the subacute form of the disease. Tonsilitis is more frequently observed in the beginning of an attack. Erythema, cutaneous nodules, and endocarditis are oftener observed. The joint symptoms are, however, so slight in some cases as to be readily overlooked.

Complications and Sequelæ.—Hyperpyrexia is most frequent in the first attack and during the second week of the disease. The temperature frequently reaches 108° F. (42° C.) or even 110° F. (43.5° C.) in fatal cases. Delirium is often associated with it and may deepen into stupor or coma, but in many cases the mind remains clear. The pulse is usually rapid and feeble and the prostration becomes extreme.

Cardiac Affections.—Endocarditis is the most serious and unfortunately one of the most frequent complications. The liability to it is proportionate to the number of attacks, but decreases with the age of the patient. The mitral valve segments are most frequently affected, the aortic next; the pulmonary and tricuspid valves are seldom involved. The most serious results of the disease are not generally realized until the development of chronic valvular lesions and failure of compensation have developed months or years after.

Pericarditis may occur independently or it may be associated with endocarditis. It is usually simple, fibrinous or serofibrinous, but it may become purulent, especially in children.

Myocarditis is infrequent, and when present it is generally associated

with endocarditis. It consists of fatty or other degeneration of the muscle fibers. Acute dilatation of the heart has been observed.

Catarrhal pneumonia and *pleurisy* are sometimes associated with the endocardial disease. Bronchitis is not uncommon. Rapidly fatal pulmonary congestion has been observed in a few instances.

Cerebral Complications.—*Delirium*, as already stated, is sometimes developed by the high temperature or by the action of the toxins. Coma sometimes succeeds it or may develop independently of it. It frequently precedes the fatal termination of the disease by only a few hours. It sometimes develops after convalescence has begun, and is occasionally due to uremia.

Convulsions seldom occur. They may precede the coma or they may occur independently, especially in alcoholic subjects.

Chorea has sometimes been observed as an associated disease. It is not always a result of the rheumatism. The joint pains and tenderness in children affected primarily with chorea are, perhaps, of a different nature in some cases. Chorea due to embolism following rheumatic endocarditis may not develop until weeks or months after the attack.

Cutaneous Affections.—Sudamina, erythema, petechiæ, and ecchymoses may be seen. The most interesting complication of this kind is the so-called peliosis rheumatica. In it purpuric spots, with or without urticaria or erythema, accompany the rheumatic pains. The relation of the affection to rheumatism is doubtful.

Subcutaneous Nodosities.—During and after the disease, subcutaneous nodules varying in size from a small shot to a pea, firm, but movable, are in rare cases found attached to the tendons of the fingers, hands, wrists, at the edge of the patella, or over the elbows, maleoli, scapulæ, or spines of the vertebræ, especially in children and young adults. The skin is elevated, but not tender. They develop rapidly, especially when chronic endocarditis is present, and may remain for months. They are oftener seen in England than in America and are more characteristic of gout and arthritis deformans. (See Heberden's Nodosities.)

Conjunctivitis and iritis often recur with each rheumatic attack, and are amenable to the same treatment. Cystitis, orchitis, and other affections of the genitourinary organs, muscular atrophy, thyroiditis, and other more or less accidental complications have been noted.

Gastritis, profuse sweating, insomnia, delirium, and extreme prostration are sometimes induced by the alkaline treatment.

Diagnosis.—The intense painfulness and tenderness of the joints, with the swelling, wandering character of the affection, and the high temperature, seldom leave doubt as to the diagnosis. It is probably incorrect to apply the term rheumatism to the secondary arthritis which often complicates scarlatina, pyemia, and many other acute diseases, since it is doubtless due to a different kind of intoxication. It may usually be distinguished by the presence of the causative infection. Pyemia is to be distinguished by the frequent chills, intermittent character of the temperature, the often recognizable suppurative processes, and by the dusky or icteric color of the skin as compared with the anemia of rheumatism.

Gonorrheal rheumatism is generally confined to a single joint. Although persistent, it is not accompanied by so great prostration or

sweating. A polyarthritic form occasionally occurs, but the inflammation is not confined to the joint, and the swelling is generally fusiform.

Acute osteomyelitis or *necrosis* affecting the lower end of the femur or the tibia may cause doubt. In either affection, however, the shaft and epiphyses are affected, and not the joints. Rigors frequently occur, but sweats are uncommon. The constitutional as well as the local symptoms are severe. An early correct diagnosis is extremely important for the adoption of surgical treatment.

Scurvy with symptoms grossly resembling rheumatism has only recently been recognized as a comparatively frequent affection of infants. It affects the shaft of the bone and not the joints, however, and it is usually confined to a single locality, often an unusual one for rheumatism.

Gout is generally confined to a single, small joint, especially the great toe. When it invades several large joints it is difficult of distinction. The age, family history, and habits of the patient are of value, and the discovery of tophi, little nodules about the joints, and an excess of uric acid in the urine is distinctive.

Prognosis.—The prospect for recovery from the immediate disease is generally good, but the danger of cardiac complications and the liability to recurrences months or years after are always to be regarded. The joints generally recover completely without more than temporary stiffness. Acute cases may always subside into a subacute form and finally become chronic. Endocarditis is usually followed by permanent valvular insufficiency or stenosis. Death, when it occurs during an attack, is generally due to hyperpyrexia, myocarditis or resultant acute dilatation, pneumonia, or pleurisy.

Treatment.—The suffering is greatly mitigated by placing the patient upon a smooth, elastic mattress, in a quiet room admitting sunlight. He should have a flannel gown and should lie between blankets. Frequent changes are necessary on account of profuse sweating. The diet during the febrile stage should be limited to milk and fluid or semi-fluid articles. The thirst demands an abundance of pure water or lemonade at short intervals. Broths and soups may be given, but beef-juice should be omitted.

Local Treatment.—The joint should always be kept warm and at rest. This may be done by wrapping it in flannel or cotton batting, and fixation in a splint is often of great benefit. Hot-water bottles may be applied. Relief is afforded in some cases by hot fomentations and applications of chloroform, aconite, or chloral liniments and lotions and in others by cold compresses and ice-bags. Blisters often afford relief when applied below the affected joint, and the Paquelin cautery, lightly applied, is probably better. But such measures are not often necessary. In mild cases an ointment of salicylic acid (2 per cent) freely applied to the affected joints is often all that is necessary. Another excellent application is composed of equal parts of guaiacol and glycerin. The oil of wintergreen (gaultheria) may be applied pure. Methyl salicylate (50 to 100 drops) has recently been recommended.

Medication.—The alkaline treatment is almost universally employed. The salicylates are regarded by Strümpell and many other authorities as specifics to such an extent that the diagnosis may be called into question when they fail to cure. Either the acid or one of its salts may

be employed. Salicylic acid should be given in 10-grain (0.6) doses, in capsules or tablets, every hour until 1 or 2 drams (4.0—8.0) have been taken. The sodium or ammonium salicylate should be given freely, gr. xv to xx (1.0—1.25) every two hours, until the pain is relieved or physiological effects, tinnitus, vertigo, or nausea, are produced. The drug should always be chemically pure. Some prefer that made from wintergreen. Strümpell advises giving a dose of ʒj to ʒjss (4.0—6.0) three times a day. Prompt relief from the pain is generally afforded. The doses may then be reduced in size or in frequency. In many cases the disease is subdued to so great an extent within two or three days that the drug may be discontinued. It is chiefly in cases that fail to respond promptly that a continued use of the salicylates proves of no benefit. It is ordinarily considered better to discontinue their administration in any case as soon as the pain has been entirely relieved, in order not to increase the anemia, which is already rapidly developing. Potassium bicarbonate in 20-grain (1.25) doses, given along with the sodium salicylate, increases its action. It may be continued after the sodium salt has been discontinued, alone or with half-dram (2.0) doses of potassium acetate.

Disagreeable effects are not infrequently produced by the salicylates. The tinnitus and deafness are often extremely annoying and may be attended with vertigo and epistaxis. Delirium, dyspnea, and a peculiar nervous stimulation are produced in some cases. Many patients object to the taste, however disguised, and in some it produces extreme nausea. These symptoms may be relieved in a measure by sodium bromid, gr. xx (1.25); or the oil of wintergreen may be employed in doses of ♏xx (1.25) every two hours, in milk or emulsified with mucilage. Salol has not proved efficient in the hands of most observers. Salophen, gr. xv (1.0), has been recommended, but it is inferior to the salicylates. Sodium or potassium iodid is often beneficial as convalescence approaches.

It is seldom that the salicylates fail to afford relief from the suffering, but in some cases morphin, gr. ⅛ to ¼ (0.008—0.016) hypodermically, is required. Phenacetin, gr. v to x (0.35—0.65), often affords relief and aids the action of the salicylates.

Menzer has recently reported good results in acute and chronic rheumatism from the injection of a streptococcus serum obtained from culture on ascites fluid of micrococci removed from the tonsils.

During convalescence, iron and tonics should be administered to overcome the anemia. The continued adminstration of iron sometimes appears to prevent a recurrence in persons subject to repeated attacks. The patient should avoid exposure, but fresh air and sunshine are beneficial. The diet should be nutritious, but without meat until convalescence is complete. The rheumatic subject should, as a rule, indulge sparingly in nitrogenous food and malt liquors.

GONORRHEAL INFECTION.

Definition.—An infection caused by the gonococcus of Neisser and manifested by symptoms of general toxemia or of localized inflammation, especially in the joints.

Etiology.—The condition is due either to the entrance of the gonococcus into the blood or to the absorption of the toxins. It occurs in about 10 per cent of all cases of gonorrhea, and is most frequently seen in young men the subjects of gonorrheal urethritis. It may, however, occur in individuals of any age or either sex in the presence of the specific cause. Taylor maintains that infection from urethritis is unusual until the posterior urethra has become involved. It may result from the vulvovaginitis of children. Injury favors the localization of the disease in a joint. Exposure to cold and wet is not recognized as an etiological factor. Individual susceptibility is more important. The disease is entirely independent of rheumatism or the rheumatic diathesis. The gonococcus has been repeatedly found in the blood, in the affected articulations, and in the pericardium when involved.

Morbid Anatomy.—When death results from toxemia, the changes are those of septicemia. An original source of infection is usually found in the urethra or possibly in a suppurating gland.

The Articulations.—Inflammation and thickening of the capsular ligament and synovial membrane are the distinguishing features. There may be much or little effusion into the joint. The fluid is generally turbid owing to the presence of fibrin and leucocytes; suppuration is unusual, but is sometimes encountered, particularly in the wrist and knee joints. Mixed infection with streptococci, staphylococci, or pneumococci is not uncommon. Hydrarthrosis occurs, especially in the knees. Edema is peculiar to the wrist and ankle. The inflammation is rarely limited to the articulation, but it extends for a variable distance along the tendon sheaths or periosteum above and below the joint. As a result, the joint has a fusiform appearance. The inflammation is limited to these structures in some cases and the joint is not affected. Fibrous thickening and adhesions are a more constant and persistent result than in acute rheumatism.

Clinical Forms and Symptoms.—1. *Septicemic Form.*—This may occur as early as the second week of the primary gonorrhea, or as a result of secondary infections from it. It may follow any of the recognized local lesions of the disease—urethritis, conjunctivitis, vaginitis, endocarditis, abscess in the prostate or other glands. The symptoms are those of septicemia or pyemia. There is often a slight elevation of temperature, however, in the beginning of a specific urethritis, which is not necessarily of this character.

2. *Gonorrheal Arthritis.*—The joint involvement does not generally begin until the fourth week, of a gonorrhea. It has been observed, however, in the second or third week, and after several months have elapsed and the urethritis has become chronic. The clinical manifestations are exceedingly variable and most persistent in character. One or many joints may be involved. A migratory painfulness of the joint may be the only symptom. In other cases, several joints become simultaneously swollen and painful, as in subacute rheumatism. There is moderate fever, as a rule.

The typical acute gonorrheal arthritis is a monarthritis. The swelling is often extreme and the pain severe. The fever may be moderate. Suppuration of the joint occasionally occurs. The disease may become chronic, or it may run a chronic course from the start. The periartic-

ular form in which the inflammation is confined to the tendon sheaths is seen especially in the knee and elbow or along the tendo achillis.

3. *Gonorrheal Endocarditis.*—This may occur as a complication of the other forms, or independently, even in the absence of articular involvement. It is often ulcerative in character and fatal in its result. Pericarditis occasionally occurs.

Complications.—Endocarditis, pericarditis, and pleurisy may occur. Cerebral complications have been observed. Bursitis and tenosynovitis are not uncommon. The muscles and fascia, especially of the palm and sole, may become infected. Iritis is sometimes observed.

Diagnosis.—The differentiation between a gonorrheal arthritis and an intercurrent rheumatic arthritis is often impossible without the discovery of the gonococcus in the joint fluid. The diagnostician may be misled when the patient denies the existence of gonorrhea. The most distinctive features of gonorrheal arthritis are the involvement of but one or at most two or three joints or the absence of migratory tendency, the fusiform character of the swelling, the tendency to invade the tendon sheath, and the extreme persistence of the disease.

Prognosis.—Notwithstanding the persistence of the disease, ultimate recovery is the rule. A greater or less degree of anchylosis often remains for a time. The septicemic form and the pericarditis may prove fatal. Recurrence is not unusual, especially when a fresh attack of gonorrhea is contracted. Mixed infection adds gravity to the prognosis.

Treatment.—The thorough treatment of the primary source of infection is important. The condition of the posterior urethra should be looked into. The salicylates and alkalis are of little or no benefit. They may be employed, however, when the diagnosis has not been fully established. Potassium iodid has not proved of value. The administration of tonics, particularly of iron and arsenic, has proved the most satisfactory treatment. Good food and fresh air are highly advantageous. During the acute stage the patient must be kept at rest and the joint should be immobilized. The hot-air treatment has proved of benefit in the later stages of the disease. Blisters, cauterization, and counter-irritants have been recommended. In persistent cases the joint cavity may be aspirated under careful antisepsis, and a 1:2500 solution of mercuric chlorid or a 1:50 solution of carbolic acid may be injected. If suppuration occurs, vigorous surgical measures must be promptly adopted.

SYPHILIS.

LUES VENEREA, THE POX.

Definition.—A chronic infectious disease, usually of venereal origin, characterized by a great variety of pathological lesions and clinical manifestations corresponding with the stage of the disease and the part affected. It may be hereditary (congenital) or acquired.

Etiology.—The specific cause has not been definitely determined. The bacillus of Lustgarten is often found in the lesions. It is a straight or curved rod having slightly enlarged ends and measuring 3 or 4μ in length. It is probably the same as that recently found by Max

Joseph in the semen of syphilitics and propagated by cultivation on sterile normal placenta. The disease is peculiar to man, and susceptibility is probably universal.

1. *Accidental Infection.*—Inoculation most frequently occurs through the skin or mucous membrane of the genitalia as a result of sexual congress. It may occur anywhere that the virus comes into contact with a tissue whose continuity is broken. The term syphilis insontium is applied to the disease when innocently acquired. The virus may be conveyed by means of such contaminated articles as drinking-vessels, towels, bed-linen, rags, razors, pipes, dental instruments. It has been communicated by kissing, tattooing, vaccination with humanized virus, and the introduction of an infected hypodermic needle. The infant may become infected by the kiss of a syphilitic person and convey the virus to the nipple of the mother. The hand of the physician has been inoculated during surgical and obstetric work.

2. *Inherited Infection.*—This may be transmitted from either parent in whom the disease is active at the time of conception. Tertiary syphilis of the parent does not beget in the offspring active syphilis, but a feeble, cachectic constitution with great liability to mental defects and physical deformities. The disease may be transmitted to the fetus through the placenta when the mother has become infected during her pregnancy.

Pathology.—*The Chancre.*—The primary lesion consists of a circumscribed infiltration of the connective tissue with granulation and epithelioid cells, with an occasional giant-cell and a few bacilli, usually found in the center of the infiltration. Changes occur also in the smaller blood-vessels, nerve fibers, and lymph-vessels immediately around it.

Secondary Lesions.—These are of the greatest variety, including cutaneous eruptions, mucous patches, condylomata, affections of the eye, nerves, and viscera.

Tertiary Lesions.—The lesion characteristic of this stage is the gumma, the most distinctive of all syphilitic formations. It belongs to the granulomata and may be either infiltrating or circumscribed in character. It may originate in the connective tissue of any structure of the body. It consists of an infiltration of a greater or less area of the tissue, with small round or polyhedral cells in which new blood-vessels are formed. Subsequent changes lead to the formation of ulcers in the superficial tissues or to caseation, amyloid degeneration, or sclerosis in the internal organs. Large cutaneous syphilids and vascular sclerosis are common features of this stage. The lesions are usually symmetrical.

Symptoms.—**Acquired Syphilis.**—The clinical manifestations are usually divided for description into three stages, although the line of separation is not always clearly defined. Following inoculation there is a period of latency of three or more weeks during which there is no evidence of the disease.

Primary Stage.—The initial lesion appears as a small papule which gradually enlarges and breaks in the center, to form an ulcer with indurated edges. The nearest lymph-glands become enlarged and indurated; they seldom suppurate.

Secondary Stage.—This begins with the first evidence of constitutional involvement, from six weeks to three months after the appearance of

the chancre. A mild fever frequently develops; it runs a continuous course, seldom reaching 102° F. (38.8° C.), but it is sometimes severe and may run up to 104° F. (40° C.). Anæmia is often an early symptom, the blood-count showing 3,000,000 corpuscles to the cubic millimeter or less. In some cases the integument assumes a slightly yellowish, cachectic hue.

A more or less distinct roseolar rash appears on the chest, abdomen, and anterior surfaces of the arms, sometimes on the entire body, but seldom on the face, and persists from two to three weeks. A papular eruption (papular syphilid), resembling acne, sometimes appears on the face and trunk. Less frequently there is a pustular rash which has been mistaken for smallpox, or a squamous syphilid resembling psoriasis, though less scaly. Mucous patches, flat warty excrescences, showing a tendency to ulcerate, appear at the same time on the mucous membrane of the mouth, lips, tongue, or throat, or on moist surfaces of the skin, as at the angles of the mouth. Condylomata, warty outgrowths of the papillæ, are often seen at the junctions of the skin with the mucous membranes, or upon surfaces which are kept moist by contact with adjacent surfaces, as in the gluteal and anal folds. The lymph-glands of the entire body become more or less indurated. Deep-seated osteoscopic pains are complained of at night during this stage, and the anterior surface of the tibia may become slightly swollen, roughened, and sensitive to pressure. The scalp may also become tender, and there often develops a point of extreme sensitiveness to firm pressure near the upper end of the sternum.

Iritis is not uncommon from the third to the sixth month of the disease. Choroiditis, retinitis, otitis, laryngitis, and other inflammatory affections are sometimes produced. The hair falls out, usually in patches, sometimes as a general alopecia, including the eyebrows and other regions; brittleness of the finger-nails is often observed.

Tertiary Stage.—There is no clear mark of distinction between this stage and the secondary. It generally begins in from three to six months after the beginning of constitutional infection, unless delayed by treatment. It may be preceded by a short period of latency. The most distinctive features are the appearance of scattered syphilids which manifest a tendency to produce deep ulcerations, and gummatous growths in the skin and subcutaneous tissues, muscles, and internal organs. Amyloid degeneration and sclerosis are common. The internal lesions are largely a result of these processes.

1. *Digestive System.*—Fissures, ulcers (mucous patches), or gummata may form in the tongue, causing much enlargement and interference with deglutition. The tonsils may be swollen and ulcerated, and gummata sometimes form in the posterior wall of the pharynx.

The esophagus and stomach are seldom affected, but stenosis occasionally results from the pressure of large gummata. Ulceration occurs at times in the intestine; symptoms may be absent, or there may be tenderness and diarrhea. The rectum is a common seat of cicatricial stenosis, especially in women. The constriction usually forms above the internal sphincter and produces alternating constipation and diarrhea, often ribbon-like stools, and reflex nervous manifestations.

In the liver the disease is manifested by: (*a*) Diffused hepatitis, a

small-celled infiltration with hyperplasia of the connective tissue producing enlargement or contraction; (*b*) gummata, sometimes large enough to be recognizable through the abdominal wall; (*c*) perihepatitis with thickening of the capsule, frequently accompanied with pain and tenderness; and (*d*) amyloid degeneration with marked enlargement. These conditions are frequently attended with slight icterus, emaciation, ascites, and enlargement of the spleen and symptoms on the part of other organs.

2. *Respiratory System.*—Ulcers of the larynx sometimes develop and may destroy the vocal cords or cause necrosis of the cartilages; the epiglottis is sometimes involved in the destructive process. Syphilis of the lung is not common, but gummatous and sclerotic changes occasionally occur. The lesions are usually confined to the base and may involve more than one lobe. Bronchiectasis may result from the cicatricial contraction. The symptoms suggest tuberculosis, but the disease is of slow progress and can generally be recognized by the location in the base, the absence of tubercle bacilli, and the history or evidences of syphilitic infection.

3. *Circulatory System.*—Vegetative endocarditis, gummatous myocarditis, amyloid degeneration, and fibrous induration are the usual, though infrequent, cardiac lesions. The coronary arteries may be obliterated or aneurism may be produced. The arterial system is especially liable to syphilitic changes in the nature of obliterative endarteritis. Arteriosclerosis and the formation of gummatous deposits in the adventitia are particularly frequent in the cerebral vessels. Syphilis is probably the most common cause of aneurism. .

4. *Nervous System.*—Either the brain or the cord may be affected. Gummata form especially in the meninges and cause, chiefly through pressure, a localized meningitis, encephalitis, or myelitis with degenerative changes and softening of the nerve tissue. Hemorrhage is sometimes produced. The gummata may, through degenerative changes, become fibrous, caseous, cystic, or calcareous. The symptoms produced are those of tumor affecting the brain or cord.

5. *Renal System.*—The kidneys are also the seat of gummatous formations, producing symptoms of nephritis, albuminuria, edema, sometimes hematuria. Amyloid degeneration is not infrequent, in association with the same condition in other organs, especially the liver, intestines, and spleen. Anemia and emaciation are prominent symptoms; the skin becomes waxy. A large quantity of clear urine is voided which contains albumin and tube-casts. Dropsy and diarrhea frequently contribute to the inevitably fatal course of the amyloid disease. The bladder is not often the seat of syphilitic disease, but perforating ulcers are thought to be sometimes of this nature. Orchitis of a fibrous or gummatous nature sometimes occurs. Epididymitis is rare.

Metasyphilitic Affections.—Syphilis renders the subject liable to certain diseases not necessarily of syphilitic nature, as locomotor ataxia, epilepsy, paralytic dementia, and pernicious anemia. Epilepsy beginning in adult life independently of trauma is almost invariably of this character.

Congenital Syphilis.—The firstborn of syphilitic parents is generally premature and stillborn. When, however, the disease is present at birth

in a living child, it has usually the form of a vesicular or bullous cutaneous eruption with enlargement of the liver and spleen. The child is feeble and emaciated, has the snuffles, its lips are fissured, and the joints may be enlarged. In other instances the infant appears healthy at birth, and thrives for a month or six weeks, then develops a nasal catarrh (syphilitic rhinitis), which interferes with its nursing and often leads to ozena, necrosis of the nasal septum, and the production of a characteristic deformity. Other lesions soon appear, particularly an erythematous, eczematous, or papular eruption on the nates. The child becomes weak and emaciated. It is often restless and sleepless at night. Its cry is feeble and high pitched on account of the weakness; the face appears old and wrinkled. Gastrointestinal disturbances often develop and hasten the termination of the unfortunate life.

The child sometimes survives, but it never becomes vigorous. At the age of twenty years it has often the appearance of twelve (infantilism). The skin is sallow, the hair scant, and the teeth deformed. The upper central incisors are notched, short, and wedge-shaped, narrower at the cutting edge than at the gum (Hutchinson teeth). The cranium and the long bones are often deformed; the forehead is usually broad. Owing to a congenital weakness of blood-vessel walls, large blue dilated veins are often seen upon the head and neck. Many other lesions peculiar to the disease, but not confined to the congenital form of it, are often observed in the later life of these subjects.

Diagnosis.—The features which distinguish the primary sore, or chancre, are its appearance not less than three weeks after the inoculation, its round or slightly oval shape varying in size from a split pea to that of a ten-cent piece, its red and indurated edges, and the absence of spreading ulceration. The *chancroid* is differentiated by its earlier appearance, irregular shape, flat edges, and early ulceration. The syphilitic sore is single; there may be several chancroids. The chancroidal bubo suppurates, but there are no other secondary lesions. The secondary lesions of syphilis are typical, especially when a history of the primary sore can be obtained. In women, however, this is often impossible. A roseolar eruption with mucous patches and enlargement of the lymph-glands, with moderate fever or normal temperature, can rarely be attributed to any other affection.

The chief difficulty is encountered in the diagnosis of the visceral lesions of the tertiary period. Unfortunately the results of direct interrogation are less to be relied upon than in any other disease. The syphilitic will lie to his own hurt, and condemn the physician who accepts his statement as true. Women are frequently innocent victims, and it is often wrong to arouse a suspicion of their condition. In such cases the diagnosis must often be based upon the history of a roseolar rash, mucous patches, sore throat, possibly an iritis, as an evidence of which a contracted pupil may remain. There is, perhaps, a history of nocturnal osteoscopic pains, loss of hair, a paronychia of several fingers, and there may have been one or more miscarriages. Examination may reveal cicatrices in the mouth, throat, the inguinal region, or over the tibia, a deformed or perforated palate. The earlier cutaneous lesions are symmetrical in location; the later are not always so. The tertiary rupia produces characteristic, prominent crusts resting upon deep ulcers.

The vesicular lesions of syphilis when found upon the hands are often confined to the palms; when on the feet, to the soles; they do not itch. These features exclude papular eczema and other itching eruptions. Acne is accompanied by comedones, and the sores are confined to the sebaceous follicles. They are less inflamed and do not form ulcers.

Congenital Syphilis.—The appearance of snuffles and a cutaneous eruption on the nates of a child within the first three months is not to be mistaken for any other disease. Later the anemia, emaciation, the peculiarly aged facies, especially if accompanied with enlarged joints and wedge-shaped teeth, are pathognomonic.

The *therapeutic test*, made by the administration of large doses of potassium iodid, is of greater value in the cutaneous than in the visceral forms of the disease. The drug is well borne by a syphilitic patient and causes rapid improvement of the condition, but the test is not infallible.

Prognosis.—The prognosis is good in the early stages, except in alcoholic or cachectic patients or those debilitated by age or disease. Much depends upon beginning the treatment early. Recovery is often complete after three years' treatment. The recovery should not be regarded as permanent, however, until at least one year has elapsed without a reappearance of the symptoms after the discontinuance of medication.

When treatment is begun in the later stage and when relapse occurs during persistent treatment, little hope can be entertained of ultimate recovery. One of the most difficult questions which frequently confront the physician is that of the marriage of the syphilitic. This should be unconditionally condemned until at least a year has passed without relapse and without medication. It were better, indeed, that syphilitics never married.

The prognosis of inherited syphilis is always bad. The earlier the disease appears the less is the prospect of life.

Prophylaxis.—Syphilis is theoretically a preventable disease and one that could be eradicated. Practically it is not so. Few problems have received so much study from the beginning of the world to the present time as that of the social evil, but the solution is not yet. The systematic inspection of prostitutes has proved only partially successful. Much of the difficulty arises from the recklessness of young men during the early stages of the disease. It is the duty of the physician to warn his patient of the certainty of communicating the disease by contact or by the use of the same drinking-cups, towels, or other articles. The criminality of voluntarily or carelessly communicating the disease to another should be impressed upon his mind.

Treatment.—The primary sore requires little treatment further than cleansing and the application of calomel or other dry powder twice or thrice daily. It is customary with most physicians to delay the constitutional treatment until the diagnosis has been fully established by the appearance of secondary manifestations. When this has been done, it is less difficult, as a rule, to convince the patient of the correctness of the diagnosis and of the necessity of continued treatment after the disease has been gotten under control. As soon as the roseolar rash has appeared, however, the most energetic treatment should be instituted.

Mercury and potassium iodid are specific remedies. The former is

especially valuable in the secondary stage, the latter in the later manifestations of the disease. The iodids of mercury are much employed, however, during the tertiary period.

Inunction Treatment.—It is generally best to begin the treatment with mercurial inunction. One dram of mercurial ointment should be rubbed into the skin for a half-hour or longer every evening for two or three weeks, substituting a warm bath for the inunction every fifth or sixth evening. The sides of the chest and abdomen and the inner surfaces of the arms and thighs, places where the skin is thin, should be selected on successive evenings.

Internal Treatment.—Mercury may be administered by the mouth. The blue pill (massa hydrargyri) or mercury with chalk (hydrargyrum cum creta) may be given in dose of 1 grain (0.06), preferably combined with ⅛ or ¼ grain (0.008 or 0.016) of opium to prevent catharsis. The mercuric chlorid is employed in doses of 1-40 to 1-20 (0.0016—0.0032); the protiodid ¼ grain (0.016), or the biniodid, gr. 1-20 increasing to 1-12 (0.003 to 0.005).

The *subcutaneous treatment* is sometimes to be preferred. The mercuric chlorid is generally employed, ⅓ grain (0.02) being injected deep into the gluteus or other muscle once a week. One or two grains (0.06—0.13) of calomel in 20 minims (1.23) of glycerin, or ten drops of a mixture of equal parts of metallic mercury and lanolin containing 2 per cent of carbolic acid, may be injected in the same manner.

Fumigation is seldom employed in this country. For its administration the patient is placed on a chair having a perforated seat, then covered with a blanket. Perspiration is started by the heat from a spirit lamp placed under the chair. After this has been accomplished, ʒ ss (2.0) of calomel is vaporized by placing it in a spoon and holding it over the alcohol flame. The patient then goes to bed wrapped in the blanket.

In the later stages of the disease, potassium iodid is most relied upon. It should be given in doses of from 15 to 30 grains (1.0—2.0) or more, well diluted, three times a day for several months at a time. When the result is not altogether satisfactory, the dose may be increased so long as iodism is not produced, or the mixed treatment may be employed, combining about 1-40 grain (0.0016) of the mercuric chlorid with 10 to 15 grains of the potassium iodid. The action of the iodid is increased by giving it in a large draught of hot water. Some physicians occasionally interrupt the treatment with the potassium iodid by giving one of the iodids of mercury.

Whatever the plan adopted, the treatment should be continued without interruption for fully a year. An occasional interval of a week without treatment may then be allowed. Two years of treatment is the minimum of time required for a cure; three years of persistent medication is much safer.

Treatment of Hereditary Syphilis.—Mercurial treatment of the mother during her pregnancy is of benefit to the child and may enable it to survive. The infant may be nursed by the mother when she is in physical condition to provide it with nourishment, since it is a well-known law (Colles's law) that the mother does not become inoculated even if not previously syphilitic; but it must never be given to the wet-nurse,

for it will surely inoculate the nipple. The subsequent treatment of the infant is based on the same principles as that of adults. Calomel or mercury and chalk may be given in doses of 1-10 grain (0.006) t. i. d. Inunctions or baths are a less certain mode of introducing the mercury into the system. For lesions corresponding to those of the tertiary period, the iodids or the mixed treatment should be employed. The sirup of the iodid of iron is an excellent remedy in these cases, since it counteracts also the anemia.

The administration of tonics, particularly of iron and codliver oil, is advantageous in advanced cases. Cases that have persistently resisted vigorous specific treatment not infrequently begin a rapid improvement after resort to these remedies.

Precautions.—During treatment with mercury the patient should abstain from alcohol and tobacco and he should not eat freely of acid fruits and salads. The mouth and teeth should be cleansed after each meal, preferably with a 5 per cent solution of potassium chlorate. Upon the first indication of ptyalism—an increased flow of saliva, soreness of the gums, fetid breath, or diarrhea—the mercury must be discontinued for a few days. When symptoms of iodism appear—coryza, headache, drowsiness, acne, erythema, or albuminuria—this remedy must be given in reduced doses or temporarily discontinued.

TUBERCULOSIS.

Tuberculosis is the most universal of all diseases, prevailing in all parts of the world at all seasons and among all races. It attacks also many of the lower animals. Among domestic animals it is most frequent in cattle, next in young swine; sheep, horses, dogs, and cats are less frequently attacked. Fowls and fish are not exempt. Wild animals are seldom affected, but when domesticated they become exceedingly susceptible to it. Rats and mice acquire the disease. Among human beings it is more destructive than all the other communicable diseases combined, causing 14 per cent of the entire mortality. This fact affords only a partially correct idea of its prevalence, however, for many tuberculous persons die of other diseases, and the lesions are often found after death in those who were not known to be affected during life. The mortality from it in different countries corresponds very closely to the population. Every organ and every tissue of the body is liable to the disease, but the lungs are more frequently attacked than any other structure. It is customary, therefore, to regard tuberculosis as a general affection having localized lesions.

I. GENERAL TUBERCULOSIS.

Definition.—An infectious disease caused by the bacillus tuberculosis of Koch, the entrance of which leads to the formation of tubercles or to diffused infiltrations of tubercular tissue that frequently undergo subsequent caseation or sclerosis, and sometimes calcification.

Etiology.—*The Bacillus.*—The bacillus tuberculosis, discovered by Koch, in 1881, is the specific cause of tuberculosis in all of its many manifestations. The disease can be produced by no other organism. The bacillus is a slender, nonmotile, aërobic rod, measuring from 1.5 to 4μ in length. It is often found in clusters. Branching forms are exceptionally seen, and it has sometimes a beaded appearance in stained preparations, which has been incorrectly attributed to the presence of spores. Probably the most distinctive feature of the bacillus is the

slowness with which it takes up the analin stains and its equal reluctance to part with them, even under the influence of mineral acids of sufficient strength to decolorize all other bacteria. It is cultivated with some difficulty, growing best on blood serum and only at a temperature of 37° C. It is often overcome in cultures by the more exuberant growth of other bacteria, but within the body it is remarkably hardy, growing luxuriantly in the presence of staphylococci, streptococci, and other organisms. Exposure in water to a temperature of 60° C. destroys it in 15 minutes, although it is capable of resisting ordinary desiccation for months. Cold has no effect upon it. The rays of the sun are fatal to it within from 15 minutes to several hours according to the season and the character of the sputum or other substance in which it is embedded. Diffuse daylight near a window is said to destroy its vitality within a week. The bacillus is believed to be strictly a parasite, as it is not known to find conditions suitable for its propagation outside of the body of a living animal, except in culture-media.

The recent investigation of the apparent relation of the tubercle bacillus to the ray fungus of actinomycosis, although properly belonging to the department of bacteriology, is of much interest in this connection. It has been found that when this bacillus has been passed through the bodies of such cold-blooded animals as the frog, it adapts itself better to a saprophytic existence, growing vigorously upon artificial media at ordinary temperatures, while its virulence is reduced. Regularly branching forms, sometimes producing threads, are commonly seen. As Lubarsch suggests, the appearance is that of a reversion to an original saprophytic state. Similar branching forms have been found upon grass and in cow's dung. The question is therefore raised by Hektoen whether the tubercle bacillus may not be a parasitic form of some of these organisms closely related to the ray fungus and existing naturally upon grass and elsewhere. The case of streptothrix bronchitis reported by Musser seems to support this theory.

Chemical Products.—The bacillus yields a series of chemical products regarding the nature of which comparatively little is known. The most important are the fluid and precipitated toxin, the aqueous tuberculin, best known in the form of Koch's tuberculin, which is a glycerin extract, and the fat-free bacilli obtained by precipitation. An albumose and a ptomain have also been isolated.

The *virulence* of the bacillus is very different when the latter is obtained from different sources or when it is propagated on different culture-media. Prolonged cultivation causes marked reduction of virulence.

Distribution of Bacilli.—The bacilli may be found in the blood in acute tuberculosis, in the sputum when the respiratory passages are involved, in the urine, feces, and other discharges from tubercular foci. They are found also in all tubercular lesions, their numbers corresponding closely to the activity of the disease. In the lymph-glands and other structures in chronic tuberculosis they may be unrecognizable by staining methods, although they still respond to cultivation and inoculation into the lower animals.

Modes of Infection.—In a great majority of cases the disease is undoubtedly communicated directly or indirectly from person to person. To what extent tuberculous animals contribute to the dissemination of

the disease among human beings is still a matter of discussion. Cattle are looked upon by some writers as important sources of infection, particularly through their flesh and milk, notwithstanding the fact that Koch has questioned the possibility of the transmission of bovine tuberculosis to man. The immediate avenue of invasion is in a majority of cases the respiratory passages, but the infection may be hereditary or it may occur under favorable conditions through any of the cutaneous or mucous surfaces of the body. The infective agent is generally acquired through inhalation, ingestion, or inoculation.

1. *Hereditary Transmission.*—There are three ways in which the transmission of tuberculosis from parent to offspring is theoretically possible, namely: (*a*) By the sperm, (*b*) by the ovum, and (*c*) the blood of the mother through the placental circulation. Tubercle bacilli have been found in the semen, but, as Osler remarks, the chances are extremely small that the bacillus should lodge in the individual spermatozoön which fecundates the ovum, and they appear still smaller when we consider that the spermatozoön is made up of nuclear material which the tubercle bacillus is never known to attack. No case has been recorded in which hereditary transmission from the father was an inevitable conclusion from the facts in the case.

The same objections are almost equally applicable to the theory of transmission through the ovum, for it is almost inconceivable that the ovum should survive the entrance of the bacillus. Baumgarten, however, detected a bacillus within the ovum of a rabbit which he had artificially impregnated with tubercular semen. That the fetus may become infected from the blood of a tuberculous mother is possible, and the view that such infection occurs is supported by the fact that tubercular lesions have been found in the fetus. It is not an accident which is likely to occur often, however, for the bacilli are rarely found in the general circulation except in the rapidly fatal miliary form of the disease or as a result of the perforation of a blood-vessel by a tubercular infiltration. It is claimed by some writers that the placenta is always tubercular in these cases, and tubercles have been found in it in several instances. But the value of all these speculations is minimized by the statement of Hahn, that only 20 authentic cases of congenital tuberculosis have been recorded. In nearly all cases regarded as congenital a period of two weeks or more has elapsed between the birth of the infant and the discovery of the disease. There is a possibility, therefore, that infection of the infant may have occurred through inhalation or ingestion of bacilli during the first days of life. The bacilli are often, no doubt, implanted upon the lips of the infant through the kisses of the tuberculous mother or through the application of her handkerchief to its face.

Those who still hold to the doctrine that tuberculosis is to any great extent inherited maintain that the bacilli in many instances remain dormant in the body for a great length of time and ultimately become active when the individual's power of resistance is in some way lowered. In this regard the disease is comparable to syphilis, which frequently exhibits this type of latency. The comparison does not hold, however, when we reflect that tuberculosis frequently passes over the second generation, to appear in the third, a fact which is not easily accounted for on the theory of latency.

There can be no doubt that a vulnerable type of constitution is often transmitted to the offspring of tuberculous parents, and it is highly improbable that anything more than this is ordinarily handed down. And when we add to this enfeeblement of the power of resistance, the constant exposure of the infant to the many sources of infection from the tuberculous mother, we perhaps fully account for the frequent development of the disease during the first years of life.

2. *Inhalation* of dust to the particles of which bacilli have become adherent is probably the most prolific source of infection. The dust becomes contaminated for the most part through the expectoration of consumptive persons, but the sputum itself, after drying, and becoming pulverized, may be carried by currents of air. The extent to which infection is possible through the medium of the sputum becomes apparent when we reflect upon the enormous prevalence of the disease and the incomprehensible number of bacilli which each tuberculous person is capable of producing. As estimated by Vaughan, one person in every 60, or 1,050,000, of the entire population of the United States is tuberculous, and Nuttall estimates that each tuberculous individual may throw off from one and a half to more than four billions of bacilli in 24 hours. Examination of the dust from the walls, floor, and furniture of apartments occupied by consumptives almost invariably reveals the presence of large numbers of virulent bacilli. The air of such apartments, especially after sweeping and "dusting," contains them. Guerard reports the occurrence of 541 cases in 248 dwellings in a single ward of the tenement district of New York city, in which there was a total of 663 dwellings. Tuberculosis has repeatedly been introduced and has become prevalent in regions which had previously been exempt from it, by the arrival of those affected with it. Health resorts are converted into hotbeds of the disease to a considerable extent by the infection of the original inhabitants. Our Indian tribes were free from tuberculosis until the arrival of the white man; a part of their present susceptibility must, however, be attributed to the adoption of civilized manners of dissipation.

The universal dissemination of the disease through the contamination of the air is largely attributable to carelessness. The danger of infection could be greatly diminished through proper disposal of the sputum. It appears, in fact, that the disease has become slightly less prevalent as a result of the more general dissemination of the knowledge that a tuberculous person is a source of danger to those who come into contact with him. The breath of the consumptive is not in itself infective, but in coughing and sneezing, even in talking (Flügge), these persons throw off fine particles of sputum so minute that they float in the air. The inspiration of these particles is a possible source of infection to another person. Flügge, indeed, regards this as a more positive means of transmitting the disease than the inhalation of dry dust.

3. *Ingestion.*—This mode of infection is usually considered with especial reference to the ingestion of the meat or milk of tuberculous cattle. But it is probable also that the bacilli often gain entrance to the alimentary canal through the contamination of other articles of food, for example through virus carried by flies or other insects. The danger of infection through the eating of tubercular meat is compara-

tively small, since it would be possible only when the bacilli were actually present in the flesh consumed and when they escaped destruction by the processes of cooking and digestion. Koch aroused much discussion by his statement at the British Congress of Tuberculosis, in 1901, that the danger of the transmission of bovine tuberculosis to man is very slight. He estimated the extent of infection by the milk and flesh of tuberculous cattle as hardly greater than that from hereditary transmission. The comparative frequency of primary intestinal tuberculosis in young children is attributed chiefly to the drinking of milk containing bacilli. It has been found that these may be present in the milk of an animal whose udder is perfectly healthy and in which the disease is dormant to such a degree that it can be recognized only by the tuberculin test. It should be remembered also that the grass bacilli closely resemble those of tuberculosis and cannot be distinguished by the usual staining test.

Demme records an interesting observation that illustrates the certainty of deglutition infection from the entrance of the bacilli of human tuberculosis. Four infants died in succession of primary tuberculosis of the intestine, under the care of a nurse who was suffering from tuberculosis of the jaw, with a fistulous opening into the mouth. It was found that the nurse was in the habit of placing the food for infants in her own mouth before giving it to them.

Deglutition infection is also a common form of autoinoculation among pulmonary tuberculous patients. Intestinal tuberculosis commonly develops during the late stages of the disease as a result of the swallowing of sputum.

4. *Inoculation.*—The disease is seldom communicated by direct inoculation; and when this does occur, the resultant infection usually remains localized. Any broken surface of the skin may become inoculated. It is therefore a not infrequent result of injury to the hands in post-mortem work (the post-mortem wart). It is seen also on the hands of those who handle and wash the clothing and other articles of the tuberculous patient. It is interesting to note, also, that such inoculation is by no means uncommon among farmers, butchers, and tanners who handle the meat and hides of infected cattle, and that veterinary surgeons have repeatedly been inoculated from diseased cattle. Inoculation has less frequently been produced through the piercing of the ears, tattooing, and the bite of a tuberculous person. It has repeatedly followed the rite of circumcision, the final act of which is the sucking of the wound; tubercle bacilli have been demonstrated in several instances, both in the wound and in the mouth of the operator. Inoculation of tuberculosis has been attributed also to sexual intercourse.

Predisposing Influences.—Inherited vulnerability of constitution is perhaps not of so great importance as it was formerly believed to be. The tendency since the discovery of the specific germ of tuberculosis has been to minimize the importance of heredity. It is generally stated, however, that a history of tuberculosis among the ascendants of the patient is found in at least 25 per cent of cases when the parents alone are considered, and in about 60 per cent when the grandparents also are taken into account. It is believed that the vulnerability of type is more certainly handed down by the mother than by the father. Some families

show a more or less continuous prevalence of the disease through five or six generations, but on the other hand it would be difficult to find a family which had passed through so many generations without acquiring the taint. So much depends upon the environments of the individual, his nutrition and habits of life, and above all upon exposure to infection, however, that the influence of heredity will probably remain indefinite. It has frequently been observed that children which have been removed from the parents and placed under good conditions have escaped the disease while those that remained with the tuberculous parent have become infected.

Individual Peculiarities.—The "phthisical habit" has been recognized since the time of Hippocrates, yet the disease is not uncommon among persons of robust frame and free from the white skin, blue eyes, transparent conjunctivæ, and winged scapulæ which are regarded as typical of tuberculous tendency. The long and flat, or narrow, thorax, with a straightness of the upper ribs and an obliquity of the lower, conditions unfavorable to the full expansion of the lungs, are not without influence. But the "scrofulous frame" is now regarded only as an indication of vulnerability. More important from an etiological standpoint, perhaps, is the tendency to catarrhal inflammation so often seen in these individuals, which Beneke attributes to imperfect development of the heart with hypertrophy of the whole arterial system, the pulmonary artery being relatively wider than the aorta and thus favoring increased intrapulmonary blood-pressure.

Environment.—The influence of environment becomes apparent not only in those individuals who live in an atmosphere charged with tubercular virus, but to almost as great an extent in those who are deprived of sunlight and fresh air. Animals allowed to run free in the open air after inoculation with tubercular virus sometimes recover, while those that are confined in a dark, damp atmosphere quickly succumb; and the same is true of human beings. It is due largely to the influence of environment that the disease is more prevalent in large cities than in the country, and among the poor rather than among those in comfortable circumstances. Bad food adds to the evil effects of bad hygiene.

Climate and Season.—Climate and season influence to some extent the development, and yet more the progress, of the disease. This influence is probably inferior, however, to that of the sunlight and fresh air, or the cold, wet, and temperature-changes that belong to all climates. Devitalized air—air that has been breathed over and over again in small, unventilated sleeping-apartments—undoubtedly exerts a powerful influence in the production of susceptibility to infection. The disease is less prevalent, it is in fact almost unknown, in a few sparsely settled mountainous or desert regions, but this is due probably more to the absence of the infectious agent or to the outdoor life of the inhabitants than to the climate. A region which proves beneficial to strangers will not always confer immunity upon its inhabitants. Many regions have from time to time been pronounced free from the disease, but at the present time the ratio of cases, to the population of any district is very nearly the same in all parts of the world.

Age.—The influence of age is recognizable rather in the tendency to the involvement of certain structures than in the general susceptibility

to infection, for no age is exempt from the disease. During infancy and childhood the bones, lymph-glands, meninges, and intestines are more frequently attacked. From the fifth to the tenth year, which is usually the period of greatest outdoor activity, there appears to be a lull in the development of the disease. From 15 to 40 we see the pulmonary, pleuritic, laryngeal, and peritoneal forms, and in more advanced life fibroid phthisis.

Sex.—Women are somewhat more frequently attacked than men. But, aside from the influence of indoor life common to women and the probable influence of pregnancy and lactation in lowering the power of resistance, there is little or no difference in the susceptibility of the sexes.

Race.—Of all races, the Hebrew is the most nearly immune. The Irish are extremely susceptible. Among negroes in our country the disease is becoming more prevalent and more fatal.

Occupation.—Those constantly confined to small, dark rooms and to a sitting posture, as tailors and shoemakers, or those much exposed to the vicissitudes of the weather are more frequently attacked than others. Occupations which necessitate the inhalation of dust, as those of grinders and polishers, stonecutters and coal miners, predispose by the constant irritation of the respiratory passages.

Alcoholism.—Chronic alcoholism lowers the resisting power and is an especially potent factor in those whose constitution, environment, and occupation are already favorable to infection.

Disease.—Bronchial catarrh is one of the most frequent conditions precedent to tuberculosis. The sequence of whooping-cough, measles, or influenza, bronchitis, bronchopneumonia, and tuberculosis is often observed. A catarrhal condition of the pharynx or tonsils favors infection, especially in children. Smallpox and syphilis are also thought to increase susceptibility. And tuberculosis frequently bears the relation of a terminal infection to diabetes, valvular disease of the heart, aneurism, hepatic or renal cirrhosis, and other for the most part chronic debilitating affections. Cancer and tuberculosis, at one time thought to be antagonistic to each other, have more recently been found associated, not only in the same person, but even in the same organ.

Trauma.—Injury of the lungs, meninges, or bone, and more particularly of the joints favors the localization of the disease in them. And injury or operation on a tuberculous joint has not infrequently been followed by a dissemination of the disease and the development of miliary tuberculosis.

Morbid Anatomy of Tubercle.—After the tubercle bacilli have gained entrance into the tissues they rapidly multiply. The irritation of the tissues by the toxins results in the production of tubercles, small nodular granulomatous formations, not characteristic, but so common to this disease as to have given it the name tuberculosis. Tubercles are therefore a result rather than a part of the disease-process. The formation of a tubercle consists: (1) In the proliferation of cells from the endothelium of the blood- and lymph-vessels, perhaps also from epithelium, producing epithelioid cells, in which the bacilli may usually be seen; (2) around this an infiltration of leucocytes from the blood-vessels of the vicinity. (3) Giant-cells usually appear among the infiltrated cells, their number generally standing inversely to that of the bacilli present.

(4) A reticulum is formed from the fibrous tissue of the region and constitutes the external zone of the tubercle. Almost the entire process of tubercle-formation may be regarded as an effort on the part of the system to shut off the bacilli from the surrounding tissues. The nodule is not provided with blood-vessels, hence its nutrition is poor, and degenerative changes soon occur. The most common change is caseation, but sclerosis or calcification is not unusual.

Caseation begins at the center of the tubercle and invades the entire nodule. When several tubercles lie in proximity, it may extend to the entire mass. Calcification is a subsequent change and consists in the deposit of lime salts in a tubercle which has undergone caseation.

Sclerosis affects especially the outer zone, but the entire mass may be converted by it into a firm, fibrous, scarlike tissue. Caseation is a destructive process; sclerosis is constructive. The former tends to the formation of cavities; the latter to the limitation and final destruction of the tubercular process. The tubercle which is visible to the naked eye consists of a collection of small miliary tubercles, of microscopic size, or, as they are sometimes called, submiliary tubercles.

Tubercular Infiltration.—The entrance of bacilli is not invariably followed by the formation of distinct tubercles; for in some cases it produces a diffuse inflammation. Microscopic examination of the involved areas shows numerous non-vascular collections of cells without distinct nodular arrangement, the only separation being a round-celled infiltration. These large collections of cells may be the result of the coalescence of many smaller areas of infection. Coagulation necrosis soon follows their formation, and a large area of caseation (the so-called caseous pneumonia) is the result. This condition is most frequent in the lungs and the area affected may be small or large, involving only a few lobules or an entire lobe.

Distribution of Tubercles.—Tubercles may be found in every structure of the body except, perhaps, the teeth. The skin, subcutaneous tissue, the cancellous tissue of bone, and the mucous membranes, especially of the respiratory passages, but not seldom those of the alimentary and genitourinary tracts; the serous and synovial membranes and the pia mater are frequent locations. Among organs, the lungs are most frequently affected, but the liver, spleen, kidneys, testes, and lymph-glands are often attacked. The dura mater, ependyma, and endocardium are seldom affected. The brain, spinal cord, adrenals, and prostate are also among the less frequently affected regions, and the heart, salivary glands, pancreas, the mammæ, ovaries, thyroid, and voluntary muscles are among the rarest of all locations. The secondary changes are also to a great extent peculiar to certain regions. Caseation is most frequent in the lungs and lymph-glands; calcification is common in the lymph-glands, but less so in the lungs. Sclerosis is sometimes found in the pulmonary tubercles, but it is more common to those of the peritoneum.

From the original focus the tubercular virus is distributed: (*a*) Directly to the contiguous tissue or through the lymph-vessels; (*b*) in the lung also by aspiration, the infective material being drawn into bronchi which were previously unaffected; and (*c*) through veins and arteries whose walls have been infiltrated or perforated, often producing general miliary infection.

Miliary Tuberculosis (Acute) of the Lung.

The miliary tubercles, small and irregular in shape, are distributed throughout the lung—more abundantly in the upper and middle thirds.

The blood-vessels are injected with blue gelatin, so that in this photographic reproduction of the specimen the uninvolved portions of lung are dark, while the tubercles—in which the blood-vessels are compressed or obliterated—are light.

(By permission, from "Delafield and Prudden.")

Secondary Inflammatory Process.—The inflammation excited by the presence of the bacilli in the tissues is not always limited to the production of tubercles, for, beyond the nodular mass, there is frequently a proliferation of cells, with the production, in the lungs, of fibrinous or catarrhal pneumonia; or a proliferation of fibrous tissue and the production of so-called fibroid phthisis; in the blood- and lymph-vessels degenerative changes are often produced. Mixed infection is common, and the result is generally suppuration. A sterile cold abscess is not infrequently formed without the entrance of pyogenic micro-organisms, doubtless as a result of the irritation by the tubercle toxin.

1. ACUTE TUBERCULOSIS.

ACUTE MILIARY TUBERCULOSIS, GENERAL TUBERCULOSIS, ACUTE DISSEMINATED TUBERCULOSIS.

Definition. —A rapidly fatal acute tuberculous infection due to the dissemination of bacilli through the blood-vessels and lymphatics, with the production of countless miliary tubercles in various organs and tissues.

Etiology.—This form of the disease is almost always a result of autoinfection, often from a focus which is not recognizable during life. This focus is most frequently found in the lungs, pleura, lymph-glands, bones, joints, or kidneys; but nowhere more uniformly than in the tracheal and bronchial glands. Ponfick found it in the wall of the thoracic duct, and Weigert traced it in several instances to the perforation of a caseous bronchial gland into the pulmonary vein. A similar communication between a tubercular lymph-gland and a vein has been repeatedly demonstrated.

The disease occurs more frequently in children than in adults. In some cases the general infection is so sudden, particularly when it follows an acute infection like measles or whooping-cough in a previously healthy child, that it is not easily accounted for. A more or less prolonged attack of bronchial catarrh very often intervenes between the two infections.

Morbid Anatomy.—The tubercles may be so uniformly distributed that almost every organ and tissue of the body is involved; as a rule, however, they are more numerous in some regions than in others. The lungs, bronchi, liver, spleen, kidneys, and lymph-glands, the pleura, pericardium, peritoneum, and meninges are commonly affected, sometimes also the choroid coat of the eye, the bone marrow, especially that of the sternum, ribs, and vertebræ. The tubercles are for the most part small, from 1-500 to 1-250 inch in diameter. They are usually discrete, but they sometimes form large aggregations distinctly visible to the naked eye. They do not show secondary changes, as a rule, for the progress of the disease is so rapid that there is no time for such changes. (See Plate II.)

Symptoms.—Acute general toxemia is the most striking feature of the disease. There is sometimes a predominance of symptoms on the part of the lungs, cerebral meninges, peritoneum, or other structure, corresponding to an equally predominant invasion of these regions by the tubercles and giving rise to more or less distinct forms of the dis-

ease, as the pulmonary, meningeal, peritoneal, etc. They all belong to the same general infection of the system, however, and the local lesions merely add a few special features to the general symptomatology.

(1) *General or Typhoid Form.*—The invasion is generally slow, often so similar to that of typhoid fever as to lead to a suspicion of that disease. After gradually increasing malaise, headache, loss of appetite, constipation, and perhaps chilliness, the temperature gradually rises to 103° or 104° F. (39.5°—40° C.); prostration rapidly ensues, and anemia and emaciation soon follow. The pulse becomes accelerated, often to 140 or more, and the respiration is rapid and labored, often from 60 to 80 in children. Cheyne-Stokes respiration often develops toward the end. The cheeks are flushed, and the face often becomes dusky. The tongue is dry, often brown; and delirium of a quiet, muttering type may early develop. There is usually a slight cough, due to bronchitis. In some cases, the onset is more sudden. The irregularity of the fever is a distinctive feature of the condition. The morning remission usually amounts to 2° or 3° F. (1°—1.5° C.), but occasionally the fever is intermittent and the temperature in the morning may be subnormal. On the other hand it is not uncommon to find the morning record higher than that of the evening. Rarely there is very slight fever throughout the disease, and afebrile cases have been described. Albumin and pepton are found in the urine. Sudamina are frequent and an eruption of herpes is often found upon the lips. As the disease progresses, the patient sinks into a stupor; diarrhea may develop, with involuntary evacuations, and cyanosis often becomes extreme. Occasionally, however, the mind remains clear to the end.

(2) *Pulmonary Form.*—In this form, symptoms indicating the especial involvement of the lungs are added to those just described. The cough is more annoying; it has often existed for several months before the acute onset. There is usually a mucopurulent expectoration, sometimes containing traces of blood. Hemoptysis occasionally occurs. Dyspnea develops early, and cyanosis may be a prominent feature from the start; the blueness of the lips and nails is often striking. The physical evidences of pulmonary involvement are not so great as might be anticipated. Areas of distinct dullness are exceptional. The percussion note may seem to lack resonance, but this condition is not confined to any region of the chest, and there is no means of comparison. In children a slight dullness may be detected at the base of the lung, or areas of increased or slightly tympanitic resonance may suggest the presence of solidification in other areas. Auscultation usually reveals sibilant or subcrepitant râles. Tubular breathing may also be heard. The spleen usually becomes enlarged toward the close of the disease.

Diagnosis.—The leading points of differentiation in the general form of the disease are the absence of localized lesions, the irregular temperature, rapid pulse and respiration, marked dyspnea, possibly cyanosis, and the rapid progress of the disease.

In *typhoid fever* the temperature is more regular, the respiration less rapid and free from marked dyspnea or cyanosis. Epistaxis frequently occurs in the beginning, and diarrhea is more frequent. The diazo reaction is common to both diseases, but the rose-spots are very rarely seen in tuberculosis, and when seen they do not occur in successive

crops and are not typical in form. Herpes is more frequent in tuberculosis, and the tubercles may be found in the choroid. The lesions of typhoid fever and tuberculosis may, however, be found in the same person. The Widal test is the most valuable means of differentiation in a doubtful case. The presence of leucocytosis favors tuberculosis, but depends upon the existence of suppuration in either disease.

In the pulmonary form the diagnosis is usually rendered less difficult on account of the tuberculous history in the family, the existence of a cough before the present illness, possibly also a recent attack of measles, whooping-cough, or influenza; and the bacilli may often be demonstrated in the sputum.

Malaria.—It is only the remittent form that resembles tuberculosis. The temperature is more regular, as a rule; but the greater enlargement of the spleen and the presence of the plasmodium in the blood are more reliable features.

Cerebrospinal Meningitis.—In this disease the pulse and respiration are usually less rapid, the onset is more sudden, and the nervous manifestations, hyperesthesia, nystagmus, and disturbance of the reflexes are more prominent. Kernig's sign is not present in acute tuberculosis unless the meninges are distinctly involved.

Capillary bronchitis may simulate acute tuberculosis in the beginning, but the pulse and respiration are not so rapid; dyspnea is less marked and the cough more troublesome. The fever does not usually become so high, and the prostration is not so great.

2. ACUTE MENINGEAL TUBERCULOSIS.

Basilar Meningitis, Acute Hydrocephalus.

Definition.—A form of acute tuberculosis in which the cerebral meninges are especially involved.

Etiology.—Fully 50 per cent of all cases of miliary tuberculosis affect the meninges. The causes of this form are therefore the same. It is seen much more commonly in children between the ages of two and seven, but may occur at any time of life. It usually follows an involvement of the bronchial, mesenteric, or other lymph-glands or of the middle ear, and there may be a history of trauma. In some cases, however, the affection of the meninges appears to be the primary lesion. It has been suggested that the bacilli may reach the meninges in such cases through the cribriform plate of the ethmoid. The ten cases of acute tuberculosis, mostly of the meninges, reported by Reich are of interest in this connection, since the disease followed the mouth-to-mouth resuscitation of stillborn children by a tuberculous midwife.

Morbid Anatomy.—The membranes of the base are primarily and chiefly involved, but the tubercles may extend to all parts of the brain. The parts about the optic chiasm, along the larger blood-vessels and nerve-trunks, and over the tempero-sphenoidal lobes are especial points of attack. Less frequently the lesions are found on the convexities of the hemispheres. The pia is intensely hyperemic and the blood-vessels are all engorged. The walls of the vessels are not infrequently invaded by the tubercles, and thromboses sometimes result. The surface of the

pia is usually covered with a turbid, viscid, serous or fibrinopurulent exudate, and miliary tubercles are more or less profusely distributed over the affected surfaces. The lateral ventricles usually become distended with fluid, sometimes amounting to several ounces (acute hydrocephalus), and as a result the hemispheres may become flattened. The ependyma may be softened and the septum lucidum and fornix may be broken down. The brain substance becomes edematous and infiltrated with leucocytes. Red softening, rarely white, and punctiform hemorrhages are found. The association of acute and chronic tuberculosis of the meninges has been observed. In some cases the spinal meninges, especially in the cervical portion, have been found extensively involved. The other structures of the body are always involved in the disease.

Symptoms.—There is generally a history of tuberculosis in the family and of a recent attack of measles or other acute infection, followed during several weeks or months by a gradual decline of health. During the week or two preceding the acute onset of the affection there is often a complete change in the disposition of the child. It has become peevish, fretful, perhaps quarrelsome; the appetite has been lost and emaciation has become apparent. The course of the disease is divided into three stages, embracing a period of nervous excitement, a transition, and a stage of paralysis.

(1) *Stage of Excitement.*—The onset is usually gradual, often with a basilar headache, which increases in severity until it becomes agonizing. Persistent vomiting without regard to the ingestion of food is a marked feature, and there is usually moderate elevation of temperature, seldom exceeding 102° or 103° F. (39.0°—39.5° C.). At first on account of pain, but later on account of contraction of the cervical muscles the head sinks into the pillow. Very often the child grasps its head between its hands as if in great pain. Every now and then a loud shrill cry, known as the hydrocephalic cry, is uttered. In some cases the child screams continuously for days, or until the voice is lost from hoarseness and exhaustion. Obstinate constipation is a characteristic symptom, but diarrhea sometimes occurs in young infants.

The course of the fever is irregular. The evening record frequently exceeds the morning by 3° or 4° F. (1.5°—2.2° C.). The pulse, at first rapid, becomes slow and feeble; generally irregular. The respiration may be little disturbed. The sleep is restless and may be disturbed by muscular twitchings or nervous starts, and the child often awakes in terror. The pupils are usually contracted during this stage.

Occasionally the onset is more violent with, perhaps, a convulsion, rapid rise of temperature, and maniacal delirium, sometimes leading to a fatal termination within a few days. These cases are more frequently encountered in adults or in children who have been for a long time tuberculous. They are as a rule associated with involvement of the convexity of the brain. Cases are also encountered in which the disease pursues a more chronic course, marked by psychical disturbances of a type that may arouse suspicion of a brain tumor. Convulsions and paralyses do not appear until a late period.

(2) *Transitional Stage.*—The symptoms of irritation subside and there may be a deceptive promise of recovery. The vomiting ceases and the headache is no longer complained of, but the child remains dull

and listless. It may even become delirious at night. The constipation persists, and the abdomen becomes retracted and boat-shaped (scaphoid). The temperature is variable, but seldom exceeds 102° F. (39° C.). The pulse is still irregular and the respiration is often broken by sighs. The retraction of the head persists, and opisthotonos is not uncommon. The hydrocephalic cry is occasionally uttered. The pupils are dilated or uneven; one large, the other small. Strabismus or ptosis may develop from paralysis of the extrinsic muscles. Tubercles may be found in the choroid coat of the eye. Livid spots of considerable size may appear in the face, and a red line (tache cérébrale, or Trousseau's mark) appears in the skin after the finger-nail has been drawn over it, but this is not characteristic of the disease. Convulsions sometimes occur, or the muscles of one side or of a single member may become either rigid or paralytic. A tetanic spasm sometimes seizes a single limb, and it may persist for several days. Choreic movements and tremors are not unusual.

(3) *Stage of Paralyses.*—A progressively deepening coma supervenes until the child can no longer be aroused. Convulsions may still occur; and when the meninges of the cortical motor area are involved, the seizures may assume an epileptiform character. Spasmodic contractions often occur in the muscles of the neck and back, or they may be confined to the arm and leg of one side. Paralyses then develop. They may be either monoplegias or hemiplegias, the latter depending, as a rule, upon involvement of the cortical branches of the middle cerebral artery or upon softening in the internal capsule. Facial paralysis is the most common form of monoplegia. It is sometimes associated with paralysis of the extremities, the parts supplied by the third nerve, and the hypoglossal nerve of the opposite side. This association of paralyses is known as the syndrome of Weber. The lesion is in the lower, inner part of the crus. Optic neuritis may also be found. The pupils again become contracted, the eyelids remain partially open, and the globe is rolled upward.

Toward the close of the disease the temperature often becomes subnormal; in exceptional cases as low as 93° or 94° F. (33.0°—34.0° C.); occasionally, however, there is, shortly before death, a rapid rise to 106° or even 110° F. (41.0°—43.0° C.). The pulse becomes rapid, and the child sinks into a typhoid state with a dry tongue and low delirium. Leucocytosis is not infrequently present throughout the course of the disease. The duration of the disease is from two or three to four weeks, seldom longer.

Diagnosis.—The diagnosis is seldom difficult when the character of the invasion and the distinctive features already referred to are fortified by a history of tuberculosis in the family or the presence of a tuberculous lesion in another part. But in young infants many other disturbances, as a gastroenteritis, may excite rapidity of the pulse and respiration or convulsions. The train of symptoms is, however, entirely different. In gastroenteritis, diarrhea is present and the fontanels are depressed; they are usually prominent in meningitis. The hydrocephalic cry and irregularity of the pulse and respiration are absent.

Lobar pneumonia in young children is often mistaken for meningitis, but less frequently, perhaps, for the tubercular form of it. In it the onset

pia is usually covered wi… and paralytic mani-
exudate, and miliary tub…
the affected surfaces. T… may cause meningeal
with fluid, sometimes … stive of meningitis; the
alus), and as a result … Puncture of the drum
ependyma may be soft… establishes the diagnosis.
broken down. The bra… the tubercular form is
with leucocytes. Red … cases, but especially by
rhages are found. The … n the fluid obtained by
the meninges has bee… may be obtained in it. In
especially in the cervi… creased, but the withdrawal
The other structures … of the tubercular form of

Symptoms.—There … ably fatal. Recovery is so
and of a recent ac…
during several week… been reported the diagnosis
the week or two p… however, a case has been
a complete change … the demonstration of the
fretful, perhaps qu… fluid. Calomel was the only
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stages, embracing … attempt more than the relief
stage of paral… should be placed upon a soft
(1) *Stage of* … should be disturbed as little as
basilar headache … lessness sodium bromid, gr.
Persistent vom… (0.1—0.2), may be given to a
feature, and th… the meningeal form, opium in
exceeding 101° … codein, or morphin should be
pain, but late… nervous manifestations. It may
head sinks into … the rectum and drugs hypo-
its hands as … allable or the delirium active.
known as the … The constipation may be
screams contin… repeated doses of calomel,
and exhaust… cent magnesium citrate is gen-
but diarrhea … should consist chiefly of milk with

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…SERCULOSIS.

…LYMPHATIC SYSTEM.

…AR LYMPHADENITIS, SCROFULOSIS.

… more common between the first
… or adult life. It may occur,
… the bacilli gain entrance through the
… bronchial tubes, or intestine,
… ment. The condition is therefore
… mucous membranes, and more
… a tubercular tendency, both of which
… Rachitic children are also readily
… follow the inhalation of dust. It
… cough, measles, or influenza.

Morbid Anatomy.—The enlargement of the affected glands is due chiefly to an increase in the number of their lymphoid cells. These cells also appear swollen and their nuclei are large. Miliary tubercles, sometimes containing giant cells and bacilli, are also found in the glands. When the process is acute, the glands occasionally become hyperemic; suppuration is unusual. Later the tuberculous tissue may undergo caseation and calcification. When suppuration occurs it is usually without the presence of pyogenic organisms, and the pus is sterile. In the neck a fistulous opening sometimes forms through the skin for the evacuation of the pus. The bronchial glands often become enormously enlarged. Death may result from perforation and evacuation of their contents, after suppuration, into the trachea or bronchi; from compression of the esophagus; from erosion of the pulmonary artery or aorta or, more remotely, from rupture into the mediastinum or pleural cavity. The bronchial glands are probably the most frequent source of the acute dissemination of miliary tubercles.

Symptoms.—(1) *General Adenitis.*—This form is characterized by a more or less general enlargement of all the lymph-glands of the body. It is most frequently seen in the negro and especially in connection with pulmonary tuberculosis. The condition is usually accompanied by high fever and often runs a rapidly fatal course. It frequently resembles Hodgkin's disease. In infants and children a general adenitis is encountered in which one group of glands is involved after another, ultimately terminating in a fatal invasion of the meninges.

(2) *Cervical Adenitis.*—This form is the most common in children. The swelling involves the submaxillary and anterior cervical glands, less frequently the posterior cervical. It is generally unilateral in the beginning, but both sides are not infrequently affected. The individual glands are much enlarged and firm. In size they vary from as small as a pea to as large as a walnut. They are not usually painful or sensitive to pressure. Large masses may be formed, which entirely obliterate the lines of the neck. It was probably from this deformity that the disease received the name scrofula, from *scrofa*, a pig.

Coryza and acute nasopharyngeal catarrh are common accompaniments of the adenitis in these cases, and any influence which aggravates the catarrh tends to increase the enlargement of the glands. Moderate fever is often associated with the acute catarrhal symptoms. Slight inflammation of the glands may occur and ultimately pass into suppuration. Anemia soon appears and emaciation follows in most cases. The general condition is poor; wounds heal slowly, and there is a tendency to conjunctivitis or keratitis, otitis and eczema. After the disease has affected for some time the cervical glands there is frequently an involvement of the supraclavicular, axillary, and bronchial, and this extension of the disease is usually followed by pulmonary tuberculosis. On the other hand, the condition may terminate in recovery after months or years. It is an interesting fact that persons who show scars of early tubercular adenitis in the neck are seldom the victims of pulmonary tuberculosis in later life.

(3) *Tracheobronchial Adenitis.*—Enlargement of these glands ordinarily produces cough, dyspnea, or asthmatic seizures. Auscultation sometimes reveals the evidence of compression to the extent of a roughened inspir-

...ration. A venous purr may ... the upper portion of the ster... back. Extreme enlargement ... with more or less complete ...rticula have also been attrib... or lung by direct extension ...sis, as a result of the rupture ...op at any time. The same ...foration. Bronchopneumonia

... tuberculous adenitis produces ...senterica. In it the mesenteric ...ged and indurated and often ...tion, and very rarely suppura... the infection occurring from the ...commonly secondary to other ... a child, becomes anemic and ... wrinkled and the hair falls out. ... may be attributed to the dis... feature. Diarrhea, with large, ... and the abdomen is tympanitic. ... when meteorism is not present ... through it. The peritoneum ...gement of the mesenteric glands ...berculosis.

... disease is rarely difficult of rec... enlargement, and tendency ... doubt of the character of the ...ntiated from syphilitic, lymph... adenitis.

... and epitrochlear region are ... Suppuration rarely occurs ...syphilitic treatment promptly

... The glands are more movable, ... or undergo caseation. It is ... disease affects groups of glands; it ... rare in the submaxillary or

... may resemble that of tuber... by the profound anemia and

... is much more rapid and the ... disease.

... accessible and those nearest the

... the treatment of general ... the chief ... of ... has ... and ... administration

of sirup of the iodid of iron often assists in reducing the swelling. Cod-liver oil is especially useful in children. Careful removal of the glands has been performed with benefit, but it has undoubtedly been the means of disseminating the virus. It should be attempted only when rapid increase of size or suppuration threatens to produce rupture of the glands.

2. TUBERCULOSIS OF THE SEROUS MEMBRANES.

(1) General Serous-membrane Tuberculosis.

This form of the disease may be only a part of an acute miliary tuberculosis, or it may be an independent form of tuberculous infection, without visceral involvement. The serous membranes may become involved simultaneously or, as is more generally the case, in rapid succession. (*a*) The disease may be acute, the infection being derived from the bronchial or mediastinal lymph-glands, or in women from the Fallopian tubes. The pleuræ and peritoneum are generally involved. (*b*) A chronic form occurs also in which there is exudation and the formation of cheesy masses in the pleuræ and peritoneum, and it is not infrequently accompanied with inflammatory and suppurative processes. In extremely chronic cases the tubercles become hard and fibrous, the membranes become much thickened, and little or no exudation occurs. The pericardium may be implicated in either of these forms of the disease.

(2) Tuberculosis of the Pleura.

Etiology.—Fully one-third of all cases of acute, nontraumatic pleurisy are of tubercular origin. Some authors regard all such cases as of this character, since a very large number of them respond to the tuberculin test and others are followed by pulmonary tuberculosis a few months or possibly several years afterward. But on the other hand there can be no doubt that many acute cases entirely recover. The condition may be: (*a*) Primary and independent of other tuberculous infection; (*b*) a part of an acute miliary tuberculosis; (*c*) secondary to the ordinary form of pulmonary tuberculosis, or to a tubercular process in the cervical or bronchial glands, or the bodies of the vertebræ; or (*d*) it may be a part of a general involvement of the serous membranes. Probably not all instances of the development of pleuritic inflammation in the course of chronic pulmonary tuberculosis are of a tuberculous nature, however, for they frequently leave only fibrous adhesions between the two layers, without discoverable tubercle-formation.

Symptoms.—The infection is generally unilateral and it may pursue an acute, subacute, or chronic course. Such prodromal symptoms as cough, mucopurulent expectoration, anemia, emaciation, sometimes an occasional hemoptysis, may precede either of the forms for many months, especially when the disease occurs in the course of chronic pulmonary tuberculosis.

Acute Form.—The actual onset of the pleuritic involvement is often announced by the occurrence of a sharp stitch in the side or by a distinct chill. An exudate quickly forms in most cases, and, partly at least as a result of it, there are cough, dyspnea, and moderate elevation of

temperature, usually from 101° to 103° F. (38.5°—39.5° C.). The exudate may be serous or serofibrinous in the beginning, but frequently becomes seropurulent, sanguinolent, or purulent as the disease progresses. A purulent change is invariably induced by tapping without proper antiseptic precautions. The most characteristic fluid is slightly greenish, opalescent, seropurulent, and contains granular fat and a few leucocytes. Tubercle bacilli and other bacteria may be present, or the fluid may be sterile. In many cases, after a course of from three to six weeks, the fever and pain subside and the exudate is gradually absorbed. In other cases the disease becomes chronic.

The *subacute* and *chronic forms* may follow the acute or they may develop so insidiously as to appear chronic from the first. After a long period of cough, emaciation, and loss of weight and strength, slight pleuritic pain may be complained of, or physical examination may first reveal impaired expansion of the affected side of the chest, with an accumulation of fluid in the pleural cavity, or dullness due to thickening of the pleura. The fluid may be serofibrinous or purulent. The thickening of the pleura varies from ¼ to 1 inch (0.5—2.5 cm.) and there is sometimes only a small quantity of a thick caseous fluid at the base of the cavity. The pleural cavity may, indeed, be almost completely obliterated through hyperplasia of the membrane and the formation of adhesions between the parietal and visceral layers. A most virulent type of empyema or pyopneumothorax is developed in these cases by the rupture of an abscess cavity or a softened caseous nodule in the lung. In the less fatal cases of primary tuberculosis of the pleura the lung may ultimately become involved or an acute miliary tuberculosis may be instituted.

Diagnosis.—The diagnosis of pleurisy is considered under the diseases of the pleura. The tuberculous character of the condition is sometimes determined with much difficulty if the disease be primary, but in the presence of pulmonary or other recognized tuberculous infection there is always a strong probability of its tubercular nature. The presence of blood or tubercle bacilli in the exudate or the discovery of bacilli in the sputum is highly confirmatory of the diagnosis. When no other means is sufficient for the determination of the condition, the successful inoculation of a guinea-pig with the aspirated fluid will establish the diagnosis.

Treatment.—This combines the general methods of treating tuberculosis and those for the relief of pleurisy.

(3) Tuberculosis of the Pericardium.

Etiology.—The pericardium is much less frequently affected than the other serous membranes. It is usually secondary to disease in other parts, perhaps never primary in character. It may be a part of a miliary tuberculosis or it may result from tuberculosis of the mediastinal or bronchial glands, or it may be a direct extension from disease in the pleura or lung. Either layer of the pericardium may be involved and the disease may be either acute or chronic, simple or purulent. The quantity of the exudate varies from a little more than normal to as much as 64 ounces (Musser). The disease is sometimes latent

and may be discovered only at autopsy. As in tuberculous pleurisy, the membrane may be greatly thickened and the layers of the pericardium may become more or less completely adherent. As a result of the latter condition hypertrophy and dilatation of the heart are produced, and valvular insufficiency often results from this change. A loud, blowing systolic bruit is often heard at the apex, corresponding in time to that of either a stenosis or an insufficiency.

Treatment.—The treatment consists in the application of the measures employed in a nontuberculous pericarditis and those for the general treatment of tuberculosis.

(4) Tuberculosis of the Peritoneum.

Etiology.—The condition is probably never primary in its origin. It may be secondary to tuberculous disease of the endometrium or Fallopian tubes, the intestine, or mesentery. It has also been traced to disease of the epididymis, the vesiculæ seminales, or prostate. It may be a part of a miliary tuberculosis, the bacilli reaching the peritoneum through the blood; or of a general tuberculosis of the serous membranes. It may result from the perforation of a tuberculous focus that has undergone softening or suppuration in any of the adjacent organs. Trauma of the abdomen is thought to have an influence in producing localization of the infection in the peritoneum. Cirrhosis of the liver and hernia are thought to favor its development. The disease may occur at any age, but is especially frequent in children, when it is usually a result of the extension of infection from the intestine or mesentery. Its occurrence in the two sexes is probably about equal, since it has been noted more frequently by the gynecologist than by the surgeon; but in men it is oftener found on the post-mortem table.

Morbid Anatomy.—The tubercles are usually numerous, studding the entire surface of the peritoneum; more rarely they are confined to a circumscribed area. The character and the quantity of the exudate vary considerably, and the other conditions correspond to these features. When the disease is a part of an acute tuberculosis, the peritoneum is studded with young, gray, translucent tubercles, smaller than pinheads. In a less acute form the tubercles show a tendency to become clustered into patches or nodular masses, and the peritoneum is often much thickened. It is also hyperemic in many cases, and covered with a layer of fibrin. The subsequent changes may be of a caseous, suppurative, ulcerative, or sclerotic character, with the production of a variety of lesions. The tubercles are often opaque, yellowish, and caseous, and the larger masses that are formed may undergo caseation or suppuration. The omentum is especially prone to involvement and often becomes shrunken into a dense roll. The mesentery also becomes greatly thickened by a fibrous increase and may draw the attached intestine into a firm, tumor-like mass. The tissues are frequently much indurated and pigmented. The skin also shows pigmentation in many of the more chronic cases. The exudate is generally abundant in the acute form, but it may be much or little in the chronic. In character it may be either serous, fibrinoserous, or hemorrhagic; it is not often purulent. The adhesions are numerous in chronic cases. They some-

times completely encapsulate the exudate, forming several isolated sacs. The tubercles may be concealed between the adherent surfaces. Large masses are sometimes formed which are readily mistaken on palpation for cancerous formations. The wall of the intestine is sometimes perforated within these circumscribed areas.

A localized tubercular peritonitis sometimes occurs, producing large caseous, often pigmented masses on the surfaces corresponding to the localization of tuberculous ulcers within the bowel, or to similar tuberculous formations in the Fallopian tubes; likewise on the inferior surface of the diaphragm when the pleura or pericardium is affected.

Symptoms.—The condition may be either acute or chronic: (*a*) *Acute Tubercular Peritonitis.*—In the very acute cases the onset may be sudden and violent, with intense pain, great tenderness, meteorism, and vomiting. Constipation is usually present, unless diarrhea is produced by the simultaneous presence of tubercular enteritis. These cases resemble acute enteritis, appendicitis, hernial strangulation, the perforation of a typhoid ulcer, or acute obstruction.

The course of the disease may be intermittent, attacks of severe pain alternating with intervals of almost complete relief. Fever is usually present, often reaching 103° or 104° F. (39.5° or 40.0° C.). With the formation of the exudate, the abdomen becomes more distended and the presence of fluid may be recognized by dullness and fluctuation. The other symptoms of acute general peritonitis—rapid pulse, distended, motionless abdomen, dorsal decubitus, elevated knees, and anxious, Hippocratic facies—are generally noted in acute cases.

In less acute cases the symptoms develop slowly and the pain is less severe. There may be a gradual rise of temperature, possibly to 103° F. (39.5° C.), and the clinical picture may be that of typhoid fever. In some instances the disease remains for a long time latent until the ascites causes it to be recognized. The exudate may become so abundant as to interfere with respiration and cause embarrassment of the portal and renal circulation. Subacute cases are particularly liable to pass into the chronic form.

(*b*) *Chronic Tubercular Peritonitis.*—This form corresponds to the caseous, suppurative, and sclerotic conditions referred to under Morbid Anatomy. The abdomen often becomes firm and indurated. The shrunken omentum and intestine may often be felt through the abdominal wall as large, tumor-like masses. The fever is usually slight or it may be absent; a subnormal temperature is not uncommon, the oscillation often being confined for days between 95.5° and 97° F. (35.2° and 36.2° C.). When, however, suppuration occurs, the temperature rises and pursues an irregular course. With the progress of the disease, anemia and emaciation become more and more pronounced, with corresponding loss of strength and reduction of weight. More or less complete occlusion of the bowel may develop at any time or the intestinal wall may become tubercular and diarrhea often sets in with bloody dejections. Intestinal perforation may take place and cause a sudden, violent exacerbation of the symptoms. Leucocytosis is generally absent in all forms of tubercular peritonitis.

Diagnosis.—The condition, especially when discovered at operation, is to be distinguished first from the non-tuberculous nodular growths

that are occasionally found upon the peritoneum. The differentiation can be made in many instances only by the demonstration of the bacillus tuberculosis or the tuberculin test. The condition is perhaps often mistaken for tubercular peritonitis. In the acute cases the differentiation is usually to be made from typhoid fever, appendicitis, strangulated internal hernia, and intestinal obstruction; and in chronic cases from ovarian cyst and other abdominal tumors with fluid contents, as well as from chronic peritonitis of nontuberculous nature just referred to.

Typhoid Fever.—This is excluded by the absence of epistaxis, enlargement of the spleen, or roseola, and the negative reaction of the Widal test.

Appendicitis.—In this disease the abdominal distention is more unilateral, the right rectus is tense, the right knee alone elevated; and the presence of a sausage-like tumor in the ileac fossa, with tenderness at McBurney's point, is distinctive. Leucocytosis is usually present.

Hernial Strangulation.—This condition or obstruction from any other cause develops suddenly; the patient may be free from suspicion of tubercular disease. The pain is localized and paroxysmal; tympanites is marked. The most valuable symptom is feculent vomiting, which generally sets in within a few hours.

Acute Enteritis.—This is liable to cause confusion only when it occurs in a tuberculous subject. The profuse diarrhea tends to reduce rather than to increase the abdominal distention, and there is seldom marked elevation of temperature or the typhoid state.

Abdominal Tumors.—The differentiation of these growths, of whatever kind, is to be made chiefly by a study of the etiology. In the presence of an evident source of tubercular infection, in the lung, genitourinary tract, or elsewhere, the condition is most likely to prove tubercular. Ovarian cyst is generally slower in growth, and malignant neoplasms more rapid, than tuberculous nodules. Febrile attacks and digestive disturbances are less frequently observed in connection with them.

Prognosis.—The chance of permanent recovery is exceedingly poor. Spontaneous recovery sometimes occurs in cases regarded as tuberculous, and laparotomy frequently gives relief. Cures have been claimed from it in from 70 to 80 per cent of cases, but relapse is the rule in cases of known tuberculous character, sometimes after months or years of apparent freedom from the disease.

Treatment.—The treatment is constitutional, symptomatic, and surgical. The former embraces the methods for general tuberculosis. Rest is essential. The food should be the most nourishing and as largely as possible of meat. Pain may be relieved by hot fomentations, poultices, ice-bags, the application of iodin or turpentine stupes; but opiates may be required. Constipation calls for laxatives; excessive diarrhea, for opiates and astringents. Guaiacol, iodoform, and salol may be used as intestinal antiseptics, and they possibly exert an influence on the disease. Withdrawal of the fluid by repeated aspiration under strict asepsis has proved efficient in some cases, but laparotomy is generally resorted to when other methods fail. In children the administration of sirup of the iodid of iron and the application of guaiacol in glycerin to the abdomen have sometimes proved beneficial.

3. TUBERCULOSIS OF THE RESPIRATORY ORGANS.

(1) Tuberculosis of the Nose.

The nasal cavities are rarely the seat of tuberculosis. It is not infrequent, however, to find the bacilli within the nostrils of healthy individuals, and this fact probably indicates that this mucous membrane is less susceptible to infection than that of the lower respiratory passages. When the disease occurs, it is usually secondary to tuberculosis elsewhere.

Symptoms.—The condition is one of catarrh, with more than the ordinary tendency to hyperplasia of tissue and the formation of ulcers. These in turn lead to the formation of large crusts and frequent epistaxis, especially upon removal of the accumulations.

The condition is usually a part of a late tuberculosis and not, therefore, of long duration. The treatment consists of the removal or destruction of the nodes and ulcers, but as the disease is attended with little or no pain the adoption of painful measures can hardly be advised.

(2) Tuberculosis of the Larynx.

Etiology.—The disease is rarely primary in character, and, when it is so, it probably originates from inoculation with bacilli in the inspired air. As a secondary infection it is not uncommon, especially as a late complication of pulmonary tuberculosis. The infection may originate from the sputum which passes over the larynx from the lungs or it may be conveyed through the blood-vessels or lymphatics. A lesion is necessary to permit the entrance of the bacilli, and such a lesion may no doubt be produced by violent coughing.

Morbid Anatomy.—The mucous membrane becomes swollen, particularly over the arytenoid cartilages, and tubercles form in it, at first in the vicinity of the blood-vessels. Hyperemia does not, however, appear until comparatively late. The tubercles group themselves into a few small nodules, as a rule, then undergo caseation and break down, leaving shallow ulcers of unequal size and irregular shape. These are situated, in most cases, over the arytenoid cartilages, on the vocal cords, and on the epiglottis. The adjacent mucous membrane becomes thickened, especially over the arytenoids. Later the ulceration may lead to the total destruction of the vocal cords and epiglottis and the development of perichondritis, sometimes with exfoliation of cartilage. The disease shows a tendency to spread in all directions. It may extend laterally and upward to involve the fauces, pharynx, and tonsils, or downward over the cricoid cartilages. Stenosis of the larynx is one of its infrequent results, being produced by the contraction of cicatricial tissue following the ulceration.

Symptoms.—The involvement of the larynx is usually announced by a huskiness of the voice that increases to a decided hoarseness and in extreme cases to complete aphonia. Deglutition and phonation become difficult and painful. The cough is at first not very severe, but later, when ulceration has occurred, it becomes extremely troublesome, hoarse, and ineffectual, often paroxysmal. Dyspnea is a frequent accompaniment of the condition. When the epiglottis becomes exten-

sively ulcerated, swallowing is correspondingly difficult. Food often enters the larynx and causes paroxysms of coughing and threatened suffocation. Laryngoscopic examination reveals a characteristic thickening and pallor of the mucous membrane, or, later, an extensive destruction of tissues. The ulcers are shallow, have an irregular outline, and the base is usually covered with a gray, necrotic exudate. The vocal cords are thickened, usually ulcerated.

Diagnosis.—The disease is to be distinguished chiefly from catarrhal and syphilitic laryngitis. Catarrhal laryngitis may occur in a tuberculous patient. The differentiation is often difficult, since the presence of bacilli may be only accidental. Ulceration is seldom so extensive, and the peculiar pallor is not usually present. From the syphilitic form the tubercular is to be differentiated chiefly by the history of the case, the presence of tubercle bacilli, and the absence of general glandular enlargement.

Prognosis.—The prospect for cure is not only bad in all cases, but the development of laryngeal tuberculosis in the course of the pulmonary disease adds an element of great gravity to the case.

Treatment.—The general treatment is that of the underlying pulmonary tuberculosis; nothing is so important as fresh air and light, with tonics and nutritious food.

Local Treatment.—The ulcers should be kept clean with an alkaline spray. Astringents may be applied in the same manner. A solution of menthol and camphor gives great relief. Creosot should sometimes be added to the solution. In advanced cases cocain may be required, particularly at mealtime, to prevent the pain of swallowing. Insufflation of iodoform after thorough cleansing is thought to promote healing. Extensive ulceration requires the care of a specialist who may be able to improve the condition by curetting and applying caustics, silver nitrate or lactic acid. After destruction of the epiglottis the patient is able to take nourishment only in fluid form, sometimes by sucking it through a tube with the face downward, but generally only through the stomach-tube.

(3) Tuberculosis of the Lungs.

PULMONARY TUBERCULOSIS, PHTHISIS PULMONUM, CONSUMPTION.

Infection of the lungs arises most frequently from the inhalation of bacilli-laden dust; less frequently through the blood- or lymph-vessels. The conditions developed are not identical. When the bacilli have entered through the bronchial tubes, the primary lesions are usually found in the smaller bronchi and bronchioles. Their walls become infiltrated with granulation cells and surrounded by layers of lymphoid and epithelioid cells forming peribronchial granulations which are often found in a state of caseation. The lumen of the tubes becomes closed by a caseous mass of desquamated epithelium. The condition is not confined to single groups of alveoli, but affects more distinctly lobules, sometimes even an entire lobe.

When the bacilli reach the lungs through the blood or lymph, the primary lesions are located in the walls of the alveoli, capillary vessels, or the connective tissue of the interalveolar septa. Tubercles of the

miliary type, small, microscopic collections of cells, are formed within a few days, but soon coalesce to form larger masses. The subsequent changes in them are the same as have been previously described under the head of Morbid Anatomy of Tubercle, on page 175. The condition may be either localized or general throughout the lungs. When the disease is a part of a general tuberculous infection, the tubercles are found in all parts of both lungs. When the process is localized, it is generally confined to the apex of one lung; a little more frequently (about as 7 to 5) in the left; next most frequently in one lower lobe, near the base or in the middle portion, as a rule. From the area originally involved the disease gradually extends until it invades the greater part of both lungs. The mode of extension is either: (*a*) Directly to contiguous tissue, through which numerous foci are frequently merged; (*b*) by transmission through the blood-vessels or lymph-channels to more distant parts of the lungs; or (*c*) by the inoculation of other parts from the sputum in its passage outward or through its aspiration into adjacent lobules.

There are three principal forms of pulmonary tuberculosis, the acute pneumonic, the chronic ulcerative, and the fibroid.

(*1*) *Acute Pneumonic Tuberculosis.*

PNEUMONIC PHTHISIS, CASEOUS PNEUMONIA, PHTHISIS FLORIDA, GALLOPING CONSUMPTION.

Of this disease there are two forms, the pneumonic and the bronchopneumonic, distinguishable clinically and by their pathological lesions.

(*a*) *The Pneumonic Type.*—The process may be confined to a single small area, perhaps to one apex, but it may involve an entire lung. It is usually rapid in its progress. The affected area becomes solidified, heavy, and airless, much as in lobar pneumonia. The solidification is due to the infiltration of the alveolar septa with serum and leucocytes and the filling of the air-cells with an exudate consisting chiefly of proliferated and desquamated epithelial cells. The entire area subsequently undergoes caseation, and small cavities are formed, except in the more rapid fatal cases. The infiltrated area may appear so nearly homogeneous on post-mortem examination as to render the recognition of the tubercles quite impossible without the microscope. As a rule, however, the character of the lesions will be revealed by the finding of the more chronic changes in other parts of the lungs. The bronchial glands are always enlarged, and the pleura over the affected areas is covered with fibrin or caseous matter. The surrounding portions of the lungs may be hyperemic, but there is never any evidence of a tendency to resolution.

Symptoms.—The disease may develop insidiously in a person who has been debilitated by illness, alcoholism, or overwork and exposure, or it may be announced by a chill. It sometimes occurs in individuals in apparently good health. A severe cough develops, the sputum becomes mucopurulent, sometimes "rusty," and often contains bacilli and later elastic tissue from the disintegrating lung. The temperature rapidly rises, perhaps to 103° or 104° F. (39.5°—40° C.). There is pain

in the side. Sweating usually occurs at frequent intervals, especially at night. The pulse is accelerated, the breathing becomes rapid, and dyspnea may become urgent. Physical examination reveals consolidation, indicated by dullness, increased fremitus, absence of vesicular murmur and tubular breathing. The urine may show the diazo reaction. The condition is almost identical with that of lobar pneumonia, but the crisis does not occur and the condition becomes progressively worse. The rusty sputum becomes changed into the "prune-juice" expectoration, the prostration becomes extreme, the feet become edematous, and cyanosis may develop. Death sometimes occurs as early as the second or third week, but it may be delayed as long as two or three months. Occasionally the acute symptoms subside and the case progresses as one of chronic tuberculosis.

Diagnosis.—When the disease occurs in one who has not previously been the subject of recognized tuberculosis, the differentiation from lobar pneumonia may be for a time impossible. The condition may not be suspected, in fact, until the disease is found to be growing worse instead of undergoing resolution after the period of the expected crisis has passed. By this time, however, it may be possible to demonstrate the bacilli in the sputum. The occurrence of repeated chills or frequent chilly sensations, and more particularly the character of the temperature curve, may arouse suspicion, for the oscillations are usually greater than in lobar pneumonia, often amounting to 2° F. (1.1° C.).

(*b*) *Bronchopneumonic Type.*—This form of the disease is more common than the pneumonic, and is met with especially as a sequel to the bronchitis of measles, whooping-cough, diphtheria, or other acute infection in children who are predisposed to tuberculosis. Not infrequently, indeed, the disease is the rekindling of a tubercular process already existing in the individual. The process begins in the smaller bronchi, which become filled with a cheesy accumulation of desquamated epithelium. As a result, the alveoli are closed, and a catarrhal pneumonia is virtually established. In the beginning the affected areas are hyperemic, but later they become opaque and caseous. The consolidation is usually confined to more or less isolated areas between which the lung still contains air, but in extreme cases an entire lobe may become almost solidified. The bronchial glands are usually much enlarged. In another class of cases the affected areas are small and confined to different parts of both lungs, generally to the apices. A similar condition sometimes results from the aspiration of blood and the contents of tubercular cavities into the unaffected portions of the lungs during a hemoptysis. Mixed infection sometimes occurs when other organisms gain entrance, but in many cases the bacillus tuberculosis alone is found.

Symptoms.—In children the disease frequently engrafts itself upon a bronchial catarrh without for a time exciting unusual symptoms further than a prolongation of the condition. On close observation, however, the child will be found to have a fever, with flushed cheeks toward evening (hectic fever). The breathing becomes more rapid and the expectoration more abundant. Emaciation also develops with much rapidity. On physical examination slight dullness may be detected at one or both apices, occasionally in other parts of the lungs.

In other cases the onset is more abrupt. The child may be in an enfeebled condition from previous illness or it may be the subject of rickets or of unrecognized tuberculosis. There is a sudden rise of temperature and severe cough, with rapid solidification of one or both apices, and numerous subcrepitant râles are heard on auscultation. These cases sometimes terminate fatally within three or four days, and without microscopic examination of the lesions there may be nothing to indicate the tubercular character of the bronchopneumonia.

In adults the disease may attack an individual in good health, but it is much more frequently observed in those who have been debilitated or those who are the subjects of tuberculosis. The attack generally begins with a succession of chills or chilly sensations, followed by high fever, rapid pulse and respiration. Sometimes there is hemoptysis. Emaciation and loss of strength and weight rapidly follow. The physical signs in the early stage of the disease are only those of bronchopneumonia, but later the areas of dullness become distinctly recognizable. As the disease progresses, the fever becomes irregular, sweating is often profuse, and frequent chills may occur. The case may terminate fatally within three weeks, or it may gradually subside into a chronic condition lasting for several months.

(2) *Chronic Ulcerative Tuberculosis of the Lungs.*

Morbid Anatomy.—In the great majority of cases the lesions are first found in one or both apices, usually an inch or more below the summit. From this point the process passes rapidly or slowly downward. This extension of the disease is probably in part a result of the aspiration of the sputum or caseous matter into the bronchial tubes. The new growth of tubercles is then located in the tapering extremity of the terminal bronchus, near the entrance to the infundibulum. From the smaller bronchi the process may extend upward, however, to the larger tubes. The disease extends also directly from the affected center to adjacent tissue. It may follow the lymph channels, producing a chain of young tubercles radiating from the primary center, and it may be carried by the blood. Autoinfection no doubt occurs as the result of the inhalation of dried sputum from the patient's own clothing, or possibly from some other source. The disease thus progresses from one region to another until an entire lobe, an entire lung, or a greater part of both lungs becomes converted into a mass of tubercular tissue in different stages of growth and degeneration.

The processes of infiltration, caseation, and sclerosis have been considered under the head of Morbid Anatomy of Tubercle. As a result of these processes many different conditions are produced. Caseation leads to necrosis of the tissues and ulceration, with the formation of cavities. This frequently occurs in the wall of a bronchial tube. As a result, the wall is rendered thin and less resistant to the expansive force of the air in coughing. The walls stretch and form fusiform dilatations (bronchiectatic cavities). The further destruction of the tissues is often hastened by the entrance of pus-forming germs into these cavities. The necrotic destruction may begin, however, among the air-cells, particularly in the apex. Recent cavities, still in a state of formation, have uneven, ragged surfaces or they are lined with caseous débris. Such

Diffuse and Focal (Chronic) Pulmonary Tuberculosis—
"Chronic Phthisis."

In the upper third of the lung there is tuberculous broncho-pneumonia with commencing ulceration of small bronchi; nearly complete consolidation from the extension and coalescence of small tuberculous foci and diffuse formation of fibrous tissue.

In the lower third of the lung are irregular, dense, sharply outlined tuberculous foci (chronic miliary tubercles).

In the middle third there is tuberculous pneumonia of the exudative type, the incompletely consolidated areas having become, in part, caseous.

The less involved portions of the lung in this, as in the other injected specimens, are the darker.

(By permission; from "Delafield and Prudden.")

cavities may surround a small blood-vessel or a bronchus, and the end of a small bronchial tube sometimes protrudes into them. A blood-vessel thus surrounded becomes inflamed, and an endarteritis obliterans is developed, often completely closing the vessel. Old cavities are usually smooth and lined with firm (pyogenic) membrane, upon which pus is constantly formed. The largest cavities are generally a result of the coalescence of several smaller ones. In this manner an entire lobe, almost an entire lung, may be excavated.

When the tubercular process is situated near the surface of the lung, the pleura invariably becomes inflamed and later infiltrated with tubercles. A small cavity immediately under the pleura sometimes breaks through it and produces a pneumothorax, by permitting the escape of air. This accident is generally prevented, however, by the inflammatory process, which rapidly forms adhesions between the two layers of the membrane. This adhesive inflammation is sometimes so extensive that almost the entire pleural cavity of one side becomes obliterated. Very rarely a cavity evacuates its contents through the chest-wall.

Sclerosis is a reparative process, but it seldom results in the complete repair of the tubercular lesions, except, perhaps, in the earliest stage. In some other instances it separates the caseous masses from the surrounding tissue by a firm wall, and occasionally it closes small cavities after they have discharged their contents. Calcification may follow either caseation or sclerosis, but more frequently the former. Its occurrence does not always indicate an arrest of the tubercular process, for living bacilli may remain in the periphery of the calcified nodule. An awakening of the process with the destruction of surrounding tissue sometimes causes the discharge of small calcified masses in the form of the so-called lung-stones.

The bronchial glands are also implicated in all cases. In the more acute they become swollen and edematous; they are nearly always tubercular. Caseation occurs in them, and, in the chronic cases, calcification. Suppuration sometimes occurs.

Other Organs.—Important changes occur in other organs, especially the larynx, intestine, liver, spleen, kidneys, pericardium, and cerebral meninges. Many of the changes are tubercular in character. As a result, no doubt, of the toxemia and anemia, degenerations are common, especially in the organs just enumerated. The liver is the seat, also, of extensive fatty infiltration. Amyloid degeneration is of common occurrence in the more chronic cases and affects particularly the intestines, liver, spleen, and kidneys.

Symptoms.—*Mode of Onset.*—(*a*) In many cases the disease remains for a time latent. Considerable progress is often made by it before the infection is recognized. In some instances it advances to the formation of a cavity in one of the apices before the patient realizes his illness. In other cases the greater prominence of symptoms on the part of other organs may not only mask the pulmonary condition, but it may for a time mislead both the patient and his physician. The most common of these conditions are the tubercular affections of the bones and joints, caries of the vertebræ, ribs, sternum, or lymph-glands, lumbar and psoas abscesses, otitis, and anal fistula.

(*b*) *With Cough.*—In probably the greatest number of cases the

disease begins with the symptoms of bronchitis. The patient has perhaps suffered for years from a nasopharyngeal catarrh, with great susceptibility to "cold." These attacks grow more severe, or after some unusual exposure a severe bronchial catarrh develops. The sputum becomes mucopurulent, and there may be a slight elevation of the evening temperature. Dyspnea is often a noticeable accompaniment, and it sometimes assumes a paroxysmal form resembling asthma.

(*c*) *Anemia* is often one of the earliest symptoms. It is recognizable, however, by the cardiac palpitation, indigestion, or perhaps amenorrhea which results from it, rather than by the appearance of the patient. The blood usually shows reduction of hemoglobin, of 25 per cent or more, and leucocytosis, becoming more pronounced as the disease progresses. The red corpuscles are often normal in number, and the blood-plates greatly increased.

(*d*) *Dyspepsia.*—Many cases begin with an acid dyspepsia, characterized by eructations, vomiting, or pain and a sense of burning in the stomach. The connection of this symptom with the affection of the lung is probably often overlooked.

(*e*) *Hemoptysis.*—The existence of the disease is often announced by a more or less profuse hemorrhage of the lungs. Repeated hemorrhages sometimes occur. After a hemorrhage the disease sometimes remains for a long time quiescent, but in most cases the tubercular process advances with greater rapidity.

(*f*) *Pleurisy.*—The first symptom to attract attention in some cases is pleurisy, with dry friction-sounds over the apex. The disease may also follow a pleurisy with exudation. The cough persists after the effusion has been absorbed, and a localized tubercular process may soon become recognizable.

(*g*) *Chills.*—In some cases the onset is accompanied with chills, fever, and sweating not unlike those of malaria or sepsis. It is not uncommon to elicit a history of slight chills at a time corresponding to the beginning of the infection in cases that have been characterized by a greater prominence of other manifestations. The hectic flush of the cheeks in the afternoons soon becomes apparent in these cases, and a close watch of the temperature at short intervals will reveal an irregular curve quite unlike that of malaria, though more like that of sepsis.

(*h*) *With Laryngeal Symptoms.*—In a fairly large group of cases there is a history of periodic hoarseness or aphonia preceding the recognized onset of the disease. It is only in a small minority of these cases, however, that the larynx is the primary seat of the infection, and such manifestations disappear more or less permanently after the pulmonary disease has become far advanced.

(*i*) *Enlarged Lymph-Glands.*—Tubercular enlargement of the lymph-glands of one side of the neck, particularly those of the supraclavicular region, and often associated with enlargement of the axillary glands of the same side, has often been recognized for months or years before the development of recognizable lesions in the lungs.

Typical Course.—From the great variety of clinical pictures presented in this heterogeneous disease it is difficult to select one that can be called typical. Few cases are alike, yet there is a train of symptoms that are more or less common to a majority of them.

The classical description of the disease divides its course into three stages, designated by the old Latin writers: phthisis incipiens, phthisis confirmata, and phthisis desperata. These stages correspond to the more modern description of the development of tubercles, their softening, and the formation of cavities. And although this division is little employed at the present time, it is more or less clearly apparent in many cases.

In the early stage there is the persistent cough. It may be a persistent hacking little noticed by the patient during his busy moments, and it may become troublesome only when he lies down or arises in the morning. The expectoration is generally slight and of a clear mucous character; occasionally it is streaked with a little blood. The appetite is lost or becomes capricious, and the digestion becomes feeble, the bowels are generally constipated, but diarrhea sometimes develops. The patient grows anemic and he loses flesh; his strength fails, and slight exertion causes dyspnea and rapid breathing. The heart's action also becomes irritable. Slight elevation of temperature may generally be observed at this time, but not always at any definite time of the day. In many cases there is the cachectic flush with slight fever toward evening. An elevation of the temperature on the affected side amounting to 1° or 2° F. (0.5°—1.5° C.) has been observed by Peter, but it is not invariably present. Night-sweats are often an important symptom. All these manifestations may develop in the course of a few weeks, or they may occupy as much as two or three years. Recovery is possible in the less rapid cases, but as a rule there are alternating periods of improvement and decline. As the disease progresses, the patient's appearance becomes distinctive. The face becomes drawn or pinched, the expression anxious; the cheeks appear hollow and the eyes sunken, though still bright; the skin becomes sallow, sometimes appearing stretched; the finger-ends become thick or clubbed and the nails incurvated (Hippocratic fingers) and blue. The patient, always hopeful, still boasts of his strength, while his cheeks are flushed and his breathing short. Every cold contracted, every attack of indigestion, every fatigue, hastens the decline. Winter is the worst season for the consumptive. The cough then becomes more severe, the expectoration more abundant and more purulent. Microscopic examination of the sputum reveals numerous bacilli and frequently the elastic tissue from the lungs. Numerous micrococci are also present in some cases. From the beginning of cavity-formation, much depends upon the physical endurance and resoluteness of the patient. Many persons, through sheer determination, continue their usual pursuits until the most advanced stage is reached; others yield more readily to the growing inclination to rest. Sooner or later in all cases there comes a time when the bed becomes the mercy-seat. Each day it is longer occupied and returned to with less reluctance. The fever becomes high, the emaciation advances more rapidly, and the weakness grows more extreme. The approach of death becomes more and more apparent to all but the patient. The end is often hastened by a colliquitive diarrhea which generally indicates the implication of the intestine in the tubercular process, but in other cases the decline is long drawn out and life hangs as by a thread for days and weeks. The end comes peacefully in a coma or amidst pitiful strug-

gles against the inevitable. The average duration of the disease in 1,000 cases among the upper classes in England was found by C. J. and C. T. Williams to be 7 years and 8.72 months. Among the lower classes it is much shorter.

Special Symptoms.—*Local.*—(1) Cough is one of the most constant symptoms throughout the disease. In the beginning it may be so slight as to attract little attention; the patient is often little aware of it, but later it is often so distressing as to interfere with sleep. It is often out of proportion to the evidences of pulmonary involvement, being sometimes excessive, sometimes unaccountably mild. It is at first bronchial in character, but after the formation of cavities it becomes paroxysmal, and it is aroused more particularly by changes of position, as when the patient lies down or arises from sleep. Not infrequently the paroxysms induce vomiting. The sound of the cough is often peculiarly hollow. It becomes husky or hoarse when the vocal cords are involved.

(2) *The Sputum.*—(*a*) The quantity of sputum is exceedingly variable. In some cases, even after months of constant cough, there may be scarcely a trace of it, while in others the expectoration is profuse from the beginning. After large cavities have formed, the quantity sometimes becomes enormous; a pint (500 c.c.) may be expectorated in 24 hours. The sputum at first consists of clear, glary mucus from the bronchial tubes, or it may consist largely of alveolar epithelium in a state of myelinic degeneration. It contains numerous small air bubbles and floats on the surface of water. The appearance of small grayish or yellow, purulent masses is more distinctive, for it is in them that the tubercle bacilli are most numerous. As the caseous nodules in the lung begin to soften, the sputum becomes more abundant and assumes a more uniformly purulent appearance. It is often expectorated in coinlike (nummular) masses which sink in water. The only pathognomonic feature of tuberculous sputum, however, is the presence of the bacilli. When these exist in it, the case is always one of tuberculosis. They may be few or many. When only one or two are found or when they are absent from the sputum of a suspicious case, repeated examinations should be made, for the presence of a few may be accidental, and their supposed absence may be due to faulty technique in the collection of the sputum or in the process of staining. (For methods of staining, see p. 748.)

FIG. 13.—Tubercle bacilli in sputum.

Other bacteria are not infrequently found, particularly when large cavities exist. They are especially streptococci, occasionally staphylococci and pneumococci. Sarcinæ are sometimes present, and such fungi as the aspergillus even more frequently.

(*b*) *Elastic Tissue.*—This is found only after the affected tissue has begun to disintegrate. It may be derived from the bronchial tubes, the alveoli, or the walls of the arteries. That from the bronchi forms an elon-

gated network, or several long, slender fibers may lie close together; that from the blood-vessels may have the same appearance, but thin sheets, like fragments of the intima, are sometimes found. The alveolar elastic tissue is generally branched and it may retain the outline of the air-cells, as in Fig. 15.

(*c*) *Blood.*—This is sometimes only sufficient to tinge the sputum, and it may be recognized only on microscopic examination. When hemoptysis occurs, blood is the chief element of the expectorated matter. It has a bright red color and is usually so intimately mingled with air as to appear frothy.

FIG. 14.—Elastic tissue in sputum.

(*d*) *Calcareous Particles.*—These are only occasionally found and their discovery is of little significance further than the fact that there must be some disintegration of lung tissue to permit their escape from the tissues in which they were embedded. They represent calcified tubercular nodules. In size they vary from a millet-seed to a cherry. Only one or quite a number may be coughed up. They occasionally originate in a bronchial gland which has ulcerated into a bronchial tube.

(3) *Pain* is not usually a prominent symptom. It may be absent throughout the disease. In many cases there is, however, a constant sense of discomfort in the affected part of the lung, which becomes a more or less severe pain upon coughing. Sometimes there is sharp, lancinating pain as a result of involvement of the pleura. When the cough is very troublesome, the lower portion of the chest often becomes painful, in part, perhaps, from the muscular exertion. Periodic attacks of pleurisy or intercostal neuralgia are not uncommon during the disease.

FIG. 15.—Elastic tissue with epithelium and bacteria.

(4) *Dyspnea* is often absent except as it may result from exertion. In some cases, on the other hand, it is a prominent symptom from the beginning; it may even become less pronounced as the disease advances. In the more acute cases the respiration often becomes rapid, but when the process is slower it may be but little accelerated, even after an entire lung has become solidified. A cardiac dyspnea may develop in cases complicated with hypertrophy or rapid action of the heart. In some cases occasional attacks resembling asthma occur.

(5) *Hemoptysis.*—Hemorrhage of the lungs occurs in from 60 to 80 per cent of cases. It is nearly five times more frequent in men than in women. It occurs early in the disease, often before the existence of recognizable lesions; or late, after the formation of large cavities. The early hemorrhage sometimes follows a gradual decline of health,

with anemia, slight cough, or indigestion, but it often attacks without warning young healthy individuals free from predisposition to tuberculosis or recognizable tuberculous taint. Although such hemorrhages are regarded as of tuberculous origin, physical examination fails to reveal it, and the sputum often contains no bacilli. Not infrequently the patient continues in good health. In another group of cases the hemorrhage follows some unusual exertion, as swimming or athletic sport; no recognizable lesion may exist, but the sputum contains bacilli. The quantity of blood is usually small, only a dram or two, perhaps; fatal hemorrhage is exceedingly rare. In most cases repeated hemorrhages occur. They are sometimes so frequent as to justify the appellation hemorrhagic phthisis, given to the condition by some authors.

Late hemorrhages are more frequently profuse; a pint or more of blood may be lost within a few minutes, and a fatal syncope may result. The blood usually comes from the erosion of an artery or the rupture of a small aneurism within a cavity. A fatal termination is more apt to follow a succession of profuse hemorrhages. Thirst and dyspnea result from the loss of blood. The pallor which is present is in part anemic, in part a result of the alarm occasioned by the hemorrhage. In some instances the blood is retained within a large cavity and is not ejected. The other symptoms are dyspnea, thirst, sometimes sighing or yawning, and a bloodlike odor may be detected. After a hemorrhage, blood continues to appear in the sputum for several days, but its color becomes darker. In some cases an oozing from small vessels within a cavity may keep up the expectoration of bright arterial blood for a much longer time, and the appearance of hemorrhage is maintained. The more remote effects of hemorrhage are very different in different cases. Improvement sometimes follows, for a time at least, but in a majority of cases the progress of the disease becomes more rapid.

General Symptoms.—(1) *Fever.*—This is one of the most important features of the disease, its presence being especially valuable in prognosis, for fever denotes waste and loss of strength; its absence indicates a possibility of improvement. In the early stages, the presence of slight fever is apt to escape observation, unless the temperature be taken at comparatively short intervals, as once every two hours. The highest temperature usually occurs between 2 and 6 p.m., the lowest between 2 and 6 a.m. In not a few cases the temperature becomes subnormal in the early morning hours. Yet many cases reach an advanced stage without fever. Frequent observations will usually show slight elevation of temperature after exercise or excitement of any kind. The fever of the early stages is probably of different origin from that of the late stages. It is doubtless due to the tuberculization or advance of the tubercular process within the lungs, being produced, no doubt, by the absorption of toxins, since the same effect is produced by the injection of tuberculin. In the late stages it may be due to the same influence, but it is often septic in character, and possibly arises from mixed infection and absorption of other toxins than those of the tubercle bacillus. The fever may be either remittent or intermittent in type. Either form may occur in either stage, and the two sometimes alternate as the processes in the lungs change from time to time. The occurrence of a daily chill followed by fever and sweating is not uncommon, and it is, perhaps,

often mistaken for malarial infection. The fever of the advanced stages is more apt to be continuous; and the more rapid the processes of softening and suppuration, the less will be the fluctuation of temperature. A fluctuation of only a degree or two is also suggestive of the presence of tubercular pneumonia, which may develop at any time during the course of the disease. A wide range from day to day is highly characteristic of tuberculosis, for there is often a difference of from 3° to 5° F. (1.5° to 2.5° C.). The curve may be constantly above normal or it may drop several degrees below in the night-time. After hemoptysis the temperature is often higher, possibly in part from the absorption of altered blood, but often on account of the catarrhal pneumonia that is developed.

(2) *Sweating.*—Profuse sweating is often a serious symptom. It most frequently assumes the character of night-sweats, occurring as the fever drops in the early morning hours, but often also during a nap in the daytime. It is more common after the disease has become advanced, but it may be present from an early stage. Some patients are so fortunate as to escape it altogether.

(3) *Pulse.*—In acute cases the pulse becomes rapid, from 100 to 120 or higher. The rate does not always correspond to the temperature, and the acceleration may not develop until several days after the discovery of fever. In chronic cases the pulse may remain normal, full or small, regular or irregular; it is generally regular, but weak and soft. A capillary or venous pulsation may sometimes be seen, the latter especially on the backs of the hands. A dicrotic pulse is oftener observed in this than in any other chronic disease (Vierordt).

(4) *Respiration.*—A careful count usually shows increased activity of respiration. It may be slight in early, mild cases. When there is involvement of a large area of lung tissue and when fever develops, however, the breathing becomes more accelerated. The ratio of the respiration to the pulse is usually maintained. An intensely fetid odor of the breath is not infrequently noticeable. It is usually indicative of a mixed infection.

(5) *Emaciation.*—Next to the temperature chart, the record of weight gives the most valuable indication of the progress of the disease. A gradual decline is the rule, although it may amount to so little as not to be observed without actual weighing. In febrile cases the emaciation progresses with greatest rapidity.

(6) *Psychical State.*—One of the most striking features in many cases is the hopefulness of the patient, a pleasing delusion of recovery which clings to the last and should not be dispelled.

Physical Signs.—(*a*) *Inspection.*—The typically phthisical chest is long and narrow, or broad and flat, with abnormal straightness of the upper ribs and obliquity of the lower. The scapulæ are winged. In the long, narrow chest the intercostal spaces are usually wide; in the flat chest they are sunken and the sternum may be depressed, the lower portion often deeply concave. The supra- and infraclavicular spaces of the affected side are often more depressed than those of the normal side. The diminished expansion of the defective side is also distinctly noticeable, particularly if looked at from above. Osler calls attention to the importance of observing the condition of the precordia, as a wide area

of impulse, particularly in the second, third, and fourth interspaces, is often associated with chronic tuberculosis of the left apex.

(*b*) *Palpation.*—The disparity of expansion can be better recognized by this method, especially by standing behind the patient and placing the thumbs in the supraclavicular and the fingers in the infraclavicular region. An increased vocal fremitus can often be detected over the affected area before it is revealed by other methods. Allowance must be made for the normally stronger fremitus of the right side. It is more markedly exaggerated over a cavity. Thickening of the pleura and the presence of fluid in the pleural cavity diminish or entirely arrest the transmission of the fremitus.

(*c*) *Percussion.*—It is seldom that much can be learned from percussion in the incipient stage. In some cases slight dullness may be elicited, over the affected apex, by percussion upon the clavicle or above or below it. Percussion with the breath held in full inspiration is more certain to bring out dullness. The note is often slightly tympanitic (tympanitic dullness). Absolute flatness is obtained only over large areas of consolidation; a full tympanitic note or a cracked-pot sound, only over thin-walled cavities of considerable size. Neither of these sounds is trustworthy in children, however, since they may be brought out by forcible percussion of the normal chest. The sense of increased resistance imparted to the finger is often of as much value as the tone, and it is often more easily recognized. A fibrillary contraction, or mounding, of the pectoral muscle (myoidema) is often an interesting phenomenon during percussion. It is, however, without especial relation to the disease.

(*d*) *Auscultation.*—Every deviation from the normal vesicular murmur and every adventitious sound may be heard in the course of the disease. In the early stages the evidence obtained from auscultation is not always conclusive. There is at first, perhaps, only a slight feebleness of the vesicular murmur, or it may become entirely inaudible owing to the diminution in the amount of air entering the air-cells. A little later the murmur becomes higher in pitch or harsh, and with this the expiratory murmur is slightly prolonged. The rhythm often becomes wavy or jerky, or it is described as a cog-wheel respiration. A slight pleuritic friction is sometimes heard for a considerable length of time before other evidence of the disease becomes apparent. This sound is distinguished with difficulty, however, from the dry, subcrepitant, almost crepitant râles that are sometimes heard at the end of a full inspiration in this early stage. When moist râles are heard in the initial stage, they are usually due to the coexistent bronchitis. Coughing sometimes removes the râles, sometimes it serves to render them more audible.

As consolidation becomes more fully developed, the respiratory murmur is replaced by tubular breathing, the vocal resonance is increased, and bronchophony may be heard. The whispered voice is also rendered higher in pitch and more concentrated. Complete consolidaton of a large area sometimes arrests all sounds, owing to the loss of respiratory movement. As a rule such silence is indicative of hydrothorax, especially when it is confined to the lower part of the chest. The moist râles of bronchitis are often heard with greater distinctness through an area

of solidification. With the occurrence of softening, the moist râles become louder and more numerous, usually coarser in quality.

The formation of a cavity, after it has attained the size of a walnut or larger, leads to the production of many more or less distinctive sounds. The respiratory murmur becomes cavernous or amphoric in character over the cavity. The vocal resonance is increased, concentrated, and raised in pitch, not infrequently attaining the quality of pectoriloquy, when the voice seems to emanate directly from the chest. If the cavity be surrounded by solidified lung there is often a peculiar combination of bronchophony and pectoriloquy. Egophony is sometimes heard. A closed, full, cavity yields no respiratory or vocal sound, but when open and only partially filled it usually produces numerous moist râles, yielding bubbling, gurgling, or hissing sounds, sometimes a metallic tinkle or crackling. The succussion sound has been obtained in a few instances from cavities of unusually large size. Pleuritic friction sounds may be established at any time in any region as a result of the involvement of the pleura. Over the opposite lung and over unaffected portions of the diseased one, the vesicular murmur becomes high in pitch and slightly roughened, sometimes puerile, as a result of so-called compensatory emphysema or hypertrophy.

A pleuropericardial friction is sometimes heard over the cardiac region when the intervening lung is involved. The heart sounds are often transmitted through a cavity with unusual distinctness.

Complications of Pulmonary Tuberculosis.—The larynx not infrequently becomes tubercular as a result of inoculation with the virus from the affected lungs. Pleurisy is almost invariably present sooner or later. It may be simple or tubercular, dry or accompanied by effusion. The fluid may be serous, purulent, or hemorrhagic; rarely chylous. Pneumothorax is a not infrequent complication as a result of the rupture of a cavity. It may prove fatal within a few days. Pyopneumothorax is less frequent, and it is often a less fatal complication. Pneumonia frequently occurs as a terminal affection. It is often exceedingly difficult to determine whether the disease is tubercular in character or due to the pneumococcus. Gangrene of the lung occasionally develops. A slight dilatation of the air-cells, usually referred to as compensatory emphysema, is found in the unaffected parts of both lungs.

Endocarditis is infrequent, although murmurs are often heard at the apex or over the pulmonary valve. Vegetations occur most frequently on the tricuspid valve.

Various complications are met with on the part of the alimentary canal. The appetite is early lost in many cases; there may be a loathing of food, nausea, or vomiting. The tongue is usually furred, and a red line is often seen upon the gums which was once erroneously thought to be characteristic of the disease. Catarrhal or aphthous ulcers may occur in the mouth, esophagus, or stomach. Gastric dilatation is occasionally observed, and there may be marked changes in the mucous membrane with corresponding alterations of function. The acid secretion may be increased or diminished. In other cases, however, although there may be persistent indigestion, anorexia, and vomiting, comparatively little that is abnormal can be found in the stomach. The per-

sistent vomiting in such cases is often simply a result of excessive coughing.

Constipation is usually present, but diarrhea may occur at any time. Late in the disease it is frequently due to tubercular disease of the intestine. In some cases, however, even late, it is the result of simple catarrhal, ulcerative, or amyloid disease. Hemorrhoids are often present in tuberculous cases and prove persistent when there is much coughing. Anal fistula occurs in about 3.5 per cent of the cases of pulmonary tuberculosis.

Albuminuria is not unusual, either as a result of the fever or of nephritis. In the latter form, occurring late in the disease, edema generally develops, and casts are found in the urine. Amyloid disease of the kidneys, a not infrequent complication, produces similar changes, and the urine is copious and of light color. Pyelitis and cystitis, with pus and sometimes blood in the urine, are often late complications. Tuberculosis of the testicle is often encountered.

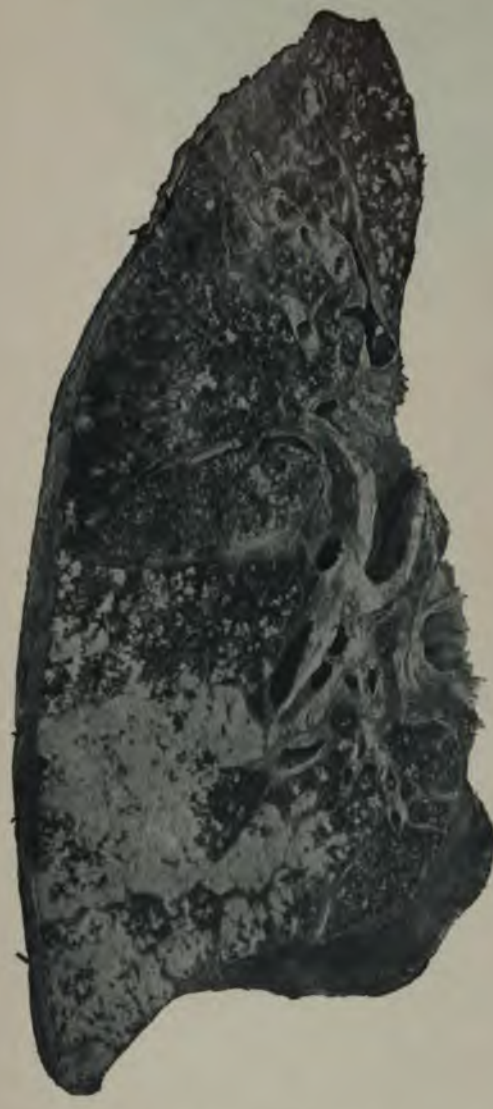
A peculiar hypertrophy of the mammary glands is occasionally seen, especially in the male subject of chronic tuberculosis.

In addition to the anemic pallor, the cachectic flush, and the cyanosis which are frequently seen, the skin often shows pigmentation over the chest (cloasma phthisicum), especially when the peritoneum is involved; or the brown stains of pityriasis versicolor on the chest and back. The hair of the head and beard often becomes harsh and dry, and the nails brittle.

Such diseases as typhoid fever, measles, erysipelas, and the other acute infections may occur in tuberculous patients, but they bear no relation to it. Tuberculosis, especially of the serous membranes, is a not infrequent terminal affection in chronic heart disease and in chronic arthritis. Mitral and more especially pulmonary stenosis and aortic aneurism apparently predispose to tuberculosis of the lungs; and the immunity of patients with chronic heart disease to tuberculosis, at one time thought to exist, is not well established.

(3) *Fibroid Phthisis.*

This term is applied to a form of chronic tuberculosis of the lungs in which the hyperplasia of connective tissue predominates. It may result from the process of sclerosis accompanying the tubercle-formation or it may be the result of the engrafting of tuberculosis upon a lung that is already in a state of chronic fibrosis from pneumonokoniosis or other occupational irritation, or of the so-called chronic interstitial pneumonia. In like manner, it may follow chronic tubercular bronchopneumonia or pleurisy. In the primarily tubercular form it usually begins in the apex along with the tubercular process and advances with it to the inferior portions of the lung. The condition produced varies from a moderate increase of the fibrous tissue confined to a single apex to the almost complete transformation of the entire lung into dense, hard, scarlike tissue, with here and there bronchiectatic or necrotic cavities, lying open or filled with caseous or calcareous débris. In many cases it can be distinguished from the nontubercular form of fibroid phthisis only by the demonstration of tubercle bacilli, but ordinarily



Diffuse (Chronic) Pulmonary Tuberculosis—"Chronic Phthisis."

In the upper half of the lung there are scattered miliary tubercles and irregular areas of consolidation, with a diffuse formation of fibrous tissue; the pleura is thickened. A large portion of the lower lobe is densely consolidated from tubercle tissue and exudate with coagulation necrosis of the involved regions. These regions are light in color, dense, hard, and bloodless. Such dead caseous areas may persist for some time, or may soften and disintegrate, giving rise to cavities.

(*By permission, from "Delafield and Prudden."*)

numerous tubercular nodules can be distinguished in the lesions. (See Plate IV.) The tissues are usually deeply pigmented. When the primary irritation has reached the lung through the bronchi, as in cases caused by the inhalation of dust, the fibrosis is largely peribronchial; and when upon the surface, as in chronic tubercular pleurisy, the fibrosis is confined to the periphery of the lung.

Symptoms.—The clinical manifestations are usually those of a rather mild, slowly progressing pulmonary tuberculosis—cough, abundant, purulent expectoration, dyspnea on exertion, gradual emaciation. The patient usually acquires a peculiarly dull, dusky facies, with thick bluish lips, often with puffy eyelids and congested conjunctivæ. Fever is not common. When large bronchiectatic cavities exist, the sputum may have a fetid odor which is communicated to the breath, and it may be found to contain, in addition to the bacilli and other organisms, pus-cells, elastic tissue, and peculiar acicular (Charcot-Leyden) crystals. Hemoptysis is occasionally the direct cause of death. Both lungs are often involved. It is in this form of tuberculosis that the clubbed fingers are most markedly developed.

The *physical signs* are often distinctive. The affected side is depressed from the shrinking of the lung; the shoulder may be lower and the chest-wall distinctly sunken. The expansion is much reduced. The affected area yields marked dullness on percussion, and all the sounds due to consolidation and cavity-formation may be heard upon auscultation. The areas of cardiac dullness and impulse are usually increased, owing to hypertrophy of the right ventricle, and the heart may be displaced by the contraction of the fibrous tissue and the formation of pleuritic adhesions. Amyloid disease of the viscera is especially frequent after this form of tuberculosis. Chronic passive hyperemia of the liver is a frequent result of the obstruction of pulmonary circulation, and dropsy develops from the enfeeblement of the heart's action.

4. TUBERCULOSIS OF THE CENTRAL NERVOUS SYSTEM.

The brain is more frequently the seat of tubercular infiltration than the cord. In both locations the disease is usually associated with tubercular meningitis. An interesting form, more frequent in children, is that in which a single large tubercle is developed in the brain substance. Symptoms of localization corresponding to those of other brain tumors are produced. Multiple tubercular formations are also met with, most frequently in the cerebellum, next in the cerebrum, seldom in the pons or cord. They vary in size from a pea to an orange, rarely even larger. They show the usual changes—caseation, calcification, sometimes liquefaction. They are generally attached to the meninges when of large size, and they not infrequently induce inflammation of these membranes. They are usually associated with tuberculosis of other organs, especially of the lungs.

5. TUBERCULOSIS OF THE CIRCULATORY SYSTEM.

The myocardium and its membranes may be involved in acute miliary tuberculosis, and caseous nodules are occasionally found in them in chronic pulmonary tuberculosis as a result of direct extension of the

disease. True tubercular endocarditis is probably less frequent than that due to mixed infection with the streptococcus and the staphylococcus. The arteries have never been found to be the seat of primary tuberculosis, but they often become the seat of secondary invasion. The intima and muscular coats become thickened and the lumen may be obliterated. The larger vessels are frequently involved in the suppurative disintegration of the bronchial, mediastinal, and other lymph-glands. Infection of the arteries from the blood is rare, but Osler records a case observed by Flexner in which a large solitary tubercle was found in the aorta independently of external connection.

6. TUBERCULOSIS OF THE DIGESTIVE SYSTEM.

The Mouth.—The mucous membrane of any part of the mouth may become inoculated with tubercle bacilli from the sputum of a tuberculous patient. A tubercular infiltration followed by the formation of rough, irregular ulcers, usually having a caseous base, may occur on the lips, on the sides or dorsum of the tongue, or elsewhere. Anders records an apparently primary lesion on the lip. Such ulcers resemble a chancre or epithelioma, and are to be distinguished from them chiefly by the history of their growth, the presence of tuberculosis in the patient, the presence of bacilli in the tissues, and the effects of antisyphilitic treatment.

The *tonsils* are probably oftener the avenue of entrance to the bacilli than is generally realized, particularly in cases in which the cervical lymph-glands are involved. The inoculation generally occurs from the sputum or possibly from tubercles in the food. The tonsil becomes enlarged by the tubercular infiltration, but the condition is distinguished with great difficulty from a simple follicular enlargement. Ulceration usually follows.

The *pharynx* becomes involved either by direct extension from the larynx and epiglottis or from the lungs. The wall of the pharynx is often closely studded with tubercles or it may become ulcerated, and deglutition is then extremely painful. The disease frequently involves at the same time the hard or soft palate. The adenoids of the nasopharynx have occasionally been found tubercular.

The *esophagus* is seldom involved except in the small upper segment as a result of direct extension from the pharynx or larynx. Any part of it may, however, become infected directly from the bronchial glands, the vertebræ, or by inoculation from the sputum.

The Stomach.—Tuberculosis of the stomach is also exceedingly rare, probably owing to the protective influence of the acid gastric juice. But single or multiple tubercular ulcers are occasionally found in its mucous membrane, and perforation has occurred in a few instances. The symptoms of the tubercular ulcer are not distinctive. Intense pain soon after eating, vomiting, especially hematemesis, are usually indicative of it. The tuberculous character of the patient should arouse suspicion of the tubercular nature of the disease.

Tuberculosis of the Intestine.—The intestine is much more commonly the seat of tuberculosis than the upper part of the alimentary canal. The disease is primary, however, in only about one case in a thousand adults. It is a little more frequent in children. As a secondary affection

it is probably most frequently a result of the swallowing of sputum, but the infection may be conveyed from other centers through the blood or lymph circulation. In a third group of cases it results from the direct extension of the disease from the peritoneum, ovaries, or abdominal glands. Secondary involvement of the intestine occurs in nearly half the cases of pulmonary tuberculosis, usually as a late complication.

Morbid Anatomy.—The lesions are remarkably uniform in character. There is catarrhal inflammation of the mucosa, with hypertrophy of the villi, and later more or less extensive ulceration. The primary lesions are often situated in the solitary and agminated follicles. These become swollen and ulcerated as in typhoid fever, but the ulcers are less regular in outline, usually have a caseous base, and their long axes soon become transverse to that of the intestine. Little chains of tubercles extend outward toward the mesentery, sometimes even to the mesenteric glands, and the ulceration follows their course. A cluster of young tubercles is usually found in the serous coat immediately opposite the ulcer in the mucosa. The ulcers show no tendency to heal. The disease is generally situated in the lower part of the ileum and cecum and upper part of the colon. A large area of the bowel is often involved, but in some instances it is confined to a limited portion, as the ileocecal region. The appendix may also become involved. Large tumor-like masses consisting of the intestine and caseous nodules are sometimes formed which more or less completely obliterate the lumen. Perforation often follows, leading to a fatal acute peritonitis or to the formation of a fecal fistula. Stenosis from cicatrization is a less frequent result. Fistula in ano is commonly a result of localized tuberculosis of the rectum, occasionally primary in character.

FIG. 16.—Tubercular ulcer of the intestine. (Med. and Surg. History of the War of the Rebellion.)

Symptoms.—Diarrhea is the most common manifestation of intestinal tuberculosis. The stools contain much mucus and often pus and blood. The bacilli are numerous in them, but they cannot be regarded as diagnostic of the intestinal lesion, since they may have passed through with the sputum. Fever, colicky pains, tenderness, and meteorism are usually present. The emaciation becomes extreme. But the most advanced tubercular lesions are sometimes found in the bowel after death in cases which exhibited no evidence of intestinal involvement during life.

Diagnosis.—The diagnosis of primary intestinal tuberculosis is extremely difficult. The persistent, profuse watery dejections, containing much mucus and occasionally pus and blood, without other recognizable cause, and attended with abdominal pain and tenderness, especially if the pain and tenderness be confined to the ileocecal region, are sufficient to arouse suspicion of the disease. The presence of fever, rapid emaciation, and the discovery of bacilli in the dejections support the diagnosis,

but the reaction to the tuberculin test is of more positive value. When a tubercular focus can be recognized elsewhere, the diagnosis is much less difficult.

Tuberculosis of the Liver.—The liver is always involved in acute miliary tuberculosis, but the lesions are often microscopic in size and may be concealed by the pale color due to fatty degeneration. Localized or disseminated tuberculosis also occurs. In the former, the solitary large tubercle is sometimes found, or there may be more or less numerous smaller nodules. These sometimes undergo softening or complete caseation and necrosis, breaking down to form abscesses which may give the organ a honeycombed appearance. In another class of cases there is marked sclerotic increase of the connective tissue accompanied, perhaps, with advanced fatty change. The nodular formation often follows the course of the bile-ducts. Perihepatitis is sometimes induced, with the production of ascites.

Tuberculosis of the Pancreas.—The disease is always secondary in origin, and occurs in about 5 per cent of all cases of general tuberculosis in children; it is rare in adults. Both the miliary and localized forms are encountered, the latter sometimes producing large abscess cavities. The infection is supposed to be carried either through the blood or lymph, or by way of the pancreatic duct from the intestine.

The *spleen* is usually involved in general tuberculosis, and sometimes secondarily in chronic pulmonary tuberculosis. The foci often become caseous and abscesses are sometimes formed.

7. TUBERCULOSIS OF THE GENITOURINARY SYSTEM.

Tuberculosis may attack any part of this system, or it may involve all the organs simultaneously, particularly in the general miliary form of the disease. It is comparatively seldom that a single organ or viscus is affected. Probably the most frequent location of the primary lesion is the epididymis; but it is occasionally found in the testicle proper. The extension of the disease from one center to another is accomplished by various routes. The bacilli readily pass downward from the kidneys and probably upward through the urethra to the bladder, in the same manner as they pass through the vagina and uterus to the Fallopian tubes. They are sometimes conveyed through the blood from the lungs or other remote centers of infection, probably also by the lymph-vessels in some instances. The disease may extend directly from the intestine or peritoneum to the bladder, vesiculæ seminales or Fallopian tubes, or from the bodies of the vertebræ to the kidneys. Infection through sexual intercourse is regarded as possible. Genitourinary involvement has been found also in the fetus, illustrating the possibility of hereditary transmission. The disease is three times more common in men than in women, and it usually develops between the ages of 20 and 40. Its progress from region to region is generally remarkably rapid. The extension may be direct, as when it extends by the formation of a line of tubercles through the ureter from the kidney to the bladder, and surface inoculation is supposed to occur in some instances. Whether or not the bacilli ever pass upward from the bladder to the kidney is an unsettled question, as it is thought improbable that they could be carried against the constant flow of urine through the ureter.

Tuberculosis of the Kidneys.—The disease may be primary, but it is much more frequently secondary to tuberculosis of other organs. One or both kidneys may be affected. Men are more frequently affected than women, and generally in middle life, except by the miliary form, which may occur in any age. The kidneys are involved in about half the cases of genitourinary tuberculosis.

Morbid Anatomy.—In general miliary tuberculosis, miliary tubercles are found scattered throughout the organ and beneath the capsule, while in other cases they are often confined to the pelvis and papillæ, producing pyelitis. It sometimes happens after death from pulmonary tuberculosis, that a few tubercles are found in the kidneys, which had produced no recognizable disturbance during life. As a rule, the upper extremity of the ureter, and in more chronic cases the lower portion of it and perhaps the bladder and prostate, are involved along with the pelvis of the kidney. In quite a large group of cases the testicles are likewise affected. As a result of caseation and softening, cyst-formation or pyonephrosis is induced. By the coalescence of several nodules, large caseous masses are formed. One kidney is often found in a more advanced stage of the disease than the other.

Symptoms.—In acute miliary tuberculosis the involvement of the kidney seldom attracts attention. In the chronic form, indeed, there may be few symptoms until a late stage has been reached. Advanced lesions have been found post mortem which were not suspected during life. The manifestations are then characteristic of pyelitis. When the kidney is much distended by tubercular formations, as in pyonephrosis, there are usually constant pain and tenderness in the region. The pain may become agonizing at times, but in other cases it is never severe. In some cases there is a tumor-like prominence over the region of the affected organ and fluctuation may be detected. There may be also pain in the bladder. Micturition becomes frequent, and the urine, generally of acid reaction, often contains pus and blood. Albumin is never absent, and, in addition to pus-cells, epithelium, sometimes caseous masses, and bacilli are found in it. Casts cannot usually be recognized. Irregular fever and occasional chills are often observed. The constitutional symptoms are naturally more pronounced when both kidneys are involved. The intermittency of the manifestations is strongly characteristic. In most cases, even when advanced, there are intervals of freedom from fever, pain, and swelling, with secretion of clear urine, sometimes lasting for several days or weeks, followed by a return of the symptoms. In this manner the disease may run along for several years without greatly reducing the patient's strength, but emaciation is generally progressive. In a few cases old caseated or calcified masses have been found in the kidneys after death from other diseases, indicating that spontaneous arrest of the disease had occurred.

Diagnosis.—The persistency and intermittent character of the disease strongly point to its tubercular nature, but in most cases the diagnosis is difficult unless the patient be submitted to the tuberculin test. The discovery of tubercle bacilli in the urine in repeated examinations may be regarded as proof of the tubercular character of the lesions, but the smegma bacillus must be excluded. Fortunately this may readily be done, since it is decolorized by immersion for a minute in alcohol after

staining in carbol-fuchsin. But the tubercle bacilli are not generally discovered in more than half the cases. It is usually more accurate to examine the urine that has been withdrawn by the catheter, and the precipitate should be obtained by centrifugation. Before operation is resorted to, the urine from each kidney should be examined separately.

Cystitis can generally be recognized by the alkalinity of the urine, the greater quantity of mucus, and the less frequent appearance of blood, as well as by the absence of tenderness, pain, or swelling in the region of the kidney.

Cysts, *calculi*, and *tumors* of the kidney can generally be excluded by the presence of a tuberculous history or proof of such infection in some other organ. Copious hemorrhage and the presence of a large tumor, although possible in tuberculosis of the kidney, are more strongly indicative of pyelous nephritis or malignant disease. The latter class of diseases shows more rapid progress.

Treatment.—The general treatment of the tuberculous condition is highly important in primary genitourinary cases, in order to favor a spontaneous arrest of the disease. The urine may be rendered less irritating and the pain consequently diminished in some cases by administration of alkalis, as sodium benzoate, gr. x to xv (0.65 to 1.0), well diluted, every four hours, or urotropin, gr. v to x (0.35—0.65), thrice daily, and by the drinking of a large quantity of water.

Tuberculosis of the Ureter, Bladder, and Urethra.—The disease is seldom primary; it is usually an extension from the kidney. In the ureter the lesions are either at the upper extremity or they are confined to a small area just above the vesical extremity. The bladder is most frequently infected through the urine from the diseased kidney or from the testicle, seminal vesicles, or prostate, the virus probably being carried through the lymph-vessels. The disease may be communicated, however, by direct extension from the peritoneum, intestines, Fallopian tubes, ovaries, or vagina. The urethra is rarely involved and probably, in most cases, secondarily to one or more of the associated viscera.

Symptoms.—The symptoms are those of cystitis—frequent micturition, especially at night, alkalinity of the urine, pus being generally present, sometimes with hematuria. Pain in the penis, especially in the glans, is often complained of, and it is sometimes severe. Vesical tenesmus often occurs. Tenderness may be elicited by pressure over the bladder, or by rectal examination. Cystoscopic examinations may reveal the tubercles beneath the mucous membrane at an early period, but later large or small ulcers are to be seen. The testes, prostate, and other organs should always be examined for the presence of tuberculosis in them.

Tuberculosis of the Prostate.—The prostate may become involved by extension from any of the surrounding viscera in the manner that has been described. The disease is generally associated with that of the kidneys or bladder, but it is occasionally primary in origin. The prostate seldom escapes when the testicle and seminal vesicles are affected. There is often a history of previous attacks of gonorrhea. The gland becomes much enlarged from the growth of tubercular nodules; caseous softening often leads to the formation of an abscess with many sinuses, or there may be extensive formation of new fibrous tissue. The irritability

of the bladder is often extreme, with painful or more or less obstructed micturition, and catheterization is extremely painful. The enlarged lobes of the prostate can be felt upon rectal examination.

Tuberculosis of the Testicle.—The disease is comparatively frequent in infants and young children; it has been found in the fetus, and it is by no means rare in the adult. It appears first in the epididymis of the adult, less frequently in the substance of the testicle or in the vesiculæ seminales. It is generally secondary in character. One or both testes may be involved. The growth is generally rapid, extension from one location to another equally so, and in most cases the seminiferous tubules and vas deferens soon become involved. The tubercles may be present for a considerable time without undergoing caseation, and the appearance often suggests sarcoma, but the growth is less rapid than that of sarcoma. The testicle is usually less nodular than that of syphilitic disease. In syphilis, too, the vas deferens escapes, and the swelling soon becomes free from pain. The therapeutic test should be employed in obscure cases. Spontaneous recovery undoubtedly occurs in rare instances, but the disease is more apt to become generalized, especially after operation.

Tuberculosis of the epididymis is recognized by the presence of painful and sensitive nodular enlargement, especially at the head of the loop.

Tuberculosis of the Female Generative Organs.—Tubercular ulcers of the vulva have been observed in a very few instances. The disease is not uncommon in the Fallopian tubes, but it is comparatively rare in the ovaries, uterus, and vagina. It may be primary, but it is generally an extension from other centers of infection. Tubercular salpingitis is recognized by a characteristic enlargement of the tube with thickening and infiltration of the walls; caseous masses are usually found in the interior. Both tubes are ordinarily affected. The tubercles may be recognizable only upon microscopic examination of the specimen. The fimbriæ are generally bound down by adhesions to the ovaries, and the disease extends in most cases to the endometrium, very often also to the peritoneum. Large abscess-cavities are sometimes formed.

Tuberculosis of the ovary is always secondary. Extensive caseation and abscess-formation are its chief characteristics. Primary tuberculosis of the uterus has been described, but the disease is usually associated with pulmonary or other involvement. The disease generally assumes the form of ulcerative endometritis, with an accumulation of caseous and purulent débris in the cavity. The muscular substance is sometimes infiltrated. Tubercular disease of the cervix is generally a result of infection from the vagina. Communication of the disease by sexual contact is supposed to be possible.

Tuberculosis of the Mammary Gland.—The mammæ are seldom affected. In some instances the disease is primary, the bacilli reaching the glands through the blood or through the lactiferous ducts, while in other instances it originates in the skin and afterward extends to the gland. It has been seen mostly between the ages of 40 and 60, and only once or twice in the male. Distinct tubercular nodules are formed which tend to unite and form large caseous masses. The disease usually remains localized for many years. The axillary glands and other regions are generally simultaneously involved when the disease is not

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ment. The discovery area establishes pulmonary form, st important of all ly advanced stage nothing, but their of the infection. is still employed by sure, but a majority any instances its use tion is continued into nfirmed at the expense ease. The reaction is existence of tuberculosis loyed with the utmost s. It is highly probable s have arisen from the initial dose, given hypo- in distilled water. This mg. are given every 24 ases within from 4 to 12 et until several injections by an elevation of tempera- 5°—40° C.), with headache, in the limbs, and languor. tubercular processes wherever ds, and joints, for example, and in pulmonary tuberculosis sputum becomes more abundant times also caseous fragments. ases are not acted upon. The the fact, repeatedly observed, by persons and fails in those tuberculous. Of the two tests, more valuable. demonstration of tuberculosis. ions as the destruction of bone several large areas of solidification when the revelation is of the most an area which is sufficiently large almost always readily recognizable ussion and auscultation. ology of the disease and of the to recognize the disease in a great The examination of the sputum, since it is the most positive means

of differentiating most of the other diseases which may be confounded with tuberculosis—notably, bronchiectasis, chronic interstitial pneumonia, syphilis, and malignant disease of the lung.

Bronchiectasis.—In this condition cavities are formed and there is an exceedingly copious, often fetid, purulent expectoration, particularly in the morning. The diagnosis rests for the most part upon the absence of bacilli. But bronchiectasis is probably in most instances a part of the tubercular process in the lungs.

Chronic Interstitial Pneumonia.—In this disease there is usually a history of long-standing bronchitis, a previous attack of pneumonia, bronchopneumonia, chronic pleurisy, or a pneumonokoniosis. There may be a marked deformity of the chest, dyspnea, cough, and free expectoration. The patient has a dull, heavy expression, with thick lips and dusky complexion. Percussion and auscultation reveal a general, or quite extensive, involvement of the lungs. The general condition of the patient is often better than would be compatible with tubercular disease. Anemia, emaciation, and debility are not marked, and there is no fever. In some cases, however, the diseases are associated in the same subject.

Syphilis of the Lung.—The history of the patient, if obtainable, and the absence of bacilli should point to the possibility of syphilis. Syphilitic lesions are seldom found in the apex, more frequently down near the roots of the lung. They are not usually accompanied by so profound emaciation, and the anemia may be less marked. In some cases the diagnosis must rest until the effect of antisyphilitic treatment has been tried.

Malignant Disease.—Sarcoma and carcinoma both affect the lung secondarily, the former following possibly remote disease in the line of venous communication, the latter a less distant growth, connected by the lymph-vessels. Or there may be the history of a recently removed tumor. More than one area of dullness is usually found, corresponding to an equal number of tumors. The progress of the disease is rapid and generally febrile. Tubercle bacilli are not to be found.

Finally, such conditions as anemia, heart disease, gastritis, and nephritis are to be excluded only by careful examination, with full appreciation of the blood-changes and the gastric, cardiac, and renal disturbances which commonly attend tuberculosis at one or another stage.

Phthisiophobia.—Cases are by no means uncommon in which there is a hypochondriacal dread of tuberculosis. Cough, thoracic pains, and great weakness are the usual complaints, but there is entire absence of the physical signs, and the psychical state of the individual stamps itself upon every symptom.

Prognosis of Tuberculosis.

There are no fast rules by which to estimate the prospects of a given case. Much depends upon the location of the disease, its extent, its virulence, the patient's power of resistance, and the measures taken to arrest the disease. The prognosis is always more unfavorable in a case that has pursued an active course. A tuberculous tendency decreases resistance. A good appetite and vigorous digestion, added to previous good health and an absence of depressing influences, increase the chance

of recovery—but it is always a chance; a majority of the cases of supposed recovery relapse sooner or later, and the apparent cure proves to have been but a quiescent period. A slow, afebrile beginning, with pleurisy, is generally followed by a protracted course. The most unfavorable symptoms are persistent fever, pronounced anemia, especially chlorosis, loss of appetite, and feeble digestion. The number of bacilli found in the sputum is of little importance in prognosis, for they may originate in a small focus and they may continue for months after arrest of the process. Repeated hemoptysis is usually unfavorable. The same is true of night-sweats, persistent diarrhea, and progressive loss of weight. The development of acute pneumonia of any type often converts a quiescent case into a rapidly fatal one.

Marriage during the existence of active tuberculosis is always harmful, and especially to the woman, since pregnancy and lactation greatly hasten the progress of the disease. Marriage after recovery from tuberculosis has often resulted happily. Such persons should be taught the necessity of giving their offspring all possible benefit of nutritious food, outdoor life, and judicious gymnastic training. Advice against marriage is seldom gratefully received and rarely heeded.

Prophylaxis of Tuberculosis.

The enormous numbers of bacilli which a tuberculous patient is capable of throwing off daily has been referred to. The importance of measures for the prevention of contagion is apparent. It is the duty of physicians everywhere to cooperate with and to assist the boards of health in all proper efforts to this end. Political and professional prejudices should be forgotten in matters affecting public health. The reporting of cases of tuberculosis is required by law in most of our cities, but in too many places the matter ends with the filing of the report. In other places a list of instructions in the nature of the disease and the measures to be taken for the protection of others is sent to the patient through his physician. The list should contain directions for the collection and destruction of sputum and emphatic warning against promiscuous spitting in public places and conveyances. The use of the "spit-cup," or of a china cuspidor always half-filled with a solution of corrosive sublimate, 1:500 or stronger, should be insisted upon. The patient should be cautioned against the danger in swallowing the sputum. He should occupy a room separate from the apartments of his family, one receiving a large supply of sunlight and air. Flies should be excluded from it, for they are carriers of bacilli. The bedclothing should be exposed for several hours each day to the air and sunshine. Occasional disinfection of the room and its contents with formaldehyd is beneficial. The irritating fumes of the disinfectant can be removed by sprinkling a little aqua ammonia about the room. When these requirements cannot be carried out, it is better to have the patient removed to a sanitarium, where he will be placed under strict rules both for his own treatment and for the protection of others.

The inspection and regulation of the milk and meat supplies are also of importance to the public. Much good has unquestionably been done in late years by the agitation against filthy dairies and by the systematic inspection of milk, cows, and market meat.

Individual Prophylaxis.—This should begin at the time of a child's birth. The infant should not be nursed by its tuberculous mother. If a wet-nurse is not obtainable, the greatest care should be exercised in the selection of an artificial food. A modification of fresh cow's milk is better than a substitute, and milk should constitute a large part of the child's diet during the whole of childhood. The clothing should be carefully adapted to the changes of the weather. Light flannel garments of open texture should be worn. Such children should be kept as much as possible in the open air. They should be early started in a course of gymnastics directed especially to the development of the respiratory muscles, and athletic sports should be encouraged. They should be taught to bathe daily in cool water. Cold sponging of the throat and chest morning and evening, or, better, of the entire body at least once a day, has a decided effect in warding off "colds." Especially important is the ventilation of the bedroom at night. The windows should never be closed; the more nearly the air in the sleeping apartment can be made to correspond to that of the outside, the better. The greatest care should be exercised to avoid catarrhal affections. The nose and throat should be frequently examined with reference to catarrh, the presence of adenoids, and hypertrophied tonsils; and it is important that complete recovery be secured after the acute infections of childhood. The resulting anemic condition should be as speedily as possible overcome by the administration of iron, arsenic, and codliver oil or malt. Young persons having tuberculous tendencies should reside in the healthful climates of the West rather than attempt to combat the disease against the odds imposed by the long, severe winters of the Northern and Eastern States.

Treatment of Tuberculosis.

The treatment of tuberculosis resolves itself into the cure (care) of the patient, rather than the adoption of measures for the destruction of the bacillus, for in attaining the former end we do more than is possible by any other means toward the accomplishment of the latter. The biological processes of the body, properly supported, are capable of overcoming the infection in probably a majority of cases. For their support the best measures are secured by the regulation of the patient's habits and mode of life, his food and all that contributes to an increase of nutrition. The earlier these measures can be adopted, the greater will be the chances of success. Unfortunately many cases that the physician encounters have been neglected until all prospect of recovery has vanished.

When a case is seen in its incipiency, a complete change in the life of the individual should generally be made. More hours should be given to leisure, more hours spent in the open air. All employment would better be given up, if possible, and the first winter, at least, should be spent in a warm climate where the patient can remain in the open air day and night. He should not return north before the month of May. Permanent removal to a suitable climate is of course to be recommended. But a majority of persons are not so situated as to make such changes without undergoing hardships more than equivalent to the benefit to be derived. These must be treated at home. If they reside in a larg

city, removal to the suburbs is advisable, and the farther out the better. The patient should be taught to practice respiratory gymnastics, deep breathing in the open air or at an open window several times a day for many minutes at a time, and to strengthen the respiratory muscles by the use of light dumbbells and "pulleys." There is no better exercise for the breathing than rapid walking or moderate running in the open air, providing it does not cause an elevation of temperature, and the distance may be increased a little each day. Exercise should never be carried to the point of fatigue. The patient should take a rapid cold sponge bath every morning. It should be taken in a warm room, the patient standing upon a rug, not in the tub. If strong enough, he should rub his own body. After the bath the skin should be rubbed into a glow with a crash towel. This may be followed by a rapid sponging with dilute alcohol. After breakfast, whenever the weather will permit, the patient should take a walk in the open air. A daily sun-bath should be taken when possible, summer and winter, the entire body being exposed to the rays of the sun in a solarium or at an open window. If this cannot be done, the patient should sit in the sunshine for several hours; even in bad weather he should sit in the open air on a veranda. These measures must, of course, be undertaken gradually at first.

If the patient is too ill to leave his room, he should still be given all possible advantage of the air and sunlight.

Dietetic Treatment.—One of the most favorable results of a change to outdoor life is often seen in the stimulation of the appetite. The food should be of the most nutritious kind and should contain as much fat as the system can assimilate. Food should generally be given at shorter intervals than in health, the regular meals being supplemented by a light lunch in the middle of the forenoon and afternoon and before retiring. A glass of rich milk may be taken during the night. A half-dozen or more eggs, soft-boiled, poached, or raw with sherry, may be taken daily, if digestion is good. There is no better form of fat than cream or butter. Nitrogenous food, as a rule, agrees better with the patient and enables him better to combat the disease. If the theory that uric acid in the blood antagonizes the tubercle bacillus be true, there is no better way of securing this condition of the blood than by the exclusive meat diet, or a near approach to it.

In many cases the digestive processes appear slow. The ingestion of a full meal arrests appetite for an entire day, and the bowels are usually constipated. In such cases digestive ferments may be given with advantage; in some cases pepsin, in others a diastase and malt extract. The occasional administration of 1-10 gr. (0.006) of calomel for a few days often aids the digestion. The existence of fever does not contraindicate a liberal diet. When, however, filling the stomach induces coughing, and this in turn vomiting, a liquid diet should be taken in small quantities at intervals of two or three hours. Beef, chicken, or other broths may be added to the list. In some cases forced feeding must be practiced. As much as a quart of liquid food, chiefly milk, is introduced into the stomach two or three times a day by means of a tube. The method is applicable especially to cases in which there is a great aversion to food, or in which there is so great destruction of the epiglottis as to render deglutition impossible.

Climatic Treatment.—The selection of a suitable climate depends largely upon the condition of the patient and the stage of the disease. Advanced cases do better at home. They rarely receive benefit from a change of climate that compensates them for the hardships of travel. Poor people in any stage are generally better at home. Some cases show greater improvement in a high, dry atmosphere, while others improve in the pure damp air of the seashore. Cases that have advanced to cavity-formation and those accompanied with repeated hemorrhages should, as a rule, seek a warm equable climate of low altitude, as in the resorts of Georgia, South Carolina, and the east coast of Florida, Bermuda, or southern California. These places are also better for persons who are compelled to return north in the summer. Many advanced cases have made remarkable improvement in Colorado, New Mexico, Arizona, and other Western States, but, as a rule, a return to a place of low altitude causes an awakening of the disease, so sharp that a return to the high altitude proves of no benefit. Many patients and some physicians fall into the error of assuming that climate alone can effect a cure. It is only when a change of climate is supplemented by home comforts, with an abundance of proper food, that great benefit can be expected from it, and the measures which prove of advantage in home treatment are all the more efficient when aided by the pure, exhilarating atmosphere of Colorado. It is not generally advisable to send a patient to a place where he is an entire stranger, without a companion, unless he has been accustomed to travel, for the homesickness is often worse than the disease.

General Medical Treatment.—No drug has yet been discovered which is capable of acting directly upon the tubercular process. The few remedies which deserve mention act solely by improving the condition of the patient, by stimulating his nervous system, enriching his blood, and thus increasing his power of resistance, or possibly by rendering the tissues less susceptible to invasion. The principal remedies are creosot, iron, arsenic, strychnin, hypophosphites, and codliver oil.

Creosot.—This remedy has outlived a thousand in the estimation of the profession, although it is recognized as having no specific action on the disease. In many cases it quiets the cough, diminishes the expectoration, increases the appetite and digestion. It may be taken in milk, sherry, hot water flavored with an essential oil, as cinnamon or cloves, or in capsules. The dose should not exceed ℥j to ij (0.06—0.12) after each meal, in the beginning, but it may be increased one drop a day until ℥x to xx (0.60—1.25) are taken, providing that the stomach continues to tolerate it. Only a pure creosot, made from beechwood tar, should be administered. This remedy has been employed also for inhalation, for subcutaneous, intratracheal, or intrapulmonary injection and by the rectum.

Some clinicians prefer guaiacol to creosot. It is especially suitable for administration to children by inunction. It may be given hypodermically in oil. It is often not so well tolerated by the stomach. When neither of these remedies can be taken by the mouth, their carbonates should be employed. The best time for administration is probably two hours after each meal. In intestinal tuberculosis the creosot acts well when given in keratin capsules, which do not dissolve until they reach the intestine.

Iron.—This remedy is useful only for the relief of the anemia and especially in children. The sirup of the iodid is the best remedy for the tuberculous adenitis. In chlorotic anemia, Blaud's pills, gr. iij (0.2), or the tincture of the chlorid, ♏x (0.60), should be employed.

Arsenic.—This is, as a rule, the best remedy for the anemia, acting more promptly and more certainly than iron. Three to five drops of Fowler's solution are given after each meal. The dose may be gradually increased until the effect is recognizable, but large doses are not generally required.

Strychnin is useful as a general tonic, but it is especially indicated when the heart's action becomes irregular or weak. A dose immediately upon awaking often gives the patient strength to take his morning's bath. It should be given in doses of gr. 1-60 to 1-20 (0.001—0.003). It may be combined with iron and arsenic or given in tablet form.

Hypophosphites.—The hypophosphites of lime and soda are believed by some physicians to exert a tonic influence and to reduce the expectoration. They probably have no other action.

Codliver oil has in some cases a decided influence in increasing the nutrition and in relieving cough. It acts best in children and in the incipient stage. Some physicians prefer the pure oil, others an emulsion. The dose should not be too large and can best be regulated by its effects upon the appetite. From a teaspoonful to a tablespoonful two hours after meals is the quantity usually administered. Whenever the appetite fails, the dose should be reduced. In the advanced stages it is of no benefit, and when diarrhea or fever is present or digestion is feeble it is contraindicated.

Urea.—The fact that tuberculosis and gout are seldom associated in the same individual or family led Henry Harper and others to administer pure urea in doses of gr. xx (1.30) or more four times a day. Excellent results were reported from the treatment, especially when it was combined with a diet favoring the production and retention of uric acid in the system, but the investigations of Pearson indicate that it is of little benefit in tuberculosis of the lungs. Its action is more certain in tuberculosis of the joints.

Treatment of Special Symptoms.—*Fever.*—There is probably no other type of fever which is so difficult of control as that of tuberculosis. The coal-tar antipyretics control the temperature only temporarily, and their depressing effect is undesirable. Quinin cannot usually be taken in sufficient doses to be of benefit without deranging the digestion. In most cases more can be accomplished by rest in the open air and by cool bathing and sponging than with drugs. Osler speaks favorably, however, of 2-gr. (0.13) doses of antifebrin every hour for three or four hours before the rise of temperature takes place.

Sweating.—The aromatic sulphuric acid, ♏xx—xxx (1.25—1.80) t.i.d., often arrests sweating in the early stages. Later, atropin, gr. 1-100 (0.0006) morning and evening or gr. 1-60 (0.001) at bedtime, is more effective for night-sweats. Other remedies are: agaricin, gr. 1-6 (0.01), muscarin (1 per cent solution) ♏v (0.30), and picrotoxin, gr. 1-60 (0.001).

Cough.—When the cough is not so severe as to interfere with sleep it serves a useful purpose and should be let alone. In the distressing

night-cough, however, it is necessary to administer remedies for its relief. In many cases, heroin, gr. 1-12 (0.0056), taken before retiring, is sufficient for the night; but in other cases it fails. It is then better to give: Dover's powder, gr. ij to v (0.13—0.32); codein, gr. ⅛ to ¼ (0.008—0.016); or morphin, gr. 1-16 to ⅛ (0.004—0.008). When the cough is dry, the morphin may be given in a mixture of which each dose contains also the dilute hydrocyanic acid, ♏ij to iij (0.12—0.18), or cherry-laurel water, ʒ ss (1.80). In all cases of persistent cough, the posterior nares, pharynx, and larynx should be examined, for it will often be found that a small fissure, granulation, or ulcer is the principal cause of the cough, and that a few applications of silver nitrate give the most gratifying relief. Inhalations of creosot, benzoin, or tar, or spraying the throat just before retiring with a strong (5—10 per cent) solution of menthol, often prevents the cough for several hours, and it may be repeated during the night. An elongated or congested uvula is an occasional cause of persistent cough.

Appetite.—The appetite is often much improved by the administration of the bitter tonics, especially the compound tincture of gentian, ʒ j to ij (3.75—7.50), shortly before meals. Moderate exercise in the open air is often sufficient.

Diarrhea.—In the early stages of the disease, diarrhea can usually be arrested with large doses of bismuth and regulation of the diet. Later, opium and astringents are generally required. The lead and opium pill, gr. iij to v (0.20—0.30), or gallic acid, gr. iij to x (0.20—0.65), may be employed. Salol, thymol, naphthol, and other intestinal antiseptics are also employed. It is sometimes necessary to reduce temporarily the quantity of fat taken by the patient or to restrict his diet to milk. If the intestine has become tubercular, however, it is useless to temporize, and morphin should be given at short intervals in sufficient doses to hold the diarrhea within bounds; it cannot be entirely overcome.

Hemoptysis.—The patient should be immediately placed in a recumbent posture and kept absolutely quiet. He should not be asked questions or permitted to speak. Morphin, gr. ¼ (0.0016), should be administered hypodermically, and an ice-bag should be placed upon the chest, over the point at which the bleeding is going on. When the pulse is full and bounding, it may be reduced by the cautious administration of aconite. One-half minim (0.03) doses of the fluid extract may be given every half-hour for perhaps two or three hours, but only until the effect becomes apparent. The most important point in the treatment, however, is to keep the lung at rest, and this can be done by the administration of an opiate in sufficient doses to keep the patient in a quiet sleep for 24 to 48 hours. When a large quantity of blood has been lost, the saline infusion should be employed; transfusion is sometimes necessary.

Strapping.—When the hemorrhage persists, this method may be advantageously employed: A pad is placed in the axilla and over the femoral veins, and secured with a strap drawn just tight enough to arrest the flow of venous blood without affecting the arterial circulation. Only two or three extremities should be thus treated at one time, and one compress should be removed to the free extremity every 15 minutes. In this manner a considerable quantity of blood can be withdrawn from the general circulation.

Pneumatic Treatment.—The pneumatic cabinet is now seldom employed, since its effect is believed to be only psychical. The patient was placed in a hermetically closed chamber and inhaled rarefied air. Complete expansion and collapse of the air-cells was believed to be secured at each respiration.

Specific Treatment.—The treatment with tuberculin has been almost entirely abandoned everywhere, on account of unfortunate results which were repeatedly observed after its administration. The new tuberculin (TR) has been found even more dangerous than the original. When used, it is injected in doses of 1-1000 to 1-500 milligram once or twice a week, gradually increasing the dose until slight reaction is obtained.

A serum obtained from animals, especially the horse, which have been rendered immune by repeated inoculations, has been tried during the last few years, but while it apparently exerts a beneficial effect on the fever and sweats, the results have not confirmed the hope that a cure had been found.

LEPROSY.

LEPRA.

Leprosy is one of the oldest known contagious diseases. Although it is now much less prevalent than it was several centuries ago, it is still common in India and China. It is rarely seen in the United States, but it is not infrequent in the West Indies; and in the Hawaiian Islands it is so common that the Island of Molokai has been reserved for the segregation of its victims. Foci of the disease have existed for a number of years also in New Brunswick and Nova Scotia, and leprous immigrants have recently reached Minnesota. The disease is occasionally seen in the South, especially at New Orleans, and among the Chinese along the Pacific Coast.

Definition.—A chronic infectious disease caused by the bacillus lepræ and characterized by the formation of granulomatous nodules in the skin and mucous membranes; changes in the nerves, which lead to anesthesia and trophic disturbances in the skin and other tissues; and constitutional disturbances.

Etiology.—The bacillus lepræ of Hansen closely resembles that of tuberculosis in form and staining, but a relation between the two organisms has been neither proved nor disproved. The leprosy bacillus has not been reproduced by inoculation, but Von Babés and Kalindero report that lepers react in the same manner as tuberculous persons to tuberculin. An individual susceptibility is believed to be necessary to the development of the disease. Men are more frequently affected than women and oftenest in the third decade of life. There is no racial immunity, but the dark races are more generally affected than the light.

Three modes of communicating the disease are recognized; namely, by inoculation, by contagion, and by heredity. The slow development of the lesions is the chief obstacle to the recognition of the source of infection.

(1) *Inoculation.*—A criminal in Hawaii was inoculated with leprous tissue in 1884, and died of the disease six years afterward, but there remained a possibility of his having contracted it from some other source. A leprous nodule at the point of inoculation was, however,

regarded as evidence of the success of the experiment. The disease is believed to have been conveyed by vaccination. It is an interesting fact that leprosy and mosquitoes arrived in Hawaii simultaneously, presumably from China.

(2) *Contagion.*—The bacilli are found in great numbers in the saliva and mucus from the mouth and nostrils of individuals having leprous lesions in those parts and in the open sores wherever situated. Hence the custom of kissing, or that of "rubbing noses," as practiced in Hawaii, has been looked upon as a common mode of conveying the virus. This mode is especially insisted upon by Sticker, who found the primary lesion commonly in the nose. Bacilli are found also in the urine and milk of lepers. The contagiousness of the disease has been recognized from the time of Moses and has been established by numerous examples.

It is probable that close contact is necessary to contagion, since physicians and nurses usually escape infection, although coming into ordinary contact with the disease for many years.

(3) *Heredity.*—The old belief that the disease is transmitted by heredity has been abandoned, since the lesions are not found at birth, and the opportunities of contagion during infancy and childhood are many.

The disease has long been attributed to the eating of fish, but there is little evidence that the bacilli can be derived from this source. It is doubtless often conveyed by cups, pipes, and other articles which have been used by the leper.

Morbid Anatomy.—The typical lesion is a granulomatous nodule of variable size, usually termed a tubercle, in the skin or a mucous membrane. This consists of epithelioid, lymphoid, and giant cells in a connective-tissue stroma, within and between which are numerous bacilli. In the anesthetic type of the disease the bacilli enter the peripheral nerve fibers and produce neuritis. They have been seen also within the cells of the spinal cord. The face and hands and in some cases the integument of a greater part of the body become disfigured by large nodules. Later these nodules show a tendency to soften; they frequently separate and form large ulcers which persist indefinitely and gradually invade surrounding tissues. The fingers and toes often slough away. The mucous membranes, particularly that of the larynx, the cornea, and conjunctiva, are frequently invaded and to a great extent destroyed. The internal organs, especially the lungs, liver, spleen, are often the seat of nodular formations.

Symptoms.—The first symptom observed in many cases is a catarrh, a "cold," or rhinitis, with sneezing and itching of the nose. This is thought to render active bacilli which may have been latent. Other prodromal symptoms, as digestive disorders, anorexia, sometimes epistaxis and prostration, are often observed. After this the disease develops in either of two forms, the tubercular or the anesthetic, but as a rule both forms are later found in the same patient.

1. **Tubercular Leprosy.**—The disease appears in the skin as an erythematous, distinctly defined, or macular rash, often with hyperesthesia. Burning pain and itching are commonly present. The color of the affected area becomes dark from a deposit of pigment, but later it often loses its color and becomes "white as snow" and totally anesthetic

(lepra alba). This stage may be repeated more or less constantly during the course of the entire disease. A remittent type of fever is almost invariably present in the beginning of this form of the disease. Nodules then make their appearance in the affected areas when they are situated on any part of the body except the scalp. They are especially large in growth upon the face, arms, and legs, often reaching the diameter of ¾ inch (2 cm.), and sometimes coalescing to form yet larger prominences. The hair of the face falls out. The eye and the mucous membranes of the mouth, pharynx, and larynx are often invaded. The face acquires a lion-like appearance, which has given the name leontiasis to the condition. More or less destructive ulceration often occurs in and between the nodules, ultimately destroying the bridge of the nose, the eyeballs, fingers, and toes. The appearance of the patient becomes most loathsome, but owing to the anesthesia the suffering is not usually great. The progress of the disease is extremely slow.

2. **Anesthetic Leprosy.**—This form of the disease, although undoubtedly of the same nature as the tubercular, frequently bears no resemblance to it. In it areas of hyperesthesia, pain, or numbness replace those of erythema. If the peripheral nerve trunks could be examined at this time, they would be found enlarged by the formation of small nodules which can sometimes be felt through the skin. Soon after the development of pain there may be a formation of small bullæ as a result of trophic changes in the skin. The hair of the affected area falls and the perspiration is arrested, so that the skin is always dry. Following the bullæ, ulcers are formed which soon become necrotic and fetid. Peculiar contractures of the hands and feet often occur owing to the affection of the muscles, and a perforating ulcer is not infrequently formed, especially on the foot. The fingers and toes, as in the tubercular form, become necrotic and drop off. An entire hand or foot is sometimes amputated. But the disease may last many years before these destructive manifestations appear, and, indeed, many years afterward.

Diagnosis.—The peculiar, erythematous, hyperemic, or anesthetic pigmented patches are characteristic of no other disease. After the nodules, ulcers, anesthesia, or contractures have become well developed, there is no possibility of error. The microscopic examination of an excised nodule establishes the diagnosis. The bacilli may be found in the nose or mouth. Since animals cannot be inoculated with them, this is one of the best methods of excluding tuberculosis.

Prognosis.—The disease is incurable. It almost invariably progresses to a fatal termination. The tubercular form usually lasts about 10 years, the anesthetic 20. Death sometimes occurs comparatively early from the involvement of the larynx and the development of aspiration pneumonia. Spontaneous cure is sometimes observed, especially in the early stages. The duration of the disease depends much upon the strength of the individual.

Treatment.—Isolation and segregation are the only prophylactic measures. Fortunately there are many institutions for this purpose in leprous countries. The treatment of the patient is confined to the administration of remedies to retard progress. General tonics, abundant nutritious food, and frequent bathing are the chief elements of treatment.

Arsenic, potassium iodid in large doses, gurjun oil in ♏x (0.60) doses, and chaulmoogra oil in doses gradually increasing to ʒiij (11.0) are most employed.

TETANUS.

LOCKJAW.

Definition.—An acute infectious disease caused by the tetanus bacillus and producing as its chief symptom a tonic spasm of the muscles, especially those of the jaw and neck.

Etiology.—The tetanus bacillus, the recognized cause of the disease, is a short, motile, club-shaped, spore-bearing bacillus, one extremity of which is expanded into a little head or bead. It is capable of independent existence in damp earth, as that of cellars and gardens, and in manure. Hence the disease is especially liable to follow injuries of the hand or foot by objects embedded in foul earth. It is a particularly common result of injuries by the toy pistol, the virus probably being present on the hands before injury. The disease often seems to be endemic in certain places. It has assumed epidemic proportions in some instances, particularly during wars. An idiopathic form of it is generally recognized, in which it develops in the absence of recognizable trauma. The tetanus of the newborn babe (trismus neonatorum) is generally regarded as of this character, but it is possible that infection sometimes takes place through the umbilicus.

Morbid Anatomy.—The lesions found after death are not distinctive. The bacillus has been found in the discharge from wounds, in the spinal cord, and in the peripheral nerves near the site of inoculation. The brain and cord are hyperemic; the nerve-cells are in a state of granular degeneration, and perivascular exudations are usually found.

Symptoms.—The incubation generally lasts from 10 days to two weeks. The onset may be marked by chilly sensations, rarely by a distinct rigor; but very often stiffness of the jaw and sides of the neck is first noticed on account of the interference with swallowing. The stiffness soon develops into a tonic spasm affecting the jaw and back of the neck, tightly closing the mouth and producing lockjaw. In most cases there is a contraction of the muscles of the face, raising the eyebrows, wrinkling the forehead, and drawing down the angles of the mouth, thus producing the so-called sardonic grin. The pupils are generally contracted. In children the spasm may be confined to the muscles of the face and jaw. In adults the muscles of the back of the neck or those of the entire spinal region become rigid, drawing the head back so that the body rests upon the head and buttocks (opisthotonos), or in extreme cases on the head and heels (orthotonos). In some instances the muscles of only one side are affected (pleurothotonos). A rigidity of the abdominal muscles, with flexion forward (emprosthotonos), has been observed.

In many cases the tonic spasms are interrupted by paroxysms in which the contracted muscles become still more rigid, or in which a clonic spasm occurs. The muscles jerk violently and often raise the patient from his bed. Rupture of a muscle is not unusual during the seizures. The suffering at these times is intense, although the tetanic contractions may not be painful. The skin is generally bathed in a cold

sweat. The spasms may be induced, as in hydrophobia, by the most trivial irritation, a draft of air, noise, or jarring of the bed. Fever may be present or absent. The temperature may reach 105° or 106° F. (40.5°—41° C.), and often runs up to 110° F. (43.3° C.) shortly before death. A peculiar form of the disease is that known as head-tetanus, which usually results from wounds on one side of the head. The symptoms are paralysis of the face muscles on the side of the injury, and difficulty in swallowing on account of rigidity of the jaw.

Diagnosis.—These conditions, following a trauma, could not well be mistaken for any other disease. In strychnin-poisoning, spasms occur, but they affect the entire body, including the arms, which are seldom affected in tetanus. In the intervals between the spasms, the muscles become relaxed.

In *hysteria* the convulsions are usually general in character and the temperature is normal. Complete relaxation occurs in the intervals.

In *tetany* the spasm involves the face, hands, and feet. If there is rigidity of the jaw, it is one of the late manifestations.

In *hydrophobia* there is difficulty in swallowing, but the jaw is not rigid and there is no opisthotonos.

Prognosis.—The mortality in traumatic cases is from 80 to 90 per cent. In acute cases death usually occurs in from one to seven days. When the onset is gradual and the case lingers for a week, the prognosis becomes more favorable. Infants invariably die.

Treatment.—When the patient is seen immediately after receipt of the injury, thorough cauterization may be practiced with hope of destroying the virus. Complete excision of the wound is a more certain measure. But after the disease has developed, little can be anticipated from either practice. Good results have followed the early injection of an antitoxic ("antitetanic") serum in a few cases. Bacelli and others have also employed successfully a 2 or 3 per cent solution of carbolic acid, the quantity of the acid used aggregating gr. ½ to ⅔ (0.03 to 0.04) in each 24 hours. The spasms should be controlled, as far as possible, by the administration of chloroform by inhalation, and by the internal administration of the bromids and chloral, or by morphin hypodermically if necessary. The patient should lie in a quiet, darkened room, carefully protected from injury during the seizures. Nourishment must be given by the rectum or by means of a catheter passed through the nostril.

INFECTIOUS DISEASES OF DOUBTFUL NATURE.

FEBRICULA.

SIMPLE CONTINUED FEVER, EPHEMERAL FEVER.

Definition.—A mild fever of short duration, due to a variety of causes and unattended with definite lesions. The term ephemeral fever is generally restricted to the class of cases which last only a day; while febricula or simple continued fever is applied to those which last from 2 or 3 to 14 days.

Etiology.—With reference to cause, there are three groups of cases—the gastrointestinal, the nervous, and the infectious.

(1) *Gastrointestinal Group.*—Most cases are due to a disturbance of digestion. This is especially frequent in children. It is doubtless often of the nature of ptomain-poisoning, due to the absorption of poisonous substances from the intestinal canal, following the ingestion of decomposed food.

(2) *Nervous Group.*—Nervous excitement or exhaustion is a common cause. This frequently results from exposure to excessive heat (insolation), worry, the excitement of receiving visitors while ill from another disease, or removal to a hospital. In nervous persons it may result from witnessing a serious accident to another, rarely from meditating upon some public calamity. It is possible that many cases in which fever follows exposure to foul odors or sewer gas are of this type, since those constantly exposed to them are rarely affected; other cases are perhaps due to ptomain-poisoning conveyed by the gas, and belong to the next group.

(3) *Infectious Group.*—This includes abortive cases of the infectious diseases, cases that begin with the initial symptoms of typhoid fever, measles, scarlet fever, or other infection, but gradually subside after a few days, and cases in which the exanthem fails to appear. In it may be included also the mild fever that occurs in some cases of rheumatism, pharyngitis, tonsilitis, bronchitis, and lymphadenitis.

Symptoms.—Fever is the essential symptom. In some cases it sets in abruptly, in others it is preceded by slight malaise. There may be slight chilliness, flushing of the cheeks, headache, pain in the back and limbs, loss of appetite, furred tongue, constipation, restlessness and insomnia. The fever ranges from 101° to 103° F. (38.0° to 39.0° C.). The usual accompaniments of temperature elevation are present, as thirst, depression, and concentration of urine. High fever, with delirium, is sometimes observed in children.

Diagnosis.—The diagnosis is to be arrived at by exclusion. Febricula should be the last item in the list considered. It is rather a name to be given to a febrile condition which is unaccompanied by pathological lesions to justify another diagnosis. Too often, perhaps, it implies an inability to arrive at a correct solution of the case. *Typhoid fever* is distinguished by the severity and duration of the prodromal symptoms and gradual rise of temperature; *malaria*, by its periodicity and the presence of the plasmodium; *sepsis*, by the presence of suppuration, repeated chills, and sweating. In the exclusion of other conditions, it is necessary to examine the ear, throat, chest, and all parts of the body. The skin should be examined daily for an exanthem.

Prognosis.—True febricula always terminates in recovery.

Treatment.—Rest in bed, the administration of a laxative, cooling, acidulated drinks, and liquid diet are generally all that is required. If the fever is above 101° F. (38.0° C.) it may be reduced by cool sponging, a bath of 75° F. (25.0° C.), or a few doses of phenacetin.

ACUTE FEBRILE JAUNDICE.

WEIL'S DISEASE.

Definition.—An acute febrile disease, probably a specific infection, accompanied by fever, jaundice, nephritis, and pains in the muscles.

Etiology.—A bacillus proteus fluorescens has been described as the specific cause by Jaeger, but there is still much doubt as to the identity of the disease. Some writers regard it as simply a febrile icterus due to one of several causes. It usually occurs in summer, oftenest in men between 30 and 50 years of age. It sometimes attacks a number of persons in the same locality, as a group of soldiers, or those of the same occupation, notably butchers.

Morbid Anatomy.—There is usually hyperemia of the intestines, liver, and spleen and a mild acute desquamative nephritis, lesions which cannot be regarded as typical.

Symptoms.—The onset is generally abrupt, with chill, headache, and pain in the back and legs and often in the muscles, especially of the cheek. The fever is remittent. Jaundice appears on the second day and may deepen on succeeding days. Gastrointestinal disorders are occasionally present. The stools are sometimes clay-colored. The fever lasts about a week and subsides by lysis; it does not usually exceed 103.5° F. (39.5° C.). Nervous symptoms sometimes develop, as restlessness, occasionally delirium or coma. The liver and spleen become enlarged, and albuminuria is present with casts, epithelium, rarely blood-cells.

Müller has described a form in which jaundice is absent. An epidemic of it occurred in 1891 near Breslau. It has been described also under the name Schlammfieber. Recovery usually occurs.

Treatment.—The treatment is that of catarrhal jaundice.

GLANDULAR FEVER.

Definition.—An acute infection of childhood distinguished by fever, and swelling of the cervical lymph-glands.

Etiology.—The specific cause is unknown. The disease has been recognized in epidemic form in a few instances, notably by Pfeiffer, in Germany, in 1889, and by West, in Ohio, in 1893. It attacks children between the seventh month and the thirteenth year. Outbreaks are sometimes limited to all the children of a single family.

Symptoms.—The onset is sudden, the first symptom usually being pain and tenderness over the region of the cervical lymph-glands, aggravated by moving the head. There are sometimes abdominal distress, nausea, constipation, and fever reaching 102° or 103° F. (39.0°—39.5° C.). On the second or third day the glands, especially those of the carotid region, and in the majority of instances also the postcervical, axillary, and inguinal, become much swollen and tender. The internal glands—the bronchial, tracheal, and mesenteric—may be affected. The tonsils are red and swollen in some cases. The liver and spleen are enlarged. Otitis, bronchitis, and acute nephritis have been observed as complications. The fever usually lasts only a few days; the swelling of the glands continues about two weeks, then slowly subsides. Suppuration is extremely rare.

The ***prognosis*** is good.

Treatment is symptomatic. Hot or cold applications may be employed to relieve pain. Park warns against purgative doses of calomel.

Diarrhea should not be too hastily checked. Tonics including iron are required in convalescence.

MOUNTAIN FEVER.

Definition.—A peculiar febrile condition induced by ascent to high altitudes. The condition is not specific, and there is very little reason to regard it as a distinct disease.

Etiology.—The only cause is the influence of the rarefied atmosphere. Several other diseases have been repeatedly reported as mountain fever. These have been for the most part atypical cases of typhoid fever. A mountain anemia due to the anchylostoma has also been described under this name.

Symptoms.—The ascent to moderate heights, less than 15,000 feet, causes in some persons a feeling of giddiness with moderate dyspnea, which precludes exertion. Ascent to greater heights, as described by Whymper in his ascent of Mt. Chimborazo, causes at a height of 16,664 feet headache, gasping for breath, dryness of the throat, intense thirst, loss of appetite, and general malaise, with slight elevation of temperature, in this instance 100.4° F. (38° C.). The condition lasted for three days. Epistaxis and hemoptysis occasionally occur at even lower levels. Zangger has recently shown that rapid ascent to high altitudes is attended also with great danger of cardiac thrombosis and pulmonary embolism, especially in elderly persons. These seizures often do not develop until two or three days after descent.

Treatment.—The febrile condition calls only for rest. Return to a lower altitude gives immediate relief. The system becomes accustomed to the atmospheric condition in a few days.

"SPOTTED FEVER OF THE ROCKY MOUNTAINS.

BLACK FEVER, BLUE DISEASE.

Under this provisional name an interesting disease has been described as observed in the Bitter Root and Solo valleys of Montana and Idaho, by E. E. Maxey, in the *Medical Sentinel*, October, 1899, and by L. B. Wilson and W. M. Chowney, in the *Journal* of the American Medical Association, 1902, Vol. xxxix. p. 131.

Definition.—An acute infectious disease of the Rocky Mountain region, characterized by chills, fever, prostration, slight jaundice, and an eruption of macules which often coalesce to form a marmorated surface.

Etiology.—The specific cause is believed to be a protozoön found in the red blood-corpuscles. The medium of infection is thought to be a species of tick, and the gopher perpetuates the disease. It is not contagious. It occurs only in a limited area of 40x20 miles and only from March to July. It attacks persons of any age and of either sex; the Indians are apparently immune.

Morbid Anatomy.—The marmorated appearance of the skin persists, and the bites of the tick can usually be found. The liver and spleen are moderately enlarged; the other organs normal. Petechiæ are some-

times found in the pericardium. The hematazoön is found in the blood, liver, and spleen.

Symptoms.—In some cases there is a short period of malaise. The invasion is with marked chill, which recurs at irregular intervals with decreasing severity. In the beginning there is severe pain in the head, back, bones, joints, and muscles, and great prostration. Constipation is usual. The skin is dry, often puffy, but it does not pit. The tongue is heavily coated, becoming brown and fissured. Sordes are pronounced. The appearance is like that of typhoid fever. The temperature rises after the chill to 103° or 104° F. (39.5°—40° C.) and gradually increases for five to seven days to 105° or 107° F. (40.5°—41.5° C.), and declines by a lysis of about two weeks. It sometimes becomes normal or subnormal shortly before death. Low muttering delirium may occur at this stage. The pulse, at first slow and strong, increases up to 150 and becomes feeble. Both pulse and respiration are rapid beyond the ratio to the temperature. Nausea and vomiting generally develop in the second week and continue to the end in fatal cases. There is moderate anemia and leucocytosis.

The macular, rose-colored eruption appears by the second to the fifth day on the wrists, ankles, and back and rapidly spreads to the entire body. The spots are at first discrete, 1 to 5 mm. in diameter, but soon become dark and coalesce, giving the skin a mottled appearance, especially on dependent parts. It sometimes remains discrete; occasionally it is absent in mild cases. Slight jaundice is present, noticeable especially in the conjunctivæ. The skin becomes glazed in the second week, and a slight desquamation begins in the third. The eruption vanishes with the fever. Gangrene sometimes sets in shortly before death, in the elbows, fingers, toes, lobes of the ear, and elsewhere. Hypostatic pneumonia may develop. The disease lasts about four weeks.

Prognosis.—Mild cases are encountered, but in well-marked cases the mortality is about 80 per cent.

Treatment.—No specific treatment has been proposed, further than that of other febrile diseases, particularly typhoid fever.

MILIARY FEVER.

SWEATING SICKNESS.

Definition.—An acute epidemic infection characterized by profuse sweating and an eruption of miliary vesicles.

Etiology.—The disease has not been recognized in this country, but it occasionally assumes the form of a limited epidemic in France, Italy, and Austria. A fatal type of it prevailed in England in the fifteenth and sixteenth centuries. It is more frequently encountered in females. The specific cause is not known. The epidemic usually spreads with great rapidity.

Symptoms.—The onset is sudden, with fever, frequent sweating, epigastric oppression, and marked prostration. An erythematous rash appears, and by the third or fourth day this gives place to an eruption of miliary vesicles, chiefly on the neck and axillæ, sometimes on the mucous membranes. In mild cases the fever is slight, but in severe

cases there are evidences of intense toxemia, high fever, profound prostration, delirium, and sometimes hemorrhages. Death may occur within a few hours. Pregnant women abort and usually die. The average mortality is only 8 or 9 per cent, but in epidemics it has been higher.

Treatment.—The treatment is chiefly symptomatic. Quinin is generally prescribed.

INFECTIOUS DISEASES COMMON TO MAN AND LOWER ANIMALS.

GLANDERS.

FARCY.

Definition.—An infectious disease caused by the bacillus mallei usually affecting the horse or other animal of the same species, and occasionally acquired by man through inoculation. It occurs in two forms: True glanders, which affects the nasal cavity, and farcy, in which nodules appear beneath the skin. Either form may be acute or chronic.

Etiology.—The bacillus mallei is a short, non-motile, rod which closely resembles the bacillus tuberculosis. It is found in the nodules, ulcers, and abscesses, and in the discharges from them, and possibly in the blood and urine. Infection usually occurs through the inoculation of an abraded surface by the nasal mucus of an affected horse, ass, or mule. It may be caused by the inhalation of the dried mucus or by transmission from man to man.

Morbid Anatomy.—A granulomatous nodule of variable size is the characteristic lesion. In man these are usually small and consist of lymphoid and epithelioid cells, within and between which the bacilli may be seen in microscopic section. They rapidly disintegrate and leave ulcers and abscesses in the skin, subcutaneous tissue, muscles, and viscera.

Symptoms.—An acute and a chronic form are recognized in both the true glanders and in farcy. The symptoms usually appear within three or four days after inoculation, less frequently after a week or two.

1. *Acute Glanders.*—The first manifestations of the disease are generally of a constitutional character—fever, with headache, and pain in the extremities, sometimes suggestive of typhoid fever. The point of inoculation becomes swollen, painful, and surrounded by an area of hyperemia and lymphangitis. Within two or three days small nodules appear in the nose, which break down and discharge a saneous, mucopurulent fluid. The nose and face become greatly swollen. Papules and vesicles appear singly or in groups on the face, sometimes also about the joints. These soon change into pustules, like those of smallpox, but larger, as a rule. Ulcers then form, which sometimes lead to necrosis, with an exceedingly offensive discharge. The nearest lymph-glands become much enlarged and the disease often extends to the pharynx, mouth, larynx, and bronchi. Broncho-pneumonia may follow. The constitutional symptoms are severe and often assume a pyemic character, with chills, fever, and sweating, vomiting, diarrhea, and profound prostration.

2. *Acute Farcy.*—The first manifestation is usually a local, painful swelling at the point of inoculation, with hyperemia and lymphangitis.

Nodules (farcy buds) form, especially along the course of the lymphatics, and rapidly produce abscesses. Abscesses may form also about the joints and in the muscles. The nose is not affected, and the superficial ulcers of the skin are absent. The constitutional symptoms are those of sepsis.

3. *Chronic Glanders.*—This form is rare and difficult of diagnosis. There is usually a fetid, saneous, or mucopurulent discharge from ulcers in the nose which resembles that of syphilitic ozena. Subcutaneous nodules may also form, but they produce little local or constitutional reaction. The larynx is sometimes affected.

4. *Chronic Farcy.*—The nodules generally form on the arms or legs, then break down into ulcers and abscesses which persist for months. Local and constitutional disturbances follow.

Prognosis.—The acute forms of the disease usually terminate fatally within 8 to 15 days. Recovery from farcy has been reported. Chronic glanders terminates fatally in from 2 to 3 weeks as a result of bronchopneumonia or sepsis. Chronic farcy may last for several months, ultimately ending in death from exhaustion or sepsis. Recovery sometimes occurs.

Treatment.—The site of the original inoculation should be promptly destroyed with the hot iron or pure nitric acid. The farcy buds should be incised and cauterized or injected with a 1:20 solution of carbolic acid. In glanders the nostrils should be frequently irrigated with a 1:1000 solution of mercuric chlorid. The strength of the patient must be supported by the free administration of stimulants, animal food, and full doses of strychnin. A toxin, mallein, prepared from cultures of the bacillus, has been employed in the treatment of animals and for diagnostic purposes.

HYDROPHOBIA.

RABIES.

Definition.—An acute infection of man and animals, generally communicated through inoculation and manifested by spasms of the muscles of deglutition and respiration, with other indications of disturbance of the central nervous system.

Etiology.—The disease is peculiar to warm-blooded animals, particularly the carnivora. The dog, cat, wolf, fox, and skunk are especially liable to it, but many other animals are susceptible to inoculation, notably the horse, ox, and pig. The disease is probably more prevalent in Russia and Siberia than elsewhere. It is communicated to man chiefly through the bite of a rabid animal. Bites on the exposed parts of the body are especially dangerous; since the clothing, when penetrated by the teeth, may remove much of the virus. The saliva of a rabid animal, whether violent or "dumb," is capable of inoculating an abraded surface.

The specific micro-organism has not been discovered, but the toxin has been separated from the nerve substance, saliva, and other secretions of affected animals. The virus of the wolf is said to be first in order of virulence, that of the cat second, that of the dog third. Children are more susceptible than adults. It has been estimated that only 15 per

cent of the persons bitten take the infection. The disease is somewhat more prevalent in the hot weather of summer, but it may be encountered at any season.

Morbid Anatomy.—The lesions are found almost exclusively in the brain, medulla, and cord, but are not specific in character. There may be congestion of the nerve tissue, with perivascular exudation of leucocytes and minute hemorrhages, especially in the medulla. More or less extensive hyperemia of the mucous membranes of the respiratory and digestive tracts may be found. The presence of the virus in the nerve tissues may be demonstrated by the inoculation of rabbits.

Symptoms.—The incubation is six weeks or longer; it is shorter in children than in adults. The more extensive the wound and the deeper the penetration of the tissues, the more promptly does infection occur. The course of the disease may be studied under three stages, known as the initial or premonitory, the spasmodic, and the paralytic.

1. *Initial Stage.*—A feeling of numbness, irritation, or pain in the cicatrix of the wound is often the first symptom. The patient becomes morose, melancholy, depressed, and irritable; he loses his appetite and cannot sleep. He is particularly sensitive to light and sound. His voice becomes husky and he has difficulty in swallowing. There may be slight fever.

2. *Spasmodic Stage.*—A distinct spasm of the muscles of the larynx and mouth develops and increases the difficulty of deglutition. The spasm is provoked by efforts to swallow or by anything that suggests it, particularly by the sound of running water; hence the fear of water (hydrophobia), which is often a prominent symptom. Extreme anxiety is depicted in the face of the patient and a sense of suffocation adds to his suffering. Maniacal manifestations are frequently engrafted upon the spasmodic seizures, during which peculiar sounds are uttered and saliva is sometimes ejected, but the patient's mind is clear in the interval and he frequently converses intelligently in regard to his condition; he may even express fear of causing injury to others. There is rarely any attempt to inflict injury. In some cases the temperature remains normal or is slightly subnormal, in others it rises to 102° or 103° F. (39.0°—39.5°C.). This stage usually lasts from one to three days, but death may result from asphyxia before its expiration.

3. *Paralytic Stage.*—In this the spasms cease and the patient sinks into a state of unconsciousness; the heart becomes more and more feeble until death supervenes.

Pseudohydrophobia (Lysophobia.)—This is a neurotic or hysterical condition in which the symptoms of hydrophobia are simulated, usually by a person who has been bitten by an animal supposed to be rabid. It may develop months or years after the injury. In most instances the picture is overdrawn and there is a display of dramatic action not seen in the real disease. The patient believes his condition serious and so declares it. Paroxysms occur in which he grasps at his throat and asserts his fear of water and inability to swallow. He often imitates the sound of the animal and foams at the mouth. The condition lasts longer than hydrophobia, as a rule, but it ends in recovery. Death may result, however, from the neurasthenic condition, which is usually the predisposing cause of the attack.

Diagnosis.—The chief difficulty is the exclusion of pseudohydrophobia. The apparent severity of the symptoms is often less than in the latter condition. When the person has been bitten, the animal should be killed and the medulla removed for investigation. Subdural inoculation of a rabbit produces the disease in 20 days. When this is not practicable, the animal should be kept in comfortable quarters and its condition watched.

Prognosis.—The mortality, as stated by different authorities, varies from 40 to 80 per cent. When the disease becomes fully developed it is almost invariably fatal.

Prophylaxis.—Although the disease is uncommon in this country, it could be still further prevented, as in England and Germany, by the muzzling of dogs and the extermination of useless curs. Preventive inoculation has been practiced, particularly in France. Pasteur found that by passing the virus through successive inoculations in rabbits its virulence was so increased that the period of incubation was reduced to seven days. Preservation of the spinal cords of these rabbits in a drying-chamber for from 12 to 15 days progressively reduced the virulence. After testing the method on animals he applied it to man, using on successive days a stronger virus until the individual became immune even to the virus previously inoculated by a rabid animal. This method is still employed, but notwithstanding the continued favorable reports, many authorities doubt its utility.

Treatment.—The wound should be immediately cleansed and thoroughly cauterized with caustic potash or pure carbolic acid, and treated as an open wound. After the disease has developed, nothing can be done further than to relieve the suffering. The room should be quiet, moderately dark, and free from drafts. The spasms may be moderated by morphin hypodermically or chloroform inhalation. Milder remedies are useless. A cocain spray has been found to relieve the spasm and sometimes to enable the patient to swallow. When this fails, nourishment must be given by the rectum. Treatment with the serum of animals rendered immune has proved unsatisfactory, except perhaps in animals of the same species.

ANTHRAX.

SPLENIC FEVER, CHARBON, WOOL SORTERS' DISEASE, RAG PICKERS' DISEASE.

Definition.—An acute infectious disease caused by the bacillus anthracis, in some cases presenting lesions in the skin, in others affecting the internal organs.

Etiology.—The disease is very prevalent and highly fatal among horses, sheep, and cattle in some localities. It is much less common in America than in Europe and Asia. It is communicated to man by accidental inoculation, as a rule. The bacillus is one of the most familiar of those used for laboratory experimentation and need not be described. The spread of the disease is due chiefly to the remarkable vitality of the spores, which are capable of retaining life for years in a condition of dryness and are not destroyed by a temperature of 212° F. (100° C.) for several minutes. The victims of infection are, as a rule, persons

whose occupation requires them to come into contact with infected animals or to handle the products of such animals. It is therefore most frequently seen in hostlers, dairymen, shepherds, farmers, butchers, sorters of hair and wool, mattress-makers, tanners, and furriers. It has been produced by eating the meat or drinking the milk of diseased cattle. The inoculation ordinarily occurs at an abraded point on the skin or a mucous membrane, but may result from the inhalation of dust, particularly that from infected horsehair. Insects are regarded as possible carriers of the bacillus. The poison cannot penetrate the unbroken skin, but a lesion is not thought to be necessary when the respiratory or intestinal mucous membrane is the avenue of entrance.

Symptoms.—The incubation does not exceed three days. There are two principal types of the disease, one exhibiting external, and the other internal, lesions.

1. **External Anthrax.**—This form is again divided into two kinds, malignant pustule and malignant edema.

(*a*) *Malignant pustule* is the most frequent form of the disease. It is seen especially on the hands, neck, and face, sometimes on the lower extremity, as a result of direct inoculation. In a few hours, or not until a day or two, after inoculation, a burning and itching sensation is felt. A papule develops having a purple center, and this is soon converted into a vesicle from which a bloody serum is discharged. By the end of 36 hours the area of the original vesicle is converted into a bluish black necrotic mass which is usually surrounded by a hyperemic zone studded with small vesicles. A painful lymphangitis and phlebitis form around this, with intense swelling of the tissues. The resulting constitutional disturbances are generally severe. The temperature rises rapidly, often to 105° F. (40.5° C.), and there are usually persistent vomiting, profuse sweating, and great weakness. Delirium sometimes supervenes, but the mind frequently remains clear. Many writers refer to the absence of anxiety on the part of the patient, even when his condition is critical. The temperature sometimes declines and becomes subnormal, while the patient sinks into a collapse that usually proves fatal. Death often occurs within the first three or four days. When the symptoms are of only moderate severity, recovery is possible. The pustule sloughs away and the wound heals by granulation. In the mildest cases the swelling is slight, and the original papule dries into a crust that separates in the course of a few days.

(*b*) *Malignant Edema.*—This is seen, for the most part, in loose connective tissue, as on the eylid, lip, neck, forearm, hand, or thigh, sometimes in the mucous membrane of the mouth or tongue. No papule or vesicle is formed; the skin may not be reddened, but it becomes intensely edematous. The infiltration rapidly spreads; bullæ sometimes form upon it and lead to gangrene and sloughing. Intense sepsis develops, sometimes preceding the local manifestations.

The ***diagnosis*** of either form is based upon the appearances described and the occupation of the individual. The bacilli may be found in the fluid from the pustule or edematous area, and later, sometimes only immediately before death, in the blood.

The ***prognosis*** is especially grave when the pustules are situated on the head or neck. The mortality is then 25 per cent; when the lower

extremity is affected, it is 5 per cent. The edematous form is inevitably fatal.

2. **Internal Anthrax.**—There are also two kinds of internal anthrax, designated intestinal, and pulmonary or cerebral. They may occur independently or they may be associated with malignant pustule or malignant edema.

(*a*) *Intestinal Anthrax.*—This type was formerly known as mycosis intestinalis. It is the result of eating the meat or drinking the milk of infected cattle, of inhaling and swallowing the bacilli from different sources, or of inoculation of the mouth. The invasion is usually acute, with chill, fever often reaching 106° F. (41.0° C.), languor, and severe pain in the head, back, and legs. Gastrointestinal disturbances soon follow, especially vomiting and diarrhea, often bloody in character. Dyspnea, cyanosis, and great restlessness are present. Muscular spasms or convulsions often supervene. There may be hemorrhages from the mucous membranes, and petechiæ or other hemorrhagic lesions may be found in the skin and gums or other mucous membranes. The blood is dark and slow to coagulate. The disease always terminates fatally in from two to seven days.

(*b*) *Wool-Sorters' Disease.*—This form results from the inhalation or swallowing of dust impregnated with the bacilli from infected wool or hair, often after it has been imported from distant countries. There is often no external lesion. The onset is with chill and great prostration, pains in the back and legs, oppression in the chest, rapid respiration, cough and dyspnea or a sense of suffocation (pulmonary anthrax). The fever is usually moderate, 102° to 103° F. (39.0°—39.5° C.). The pulse becomes rapid and feeble. Violent delirium sometimes develops. Bacilli have been found in the capillaries of the brain (cerebral anthrax). In less severe cases there may be diarrhea and other symptoms on the part of the alimentary canal. The patient soon sinks into a fatal collapse. Tetanic spasms often occur shortly before death. The rag-picker's disease is usually of this type.

The ***diagnosis*** is difficult unless the probability of infection is known. The occupation of the patient should always arouse suspicion, and the bacilli may generally be found in the sputum, blood, or other fluids of the body.

Prognosis.—The prognosis of anthrax is always grave. In the pustular form, early treatment may save the patient, but, after general systemic infection has occurred, death is inevitable.

Prophylaxis.—Theoretical methods of prophylaxis, involving the disinfection of wool and hair and the use of antiseptic solutions by the workmen, are difficult of application among the classes usually affected. Diseased animals should be destroyed and their carcasses should be burned. Burial is unsafe. Preventive inoculation of animals with an attenuated virus has been practiced in France with much success.

Treatment.—This is for the most part surgical. The point of inoculation should be immediately destroyed with nitric acid, carbolic acid, or the actual cautery. The entire area may be excised early, and the resulting wound thoroughly cauterized. If too extensive, the area may be freely incised with repeated cross-cuts and treated with strong caustics. Subcutaneous injections of carbolic acid into the surrounding skin

have proved successful in many cases. The system is remarkably tolerant of the acid in this disease; 15 grains (1.0) have been injected in a day. The application of very hot poultices or ice-bags at short intervals seems to inhibit the growth of bacilli in the superficial tissues, since the more resistant spores do not form in the body. Extensive lesions should be treated with mercuric chlorid (1:500) and covered with an ice-bag or poultice. When the legs are edematous, they should be incised to evacuate the poisonous fluid. In the internal forms of the disease we can only hope to contribute to the comfort of the patient.

ACTINOMYCOSIS.

Definition.—A chronic infection caused by the streptothrix actinomyces, or ray fungus, frequent in cattle, but rare in man.

Etiology.—The fungus is found in the pus from the affected area, in the form of bright yellow granules from 0.5 to 2.0 mm. in diameter. These are composed of masses of cocci and radiating threads with bulbous extremities. Several atypical, polymorphous forms of the ray fungus have been recognized, some of which approach very closely in morphology to the bacilli. Infection generally takes place in the skin directly from a diseased animal or in the mouth through eating infected meat or cereals. The presence of such lesion as a carious tooth is necessary to the entrance of the fungus. Men are more exposed to infection than women.

Morbid Anatomy.—Granulomatous tumors are formed consisting of lymphoid and epithelioid cells with an occasional giant-cell. These sometimes disintegrate to form abscesses, but they do not involve the neighboring lymphatics.

Symptoms.—The disease invades the alimentary canal, the lungs, the skin, or the brain, and produces symptoms peculiar to each location.

1. *Alimentary Canal.*—The jaw is usually affected. It becomes greatly swollen, and sinuses burrow from it into the face and neck. The tongue, intestine, or liver may be involved primarily or by metastasis. The fungus has been found in a diseased appendix and in a pericecal abscess.

2. *The Lungs.*—Three forms of pulmonary actinomycosis are recognized: (*a*) A chronic bronchitis in which the fungus appears in the sputum. (*b*) A miliary form in which nodular masses of the fungus are surrounded by granulation tissue, resembling miliary tuberculosis. (*c*) Extensively destructive lesions causing bronchopneumonia, interstitial changes, and large abscesses. The ribs, sternum, and vertebræ may be eroded.

3. *The Skin.*—Suppurating nodules are formed which lead to extensive ulcers and fistulæ of an exceedingly chronic character.

4. *The Brain.*—The brain may be involved primarily or by metastasis. Few cases have been observed. The symptoms are those of tumor or abscess.

All forms of the disease are accompanied by constitutional disturbances of a septic nature. Persistent cough distinguishes the pulmonary form; headache, epilepsy, or other symptoms of brain tumor, the cerebral form. Various metastases develop.

The ***diagnosis*** depends upon the discovery of the fungus, which is readily recognized.

Prognosis.—Cases that are within the reach of surgery may recover. The internal forms of the disease are generally fatal.

Treatment.—The surgical treatment consists chiefly in the incision and thorough evacuation of abscesses and the removal of diseased bone and other tissues. Potassium iodid, gr. lx (3.88), daily has been recommended as curative in internal cases. It should not be relied upon to the exclusion of surgical measures.

PSITTACOSIS.

Definition.—An acute infectious disease of birds, which, when contracted by man, produces lesions in the respiratory organs, with fever and other manifestations of toxemia.

Etiology.—The disease has been studied especially in Paris, where it was introduced in 1892 by a cargo of parrots and parrakeets. The bacillus of Nocard is believed to be the specific cause. This is a rapidly motile rod having from 8 to 12 cilia. It belongs to the paratyphoid or paracolon group. It differs from the colon bacillus, and at the same time resembles the typhoid, in being more virulent, actively motile, in not fermenting lactose or coagulating milk, and not producing indol. It differs from the typhoid and resembles the colon in its growth on gelatin and potato or upon old cultures of the typhoid bacillus. The disease is communicated directly from the bird, by the feathers, cage, or other articles soiled with the dejections, less frequently from another individual.

Morbid Anatomy.—The lesions are in the beginning those of bronchial catarrh, occasionally accompanied with a membranous formation like that of diphtheria in appearance. Later there is the formation of isolated areas of consolidation in both lungs like those of bronchopneumonia.

Symptoms.—The incubation is probably from 7 to 12 days. The invasion is preceded by malaise, headache, pains in the back and limbs, sometimes by epistaxis, nausea, and vomiting. Constipation is usually present, occasionally diarrhea. The onset is often sudden with a chill or chilly sensations and rapid rise of fever, reaching 103° or 104° F. (39.5° or 40.0° C.) on the second day, and profound prostration. The pulse is accelerated. The fever subsides on the fourth or fifth day, and there is an afebrile period of variable duration. This is followed by a return of the fever, and this in turn by an afebrile period. Thus the disease continues sometimes for 15 or 20 days. The last defervescence lasts two or three days. The physical signs are those of bronchopneumonia. Delirium, usually mild, occasionally violent, may be present. The spleen is slightly enlarged, and febrile albuminuria is present. The course of the disease is like that of relapsing fever, but the relapses are attributed to the involvement of additional areas of the lungs.

Prognosis.—The mortality varied in the cases recorded between 20 and 40 per cent.

Treatment.—The patient should be isolated in a well-ventilated room to prevent spread of the infection. The treatment is wholly sympto-

matic. The strength is to be maintained by nutritious liquid diet and free stimulation. Cold baths or sponging is recommended for the reduction of the temperature.

MILK SICKNESS.

Definition.—An infectious disease of man and cattle, characterized by constipation and severe nervous phenomena. It was formerly prevalent west of the Alleghanies. In cattle it is known as "the trembles."

Etiology.—The specific cause is unknown. It is supposed to reside in the earth, since the disease has almost entirely disappeared with the clearing of the forests. The milk and flesh of affected cattle are extremely poisonous to other animals, hence it is believed that meat, milk, butter, and cheese are the chief carriers of infection to man. Adult males are most frequently affected.

Symptoms.—The incubation is short and characterized by malaise, headache, and indigestion, followed by burning pain in the stomach and vomiting. There is slight fever, great thirst, and usually obstinate constipation. The tongue becomes dry and swollen and the breath has a characteristically fetid odor. A typhoid state sometimes supervenes, with great restlessness, irritability, sometimes delirium, which may terminate fatally in convulsions or coma. The duration of illness is variable. Death may occur in two or three days, or after three or four weeks.

Diagnosis.—The diagnosis is made with difficulty, especially the exclusion of ptomain and fungus poisoning, unless the source of infection can be traced to cattle having the trembles.

Treatment.—This is purely symptomatic. The constipation should be promptly overcome in order to eliminate the poison. Stimulants are indicated.

FOOT-AND-MOUTH DISEASE.

EPIDEMIC STOMATITIS. APHTHOUS FEVER.

Definition.—A highly virulent, febrile disease of cattle, occasionally contracted by man in the form of a vesicular eruption of the mouth or a miliary, sometimes pustular eruption of the hands.

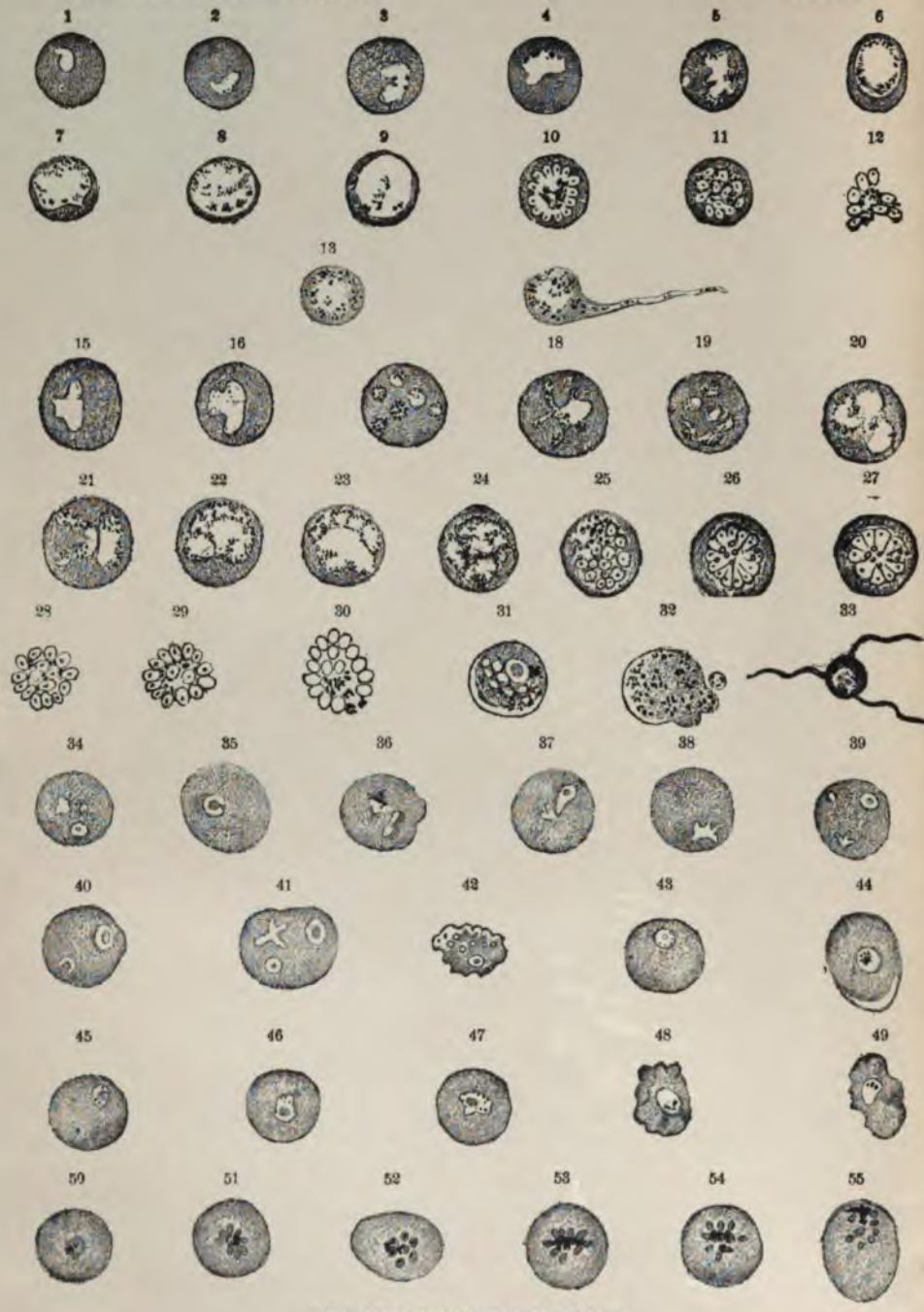
Etiology.—The disease is most frequent in cattle, sheep, goats, and swine. Among cattle it has been especially severe in Texas. It is contracted by those who work with the diseased animals, especially by milkers, but sometimes also through drinking the milk or eating the butter from the affected cows. It is believed to be caused by an exceedingly small micro-organism.

Symptoms.—After an incubation of four or five days, the disease begins by a more or less distinct chill, with fever, and followed by prostration. Vesicles similar to those of aphthous stomatitis soon form upon the mucous membrane of the mouth, including the tongue and lips, and often upon the pharynx. All the associated symptoms of the latter disease, including redness, heat, and pain, are present, and the flow of saliva is greatly increased. The hands and fingers usually show a papular or pustular eruption.

Diagnosis.—The disease is usually recognized without difficulty, owing to the prevalence of the affection among cattle in the vicinity.

Prognosis.—Recovery within a week or ten days is the rule, but fatal cases have been reported.

Treament.—A serum produced by Löffler seems to be protective to animals. The best means of protecting human beings are the thorough boiling of the milk and the use of antiseptics by those having to come into contact with diseased cattle. The disease of the mouth should be treated as an ordinary aphthous stomatitis.



THE PARASITES OF MALARIA.

(Marchiafava and Bignami in "Twentieth Century Practice.")

EXPLANATION OF PLATE V.

FIGS. 1–14.—The hematozoa of Quartan Fever; Figs. 1–9, Progressive endoglobular development of the quartan parasite; Figs. 10 and 11, Endoglobular fission forms; Fig. 12, Free sporulation; Figs. 13 and 14, Free pigmented forms, one flagellated.

FIGS. 15–33.—Hematozoa of Tertian Fever: Figs. 15–24, Progressive endoglobular development of the tertian parasite; Figs. 25–27. Endoglobular fission forms; Figs. 28–30, Free sporulation; Figs. 31–33. Free pigmented forms, one flagellated.

FIGS. 34–55.—Hematozoa of Estivo-autumnal (quotidian) Fever; Figs. 34–50, Endoglobular development of the quotidian parasite; Figs. 42, 48 and 49, parasites in altered red blood corpuscles (brassy bodies); Figs. 51–55, endoglobular forms in sporulation.

SECTION II.

Diseases Due to Animal Parasites.

PROTOZOAN DISEASES.

MALARIA.

INTERMITTENT FEVER, CHILLS AND FEVER, FEVER AND AGUE, SWAMP FEVER, PALUDISM.

Definition.—Infection by the plasmodium malariæ of Laveran, with the production of a febrile disease, of which the following are the principal forms: (*a*) Intermittent fever, in which there are paroxysms of chill, fever, and sweating at regular periods; (*b*) estivo-autumnal fever, a continued, remittent fever; (*c*) pernicious, rapidly fatal forms; and (*d*) a malarial cachexia, a chronic form showing profound anemia and enlargement of the spleen.

Etiology.—1. The plasmodium malariæ, discovered by Laveran in 1880, is the specific cause. 2. The only demonstrated means by which the infection is produced in man is through inoculation by the mosquito. Egbert, of Honduras, maintains that fleas may also introduce the parasite, since he has observed the disease in localities where this insect is abundant and mosquitoes unknown.

The Parasite.—The plasmodium is a motile, protozoan parasite belonging to the hematozoa, or hemocytozoa (Thayer). Our knowledge of it is limited almost entirely to the phenomena which occur within the human body and that of the mosquito. Attempts to cultivate it in artificial media have failed. It is regarded as asexual in man, but it is believed to attain sexual maturity in the mosquito. The cycle of its existence is probably completed in the water of swamps and stagnant pools, which are at the same time the birthplace and grave of the mosquito. Similar protozoan forms have been found in the red blood corpuscles of fish, turtles, snakes, and birds inhabiting the same regions.

Three forms or species of the plasmodium have been recognized: (1) The parasite of tertian fever, (2) the parasite of quartan fever, and (3) the parasite of estivo-autumnal fever. All of these present different appearances peculiar to stages in their development. (See Plate V.)

The Tertian Parasite.—The young parasite enters the red blood-corpuscle in the form of a small, nucleated, hyaline, ameboid body. As it grows it almost completely fills the corpuscle. At the expense of the hemoglobin of the cell, it accumulates pigment which may be seen as small, dark granules in active motion within it, but ultimately becoming collected into a close mass, usually near the center of the parasite. As

a result of this action, the blood-corpuscle becomes decolorized and expands to accommodate the increasing size of the plasmodium. A process of sporulation now takes place. The protoplasm of the ameba becomes opaque, and soon radial striations may be seen extending inward from the periphery nearly or quite to the mass of pigment at the center. The original parasite is thus divided into from 12 to 20 small segments, each of which is destined to become a young plasmodium. The enveloping red blood-corpuscle has been destroyed by this time and the young parasites are set free in the blood. One of the most remarkable features is that the parasites of the same group or family all mature at the same time, and thus give the characteristic periodicity to the clinical manifestations. The cycle of the tertian parasite within the human being is completed in 48 hours, hence the paroxysms occur at regular intervals of this length, or on alternating days. It not infrequently happens that two groups of the same parasite are present at the same time, one maturing every day and producing a quotidian type of the disease.

The Quartan Parasite.—This is very similar to the tertian, but it is somewhat smaller, less active, and the pigment which it accumulates is usually more abundant and darker in color. The blood-corpuscle, instead of expanding, appears shrunken around it and acquires a greenish shade. In the process of sporulation, only from 5 to 10 segments are formed, and these usually collect around the pigment mass in the form of a rosette. Their cycle is of 72 hours' duration. When only a single group is present, the paroxysms occur on every fourth day; when there are two groups, the paroxysms occur on two succeeding days, followed by an interval of one day. More than two generations with corresponding irregularity of clinical manifestations are sometimes observed.

The Estivo-Autumnal Parasite.—This parasite is described under two forms: (*a*) The quotidian estivo-autumnal parasite, and (*b*) the malignant tertian estivo-autumnal parasite. Each of these appears at first as a small, hyaline body, smaller than that of the tertian parasite, then as a pigmented body, and later as a segmenting body. Crescentic, ovoid, and flagellate bodies also appear later. (See Plate VI.) Marchiafava considers them the beginning of a life cycle which is completed in the mosquito. The flagellate forms may be seen also in connection with the tertian and quartan parasites and is believed to be concerned in the process of reproduction.

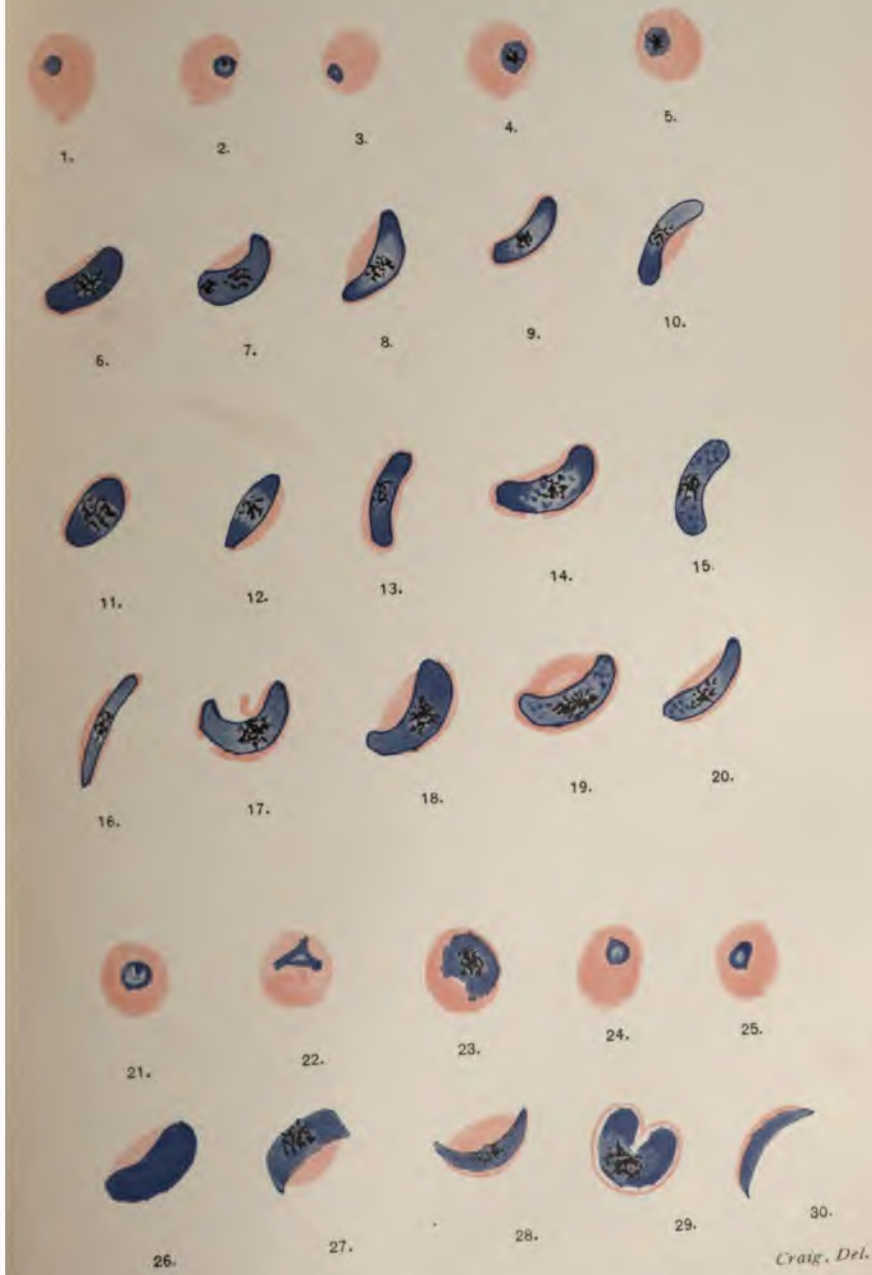
Phagocytes.—The liberation of segments gives rise to an energetic phagocytosis. The polynuclear neutrophile cells attack and devour with great activity the segments, flagella, pigment, and fragments of the disintegrated corpuscles. A single phagocyte may envelop several parasites.

The Mosquito.—So far as is now known, the plasmodium undergoes development only in the species of the mosquito belonging to the genus Anopheles. Of these there are two varieties: (*a*) Anopheles claviger and A. pictus, recognized by the yellow color of their bodies and spotted wings; and (*b*) Anopheles bifurcatus and A. nigripes, smaller, dark brown or brownish yellow insects without spots on the wings. The anopheles alight with the body, proboscis, thorax, and abdomen in a straight line and often perpendicular to the surface upon which they rest. It is only the female, especially the A. claviger, that transports the plasmodium;

PLATE VI.

STAINED INTRACORPUSCULAR AND CRESCENTIC FORMS OF THE ESTIVO-AUTUMNAL PARASITES.

Note the polar staining of the crescents, the irregular staining of the protoplasm, and the eosin-stained border. Some of the crescents have lost the eosin-stained rim and stain almost uniformly throughout.



Stained forms of the Estivoautumnal Parasites.—*Craig.*

the male is vegetarian in his diet. After the female has filled herself with blood, she flies to a dark, sheltered place, near stagnant water. After about six days she alights upon the water and deposits her eggs. She commonly dies in the water beside the eggs. These are oval and float with their ends in contact. The young larvæ often devour the body of their dead parent in their search for nutriment. From the larval stage they pass into the nymphal, during which they float upon the water. It is distinctive of the class that they do not sink below the surface (Fig. 17). Finally the shell cracks and the young mosquito is liberated. Manson believes that the larva is infected by eating the body of the mother. The cycle of the plasmodium's development within the mosquito is well illustrated in Plate VII.

It is only since these facts have been discovered that a proper estimate can be placed upon the various theories that have been held heretofore in regard to the causes of malaria. All the geographic, seasonal, and telluric influences which have been regarded as especially favorable to the production of the disease may now be summed up in the single statement that all conditions which favor the propagation and activity of the anopheles are favorable to malaria. Where mosquitoes do not exist, there is no malaria. The presence of the anopheles does not invariably signify a danger of malarial infection, however, for Nuttall and others have shown that the disease has disappeared from districts in England formerly malarious, although the anopheles and swamps remain. Occasional cases are attributed to the transfer of infection by the mosquito to healthy persons, from others who come to the region while infected.

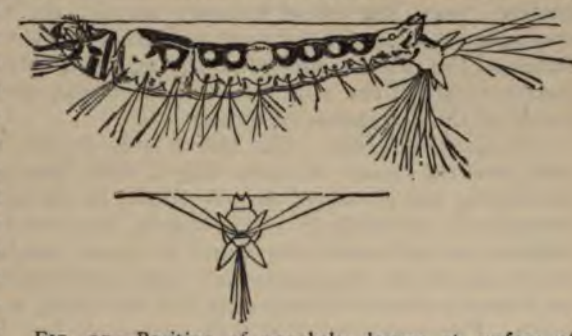


FIG. 17.—Position of anopheles larvae at surface of water. (After Howard, Bulletin U. S. Dept. of Agriculture.)

Marshes in which there is an abundance of decaying vegetable matter are especially favorable to the production of malaria. The turning up of soil by plowing has frequently been followed by the appearance of the disease in the vicinity, possibly, as has been suggested, on account of permitting the formation of small pools to serve as the breeding-places of mosquitoes. The fact that persons living in the lower stories of dwellings are more frequently attacked than those on the upper floors is explained by the low flight of the insect. The same fact accounts for the absence of the disease at high altitudes and on mountain-tops. The influence of season, of winds, forest trees, and the danger of night air are all apparent. High temperature favors the production of the disease, but the drying of swamps checks it for a time. A. F. A. King has recently advanced the theory that bright sunlight is of greater importance than heat, especially in the life cycle of the plasmodium after it has entered the blood.

In the United States, the malarial districts are found especially along the southern Atlantic and Gulf coasts and up the Mississippi and its larger tributaries. But the disease is much less prevalent than it was a half-century ago, chiefly, perhaps, on account of the redemption of swamp lands for the purposes of agriculture. It is more common in the country than in cities. Its virulence is much greater in the tropics and in subtropical climates than further north.

Age, sex, and other individual peculiarities are unimportant, since exposure is the principal feature, and very few persons are immune to either the mosquito or the plasmodium. The negro is not so susceptible as the Caucasian. King explains this on the supposition that the negro's dark skin prevents the penetration of light to the interior of the body and thus inhibits the sporulation of the plasmodium. Men are five or six times more frequently affected than women, on account of greater exposure. It has been asserted that the anopheles rarely enter dwelling-houses.

Morbid Anatomy.—We know little of the lesions produced by the milder forms of malaria, for they are seldom fatal. In severe or protracted cases, the blood becomes anemic, the spleen is enlarged, and spontaneous or traumatic rupture has been observed. In fatal pernicious forms of the disease, and after the malarial cachexia, the changes are in part a result of the alterations of the blood, and in part, doubtless, a result of the formation of toxins.

Pernicious Malaria.—The blood is hydremic, the red corpuscles are pale, and the serum is often tinged with hemoglobin. Red corpuscles containing the parasites may be found in the blood-vessels of all parts of the body, particularly in the spleen, bone marrow, and brain. Extracellular parasites are also found in great numbers. Along with these forms, numerous phagocytes are seen everywhere; they sometimes form an almost complete occlusion of the arterioles, especially in the liver and brain. Pigmentation is almost universal, but becomes extreme in the spleen and brain, affecting moderately also the kidneys. Areas of necrosis may be seen in the liver and elsewhere, especially in the estivo-autumnal form. Small punctate hemorrhages may be found in the same regions. The spleen is enlarged to a variable degree; in recent cases it is soft; after repeated attacks it becomes firm (ague cake). The liver is moderately enlarged, and capillary thromboses have been found in it. The kidneys show cloudy swelling or fatty degeneration; and after severe hematuria, there is often hemorrhage into the glomeruli and necrosis of the tubular epithelium.

Malarial Cachexia.—Death is usually a result of anemia or hemorrhage. All the organs are found anemic. The pigmentation is general, but the coloring matter is deposited around the blood-vessels and in the spleen along the trabeculæ. It is found also in the peritoneum, around the blood-vessels of the mucous membranes of the stomach and intestines, and between the gland-cells of Peyer's patches. The spleen may weigh 8 or 10 pounds. It is dense and the cut surface has a uniform or mottled brown color. Enlargement and pigmentation of the liver and kidneys are also common. The liver may become sclerotic.

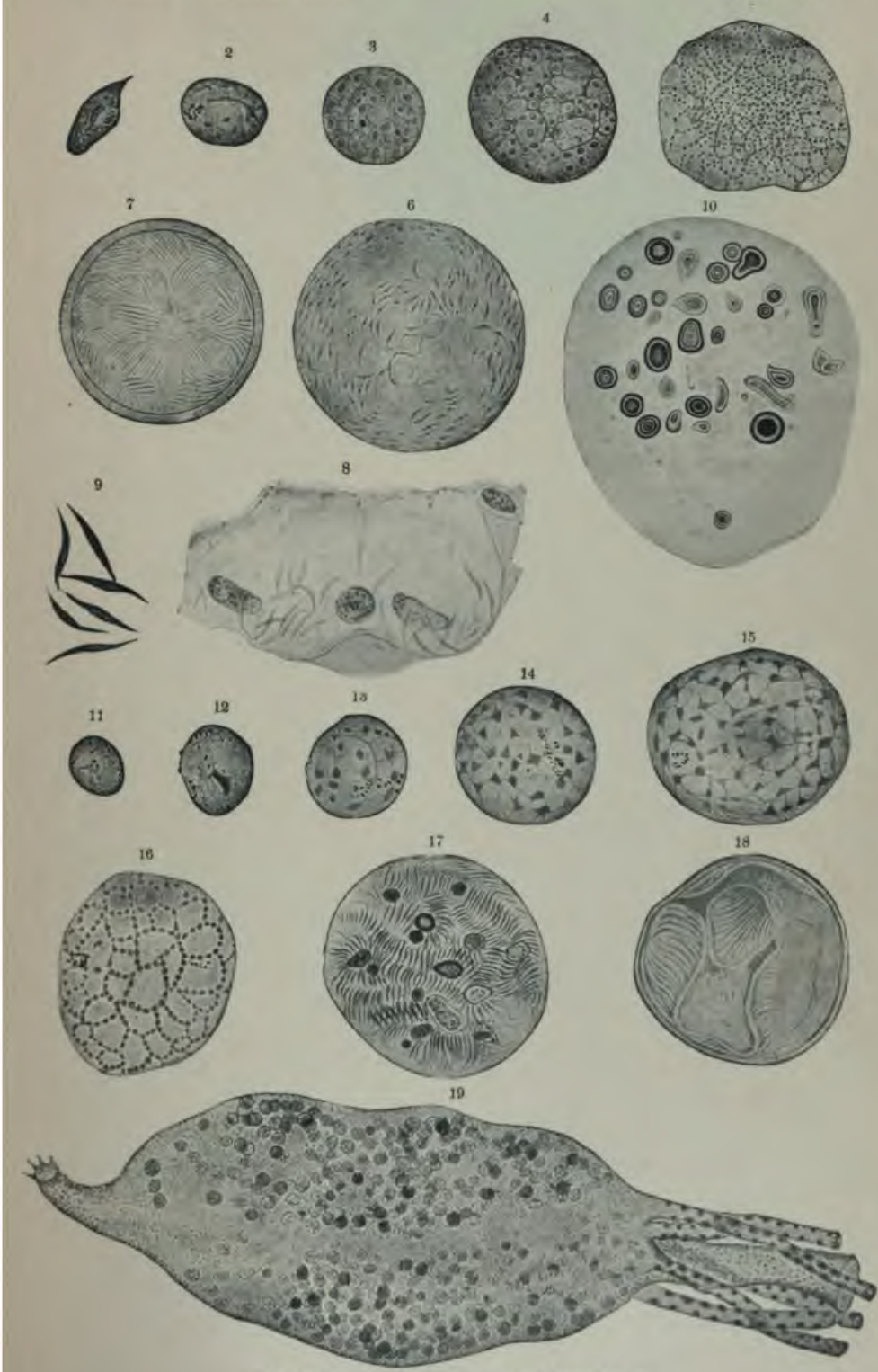
Clinical Forms of Malaria.—1. **Tertian intermittent fever** is due to a parasite which matures and gives rise to a paroxysm every 48 hours.

EXPLANATION OF PLATE VII.

FIGS. 1–10.—Development of crescents in the Middle Intestine of the Anopheles claviger; Fig. 1, Crescent in the wall of the middle intestine a little less than two days after the Anopheles has sucked the blood of a sufferer from malaria. The parasite preserves its spindle shape, resembling perfectly the form which it may assume in the blood of man. Figs. 2–5, Forms of progressive development, surrounded by a very thin hyalin capsule, showing phases of successive division of the nucleus. The nuclei of the parasitic body in Fig. 5 are small and very numerous; Fig. 6, Forms of complete development of the crescenting sporozoön. Within the capsule are seen numerous sporozoites. Fig 6 is from the infected intestine of the Anopheles cut *in toto*, after being embedded in parafin; Fig. 7, A mature sporozoön containing a large number of sporozoites, seen in an unstained fresh specimen; Fig. 8, Salivary gland of Anopheles, the cells of which contain numerous sporozoites; Fig. 9, Mature sporozoites; Fig. 10, A large capsule containing many brown bodies of varying form and structure.

FIGS. 11–18.—Developmental forms of the parasites of ordinary tertian in the middle intestine of the Anopheles claviger; Fig. 11, Tertian bodies in the substance of the middle intestine less than two days after the insect had sucked the blood of a patient with tertian fever; Figs. 12–16, Later developmental forms of the tertian sporozoön, showing successive division of the nucleus of the parasite; Fig. 17, Mature tertian sporozoön containing numerous sporozoites and the residua of segmentation (semi-schematic); Fig. 18, Mature tertian sporozoön containing many sporozoites and residua of segmentation, seen in an unstained fresh preparation.

FIG. 19.—The middle intestine of a specimen of Anopheles claviger captured in a cabin in Ostia (a region where grave malaria prevails), occupied by several peasants suffering from malaria. It contains an enormous quantity of cystic bodies, the greater number of them mature and enclosing sporozoites. They are scattered throughont the entire length of the middle intestine, but are more numerous in the middle third. The figure is semi-schematic.



MALARIA PARASITE IN THE MOSQUITO.

(*Marchiafava and Bignami, in "Twentieth Century Practice."*)

When two groups of these parasites are present, one group matures every day, causing a paroxysm every 24 hours. The disease is then known as double tertian or quotidian (daily) intermittent fever. This is the most prevalent type of intermittent fever in many regions. Other forms are rarely seen in the Philippine Islands.

2. **Quartan Intermittent Fever.**—The parasite causing this type of the disease matures in 72 hours. Three kinds of infection are recognized—single, double, and triple. In the single form there is a paroxysm on the first and one on the fourth day. In the double form there is a paroxysm on the first day, none on the second, one on the third, and one on the fourth. In the triple quartan three groups of the parasites mature on different days, producing a paroxysm every day, or a quotidian quartan intermittent fever. This form is usually recognizable by the very unequal intensity of the paroxysms.

3. **Estivo-Autumnal Fever.**—This name is sufficiently appropriate in our country, where the disease prevails almost exclusively during the summer and autumn; but in many other regions, especially in tropical and subtropical climates, it is a perennial disease. Two forms are recognized, a quotidian and a tertian. Some writers have maintained that the former is a double tertian, but the more prevalent view refers them to different plasmodia, one maturing in 24 hours, the other in 48. This type of the disease differs from the intermittent chiefly in the greater length of the paroxysm and relative brevity of the interval. It also shows a tendency to become remittent, the intermissions often becoming incomplete. As a rule, it is of much greater severity. It is only in this type of malaria that crescents are found in the blood. The term pernicious remittent fever is applied to the disease when such grave symptoms as hematuria, coma, severe gastrointestinal disturbances, and jaundice appear.

4. **Remittent Fever.**—This form is attributed to the sporulation of more than one group of estivo-autumnal parasites at irregular intervals or to the presence of two or more different species. It may begin with an intermittent type of fever, but the intermissions soon disappear, and the pyrexia becomes continuous, with more or less marked remissions. Hyperpyrexia is not unusual.

5. **Malarial Cachexia, or Chronic Malaria.**—This may result from the frequent repetition or long duration of any of the other forms of malarial infection. There may be no parasites in the blood. The fever is very irregular or it may be absent. The spleen becomes much enlarged. The blood is extremely anemic and contains much pigment.

Symptoms.—The period of incubation is generally stated as from a few hours to several months. In the few instances in which the inoculation by the mosquito has been watched, the period varied from 16 to 19 days in the intermittent form and from 9 to 12 days in the estivo-autumnal. It is believed to be longest in the quartan and shortest in the remittent. A gradually increasing interval often occurs between attacks of the disease, which is attributed to the development of a systemic or therapeutic immunity.

Of the different forms as they are observed in America, the tertian is the most frequent; the double tertian is probably more frequent than the single. The estivo-autumnal is often seen, but the quartan is rare. The estivo-autumnal is the most virulent, particularly in the tropics.

Prodromal symptoms are usually felt. The individual who has previously suffered from the disease can generally predict the approach of a paroxysm, from a feeling of lassitude, headache, pain in the extremities, often accompanied by yawning and stretching. Nausea and vomiting may occur; bronchitis is often present. In fully two-thirds of the cases the paroxysms occur before noon; they are almost never seen at night.

I. *Symptoms of Intermittent Fever.*—The paroxysms, whether tertian, quartan, or quotidian with reference to periodicity, are all of the same character. The typical paroxysm lasts from 8 to 12 hours and consists of a chill, fever, and sweating. It is thus divided into three periods, known as the cold, the hot, and the sweating stages.

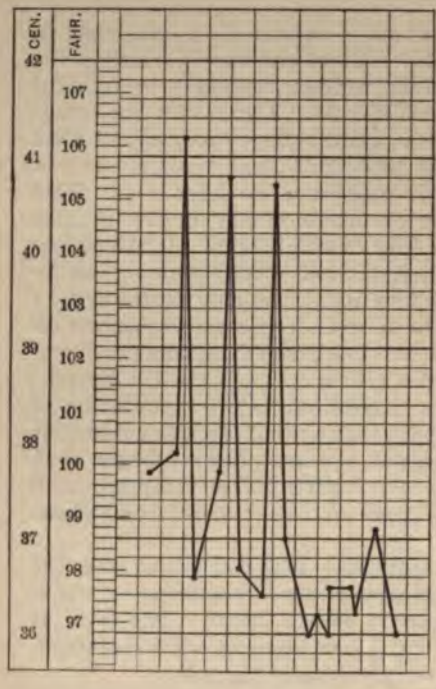


FIG. 18.—Quotidian fever. (Seguin.)

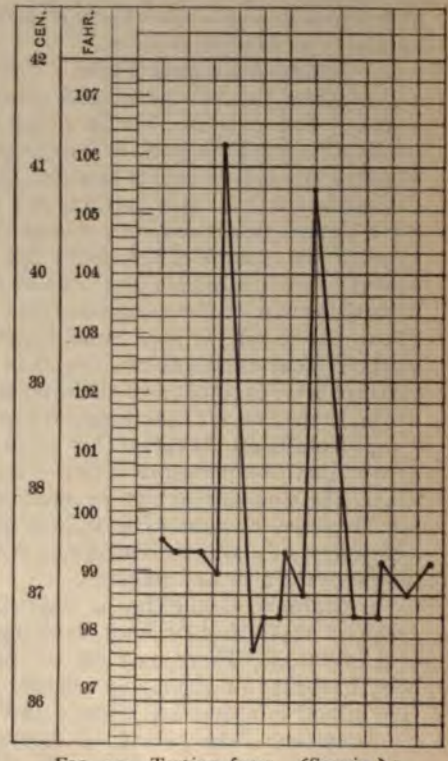


FIG. 19.—Tertian fever. (Seguin.)

1. *The Cold Stage.*—The chill is usually severe and lasts from 15 to 45 minutes. The shivering involves the entire body, and the teeth chatter. The temperature of the skin is reduced. The face and hands, often the entire body, become cyanotic, and the patient complains of intense cold, notwithstanding the warmest coverings. The general temperature is, however, increased during the chill, often reaching 104° to 106° F. (40.0°—41.0° C.). The pulse is rapid and of high tension. The feeling of coldness may continue for a time after the rigor has ceased. Rarely, the chill is omitted—"dumb ague." In children a convulsion may take its place.

2. *The Hot Stage.*—This gradually develops after the chill. As the skin becomes warm, the sensation of cold gives place to that of intense heat. The face and hands become flushed and the entire body becomes burning hot. The heart's action is often violent, the pulse full and bounding. Severe headache, great thirst, and restlessness are not unusual; there may be delirium of short duration. The course of the fever is characteristic. After attaining its maximum within one or two hours, it begins to decline within from a half-hour to three hours, and reaches the normal, or a point 1° to 2° F. (0.5°—1.0° C.) below normal, within 8 to 12 hours from the beginning of the attack.

3. *The Sweating Stage.*—Sweating begins with the decline of the temperature. As a rule, it is profuse. With it the headache and feeling of discomfort subside and the patient often falls into a refreshing sleep.

The paroxysms usually occur at the same hour each day. When, however, they are increasing in severity they frequently come on from a half-hour to an hour earlier, and when they are decreasing in severity they may be delayed from one to several hours.

Other symptoms frequently appear, especially after the occurrence of several paroxysms. There is a recognizable enlargement of the spleen after almost every paroxysm. This at first subsides in the interval, but it soon becomes constant and progressive, and does not subside for several weeks after recovery. The organ may be sensitive to pressure. The enlargement of the liver is seldom recognizable in this type of the disease. The urine shows the febrile changes, concentration with increase of color and solids, rarely albumin. A marked diminution of the quantity of urea has been noted five or six hours before the chill. Herpes appears at the angles of the mouth or on the alæ of the nose in about one-fourth of the cases. "Masked malarial fever" is sometimes encountered, in which the paroxysms are replaced by violent attacks of neuralgia, affecting the supra- or infraorbital, intercostal, sciatic, or other nerves, lasting 8 to 12 hours and recurring at regular intervals.

II. *Symptoms of the Estivo-Autumnal (Remittent) Fever.*—This form may be preceded by symptoms of much the same character as those of the intermittent type. A few tertian paroxysms may occur. In many cases, however, the invasion is more insidious, the chill is mild or it may be absent. The temperature rises more gradually, and during the first few days it may not attain a great height. One paroxysm is said to "anticipate" another; that is, a second paroxysm comes on before the temperature of the first has fully declined. There is, therefore, no complete intermission. After a few days the temperature becomes continuous in character and the remissions may become comparatively slight. Nausea and vomiting are often prominent symptoms, and bronchitis is usually present. There is frequently also diarrhea with adbominal tenderness and discomfort. The pulse becomes rapid, often 120 to 130, small and feeble. Headache, restlessness, and insomnia are common, and there may be slight delirium, especially at night; stupor and coma may follow. Profuse sweating occurs, usually at night, and the temperature declines to a variable degree; but it rises again in the morning, without chill or other evidence of a renewal of the paroxysm. The temperature often reaches 105° or 106° F. (40.5°—41.0° C.). The duration of the disease is indefinite. Some cases yield readily to treatment, while others are ex-

ceedingly refractory, lasting from two to four weeks or longer, and relapses are common.

A grave, pernicious type of the fever may develop, and the general appearance, particularly of the face and tongue, and the temperature curve, may all conform to the features of typhoid fever. The name typhomalarial fever was formerly applied to these cases by many clinicians. The more prolonged cases are frequently accompanied with jaundice, which varies from a slight tingeing of the skin, with light yellow conjunctivæ, to the deeper saffron shades (bilious remittent fever). A catarrhal duodenitis, with the usual symptoms, sometimes develops during the course of the fever or during convalescence.

III. *Symptoms of the Pernicious Form.*—The course of this form differs from that of the intermittent or remittent chiefly in its severity, and more particularly in the occurrence of severe gastrointestinal, hemorrhagic, or cerebral manifestations. In periodicity it may conform to either type, but this feature is often lost. It is not a common form of the disease in America, being encountered for the most part in the warmer climates, notably in the West Indies and the tropics. Many cases have been observed among the soldiers returning from the Philippines. Three principal types are recognized:

1. *The Algid Type (Congestive Chills).*—This form is characterized by profound disturbances on the part of the gastrointestinal tract, often with thrombosis of the smaller vessels of the mucosa, with necrosis and ulceration. The disease may begin with a chill or chilly sensations, usually accompanied with nausea, vomiting, and great prostration. This is soon followed, in most cases, by a profuse diarrhea. The discharges are copious and watery, like those of cholera; or they may be dysenteric, containing blood and mucus. The temperature is usually normal or subnormal; fever is unusual. The pulse is accelerated and feeble, the breathing rapid and labored. The body is frequently bathed in a cold, clammy perspiration. A fatal collapse may supervene within a few days, or jaundice may develop and the case may thus resemble one of yellow fever.

2. *Comatose Type.*—The attack may be ushered in with a chill, followed for a few days by fever, but ordinarily the chill is absent and the patient passes directly into a state of stupor or delirium that merges into a profound coma. Hyperpyrexia is the rule, the temperature reaching 106° or 107° F. (41.0°—41.5° C.). The respiration is rapid, feeble, and superficial; it may assume a Cheyne-Stokes character. The condition may prove fatal within the first 48 hours, or a partial recovery may be followed by a fatal relapse. It is in this form of the disease that the cerebral vessels become obstructed by the parasites.

3. *Hemorrhagic Type.*—The malarial infection is manifested by a tendency to hemorrhages, particularly from the kidneys and the mucous membranes. There may be an initial chill and elevation of the temperature, but these are often absent. In one class of cases hemoglobin alone appears in the urine (hemoglobinuria), while in another class blood-cells are also found (hematuria). Casts are also discovered in some cases. The gums are usually swollen and bleed spontaneously or upon slight irritation. There may be also bleeding from the nose, lungs, stomach, and intestines, and ecchymoses appear in the skin. Jaun-

dice is a common accompaniment. In the United States this form of the disease is confined chiefly to the Gulf Coast. It is more common in the West Indies and in tropical countries. It is sometimes seen in individuals who have suffered from severe and prolonged attacks of malaria. In such persons there are usually periodic attacks in which the urine becomes scant and discolored by hemoglobin. The fatal "black-water" fever of the Gold Coast, in Africa, belongs to this type. The predominance of special symptoms has led to the adoption by different writers of such names as cardiac, choleraic, diaphoretic, gangrenous, gastric, pleuritic, pneumonic and syncopal, pernicious malaria.

IV. *Malarial Cachexia (Chronic Malaria).*—This condition develops after frequently repeated or very prolonged attacks of intermittent or remittent fever. It sometimes appears in the residents of malarious districts who have not suffered from distinct paroxysms (latent malaria). The condition is seldom seen in the Northern States, but it is not uncommon in Cuba, and it is exceedingly prevalent in Hawaii, where it often adds an element of much gravity to other diseases. It may assume the form of recurrent paroxysms separated by an interval of two, three, or four weeks, and lasting for months; or there may be ill-defined manifestations of various kinds. The most characteristic symptoms are profound anemia and enlargement and firmness of the spleen, often almost equal to that of leukemia. Fever is not always a feature of the condition, but it is more usual than chills. The blood-count shows an extreme anemia. There may be only 1,000,000 corpuscles to the cubic millimeter. Free pigment granules, crescents, and flagella are often found. The skin is usually of a dull, dusky yellow or "muddy" color, or the jaundice may be as deep as saffron. The urine frequently shows the presence of bile pigments. The tongue is pale, broad, flat, and flabby, showing the indentations of the teeth and coated with a white fur. The breath is foul, and the digestion is weak. Constipation, sometimes alternating with diarrhea, is the rule. The stools are light in color, sometimes chalky. Lassitude, mental depression, and muscular weakness, sometimes accompanied by aching pains, are common symptoms. The circulation is weak, and edema of the ankles often appears in the evenings, especially after long standing. Periods of fever occur, but in some cases the temperature is for the most part slightly below normal. Hemorrhages from the mucous membranes or into the skin, retina, and other tissues are occasionally observed, and a distinctly scorbutic condition may develop.

The malarial cachexia is one of the most persistent forms of malarial infection. Change of climate may give temporary relief, but a return to a malarious district is promptly followed by a relapse. This fact has been well exemplified in the cases of soldiers who contracted the disease in Cuba, recovered at home, and relapsed upon reaching the Philippines.

Complications.—Complications arise in about 10 per cent of all cases. They are, in the order of frequency: enteritis, nephritis, rheumatism, typhoid fever, lobar pneumonia, jaundice, and dysentery (Anders). A number of other conditions may result more or less directly from the infection, among them such paralyses as hemiplegia and aphasia; spinal irritation; optic neuritis and retinal hemorrhages, amblyopia and other disturbances of vision; pulmonary congestion, and asthma.

Diagnosis.—It is only in the estivo-autumnal type of malaria, as a rule,

that the diagnosis is difficult, even without examination of the blood. This form so frequently corresponds in its symptomatology to typhoid fever, pyemia, or pneumonia that its recognition may be difficult until the plasmodium, crescents, flagella, or pigment granules have been discovered in the blood.

Typhoid fever does not usually begin with a severe chill, the rise of temperature is gradual, the plasmodium is absent, and the Widal test is usually positive.

Pyemia is characterized by chills, fever, and sweating at less regular intervals; a focus of suppuration can generally be found; there is leucocytosis, and the plasmodium is absent. The spleen is not so greatly enlarged. *Septicemia*, if chills be present, conforms in character to pyemia.

Acute tuberculosis presents symptoms resembling pyemia rather than malaria. Physical examination usually reveals a pulmonary lesion, and the bacillus tuberculosis may be found in the sputum.

Prognosis.—Malaria is attended with a high mortality only in the tropical countries, and this pertains especially to the pernicious estivo-autumnal type. In any case the prognosis depends chiefly upon the number of previous attacks, the severity of the type, and the promptness of treatment. Repeated attacks very often leave permanent enlargement of the spleen, persistent anemia, disordered digestion, and impairment of one or more functions, especially of the nervous system. Complete recovery seldom occurs in the tropics.

Prophylaxis.—This consists in: (1) The destruction of the parasite, or of its intermediate host, the mosquito; (2) the prevention of inoculation; and (3) fortification of the system against the development of the parasites after they have gained entrance. Theoretically the plasmodium may be destroyed by the drainage of swamps and stagnant pools. The extermination of the mosquito is a more positive means of preventing the disease. This is to some extent accomplished by drainage. Fish destroy the ova and larvæ, but, unfortunately, they can rarely live in the marshes. Petroleum has been found to be the best agent for destroying the mosquito. A mere film of it upon the surface of the water quickly kills the larvæ and nymphæ. One barrelful is sufficient to cover a surface of 96,000 square feet. It should be used early in the spring. Mosquitoes should be excluded from dwellings and tents by the use of bars and screens. They may be to some extent driven away by the diffusion of such odors as pennyroyal, nutmeg, or camphor, or by the application of volatile oils to the skin. The system is best fortified against the parasite by the administration of quinin, gr. iij or iv (0.20—0.25), morning and evening, or three-drop doses of Fowler's solution of arsenic after each meal, during the season of greatest exposure.

Treatment.—The patient should go to bed as soon as a chill commences. There is no means of arresting it, but the suffering may be moderated by hot drinks and the application of dry heat to the body. The administration of aromatic spirit of ammonia, ʒ ss (1.85) or chloroform, ♏xv (1.0), in hot coffee or lemonade affords some relief. The body should be sponged with cool water during the hot stage, and dried with a warm towel during the sweating.

Quinin is a specific for the disease. It must be given in doses and at intervals appropriate to the form of the disease to be treated. In

the intermittent type it is not necessary to give the remedy at regular intervals between the paroxysms. One dose of from 10 to 20 grains (0.65—1.30) five hours before the anticipated chill is of more advantage than several times as much given at another time. This method is largely used in hospitals, and it is seldom that a second paroxysm is seen. Some physicians prefer to give a similar dose immediately after the paroxysm, others give it in doses of 5 grains (0.32) every three hours. It should be given in capsules or wafers or in solution. In children and sometimes on account of irritability of the stomach, it must be given by the rectum. The old practice of beginning the treatment with a calomel purge is often beneficial, as it frequently removes a condition which interferes with the absorption of the quinin.

The treatment should be continued until the spleen has returned to its normal size. Arsenic may be administered (3 drops of Fowler's solution t. i. d.) for several weeks after discontinuance of the quinin.

In the remittent estivo-autumnal type of the disease it is often impossible to anticipate the paroxysms. The quinin must then be given at regular intervals and in sufficiently large doses to produce physiological effects in the shortest time possible. It often happens, particularly in the pernicious form, that the remedy is not retained or, if retained, it fails to produce the desired effect. It is then better to administer it hypodermically in the form of the hydrobromate, hydrochlorid, or bisulphate, in doses of 15 to 30 grains (1.0—2.0) once or twice a day. The administration of a calomel purge at the beginning is advisable also in this form. There has been much discussion in regard to the action of quinin in hematuria. Some writers, especially in the North, claim benefit from it, while others, in warm climates, assert that it is generally harmful.

The unpleasant symptoms of cinchonism may be lessened by the administration of 20 to 30 grain (1.5—2.0) doses of potassium bromid. It is often necessary to give opium in the form of Dover's powder, codein, or morphin hypodermically, for the restlessness. Other remedies are required in some cases: Stimulants for prostration; iron and arsenic, codliver oil and malt, for the anemia; opium and bismuth for the diarrhea. Other remedies have also been employed to combat the disease, notably iodin, ammonium chlorid, salicin, and eucalyptol. Warburg's tincture is also highly esteemed by some writers, and often produces effects not easily accounted for. Most of these remedies are applicable, however, only to the interval or to the chronic and cachectic conditions.

Quite recently Gautier has found a powerful specific for the disease in disodic methylarsenate. It may be administered by the mouth, but is more active when given hypodermically in the dose of gr. ¼ to ½ (0.05—0.10). Recovery has been observed to follow one or two injections after quinin had failed to arrest the disease.

DYSENTERY.

THE BLOODY FLUX.

Dysentery is one of the oldest of known diseases. Although it is, in the strict sense of the term, ubiquitous, occurring everywhere, it is most common and most virulent in the tropics, where it frequently becomes epidemic and more fatal than the cholera. In army life it is most dreadful. It is encountered in all parts of the United States, but it

has been most fatal in New England. Epidemics are not frequent in this country. The disease becomes less prevalent with the progress of sanitary improvements. In the West Indies, and more particularly in the Philippine Islands, it constitutes a large per centage of the total illness.

Definition.—An infectious disease, endemic in the tropical zone, sporadic or epidemic in the temperate; characterized pathologically by catarrhal, croupous, or ulcerative inflammation of the large bowel, and clinically by frequent mucous, serous, or bloody dejections, accompanied with tormina and tenesmus and more or less severe general symptoms.

Etiology.—The direct cause of the disease is probably a micro-organism in all cases. The ameba coli and the bacillus dysenteriæ are recognized causes. Their relation to forms of the disease will be considered under separate heads. The predisposing causes may be considered together. One of the most important of these is the contamination of drinking-water with the dejecta of dysenteric patients, through defective drainage or otherwise. That there are other modes of conveying the infection is highly probable. That the disease is contagious is possible, though not probable. Disturbances of digestion, particularly as a result of eating improper food, unripe fruit, imperfectly cooked vegetables; constipation, and starvation are important factors. Crowding and imperfect ventilation, fatigue, loss of sleep, anxiety and chronic alcoholism, favor infection.

Age and Sex.—No age is exempt, but males are somewhat more frequently attacked than females, probably on account of greater exposure. There is no racial immunity.

Climate and Season.—The disease is more frequent in hot seasons in all climates. It has prevailed most extensively in the United States during August and the succeeding fall months. Atmospheric moisture appears to favor its spread when associated with either extreme heat or cold. Sudden changes of temperature, as when cool nights succeed to hot days, also favor it.

Forms of Dysentery.—The marked difference in the severity of different cases, and, perhaps, more particularly the discovery of two very different micro-organisms in relation to the disease, have led to a separation of the cases into different groups. It is probable, however, that more accurate study will result either in establishing the identity of the different forms or in giving us a better classification. Four varities of dysentery are now recognized: (1) Acute catarrhal, or sporadic; (2) amebic; (3) diphtheritic, or that due to the bacillus dysenteriæ; and (4) chronic dysentery, which may follow either of the preceding forms.

1. Acute Catarrhal Dysentery.

Etiology.—This form is probably due to a specific micro-organism. It not infrequently occurs as a secondary affection in the course of such diseases as tuberculosis, syphilis, chronic nephritis; or follows scurvy, cholera, malaria, and other affections. The investigations of Flexner and Strong indicate that all these cases are due to the bacillus dysenteriæ described by Shiga. Park and Carey believe, however, that a group of bacilli, rather than an individual organism, is embraced under this term.

Morbid Anatomy.—The lesions are most frequently found in the rectum and flexures of the colon. They are sometimes limited to the rectum (proctitis). The affected mucous membrane is hyperemic and swollen and is usually covered, in areas of variable extent, with mucus, pus, and blood. The epithelial layer is absent, and the exfoliation may have extended to the glands. Superficial ulcers are found, and occasionally a few which have extended through the submucosa. Punctiform hemorrhages may be discovered in the mucous membrane and submucosa. The solitary follicles are swollen and sometimes distended with pus, or they may have burst and given place to ulceration.

Symptoms.—The disease may be mild or severe. The milder cases often begin with the symptoms of an ordinary diarrhea, free watery passages with scybala. Tympanites and abdominal pains develop; the passages become small and painful, and they are composed chiefly or entirely of blood-stained mucus. The microscope reveals a granular débris, red and white blood-cells, and degenerated epithelial cells which are sometimes mistaken for amebæ. A large number of bacteria are usually found, and the cercomonas intestinalis is sometimes seen. Griping pains (tormina) are an almost constant feature. They begin usually at the umbilicus and extend to the region of the ascending or descending colon, causing urgent desire for evacuation. This is attended with intense involuntary straining (tenesmus), and followed by burning pain in the anus. Vesical tenesmus is often added to the patient's suffering. Prolapse of the rectum often occurs, especially in children. The abdomen becomes flat and highly sensitive to pressure, particularly over the colon. There is little constitutional reaction and little or no fever, as a rule. Improvement begins in from three to five days, and recovery may be complete in a week or 10 days.

Severe cases usually last from two to three weeks. The symptoms are those of the milder form, but more severe, particularly at night. The discharges are usually from 20 to 40 in 24 hours; they sometimes reach 150 to 200 within that time. The dejections consist of a serous fluid containing flakes or masses of mucus and blood. After ulceration has begun, usually about the fifth or sixth day, pus appears in them. The temperature reaches 101° to 103° F. (38.5°—39.5° C.) or higher. The pulse is accelerated and often becomes feeble. The prostration may be extreme. The tongue becomes dry and the thirst imperative. The patient suffers intensely from loss of sleep, not due to ordinary insomnia, but to the intense suffering. When this is not relieved, delirium sometimes develops. When recovery occurs, the symptoms gradually subside, and fecal matter again appears in the dejections. Death may result from the physical and mental exhaustion. The disease sometimes passes into a chronic condition.

2. Acute Amebic Dysentery.

Etiology.—This form of the disease is endemic in India, Japan, the Philippine Islands, and other tropical regions; hence it is often referred to as "tropical dysentery." But it is by no means peculiar to the tropics. The other forms are frequent there, and sporadic amebic dysentery is encountered in different parts of the United States. It is due to the

ameba coli, a peculiar, irregularly shaped body from one and a half to four times as large as a red blood-corpuscle, having a pale nucleus, vacuoles, and active pseudopodia (Fig. 20). Other micro-organisms, especially the pus-formers, are often present in the lesions. The etiological relation of the ameba to the disease was disputed by some writers before the discovery that there are at least two other forms of ameba.

Morbid Anatomy.—The lesions are commonly located in the sigmoid flexure, but they are not infrequently found also in the rectum and flexures of the colon. The intestinal wall is thickened and hyperemic, particularly the mucous membrane, which is also covered with bloody mucus. Nodular prominences appear on the mucosa, which are due to edema and cellular infiltrations of the areas in the submucosa. Necrosis follows, with the production of cavities, which often communicate with each other and soon reach the surface, forming large ulcers. Amebas and other organisms are found clinging to the surface of the ulcer and often in the intertubular spaces. Follicular ulcers are also present, but they are often concealed by necrotic (gangrenous) masses. In some cases abscesses are found in the liver, sometimes also in the lung, usually in the lower lobe of the right, and these may be found to communicate through the diaphragm with an abscess in the liver.

FIG. 20.—Amebae dysenteriae from an abscess of the liver.

Symptoms.—The course of the disease is divided into two stages, the catarrhal and the ulcerative.

Catarrhal Stage.—This often begins with a slight chill and moderate fever, but the temperature declines in a few days unless serious intestinal, hepatic, or pulmonary lesions develop. There may be at first a simple diarrhea, but the dejections soon become dysenteric. In some cases they are of this character from the beginning, with little or no odor and an alkaline reaction. The amebas are found in the bloody mucus. The pain, tenesmus, and other symptoms are similar to those of the catarrhal dysentery, but the tenesmus is not usually so severe. The diarrheal stage may be absent, or may be so mild as to be unheeded, and the ulcerative stage may begin abruptly.

Ulcerative Stage.—The symptoms are profound, often resembling cholera in severity. The evacuations become more and more numerous; they contain much mucus and blood, and soon become fetid. When gangrenous destruction of the mucous membrane occurs, the temperature often declines to normal or becomes subnormal. The extremities then become cold. Hemorrhages and perforations of the bowel occasionally occur. The patient may die in collapse due to the intensity of the disease; from exhaustion, or from the formation of abscess in the liver or lung. But the course of the disease is very indefinite. Remissions and

exacerbations are common and the condition often becomes chronic and lasts for several months. The convalescence is always slow.

3. Diphtheritic Dysentery.

Etiology.—This form of the disease is endemic in the Philippines and other tropical regions; it occasionally becomes epidemic there and in other parts of the world, and sporadic cases are occasionally observed in this country. A very large part of the dysentery in our civil war was of this character. It has been observed also in a secondary relation to various acute and chronic diseases, notably pneumonia, chronic endocarditis and chronic nephritis. It is caused by the bacillus dysenteriæ, a slender rod, which is usually found in the intestinal contents and in the mesenteric glands. Flexner in his studies of Philippine dysentery, found two bacilli, one present in all cases, the other only in the most acute. Characteristic lesions and symptoms are produced in animals inoculated with the bacillus and by the injection of a toxin separated from the cultures. One human experiment is recorded. A healthy Indian criminal condemned to death voluntarily ingested a bouillon culture in warm milk and developed the disease with typical symptoms. The bacilli, but no amebas, were found in the stools. Recovery occurred and the lesions were not seen.

Morbid Anatomy.—The lesions may involve the entire colon and the rectum, or they may be more limited. In about a third of the cases they extend from 10 to 15 cm. into the ileum, a condition not observed in amebic dysentery. Peyer's patches and the solitary follicles may be slightly enlarged. The wall of the colon is thick and edematous to an extent corresponding to the severity and duration of the attack. The surface of the mucous membrane is red or of a reddish brown color and covered with a more or less tenacious pseudomembrane composed of fibrin or mucin filaments inclosing blood-corpuscles and epithelium. Nodular prominences are formed in the mucosa, especially in the lower part of the colon, as in other forms of the disease. One of the most striking features of the morbid anatomy is the enormous thickness of the colon walls. In many cases, no ulcers are found. There is only an irregular, superficial erosion of the epithelial surface, sometimes involving the entire thickness of the mucosa. Under the microscope the section often has the appearance of having been trimmed with a pair of scissors. The capillaries are enlarged and hemorrhagic areas are often found. The mesenteric vessels are often distended.

Symptoms.—In the primary form of the disease the incubation does not exceed 48 hours. The onset is sudden, often with chill and fever reaching 102° to 104° F. (39°—40° C.); rapid pulse, from 120 to 150, as the disease progresses. The stools, as in the other forms of the disease, are small and consist of mucus and blood, becoming more frequent, the pain and tenesmus also increasing. The tongue usually has a whitish fur. The thirst is excessive. The abdomen is not, as a rule, distended, but it is extremely sensitive to pressure, especially along the colon. The urine is gradually reduced in quantity and often becomes albuminous. The liver and spleen retain their normal size in most cases. In fatal cases the dejections often become less frequent and the patient

may sink into a state of profound collapse. But the course of the disease is exceedingly variable. Severe cases rarely recover; death usually occurs from the fourth to the tenth day. Convalescence is slow and relapses are common. They are often precipitated by errors in diet or exercise.

The secondary or terminal form of the disease may for a time be obscured by the manifestations of the affection which it complicates, or by the irregularity of its symptoms. Not infrequently there are several large watery dejections daily without mucus or blood, or the mucus and blood may be so small in quantity as to be overlooked. The bacillus has been discovered in cases occurring in the course of chronic nephritis, by Flexner and Strong.

Chronic Dysentery.

Either of the acute forms may pass into the chronic. The disease, especially the amebic form, frequently pursues a subacute course.

Morbid Anatomy.—Any or all of the lesions may be found as they have been described under the several acute forms. The ulcers are usually numerous, some of them in a state indicating reparative changes, others showing little vitality. As a result of the continued hemorrhages and the disintegration of blood, the ulcers are generally pigmented. The submucosa and muscular coats of the bowel are much thickened. Here and there among the ulcers small follicular cysts are often observed. In some cases an apparently excessive reparative process has led to the development of much new connective tissue, the contraction of which has given the mucous membrane an irregular, uneven surface, showing in some places deep depressions, in others polypoid prominences. The lumen of the bowel is often much narrowed; complete strictures are not common. In exceptional cases, no ulcers are found.

Symptoms.—The course of the disease is not at all uniform. Many of the symptoms of the acute forms of the disease are absent or they are present in a modified form. Pain and tenesmus are seldom prominent features. Many cases bear a close resemblance to the lienteric type of chronic diarrhea, the passage of undigested food being a prominent symptom. True dysenteric manifestations are seen for the most part only during exacerbations. At such times from three to twelve large, liquid dejections occur during the course of 24 hours. They are often frothy, especially if the patient's diet has been largely of a starchy nature. The character of the symptoms in any case depends largely upon the nature of the food. A mixed diet, as a rule, produces large watery stools. Mucus is usually present in variable quantity. In the severe cases, particularly those of the amebic form, pus and blood are often discharged. As a rule, however, neither blood nor shreds of necrotic tissue are found in the dejections. In another class of cases the stools are semifluid, pultaceous, and of a yellow or brownish color due to the presence of bile. Scybala are seldom seen, except in cases in which the dysentery alternates with constipation. These cases occur for the most part when the disease is confined to the lower part of the bowel. Flatulence is often an aggravating symptom. The tongue is usually red and glazed, seldom coated. In protracted cases it becomes dry and fissured. Slight tenderness may be elicited along the course of the ileum. Emaciation and anemia become extreme in the most chronic cases.

Complications and Sequelæ.—The complications and sequelæ of acute and chronic dysentery are probably more numerous than those of any other disease. The loss of blood and impairment of digestion rapidly lead to malnutrition and anemia. Other conditions result in part from these, in part from toxic or septic infection, possibly from the direct action of the bacteria. The following complications have been noted: Catarrh of the stomach and small intestine, perforation of the bowel and peritonitis, prolapse of the rectum, hemorrhoids, perineal abscess and fistula, acute bronchitis, pleurisy, pneumonia, abscess and gangrene of the lung, abscess of the spleen, endocarditis, pericarditis, pyelophlebitis, parotitis, convulsions, meningitis, cerebral embolism with hemiplegia and aphasia, thrombosis of the cerebral sinuses, monoplegias and paraplegia due to neuritis; albuminuria, chronic nephritis, anuria, edema, ascites, anasarca, erysipelas, and various forms of arthritis. The disease is sometimes associated with tuberculosis, scurvy typhoid fever, typhus, malaria, or other diseases.

Diagnosis.—When the dysentery begins with frequent small dejections consisting largely of mucus and blood, accompanied with tormina and tenesmus, as is usually the case in the acute catarrhal form, the diagnosis is not difficult. The diphtheritic form often resembles typhoid fever so closely as to render a distinction difficult. It is seldom, however, that the initial symptoms of the latter disease are so severe. Blood does not appear in the stools so early, if at all, and it is usually in the form of a profuse hemorrhage. The presence of the rose spots, and the serum test, complete the diagnosis. In the amebic form the discovery of the parasites in the stools establishes the character of the disease. They should be searched for in all cases of persistent atypical diarrhea. Leucocytosis is not usually present unless complications have developed.

Prognosis.—The disease is extremely fatal unless the treatment be prompt and thorough. Not a little depends upon the character of the disease in the given case, the constitution of the patient, and his hygienic surroundings. The mortality at different times and in different places has varied from less than 10 to more than 90 per cent. In the tropics this range is still observed in some instances. In Manila recently it was only 9.5 per cent.

Prophylaxis.—This is practically the same as that of typhoid fever. Thorough sanitation is the most efficient means of exterminating the disease. During epidemics raw food should not be eaten and the drinking-water should be thoroughly boiled. The disinfection of dejecta should be rigidly practiced.

Treatment.—Each form of the disease has its appropriate treatment. The acute catarrhal form is often promptly relieved by the administration of a purge. The effervescent magnesium citrate, Rochelle salts, a half-ounce in a glassful of water every hour, an ounce of castor oil to which may be added 15 drops of deodorized tincture of opium, to relieve the griping; either remedy may be employed. The purge need not be repeated unless the presence of scybala or continued abdominal distress and desire for evacuation indicate that the bowel has not been thoroughly freed of its irritating contents. After thorough evacuation, an opiate should be administered, as the deodorized or camphorated tincture of opium, Dover's powder, or the lead and opium pill. If tenesmus continues, laudanum, ʒ ss (1.85), in starch-water, or a suppository

containing cocain, gr. ¼ (0.016), or morphin, gr. ½ (0.032), may be inserted into the rectum. Morphin hypodermically is sometimes required.

Ipecacuanha is one of the oldest and often the most valuable remedy, particularly in the amebic form of the disease. Its action is almost specific when it is employed at the very beginning of the attack. The patient should abstain from all food for at least three hours before the remedy is administered, then take 20 drops (ɱlxij—0.75) of the deodorized tincture of opium. A half-hour later, when the effect of the opium is becoming apparent, gr. xx to xxx (1.30—2.0) of the powdered ipecacuanha are given, preferably in pills or capsules, with as little water as possible. Keratin capsules, which do not dissolve in the stomach, may be used. The patient must lie quietly on his back, not even speaking, in order to prevent vomiting. The saliva must not be swallowed, but should be removed on a cloth with the assistance of the nurse. If vomiting occur, the dose should be repeated in an hour or two, after nausea has ceased, otherwise not until the following day.

Bismuth in large doses, ʒ ss to ʒ j (2.0—4.0) every two or three hours, has been found beneficial, especially in chronic cases. Astringents are usually harmful.

Irrigation Treatment.—This has proved one of the most effective methods of treatment. The chief difficulty in its way is the great irritability of the bowel, especially in acute cases, which prevents the retention of the injected fluid. This may be in a measure overcome by the preliminary introduction into the rectum of a cocain suppository or a few drops of a 4 per cent cocain solution. A hypodermic dose (⅛ gr.; 0.008) of morphin may be given unless a poisonous antiseptic solution is to be employed. The irrigation is made through the long rectal tube, using from 2 to 6 pints (1 to 3 liters) of the solution at a temperature of 100° F. (37.5° C.). Pure water or a saline solution may be used first to cleanse the bowel, then an antiseptic or astringent solution. The solutions generally employed are: Mercuric chlorid (1:5000), quinin (1:2500), carbolic acid, tannic acid, zinc sulphocarbolate, salicylic or boric acid (1:2000 or stronger). Care must be exercised to thoroughly withdraw the solution from the bowel, especially if poisonous. Silver-nitrate solution is one of the most reliable remedies, particularly in chronic cases. It should be used in large quantity, 2 to 3 pints, containing gr. xx to xxx (1.3—2.0) to the pint. Irrigation should be practiced once or twice a day.

The diet should be largely fluid in character, consisting of milk, meat broths, beef-juice, junket, albumin water, milk-toast, and thoroughly boiled rice. During convalescence solid food must not be allowed too soon, and the patient should not be permitted to leave his bed or in any way to exert himself until all indication of intestinal ulceration has disappeared.

SMALLPOX.

VARIOLA.

Previous to the introduction of inoculation by Lady Mary Wortley Montagu and vaccination by Edward Jenner, smallpox was one of the worst scourges of humanity. It has prevailed from the earliest antiquity in India and China, and to such an extent in other countries that its origin cannot be determined. It was brought to the West

Indies in 1507, to Mexico in 1520, and to the United States in 1649. From its original foothold in Boston, it spread gradually westward until it reached the Pacific coast about one hundred years later. The name smallpox was given to the disease to distinguish it from syphilis, the "great pox."

Definition.—An acute infectious disease characterized by a sudden onset with violent pains in the head and back, rapid rise of temperature, followed by a remission, and an eruption which passes through the stages of papule, vesicle, pustule, and crust, sometimes complicated by cutaneous and visceral hemorrhages. The mucous membranes may also be affected.

Etiology.—Smallpox is one of the most virulently contagious diseases. Susceptibility to it is all but universal. The negro and other dark races and the aborigines of all countries are especially predisposed to it. All persons unprotected by vaccination are almost certain to be attacked after even brief exposure. Natural immunity seems to be possessed by a few individuals, for there have been instances in which persons resisted both smallpox and vaccination. The immunity conferred by an attack is not always permanent, but there are few well attested instances of a second or third attack.

Age and Sex.—The disease spares no period of life, but it is relatively more fatal in young children. Infants have been born with the eruption or with the scars. The fetus is sometimes attacked in utero, when the mother is suffering from the disease; but it may escape and can then be saved by vaccination immediately after its birth. Nurslings are believed to be less susceptible than infants of a year or more. The disease is sometimes contracted in extreme old age. Sex bears no relation to susceptibility.

The Contagium.—The ultimate source of infection in all cases is an individual suffering from the disease. The contagium exists in the cutaneous lesions and doubtless in the blood and secretions, probably also in the excretions, of the patient. It is given off in the exhalations from the lungs and skin, but it is nowhere more virulent, perhaps, than in the crusts, which are believed to be a frequent source of infection. The communication of the disease may be direct or indirect. The smallpox patient is always a source of infection from the first appearance of the eruption to the completion of desquamation. It is doubtful whether he gives off the infection before the appearance of papules, but in one instance the disease was communicated by grafts of skin taken from an individual who developed the disease a few hours later (Thompson). The most contagious periods are the stages of suppuration and desiccation. The disease may be transmitted by the clothing, bed-linen, or furniture; by anything that comes into contact with the patient. The contagium is retained by such fomites for years if protected from the air, and may be carried to a great distance. It has thus been transported across the Atlantic in baled rags. It clings tenaciously to a locality and is retained for some time in the body after death. It is doubtless carried to a considerable distance in air currents. Observations made at the Bradford, England, fever hospital indicate that it may be thus transmitted for a mile.

Bacteriology.—The protozoön organism recently isolated by Councilman, McGrath, and Brinckerhoff will doubtless prove to be the specific

cause of the disease. It is about 12μ in diameter, has two cycles of development, and produces spores 1μ in diameter. The sexes appear to be distinct, one of them developing only in the cytoplasm (protoplasm) of a tissue cell, the other in the nucleus. Numerous micrococci, probably pyogenic in character, have been found in the pustules.

The avenue by which the infection enters the system is not known, but it is probably the respiratory passages in most instances. The type of infection is not always transmitted, for the mildest case of varioloid may give rise to the most virulent form of smallpox in another person.

Morbid Anatomy.—*The Eruption.*—The eruption passes through four stages—the papule, vesicle, pustule, and crust. The papule corresponds to an area of hyperemia in the rete mucosum, which later gives place to a coagulation necrosis at the beginning of vesiculation. The filling of the vesicle with clear serum is due to the infiltration of the intercellular spaces with lymph containing leucocytes and fibrin filaments. The interior of the vesicle has a reticulated framework. An accumulation of pus-cells converts the vesicle into a pustule. The depression in the center of the pustule corresponds to the original area of coagulation necrosis. In the hemorrhagic form of the eruption, blood-corpuscles are found in the vesicles and in the deeper layers of the epidermis around them. The vesicle often surrounds a hair follicle. The depth to which the suppuration extends into the derma determines the extent of subsequent pitting. The entire process is due to the presence of bacteria. The eruption is found after death, not only in the skin and visible mucous membranes, as those of the mouth, tongue, cheeks, palate, pharynx, and larynx, but to a variable distance down the esophagus, trachea, and bronchi, sometimes in the rectum. Peyer's patches are not infrequently enlarged. Owing to heat and moisture, the mucous membrane eruption is soon converted into ulcers. Edema and membranous growth are often associated with the ulceration in the larynx. The laryngeal cartilages may be involved by a deep extension of the ulceration.

The blood-corpuscles form irregular clumps instead of rouleaux under the cover slip. Leucocytosis is generally pronounced at the height of pustulation, but rapidly declines with desiccation. The heart sometimes shows parenchymatous or fatty change. Endocarditis and pericarditis are frequent.

The changes in the liver are those accompanying febrile diseases, fatty degeneration, hyperemia, and migration of leucocytes.

The spleen is enlarged and firm in the hemorrhagic form of the disease. The kidneys may show cloudy swelling or coagulation necrosis, and nephritis sometimes develops during convalescence. Orchitis is sometimes found. In the hemorrhagic form, extravasations of blood are found in the parenchyma of various organs, in the connective tissues, and on the surfaces of the serous and mucous membranes, in the muscles, bone marrow, and elsewhere.

Symptoms.—Three forms of the disease are recognized: 1. Simple smallpox, variola vera; 2, malignant smallpox, variola maligna; 3, varioloid, smallpox modified by vaccination. They are all, however, different forms of the same disease, differing chiefly in severity.

Variola Vera.—***Incubation.***—The average duration of incubation is 12 days; it may be as short as 8 days or as long as 15, rarely longer. After inoculation the disease may appear in 48 hours. There are usually no prodromal symptoms.

Initial eruptions occur during the stage of invasion in from 10 to 16 per cent of cases and are often of great diagnostic value. Two forms of eruption are thus seen: (*a*) An erythematous rash, which may be diffuse, resembling scarlatina or erysipelas; and (*b*) a macular eruption resembling measles or urticaria. They are usually limited to the lower abdominal region, inner surface of the thighs, sides of the thorax and axillæ, or inner sides of the arms, but occasionally appear on the extensor surfaces, especially near the elbows and knees. The purely erythematous rash is of prognostic importance also, since it is almost invariably followed by a mild type of the disease. A petechial eruption appearing at this early period is generally of grave import, being followed, as a rule, by the malignant form.

Invasion.—The onset of the disease is generally announced by a chill or a succession of chills, with violent frontal headache, intense pain in the lumbar region and extremities, and persistent vomiting. In children the chill is generally replaced by one or more convulsions. Loss of appetite and thirst are constant accompaniments of the disease. Vertigo and syncope are frequently present; the tongue is furred, the breath is fetid, the throat sore, and the bowels are usually constipated. The temperature rapidly rises, reaching 103° or 104° F. (39.5°—40.0° C.) on the evening of the first day, and remaining high, often 104° or 105° F. (40.0°—40.5° C.), until the eruption appears. The pulse is rapid, 100 to 130, but full; seldom dicrotic. The respiration is rapid and labored, often out of normal ratio to the pulse. The face is flushed, the eyes are bright and clear, and the conjunctivæ are congested. The patient is generally restless and anxious, and often becomes delirious within the first few days. Profuse sweating occurs in many cases. The stage of invasion lasts three, sometimes four days, but its intensity is not an indication of the severity of the subsequent course of the disease, for the initial symptoms of varioloid are often severe, especially in women and children.

Stage of Eruption.—Four types of eruption may be encountered. 1. The discrete; 2, the confluent; 3, the hemorrhagic; and 4, the verrucose.

1. *Discrete Type.*—The eruption makes its appearance late on the third or on the fourth day. It comes out first upon the forehead, at the margin of the scalp, and on the wrists. Occasionally it appears also on the hands, sides of the neck, and upper lip. It appears first in the form of little, round, slightly elevated, pale red blotches, which feel to the touch like bird-shot in the skin, often before they can be distinctly seen. They can sometimes be seen in the mouth ten or twelve hours sooner than upon the forehead. In a few hours these original blotches become darker in color and distinctly papular. By the end of 24 hours they may be found on all parts of the body. They are usually less numerous upon the trunk than upon the extremities; the hypogastrium sometimes escapes, and the inner sides of the thighs seldom show a mature eruption.

On the fifth or sixth day of the disease, as a rule, the papules become converted into vesicles containing clear serum. These appear full and have an almost glistening appearance in a good light. When punctured, they do not collapse. Within a day or two they become umbilicated, a small indentation appearing in the center of each, and the fluid becomes gradually less translucent, rendering the pock opalescent and finally opaque. By the ninth or tenth day of the disease the vesicles are thus changed into pustules. The umbilication is then lost, and the pock assumes a full, globular form about the size of a split pea or larger. Surrounding each pustule is a zone of hyperemia, the "halo"; and the skin between the pocks is reddened, painful, and exquisitely sensitive, particularly on the face, hands, and feet. The transformation of papule into vesicle, and vesicle into pustule, begins on the face and follows the same order of progression as the original eruption.

With the appearance of the eruption the temperature falls nearly or quite to the normal; the pain and other symptoms subside. When the pustules mature, the temperature again rises nearly or quite as high as in the stage of invasion, and the other symptoms return, often with even greater severity, but, as a rule, the temperature begins to decline again in from 24 to 48 hours.

The swelling of the face is so great at this time that the eyes are closed. The maturing of the pustules requires about three days. In some cases by about the eleventh day the pustules begin to rupture, either spontaneously or by accident, and sometimes the pus seems to ooze from them without actual rupture. This gives the patient a most loathsome appearance and a peculiar, fetid odor. After this, desiccation rapidly progresses. The crusts are usually formed by the fourteenth or fifteenth day of the disease, by which time the temperature has usually fallen and convalescence is fully established. Desquamation begins about a week later, the twenty-first to the twenty-fifth day, first on the face, and follows the order of the eruption. In severe cases the fever sometimes continues through the third and fourth weeks.

The eruption on the mucous membranes soon becomes converted into more or less confluent ulcers, as stated under Morbid Anatomy. The lesions are seen especially in the mouth, tongue, soft palate, cheeks, the conjunctivæ, in the nose, pharynx, rectum, and vulva, and are usually accompanied with intense inflammation of the surrounding areas, seriously interfering with the function, and sometimes leaving such permanent damage as the loss of sight.

2. *Confluent Type.*—The confluent type of eruption is seen, as a rule, only in the most severe cases. It generally appears as a numerous collection of discrete papules, usually on the second day of the disease, instead of the third or fourth, and spreads in the same manner as does the discrete form. The confluence is due to the coalescence of groups of vesicles. Large blebs are thus formed, which rupture rapidly, and the entire face is sometimes covered by one large, firm crust. All the symptoms are more severe and more protracted than in the discrete type, and there is little or no remission at the appearance of the eruption. Sepsis is manifested in many cases by the occurrence of repeated chills and hyperpyrexia, and not infrequently this is the cause of a fatal issue. The nervous manifestations are also more prominent. The de-

lirium may become violent, often with a suicidal tendency, or it may pass into coma. The cervical glands become markedly swollen in most cases, and salivation and diarrhea often add to the gravity of the situation. Desiccation does not begin until the third or fourth week, and the crusts separate in large masses. Osler refers to entire molds of the hands and feet.

3. *Hemorrhagic Type.*—This is an alteration in the character of the eruption which is due to the escape of blood into the vesicles or pustules. The initial hemorrhagic eruption, when it appears in the malignant form of the disease, is generally accompanied by profuse hemorrhages from almost every part of the body (see Malignant Smallpox). It helps to make up the symptomatology of the disease that is described as purpura variolosa; hemorrhagic, or black, smallpox.

Another form of hemorrhagic eruption is seen when hemorrhage occurs into the vesicles or pustules at any time during the course of the disease (variola hemorrhagica pustulosa). It is especially likely to occur in alcoholic subjects, or others in a debilitated condition, but it is sometimes encountered in robust young men. It may result from leaving the bed too soon, and is then generally confined to the lower extremities. It is not usually a fatal complication.

4. The *verrucose eruption* is a rare form in which the vesicles partially dry and remain adherent to the skin, particularly on the face, resembling horny warts.

A crystalline eruption is also described in which the vesicles rapidly dry without undergoing pustulation.

Malignant Smallpox (Variola Maligna).—This form is characterized by profound alteration of the blood, with resultant purpuric or hemorrhagic eruptions and hemorrhages from the mucous membranes and into the substance of various organs. Klebs, Unna, and others attribute the cutaneous hemorrhages to blocking of the vessels of the skin by bacteria. Another explanation is that an acute hemophilia is established through a dissolution of the blood by the infectious agents (hematolysis), and hematin is deposited in the substance of the skin and other tissues. In some cases death occurs before an eruption appears. The shotlike feel of the skin may be found in such cases.

The onset is unusually severe. On the evening of the second or third day, as a rule, a diffuse rash appears in the groin, inner surfaces of the thighs, axillæ, or one of the other regions already referred to, with small punctiform hemorrhages. The fever is usually slight or it may be absent. The eruption extends and soon becomes hemorrhagic; ecchymoses appear on the mucous membranes, including the conjunctivæ. Profuse hemorrhages from various sources, as the nose, mouth, bowels, stomach, kidneys, even from the eyes, rapidly sap the vitality of the patient. The face becomes enormously swollen, purple in color, the conjunctivæ project over the shrunken corneæ. Retinal hemorrhages frequently destroy the sight. The patient rarely survives longer than the fifth day. The mind may remain clear to the last; as Curschmann remarks, "Only a few patients are so fortunate as to fall speedily into delirium or coma."

Varioloid (variola benigna) is a form of smallpox which has been rendered mild and comparatively harmless by previous vaccination,

rarely, perhaps, as a result of natural insusceptibility or a previous attack of the disease. In most cases the disease differs from true smallpox only in the lighter character of the symptoms, but cases have been described in which the eruption failed to appear, or rapidly underwent resolution after reaching only the stage of papulation or vesiculation. Osler, in his large experience at Montreal, failed to encounter a case without eruption.

The invasion may be severe, with chill, headache, backache, vomiting, and a rapid rise of temperature to 103° F. (39.5° C.), sometimes higher. The papular eruption appears at the end of the third day, and the temperature and other symptoms recede. The papules are, however, few in number, often not more than a dozen, mostly on the face and hands, with perhaps one here and there over the trunk. Vesiculation and maturation of the pustules progress rapidly, and there is usually no second rise of temperature. Desiccation begins from the fifth to the seventh day. The crusts are shallow, and pitting does not usually remain.

Complications and sequelæ are numerous. *Septicemia* and *pyemia*, with resultant abscesses, or a general furunculosis, are not uncommon, yet more so than might be anticipated in a disease attended by so extensive suppuration. Fatal pyemia sometimes develops during the stage of desiccation. Pigmentation of the skin occasionally remains after the disease. A second (recurrent) eruption has been described. Lobular pneumonia is a frequent, often fatal complication, particularly in children; pulmonary congestion and pleurisy are common in some epidemics and may result in empyema; lobar pneumonia is rare. The laryngitis, so commonly present, sometimes leads to fatal edema of the glottis, rarely to necrosis of the cartilages. Myocarditis is the most frequent of the heart complications and may be associated with endarteritis of the coronary artery; a systolic apex murmur may be heard during the height of the fever. The initial vomiting rarely persists; diarrhea is often present; parotitis occasionally develops. Orchitis and ovaritis have been observed. There are sometimes sequelæ on the part of the nervous system during convalescence; cerebritis, peripheral neuritis, paraplegia, and other paralyses probably due to peripheral neuritis or diffuse myelitis, optic neuritis, and rarely insanity, are encountered. Conjunctivitis is always present in severe cases; retention of the purulent secretion often leads to keratitis, ulceration, and perforation; iritis and glaucoma may develop. Otitis media sometimes occurs. Nephritis is seldom encountered, although albuminuria is often present.

Diagnosis.—Error is most likely to occur in the absence of a known source of infection or when, as may happen in varioloid, the initial symptoms are unusually mild. When the disease is prevalent, the history of a chill followed by high fever, 103° to 106° F. (39.5°—41.0° C.), with vomiting, severe headache, and lumbar pain, should always arouse suspicion of the disease. The infections most likely to be confounded with smallpox are measles, scarlatina, and chickenpox; typhus, cerebrospinal meningitis, and a few other conditions may occasionally be suggested.

In measles the initial symptoms are not so severe and the temperature rises gradually; when the eruption appears, the papules are soft and

cannot be felt when the skin is stretched, as can those of smallpox. In the latter disease, coryza, photophobia, injection of the conjunctivæ, cough, and Koplik's spots are all absent. The initial papular eruption of smallpox is sometimes identical with that of measles.

In *scarlet fever* the onset is sudden with high temperature, nausea, vomiting, and headache; backache is not usually complained of. The initial erythematous eruption of smallpox is generally pale and the strawberry tongue is not seen. Scarlet fever is more uniformly a disease of childhood.

Chickenpox is distinguished with most difficulty from varioloid, particularly when both diseases are prevalent at the same time. It is distinguished by the character and location of the eruption and the course of the disease, which are more fully considered in the Diagnosis of Chickenpox, page 267.

Cerebrospinal meningitis resembles smallpox only in the severity of the onset, with headache, backache, vomiting, and rapid rise of temperature. It may be excluded by the rigidity of the neck muscles, retraction of the head, the taches cérébrale, muscular twitchings, and the character of the eruption, which does not become vesicular.

Typhus fever closely resembles smallpox in its invasion, but it is a much less frequent disease; the eruption appears on the chest and abdomen, first as maculæ, which later become converted into petechiæ; they do not give the sensation of shot to the touch, vesicles and pustules do not occur, and the temperature continues to rise after the appearance of the eruption.

Glanders has been mistaken for smallpox; but it is a rare disease, seen only in those caring for horses, and ulceration very speedily occurs in the cutaneous infiltration. The febrile symptoms are much the same.

Syphilis.—The areolar and pustular eruptions of syphilis have been mistaken for variola. The history of the disease, absence of sudden invasion, slight if any elevation of temperature, and the enlargement of inguinal glands, generally suffice for differentiation.

Ptomain poisoning, with high fever, vomiting, diarrhea, headache, and an erythematous rash, may resemble smallpox; but the course of the affection is altogether different after the first day, and the cause of the poisoning can generally be discovered.

Medicinal rashes rarely cause confusion. That of potassium iodid may resemble variola pustules, and croton oil has been applied with intent to deceive, but all other symptoms are absent.

Prognosis.—The mortality in persons unprotected by vaccination is 25 to 35 per cent, sometimes higher. The mortality of varioloid is a little more than 1 per cent. The statistics of 15,000 deaths show the following rates: Among unvaccinated, 35 per cent; among those having one vaccination scar, 7.73 per cent; two scars, 4.7 per cent; three scars, 1.95 per cent; four or more scars, 0.55 per cent. Much depends upon the character of the epidemic, the constitution and age of the patient. Children under five years, as a rule, succumb. The malignant form is almost universally fatal; cases in which the eruption is purpuric may recover. The confluent eruption indicates a less favorable prognosis than the discrete, and the gravity of the disease can, as a rule, be estimated by the intensity of the eruption on the face and hands.

When the fever increases after the appearance of the eruption, the prognosis is unfavorable. Delirium and convulsions are grave symptoms. Severe laryngitis adds danger through liability to the development of edema of the glottis. Pregnancy renders the case less hopeful; abortion usually occurs.

Prophylaxis.—Effective prophylaxis can be secured only through universal, compulsory vaccination. The disease was almost eradicated from Germany by the vaccination law of 1874.

As soon as the disease is suspected in a case, the patient should be isolated. It is generally better for him, and always safer for the community, to have him placed in a special hospital. Every member of his household should be immediately vaccinated. If he is kept at home, a large, well-ventilated room should be prepared by the removal of carpet and all unnecessary furniture and drapery. Only the physician and nurse should be permitted to visit the patient. The physician should protect himself from the danger of conveying the disease, by wearing a linen gown and oilcloth cap, and he should make his visits brief.

The urine, feces, and all discharges should be disinfected with as much care as those of typhoid fever, by the addition of mercuric chlorid solution (1:500), and the clothing of the patient and nurse should be disinfected by steam or by soaking in a 1:20 solution of carbolic acid, followed by boiling. All utensils, bedclothing, everything that comes into contact with the patient must be thoroughly disinfected or destroyed by fire after the recovery of the patient. The room should also be thoroughly disinfected with formaldehyd vapor for 24 hours, and the walls, ceiling, floors, and bed should be scrubbed with corrosive sublimate solution, 1:1000.

Treatment.—The diet of the patient should be of the most nourishing character. During the febrile stage it must be liquid in form—milk, broths, and an abundance of water, lemonade or other fruit-juices in water. During the remission of the fever, eggs, toast, and soups may be added; in the stage of suppuration, stimulants should be given to meet the decline of strength. Pledgets of ice are gratifying and soothing to the throat. Ice-cream may be allowed when the stomach will tolerate it. The fever and prostration may be counteracted by cool baths or cold sponging; antipyretics should be administered with caution, if at all, on account of their depressing effect. Vomiting is to be checked by the administration of champagne, dilute hydrocyanic acid, cocain, or small doses of calomel in the absence of diarrhea. Excessive diarrhea may be controlled by the camphorated tincture of opium, bismuth or lead acetate with opium. The nervous manifestations call for the bromids, stimulation, and baths of 70° F. (21.0° C.) or cold sponging repeated as often as the temperature rises to 103° F. (39.5° C.). Morphin may be required to relieve the pains.

The Eruption.—Much can be done to relieve the painfulness of the eruption, and many measures are employed to limit the pitting. Frequent bathing with a 1:5000 solution of corrosive sublimate or 1:20 of carbolic acid is one of the best measures. Hydrogen peroxid may be employed to cleanse away the pus. The addition of gylcerin to any of these solutions is recommended by some writers. Ichthyol collodion is probably the best application for the face and hands. Masks of lint

saturated in the bichlorid solution should be constantly worn on the face and hands and covered with oil-silk, for not a little of the benefit to be derived from them is due to the exclusion of light. All crusts, not overlooking those of the nose, should be kept thoroughly moist, and scratching should be prevented by restraint of the hands or by thoroughly enveloping them in the moist lint. Applications of oil, vaselin, or glycerin to the crusts are preferred by some authors. The hair should be closely cut.

The eyes must be carefully looked after and cleansed three or four times a day, irrigated with boric acid solution, and vaselin should be applied to prevent adhesion of the lids. A spray of corrosive sublimate 1 : 1000 has been recommended for use in the nose, mouth, and ears.

In the hemorrhagic form of the disease, nothing can generally be done, but full doses of ergot, tincture of ferric chlorid, gallic acid or turpentine may be tried. Tracheotomy may be resorted to when edema of the glottis develops. During convalescence, the patient should bathe often, sponging with carbolic acid or other antiseptic solution. His danger to others ceases only after the skin has become entirely free from all traces of crusts.

VACCINATION.

Definition.—The artificial production of immunity to smallpox by inoculation with the virus of vaccinia, or cowpox.

The nature of vaccinia has not been determined. Many of the best authorities regard it as a form of smallpox modified by transmission through the body of the cow, while others look upon it as a distinct disease.

The lymph for use in vaccination is obtained from vesicles on the udders of young heifers or calves before the lymph has become turbid. The virus is then dried on ivory "points" or hermetically sealed in glass tubes. The danger of inoculating with other diseases by the use of humanized virus—lymph obtained from a child after vaccination, or from the dried crust—is so great that it should never be employed except in case of emergency. When this arises, the lymph should be taken, if possible, from a clear, unbroken vesicle on the eighth day after vaccination, the utmost care being exercised to obtain it from a robust, healthy child free from tuberculous or syphilitic taint.

The Method.—The place selected for scarification is usually on the outer side of the left arm over the insertion of the deltoid. In girls the leg is sometimes selected for esthetic reasons. This is then cleansed and rendered aseptic. The skin is stretched between the fingers and a spot about an eighth of an inch in diameter is scraped with a clean lancet, the ivory point, or a needle until serum begins to ooze. Blood should not be drawn. The charged end of the point is then dipped in clean water and rubbed over the scarification until the coating has been removed. It is advisable to continue the friction until a slight blush appears in the adjacent skin. The vaccination is allowed to dry before the clothing is adjusted. After the arm has become inflamed and painful, the comfort of the child may be added to by the application of a shield or a linen patch greased with white vaseline and covered with a pledget of cotton. In case the pock becomes ruptured,

the arm should be bathed with an antiseptic solution to prevent inoculation of the surrounding skin by the pus discharged from it. In the presence of an epidemic it is safer to scarify two or three places, inoculating each with a different point, unless the freshness of the virus is positive; in ordinary cases one scarification is sufficient. Infants are usually vaccinated in the second or third month; after exposure it should be done immediately without regard to age. A child should be vaccinated not later than the tenth year, but in times of danger it is better to repeat the vaccination at any time, even after but one or two years have elapsed, until three or four successes have been secured.

Symptoms.—The history of the lesion produced by vaccination is in a measure similar to that of a single smallpox papule. On the third or fourth day after vaccination, a small red papule appears, surrounded by a hyperemic areola. This increases in size until the fifth or sixth day, when it changes into a vesicle with a depressed center. The vesicle enlarges and the umbilication becomes deeper until by the tenth day, as a rule, the pustule has become mature. About the eleventh or twelfth day the hyperemia begins to subside and desiccation begins. The scab separates about the 21st to the 25th day.

The constitutional symptoms are usually mild. On the fourth or fifth day there is often a slight elevation of temperature, which may persist for a few days. Nausea and vomiting sometimes occur. The lymph-glands nearest to the site of vaccination become enlarged and sensitive.

Irregular Symptoms and Complications.—In some cases the formation and progress of the pock are unusually rapid, in others they are delayed beyond the usual time. Injury of the sore may cause unusual inflammatory extension and ulceration. A recurrence of the pock has been seen in a few instances. A more or less protracted generalized pustular eruption sometimes occurs and has proved fatal to young children (generalized vaccinia). An eruption of vesicles within a limited area around the vaccination is not unusual. In unhealthy children or as a result of uncleanliness in the vaccination or in the subsequent care of the arm, deep ulceration occasionally occurs; lymphangitis and gangrenous sloughs sometimes form and sepsis may develop. Tetanus is extremely rare. Among the infrequent complications are erysipelas, urticaria, erythema multiforme, eczema, and lichen. Syphilis has been inoculated by the use of humanized virus. That tuberculosis or leprosy has ever been conveyed with the vaccine has not been definitely shown.

CHICKENPOX.

VARICELLA.

Definition.—An acute, contagious disease of children, characterized by a vesicular cutaneous eruption and mild constitutional disturbances.

Etiology.—The disease is highly contagious, often becoming epidemic, especially in schools and asylums. Spring and autumn are the seasons of its greatest prevalence, but sporadic cases occur at any time of the year. The specific cause is not known. Although peculiarly a disease of childhood, it not infrequently attacks adults. It is in no way related to smallpox, but often occurs in epidemic form shortly before, after, or dur-

ing an epidemic of that disease. One attack usually confers immunity, but recurrences have been noted.

Symptoms.—Incubation lasts from ten to fifteen days, occasionally a day or two longer. Slight prodromal indisposition is sometimes noticed.

In mild cases the invasion is not attended with constitutional disturbances, but there are generally an elevation of temperature to 100° or 102° F. (38°—39° C.), chilliness, anorexia, nausea, perhaps vomiting and restlessness, and slight muscular pains. The eruption usually appears within 24 hours, on the face, neck, and scalp; it later becomes even more abundant on the trunk and extremities, particularly on the back. It consists, at first, of slightly raised, round or oval, red papules, which within a few hours become converted into clear vesicles. These vary in size from a pinhead to a dime; the largest are found upon the forehead and back; the total number varies from a half-dozen to two hundred or more. Distinct umbilication does not occur; but by the second day, sometimes earlier, the tops of the vesicles become flattened. They become purulent within 36 to 48 hours. They are not surrounded by a distinct areola, but the external zone of the pustule may have a dark red color, particularly after it has been ruptured and converted into an ulcer. By the third or fourth day the pustules have dried into dark brown crusts which usually become detached in a few days and leave no pits, except as a result of scratching. Successive crops of vesicles appear in most cases, so that it is not unusual after the third day to find fresh vesicles among the drying pustules. The mouth is sometimes invaded by the eruption, but the vesicles are there quickly converted into open ulcers by the saliva. The conjunctiva, larynx, and trachea are seldom involved.

The temperature does not always decline with the appearance of the eruption as in variola. The only prominent symptom, aside from the eruption, in most cases, is the itching, which is sometimes most annoying. Anomalous cases occasionally occur. The vesicles may be extraordinarily large, measuring from a half to three-quarters of an inch in diameter and resembling ecthyma or pemphigus. In rare cases, owing to infection from the finger-nails, the skin around the ulcer becomes gangrenous. Cases of hemorrhagic varicella have been described. Complications are few. Erysipelas has been observed; the lymph-glands are sometimes enlarged and may suppurate; nephritis is sometimes observed, and death has been attributed to an unusually extensive involvement of the skin.

Diagnosis.—The differentiation of varicella from varioloid is often of the greatest importance and is sometimes exceedingly difficult. It is to be made for the most part by a study of the eruption, although the mild stage of invasion is of much value. In varicella the greatest number of vesicles is generally found upon the trunk. The vesicles are more superficial, not so fully globular, may be oval, are not umbilicated, but rather flat in the center, and, when punctured, they collapse. The original papules have not the shotlike feel of variola, and it is usual to find recent vesicles distributed among the pustules and crusts. Initial rashes are occasionally seen in varicella, but not so frequently as in smallpox. The eruption is never confluent.

Treatment.—The disease is so harmless that isolation is seldom enforced; it is safer, however, to at least protect delicate children. Con-

finement to the house and light diet should be ordered during the existence of the fever. Internal medication is unnecessary. To relieve the itching, the skin may be sponged several times a day with a carbolic acid solution, 1 : 20, or carbolated vaselin may be applied. Anders recommends the application of ichthyol (2 per cent) in zinc ointment after the crusts have formed. Scratching should be prevented by placing mittens on the hands if necessary.

PSOROSPERMIASIS.

Psorosperms, known also as sporozoa or gregarinidæ, are best known as they occur in the lower animals, for they have been rarely found in the human being. In some instances, small, nucleated, spherical organisms, usually termed coccidia, have been discovered in the cells of the body, especially in those of the intestine, spleen, liver, kidneys, and serous membranes. They produce small nodules which are readily mistaken for tubercles. In the liver, indeed, they sometimes form palpable tumors. Several cases of peculiar skin disease have been attributed to them, and, on the other hand, several diseases formerly so regarded have more recently been referred to other causes.

Symptoms.—Internal psorospermiasis is usually attended with indications of local disturbances in the organ affected, pain and tenderness, sometimes accompanied with diarrhea. Peritonitis may be produced. When the kidneys are affected, there is hematuria. The disease may prove fatal within a few weeks, or it may last for several years. In the cutaneous cases the nodules appear first upon the face and extend to the other surfaces of the body. The lymph-glands and lungs are later invaded, as a rule, and often contain nodular masses in which the psorosperms are found.

INFUSORIA.

Quite a number of infusoria have been discovered within the body, but they are for the most part harmless and not productive of symptoms.

1. The **cercomonas intestinalis** is a pear-shaped parasite having a sharp anterior extremity provided with a short cilium, and a broad posterior extremity to which is attached a tail-like flagellum. It has been discovered in connection with different diarrheal conditions, but is not known to have pathological properties. 2. The **cercomonas coli hominis** was observed in one instance by May, a case of carcinoma of the stomach with chronic diarrhea. The organism is about as large as a red blood-cell, spindle-shaped, the anterior end being blunt and provided with four cilia. 3. The **trichomonas intestinalis** is also a pear-shaped organism from 10 to 15μ long and 7μ broad and endowed with ameboid movement. It is found in diarrheal discharges. 4. The **trichomonas vaginalis** has been found in cases of vaginitis, but is not known to bear a relation to the disease. It is smaller than that found in the intestine.

5. The **trichomonas flagellata, caudata,** and **elongata** have been found in the mouth by Sternberg. Similar flagellate bodies have been

seen in the expectoration from gangrene of the lung, bronchiectasis, and pleurisy.

6. The **balantidium coli** is an ovoid body 7 to 10μ in diameter and surrounded by cilia. Oral and anal apertures are recognizable. Its natural habitat is in swine. Man becomes infested through contaminated water or food. It has been found in diarrheal and dysenteric conditions, but is not known to be the cause of them. The *megastoma entericum* has also been seen in great numbers in the stools of chronic diarrhea.

THE TREMATODES.

Distomiasis is the name applied to the diseases produced by the trematodes or flukes, several of which are found in man, notably: (1) The liver flukes, (2) the blood flukes, and (3) the bronchial flukes.

1. **Liver Flukes.**—The most common of these is the distoma hepaticum (fasciola hepaticum), yet Huber found only 22 cases in the literature of over a hundred years prior to 1896. It is a broad, lanceolate body from 28 to 32 mm. in length. It is found in the liver, gall-bladder, and bile-ducts. Its intermediate host is a small snail which crawls over grass and water-cresses, and this fact probably explains its occasional appearance in man, and much greater frequency in sheep. The distoma lanceolatum is a smaller parasite, 8 to 10 mm. long. It has been found in the same locations. The distoma Buski (or D. crassum) is a large fluke measuring 6 to 8 cm. in length. Other flukes occasionally found in the liver, though not in this country, are the distoma Sibericum, D. spatulatum (or sinense), and D. conjunctum.

Symptoms.—There is usually some indication of hepatic and intestinal irritation and chronic cholangitis, with resultant diarrhea, enlargement of the liver, jaundice, and sometimes ascites. Progressive emaciation is usually observed. The eggs are frequently found in the dejections, and the parasites sometimes find their way into the subcutaneous tissue.

2. **Blood Flukes** (Distoma Hematobium, Hematobia Bilharzia).—This is a flat worm with the two sucking disks on the abdomen. The male is shorter and thicker than the female. It is an exceedingly common parasite among the children in Egypt, and it is believed to enter the body more commonly through the integument while the individuals are bathing than through the medium of drinking-water, as was formerly supposed.

Symptoms.—The portal system is especially affected. The eggs are found in the veins of the small intestine and rectum, kidneys and bladder. Micturition is painful, and blood, pus, and the sharp-pointed, ovoid eggs of the fluke are found in the urine. The mucous membranes of the renal pelvis, ureter, and bladder are much thickened, and large calculi are sometimes formed. Leucocytosis and eosinophilia have been observed in at least two instances.

Treatment.—This consists in the administration of full doses of male fern or thymol. The bladder should be irrigated with a 1:1000 solution of mercuric chlorid.

3. **Bronchial Flukes.**—The distoma pulmonale (D. Westermanni) is common in Japan, China, and Formosa, where the disease becomes epidemic. It has probably never been seen in this country, except in

the cat and dog, by Ward of Nebraska. It is 8 to 10 mm. long, 5 to 6 wide, club-shaped, pointed in front, blunt behind. It has the color of the earthworm and the movements of a leech.

The ***symptoms*** are cough, reddish-brown sputum containing the ova and rarely the parasites.

In addition to these trematodes, several others have been encountered, notably the distoma ophthalmobium and the monostoma lentis, found in the eye; distoma heterophyes and amphistomum hominis, found in the small intestine.

Anneledes.—The hirudinea, or leeches, of the United States seldom cause more than a slight temporary disturbance by their bites. In Ceylon and certain parts of South America, however, large ulcers are frequently produced by the hirudo Ceylonica; and in Europe and Africa, a leech known as the herudo vorax attacks the mucous membranes of the nose, mouth, larynx, and trachea, producing severe inflammations.

DISEASES CAUSED BY NEMATODES.

ASCARIASIS.

The ascaris lumbricoides is the most common of all the human parasites and is particularly frequent in children. It is a cylindrical worm, pointed at both ends, and of a yellowish or reddish color. The mouth has three lips and the body is marked by numerous tranverse striations and four longitudinal bands. The female is usually from 8 to 12 inches (20 to 30 cm.) in length. The male is shorter and is recognized by a curved caudal extremity. The ova, often found in great numbers in the feces, are broadly elliptical, about 0.05 mm. long, and surrounded by a clear albuminous layer. The worm contains an odorous substance to which some of the symptoms are thought to be due. There is no intermediate host; direct transmission and development of the eggs have been experimentally demonstrated. The worm lives in the small intestine. Only one or two are generally present, but large numbers are occasionally found.

Symptoms.—In most instances no symptoms are observed which can be attributed to the presence of the worm. In other instances continued fever, restlessness, twitchings, even convulsions are regarded as due to the reflex irritation caused by large numbers or to the absorption of the volatile substance produced by them. Intestinal symptoms, intermittent diarrhea, abdominal pains, and foul breath are sometimes complained of. Anemia develops in prolonged cases. Such symptoms as itching of the nose, grinding of the teeth, and talking in the sleep are always associated in the minds of mothers with the presence of worms in the intestinal canal.

Disturbances of a more serious nature are sometimes caused by the migration of the parasites into the ducts entering the intestine, especially the bile-duct and to more remote parts. The pancreatic duct has been entered, intestinal ulcers have been perforated; and there is little doubt that the normal intestinal wall has been perforated by them. They not infrequently pass up to the stomach and may be vomited. In some

instances they have passed through the esophagus into the pharynx, Eustachian tube, or larynx. Fatal asphyxia has followed obstruction of the larynx; bronchitis has followed only a temporary lodgment in the trachea, when the worm was coughed out, and gangrene of the lung has been induced by their lodgment in the bronchi. The accumulation of a large roll of worms in the intestine has led to obstruction.

Treatment.—Santonin is the best remedy. It is given to a child in doses of gr. ¼ to j (0.016—0.06), or gr. ij to iij (0.15 to 0.20) to an adult, three times daily for two or three days, and followed by a purge—calomel, magnesium citrate, or castor oil. Or calomel, gr. 1-10 to ¼ (0.006—0.016), may be combined with the santonin. The santonin crystals should be mixed with sugar without trituration, since the drug is then less likely to be absorbed. Such disturbance as yellow vision (xanthopsia) is seldom observed, but the urine is sometimes discolored.

Oxyuris Vermicularis (Thread-Worm, Pin-Worm).—This is a small, white, threadlike parasite. The length of the female is 10 mm., that of the male 4 mm. It is found at any age, but most frequently in children. Its habitat is the rectum and colon. Huber asserts that the male is commonly found in the ileum. Large numbers can usually be detected in the feces of those infested. The eggs are seldom, if ever, found. There is no intermediate host, and the eggs or the parasites themselves are believed to be transferred directly by the hands of their host to others, and possibly through the medium of food or drink. The worms migrate at night and may pass out of the rectum. Great numbers are frequently found in the anus, and they often pass into the vagina. They have been found also in the nose and other regions whither they had been carried by the fingers of the child.

Symptoms.—The most frequent symptom is pruritus, which becomes so greatly aggravated at night by the wandering of the worms that the child becomes restless, sometimes almost frantic. Convulsions are occasionally produced. The patient may become anemic and emaciated. Perirectal abscesses have been induced by the irritation and scratching. Many nervous disorders have been attributed to the parasite.

Treatment.—All that is necessary in most cases is a daily enema of a cold, strong salt solution, or of infusion of quassia or aloes, for ten days. In some cases irrigation of the bowel with solutions of carbolic acid or turpentine is more effective. When these measures fail, santonin and calomel should be administered per os, as in the treatment for lumbricoid worms. The itching may be relieved by the application of carbolated vaselin to the anus.

Ascaris Alata (A. mystax) is a worm from 2 to 3 inches (4 to 8 cm.) long, of frequent occurrence in the dog and cat, but very rare in man.

TRICHINOSIS.

TRICHINIASIS.

Definition.—A disease produced by the embryos of the trichina spiralis in their migration from the intestine to the skeletal muscles, and

characterized by pain, tenderness, and swelling of the muscles, edema, and fever.

The Parasite.—The female trichina measures 3 or 4 mm., the male 1.5 mm. The latter has two hooked projections from the posterior extremity. The body is surrounded by fine striations, and around the middle portion of the intestine a collection of large cells may be seen, which is of value as a diagnostic feature. The mature worm inhabits the intestine of man and of many of the lower animals, especially swine and rats.

The larval trichina is from 0.6 to 1 mm. in length and is best known as it lies coiled in its capsule between the muscle fibers. The capsule is a clear, translucent membrane produced as a result of the irritation set up by the presence of the parasite. In the course of time it becomes calcified. In this condition the trichinæ remain dormant, but retain their vitality for many years.

Man becomes infested through eating meat containing the living larvæ. These are liberated through digestion of the capsule by the gastric juice, and pass into the small intestine. Here they acquire their full growth and become sexually mature in about three days. By the end of five days living embryos are found in the intestine. The trichina is viviparous, the female giving birth to several hundred living young, possibly to successive broods, during the five weeks of her life. But, although living embryos are found in the intestine, it is believed that those which eventually reach the muscles are born in the lymph-spaces of the intestinal wall and mesentery, especially in the Peyer's patches and mesenteric glands, since the impregnated female is known to pass out of the intestine, and the embryos have been found in large numbers in these places. Through the lymph circulation they pass into the blood, and with it they are carried to the muscles of all parts of the body. From the intermuscular connective tissues they pass into the fibers, where they reach their full growth in about two weeks. The capsule is formed, no doubt, as a result of the interstitial myositis produced by the irritation of their presence. A single embryo is usually found within each capsule, but occasionally there are three or four. The formation of the capsule requires about six weeks. The calcification is a later change, sometimes seen as early, however, as the fourth or fifth month after the invasion. It at first affects only the extremities of the capsule, but later involves the entire sac and sometimes the embryo. Every stage in the life cycles of the trichina may be studied from Plate VIII.

The common source of human infection is the meat of the hog. Raw or insufficiently cooked pork and sausage are the usual forms in which this is eaten. The danger is removed by submitting the meat to a boiling temperature. The disease has assumed epidemic form in a number of instances, all the cases originating, as a rule, from one source. Three hundred persons have been simultaneously affected. Such outbreaks have generally occurred in Germany, where the custom of eating raw ham and sausage prevails.

Morbid Anatomy.—The changes are found in the voluntary muscles. The number of parasites is enormous. When death has occurred in the fourth week, they are found mostly in the head and trunk; after the sixth week, also in the extremities. They are numerous in the intestinal

EXPLANATION OF PLATE VIII.

FIG. 1.—Muscle Trichina enclosed in a fully developed cyst, X 240. *Cy*, cyst: *Bg*, connective tissue envelop; *Fk*, fat globules.

FIG. 2.—The trichina removed from the cyst, X 400. *Oe*, esophagus; *Zk*, cell bodies; *L*, side lines; *Ov*, ovary; *Ch D*, chyle duct.

FIG. 3.—Part of the ovary, X 600. The ovary is readily distinguished from the testicle by the varying size of the germ cells.

FIG. 4.—Male intestinal trichina, X 100. *H*, testicle; *d ej*, ejeculatory duct; *Z k*, cell bodies.

FIG. 5.—Female intestinal trichina, X 90. *Ov*, Ovary; *E*, embryos; *Oe*, genital opening from which the embryos escape.

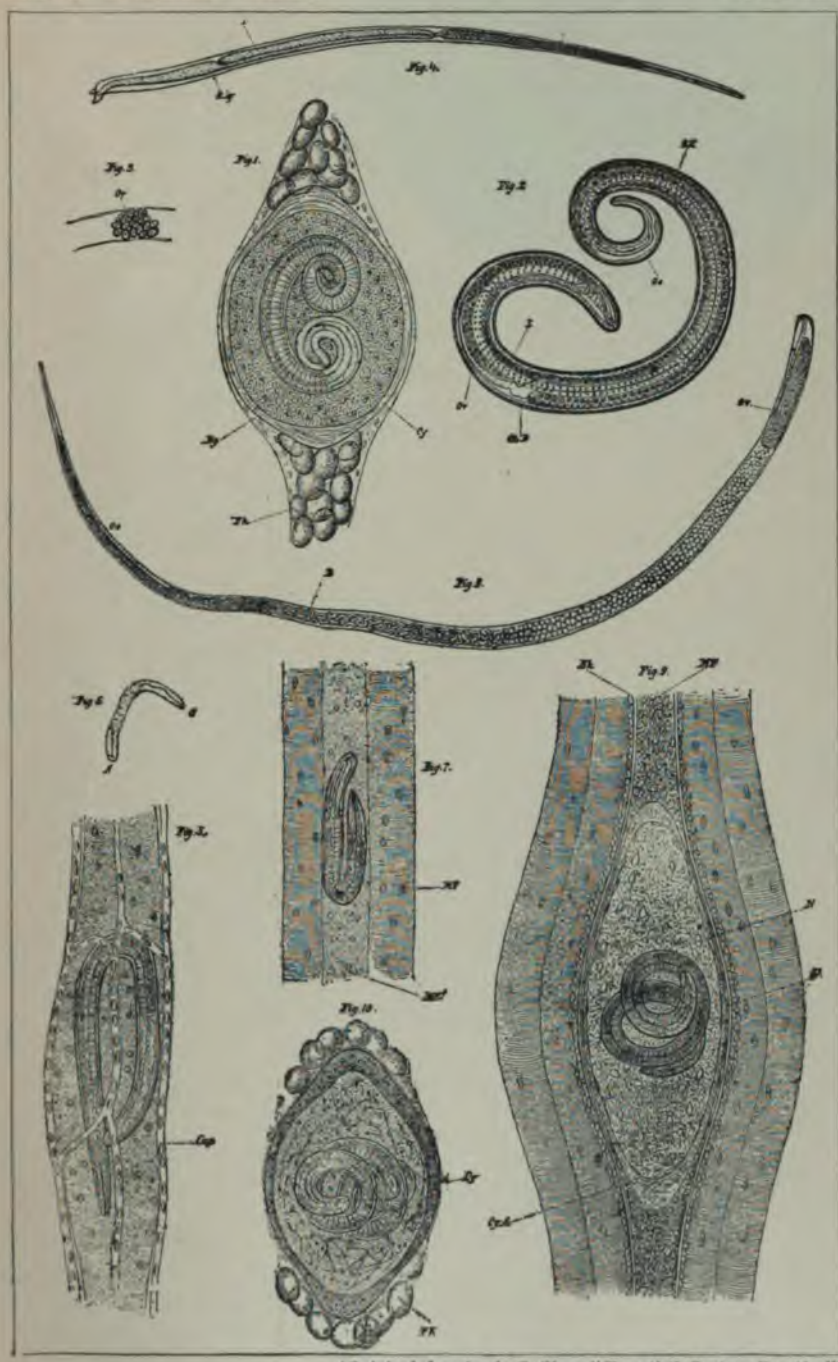
FIG. 6.—Free embryo, X 400. *O*, mouth; *A*, anus.

FIG. 7.—Embryo about three days after having entered the muscle fiber. *MF*, normal muscle fiber.

FIG. 8.—Muscle trichina about six days old, in the greatly swollen sarcolemma sheath traversed by capillary vessels, *Cap*.

FIG. 9.—Muscle trichina four weeks old, enclosed in a capsule. *Cy A*, within the sarcolemma sheath, *Sk*; *Bk*, the connective tissue capsule in process of active growth; *k*, nuclei; *MF*, contents of the sarcolemma sheath at each pole of the capsule.

FIG. 10.—Muscle trichina with calcified capsile; *Fk*, fat globules.



(*Original drawing by C. Claus, "Twentieth Century Practice."*)

TRICHINA SPIRALIS.

contents. The mesenteric glands are enlarged; the muscles grayish or brownish red. The degenerative changes are seen in longitudinal streaks from 0.5 to 2 mm. in length. Bronchitis, hypostatic congestion, and pneumonia may be found. The heart muscle is very rarely involved.

Symptoms.—The clinical history of trichinosis may be divided into two periods, the *first* embracing the disturbances occasioned by the intestinal trichinæ, the second those accompanying the general dissemination of the larvæ in the muscles. The severity of each stage varies with the number of parasites present. The gastrointestinal disturbances generally begin on the second or third day after the ingestion of trichinous flesh. They are nausea, vomiting, colic, and diarrhea. They may be so mild as to attract little attention or so severe as to simulate cholera morbus. Obstinate constipation often follows the primary diarrhea. Accompanying these symptoms there is always a sense of extreme weariness like that which follows unusual muscular exercise. All these manifestations usually disappear within a few days.

Second Period.—Sometime within the first six weeks, almost always between the seventh and tenth day, the symptoms of invasion begin. They are sometimes initiated with a chill and rise of temperature. As the myositis is developed, the muscles become swollen, stiff, hard as rubber, sensitive to pressure and motion, sometimes painful at rest. The flexor muscles are usually affected. Flexures often occur which bend the elbows and knees at an acute angle. The involvement of the diaphragm and intercostal muscles causes urgent dyspnea; and when the muscles of mastication and those of the larynx are invaded, mastication and deglutition become difficult and painful, and hoarseness is usually produced.

The *fever* is variable. The temperature may remain normal even when the muscles are extensively invaded; it may reach only 102° F. (39.0° C.) or it may rise to 104° or 106° F. (40.0° to 41.0° C.). It is then usually intermittent or remittent in its course, which ordinarily lasts from four to seven weeks. In mild cases, especially in children, it may be of shorter duration, and the muscular pains and flexures may also abate. In extreme cases they may last for two or three months. Profuse sweating occurs during the febrile stage, and sudamina often develop. Tingling and itching of the skin and such other eruptions as urticaria, acne, herpes, or furunculosis are not uncommon. In protracted cases the tongue becomes dry, as in typhoid fever.

Edema is one of the most characteristic symptoms after the seventh day. It appears in the face, especially the eyelids, and lasts, as a rule, from two to five days. It may reappear. The extremities become edematous when the muscular swelling is at its height, sometimes earlier. It does not always subside with the swelling. Ascites may develop.

The blood-count usually shows marked leucocytosis, often reaching 30,000, and about half the leucocytes in many cases are eosinophiles, but this feature is probably not so nearly universal as at first supposed.

Persistent insomnia occurs in severe cases. Headache is common, but delirium is rare. The tendon reflexes are often lost, and tremors are sometimes noticed. Mydriasis has been observed. The patient becomes markedly anemic and emaciated. Bronchitis is common, and in fatal cases pneumonia and pleurisy are sometimes found. Polyuria

occurs in some instances, while in others the urine becomes scant, of deep red color, containing albumin and sometimes casts and red blood-cells.

Diagnosis.—"The disease should always be suspected when a large birthday party, or Fest, among Germans is followed by cases of apparent typhoid fever" (Osler). The early nausea and vomiting are not characteristic, but if they have followed the eating of raw pork they should arouse suspicion. The diagnosis can often be confirmed by an examination of a remnant of the meat. Trichinæ can often be found in the stools. After the seventh day, the edema of the face, the dyspnea, the swollen, hard, tender, contracted muscles leave little doubt as to the character of the disease. A small fragment of muscle may be removed under cocain anesthesia with a harpoon devised for the purpose, or through a small incision. The parasites are most numerous near the tendinous portion of the muscle.

Typhoid fever is excluded by the painfulness of the condition, and more particularly by the leucocytosis.

Acute rheumatism affects the joints, while the swelling and soreness of trichinosis are confined to the muscles.

Ptomain poisoning produces gastrointestinal disturbance like that of trichinosis, but all the symptoms develop earlier and with greater violence, as a rule. The skin is dry, the muscles are not swollen or flexed, and edema is absent.

Cholera morbus is usually of longer duration and more violent than the diarrhea of trichinosis, and, although it is accompanied with prostration, there is not the sense of muscular fatigue.

Pseudotrichinosis.—A considerable number of reports have been published of the finding of other nematodes closely resembling the trichina, in man, lower animals, and vegetables. The differentiation can usually be made by observing the spiral-like striations and the group of large cells about the middle of the intestine of the worm.

Prognosis.—All depends upon the severity of the attack and the number of the parasites. In some outbreaks the mortality has not exceeded 2 per cent, in others it has reached 30 per cent.

Prophylaxis.—Care should be exercised in the feeding of swine. They should not be given the refuse of slaughter-houses, and rats should be excluded from their pens. The curing and smoking of the meat cannot be relied upon. Only thorough cooking, by which all parts of the meat are raised to the boiling point, will prevent the infection. Systematic inspection of meat is advisable and desirable from an esthetic point of view, but it cannot take the place of cooking. Pork and sausage should never be eaten raw.

Treatment.—When the character of the gastrointestinal disturbance is recognized early, a promptly acting purge will often remove all danger. All the anthelmintic preparations have been recommended, especially santonin, male fern, thymol, and turpentine. Sodium sulphocarbolate or salicylic acid in frequent doses in keratin capsules may be employed. Glycerin in large doses is always mentioned, but it is inferior to the other remedies. A free action of the bowels should be maintained until microscopic examination of the dejections no longer reveals the parasites. After the invasion of the muscles has begun, the treatment is

purely symptomatic. The pain and soreness of the muscles may be mitigated by the application of ice-bags or hot water. Sleep should be secured with remedies that do not constipate, as trional in doses of gr. xv to xxx (1.0—2.0). Tonics may be required later to overcome the anemia and emaciation, and passive motion or massage to restore the muscles.

ANKYLOSTOMIASIS.

DOCHMIASIS, TUNNEL-ANEMIA, BRICKMAKER'S, MINER'S, OR MOUNTAIN ANEMIA, EGYPTIAN CHLOROSIS.

This disease is produced by a parasite with many aliases, the ankylostomum duodinale; dochmius, strongylus, or uncinaria duodinalis, etc. It is a nearly cylindrical worm, from ¼ to ¾ inch (6 to 18 mm.) long, the male much smaller than the female. It is yellow or gray, and becomes red when it is filled with blood. The head is bent backward. The mouth is provided with a row of hooks by which it attaches itself to the intestine. The male has a large bursa copulatrix at its hinder extremity. The upper parts of the small intestine, especially the duodenum and jejunum, are its habitat. The eggs are oval and measure about 60μ by 35μ. These mature outside of the body under favorable conditions of temperature, and the larvæ become encysted. Thus they reach the drinking-water or food of man, and the cycle is completed. Some investigators assert that the larvæ can pass through the integument.

Symptoms.—These vary with the stage of the disease, the age, sex, and constitution of the patient. In the beginning there is often only such gastrointestinal disturbance as diarrhea and colic, but profound anemia is the characteristic symptom. It may develop so rapidly as to cause dyspnea and edema. The skin is yellowish or perhaps blanched as though from hemorrhage, and there are great weakness and prostration, with rapid pulse and palpitation. Moderate fever may be present. Emaciation is often absent except in advanced cases. All these symptoms are the result of the abstraction of blood by the parasite, although the additional influence of a toxic substance secreted by it has been suggested. Hypertrophy and dilatation of the heart are found in many cases, the apex being displaced downward and to the left.

Diagnosis.—An early diagnosis is important. It is to be based upon the rapid development of the profound anemia accompanied by gastrointestinal disorders, and confirmed by the discovery of the ova in the stools, especially in tropical countries where the disease is most prevalent. Delamere has called attention to a peculiar mark on the tongue which looks as if the patient had just wiped a penful of blue-black ink on it. He found it an early and constant sign.

Treatment.—Spontaneous recovery sometimes occurs owing to the death of the parasites. Thymol is a specific. It should be given in two doses of 3 ss to 3 ij (2.0 to 7.5) two hours apart, preferably in the morning, the first dose preceded and the second followed by a purge of

magnesia or castor oil. The treatment may be repeated in a week if necessary. During treatment the patient should remain in bed, on a milk diet, and no alcoholic beverages should be allowed (Guiteras).

FILARIASIS.

Definition.—A diseased condition caused by the filaria sanguinis hominis and manifested in many quite different lesions. The disease occurs chiefly in tropical and subtropical countries, but is occasionally encountered in the Southern States. It is very prevalent in some of the West India islands. Its distribution to different regions of the globe is shown in the accompanying map (Plate IX).

The Parasite.—No less than six species of the filaria have been described, but only three of these have been sufficiently investigated to receive general recognition.

(1) *Filaria Sanguinis Hominis Nocturna* (Filaria Bancrofti).—This is the form commonly present. The mature worm is about as thick as a human hair; the female is from 3 to 4 inches (8—10 cm.) in length, the male is a little more than half as long, and its tail has a spiral twist. The embryos are inclosed in a sheath and measure from 270 to 340μ in length and from 7 to 11μ in width. They are found in the blood at night, or whenever the individual sleeps. The intermediate host of this species and probably of the others is the mosquito. They have been found in several species of the culex and in one anopheles.

(2) *Filaria Sanguinis Hominis Diurna.*—The identity of this species rests upon the observation of it by Manson in the blood of three negroes from the Congo region. They were found in the blood only during the daytime. They are further distinguished by a granular body. Manson regards the filaria loa as probably the mature form.

(3) *Filaria Perstans.*—Only the embryos of this form are known, and they are found in the blood both day and night. They are smaller, more active than the former, and have no sheath. A mature worm has been described by Daniels as the supposed parent. Manson attributes to this form the sleeping sickness of the Congo country and possibly also craw-craw, a cutaneous disease of the west coast of Africa.

The filaria Bancrofti has been most thoroughly studied. The mature parasite has been found in the human body only about eleven times. These were found for the most part in the lymph-channels, adipose tissue, or in abscesses induced by their presence. The female gives birth to a large number of embryos which pass with the lymph into the blood. When the individual is active, they are believed to remain quiet in the blood-vessels, especially within the lungs. When the individual rests, they wander into the peripheral circulation. Hence they are found in increasing numbers from evening to morning, and disappear entirely by 9 a. m. Their life cycle is completed, as previously stated, through the medium of the mosquito. They are extracted with the blood of their host into the body of the insect. Here they undergo partial development. It is assumed that after the death of the mosquito they are liberated in the water, where they become mature and again enter the body of man through the drinking-water. From the intestine they

CHARTS SHOWING DISTRIBUTION OF GUINEA WORM AND FILARIA SANGUINIS HOMINIS.

(*Twentieth Century Practice*)

find their way into the lymph-channels. They may be present in the body indefinitely without producing symptoms, and have been repeatedly found in the blood of animals in which they produced no recognizable disturbance. When, however, the mature parasite or the ova obstruct a lymph-vessel, disturbances are produced peculiar to the location of the obstruction.

Symptoms.—The principal manifestations are the following:

(*a*) *Hematochyluria.*—This feature is manifested by an occasional passage of a milky, bloody, or chylous fluid which generally deposits a reddish clot. The urine may continue normal in quantity or it may be increased. The general health is not impaired. The condition is intermittent and may persist for many years, intervals of several weeks often intervening between the chylous discharges. Microscopical examination of the chylous urine reveals large quantities of granular fat, usually red blood-corpuscles, and sometimes the embryos. It is well to remember that the anguillula aceti has been mistaken for the filaria in urine not chylous in character, after it had been placed in a bottle previously used to contain vinegar. The greatest inconvenience is usually due to the obstruction caused by the formation of clots within the bladder. A nonparasitic form of chyluria is occasionally met with.

(*b*) *Lymph-Scrotum.*—The scrotum becomes distended with the enlarged lymph-vessels which may usually be traced a considerable distance upward. The scrotum may be much thickened. The chylous fluid flows freely from punctures, and the filariæ are found in it. Lymph-vulva was observed in one instance by Bälz.

(*c*) *Other Diseases.*—The filariæ have been found also in other conditions, among which are elephantiasis arabum, chylous ascites, filarial hemoptysis, and chylous diarrhea.

Febrile attacks are frequently observed during the course of many of these affections.

Prophylaxis.—In countries where these diseases are prevalent, all drinking-water should be filtered or boiled.

Treatment.—There is no known means of destroying either the mature worm or the embryos. The death of the parasite may or may not be followed by relief of the obstruction. The remedies which have proved most beneficial are gallic acid, quinin, and the barks of various tropical trees. When chyluria is present, the patient should abstain from fatty food. In most cases, however, little can be done beyond the treatment of the local condition.

DRACHONTIASIS.

GUINEA-WORM DISEASE.

This disease, due to the filaria medinensis, dracunculus Persarum, or Guinea-worm, is most prevalent in the East Indies and in Africa, but it is occasionally encountered in the United States, for the most part among foreigners (Plate IX.). The living male has been found only once, by Charles, in the mesentery. The female is cylindrical, 20 to 40 inches (50—

100 cm.) in length, ¾ inch (2 cm.) in diameter, and of a whitish color. Only one parasite is usually present. The avenue of entrance is probably in all cases the alimentary canal, and the medium drinking-water. It is probable also that the male accompanies the female, and with her passes out of the intestine into the mesentery, and that, after performing his sexual function, he dies. Charles found calcified remains of males in the mesentery. The female passes on through the tissues until she reaches the subcutaneous connective tissue. Here she remains quiescent for an indefinite time, coiled up like a tangled cord beneath the skin. As the time for parturition approaches, at least with an evident purpose of liberating her embryos, she usually travels downward until she reaches the ankle or foot; then thrusts her head through the overlying skin. The opening is occasionally made in the upper parts of the body. A vesicle is formed by the elevation of the epidermis over the head. This finally bursts and a small ulcer is formed, at the bottom of which the protruding head may be seen. In a short time the uterus ruptures, for there is no other way of liberating the young, and these are discharged in a milky fluid. Those which find their way into water find an intermediate host in the cyclops, a small crustacean. The parent worm soon leaves the body after the discharge of her embryos.

Treatment.—Prophylaxis consists in preventing the entrance of the embryos by filtration or boiling of drinking-water. The worm should not be molested when it first makes its appearance beneath the skin, since it is then seeking an avenue of escape from the body. It is customary in the tropics to roll the body of the worm around a smooth stick as it protrudes, winding a little more each day, but serious inflammation follows the rupture of the body. The worm has been excised entire with success, but a better method for causing its destruction is the injection of mercuric chlorid, 1 : 1000. Asafetida in large doses has been vaunted as a specific.

OTHER NEMATODES.

The following nematodes have been found once or more within the human body: Filaria labialis, F. bronchialis, F. hominis oris, F. lentis, their names signifying their location; the filaria immitis, causing hematuria, found also in the portal vein, and its ova in the walls of the ureter and bladder; and the filaria loa, found beneath the conjunctiva.

Eustrongylus Gigas (Dioctophyme Gigas).—This is a large worm, the female measuring 20 to 40 inches (50 to 100 cm.), the male about one-third as long. It is found especially in the kidneys of the lower animals, and only rarely in man. Hematuria is produced and sometimes the entire kidney is destroyed.

Strongylus paradoxus, once found in the dejecta of a man, is not infrequent in the respiratory passages of the hog.

Strongyloides intestinalis are occasionally found in the stools in the diarrhea of Cochin-China and other hot countries. The worm was formerly known as the anguillula stercoralis, anguillula intestinalis, and rhabdonema intestinale.

Acanthocephala.—The echinorhynchus gigas, or gigantorhynchus, has been found in the intestine. It is not uncommon in the hog. Its intermediate host is the grub of the cock-chafer, our June-bug.

DISEASES CAUSED BY THE CESTODES.

TENIA, TAPEWORMS, CYSTICERCI, HYDATID CYSTS.

The following general description of the family of Tenioidea accords with that of R. Hertwig: Tapeworm with scolex (head) and detachable segments (proglottides); on the scolex four sucking disks, often a rostellum with or without a row of hooklets; in the proglottides an albumin gland; the uterus ends in a blind extremity, the porus genitalis or common opening of the vas deferens and vagina, on one side of the segments. These rarely open separately on opposite sides, as in tenia canina. The embryonic stages are hydatids (measles, cysticerci) or cysticercoidi. The tenia are hermaphrodites. If a tenia ovum enters the intestine of man or an animal, the embryo migrates through the blood-vessels or lymph-vessels into the tissues, and there develops into a hydatid. The ovum of tenia solium produces a cysticercus in man or the pig; that of the tenia saginata, a measle in the flesh of cattle. Ingested cysticerci, on the other hand, develop into mature tenia in the intestine. The tenia and cysticercus require different hosts, except in the case of the tenia solium, both stages of which may occur in man. Infection may arise from the ingestion of contaminated vegetables, salads, or cresses.

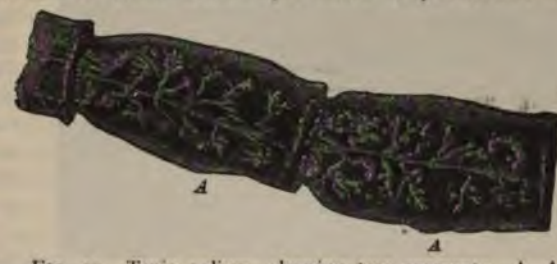


FIG. 21.—Tenia solium, showing two segments. A, A, pores.

Tenia Solium (the Pork Tapeworm).—This form of tapeworm is met with much less frequently in this country than in some parts of Europe and Asia. The meat of the hog is the usual source of infection. The mature worm is from 6 to 12 feet (2—3.5 m.) long. It reaches maturity in from three to three and a half months, after which the segments begin to appear in the stools of the host. The head is round and smaller than the head of a pin; it has four sucking disks and is armed with a double row of hooklets. Hence it receives the name tenia armata. The neck is slender, almost threadlike. The mature proglottides are about 1 cm. long and 6 to 8 mm. broad. The genital pore may be on either side. The uterus consists of a central stem which gives off at right angles from five to seven branches on each side (Fig. 21). The ova are round, brownish, and have a thick shell. When the ova enter the alimentary canal of man, the shell is digested and the embryos, each of which has six hooklets, are liberated. They most frequently pass into the liver, muscles, brain, or eye, and there develop into larvæ, or cysticerci.

Tenia Saginata.—This species is larger than the tenia solium. It is derived from beef and is the form almost exclusively encountered in this country. It is often 15 or 20 feet (4—6 m.) long. The head is larger than that of the tenia solium, square-shaped, and has four suckers, but no hooklets. It is often pigmented. The segments are also larger, and the terminal proglottides often measure an inch (2.5 cm.) in length. The median stem of the uterus resembles a central canal, from which resemblance the worm has been called tenia mediocanellata. From 15 to 35 lateral branches are given off from it, usually at an acute angle (Fig. 22). The ova are larger and the shell thicker than those of the solium. They pass out of the body, and when ingested by cattle they develop into cysticerci within the flesh. This is probably the universal source from which man becomes infected. They have been found within the human body in only two or three instances.

Bothriocephalus Latus (Tenia Lata).—This is an extremely large cestode often attaining the length of 25 to 30 feet (9 m.). The head is almond-shaped and about 2.5 mm. long. Instead of sucking disks, it has two long, deep grooves. It is unarmed. The larvæ are found in the muscles of fish, especially in the Baltic Sea, in parts of Switzerland, upper Italy, and Japan. It probably does not exist naturally in our country, but it is met with from time to time in Russian immigrants.

FIG. 22.—The uterus in a segment of tenia saginata. X3.

Tenia cucumerina (T. elliptica, dipylidium canina) is a small tapeworm often found in great numbers in the dog, less frequently in the cat, occasionally in children. The flea and louse harbor the larva. As many as 50 cysticerci have been found in a single flea.

Tenia confusa is a tapeworm about 16 feet (5 m.) long, two specimens of which were secured by Ward at Lincoln, Neb. The head is small and has, in addition to the four suckers, six or seven rows of hooklets. The terminal segments are larger than those of the tenia saginata.

Tenia nana (T. Madagascarensis, Hymenolepsis nana) is the smallest tapeworm found in man. It occurs especially in Egypt, Italy, Sicily, and Siam.

Tenia Diminuta (T. Flavopunctata, Hymenolepsis Diminuta).—This is a common parasite of rats and mice, but is seldom found in man. Its larva is found in caterpillars and beetles.

Symptoms of Tapeworm.—The tenia occurs in persons of any age, from early infancy to advanced senility. In many cases no disturbance is produced. On the other hand, much mental distress, amounting to hysteria or hypochondriasis, may be occasioned by the discovery of the

condition in a person of nervous temperament or fastidious sensibility. In other respects much depends upon the physical constitution of the individual. A ravenous appetite is sometimes complained of, or there may be nausea, vomiting, diarrhea, and abdominal pain. Vertigo, chorea, convulsions, and epilepsy have been attributed to the presence of the worm. Profound, even fatal, anemia is produced by the bothriocephalus latus, and appreciable anemia sometimes attends the other species. In most cases, however, the first indication of the parasite is the discovery of the terminal proglottides in the stools. Those of the tenia saginata sometimes escape from the rectum and attract attention by their wriggling movement.

Diagnosis.—The passage of segments leaves no doubt as to the diagnosis. The ova may also be found in the dejecta. The species of the worm can easily be determined by an examination of the segments or head.

Prophylaxis.—The surest means of prevention is the avoidance of raw or insufficiently cooked meat and unclean vegetables. Whenever beef has a raw appearance and when blood flows from the cut surface, it should be regarded as underdone. Individuals harboring tenia should be careful not to contaminate water or the soil with the proglottides. These should in all cases be burned. Care should be exercised also not to rupture the segments upon the person. The worm should not be handled; this is especially true of the tenia solium. Inspection of meat is of value, but inferior to thorough cooking, as a means of prophylaxis. The measles of beef are most readily found in the muscles of the jaw; those of the hog in the tongue, muscles of mastication, the diaphragm, neck, and shoulder. The latter are more easily recognized than those of beef on account of their more opaque, whitish color.

Treatment.—The treatment is the same for all species; some writers prefer one method, some another, for each. The treatment consists first in bringing the alimentary canal into a condition which favors the action of the chosen remedy; second, in the administration of a drug which is capable of killing or benumbing the worm; and third, in the administration of a purge to remove it. The treatment should be given immediately after the discovery of the parasite, unless so large a portion of it has recently been discharged as to render its death probable.

Preparatory Treatment.—For two days the patient should be placed on a diet which requires only stomach digestion and leaves little residue; milk, soup, beef, very little bread, and no vegetables compose a good diet; the exclusive use of milk is even better. A mild laxative should be given if the bowels are sluggish, or an enema may be administered the night before the remedy is to be taken.

Medicinal Treatment.—The teniacide should be administered in the morning, fasting, after or with a cup of coffee, but without food. The male fern is one of the most effective remedies. It may be given in the form of the ethereal extract or the oleo-resin in dose of ʒ ij (8.0), in capsules which may be coated with keratin. Unless the remedy produces active purging, a saline cathartic, a half-ounce of magnesium sulphate, or the citrate must be given. Castor oil should not be used, as it favors the absorption and poisonous effect of the drug.

Pomegranate root is an efficient remedy. It may be administered in the form of a decoction made by boiling oz. 2 (64.0) in 1½ pints of distilled water down to a pint. The entire quantity is given in three portions about a half-hour apart. It is objectionable on account of its nauseating taste. Vomiting should be avoided, as there is danger of carrying the segments up into the stomach, with a liability to cysticercus infection. The tannate of the active principle, pelleterin, should therefore be employed, since it is tasteless. It is administered in the dose of 7 to 10 gr. (40.0—65.0) dissolved in an ounce of water which may be concealed in a glass of lemonade. The purge is given an hour afterward.

Pumpkin seeds are often effective. Three or four ounces are bruised, the outer rind being removed, and mascerated for twelve hours. The entire quantity is taken, and a purge is given an hour later.

Among the other remedies recommended are cusso, kamala, the black oxid of copper, naphthalin, and thymol.

After the worm has been discharged, it should be carefully examined for the head, remembering that this is the smallest part, for unless this has been removed, the parasite will again attain full growth.

VISCERAL DISEASES.

Cysticercus Cellulosa.—When the ripe ova of the tenia solium enter the stomach, the embryos escape and soon burrow into the blood-vessels and lymphatics, whence they are carried into the tissues. The cysticercus is a small vesicle or cyst, not unlike a miniature bottle. Racemose forms are occasionally seen. Self-infection has repeatedly occurred, one individual harboring both the tenia and the cysticercus.

Symptoms.—The manifestations depend upon the number of cysticerci and their location. The most frequent places of lodgment are the muscles and subcutaneous tissue, the brain and cord, and the eye; less frequently the lungs, liver, bones, and lymph-glands. When the cysticerci are not numerous, or if they do not affect a vital or sensitive part, few or no symptoms are produced. In some cases, however, owing to excessive involvement of the muscles, pain, soreness, and stiffness are complained of in the beginning. Here and elsewhere, however, a remarkable tolerance is often begotten.

Subcutaneous Cysts.—These appear in the form of one or many, up to 1,000, round or oval, firm, movable tumors of the size of a hazelnut. They are situated on the trunk and extremities, rarely on the face. From other small *cutaneous tumors* they are distinguished chiefly by their numbers, tenseness, and mobility. *Sebaceous cysts* are immovable nodules in the skin; *lypomata* are larger and softer; *gummata* are flat, doughy, and tender.

Cerebrospinal Cysticerci.—Among the most constant symptoms are cephalalgia, epileptiform or cumulative convulsions, vertigo and psychical disturbances, with depression and confusion as prominent features. Paralyses are rare, since the cysts are located in the cortex and meninges; they may develop, however, at a late period. Sudden death has occurred in a few instances. In the fourth and lateral ventricles the cysts sometimes attain considerable size. When they press upon the

floor of the fourth ventricle, symptoms of diabetes and anomalous nervous disturbances are produced. Many vesicles may exist for an indefinite time in the silent region without manifestations.

Ocular Cysticerci.—The vesicles may occupy the anterior chamber or the vitreous, or they may lie beneath the retina or conjunctiva. The symptoms are those of irritation and interference with vision. Ophthalmoscopic examination generally reveals the parasite when it is located within the chambers.

Cysticercus of the heart is rare and is attended with no symptoms. The same is true of involvement of the liver and lymph-glands. In the bones destructive lesions are sometimes produced.

The diagnosis is usually difficult or impossible, except when the cysticerci are found in the eye or in the subcutaneous tissues, from which one can be removed for examination.

ECHINOCOCCUS DISEASE.

HYDATID CYST.

The Parasite.—The mature tenia echinococcus is found in the dog, especially in mastiffs and Newfoundlands; in cattle and other animals, particularly sheep. The hydatid echinococcus is the larva, designated the echinococcus polymorphus. The tenia is one of the smallest cestodes, ¼-inch (4 to 5 mm.) in length, having but three or four segments, of which only the terminal becomes mature; it then measures about 2 mm. in length by 0.6 mm. in breadth. The head supports a rostellum bearing from 28 to 52 hooklets in two rows; it has also the usual four sucking disks. The terminal segment becomes filled with ova, estimated at 5,000, and is then detached and discharged from the alimentary canal of the host. Through the contamination of vegetables or drinking-water, sometimes through direct contact with the dog, it reaches the human intestine. Here the embryo, which is armed with six hooklets, is set free and at once bores through the intestine-wall until it reaches the lymphatics or blood-vessels. When it reaches the portal vein it is carried to the liver, one of its most frequent lodging-places. When it enters the general circulation, it is carried to the lungs, or, passing on, may reach the brain, spleen, kidneys, muscles, or other parts.

The Hydatid Cyst.—After reaching its destination, the embryo loses its six hooklets and acquires a vesicular form, through the growth from its caudal extremity of a serous membrane which ultimately envelops it and becomes distended with a clear fluid. It thus becomes the echinococcus cyst. Its growth is slow; years sometimes elapse before it becomes large enough to produce appreciable disturbance. As usually found, the size varies from that of a pea to that of the human head, rarely larger. The cyst consists of a delicate substance, chitin, and is separable into two layers. The outer of these is laminated; the inner, known as the parenchymatous or germinal layer, is granular. The presence of the cyst occasions a reaction on the part of the tissues which results in the formation of a firm fibrous wall about it. After a variable time, usually from two to five months, little mounds appear upon the surface of the germinal layer, each having at its apex a small

depression which later becomes a cavity. These cavities enlarge and become secondary or daughter-cysts. This process of budding may take place also in the daughter-cysts and give rise to granddaughter-cysts. Either generation may develop either endogenously or exogenously. The former is the more common method of growth in man. The number of cysts formed varies from a few to several thousand. From the germinal surface of the daughter-cysts, sometimes from the mother-cyst, scolices, the heads of embryonic teniad, evelop. These appear as conical projections, each having on its free extremity a rostellum armed with a double row of hooklets and four suckers. The other extremity becomes constricted into a narrow pedicle, which later divides, liberating the scolex, thenceforth to float freely about in the interior of the capsule. Each of these scolices, from one to nine or more in each capsule, is capable of development into a mature tapeworm within the intestine of the dog. It occasionally happens that daughter-cysts are formed within the scolex. In another variety of cyst the daughter-cysts are sterile; they are then known as acephalocysts.

The Multilocular Echinococcus.—This form of cyst is encountered once in about 180 cases of the disease. The sac, sometimes of very large size, is surrounded by an exceedingly dense fibrous capsule firmly united to the surrounding tissue. It is subdivided into numerous small cavities and is filled with a thick, gelatinous, or colloid material suggestive of cancer. It is probably not due to the tenia echinococcus, but a form of the echinococcus in which there is no intermediate host. Recent investigation has shown that after the embryo has reached the liver by the same route as the other parasite pursues, a multilocular chitinous structure is formed which resembles the mature proglottis of a tapeworm. From the granular protoplasm which lines the cyst-wall within and without, there are formed not only scolices, but young parasites and ovoid embryos. If these embryos gain access to the blood-vessels or bronchioles they may develop new cysts. The new cysts remain sterile. Metastasis may also occur, the embryos being carried to the lymph-glands, lung, brain, or elsewhere.

The *fluid* of the echinococcus cyst is limpid, usually clear, of neutral reaction, and has a specific gravity of from 1.006 to 1.015. Sometimes, however, it has a pale green tint and is slightly alkaline from admixture of bile, opalescent from the presence of fatty matter or other débris; or pale red blood is present in it. Chemical analysis shows the presence of from 0.50 to 0.75 per cent of sodium chlorid and small quantities of the earthy compounds of succinic acid, inosit, and grape-sugar. Albumin is not normally present. Urea, creatin, hematoidin, and substances resembling toxalbumins and ptomains have been found in it. Microscopic examination of it usually reveals scolices, hooklets, and occasionally fragments of the chitinous membrane.

The echinococcus sometimes dies, spontaneously or as a result of some accident. Its growth is then arrested and its contents undergo retrograde changes converting them into a pasty mass which may later undergo calcification, or it may be partially absorbed. Suppuration sometimes occurs and the rupture of the abscess may lead to serious consequences.

Etiology.—Echinococcus disease is met with most frequently in Iceland, where the people live in intimate association with their dogs, and among the shepherds of Australia. No part of the world is exempt, but the disease is rare in the United States. Lyon was able to collect only 241 cases in America up to July, 1901. The disease affects the sexes alike and at all ages after infancy. Except in Iceland, perhaps, the disease is most frequently contracted through eating infested meat. Scolex-bearing cysts have been found in the flesh of the ox, sheep, hog, goat, deer, horse, and squirrel. Autoinfection is possible, although the tenia has seldom been known to enter the human intestine.

Symptoms.—Echinococci frequently exist for years without occasioning symptoms. The greater number have been found at autopsy. In most instances the first symptom to attract attention is the formation of a tumor; the direction of greatest protrusion depends upon its location. In fully half the cases the hydatid is located in the liver, next most frequently in the lung or pleura, then in the kidney, bladder or genitals, brain, spinal canal, bone, heart, blood-vessels, or other organs.

Hydatid of the Liver.—The first indication is generally that of enlargement of the organ or the protrusion of a tumor into the hypogastrium or upward into the thorax, corresponding to the location of the cyst. A large tumor pushing upward impedes respiration and sometimes displaces the heart to the left. Pushing downward, it may extend to the pelvis. When in the left lobe it may displace the spleen.

If accessible to palpation, the cyst gives the impression of a smooth, globular tumor, sometimes fluctuating, usually irregular in outline. If it be of large size and contain many daughter-vesicles and the abdominal wall be thin, we may, by grasping the tumor and exerting moderate compression with one hand and striking a quick, rather forcible blow upon it with the other, elicit a peculiar vibratory sensation which has been compared to the trembling of a bowlful of jelly, and known as the hydatid purring of Briançon. The sign is absent in about half the cases, and is not always trustworthy. Many cases are attended with pressing, undefined, or sharp pain. Pressure symptoms attend all large cysts. Prominent among these are dyspnea, cough, cardiac palpitation, indigestion, vomiting, constipation, and later ascites and occasionally varicose veins. Jaundice is not often present, unless late in the disease. There is usually no fever unless suppuration has occurred, and the nutrition of the patient is generally maintained.

Rupture of the cyst occasions a new train of symptoms, varied with the direction in which it occurs. Spontaneous recovery has followed rupture through the external abdominal wall, or into the intestine, bile-duct, ureter, or vagina, but rupture into the pericardium or inferior vena cava is necessarily fatal. The accident is usually attended with sudden, sharp pain and a variable degree of shock. Urticaria appears on the skin after rupture internally.

The *multilocular echinococcus* is generally firm, seldom fluctuates, and is often sensitive. It is commonly accompanied with enlargement of the spleen and ascites. Gastric and intestinal disturbance, hemorrhage, and jaundice are also more frequent in connection with it.

Diagnosis.—The disease is to be differentiated from cancer, amyloid disease, syphilis, cirrhosis, and abscess of the liver and occasionally from

hydrothorax, pyothorax, cystic disease of the retroperitoneal glands, ovarian cyst, enlargement of the gall-bladder, and aneurism of the aorta. In its early history the differentiation may be quite difficult. A tumor of slow growth, elastic, fluctuating, and giving the hydatid fremitus is generally an echinococcus. The diagnosis is established if the fluid withdrawn through a small aspirator needle is found to be characteristic.

Cancer is more painful, grows more rapidly, is firmer, and produces greater emaciation and cachexia.

The *amyloid liver* is hard, the edge sharp and smooth; the skin is waxy, and there is a history of suppuration or syphilis.

Syphilis must be excluded by the history and evidences of previous lesions.

Cirrhosis reduces the size of the liver, roughens its surface, and follows chronic alcoholism. The ascites is usually greater.

Hepatic abscess is attended with fever and other evidences of sepsis. The needle withdraws pus; but suppuration may occur in the hydatid cyst. The differentiation is then not important.

Hydrothorax and *pyothorax* are recognized by the level line of dullness, changing with a change of position. In echinococcus the highest margin is usually in the axillary line.

Cystic enlargement of the retroperitoneal lymph-glands is rare and may not be excluded without exploratory incision.

Distention of the gall-bladder is recognized by the character of the fluid withdrawn.

Aortic aneurism is recognized by its location as well as by pulsation and thrill. Pulsation may be transmitted, however, to a hydatid cyst.

Echinococcus of the Lung and Pleura.—The cysts may exist for a long time in the lungs without occasioning symptoms. Later they lead to inflammation, occasionally to gangrene with the production of cavities. When communication has been established with a bronchus, small cysts or fragments of membrane and hooklets may be found in the expectoration. Hemorrhage is not infrequent, and rupture into the pleural cavity may occur. The diagnosis is rarely made during life.

When the cysts are in the pleura, the symptoms are those of hydrothorax; but the outline of dullness is often irregular and does not change when the patient lies down. Spontaneous evacuation may take place through the chest-wall.

Echinococcus of the Kidneys.—Few symptoms are observed in many cases until the cyst ruptures. The small cysts cause pain in their passage, and their presence in the urine is diagnostic of the disease. Without rupture, the character of the condition can be determined only by examination of the fluid obtained through aspiration.

Echinococcus of the brain is fortunately a rare affection. The symptoms are those of brain tumor and depend largely upon location. The character of the tumor can rarely be determined during life.

Prognosis.—This depends upon the size and location of the cyst. Unless terminated by the death of the echinococcus or by operation, the disease is ultimately fatal.

The prophylaxis is the same as that of the other diseases caused by the cestodes.

Treatment.—The treatment is purely surgical. The withdrawal of a small quantity of fluid under aseptic precautions may result in the death of the parasite, but incision is generally necessary. This should not be delayed. The method is detailed in the textbooks on surgery.

PARASITIC ARACHNIDS.

1. **Linguatilina.**—(*a*) Linguatula rhinaria (Pentastoma tenioides) is a lancet-shaped arachnid, the male about 1 inch (2.5 cm.), the female 3 inches (7.5 cm.) in length, with a tapering body marked by numerous rings. It infests the nostrils and frontal sinus of the dog, sometimes those of the horse, rarely attacking man. The larva, known as the linguatula serrata or pentastomum denticulatum, invades the internal organs, especially the liver of animals, sometimes also of man.

(*b*) Porocephalus constrictus (Pentastomum constrictum) is rare in this country. It is about ½ inch (12.5 mm.) in length and is found in the liver and lungs.

2. **Sarcoptes.**—The most important of this class is the acarus scabei, or itch mite, the cause of the disease commonly called the itch. The male measures 0.23 x 0.19 mm. and the female is almost twice as large. It is much more common in Europe than in America. It lives in burrows beneath the epidermis, but the male is seldom found. The itch mite is readily destroyed by giving the affected region a thorough scrubbing with good soap, twice daily, then applying a mild sulphur ointment.

3. **Demodex (Acarus) Folliculorum.**—This parasite, known also as the comedo mite, is about 0.4 mm. in length and occasionally occupies the sebaceous follicles, especially those of the face, and may produce acne.

4. Another form of acarus, normally a parasite of plants, produces the adobe itch of tropical countries.

5. **Leptus autumnalis,** or harvest bug, occasionally becomes parasitic by attaching itself to the skin of the legs. Much irritation may be produced by its sharp proboscis and the hooklets of its legs. It is destroyed by mercurial or sulphur ointment.

6. **Ixodes.**—Several species of the tick occasionally become parasitic to man, notably the Ixodes albipictus, ricinis, and bovis, also known as Dermacantor Americanus. Some species are also regarded as carriers of bacteria.

LARGER PARASITIC INSECTS.

1. **Pediculosis.**—Pediculi, or lice, are of three species, each of which inhabits different regions of the body. The condition is known as pediculosis or phthiriasis.

(*a*) *Pediculus capitis,* an insect of grayish, white, or brownish color, with six legs under the front part of the body. The male is about 1 to 1.5 mm. long; the oviparous female, about twice as large, produces about 80 eggs in a week. These "nits" can be seen attached to the sides of the hairs. Their presence is indicated by the itching of the head. Dermatitis and eczema are sometimes produced; large crusts are often formed in filthy persons. The plica polonica, occasionally seen on the heads of recent immigrants from Poland, is of this character.

(*b*) *Pediculus vestamentorum* (P. corporis), the large body louse or gray-back, the companion of the hobo. It inhabits the clothing, and invades the body only for the purpose of drawing blood. As a result of the itching produced by its bites, the skin of the individual is usually streaked with the recent or old pigmented marks caused by scratching, especially in those regions where the clothing fits closely to the body.

(*c*) *Phthirius Pubis.*—The crab-louse is an ovoid insect which inhabits particularly the hair of the pubic region, but occasionally invades that of the breast, axillæ, beard, and eyebrows.

Treatment.—(1) The pediculus capitis can be quickly removed by saturating the hair with coal-oil or turpentine, scrubbing with a soft soap, then saturating it with vinegar, and finally rinsing with clear water. The oil destroys the pediculus, the vinegar ruptures the ova. One or two treatments usually suffice. (2) The pediculus vestamentorum requires thorough cleansing of the body and disinfection of the clothing in a disinfecting-oven for several hours. The itching is relieved by a lotion containing sodium bicarbonate and carbolic acid (2 per cent) applied after the bath, or by the application of carbolated vaselin. (3) For the crab-louse, thorough bathing and the application of the blue mercurial ointment or an ointment of the white precipitate is curative.

2. **Cimex lectularius** (bedbug), a small, flat, nearly round, reddish brown insect 3 to 4 mm. in length, may be recognized by its peculiarly offensive odor, due to the secretion of a gland. Its bite is exceedingly poisonous to some persons, causing intense hyperemia or urticaria.

3. **Pulex Irritans** (the Common Flea).—The flea is a small, nearly black insect, the male from 2 to 2.5 mm. and the female from 3 to 4 mm. in length. The bite produces circular, bright red spots. In some individuals a marked hyperemia or urticaria is produced. Tolerance seems to be acquired by those living in localities where fleas abound.

4. **Pulex penetrans** (sand-flea or jigger) is a common parasite of tropical countries, especially the West Indies and South America. It attacks especially the feet and ankles, penetrates the skin and produces burrows which frequently suppurate, causing pustules and often enlargement of the lymph-glands. Its removal is not difficult. Application of an essential oil to the feet prevents its invasion.

Other parasites are the Dermonyssus avium and gallinæ, bird and chicken lice; culicidæ, mosquitoes and gnats; estridæ, bot-flies; and muscidæ, common house-flies. Bees, wasps, ants, and spiders sometimes act as parasites and by their bites or stings cause great and painful swelling, sometimes with toxic effects. Caterpillars occasionally cause urticaria, apparently by the irritation of their bristles. The several species of flies named, and many others peculiar to certain regions, especially the tropics, are not only annoying by their bites, but their larvæ occasionally infest wounds or sinuses, and sometimes gain entrance to the ears or nostrils, or to the vagina after parturition. The condition is known as myiasis.

SECTION III.

Diseases of the Blood and Ductless Glands.

DISEASES OF THE BLOOD.

POLYCYTHEMIA.

The term polycythemia is applied to a relative increase of the number of the red blood-corpuscles, whether this be due in reality to an actual numerical increase, or to a decrease in the volume of the plasma. It is a normal condition of the blood in the newborn before nursing begins, the red cells often reaching 6,000,000 in the cubic millimeter. An increase of a million or more corpuscles rapidly ensues upon assuming residence in a high altitude, and it is believed to become a permanent condition until the individual returns to a lower altitude.

Polycythemia occurs also in cholera and other diarrheal diseases, sometimes in typhoid fever. Relative increase of the red corpuscles may be found in chronic valvular disease of the heart, with passive hyperemia, in endocarditis, after excessive sweating, poisoning by illuminating gas or phosphorus, and after cold baths or the application of alcohol and other drugs that cause contraction of the blood-vessels.

Polycythemia with Chronic Cyanosis.—This condition, as a clinical entity, was first brought prominently before the profession by Osler, in May and August, 1903, although it had been described a year before by Saundby and Russell, of England. The condition is one of general cyanosis, in which the only pathological lesion, in addition to slight enlargement of the spleen, is a polycythemia. The red corpuscles range from 7,000,000 to 12,000,000 in the cubic millimeter. The hemoglobin is correspondingly increased; the leucocytes are normal. Congenital heart lesions, emphysema, and other causes of cyanosis were carefully excluded, and there was no dyspnea in the cases observed by Osler. A trace of albumin was found in the urine, but not sufficient to indicate disease of the kidneys. Weakness, prostration, constipation, headache, and vertigo were the prominent symptoms. The blood was black and flowed sluggishly from the ear puncture. Similar cases were reported by Cabot, Shattuck, Stockton, and others.

ANEMIA.

Definition.—A reduction of the quantity of the blood as a whole or of one or more of its cellular or chemical constituents. The greatest reduction affects the red corpuscles. These may be greatly diminished in number, or their hemoglobin alone may be deficient. Anemia may be local or general. Local anemia is considered on page 11.

General anemia may be primary or secondary in origin. (1) *Primary*, essential, or cytogenic anemia arises as an independent disease. It embraces the two affections, pernicious anemia and chlorosis. (2) *Secondary anemia* results from such causes as hemorrhage, inanition, infectious toxemia, metallic or gaseous poisons, and autointoxications.

I. PRIMARY ANEMIA.

Pernicious Anemia (Idiopathic, or Progressive, Pernicious Anemia).—A fatal form of anemia, probably of infectious origin, showing an extreme reduction of the number of red blood-corpuscles, preponderance of megaloblasts, and other changes in the cellular elements, and various resultant changes in the organs and tissues generally.

Etiology.—The disease may affect either sex at any age, but middle-aged men are oftener attacked than women or children. The disease is more common in European countries than in our own, and especially in England and Switzerland. It sometimes develops during pregnancy or soon after its termination. A clear distinction has not always been made between the pure pernicious anemia, first described by Addison, and severe forms of secondary anemia due to the presence of intestinal parasites, as the bothriocephalus latus and the ankylostoma. Atrophy of the stomach has been repeatedly observed after death, and it has been regarded as a cause in some instances. In nearly all cases there is a history of more or less prolonged disturbance of gastric or intestinal digestion, preceding the recognition of the anemia, but the lesions have not usually been so severe as to be regarded as the sole cause of the blood-changes. What the exciting cause is, we do not know.

Theory of Infection.—Many attempts have been made to discover a bacterial origin, but without definite success. A new impetus has been given to the search by the investigations of Hunter, who has found as constant features a special type of glossitis and septic lesions in the mouth, stomach, or intestines separately or in combinations. Various bacteria have been found, but no single organism has been identified with the disease, and it is highly probable that, if any, more than one species may be capable of producing infection. The site of initial inoculation is probably the tongue, but, perhaps, in some cases another part of the gastrointestinal tract. The infection is evidently a chronic one; and an important feature in its pathology, if not in its etiology, as believed by Quincke and others, is the peculiar type of hemolysis manifested in the excess of iron in the liver, spleen, and kidneys.

Morbid Anatomy.—The surface of the body is extremely pale and often has a distinct lemon hue, but emaciation is unusual. The muscles are red; most of the other tissues are pale and anemic. All the organs show fatty degeneration. The heart is large, soft, and fatty. The ventricles are usually empty or contain a little light-colored blood. Ecchymoses are generally found in the skin and mucous membranes, and small extravasations in the various other organs and tissues of the body. The serous cavities may contain an increased quantity of serum, and moderate general or localized edema is common. The liver and spleen may be normal in size or slightly enlarged, but both these organs and the kidneys contain a large quantity of iron derived from the blood.

In the liver the deposit is confined to the outer and middle zones of the lobules.

Hunter records a peculiar type of glossitis found in 25 consecutive cases, and in seven of the cases examined post mortem septic gastritis with more or less atrophy of the stomach, and in three of the cases a septic, croupous enteritis, patches of congestion, enlargement of the follicles, and localized areas of colitis.

The blood-changes are typical of the disease. During the earlier stage, blood obtained from the finger or ear is fairly normal in color. The color index is higher than in any other form of anemia, owing to a relatively large quantity of hemoglobin contained in the corpuscles. This ratio may, in fact, be normal or increased. The red cells are always greatly reduced, the average number to the cubic millimeter being 1,000,000, but in extreme cases there may be only 200,000 or 400,000. The most distinctive feature of the blood-count, however, is the great abundance of megaloblasts. Normoblasts may be so few as to be demonstrable with difficulty; they are never so numerous as the megaloblasts. Eichhorst's corpuscles (small red cells without indentation) are usually seen. The leucocytes are reduced in number, but there is a relative increase of large and small mononuclears. Poikilocytosis, or irregularity of form, is often extreme. Hyperleucocytosis may be encountered. The erythrocytes are somewhat larger than normal, and megalocytes may be numerous. Oval erythrocytes are not unusual. Degeneration of the sympathetic ganglia has been repeatedly observed, and posterior spinal sclerosis in a few cases.

Symptoms.—There is in most cases a history of repeated gastro-intestinal disturbance, occasional attacks of vomiting, constipation, or irregular diarrhea, possibly with blood in the stools, and these symptoms sometimes persist throughout the course of the disease. But the symptoms are not always so severe as to occasion pronounced illness. The beginning is generally so insidious as not to be recognized by the patient until languor and muscular weakness, with increasing faintness and breathlessness after slight exertion, pallor and waxiness of the face, call attention to it. The mucous membranes, expecially those of the eyelids, lips, tongue, and gums, become blanched; the muscles are flabby and the heart is thrown into palpitation by exertion or a nervous shock. Hemic murmurs usually develop; the pulse is full and sometimes of the water-hammer type; the throbbing of the arteries is often visible and may be felt by the patient. The appetite is lost; headache, vertigo, and restlessness become more and more constant. Retinal hemorrhage, with consequent amaurosis, sometimes occurs. The skin becomes yellow, ecchymoses appear, possibly slight edema of the ankles, and finally, from extreme weakness, the patient becomes unable to leave his bed. The course of the disease is irregular and to some extent intermittent, intervals of apparent improvement separating periods of decline. Slight fever may be observed in the evening, but the temperature may become subnormal toward the end. Many patients suffer from hemorrhages of the nose, gums, intestines, or kidneys. Various nervous manifestations supervene in about a third of the cases, including anesthesia, spinal paralysis, aphasia, or tetany. The long bones often become sensitive to pressure. The urine is pale and of low specific gravity,

except when it contains much urobilin. Albuminuria is rare, peptonuria comparatively common.

Diagnosis.—This form of anemia is distinguished from chlorosis and other forms chiefly by the extreme decrease of red blood-corpuscles and relative increase of hemoglobin, with preponderance of megaloblasts. The constant presence of glossitis, if confirmed, will prove a valuable aid to diagnosis. The greatest difficulty, as a rule, lies in the exclusion of gastric cancer with extreme anemia, but the blood-count of cancer rarely approaches that of primary anemia, and the emaciation is rapid and extreme. The chemical examination of the stomach contents may further establish the diagnosis.

Prognosis.—The prognosis of a pure pernicious anemia is very unfavorable, but more or less complete recovery has been repeatedly observed, sometimes lasting indefinitely, sometimes followed by relapses. Extreme lowness of the red blood-corpuscles and the presence of great numbers of megaloblasts are regarded as of bad prognosis, but the blood-count is too changeable to constitute a reliable guide.

Treatment.—Arsenic has proved the most valuable remedy. Its action sometimes appears almost specific. It is best given in the form of Fowler's solution, beginning with ♏ iij (0.18) t.i.d., and increasing one minim each day until ♏ xx to xxv (1.2 to 1.5) are given, unless toxic symptoms arise. The remedy should then be discontinued for a few days and resumed in reduced dose. Iron is seldom useful, but it may be tried when arsenic fails after proper trial. Outdoor life, with light exercise, abundant rest, and nutritious food, is an important adjunct to the treatment.

Hunter and his followers have observed good results from the administration of intestinal antiseptics and the injection of antistreptococcus serum. Inhalations of oxygen prove beneficial in some cases, but useless in others, and this is true of many other remedies.

Chlorosis (Green Sickness).—A primary anemia occurring chiefly in young girls and characterized by a great deficiency in the quantity of hemoglobin, and less marked reduction of the number of red corpuscles, with pallor and other evidences of the condition.

Chloranemia signifies an anemic condition of the blood, like that of chlorosis, often observed as a result of tuberculosis, syphilis, cancer, and other affections, and not infrequently in persons whose age and sex do not correspond to the definition given.

Etiology.—Girls of blond type, at the age of puberty, are more susceptible than others to the disease. Those under 12 or over 20 are rarely primarily affected, but recurrences may continue for many years. The disease is very rarely observed in young boys. Heredity or a family influence is often an important factor, and a tubercular taint is one of the most commonly recognized etiological features in families. No definite cause has yet been recognized. There is much reason to look upon the disease as a neurosis, especially the frequency of the association of vasomotor neuroses, and the fact of its ready curability. The predisposing influences are many. Poor food and a disregard of hygiene are often influential. In a majority of cases, perhaps, there is an apparently close relation between the establishment of menstruation and the inception of the disease. Hypoplasia of the heart and aorta, not infrequent

pathological conditions in these patients, were regarded by Rokitanski and others as bearing a causal relation to it. Constipation, with consequent autointoxication, tight lacing, lack of exercise, and homesickness may be mentioned among the theoretical causes that have been offered. The disease commonly develops in young European girls soon after arrival in this country.

Symptoms.—The appearance of the patient is much the same as in pernicious anemia, but the pallor is less extreme and the skin has often a peculiar yellowish green tinge. The cheeks often bear a deceptive flush, and the mucous membranes may not reveal the poverty of the blood under the excitement of the first examination. The eyes are brilliant and the scleræ have a bluish tint. The adipose tissue remains nearly normal; emaciation is exceptional. Pigmentation is often observed in certain areas, particularly about the joints. Headache, languor, breathlessness, and palpitation are commonly complained of. The appetite is lost or becomes capricious. Nausea and vomiting are easily induced. Edema of the face and ankles, with cold feet, are common. Constipation is almost constant. Gastroptosis is often observed in girls who wear the corset. Dysmenorrhea or complete suppression is commonly present, and leucorrhea may be developed. Slight fever is sometimes observed. The heart is accelerated. A soft, diffused systolic murmur is usually heard, with maximum intensity over the pulmonary valve, the second sound of which is accentuated. A peculiar venous murmur is also heard over the right jugular (*bruit de diable*). Accidental diastolic murmurs are also occasionally heard. Pulsation is often visible in the peripheral veins. Late in the disease thrombosis may develop in the veins of the lower extremities, particularly in the femoral, and rarely in the cerebral sinuses. Pulmonary embolism is a possible result of the thrombosis. Nervous symptoms may also develop, and the patients very often become melancholic or hysterical. The urine generally remains normal except that the solids are increased and temporary variations of quantity are commonly observed.

The *blood* looks pale and coagulates readily. The corpuscles, when, examined under the microscope, also appear of light color, owing to the deficiency of hemoglobin. The number of the red corpuscles is reduced, but not as a rule much below 80 per cent. The hemoglobin may be as low as 45 per cent, and occasionally it is below 40. This is the most characteristic feature of the condition. Poikilocytosis, great irregularity of size and shape of the corpuscles, may be almost as extreme as in pernicious anemia. The average size of the corpuscles is diminished. Megaloblasts are never present (Higley). Normoblasts are common, but vary in numbers from time to time. Leucocytosis of moderate degree is ordinarily noted, but it is probably not important. The lymphocytes are relatively increased. The proteids of the serum are diminished.

Diagnosis.—The appearance of the patient, her age, and the clinical history generally lead to a correct diagnosis, but they are not always sufficient to differentiate it from the chloranemia of early tuberculosis or secondary syphilis. That of the malarial cachexia or of chronic nephritis can rarely be mistaken for chlorosis.

Tuberculosis can be distinguished by a careful physical examination and the discovery of the bacillus; syphilis, by the appearance of other

characteristic lesions. The blood-examination of malaria reveals some form of the plasmodium or free pigment. Chronic nephritis is rare in young girls, and the condition of the urine is pathognomonic. From pernicious anemia the distinction is usually made without difficulty by the low color index and the absence of megaloblasts.

Prognosis.—The disease is usually promptly overcome by early treatment, but recurrences are common, sometimes after an interval of several years. In some instances, too, the most thorough and persistent treatment proves but partially effective.

Treatment.—A mild case will recover with very little treatment further than rest, the correction of any unhygienic influence, and an abundance of good nourishment, consisting largely of meat. When the disease has advanced to the stage in which there are extreme pallor, dyspnea, faintness, anorexia, and amenorrhea, the patient should generally be confined to bed for the first week or two of treatment. The diet should be easily digestible and nutritious, with beef-juice or rare beef, milk, and eggs. If the home surroundings of the patient are unsanitary, she will do better in a hospital, and removal from the city to the country is always advantageous. Iron is generally the only drug that is required, and there is probably no better preparation of it than Blaud's pills, freshly prepared, and administered in doses of from gr. v to xv (0.3 to 1.0) three times a day. Although the quantity may be more than theoretically can be appropriated, no injurious effect is observable and a cure is effected. Many other preparations, and such natural waters as Ronsigno and Levico, containing both iron and arsenic, have been highly extolled. Reduced iron and the tincture of the chlorid are often effective. It is usually necessary, in order to prevent early recurrence, to continue the chalybeate treatment for several months or a year, but the maximum dose need not be maintained. As soon as improvement has become apparent, and it is often quite prompt, the patient should be kept in the open air and sunlight a greater part of the day, if the weather permit, and given light exercise.

Other symptoms generally require treatment, particularly the constipation. A saline laxative, given regularly every morning, is generally the best remedy for this. In cases accompanied with superacidity, a milder laxative alkaline mineral water may be taken before each meal.

II. SECONDARY ANEMIA.

Secondary anemia is induced by hemorrhage, disease, or such morbid states as inanition, toxemia, and poisoning. The blood is not primarily affected.

Etiology.—(1) Hemorrhage is one of the most frequent causes. It may be: (*a*) Rapid, as in spontaneous or traumatic lesions of the blood-vessels, in the rupture of aneurisms, or in the erosion of a vessel in gastric or duodenal ulcer, cancer, or cirrhosis. The quantity of blood lost may be so great as to cause severe, even fatal, syncope. (*b*) It may be slow, as in the more or less regular bleeding of hemorrhoids, uterine disease, persistent epistaxis, or from slight injury in "bleeders." (2) Inanition. This form of anemia is more frequently seen in individuals who are prevented, by the location of malignant or other disease of the

esophagus, from ingesting or appropriating sufficient nourishment. (3) Toxemic anemia occurs in many of the acute and chronic diseases, as typhoid fever, tuberculosis, syphilis, and malarial cachexia. Chronic poisoning by lead, copper, mercury, and arsenic affords examples of toxic anemia.

Symptoms.—The general symptoms are the same, to a varying degree, as those of primary anemia. Pallor, vertigo, syncope, headache, palpitation, prostration, and other symptoms correspond in severity with that of the condition. After a hemorrhage, all the elements of the blood are at first deficient. The water is rapidly replaced, the corpuscles next, and the hemoglobin more slowly. In the anemia following disease and toxic conditions, the deficiency may affect only the corpuscles, and the proteids of the plasma, or the hemoglobin may also be greatly reduced. Different conditions are more or less peculiar to different diseases. Malignant disease generally produces irregularity in the form and size of the corpuscles (poikilocytosis); suppuration is attended with leucocytosis.

Diagnosis.—With a knowledge of the cause, the condition is immediately recognizable; without this it may be differentiated with great difficulty from primary anemia.

The ***prognosis*** depends entirely upon the character of the cause. Recovery is generally spontaneous when this can be removed.

Treatment.—The removal of the cause, often a surgical measure, is the first element of the treatment. After that, the blood condition is promptly restored by rest and nutritious diet. Medication is seldom required, but iron and arsenic may be employed in chronic cases.

LEUKEMIA.

LEUCOCYTHEMIA.

Definition.—A disease the chief feature of which is a persistent overproduction of leucocytes, with changes in the spleen, bone marrow, and lymphatic glands. The lesions are often confined more or less exclusively to either one or two of these structures.

Etiology.—The disease occurs at any period of life, from earliest infancy to extreme old age, and in either sex, but it is more frequent in men during the third decade than in any others. Women are oftener attacked between the ages of 20 and 30. Heredity is regarded as an important factor, a so-called lymphogenous diathesis existing in families. The disease prevails among all races in all parts of the world. It is more frequent among the poor and working classes. Previous ill-health is sometimes noted, but it is not essential. Among the influences thought to predispose to it are malaria, syphilis, influenza, a tendency to hemorrhage, digestive disorders, pregnancy, and lactation.

The immediate cause is unknown. The usual search for a microorganism is going on. The supposition of an infectious nature is in a measure supported by the fact that the disease is not uncommon among certain of the lower animals. A protozoön has been found in the leucocytes and plasma by Löwit, who believes that he has transmitted the disease to animals by inoculation. Certain other investigators regard it as a neoplastic disease, a "sarcoma of the leucocytes," but neither of

these views has received strong support. An injury sometimes seems to be the exciting cause.

Morbid Anatomy.—The body is extremely emaciated; it may have a yellowish or greenish tinge, and edema with serous effusions into the cavities is common. The heart-chambers and the veins are often distended with coagulated blood which may have the appearance of pus owing to the great abundance of the white corpuscles. The coagula often have a peculiar greenish tinge. Charcot's colorless, octahedral crystals often separate from the blood in great numbers after standing. The characteristic lesions are found in the spleen, bone marrow, and lymphatic glands. In the most frequent type (splenomyelogenous leukemia), the essential feature is a combination of lesions in the spleen and bone marrow. In the other (lymphatic leukemia), lesions of the lymphatic glands predominate.

The Spleen.—Changes in the spleen are the most constant. They are almost always associated with changes in the bone marrow and lymphatic glands. The spleen is enlarged, sometimes greatly, sometimes so slightly as not to be recognizable during life. (*a*) The enlargement in lymphatic leukemia is at first due to a hyperplasia of the small and large mononuclear cells. The Malpighian bodies were once regarded as the primary seat of the disease in these cases. Large, pale, lymphoid bodies are found in the organ, but they are probably neoplastic, at least not Malpighian bodies, which may be unrecognizable. (*b*) In the myelogenous form, both small and large mononuclears (myelocytes) are found in great numbers in the spleen as well as in the marrow. An inflammatory condition is usually set up which results in a chronic hyperplasia of the connective tissue. The organ then becomes sclerotic and the capsule is often greatly thickened.

The Bone Marrow.—The medulla of the bones, both long and flat, loses its fatty appearance. In acute cases it often resembles thick pus, while in chronic cases it is firmer and lighter in color. It has occasionally a dark brown color. Local swellings may be observed over the bones, which may be tender and may yield on pressure. The essential change in the marrow is a cellular hyperplasia affecting the leucocytes and varying in intensity with the type of the disease. (*a*) In the lymphatic form the lymphocytes are greatly increased, and the neutrophiles and eosinophiles are comparatively few. The increase of lymphocytes is not always excessive, however, and in some cases of this type no alteration of bone marrow can be discovered. (*b*) In the medullary form the neutrophilic myelocytes show the greatest increase. Eosinophiles are present, but not in greatly increased numbers. These lesions (*a* and *b*) are generally combined and are rarely encountered separately. The changes in the erythrocyte occur for the most part in the advanced stages of the disease and are the same in character as those observed in pernicious anemia, without, however, so great increase of the megaloblasts.

The Lymphatic Glands.—The lymph-glands are always enlarged in the lymphatic form of the disease. Those of the cervical region are most frequently involved, but the axillary, inguinal, thoracic, and abdominal glands are usually affected. The enlargement generally begins in a single gland or group of glands, and extends to others in proximity, until the condition becomes general, including the follicles of the mouth,

tongue, tonsils, pharynx, and intestines. The glands usually remain comparatively soft, but sometimes become quite hard. On section they are white or pink, but necrosis may occur, with the production of yellowish spots; suppurative softening is rare.

On microscopic examination the enlargement is found to be due to hyperplasia of the cells, blood-channels, and connective tissue. The proliferation sometimes oversteps the limits of the capsule.

The liver, kidneys, and other organs are in some cases enlarged by a leukemic infiltration. The skin is occasionally affected with an eczema, which is followed in some cases by tumor formation and ulceration. Pruritus frequently develops independently of eruptions. Changes are seldom discovered in the lungs.

Symptoms.—The onset of the disease is not usually recognized until the glandular enlargement, abdominal distention, or possibly shortness of breath can no longer be overlooked. Gastrointestinal symptoms may have preceded these manifestations, and epistaxis may have occurred. A severe, even fatal, hemoptysis or hematemesis has been the first indication of the disease in some cases. The appearance of the patient is not always distinctive. The symptomatology is generally described under the two heads of Splenomyelogenous and Lymphatic Leukemia, corresponding to the two pathological types. Both these forms may be acute or chronic in character. Extremely acute cases are rarely observed, as those terminating fatally in the initial hemorrhage.

1. **Splenomyelogenous (Splenomedullary) Leukemia.**—The most prominent feature of this, the commonest, form of the disease is a progressive enlargement of the spleen. This may be accompanied with pain and tenderness. It may be extensive, even enormous, the enlarged organ occupying more than half the adbomen and extending to the pubis, but it varies somewhat in the same case from time to time. It is larger after a full meal, and often smaller after a hemorrhage or profuse diarrhea. Pulsation, a creaking fremitus, and a murmur have been observed in it in some instances.

The first indication of the disease in some cases is pallor, a symptom which, in other cases, is often absent until comparatively late. Either subjective symptoms or objective may be wanting in a given case. In those of a given type, an initial chill is not uncommon, with fever and other appearances of an infection. Nervous phenomena are commonly present, and they are sometimes the first indication of illness. Priapism has preceded all other symptoms in some cases for days or weeks. Later headache, vertigo, and fainting-spells may result from the anemia. Facial paralysis has been observed, and rarely an optic neuritis. The pulse is generally rapid, but it may continue slow, full, and soft. The heart may be displaced upward by the enlarged spleen. Hemorrhages often occur, as already noted, and purpuric or hemorrhagic extravasations into the skin, pleura, peritoneum, retina, or elsewhere. Bleeding of the gums is common. Pulmonary symptoms are unusual, aside from the dyspnea, but a terminal pneumonia or an edema of the lungs may develop. Sudden death without recognizable cause has been observed.

Disturbances of digestion are almost constantly present. Nausea, vomiting, diarrhea, and dysentery prevail in different cases. Peritonitis is sometimes produced by the formation of neoplastic tissue, and ascites

may accumulate. The liver usually becomes enlarged in chronic cases. The urine shows no constant change, except an increase of uric acid and the xanthin bodies. Peptonuria and albuminuria have been observed, and hematuria in hemorrhagic cases. Other occasional symptoms are: exophthalmos, partial deafness or tinnitus, and various menstrual disorders.

The Blood.—The blood-changes are the most distinctive feature of the disease. In the splenomyelogenous form the principal change is the great increase of colorless corpuscles. Their ratio to the red may be as high as 1 : 10, 1 : 5, or even in excess of 1 : 1. Their ameboid movements are sluggish. Their number varies, from time to time, in the same case. The large mononuclear myelocytes are more or less characteristic, especially the eosinophilic forms. Neutrophilic myelocytes are also present, and they are often found in different stages of degeneration. The granules may be few or entirely absent in acute cases. Great differences in the size of the eosinophiles and polynuclear leucocytes are sometimes observed, and especially in the polynuclear neutrophiles. The nuclei often show indentations and usually stain faintly. Distinct karyokinetic figures have been observed in the myelocytes of the blood and marrow.

The red corpuscles may remain normal, but in advanced cases their number is generally reduced. They rarely sink below 2,000,000 to the cubic millimeter. Normoblasts are generally present in large numbers. Larger cells with paler nuclei and often showing karyokinetic figures are also present. Megaloblasts may be found. Great variations in form are also observed—oval, lanceolate, and irregular forms, sometimes having fragmented nuclei. All these changes are well shown in Plate I, A (frontispiece). The specimen was obtained from a case exhibiting enormous enlargement of the spleen (St. Mary's Hospital, service of Dr. W. E. Kiely).

The hemoglobin sinks with the reduction of the red corpuscles, and sometimes a little more rapidly. It may be as low as 30 or even 20 per cent. It often shows a tendency to crystallize on the slide.

The color index of the blood is reduced, often to 0.4 or 0.5 in the early stage of the disease, but sometimes, owing to rapid destruction of the red corpuscles, toward the termination it may rise nearly to the normal. It may, however, continue subnormal throughout the course of the disease. The blood-plates are often exceedingly numerous; they are sometimes found in groups among the other corpuscles.

2. **Lymphatic Leukemia.**—This is rare as an independent affection. It is characterized by a general enlargement of the superficial glands. These often form large masses, but they do not attain the great enlargement seen in Hodgkin's disease. Acute cases more frequently conform to this type, but chronic cases may occur. Lymphatic nodules often form in the nose, ear, throat, skin, as well as in the regions of all the external lymphatic glands.

The distinctive feature of the blood examination in this form is the great abundance of small mononuclear leucocytes, fully 90 per cent of which are sometimes lymphocytes. Nucleated erythrocytes are seldom present in large numbers. Mitosis is seldom observed. The leucocytes rarely exceed 1 : 10 of the red, although the latter may be reduced to 50

per cent. Eosinophiles are rare; myelocytes are usually absent. These features may be studied in Plate I, B.

Various disturbances are produced by the glandular and other infiltrations. Cough and dyspnea follow the enlargement of the bronchial glands; tenderness of the long bones may result from their infiltration; ocular disturbances, from infiltration of the retina, and deafness from the nodular formations in the ear. The spleen is almost always enlarged, but not to the extreme degree that is seen in the other form of the disease.

Very similar to leukemia is the highly fatal chloroma, or green cancer. It is characterized by the formation of grass-green, yellowish, or grayish-green tumors behind the eyeball, causing exophthalmos, and in other regions. The nodules resemble those of leukemia, pseudoleukemia, or multiple myeloma, another rare affection probably of the same class. In some cases changes in the blood like those of leukemia and a tendency to hemorrhage have been observed. A green coloring matter is sometimes found in the urine.

Diagnosis.—The recognition of leukemia and that of its separate forms depends upon the microscopic examination of the blood. In both forms there is great increase in the number of leucocytes, more extreme in the splenomyelogenous than in the lymphatic. Myelocytes predominate in the former, lymphocytes in the latter.

Prognosis.—The prognosis is unfavorable. Recovery is possible, but the disease usually progresses slowly to a fatal termination in the course of two or three years. Its progress is not uniform, however. Intervals of apparent improvement frequently occur. The lymphatic form is generally more rapidly fatal. The occurrence of hemorrhage, persistent diarrhea, high temperature, and edema are particularly unfavorable signs.

Treatment.—The treatment is in many respects the same as that of pernicious anemia. The patient should be given physical and mental rest, an abundance of fresh air and sunshine, and nutritious food. Arsenic is often beneficial, but it has not the specific action which it so often exhibits in pernicious anemia. It should be given in increasing doses. Iron and quinin are often of apparent benefit. Inhalations of oxygen have been followed by at least temporary improvement. The natural tendency of the disease to intervals of improvement should be borne in mind.

PSEUDOLEUKEMIA

HODGKIN'S DISEASE, MALIGNANT LYMPHOSARCOMA, MULTIPLE MALIGNANT LYMPHOMA.

Definition.—A chronic progressive form of anemia with marked enlargement of the lymphatic structures and spleen, often accompanied with a growth of lymphoid formations in the liver and other organs.

Etiology.—The disease usually attacks the young. It is not uncommon in infancy and childhood; 75 per cent of the cases occur between the ages of 10 and 40, and but few after the latter age. Men are much more commonly affected. A hereditary influence often seems probable. Previous illness, as syphilis and malaria, are possible, but very uncertain

factors. No definite cause is known. In some instances the disease develops at first locally at some point of prolonged irritation, as about a decayed tooth, or follows a chronic nasal catarrh or chronic skin disease, but, as a rule, its invasion is insidious. The disease has been regarded by some writers as an infectious granuloma similar to leprosy. Some have, indeed, divided it into two forms, one tubercular and characterized by a febrile course, the other sarcomatous. Sternberg regards it as essentially tubercular, while other investigators have looked upon the presence of tubercle bacilli in the lesions as accidental. It is possible that several diseases are still included under this heading.

Morbid Anatomy.—The lesions are found particularly in the lymphatic glands and the spleen. The lymphatic glands of the entire body are sometimes enlarged. Among the superficial glands the most prominent are those of the neck, axilla, and groin; among the deep glands, the bronchial, mediastinal, and retroperitoneal. The nodules are generally soft, but may become extremely firm. In size they may exceed an egg, many smaller nodules generally being present. Even when they are hard their interior is generally soft and often caseous. The capsule is sometimes ruptured by the increasing growth. In rare instances the sternum and vertebræ have been penetrated and the spinal cord has been pressed upon. The enlargement consists of a hyperplasia of the lymph-cells. The reticulum may be thickened, but it is sometimes almost undistinguishable in the softer nodules. The bone marrow may be pus-like, as in pernicious anemia.

The spleen is enlarged in 75 per cent of all cases, but not to the extent characteristic of true leukemia. The hypertrophy is due to the growth of grayish white lymphoid bodies varying in diameter from ⅛ to 1½ inches (0.5—4.0 cm.), and composed of lymphoid corpuscles in a fibrous reticulum. Similar lymphoid growths are sometimes found in the tonsils, thyroid gland, thymus, lungs, liver, kidneys, adrenals, and skin, and rarely in the solitary follicles of the intestine, in the brain, retina, and testicle.

Symptoms.—Enlargement of the cervical glands is usually the first symptom to attract attention; occasionally this is accompanied with similar swelling of the axillary and inguinal groups. The disease may extend next to the glands of the opposite side or to those of other groups. An acute onset and rapid progress are occasionally observed. Angina with enlargement of the tonsils has constituted the beginning of some cases. In other cases, again, the deep-seated glands of the thorax have been the starting-point, and the first symptoms have been those of pressure upon the bronchi, with dyspnea and cough; pressure on the vena cava with venous engorgement of the upper part of the body and the development of visible anastomoses, or pressure upon the cervical sympathetic, with inequality of the pupils. Very exceptionally, edema of the lower extremities, shooting pains or paraplegia, due to pressure upon the veins and spinal cord, have been the initial manifestations. With the increasing enlargement of the glands the patient becomes more markedly anemic. He rapidly loses strength, he often suffers with headache, palpitation, tinnitus, vertigo, dyspnea on exertion, loss of appetite, and other disturbances due to the anemia. The deformity produced by the enlargement of the glands of the neck is often extreme and charac-

teristic, entirely obliterating the contour of the cervical and clavicular regions. In like manner, large tumors may be formed in the axillæ and groins. The internal glands can usually be felt in a thin person. A peculiar feature is an absence of uniformity in its progress; there is often a cessation of the growth of the glands or even a reduction of their size. The clinical features of the disease are exceedingly variable, owing to differences in the extent of the lesions and their locations. Moderate fever is sometimes a prominent feature in tubercular cases. It is usually of an irregular, intermittent, type, rarely continuous.

The *spleen* is enlarged to a variable extent in most cases. The increase of size may come on slowly or with remarkable rapidity, and the same lack of uniformity is exhibited as in the lymphatic glands. In extreme cases, especially in those occurring in children, the organ may extend down to the brim of the pelvis. Some writers regard the splenic anemia of the German authors, in which the spleen alone is enlarged, as a form of pseudoleukemia.

The Blood.—In some cases the blood remains normal, except that the hemoglobin is reduced. This deficiency is seldom below 60 per cent. The eosinophiles and mononuclears are generally relatively increased, especially in febrile cases. A few myelocytes are occasionally observed. Hemic murmurs are sometimes heard over the heart. The urine generally remains normal. Bronzing of the skin is occasionally noted, approaching, in severe cases, the color of Addison's disease.

Diagnosis.—The diagnosis rests upon the blood examination, based upon the distinctions just given. Simple tubercular adenitis is more common in children, more frequently affects the submaxillary than the cervical and axillary glands, and is generally slower of extension. Yet acute cases are observed. Syphilitic adenitis is recognizable, as a rule, by the history, the presence of other symptoms, and the greater, or exclusive, enlargement of the posterior cervical glands.

Prognosis.—The prognosis is highly unfavorable. Recovery has been reported, but the progress of the disease is fatal and the intervals of apparent improvement deceptive. The glandular swellings sometimes subside almost completely shortly before death. Fever, rapid emaciation and great prostration, with pressure symptoms or hemorrhages, are unfavorable indications.

Treatment.—Arsenic is generally employed in the treatment, but its action is less marked in advanced cases. The apparent results often prove fallacious. Quinin, iron, codliver oil, phosphorus, and strychnin are of benefit in some cases. Local applications of iodin ointment or tincture, or of mercurial preparations, and the galvanic current over the tumors, have been followed by more or less continued reduction of their size. The expediency of early excision of the glands should be considered. Several recoveries have followed removal of the spleen.

PURPURA.

MORBUS MACULOSUS.

The term purpura is applied generically to a group of conditions in which, without serious impairment of health, hemorrhages occur into

the skin of a greater or less portion of the body. The hemorrhagic spots may be punctate (petechial) or of the nature of ecchymoses, seldom exceeding an inch in diameter. In color they pass from a bright red to a dark brown. They do not disappear upon pressure. Such eruptions are not of infrequent occurrence in various infectious, toxic, cachectic, and nervous conditions. The purpura is then referred to as secondary or symptomatic. Several so-called primary forms are also observed.

1. **Symptomatic Purpura.**—(*a*) *Infectious.*—The rash of typhus fever is normally petechial; the eruptions of measles, smallpox, and other exanthemata are occasionally hemorrhagic. Distinctly hemorrhagic eruptions may occur also in connection with cerebrospinal meningitis, septicemia, typhoid fever, malignant endocarditis, and in such chronic affections as leukemia, pseudoleukemia, pernicious anemia and tuberculosis. (*b*) Among *toxic* cases are those resulting from snake venom or such drugs as potassium iodid, chloral, ergot, quinin, copavia, or belladonna, or in association with jaundice. (*c*) *Cachectic* cases occur in connection with malignant disease, scurvy, chronic nephritis, and in extreme old age. (*d*) *Neurotic* purpura is met with in connection with neuralgia, hysteria, locomotor ataxia, acute and transverse myelitis. (*e*) In another group are sometimes included cases of mechanical purpura resulting from obstruction, venous stasis, and extravasation of blood from violent effort, as in whooping-cough or convulsions. (See also Hemorrhage, p. 14.)

2. **Arthritic Purpura.**—Under this heading are grouped three classes of cases, in all of which more or less involvement of the joints is generally observed:

(*a*) *Purpura Simplex.*—This is a mild form of the affection, occurring almost exclusively in children, and sometimes without arthritic manifestations. The patients are generally anemic and the condition is often accompanied with digestive disturbances and diarrhea. The joints are swollen and painful in some cases, but fever is unusual. The purpuric spots appear on the legs, less frequently on the arms and trunk.

(*b*) *Purpura Rheumatica* (Peliosis Rheumatica; Schönlein's Disease).—This affection attacks chiefly men between 20 and 40 years of age, and more commonly those who have been debilitated by previous illness. A rheumatic history may be obtained. It is generally associated with distinct articular pains and swelling which are regarded as rheumatic in nature, a supposition favored by the fact that tonsilitis, endocarditis, and pericarditis are occasional complications. Fever amounting to 102° or 103° F. (38.8°—39.5° C.) is generally present. The eruption appears about the legs and on the affected joints, but it is not uniformly purpuric. Urticaria (purpura urticans), erythema with hemorrhagic extravasation, or a vesication like that of pemphigus is sometimes met with. Edema of the feet and ankles is seen in some cases and the face may become edematous. The term febrile purpuric edema has been applied to cases in which fever is also present. The patient is anemic, loses his appetite, and becomes prostrated. Digestive disorders are common. The urine becomes scant and is often albuminous.

(*c*) *Henoch's disease* is a recurrent form of purpura, seen, for the most

part, in children. It is accompanied with gastrointestinal disturbances, slight swelling of the joints sometimes with renal symptoms, and hemorrhages from the mucous membranes, the bowels, or kidneys. Enlargement of the spleen is common. The disease is sometimes fatal, especially in adults, but as many as a dozen recurrences may take place without fatal result.

3. **Purpura Hemorrhagica** (Morbus Maculosus Werlhofi).—This, the most serious form of purpura, is generally seen in young, delicate girls, but vigorous adults are not exempt from it. Poorly nourished individuals exposed to cold and damp dwellings, and those recovering from illness, are thought to be more susceptible than others. Such toxic agents as iodin, mercury, silver, and phosphorus have been regarded as the exciting cause in some cases. An infectious origin has also been supposed to exist.

Symptoms.—The onset of the affection may be sudden and severe, usually announced by great weakness, an eruption of purpuric spots, and hemorrhages from the mucous membranes. An initial epistaxis may occur. In severe cases the patient becomes almost exsanguinated, and death may ensue from the loss of blood or from hemorrhage into the brain. Eruptions like those seen in rheumatic purpura are usually present. The blood presents nothing distinctive, except marked increase of the time required for its coagulation, often amounting to ten or fifteen minutes. After the hemorrhages there is a pronounced oligemia, and for a time the hemoglobin remains reduced.

A fulminant form of purpura hemorrhagica is sometimes encountered in which, with a profuse cutaneous eruption, but without actual loss of blood from any of the mucous membranes, a fatal prostration is produced, death sometimes occurring within the first 24 hours.

Diagnosis.—The disease is not usually difficult of recognition, but it is sometimes extremely difficult to determine whether a case is one of pure purpura hemorrhagica, a leukemia with hemorrhagic symptoms, or a toxic purpura associated with an acute infectious disease. It is sometimes impossible for a few days to arrive at a positive diagnosis. As a rule, however, the glandular and splenic enlargements differentiate leukemia, and the prodromal symptoms an acute infection.

The ***prognosis*** must always be guarded, for it is never possible to predict the course of the disease.

Treatment.—In the symptomatic form the treatment is chiefly that of the underlying condition. Sodium salicylate should be administered in rheumatic cases, and arsenic in simple purpura. The arsenic should be given in increasing doses until slight toxic effects begin to appear. Ergot, lead acetate, turpentine, aromatic sulphuric acid, gallic acid, and other agents have been employed with uncertain success to control the hemorrhages. Calcium chlorid in doses of gr. xx (1.30) every four hours has been regarded as beneficial in increasing the coagulability of the blood. When the hemorrhage is from an exposed point, as in the mouth or nose, irrigations with a 2 per cent gelatin solution, or the application of a solution of adrenalin or of the suprarenal extract should be resorted to. Fuller's success with the internal administration of thyroid extract, in full doses, in hemophilia suggests the possibility of beneficial action also in this condition. After the hemorrhages

have been arrested, the strength of the patient should be built up with tonics and nutritious food, remedies for the digestion being administered if necessary. Iron and arsenic are of service in preventing recurrences.

HEMOPHILIA.

Definition.—A hereditary constitutional affection marked by a tendency to bleed spontaneously or after slight injury. The hemorrhages are generally severe, sometimes uncontrollable and fatal.

Etiology.—In a great majority of instances the disease is recognized in early life, but it may not become manifest until as late as the twentieth year, or later. The hemorrhagic tendency is generally more marked in the male members of the family, if not confined exclusively to them. It is transmitted, as a rule, by the female to her male offspring, although she may not be herself a bleeder. The disease is handed down through families, sometimes as far as the seventh generation. It has rarely been transmitted from father to son. The disease has been met with in all parts of the world, and among people of every station. It seems to be more common, however, in the Anglo-German races, in cold climates, and it is more frequently developed in the spring and autumn. No specific cause is known.

Morbid Anatomy.—The morbid condition apparently lies in the peculiar type of blood-vessel rather than in the blood. Bleeders are generally well developed and apparently healthy, but they have delicate skin and thin blood-vessels. In some cases, at least, the middle muscular coat of the vessels has been found extremely thin. The blood is usually normal, so far as can be determined. An increase of the number of erythrocytes has been observed in some instances. The coagulability of the blood is so far reduced in some cases that 30 to 45 minutes are required for the formation of a clot. The leucocytes and blood-plates have also been found deficient.

Symptoms.—The distinctive symptom is the tendency to persistent hemorrhages, occurring spontaneously or following some trivial injury, as the scratch of a pin, a cut, or bruise. Fatal hemorrhage has followed the extraction of a tooth or the operation of circumcision. A spontaneous hemorrhage more frequently assumes the form of an epistaxis, but it may originate in the lungs, stomach, bowels, urethra, or from any of the other mucous membranes. Severe hemorrhages sometimes occur at the menstrual period or after parturition in women thus affected, but it is a remarkable fact that they are very exceptional. The bleeding in any case may continue for several days and cease spontaneously, to be followed by prompt recovery, but a condition of profound anemia sometimes remains.

Arthritic symptoms occasionally accompany the hemorrhages, the large joints becoming swollen and painful. Blood is sometimes extravasated into them. A febrile synovitis is sometimes developed, and it may leave the joints stiff and deformed. Petechiæ, ecchymoses, and large hematomata are sometimes formed in various regions as a result of the extravasations.

Diagnosis.—The diagnosis is based upon a persistent tendency to profuse hemorrhages when it can be traced to a hereditary influence. This

is more commonly possible when there is simultaneous involvement of the articulations. The distinction between this disease and the different forms of purpura is not usually difficult, except in some cases of purpura rheumatica or in the absence of a definite family history.

The ***prognosis*** is relatively less favorable in young persons. The later the disease becomes manifest, the greater is the possibility of its being outlived. Children showing the hemorrhagic tendency early seldom attain the age of puberty.

Treatment.—It is not always possible to protect the bleeder from the trivial accidents which may induce a serious hemorrhage, but surgical operations of every kind should be avoided. Even vaccination should be performed with care in order not to start a persistent oozing. The female members of the family, particularly, should be advised against marriage.

When a hemorrhage has been induced, the patient should be confined to bed and given complete rest. Ice and astringents may be applied when possible. Fuller has recently found the administration of thyroid extract immediately effective in a typical case, arresting the hemorrhage and apparently overcoming the tendency. It should be given in doses of gr. v (0.30) t. i. d. to an adult. If this remedy prove unsuccessful, resort must be had to the older remedies—iron, gallic acid, lead acetate, and other astringents and styptics. When the bleeding area is accessible, solutions of adrenalin, the suprarenal extract, or gelatin may be applied. Transfusion has not proved successful. During the intervals an attempt may be made to overcome the tendency by the administration of iron and arsenic.

HEMORRHAGIC DISEASES OF THE NEW-BORN.

Epidemic Hemoglobinuria (Winckel's Disease).—This disease occurs epidemically in lying-in hospitals, among infants from one to ten days old. The infants become jaundiced, feverish, refuse nourishment, and rapidly become cyanotic. The urine is scant, high-colored, and contains methemoglobin and albumin. Hemorrhages occur into various organs. The origin of the disease is not known; investigations have failed to establish a supposed septic cause. Acute fatty degeneration of the internal organs (Buhl's disease) is frequently associated with it.

Syphilis Hemorrhagica Neonatorum.—This affection occurs, in connection with jaundice, in young syphilitic infants. At birth, or soon afterward, ecchymotic spots appear, and hemorrhages occur from the umbilicus and mucous membranes.

Morbus Maculosus Neonatorum.—This term has been applied to fatal hemorrhages from the umbilicus, gastrointestinal canal, or nose, and to simultaneous bleeding from all these sources, during the first week or two after birth. The disease usually runs a rapidly fatal course; sometimes it is attended with fever. Intense icterus is sometimes present. Its cause and nature are unknown. The probability of infection as its cause is inferred from its general occurrence in hospitals. In the diagnosis of some cases of infantile hematemesis it is necessary to exclude the vomiting of blood which has been drawn from the nipple of the mother.

SCURVY.

SCORBUTUS.

Definition.—A subacute or chronic disease the prominent features of which are inanition, anemia, debility, a swollen, spongy condition of the gums, and a tendency to hemorrhages.

Etiology.—The disease may develop at any period of life. Infants were practically exempt from it until the practice of artificial feeding became prevalent, and it is now a common affection of early life. There is probably no difference in the susceptibility of the sexes, but men are more commonly exposed to the privations which induce the disease and are, therefore, more frequently affected. The important etiological factor in all cases is malnutrition, from improper food. The disease was formerly exceedingly common among soldiers and sailors who were compelled to subsist for a long time upon a restricted diet. Since the importance of a proper diet has become recognized, however, the disease is much less prevalent. It is still seen occasionally in the hospitals of seaports, among the inmates of penal institutions and asylums, and among the foreign population of mining districts.

There are two theories of its origin, one attributing it to the improper food, and the other to an unknown form of infection.

(1) *Improper Food.*—The precise nature of the deficiency among the ingredients of the food is not known. (*a*) It is generally believed to be an absence of the ingredients supplied by fresh vegetables and fruits, but whether it is the lack of organic or of inorganic salts is still uncertain. (*b*) The presence of toxic matter in the food as a result of decomposition is also a possible cause. Experiments by Vaughan and others lend strong support to the latter view and place the disease in close relationship with ptomain-poisoning. (*c*) That the disease is induced by the excessive eating of salted meats, at least so far as the introduction of too great an amount of sodium chlorid is concerned, is no longer regarded as tenable.

(2) *Infection.*—No specific organism has been discovered by those who regard the disease as an infection. The theory is supported mainly by the epidemic occurrence of the disease, the unsanitary conditions to which its victims have been exposed being looked upon as a predisposing cause, preparing the system for infection. Such clinical features as purpura and hemorrhages add some weight to it. There is no evidence of contagiousness.

Certain predisposing influences are well recognized, as dwelling in damp apartments, overcrowding, mental depression, physical fatigue, worry, grief, homesickness, and such diseases as malaria, syphilis, and dysentery.

Morbid Anatomy.—Extensive pathological lesions are often found, but they are not characteristic of the disease. The body may be extremely emaciated and mottled with ecchymoses, the ankles puffed with edema. The blood may be fluid or partly coagulated; degenerative changes are usually found in the larger extravasations; suppuration is rare. The blood-count shows nothing distinctive. Hemorrhagic accumulations are found in the mucous membranes, muscles, and internal organs. The gums show characteristic swelling and sometimes ulceration;

some of the teeth may have fallen out, or they may remain loosely attached. The spleen is large and soft. Parenchymatous degeneration is commonly found in the heart, liver, and kidneys. The lymphatic glands are not usually involved, but those of the mesentery may be swollen when the intestinal mucous membrane is affected.

Symptoms.—The onset of the disease is generally preceded by prodromal symptoms on the part of the gastrointestinal system, accompanied with pallor, emaciation, and weakness. Soreness of the throat or a severe internal hemorrhage may be the first indication of it. In many instances the condition of the gums is the first symptom to attract attention. This is characterized by swelling and a spongy condition which renders them liable to bleed upon the slightest irritation. The swelling begins about the incisor teeth and spreads to the other parts; in severe cases it may be so extreme as to completely conceal the teeth from view. A pseudomembranous growth is sometimes noted on the surface. Actual ulceration is not common, but the teeth become loose and are sometimes lost. The tongue becomes swollen and red, and the breath is extremely fetid. Hemorrhagic spots appear in the mucous membranes, and bleeding often occurs, usually in the form of oozing. Epistaxis occasionally occurs; hemoptysis and hematemesis are unusual. The salivary glands may be enlarged. The saliva flows in increased quantity, and, mingled with blood, it often escapes from the mouth. The skin is pale, cool, dry, and sometimes has a slightly yellow hue. Ecchymoses soon appear upon the legs, then on the arms and trunk. Petechiæ form in and about the hair follicles. Irregular nodules are often formed on the legs by effusions between the periosteum and bone, and they sometimes break down to form uncleanly ulcers. Slight injury induces hemorrhage. Edema of the ankles is usually present. In severe cases the infiltration into the subcutaneous and intermuscular tissues of the legs leads to a firm induration, and this may be followed by hyperplasia of the connective tissue, which leaves permanent stiffness and loss of motion, particularly in the region of the joints (scurvy sclerosis). Necrosis of the bones and epiphyseal separations sometimes occur in advanced cases.

The gastrointestinal tract is affected to a variable extent. Thirst is often extreme; the stomach is irritable and there is often a craving for sour or highly seasoned articles, or the appetite may be suppressed. Constipation is the rule, but diarrhea or the scurvy dysentery may supervene. The action of the heart is generally feeble, and a hemic bruit may be heard over the base. Hemorrhagic infarction sometimes forms in the lungs or spleen. The urine becomes scant, dark, concentrated, and often albuminous. Headache, lassitude, mental depression, and finally delirium or coma are often noted toward the close. Hemiplegia, convulsions, or other nervous complications may develop. Hemeralopia (day-blindness) or nyctalopia (night-blindness) is an occasional symptom, sometimes developing early in sailors. The disease generally runs an afebrile course, except as fever may result from the inflammatory processes induced by the hemorrhagic infiltrations in the organs.

Diagnosis.—The diagnosis of scurvy is based upon the history of the case, the stupor, the peculiar condition of the gums, and hemorrhages,

emaciation, weakness, and finally the prompt recovery after restoration of proper food. It is occasionally difficult, however, to exclude certain forms of purpura.

The ***prognosis*** is favorable, except in the most advanced cases or when it is impossible to remove the cause. Such complications as pneumonia, thrombosis, hemorrhagic pleurisy, meningeal hemorrhage, dysentery, or acute nephritis may lead to a fatal termination.

Treatment.—Prophylaxis consists in the supply of sufficient fresh vegetables and fruits. In lieu of this it is now required by nearly all governments that soldiers and sailors be provided with canned fruits and vegetables, lime-juice, lemons, and other antiscorbutic articles.

During the attack, the patient should be at once given as liberal a supply of fruit and vegetables as his digestion will tolerate. The juice of two or three lemons or oranges daily, with meat and fresh vegetables—potatoes, lettuce, water-cress, cabbage—causes a rapid cessation of the disease, except in the most advanced cases. It is not necessary to restrict the diet in any way, so long as the digestion is not too feeble to permit the ingestion of solid food. It is then often necessary to supply liquid nourishment, especially milk; and fruit juices must take the place of the fresh fruit. Bitter tonics and dilute hydrochloric acid are beneficial in such cases. Such symptoms as constipation or diarrhea may call for special treatment. The swollen, necrotic gums often require the application of solutions of silver nitrate, potassium permanganate, hydrogen peroxid, or carbolic acid. The hemorrhages must occasionally be treated as those in purpura.

SCURVY IN INFANTS.

BARLOW'S DISEASE.

Etiology.—The disease is directly due to the feeding of infants exclusively with condensed milk, sterilized milk, or other artificial foods. Foods which are claimed to contain all the necessary ingredients are, as a rule, most dangerous. The disease is generally encountered after the sixth month and before the twelfth, rarely so late as the fifteenth. Faulty hygiene is perhaps influential in some cases, and the disease is probably sometimes related to rickets and syphilis.

Morbid Anatomy.—The gums are little or not at all inflamed before dentition has occurred. The lesions are confined chiefly to the lower extremities. Extravasations of blood are found beneath the periosteum, sometimes under that of the tibia and fibula. Superficial necrosis may follow, and the epiphyses may separate. The bones of the upper extremities, lumbar vertebræ, and orbits have been found affected. The joints are not usually involved, but the extravasations around them give them the appearance of being swollen. Hemorrhages may occur in the internal organs, especially the lungs, spleen, kidneys, and intestinal glands. The blood shows no characteristic change, but the red corpuscles are generally reduced to a degree corresponding to the severity of the disease.

Symptoms.—The disease develops gradually, the infant showing increasing peevishness and restlessness, until it finally gives unmistak-

able signs of suffering. Its color is bad, pale or ashen. Digestive disturbances develop, and the appetite fails or there may be excessive hunger. The tongue is usually dry and coated, the breath fetid, and diarrhea is generally present. Hematuria is often observed, but other hemorrhages are exceptional, except the extravasations along the bones. The characteristic feature of the disease is the occurrence of these subperiosteal hemorrhages over the long bones, especially of the lower extremities. They are generally symmetrical. Pyriform swellings form around the diaphysis, beginning at the junction of the epiphysis and gradually decreasing toward the shaft. Separation of the epiphysis is common, and a crepitus may be obtained as a result. The swellings are painful, and the infant lies with the legs drawn up. Motion and pressure increase the pain and induce crying. The skin over the prominences becomes tense and glazed, sometimes ecchymotic. The shafts of the affected bones are sometimes permanently thickened. In the later stages of the disease the legs are held in extension usually with the toes turned outward, and the condition is spoken of as pseudoparalysis. Extravasations and edema about the orbits, often occurring early, give the infant a peculiar appearance, and the globes may become unduly prominent (proptosis), on account of hemorrhages into the orbits. The course of the disease is usually progressive so long as the improper food is continued, but recovery generally follows the adoption of a mixed diet.

Diagnosis.—The distinctive features of the disease are the peculiar painful swellings, the fretfulness, edema of the eyes, and the position of the legs. A differentiation must frequently be made between acute rheumatism, rickets, purpura, and infantile syphilis. *Acute rheumatism* affects the joints, and the swelling does not follow the shaft of the bone. Fever is a prominent symptom, and the pain and tenderness are greater. The salicylates give relief. *Rickets* can generally be recognized by the enlargement of the epiphysis, without painful swelling, the beaded appearance of the ribs, deformed chest, and square head. The abdomen is enlarged, and digestive disturbances prominent. But scurvy and rickets are sometimes associated. *Purpura* does not show the peculiar subperiosteal extravasations or the painful swelling, as a rule. Petechiæ and ecchymoses are common; the disease is unusual in infants and is of shorter duration. *Syphilis* is not accompanied with the hemorrhagic lesions, but with others, especially about the mouth, which are characteristic. The pseudoparalysis may be mistaken for true paralysis, but in the latter there is complete loss of voluntary motion, and not merely a restriction of it on account of pain.

Prognosis.—Recovery is rapid when the disease is recognized and treated early; but if neglected, it may terminate fatally.

Treatment.—The artificial food must be replaced with properly prepared cow's milk to which egg albumen and a teaspoonful of beef-juice should be added once or twice a day. From a half-ounce to an ounce of the juice of the orange, lemon, grape, or apple should be given to an infant during each 24 hours. Potatoes and baked apple may be given to an infant more than a year old, providing its digestion is not too feeble. The sirup of the iodid of iron or minute doses of arsenic have been found of benefit in overcoming the anemia. The swollen limbs should be wrapped in cotton and protected from motion and pressure.

Prophylaxis demands a more restricted use of the so-called substitutes for mother's milk. When these must be used, a food prepared with fresh milk and permitting the addition of beef-juice should be selected. Overheating the milk is also injurious.

STATUS LYMPHATICUS.

LYMPHATISM.

Definition.—A rare affection of childhood and youth characterized by hyperplasia of the lymphatic glands and tissues throughout the body, the spleen, thymus, and lymphoid bone-marrow.

Etiology.—The cause is unknown. The disease is often associated with rickets and with hypoplasia of the heart and aorta, and in quite a number of instances its presence has been revealed after sudden death.

Morbid Anatomy.—All the lymphatic structures are found in a state of hyperplasia, but most notably those of the alimentary canal, including the tonsils. The intestinal follicles often stand out prominently upon the surface. The bronchial glands are also enlarged. The lymphatic swellings are generally firm, but the spleen is soft and hyperemic, sometimes not greatly enlarged. The thymus is also soft and large and it may contain a large quantity of a milky fluid. The bone marrow often becomes red and may undergo hyperplasia. The thyroid gland may also be enlarged. Lack of development of the heart and aorta, sometimes also of the entire arterial system, has been observed.

Symptoms.—The enlargement of the external glands can be felt, that of the deep-seated glands, particularly those within the thorax, may be determined by percussion; the spleen is generally palpable. The child appears rachitic and develops slowly. Its vitality and power of resistance seem to be lowered. Sudden death has occurred in several instances under the administration of an anesthetic, after a dose of diphtheria antitoxin, or during convalescence from an acute infection. Sudden deaths while bathing or immediately after falling into the water, as well as those from unrecognized causes, have been attributed to this condition. Our knowledge of the affection is very incomplete, and further investigations are necessary.

DISEASES OF THE SUPRARENAL BODIES.

ADDISON'S DISEASE.

Definition.—A constitutional disease characterized by asthenia, feeble circulation, gastric irritability, and pigmentation of the skin.

Etiology.—The disease is a rare one, more frequent in men between 20 and 40 years of age. A few cases have occurred in infants and very old persons. Of predisposing causes, tuberculosis is regarded as the most important, but in some cases there has been a history of injury, as a blow upon the abdomen or back, caries of the vertebræ, psoas abscess, or other condition of doubtful importance, previous to the development of the disease. The immediate cause has not been fully determined. Lesions are almost constantly found in the adrenals and almost as con-

stantly in the fibers of the abdominal sympathetic nerves. There are, therefore, two principal theories in regard to the origin of the disease: (1) That it originates from an arrest of the function of the adrenal bodies, with loss to the system of their powerful internal secretion, and (2) that it is due, in part at least, to a neuritis of the abdominal sympathetic fibers. Both theories are doubtless correct, each in part explaining the phenomena of the condition. The view that the symptoms do not depend wholly upon the lesions of one organ is supported by the imperfect success obtained from the administration of adrenalin.

Morbid Anatomy.—The body is seldom anemic or markedly emaciated. The skin is diffusedly pigmented, the mucous membranes and sometimes the serous membranes in patches. The pigment is deposited in the lower layer of the rete Malpighii, where it is normally most abundant in the negro. The most important lesions are found in the adrenal bodies, in the abdominal sympathetic nerves, particularly in the semilunar ganglia.

The lesions of the adrenals are: (*a*) Most frequently tubercular, the capsules being found in many cases in an advanced state of caseation with hyperplasia of their connective tissue; (*b*) simple atrophy, or atrophy with sclerosis; (*c*) carcinoma or sarcoma; (*d*) extravasation of blood. Both bodies are usually involved in these lesions. (*e*) The disease sometimes exists without recognizable alteration of the adrenal bodies, but with changes in the semilunar ganglia or solar plexus, due to pressure or inflammation. All these conditions, however, with the exception of tuberculosis, are extremely rare.

The lesions of the nerve-fibers are generally sclerotic in character, with degenerative changes and more or less marked pigmentation. Compression of the ganglia by the hyperplastic connective tissue about the capsules can sometimes be distinctly demonstrated.

Other lesions commonly associated with those just described are enlargement of the intestinal lymph-follicles, enlargement and softening of the spleen, and parenchymatous or fatty degeneration of the heart, liver, and kidneys in some cases. The thymus gland may have failed to undergo atrophy, and it is sometimes slightly enlarged.

The interrelation of these lesions with reference to cause and effect remains undetermined, although much study has been given to the subject. While the importance of the lesions in the suprarenal bodies has become more apparent since the action of their internal secretion (adrenalin) has been demonstrated, the lesions of the sympathetic ganglia and solar plexus are looked upon by many writers as the direct cause of the pigmentations, debility, and functional disturbances on the part of the circulatory, respiratory, and digestive systems. The view is supported by the fact that pigmentation of the skin is sometimes associated with tuberculosis of the peritoneum, cancer of the pancreas, and aneurism of the abdominal aorta. Finally, advanced lesions of the adrenals have been found in cases in which the clinical features of Addison's disease were absent. These cases are explained on the hypothesis that supernumerary bodies were present.

Symptoms.—The invasion is generally so gradual that the identity of the disease is not at first apparent. Increasing weakness is usually the first manifestation, or the three principal symptoms—weakness,

Prophylaxis demands a more restricted use of the so-called substitutes for mother's milk. When these must be used, a food prepared with fresh milk and permitting the addition of beef-juice should be selected. Overheating the milk is also injurious.

STATUS LYMPHATICUS.

LYMPHATISM.

Definition.—A rare affection of childhood and youth characterized by hyperplasia of the lymphatic glands and tissues throughout the body, the spleen, thymus, and lymphoid bone-marrow.

Etiology.—The cause is unknown. The disease is often associated with rickets and with hypoplasia of the heart and aorta, and in quite a number of instances its presence has been revealed after sudden death.

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Symptoms.—The invasion is generally so gradual that the identity of the disease is not at first apparent. Increasing weakness is usually the first manifestation, or the three principal symptoms—weakness,

gastrointestinal disturbance, and pigmentation of the skin—may develop simultaneously. Sometimes the discoloration of the skin is the first indication. Cases running an acute course with all the symptoms prominent have been observed.

Pigmentation.—The discoloration of the skin progresses slowly, as a rule, and affects most markedly those regions in which pigment is normally most abundant, as the face, backs of the hands, axillæ, the mammary areolæ, abdomen, groins, the genitals, and regions where the skin has been compressed or irritated by apparel. The color varies from a light yellow to a dark brown, olive, or black. The mucous membranes most affected are the lips, mouth, conjunctivæ, and vagina. The patches have often a bluish color. Pigmentation of the mucous membranes is not, however, distinctive of this disease. Small white patches (leucoderma) are occasionally seen at different points and the palms and soles remain unpigmented. Points of deeper pigmentation, resembling moles, are sometimes scattered over the surface. Pigmentation is occasionally absent, however, throughout the disease.

The gastrointestinal symptoms are often prominent, but they may be absent. Loss of appetite, nausea, and vomiting may set in early and persist at intervals throughout the disease. Diarrhea often develops without apparent cause. Late in the disease the abdomen sometimes becomes painful and retracted. Distinct crises of severe neuralgic pain in the epigastrium or hypochondriac region are occasionally observed.

Asthenia is one of the most distinctive features and often one of the earliest. It is characterized by a progressive loss of physical and mental vigor, lassitude, and a constant feeling of fatigue without exertion. Dyspnea and palpitation or periodical attacks of extreme cardiac weakness are common. Headache, vertigo, and faintness develop later, and the disease may terminate fatally in an attack of syncope, or less commonly in convulsions or delirium. Death may result, however, from a gradually deepening asthenia. Notwithstanding the pronounced evidences of weakness, the physical condition of the patient often remains good, the muscles large and firm.

Diagnosis.—The recognition of Addison's disease depends upon the association of cutaneous pigmentation with pronounced asthenia and gastric irritability, and not upon any one feature of the disease. Error is most likely to arise from a too hasty assumption that an abnormal pigmentation is due to suprarenal disease. In arriving at a diagnosis it is necessary to exclude many conditions; among them, normal excessive pigmentation, increase due to pregnancy, arteriosclerosis, chronic valvular disease of the heart, chronic passive hyperemia of the liver, malignant disease of the pancreas, tubercular peritonitis, melanosarcoma, exophthalmic goiter, scleroderma, and the pigmentation arising from pediculosis or the prolonged ingestion of arsenic or silver. Since the disease is tubercular in a majority of instances, reaction to the tuberculin test may be obtained.

Prognosis.—The course of the disease is generally chronic, but a fatal termination may be expected in most cases within a year after its recognition. Cases have rarely lasted five or even ten years, and a few instances of recovery have been reported. Temporary remissions are occasionally observed.

Treatment.—The treatment is chiefly palliative. The irritability of the stomach may interfere with the administration of tonics for the relief of the asthenia. Dilute hydrocyanic acid, creosot, cerium oxalate, champagne and ice may relieve it. Bismuth and salol are indicated for the diarrhea. Iron and arsenic are sometimes of benefit in anemic cases. The diet must be regulated to suit the digestion and with a view of avoiding its derangement. The administration of the suprarenal extract has been followed with excellent results and apparent cure in some cases, but by complete failure in others, the result probably depending upon differences in the anatomical lesion. The dried extract may be given in doses of gr. j (0.06) three times a day, or the fresh gland may be administered, raw or partially cooked. A glycerin extract is also employed.

Other Diseases of the Suprarenal Bodies.—The adrenals are occasionally the seat of malignant, tubercular, adenomatous, or cystic disease, and they not infrequently show cloudy swelling or other degeneration in connection with the acute infectious diseases. The symptoms produced are not sufficiently distinctive to admit of diagnosis, however, and they are of interest chiefly to the pathologist.

DISEASES OF THE SPLEEN.

MOVABLE SPLEEN.

Floating Spleen, Wandering Spleen, Splenoptosis.

Etiology.—Abnormal mobility of the spleen is usually associated with enteroptosis. It is therefore due, for the most part, to the same causes, namely, a congenital weakness of attachment and subsequent pressure, as by tight-lacing or injury, the dragging of a tumor, or dilatation of the stomach.

Symptoms.—The displacement is sometimes discovered accidentally during examination for other conditions. The lower border may be as low as the brim of the pelvis in extreme cases. The normal area of dullness is then absent. Sometimes there is pain or a sense of dragging in the splenic region or in the side and back. It is generally distinguishable by its shape. The ureter, bladder, or bowel may be compressed, and the splenic vessels may be twisted. Fever, pain, and swelling are then produced.

Treatment.—The organ can generally be replaced by taxis, unless adhesions have formed. An abdominal binder should then be worn. Successful removal of the dislocated spleen has been performed, and Halsted has relieved the condition by the formation of artificial adhesions.

RUPTURE OF THE SPLEEN.

Etiology.—Rupture may occur spontaneously or as a result of trauma when the organ is intensely hyperemic, as in some cases of malaria, typhoid fever, or other conditions to which reference has been made elsewhere.

The ***symptoms*** are those of internal hemorrhage, with severe pain and collapse, which rapidly proves fatal unless promptly submitted to surgical treatment. Immediate operation offers the only possible means of arresting the hemorrhage.

ACUTE SPLENITIS.

Etiology.—No distinct line can be drawn between the intense hyperemia often associated with the acute infectious diseases, and acute splenitis. The latter condition may, however, result from injury or as an extension of inflammation from adjacent organs. Instances of perisplenitis, in which the capsule alone is involved, are occasionally met with.

Symptoms.—Pain and tenderness are present, especially when the capsule is chiefly involved, and the organ is more or less enlarged.

Treatment is seldom directed to the splenic condition, but rather to the underyling disease. Local applications should be employed for the relief of pain.

CHRONIC SPLENITIS.

This affection usually assumes the form of a chronic induration. The organ is greatly hypertrophied, and the section shows pigmentation, sometimes in successive layers. A localized form is seen also around old hemorrhagic infarcts, abscesses, and foreign bodies.

Symptoms.—These are usually limited to a feeling of weight and oppression due to the enlargement, or disturbances of the bowel, or shortness of breath as a result of upward pressure.

Treatment.—As the condition is generally due to malaria, syphilis, or leukemia, the treatment must be directed to these conditions. Excision of the enlarged spleen has been successfully performed.

INFARCTION OF THE SPLEEN.

Infarction arises from the plugging of one or more branches of the splenic artery by emboli, which may be either simple or infectious. The former generally arise from the vegetations of endocarditis or from the interior of an aneurism; the latter from malignant endocarditis, pyemia, or other severe infectious condition, especially typhoid fever. Thrombosis of the splenic vein is a possible cause.

The ***symptoms*** are seldom diagnostic. The presence of the condition may be inferred, however, when severe pain in the spleen is associated with chill, rapid elevation of temperature, vomiting, and enlargement of the organ.

ABSCESS OF THE SPLEEN.

Abscess generally results from infarction, but it may arise directly from infection by septic emboli, or rarely from rupture of a gastric ulcer, or trauma. The abscess may be minute, or so large as to convert the entire organ into a pus-sac. The abscess may rupture into the

colon, into the peritoneal cavity, or upward through the diaphragm. Fatal peritonitis follows rupture into the peritoneum. The treatment is surgical.

SPLENOMEGALY.

Splenic Anemia, Splenic Pseudoleukemia.

Definition.—A form of anemia attended with great enlargement of the spleen, without other pathological lesions.

Etiology.—The condition is now looked upon as an independent affection, but until recently it was regarded as a splenic form of Hodgkin's disease. No definite cause has been determined.

Morbid Anatomy.—The organ is enormously enlarged, smooth, firm, and deeply notched. The capsule is adherent. The histological appearances are differently described. Some writers refer to sclerosis and atrophy of the Malpighian corpuscles; others, notably Bovaird, to a proliferation of endothelial cells, not only in the spleen, but in the liver and lymphatic glands. The condition has been found in connection with hepatic cirrhosis, tuberculosis, typhoid fever, and other infections. A varicose condition of the esophageal veins has been noted in some cases, and this condition was doubtless the source of profuse hematemesis and melena in other cases. Ascites may occur independently of the condition of the liver. The anemia is usually moderate, the red corpuscles seldom falling below 3,000,000. The hemoglobin is reduced, possibly to 50 per cent, and the leucocytes may be normal or decreased. The evidences of anemia are sometimes altogether absent.

The ***diagnosis*** is based upon the large size of the spleen and the absence of blood conditions characteristic of leukemia, pseudoleukemia, or malaria, the hemorrhages of purpura, the history of syphilis, or the bone lesions of rickets.

TUMORS OF THE SPLEEN.

Sarcoma and carcinoma are occasionally found in the spleen as secondary growths, the former more frequently than the latter. Tubercles and gummata sometimes occur. The local disease is seldom of clinical importance, however, owing to the greater prominence of lesions in other organs.

The echinococcus cyst is sometimes found and must be differentiated from abscess. It is usually recognized by its slow growth, the absence of chills, fever, and leucocytosis, and by the character of the fluid withdrawn by aspiration. Hooklets may be found.

Amyloid spleen (lardaceous, sago, or waxy spleen) is marked by enormous enlargement in some cases, but the condition is usually evident from the presence of amyloid degeneration in other organs, the history of prolonged suppuration, and the typical appearance of the patient.

DISEASES OF THE THYROID GLAND.

THYROIDITIS.

Definition.—An acute inflammation of the thyroid gland, often terminating in suppuration.

Etiology.—The affection generally follows one of the acute infectious diseases, as typhoid fever, rheumatism, smallpox, or malaria, or it may develop in a gland already the seat of goiter. Traumatic cases are occasionally met with. Thyroid abscess may result from metastasis or from hemorrhage into a goiter. Pregnant women and those suffering from suppression of menstruation are more susceptible to it. Inflammation originating in a gland that is already diseased is sometimes termed strumitis.

Morbid Anatomy.—The gland is enlarged and softened. One or many abscesses may be present, affecting one lobe or the entire gland. The blood-vessels are often distended with thrombi; hemorrhages and necrotic foci are usually found. The abscesses not infrequently burrow along the larynx, trachea, or esophagus, and they may perforate either tube or cause erosion of the cartilages and necrosis of the soft tissues.

Symptoms.—There are the usual indications of inflammation—swelling, pain, and tenderness—over one orboth lobes of the gland. As suppuration becomes established, fever develops. Pressure on the vessels of the neck sometimes causes headache, vertigo, and cyanosis. The trachea may be compressed, even to the extent of a fatal strangulation.

Diagnosis.—Perichondritis of the larynx, the only condition likely to cause error, produces swelling above the thyroid region, not in it, and it is attended with greater difficulty in phonation.

Treatment.—The treatment is surgical, embracing the early evacuation of the pus and drainage. Tracheotomy may become necessary when the trachea is compressed.

GOITER.

Bronchocele.

Definition.—Chronic enlargement of the thyroid gland.

Etiology.—The disease occurs either sporadically or endemically. Sporadic cases are not uncommon in many localities of our own country, as in New England and Michigan. It is endemic in the mountainous regions of Europe, particularly in the Alps and Pyrenees, and in some parts of Asia, South America, and Mexico. An infectious influence is suggested by its occasional epidemic prevalence in these districts. The disease has been attributed to an excess of lime in the drinking-water of certain localities. Women are more frequently affected than men and usually in early adult life. It is occasionally observed in girls at the age of puberty.

Morbid Anatomy.—The anatomical lesions correspond more or less closely to the following classification: (1) Parenchymatous or hyperplastic, in which the gland becomes generally enlarged, the follicles proliferated and filled with a colloid substance; (2) vascular, in which there is marked dilatation of the blood-vessels, without hyperplasia of the gland-tissue; (3) cystic, in which the normal structure is replaced by one or more large cysts filled with colloid, amyloid, or hemorrhagic matter or the débris of fatty or other degeneration. (4) To these may be added an interstitial form in which the proliferation of connective

tissue is the chief feature. Calcareous infiltration sometimes ensues upon the other changes, particularly in the cystic and interstitial forms.

Symptoms.—The enlargement may be uniform or it may be more or less limited to one lobe, especially the right. In most cases no symptoms are produced further than the inconvenience occasioned by the tumor, which may remain small, but often attains enormous size, especially in the endemic form. Pulsation is distinctly felt and a systolic murmur may be heard over the vascular form, and fluctuation in the cystic, but the interstitial is firm and smooth. Dyspnea or aphonia may be produced by compression of the trachea or larynx in extreme cases, and the gland has been known to compress the veins behind the upper margin of the sternum, causing cerebral anemia and sometimes convulsions. The tumor generally ascends with the larynx during deglutition. The growth is slow and painless. Spontaneous recovery sometimes occurs, but sudden death has been observed as a result of hemorrhage, compression of the pneumogastric nerves, or from some undiscoverable cause.

Diagnosis.—Simple goiter is distinguishable from the exophthalmic by the absence of exophthalmos, tachycardia, tremor, and other evidences of constitutional intoxication. Abscess is excluded by the absence of pain, tenderness, and fever, and other tumors of the neck by the localization in the gland, its uniform surface, and the upward movement in deglutition.

The ***prognosis*** is ordinarily good with reference to life, but the possibility of sudden death adds gravity to the disease.

Treatment.—Medicinal treatment is generally unsatisfactory, except in the most recent cases. Potassium iodid sometimes effects a reduction of size in the interstitial form. Belladonna and ergot are useful in the vascular form, and arsenic promptly arrests the growth in young girls, as a rule. Electrolysis has also effected a cure in early cases. The eating of the thymus glands of sheep has been reported as curative. Inunctions of ointments containing iodin, mercuric biniodid, or lead iodid are beneficial.

EXOPHTHALMIC GOITER.

PARRY'S DISEASE, GRAVES'S DISEASE, BASEDOW'S DISEASE.

Definition.—A disease the principal features of which are exophthalmos, hypertrophy of the thyroid gland, rapid action of the heart, and tremor.

Etiology.—The disease may develop at any time of life and in either sex, but it is much more common in women between 20 and 30. A hereditary influence is sometimes apparent, and several cases may occur in the same neurotic family. A form of the affection has been developed during pregnancy, but pregnancy has no influence upon the previously developed disease. Emotional disturbances, as fright and worry, severe mental strain, or an acute infectious disease has repeatedly preceded the appearance of the disease. Many cases develop without recognizable exciting cause.

The principal theories of the etiology are: (*a*) That the disease is a pure neurosis; (*b*) that it is due to lesions in the central nervous system, the medulla oblongata, or the sympathetic system; and (*c*) that it is due to disease of the thyroid gland, a superactivity (hyperthyrea), the reverse of myxedema (athyrea). While all of these theories are supported in a measure by occasional anatomical findings, or by special features of the disease, the last is borne out both by changes in the gland and by experimental evidence obtained from the administration of thyroid extract. This drug in excessive dose produces rapidity of the heart's action, headache, tremor, and prostration analogous to those of the disease. Exophthalmos was observed in one instance after an overdose.

Morbid Anatomy.—The changes in the gland are those of an active hyperplasia, with proliferation of tubular spaces from the acini, often accompanied with proliferation of the epithelium to such an extent as to produce the appearance of a villous formation. The changes are compared by Berkeley to the involution of the mammary gland of a nursing mother. The cylindrical epithelium is often fatty, and the interior of the follicles is filled with a pale colloid material, supporting the theory of a hypersecretion. Changes have been discovered in the medulla and other portions of the nervous system in a few cases. Persistence of the thymus gland is observed in most instances.

Symptoms.—The disease is usually chronic. An acute form is occasionally met with, however, in which the onset is rapid, and in a few instances a fatal termination has occurred within three or four days. In such cases the heart's action becomes rapid and the arteries throb violently, uncontrollable vomiting and diarrhea set in, the eyes become prominent, the thyroid gland large, and in some cases delirium supervenes.

In the chronic form the invasion is insidious, several months or even years elapsing in some cases before the symptoms become fully developed. The cardinal symptoms are tachycardia, tremor, exophthalmos, and enlargement of the thyroid gland. They do not always appear in this order.

1. *Tachycardia.*—Rapidity of the heart's action is generally the earliest manifestation. The pulse may not at first exceed 100, but after the disease has become fully developed it often reaches 160 or 180, and may run higher under excitement. It is usually remarkably regular. The area of cardiac dullness is increased, especially late, when there may be hypertrophy and dilatation, and the impulse is strong and heaving. The larger arteries throb, and the expansile pulsation of the thyroid gland has been mistaken for aneurism. Capillary pulsation is distinctly visible through the finger-nails, and a pulsation may be transmitted to the veins in the backs of the hands. A loud systolic murmur may be heard at the apex, and various bruits at the base and over the carotid and femoral arteries.

2. *Tremor.*—A fine involuntary, muscular tremor with about eight vibrations in the second is often one of the earliest symptoms.

3. *Exophthalmos.*—Protrusion of the eyeballs may precede or follow the appearance of tremor. It may be unilateral. It is not always a prominent manifestation, but it may be so extreme that the globe is

dislocated from the orbit. In such cases the eyes are occasionally destroyed by panophthalmitis. It is generally immediately recognizable, owing to the inability of the lids to completely cover the conjunctivæ. When the protrusion is moderate, it can be determined in some cases by Graefe's sign: When the patient suddenly lowers the eyes, the upper lids move downward slowly, leaving the corneæ for a moment exposed.

4. *Enlargement of the thyroid* is rarely extreme, and it may vary to a considerable extent from time to time. It may affect both lobes or one more markedly or exclusively, especially the right. A distinct pulsation and thrill are generally felt, and a distinct murmur, sometimes double, or the *bruit de diable* may be heard on auscultation over it.

Other symptoms are usually observed, especially anemia, progressive emaciation, and weakness, sometimes fever. Great nervous irritability is not uncommon, and melancholia may ensue. The patient may become neurasthenic, despondent, rarely maniacal. Paroxysms of uncontrollable temper, with palpitation, dyspnea, rapid breathing, and violent hysterical manifestations sometimes occur. Sensations of cold and heat, profuse sweating, attacks of urticaria or pruritus, are not uncommon. Limited patches of cutaneous hyperesthesia are sometimes noted. The skin may become pigmented as in Addison's disease, or areas of leucoderma and localized edema may be observed. The electrical resistance of the skin is diminished (Charcot); the expansion of the chest is reduced (Bryson). Glycosuria and albuminuria often develop, and true diabetes has been observed. The course of the disease is very variable. Spontaneous recovery or death may occur within a period of a few days or several months, but many cases last for several years.

Diagnosis.—The disease is readily recognizable after it is fully developed, but obscure cases are sometimes encountered, as when the symptoms on the part of the stomach predominate, and before thyroid enlargement and exophthalmos have developed. Tachycardia and arterial and capillary throbbing can generally be found on examination.

Treatment.—The most essential part of the treatment in most cases is a complete change of environment and in the mode of life. The earlier these can be accomplished, the greater is the possibility of recovery. Rest of body and mind is essential; all worry and excitement must be removed. In severe cases it is better to confine the patient to bed for a time, especially after an acute exacerbation. An ice-bag often quiets the action of the heart. It should be worn constantly, over the heart or over the manubrium sterni and lower part of the neck, as advised by Osler. Prolonged galvanization, with the anode over the cervical spine and the cathode over the peripheral nerves, has proved beneficial. Drugs are for the most part unreliable, but in conjunction with rest they are often of great service. Aconite in full doses is of great benefit in some cases, and its action is aided by large doses of the bromids, ʒ ij (7.70) being given in a day. Musser has found small doses of opium curative. Digitalis quiets the heart's action and reduces the thyroid in some instances. Belladonna, strophanthus, and veratrum viride have been employed with apparent benefit by some writers, but they cannot be depended upon, and prove injurious in some cases. Arsenic and iron are indicated for the anemia. Thyroid extract is harmful and may produce alarming symptoms. Surgical treatment, embracing the removal of one

lobe, and other methods have been tried, but there is great danger of death from the anesthesia. Division of the cords of the cervical sympathetic has recently proved successful.

MYXEDEMA.

ATHYREA.

Definition.—A disorder of nutrition due to atrophy and arrest of the function of the thyroid gland, and characterized by myxomatous infiltration of the subcutaneous tissues, with dry desquamation of the skin and mental failure. Three forms of the disease are recognized: (1) Myxedema proper, (2) cretinism, and (3) operative myxedema (cachexia strumipriva).

Etiology.—Cretinism may be a congenital condition, or it may develop at any time before puberty. It is due to the absence or loss of the function of the thyroid gland. The causes of this functional arrest are not usually recognizable, but in one instance at least it followed destruction of the gland by actinomycosis. The gland is sometimes congenitally absent, and it may undergo atrophy after one of the acute infectious diseases. A transitory form of the affection has followed exophthalmic goiter. Women are more frequently affected; hereditary transmission through the mother has been observed, and several cases not infrequently occur in the same family. The disease is much less common in this country than in some districts of Europe.

Symptoms.—*Myxedema.*—The chief manifestations of true myxedema are seen in the integument and in the nervous system. Owing to the infiltration of the subcutaneous tissues, the facial expression is lost; the face appears broad and expressionless. Flattened, tumor-like masses sometimes form on the sides of the head and elsewhere. The face has usually an edematous appearance, a puffiness like that of acute nephritis. The lips and alæ of the nose are much thickened. The skin of the entire body becomes similarly affected, the surface dry and scaly. The hair is more or less completely lost from all parts of the body. The bony frame is not enlarged, as it is in acromegaly, but the soft parts are much increased. The hands and feet are broad. Large accumulations are often found in the supraclavicular regions. The movements of the body are slow, and the action of the mind is equally sluggish. The speech is slow and drawling. Headache often develops, and the patient becomes irritable. In severe cases delusions and hallucinations appear, and they may lead to a fatal dementia. The functions of the vital organs are generally unaffected, but glycosuria is not uncommon, and albuminuria sometimes develops. The temperature is normal or subnormal; the surface temperature is low. Death is usually a result of tuberculosis or other intercurrent malady. The disease may last ten or fifteen years, but an early fatal termination has been repeatedly noted.

Cretinism.—The endemic form is usually congenital; the sporadic form appears after the first year, as a rule. They are the same in nature. With the appearance of the disease, the development of the child is arrested, and the infantile appearance is often retained for many years. The growth of the body is very slow. The expression is heavy, owing to the thickening of the subcutaneous layer, as in true myxedema. The

body is often much deformed by the excessive enlargement of certain regions. The face is extremely puffy, so that it is large in comparison to other parts of the body. The tongue is thick and hangs out of the mouth. The legs remain short and become thick and stocky; the hands and feet are poorly developed. The face is pale and waxy; the fontanels remain open. The muscles are so weak that the child cannot sit or stand. The intellect remains undeveloped; the child is an idiot when the condition is congenital, or an imbecile when it appears later. Congenital cases are rare, and generally terminate fatally within the first two years.

Operative myxedema develops, as a rule, only after total extirpation of the thyroid gland, and not then in case supernumerary glands should be present. But in a few instances it has developed after partial removal. Comparatively few cases of this character have been observed in man.

Diagnosis.—The pallor and puffiness of the face may be mistaken for the edema of parenchymatous nephritis, and the diagnosis is often supported by the presence of albumin and casts in the urine. But the puffy swellings do not pit, as in edema; and the dryness and scaliness of the skin, the ragged alopecia, and the dullness of the intellect usually remove all doubt.

Treatment.—The thyroid extract or the powdered gland of the sheep is a specific remedy. It should be given in small doses at the start, but rapidly increased until gr. v to x (0.30—0.60) are given three times a day. Children can take half the adult dose, as a rule. The remedy must not be discontinued too soon. Its action is generally rapid and astonishing, but relapse follows its discontinuance in most cases. Some patients find it necessary to take an occasional dose during the remainder of life.

Tumors of the Thyroid Gland.—Adenomata, fibromata, cysts, and malignant growths are occasionally met with. The lesions of tuberculosis, syphilis, actinomycosis, and hydatids are sometimes found.

DISEASES OF THE THYMUS GLAND.

The thymus gland normally shrinks to a small, probably functionless remnant between the fifteenth and twentieth years. Comparatively little positive knowledge exists regarding the gland and its diseases. It is occasionally found enlarged after sudden death or after various diseases, but the relation of the morbid condition to the fatal issue is largely a matter of theory. Pressure of the enlarged gland upon the trachea is undoubtedly an occasional cause of asthma in children (asthma thymicum) and of laryngismus stridulus, by some authors regarded as identical, but it is by no means the only cause of these conditions. The sudden death of infants must sometimes be attributed to thymus enlargement. In some cases the pressure is probably exerted, not upon the trachea, but upon the blood-vessels or the pneumogastric nerve. The sudden death of adults while bathing or during anesthesia is believed, in some instances, to be due to this cause. The persistence of the thymus gland in exophthalmic goiter is a fact not yet fully understood. The gland has been found enlarged also in some cases of epilepsy.

Abscesses and various malignant and benign tumors are occasionally found in the thymus gland.

SECTION IV.

Diseases of the Circulatory System and Mediastinum.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—Inflammation of the pericardium, arising from infection, trauma, or extension from diseases in adjacent structures. The disease is to be studied under the following heads: (1) Acute plastic or "dry" pericarditis, (2) pericarditis with serous effusion, (3) pericarditis with purulent or hemorrhagic effusion, (4) chronic adhesive pericarditis, (5) tubercular pericarditis, and (6) cancerous pericarditis.

1. **Acute Plastic Pericarditis.** 2. **Pericarditis with Serous Effusion.**—***Etiology.***—The causes of these two forms are practically the same and may be advantageously considered together. The disease occurs at all periods of life, corresponding in a measure to the prevalence of the affections to which it bears a secondary relation. It is more frequent, however, in males. It assumes epidemic proportions only when it is associated with a widespread epidemic of an infectious disease, notably influenza or pneumonia.

(*a*) *Primary Pericarditis.*—Cases of so-called idiopathic or primary pericarditis are extremely few. They are generally met with in children in whom no evidence of previous local or constitutional illness has been recognized. It is quite probable, however, that many cases regarded as primary are in reality secondary to an unrecognized mild type of infection, or that they are tubercular in character.

(*b*) *Secondary Pericarditis.*—As a secondary affection, acute pericarditis occurs most frequently in the young and middle-aged. (1) Fully half the cases follow acute rheumatism, or they are associated with the acute tonsilitis of rheumatic subjects. The pericardial inflammation sometimes precedes the articular. (2) Less frequently, it follows other acute infections, particularly influenza, scarlet fever, or pneumonia. (3) It is not unusual after acute septic processes, as septicemia, pyemia, puerperal sepsis, malignant endocarditis, necrosis of bone, and it is not infrequently met with in the new-born infant as a result of septic infection through the navel. (4) In altered blood-states, especially gout and chronic interstitial nephritis, affecting, as a rule, individuals past 50 years of age, sometimes also in scurvy and diabetes. (5) It is often tubercular in character, occurring primarily in the general involvement of serous membranes or as a result of extension from the lungs or lymph-glands.

(*c*) As a result of direct extension of inflammation the disease is met with in pleuropneumonia, especially in children and alcoholic adults,

rarely in connection with simple pleurisy, but occasionally in suppurative myocarditis and aneurism of the aorta.

Morbid Anatomy.—The morbid process may be confined to a limited area or it may be general. In the beginning of the plastic form, the affected surface is inflamed and opaque, but smooth. A plastic exudate of variable thickness is soon thrown out, which gives it a roughened surface. When the plastic matter is abundant it is given a peculiar appearance by the movements of the heart. It is often compared to the shredded appearance of two buttered surfaces that have been forcibly separated; it is sometimes honeycombed or there may be long villous threads (the hairy heart of the ancients). There is usually a slight increase of the pericardial fluid and it may be clear or flocculent.

Pericarditis begins as a plastic inflammation with fibrinous exudate, usually most marked on the visceral layer about the base of the heart, near the origin of the great vessels. The quantity of serum that is poured out varies within wide limits, usually from 2 to 10 ounces (64.0 to 320.0), but sometimes exceeds 3 pints (1.5 liters). The fluid is sometimes slightly cloudy from fibrin, desquamated epithelium, granular detritus, or pus-corpuscles, and a few blood-cells may be found in it. As the disease subsides, the fluid becomes less and the fibrinous exudate undergoes organization, forming adhesions between the two layers of the pericardium. The myocardium is usually edematous and in some cases the inflammation extends to a variable depth into the muscular structure. The acute process sometimes passes into a chronic one without complete absorption of the serum.

Symptoms.—1. *Adhesive Pericarditis.*—Many mild cases of this type are overlooked on account of the absence of distinctive symptoms. It is only in severe cases that the subjective manifestations are sufficiently prominent to attract attention to the condition. The most important features of these cases are: (*a*) A sense of discomfort or constriction in the precordial region. Distinct pain is unusual, yet it is occasionally so severe as to resemble angina pectoris. It may be felt in the region of the heart or it may be referred to that of the xiphoid cartilage. (*b*) Palpitation with increased frequency and force of the heart's action are common in the early history of the disease, but after adhesions have formed or the myocardium has become affected the heart's action usually becomes weak. (*c*) Dyspnea is sometimes present. (*d*) Fever is generally observed, but in most cases it is a feature of the underlying infection.

2. *Pericarditis with Serous Effusion.*—Symptoms are often wanting also in the beginning of this form, but precordial pain or distress is more common than in the simple adhesive form. When, as rarely happens, the disease develops as a primary affection, there may be an initial chill with fever and acceleration of the heart and respiration; but when it is a secondary affection, these manifestations are obscured by the pre-existing disease. The fever is seldom high and it runs an irregular or intermittent course. Dyspnea develops with the formation of the effusion and constitutes one of the most significant symptoms of the condition. The patient lies on his left side, or he may be compelled to sit up in bed (orthopnea). The pulse becomes small as well as rapid, and irregular; or it may present the features of the pulsus paradoxus, becoming

extremely weak or imperceptible during inspiration. The embarrassment of the heart's action corresponds to the quantity of fluid present and the consequent pressure that must be overcome by the heart muscle in diastole. Other pressure symptoms are usually observed, as cough due to compression of the trachea, aphonia from compression of the recurrent laryngeal nerve, dysphagia from pressure on the esophagus, and distention of the veins of the neck. Nausea and vomiting may occur. The dyspnea is probably in part a result of pressure upon the left lung, especially when the pericardial effusion is excessive. The lips and fingernails are blue; cyanosis is often extreme. Such nervous symptoms as headache, restlessness, and insomnia are common, and in the later stages there may be mild delirium passing into stupor or coma. Melancholia with suicidal tendency has been noted in some cases. A type of delirium resembling that of alcoholism has been noted, even in cases that subsequently recovered. Chorea sometimes develops and epileptic seizures have been observed during paracentesis.

Physical Signs.—(1) *Adhesive Pericarditis.—Inspection* is usually negative. On palpation a friction fremitus caused by the rubbing of the roughened surfaces may often be felt, especially over the right ventricle, but it is often absent, even when the friction sound is distinctly heard.

Auscultation.—A double, to-and-fro friction sound is the most distinctive sign of acute pericarditis. Although it corresponds to the systolic and diastolic movements of the heart, the friction sound is usually slightly longer in duration than these sounds. A single friction sound is sometimes heard, and in some instances it has a distinctly triple character. The sound is dryer, harsher, and more grating or crackling than the endocardial murmur, and, like the pleural friction, it is often compared to the creaking of new leather. But it is sometimes soft and difficult of distinction. It is best heard, as a rule, at the left border of the sternum, in the fourth and fifth interspaces. Sometimes it can be heard over the apex or base, but it is not transmitted along the blood-vessels. One of the most characteristic features is the inconstancy of the sound from day to day. It may be heard at one examination and not at the next.

(2) *Pericarditis with Effusion.—Inspection.*—In children the precordial region often bulges when the effusion is large, and the left side of the chest may appear slightly enlarged, but the expansion is often markedly diminished. The apex beat cannot be seen. The diaphragm and the left lobe of the liver are often depressed and the epigastrium becomes prominent. The skin of the precordium sometimes becomes edematous. The integument and mucous membranes are pale and more or less cyanotic; the veins of the neck are distended and often show undulatory movements or distinct pulsation. The expression is anxious. The respiratory movements are rapid and often irregular.

Palpation.—The apex beat, when it can be recognized, is displaced upward and to the left, but exceptions to the rule are noted. When the effusion is abundant the impulse becomes imperceptible, and the cardiac shock is lost. Sometimes these impulses can be restored by having the patient lie on the left side or by inclining the body forward. The friction fremitus is generally lost, but it may be present at the base in large effusions. The impulse is sometimes retained when the heart is

hypertrophied or bound to the chest-wall by old adhesions. Fluctuation can very rarely or never be recognized.

Percussion is negative in adhesive pericarditis, but when effusion is present the area of dullness is greatly increased. This dull (flat) area has a characteristic outline with the patient in a sitting posture, being irregularly triangular with the base downward. This is one of the most positive signs of the condition. The normal resonance in the right fifth intercostal space, the so-called cardiohepatic angle, is also obliterated. An area of dullness is sometimes found also in the left infrascapular region. A dull tympanitic note is elicited over the portion of the left lung that is compressed.

Auscultation.—The friction sound sometimes persists, especially at the base, occasionally in a limited area about the apex; but it disappears, as a rule, when the effusion has become copious. Later, when the fluid undergoes absorption, the sound returns for a time. The heart-sounds become indistinct and distant with the increase of effusion, but the second sound may persist at the base. The respiratory murmur over the anterolateral region of the left lung, the part compressed by the distended pericardium, becomes bronchovesicular in quality. This area changes, however, when the patient assumes a different posture.

The course of the disease corresponds to the pathological condition, representing the three stages of dryness, effusion, and absorption. The duration of each is exceedingly variable in different cases, depending largely upon the cause of the affection. The disease sometimes runs a rapid course. The effusion may reach its height within two or three days, and undergo complete absorption within an almost equally short time. But it not infrequently progresses slowly through each stage, exhibiting the features of a chronic condition; in some acute cases, too, the absorption of the fluid is slow and the disease may become chronic or a purulent pericarditis may be set up. Adhesions always remain after recovery that is complete in other respects. When associated with rheumatism the disease seldom lasts more than two weeks. With the absorption of the effusion the other symptoms gradually disappear. The temperature falls by lysis, the dyspnea subsides, and the pulse becomes slow, full, and regular. If, on the other hand, the disease pursues an unfavorable course, the temperature generally rises, the dyspnea becomes extreme, the patient becomes restless, delirious, and finally comatose. When the associated myocarditis is extensive, death sometimes occurs rather suddenly in syncope.

3. **Purulent Pericarditis** (Empyema of the Pericardium).—This may follow a serofibrinous pericarditis of variable duration, or the exudate may have a purulent character from the beginning. This is true especially of tuberculous and septic cases and occasionally of cases due to the acute infections. Various micro-organisms have been found in the exudate, but for the most part those ordinarily associated with septic or tuberculous processes—the streptococcus, staphylococcus, pneumococcus, and the bacillus tuberculosis.

Morbid Anatomy.—The pericardium is greatly thickened and covered with a thick layer of fibrinous exudate, as in the adhesive form. But in addition to this, the membrane is infiltrated with fibrin and pus. Its surface is opaque, granular, and often necrotic in patches. A myocar-

ditis is usually present, or a fatty degeneration of the myocardium may be found. The quantity of pus varies from a few ounces to four pints (2 liters). Absorption of the fluid sometimes occurs, leaving a portion of the pus as a caseous mass. Subsequent calcification of the pericardium may occur.

Symptoms.—The clinical manifestations are much the same as those of pericarditis with serous effusion, but an initial rigor is more common and the chill is often repeated. The temperature runs a course indicative of suppuration, and the prostration is more profound. The disease progresses rapidly and almost always terminates fatally.

Hemorrhagic Pericarditis.—The serous effusion of a nonpurulent pericarditis is sometimes more or less distinctly tinged with blood, especially in cases associated with chronic nephritis and those occurring in extremely old persons. A typical hemorrhagic pericarditis is generally associated with the purulent form of the disease, and more particularly when this is due to tuberculosis.

Diagnosis of Pericarditis.—The frequency with which pericarditis is revealed upon the post-mortem table in cases in which it had not been suspected indicates the importance of daily examinations of the heart during the course of the acute infections, particularly rheumatism. A complaint of precordial distress or of dyspnea, or an increase of fever without aggravation of the articular inflammation during the course of rheumatism should arouse suspicion of the disease. The diagnostic sign of a pericarditis is a friction sound. This may, however, disappear after the development of effusion. Pericarditis with effusion is readily recognized in most cases, providing the heart has been previously examined, but in other cases it is sometimes determined with great difficulty. The chief source of confusion usually lies in the exclusion of a cardiac dilatation; but in this there is generally a history of chronic valvular disease, and fever, pain, and nervous manifestations are absent. The apex beat and cardiac impulse are present, often wavy in character and forcible. The area of dullness is not triangular and it does not extend so high along the left margin of the sternum, except in mitral stenosis, or so low in the fifth and sixth intercostal spaces, and it does not change with the position of the patient. There is no dull tympany, as in pericarditis, except, perhaps, in the most extreme cases of dilatation. The first sound of the heart is not lost, and instead of the friction sound one hears an endocardial murmur.

Pleuritic Effusion.—A pericardial effusion is probably oftener mistaken for a pleuritic than the reverse, in cases of excessive accumulation. The absence or feebleness of the heart sounds and the dull tympanitic note in the infrascapular region are the most distinctive features of the pericardial effusion, when the friction sound is absent; but an encysted pleuritic effusion in the anterolateral region of the chest is next to impossible of differentiation in some cases.

The distinction between a serous and a purulent effusion cannot always be determined without aspiration, a measure that is resorted to only when it is required by the condition of the patient. The character of the effusion may be inferred, however, from the associated disease. Effusion of rheumatic origin is generally serous, that of tuberculosis or septic disease is often purulent. Chills, fever, and sweat-

ing supervening upon an effusion of considerable duration point to the presence of pus.

Prognosis.—Simple adhesive pericarditis and pericarditis with serous effusion are generally followed by recovery, especially in rheumatic cases, but a purulent effusion is almost always fatal. Good results have followed surgical treatment of purulent effusion, however, in nearly 50 per cent of recent cases. When septic infection manifests itself, a fatal issue may be expected. Recovery rarely occurs in the tuberculous form of the disease.

Treatment.—From the very beginning the patient should be confined to bed and given absolute rest, physically and mentally. All excitement, especially that occasioned by visitors, should be guarded against. The object of quiet is to lessen the heart's action and thus prevent one of the most potent factors in the production of effusion. Drugs are of doubtful utility except when the pain is so excessive as to call for the administration of morphin. There is seldom, if ever, any indication for the use of digitalis, except for its diuretic action after the effusion has become profuse. Aconite is serviceable for quieting the heart, but it is not devoid of danger when the myocardium has become involved. Local applications are sometimes of great benefit; either the local abstraction of blood by cupping or leeches in robust individuals, as favored by Osler, or repeated blisters, as recommended by Pepper. Hot and cold applications are also useful. The ice-bag often affords relief from pain and quiets the heart's action. Leiter's coil or simple compresses may be substituted for it. Blisters are efficient, especially for promoting the absorption of fluid. In robust patients purgation by the administration of salts in concentrated solution is beneficial, but it should not be resorted to in asthenic cases. The action of the kidneys should be favored by the administration of potassium bitartrate or acetate or calomel and the infusion of digitalis. Potassium iodid is often of great service in effusion. It should be given in small doses at first and increased daily until gr. x (0.60) t. i. d. are taken. As soon as the effusion becomes so extensive as to cause serious dyspnea or other pronounced pressure symptoms, the fluid should be withdrawn either by aspiration or an incision. Aspiration is usually sufficient in serous effusion, but free incision and drainage are required when it is purulent. The puncture is usually made in the fourth intercostal space, near the left margin of the sternum, or an inch (2.5 mm.) from it; or in the fifth interspace, an inch and a half (4 mm.) from the sternum. The operation must be done under the strictest antisepsis and preferably by a skilled surgeon.

The diet of the patient should be wholly liquid during the prevalence of fever, and later it should be light and nutritious as in the convalescence from a febrile disease. Tonics are also indicated, especially strychin and arsenic or iron, to strengthen the heart and improve the condition of the blood. The patient should be cautioned against undue excitement and fatigue until convalescence has been fully established.

4. **Chronic Adhesive Pericarditis** (Adherent Pericardium).—***Etiology and Morbid Anatomy.***—Adherent pericardium is a common result of the acute forms of the disease. It may be partial or general. The pericardium is thick and the adhesions are firm. The cases may be separated into two groups: (1) Cases in which the pericardium and the epicardium

are united, and (2) those in which the condition is associated with chronic mediastinitis, the outer layer of the pericardium being firmly united with the pleura and chest-wall. The condition is described under the names pleuropericarditis, external pericarditis, and mediastinopericarditis. Simple adhesion of the pericardium with the epicardium is often discovered post mortem in patients who never gave a history of pericarditis. The more extensive adhesions often lead to extreme hypertrophy and dilatation of the heart, even in cases in which only a limited area of the pericardium is involved. The condition is often of tubercular origin.

Symptoms.—The clinical manifestations of the disease are generally a result of the interference with the free action of the heart caused by the adhesions. The condition is not usually recognized until marked hypertrophy has taken place. Cardiac insufficiency is later developed. In some cases the proliferative inflammation extends from the pericardium to the peritoneum and produces similar thickening of that membrane, with perihepatitis and perisplenitis. The patient usually suffers in the late stages of the disease from urgent dyspnea, and the cyanosis is often extreme after fatty degeneration and dilatation of the walls of the heart have become extensive. The heart movements, as recognized by inspection and palpation, are exceedingly irregular and there is sometimes a slight retraction or "dimpling" in the region of the apex in each systole. A pleural friction sound is often heard in addition to the pericardial, and often conceals the true condition from the examiner, unless the patient be instructed to hold his breath. The pulsus paradoxus may be present and the veins of the neck become distended during inspiration. Sudden death often occurs in this condition as a result of excitement or strong exertion.

5. **Tubercular pericarditis** is considered under the general heading of Tuberculosis, page 186.

6. **Cancerous Pericarditis.**—This term is commonly made to include both carcinomatous and sarcomatous disease of the pericardium. They are both rare affections and probably always secondary in their origin. In addition to the neoplastic tissue there is usually a serous or hemorrhagic effusion within the sac. The diagnosis is generally based upon the severity of the pain, the evidences of effusion, the cachexia, and the discovery of a primary growth elsewhere.

OTHER AFFECTIONS OF THE PERICARDIUM.

Hydropericardium (Dropsy of the Pericardium).—A noninflammatory accumulation of serum within the pericardial sac. ***Etiology.***—The condition is generally one of the features and one of the more serious manifestations of a general dropsy. It is most commonly a result of chronic nephritis, next most frequently of valvular heart disease, occasionally of pulmonary emphysema or hepatic cirrhosis. It is a common accompaniment of hydrothorax under the same etiological conditions.

The ***symptoms*** and ***physical signs*** are identical with those of pericarditis with effusion, but there is no friction, pain, or fever. The accumulation of fluid sometimes becomes extreme without producing marked dyspnea, but in other cases this is a prominent feature.

Chylopericardium, in which the accumulation has a white, milky appearance, has been met with in a few instances.

Hemopericardium.—This condition, in which the pericardium is more or less completely filled with blood, is generally a result of penetrating wounds, the rupture of an aneurism of the aorta, a rent in the heart-wall in advanced myocarditis, or the rupture of the coronary artery. The condition generally proves rapidly fatal, except in some cases of rupture of the heart in which life may be prolonged for a few hours or possibly for a few days. The symptoms are those of more or less rapid heart-failure and the signs of pericardial distention.

Pneumopericardium.—This is a rare condition in which the pericardial sac becomes inflated with air or gas. It is commonly a result of a penetrating wound of the chest-wall or a perforation of the esophagus, stomach, or lung into the pericardium, due to wound, ulcer, cancer, or tuberculosis. Gas may also be formed in a purulent exudate by the bacillus aerogenes capsulatus. A purulent pericarditis is promptly developed in cases in which it was not previously present. The ***symptoms*** are those of acute exudative pericarditis with rapid heart-failure. Percussion reveals a changeable area of dullness with tympany over the inflated area. On auscultation are heard churning and splashing sounds, sometimes friction, with feeble, distant heart-sounds. The ***diagnosis*** is generally revealed by the history of the case and the physical signs. The ***prognosis*** is grave. The case often terminates fatally within the first 48 hours.

The ***treatment*** is surgical, consisting of free incisions and drainage, or enlargement of the opening in a case of penetrating wound. While these measures are justifiable, they are seldom effectual.

DISEASES OF THE HEART.

ENDOCARDITIS.

Definition.—Inflammation of the endocardium. It may affect any portion of the lining membrane, but is usually confined to the valves. The process may be acute or chronic, and the acute is further subdivided into the simple or benign form, and the ulcerative or malignant.

SIMPLE ACUTE ENDOCARDITIS.

Etiology.—Acute endocarditis is met with at any period of life, but more frequently in children and young adults. It usually occurs as a complication of an acute infectious disease, particularly rheumatism, rarely, if ever, as a primary affection. It is probably of bacterial origin in all cases. Some previous lesion of the endocardium has always been regarded as necessary to the production of endocardial inflammation, a view based largely upon the fact that the endocardium of the right heart was sometimes found to be affected in the fetus, but the left side almost invariably in cases occurring after birth. The injury has, therefore, been attributed to the force of the blood-current. At the present time, only such impairment of the membrane is deemed to be essential as will permit the lodgment and growth of micro-organisms. It seems probable that several different organisms are capable of inducing the

disease, and that the endocardium is rendered susceptible to their action by the toxic state of the blood accompanying the acute infections with which it is associated. Acute rheumatism is the disease which above all others leads to its development. There is usually no relation between the endocarditis and the severity of the articular affection, for it often develops upon nonarticular cases. Tonsilitis in a rheumatic subject is sometimes complicated with endocarditis. Scarlet fever, measles, chickenpox, and other infections of children are sometimes followed by it. Pneumonia and less frequently typhoid fever, erysipelas, diphtheria, or smallpox may induce it, and the valves not infrequently become affected in such wasting diseases as diabetes, gout, tuberculosis, cancer, or chronic nephritis. A chronic endocarditis is liable to develop acute attacks, and the sclerotic valves resulting from chronic endocarditis are sometimes the seat of acute inflammation (acute recurrent endocarditis). The liability to such recurrence is increased during pregnancy and the puerperal state.

Morbid Anatomy.—*Vegetations*, granular or warty excrescences, varying in diameter from less than 1 to more than 4 mm., are formed upon the affected surface of the endocardium. They are most frequently found on the mitral valve, next most frequently on the aortic. They are situated just above the line of closure of the cusps or leaflets, on the auricular surface of the mitral valve and the ventricular surface of the aortic. The vegetations at first consist of leucocytes, blood-plates, and fibrin, but a proliferation of the endothelium and of the subendothelial connective tissue is shortly induced and gradually replaces the original vegetations. It is in this manner that the vegetations become "organized." A layer of fibrin and leucocytes is generally retained on the surface. The same process sometimes takes place on the mural endocardium or over the chordæ tendineæ. The vegetations are sometimes attached by a very narrow pedicle. In consequence of this loose attachment they are sometimes detached and carried away as emboli in the blood-stream, but not so frequently in the simple as in the ulcerative form of the disease. Very old vegetations sometimes become converted into firm connective tissue and are thus greatly reduced in size; their contraction often leads to deformity of the valves. They sometimes undergo subsequent softening or ulceration, owing to the impairment of nutrition occasioned by the sclerotic process. The bacteria which have been found in the vegetations or upon the surface are the streptococci, staphylococci, pneumococci, occasionally gonococci, and the bacilli of typhoid fever and influenza, the common colon bacillus, and several other organisms.

Symptoms.—Simple acute endocarditis often develops without producing either clinical manifestations or physical signs by which it can be recognized. Lesions of long standing are sometimes encountered after death in persons who manifested no symptoms during life. In the beginning there is often an elevation of temperature amounting to 102° or 103° F. (39.0°—39.5° C.), but it may be concealed by the fever belonging to the previous condition. Under these circumstances, however, there is generally a slight increase of the fever without recognizable aggravation of the original disease, and, therefore, suggesting the development of a complication. There is no pain, but the patient becomes

restless and the heart's action becomes accelerated and irregular, especially in mitral disease. The distinguishing feature of the disease is the presence of a murmur. This is at first little more than a roughening of the first sound. The character of the sound after it has developed into a distinct murmur, and the area of its greatest intensity, depend upon the valve affected and the manner in which it is affected. No greater mistake is made, and probably none more frequently, than in the assumption that every heart murmur indicates a present or previous endocarditis.

Diagnosis.—The diagnosis depends upon the sudden development of a heart murmur with irregular action in the presence of a recognized causative condition, as during the course of acute rheumatism. But when the valves are only slightly or not at all affected, there may be no murmur, and in any case the sound may be heard only at intervals. Anemic and other functional murmurs must be differentiated.

Prognosis.—The disease is not necessarily fatal, but it generally leaves the heart in an impaired condition. Each recurrence increases the liability to sclerosis, the extent of the sclerosis, the consequent deformity of the valves and their insufficiency. This in turn induces greater hypertrophy and hastens the approach of dilatation and incompetency. (See p. 335.) Many patients live to advanced age, however, after having passed through several attacks, and permanent recovery undoubtedly takes place in some cases.

Treatment.—The patient should be given completer est. Rest is important as a prophylactic measure during any illness which is liable to induce endocarditis; it is in fact the only means at our command for warding off the disease. The rapid, irregular action of the heart may often be quieted to some extent by the application of an ice-bag. Salt should not be added to the ice, for in careless hands the skin may be quickly frozen. When the disease is associated with rheumatism, the salicylates should be continued in full doses. Some writers advocate their use also in cases arising from other causes. The sulphocarbolates, ammonium carbonate, and alkalis in general are regarded as beneficial. The diet should be mostly liquid, but abundant, and stimulation is generally required. Strychnin in small doses should be given during convalescence. The patient should not be permitted to exert himself for several weeks after apparently complete recovery.

MALIGNANT OR ULCERATIVE ENDOCARDITIS.

Infectious, Septic, or Diphtheritic Endocarditis.

Etiology.—(*a*) It is now pretty generally admitted that malignant endocarditis may arise as a primary affection; (*b*) in a majority of cases, however, it develops secondarily, in a heart that has been previously affected with endocarditis, or it may follow immediately upon an attack of the acute form. In either instance it is due to the action of one of the pyogenic micro-organisms, especially the micrococcus pyogenes or the micrococcus lanceolatus. It is often closely related to a process of suppuration, as otitis media, gonorrhea, puerperal fever, but it occurs more frequently in connection with pneumonia, sometimes with erysipelas

or dysentery. In many cases the direct source of infection cannot be determined. It is not common in connection with rheumatism, nor is it encountered in cases of chorea. It is rare in measles, diphtheria, scarlet fever, smallpox, typhoid fever, and tuberculosis.

Morbid Anatomy.—In the beginning of the disease, vegetations are found upon the affected endocardium, as in simple endocarditis, but necrosis soon develops. The resultant loss of tissue causes a thinning and weakening of the valve and often leads to rents and perforations. Suppuration sometimes occurs within the vegetations, producing minute abscesses and increasing the liability to perforation or to the formation of the so-called aneurisms of the valves. Excessively large and long verrucose vegetations are occasionally found upon the valves without loss of substance. The necrotic process often invades also the mural endocardium, causing more or less complete perforation of the ventricular septum, errosion and sometimes rupture of the chordæ tendineæ, rarely of the heart-wall itself. Fragments of the vegetations are sometimes torn away and carried with the blood to produce one or many embolisms in remote parts, especially in the lungs, liver, intestines, spleen, kidneys, brain, or elsewhere. The lesions are commonly found in the mitral valves, next in the aortic, and almost as often in both, but rarely in those of the right heart. Among Osler's 209 cases, the disease was confined to the mitral valve in 77, to the aorta in 53, affecting both in 41; the tricuspid was affected in 19, the pulmonary in 15, and the heart walls in 33. Hemorrhagic pericarditis, petechial, erythematous eruptions, and general purpura are not uncommon complications. Purulent inflammation of the joints and general pyemia are possible results. The ulcerative process sometimes extends outward from the affected endocardium, along the intima of the valves, producing endarteritis, especially in the aorta.

The bacteria found in the lesions are the same as those in simple endocarditis, but the micrococcus lanceolatus and the streptococcus pyogenes are more constantly encountered.

Symptoms.—The clinical course of malignant endocarditis is exceedingly variable. Two definite types of manifestations are encountered, the one corresponding closely to those of sepsis, the other assuming a typhoid course, but many cases are more markedly intermittent in character. In some cases the manifestations of the local disease are prominent, while in others they are so slight as to be readily overlooked. The most striking features in all cases, however, are directly referable to septic infection.

The onset is usually sudden, with a distinct rigor, or, if the disease develops in the course of a febrile affection, there is a sharp increase of fever, often to 104° or 105° F. (40°—40.5° C.). The pulse becomes feeble, rapid, and irregular, often dicrotic. The subsequent temperature curve varies with the type of the symptoms in each case.

In the *septic type*, repeated chills occur, followed with profuse sweats, great prostration, and other symptoms of sepsis. The chills sometimes occur with a regularity that suggests quotidian or tertian intermittent fever. Heart symptoms of greater or less severity may be present, but very often they are so mild as to be entirely obscured by the general condition.

The *typhoid type* is more common. It is characterized by a more uniform temperature curve and greater nervous disturbance, headache, restless sleep, delirium finally passing into coma. More active cerebral symptoms sometimes appear, the case being readily mistaken for one of basilar or cerebro-spinal meningitis. Profuse sweats generally occur, and petechial, erythematous, and other cutaneous eruptions may appear. The tongue is heavily coated, sordes collect on the teeth, and abdominal distention, with diarrhea or constipation and vomiting—these are features of many cases. In this form, as in the septic, the endocarditis may be entirely overlooked; it may, in fact, be unrecognizable upon the most careful examination.

Embolisms.—The course of the disease may be suddenly changed by the development of embolism in one or more parts, with manifestations peculiar to the part affected. They are generally announced by a sudden localized pain corresponding to the location. A chill often announces the dissemination of the emboli. Following their lodgment there are evidences of inflammation, and, later, those pointing to the formation of an abscess. There is then, as a rule, tenderness on pressure, especially over the liver, spleen, or kidneys. Infarction or abscess of the lung or empyema may follow a pulmonary embolism; albuminuria, hematuria, and, later, pyuria, that of the kidney. Jaundice commonly accompanies other evidences of embolism of the liver.

Heart Symptoms.—The valvular lesions are all the more serious in their consequences because they are so suddenly developed. Congestion of the lungs is a constant result, a condition that is manifested in extreme dyspnea and marked cyanosis; edema of the lung may also be induced. These disturbances are, as a rule, more severe when the disease attacks a previously healthy endocardium than one that has been the seat of old valvular lesions, for in the latter condition the heart, already hypertrophied, is better able to overcome the circulatory derangement that is set up.

Diagnosis.—When the heart symptoms are prominent, the diagnosis is not difficult; in their absence, it may be extremely so. It is generally to be based upon the history of the previous affection, the sudden onset, with chill, high fever, sweating, and the characteristic physical signs, when present. The development of embolism throws additional light upon the case. The differentiation from a *simple endocarditis* is not difficult. In a majority of cases the symptoms resemble either general septicemia or typhoid fever. As the clinical manifestations are in reality septic there can be no differentiation between the condition and sepsis, in the absence of a history of previous valvular involvement or present cardiac symptoms.

The differential diagnosis lies between typhoid fever, malaria, and acute miliary tuberculosis.

Typhoid fever is to be excluded by the fact that the disease follows a pneumonia or other infectious disease, as well as by the sudden onset without prodromes, the rapid rise of temperature, early prostration, the irregular or intermittent course of the fever, the presence of cardiac symptoms, marked leucocytosis, and probably by the development of embolisms.

Intermittent fever and other forms of malaria are excluded by the

absence of the plasmodium from the blood, and usually, upon close observation, by the irregular periodicity of the chills and sweats.

Acute tuberculosis is attended with pulmonary symptoms, usually with enlargement of lymph-glands, and the bacillus tuberculosis may be found in the sputum. The difficulty of arriving at a differential diagnosis is greater when either of these affections occurs in the subject of a valvular lesion.

Prognosis.—The disease, when severe, always terminates fatally. The correctness of the diagnosis may generally be questioned when recovery occurs. A few undoubted recoveries have, nevertheless, been observed.

Treatment.—The treatment is that of other pyemic affections, with local applications to the precordial region when indicated. Perfect rest is essential. The strength of the patient should be maintained by a nutritious, mostly liquid diet and the regular and free administration of alcohol and strychnin. The salicylates and other alkalis are distinctly beneficial in some cases, and quinin in full doses in others. It is improbable, however, that any form of medication exerts much influence upon the morbid process. An ice-bag to the region of the heart may quiet its action and possibly to some extent reduce the inflammation in cases characterized by pronounced local disturbance.

CHRONIC ENDOCARDITIS.

Chronic Interstitial or Sclerotic Endocarditis, Chronic Valvular Disease of the Heart.

Etiology.—There are two groups of cases: (*a*) A majority of cases follow an acute endocarditis and are marked by rapid progress; and (*b*) cases which run a chronic course from the beginning. Going back a step further, the starting-point of the condition in fully half the cases is an attack of acute articular rheumatism, and, in a majority of the remaining cases, it is one of the other infectious diseases that have been referred to under the etiology of acute endocarditis. Like acute endocarditis, too, it is more frequent in young persons than in those beyond middle life. The disease more commonly attacks the mitral valve. It is probable also that many cases which appear to begin insidiously and to pursue a chronic course from the beginning, originate in some mild affection during which no involvement of the heart was recognized. In other instances, the slowly progressing sclerosis is induced by alcoholism, syphilis, chronic nephritis, gout, toxic substances in the blood, and sometimes, no doubt, by habitually excessive exercise. In a large group of cases the condition is part of a general arteriosclerosis.

Morbid Anatomy.—In this, as in the acute form of the disease, the primary lesion is in most cases the warty vegetation, but the sclerotic process reduces these excrescences to minute, hard nodules and they are often no longer discernible. The edges of the valves now appear opaque, yellowish gray, uniformly thickened, firm, and inelastic. In the aortic valve the sclerosis begins around the corpora Arantii; in the auriculoventricular valves, it begins just within the margin of the leaflets. The appearances are often very similar to those of arteriosclerosis in the aorta. As a later change, the valves become misshapen, corru-

gated, curled, and variously distorted. They may become shrunken into mere stumps, and the leaflets may become adherent over a variable portion of their lines of contact, forming an annular diaphragm. So long as this agglutination does not occur, the most extensive thickening and deformity of the segments may be found, the valves being rendered functionally useless, permitting extreme regurgitation, without occasioning appreciable stenosis. When, however, the edges have become adherent, the subsequent contraction necessarily induces some degree of stenosis. The orifice sometimes has a funnel-like appearance. In another class of cases the leaflets become firmly attached to the mural endocardium immediately back of them, or, in cases of the aortic valve, to the intima of the vessel, thus preventing them from coming into apposition to close the orifice. The chordæ tendineæ generally become involved in the sclerosis, beginning at their attachments to the valves and extending to a variable part of their length, sometimes beyond them into the papillary muscles. The edges of the valve leaflets are thus drawn together and the orifice correspondingly narrowed. Calcification of the degenerated, sclerotic tissue of the valves is a common result of the process, sometimes so extreme as to convert the entire valve into a calcareous plate. Ulceration resembling that of atheromatous disease may occur or a true ulcerative endocarditis may be set up at the edges of these plates or beneath them. The warty vegetations of acute endocarditis may also be found, and over the surface there is not infrequently deposited a layer of fibrin from the blood. The changes that occur in the valves of the right heart are identical in character with those just described, but they are much less frequent. The walls of the heart, especially those of the ventricles, are enormously thickened during the existence of compensatory hypertrophy, but, after dilatation has supervened, they are often reduced to extreme thinness. Both conditions are frequently found coincidently in different chambers.

VALVULAR HEART DISEASE.

Under this heading may be conveniently studied the results of the different forms of endocarditis of the valves.

The effect of endocarditis upon the valves is to produce either (*a*) incompetency (insufficiency, with regurgitation of blood), or (*b*) stenosis (partial closure of the orifice). Either of these conditions may exist separately in either of the valves, or they may be combined, affecting the same or different valves at the same time.

Incompetency, or insufficiency, is a condition in which the complete closure of the valve is prevented by erosion, perforation, deformity, or adventitious bands and adhesions. It permits the blood to flow through the orifice in an abnormal, reversed direction. Stenosis, or narrowing, of the valve orifice, on the other hand, prevents the normal flow of blood through it. As a result of either condition a chamber of the heart is engorged with blood; yielding to the increased blood-pressure within, it becomes acutely distended. The heart possesses a certain degree of reserve force which enables it for a time to meet the emergency, however suddenly it may occur, as it ordinarily does the distention caused by sudden active exercise. In most cases a valve lesion develops

gradually, the distention of the cavity is correspondingly slow, and the reserve force is sufficient to carry on the circulation without serious interruption until another change has had time to occur, namely, a hypertrophy of its walls. This is known as a compensatory hypertrophy. The increased muscular power of the heart, in other words, compensates for the valvular leakage. The walls become thickened and the blood is carried in increased quantity and with increased force from the enlarged cavity through the defective orifice. The loss to the general circulation that would otherwise result from either regurgitation or stenosis is thus prevented. Compensatory hypertrophy may enable the heart to perform its function with regularity for a long time, but it cannot restore the integrity of the organ. With the increase of muscular force, or working power, there is a corresponding diminution of the reserve force, and the heart is no longer able fully to meet emergencies. Unusual exertion is met with increased action, but it is the increased action of an abnormally large and strong heart; as a result, the blood-vessels become engorged, the rhythm is disturbed, and more remote disturbances follow, depending in character upon the valve affected and the nature of the lesion. This is known as a disturbance of compensation. The compensation fails at first only when the heart is called upon to perform extra work, but the failure becomes permanent if the heart is constantly subjected to undue strain. The failure is gradual, however, and for a time after the heart is unable to perform extra work it is still capable of maintaining the circulation with the body at rest. A final failure usually occurs, and the organ can no longer perform its function under the most favorable conditions.

With the decline of the muscular power in the walls of the heart, the chambers become more enlarged, and a condition of extreme and permanent dilatation is produced. A very remarkable form of compensation has been described in which the shrinkage of one valve segment through sclerosis is met with a compensatory lengthening of the other segments, but it is at least an extremely exceptional possibility.

The dilatation and loss of compensation are often greatly hastened by degenerative changes in the heart muscle. This is particularly the case when the circulation through the coronary arteries is impeded and the nutrition of the organ diminished. The same result is produced to some extent also by the anemic condition of the system, and this in turn may be added to by poor food, alcoholism, mental emotions, or any illness, especially an acute febrile disease. The special changes in the different chambers of the heart are considered in connection with the affections of each valve.

Remote Effects of Valvular Lesions.—The more direct effects of valvular disease are seen in the lungs, but changes occur also in the liver, spleen, kidneys, and, in cases of long standing, in almost every organ of the body. The pulmonary vessels are greatly distended, and as compensation is lost they become permanently dilated. Their branches often become distinctly varicosed. The circulation is sluggish and the lungs appear deeply congested. Proliferation of the fibrous tissue with pigmentation leads to brown induration. Areas of collapse are sometimes produced, and infarctions often form within the dilated vessels. The condition of the liver is that known as chronic passive hyperemia.

The organ is enlarged, the blood-vessels dilated, the connective tissue proliferated, and pigment is at the same time deposited about the central vein of the lobule. The spleen, kidneys, stomach, and intestines are also congested.

MITRAL INCOMPETENCY.

Etiology.—Mitral insufficiency is the most frequent form of valvular defect. It may occur at any time of life, but commonly affects younger persons than are the subjects of aortic lesions. It is slightly more frequent in women. In a majority of cases it is due to endocarditis following rheumatism. It may depend upon: (*a*) Changes in the segments of the valves, shortening, deformity, or retraction, with which there are generally associated a thickening and shortening of the chordæ tendineæ. (*b*) The segments may be normal, yet prevented from accurate coaptation and closure of the orifice by extreme dilatation of the ventricle or improper action of the papillary muscles. This is known as muscular incompetency. A variable degree of stenosis is usually associated with the incompetency, but less frequently in cases affecting children.

From this purely mitral condition there is to be distinguished a relative insufficiency that ensues upon excessive dilatation of the left ventricle as a result of profound anemia, myocarditis, or loss of tone in the heart muscles due to prolonged febrile or wasting disease, all of which conditions render the valve segments incapable of closing the relaxed or dilated orifice. A relative insufficiency sometimes results from a sudden destruction of an aortic segment. The left ventricle is dilated, the mitral valve rendered incompetent, and the lungs are engorged, sometimes to the extent of producing slight hemoptysis. The hypertrophy of the left ventricle, associated with chronic interstitial nephritis, occasionally yields to dilatation, and pulmonary and systemic engorgement results, which resembles that of a primary mitral lesion. In the same way the hypertrophy due to overwork, alcoholism, or pericarditis in children may lead to conditions almost identical with those of mitral incompetency.

Pathology.—(*a*) With each systole of the left ventricle, a part of the blood is thrown back through the imperfectly closed mitral orifice into the auricle. This blood, together with that entering from the pulmonary veins, produces a dilatation of the auricle. Hypertrophy ensues, but the walls of the auricles are incapable of extensive hypertrophy, and the condition may be regarded as one of dilatation alone. (*b*) An increased quantity of blood is thrown into the left ventricle with each auricular systole, causing dilatation of this chamber also. To meet this, the wall of the ventricle undergoes hypertrophy, and the normal pressure within the aorta is maintained. (*c*) The regurgitation of blood into the left auricle during each diastole of the ventricle, in addition to producing the changes described, prevents the normal flow of blood from the pulmonary veins, and these vessels are dilated. The pulmonary circulation is obstructed and (*d*) the pressure in the right ventricle is increased. As a result, this chamber becomes dilated and hypertrophied. (*e*) Dilatation and hypertrophy of the auricle are finally produced, usually

after tricuspid regurgitation has resulted from the extreme dilatation of the ventricle.

The hypertrophy of the ventricles enables the heart to maintain the normal blood-pressure until subsequent degenerative changes occur. The hypertrophy then gives place to greater dilatation, and failure of compensation ensues.

Relative incompetency, due to muscular relaxation and the other influences that have been referred to, is seldom followed by full compensation.

Symptoms.—The severity of the symptoms varies inversely with the degree of compensation. When the disease develops suddenly with the rupture of a valve, symptoms of extreme incompetency are manifested. But when the incompetency develops slowly, the reserve force of the heart and the prompt hypertrophy may maintain the circulation to such an extent that no subjective manifestations are produced. In most cases, however, the patient experiences shortness of breath, palpitation of the heart, and slight cyanosis after exertion. In some cases the cyanosis is more constant. The face is somewhat congested, the lips, ears, and finger-nails have a bluish tinge. In cases of long standing, especially in children, the fingers become clubbed. The compensation may become so perfect, however, either spontaneously or through treatment, that, although some of these evidences of the disease remain, the patient is able to pursue his usual occupation for many years. Such persons are more liable, however, to attacks of bronchitis, and slight hemoptysis is likely to occur as a result of the pulmonary engorgement.

With the failure of compensation, the symptoms become more intense and more constant, as a result of the rapid increase of venous engorgement. Dyspnea becomes constant and it is often accompanied with cough and watery, sometimes bloody, expectoration containing pigmented alveolar epithelium. Cyanosis is not always a constant feature, but it is sometimes intense, particularly after exertion or coughing. The sleep is often restless and broken by sensations of suffocation. The peripheral veins become distended, and the skin has sometimes an icteric hue. Dropsy ensues, beginning in the feet and ankles, and gradually invading the body, particularly the serous cavities. The liver is enlarged. The urine becomes scant and albuminous, usually containing epithelial, granular, or blood casts. Gastric and intestinal digestion is impaired. After repeated attacks of this character, and repeated relief by treatment, a stage is finally reached which can no longer be mastered, and death ensues from pulmonary edema or the extreme cardiac dilatation, rarely from sudden heart-failure.

Physical Signs.—*Inspection.*—The impulse of the heart is forcible and heaving during the stage of full compensation, but wavy and feeble after compensation has failed. The apex beat cannot always be seen. The precordial region is sometimes prominent in children.

Palpation.—A strong impulse can be felt beneath the sternum, over a variable distance to the right of it, and to the left as far as the axillary line in extreme cases. The position of the apex beat is not constant, but varies with the relative enlargement of the ventricles and the total enlargement of the heart. It is usually found to the left of the nipple,

sometimes in the axillary line. It may be as low as the sixth intercostal space, but is higher in extreme dilatation of the right ventricle. A thrill at the apex is pathognomonic of mitral incompetency, but it is seldom to be felt.

Percussion.—The area of percussion dullness is greatly increased, particularly in the lateral direction. In cases of long standing, with great dilatation of the ventricles, it may extend from one or two inches (2.5—5.0 cm.) to the right of the sternum to three or four inches (7.5—10.0 cm.) left of the nipple. The upper margin of the dullness is little, if any, higher than normal.

Auscultation.—The characteristic sign of mitral insufficiency is a systolic murmur heard with greatest intensity at the apex, or in many cases a little beyond it. The first sound is often entirely replaced by it. The murmur is transmitted with great distinctness to the axilla and generally to the angle of the scapula as well as a variable distance upward and downward along the spine. In many cases it is audible over all parts of the chest. It is usually of a blowing or puffing quality, sometimes ending in a musical tone, but it may be so harsh and loud as to be heard a distance of a few inches from the chest. When, however, the dilatation of the right ventricle is excessive, and the apex is pushed back from the chest-wall, the murmur may be indistinct or almost inaudible. In many cases, too, it becomes audible only after slight exertion or when the patient leans forward or lies upon his back. Sometimes it is heard best along the left margin of the sternum.

A rough presystolic murmur sometimes accompanies the systolic, probably as a result of an associated mitral stenosis. A soft tricuspid murmur of regurgitation is occasionally heard with it over the lower sternal region in cases of extreme dilatation of the right ventricle. The second sound at the apex is generally distinctly heard, and the pulmonary second sound is accentuated. It is heard in the second interspace at the left of the sternum. The loudness and other qualities of the murmur give but little indication of the character or gravity of the valvular defect. The pulse is generally irregular, and often extremely so in the beginning. After compensation becomes completely established, or under proper treatment, it may become full and fairly regular, but a moderate irregularity generally persists.

Diagnosis.—The most typical signs of mitral regurgitation are: (*a*) Increased dullness in a lateral direction, indicating great transverse enlargement of the ventricles; (*b*) a systolic murmur heard with greatest intensity at the apex, but transmitted to the axilla and back; and (*c*) accentuation of the pulmonary second sound. The condition is most readily confounded with the so-called accidental, or hemic, murmurs and less definitely understood conditions. These sounds, no doubt, originate within the ventricle, and they may be transmitted to the axilla. They are usually soft and blowing in character, and they are not associated with dilatation or accentuation of the pulmonary second sound. The history of the previous condition is also different, being in one case an infection, in the other an abnormal state of the blood.

It is not always possible to determine whether the regurgitation is due to an actual lesion of the valve leaflets or to a dilatation of the ventricle and consequent enlargement of the orifice due to overwork,

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costal space, near the left of the sternum, when the chest wall is thin. The apex beat cannot always be recognized; it is not usually displaced beyond the line of the nipple.

Palpation.—A presystolic fremitus or thrill is often felt in the fourth or fifth intercostal space, within the nipple line. It is usually short, harsh, and distinct, and terminates with a sharp shock in the usual cardiac impulse. When present it is pathognomonic of mitral stenosis. The evidences obtained from inspection are confirmed. The strong impulse beneath the sternum and in the third and fourth left interspaces can be distinctly felt; sometimes there is also a distinct impulse in the second interspace due to the pulsation of the conus arteriosus of the right auricle.

Percussion.—The area of dullness is increased tranversely, but not nearly to the extent seen in mitral insufficiency. In extreme cases it extends from two inches (5 cm.) to the right of the sternum to the nipple, or a short distance to the left of it, rarely more than an inch even when incompetency is also present.

Auscultation.—The characteristic sign of mitral stenosis, during the stage of compensation, is a presystolic murmur heard most distinctly at the apex or a short distance to the right of it. This is usually a prolonged, rough, purring, or rumbling murmur, heard just before the first sound and terminating in it. Its character and the time of its occurrence are readily understood when it is remembered that it is produced during the passage of the blood through the narrowed orifice from the auricle into the ventricle. It usually begins in the latter half of the auricular systole, but sometimes earlier, and ends at the first sound, which is generally sharp, clear, and distinct. It is often audible over only a very limited area.

The systolic apex murmur not infrequently accompanies the presystolic, owing to the commonly associated regurgitation. It is usually low and indistinct, but it may be loud and transmitted to the axilla. The pulmonary second sound is strongly accentuated and sometimes reduplicated, but the aortic second sound is generally reduced in force. A tricuspid murmur is occasionally heard over the lower sternal region or to the right of it in cases of long standing, owing to regurgitation through that valve.

After compensation has failed, the presystolic murmur and thrill are lost, but the sharp first sound and the shock usually persist. In some cases the irregular action of the heart is so extreme as to obscure the auscultatory signs.

Diagnosis.—Mitral stenosis is not difficult of recognition in a typical case, and the sources of error are few. But during the failure of compensation, when the thrill and murmur are absent, the valvular condition may be overlooked. Its existence should be suspected from the hypertrophy, the sharp first sound, the accentuation of the pulmonary second sound, and the great irregularity of the heart's action in most cases.

The Flint murmur, which is more fully described under the head of Aortic Incompetency (p. 345), should not be mistaken for that of mitral stenosis, since it is usually confined to the middle period of auricular systole, and it is heard only in association with the murmur of aortic

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l attacks of angina are attributed to changes in the nerves
rt or to pressure upon them by the sclerotic tissue associated
nterstitial myocarditis. A more or less general arteriosclerosis
nly found in the vessels thoughout the body, as a result of the
strain thrown upon them by the forcible contractions of the
phied ventricle. It is a remarkable fact, however, that in some
s, particularly in rheumatic cases, although there have been
es during life of enormous distention of the arch of the aorta,
nate, and right carotid, these vessels are found to be almost
ly normal and free from dilatation after death. A greater or less
of stenosis sometimes accompanies the insufficiency, but it
so uniformly present as in the corresponding lesion of the mitral

mptoms.—This disease often exists for a great length of time with-
producing definite disturbances. Among the earliest symptoms
ested, but often referred to some other cause, are headache, flashes
ght, tinnitus and vertigo or faintness upon rising suddenly. Slight
ion often causes palpitation and shortness of breath, and this may
ccompanied with distress or actual pain in the cardiac region. Pain
comparatively early symptom in some cases. It may be a dull
e confined to the precordial region, or sharp and spasmodic, often
iating to the left shoulder and sometimes down the arm or up the
k. It seldom radiates to the right side. Typical attacks of angina
toris occur in some cases.

After failure of compensation, symptoms of a more definite character
re induced. Dyspnea is often a most marked feature; it is usually
worst at night and compels the patient to sleep with his head high, or
sitting in a chair. The sleep is disturbed by dreams and nervous start-
or sensations of suffocation more frequently than in any other form
of valvular lesion. Cough is a common symptom, due to the engorge-
ment of the lungs, but hemoptysis and cyanosis are seldom observed
in an uncomplicated case. The patient is usually anemic. The blood-
count may fall below 3,000,000 in the c.mm. Edema of the ankles

generally supervenes; at first, perhaps, as a result of the anemia, later as a result of the failing of the circulation. General dropsy rarely ensues in the absence of extreme incompetency of the mitral valve as an associated lesion.

Intercurrent attacks of acute endocarditis are not unusual and often lead to a hastily fatal termination of the disease. Embolism is also a frequent complication. It is announced by sudden pain, perhaps associated with tenderness in the affected region, as in the spleen. Hematuria develops when the kidney is the seat of the lodgment, and paralysis when the brain is involved. The closing weeks, perhaps months, of the patient's life are most distressing. Great restlessness, delirium, and moroseness are commonly developed. The patient sometimes becomes acutely insane and he may attempt suicide. To what extent such maifestations are a result of the valvular condition and to what extent they may be referred to an associated uremia in different cases, has not been determined. Sudden death occurs more frequently in this than in any other form of valvular disease.

Physical Signs.—*Inspection.*—The cardiac impulse is strong and heaving. The apex beat is displaced to the left, but seldom beyond the anterior axillary line. It may be as low as the sixth or seventh intercostal space. The precordial space sometimes appears prominent, especially in children. The vessels of the neck throb, and in extreme cases pulsation of the superficial vessels of the entire upper part of the body, especially in the suprasternal notch, becomes visible. Ophthalmoscopic examinations reveal similar pulsation of the retinal vessels.

Palpation.—A forcible impulse is felt, except in the late stages of the disease, when it becomes softer and wavy. There is sometimes a distinct pulsation of the entire precordial region, and sometimes also in the second right intercostal space, due to the pulsation of the aorta. A depression of one or more of the left interspaces, between the sternum and mammary line, is occasionally perceptible during systole. A diastolic thrill can occasionally be felt over the base.

The pulse of aortic incompetency is characteristic. The impact is strong and jerky, often apparently full, but it immediately collapses under the finger. The forcible impulse is due to the strong contraction of the ventricle, which throws the blood into the arteries with much force, but the regurgitation permits the blood to fall back almost instantly. This peculiarity can sometimes be better recognized when the hand is held high above the head during the palpation of the pulse, thus favoring the recoil after the first impulse. On account of this feature, the pulse is often referred to as the water-hammer pulse. Another distinctive feature of the pulse is that it is delayed, a perceptible interval elapsing between the systole of the heart and the radial impulse, particularly in the advanced stage of the disease. Palpation of the vessels of the neck reveals a similar pulsation and sometimes a thrill. The pulsation and thrill commonly felt in the suprasternal notch in this disease occasionally lead to the erroneous diagnosis of aortic aneurism. A venous pulsation is occasionally observed, but it is seldom so strong as to be recognized on palpation. An arterial pulsation of the liver is occasionally noted, and less frequently that of the spleen. A capillary pulsation is not infrequently obtained either by gently compressing the

finger-nails or by drawing the finger-nail across the forehead. Beneath the compressed nail or at the margin of the hyperemic line an alternating flush and paling can be seen. It is not, however, fully pathognomonic of aortic insufficiency.

Percussion reveals a great increase of the area of dullness, greater in extreme cases than in any other valvular lesion. Its direction is more particularly downward and outward to the left.

Auscultation.—The murmur of this lesion is one of soft, blowing, sometimes musical quality, long in duration, and heard with greatest intensity, as a rule, at the base of the heart, in the middle of the sternum opposite the third costal cartilage, or along the entire right side of the sternum from the second cartilage to the xiphoid. The murmur may be harsh when the cusps have become calcified or in cases of traumatic rupture of a segment. The second sound may persist, but it is often entirely replaced by the murmur. A short, soft systolic murmur is sometimes heard at the base, but in most cases the first sound is clear and distinct until late. When a partial stenosis accompanies the insufficiency, and especially when the cusps are adherent along a part of their edges, a sharp, rough systolic murmur is heard. It must not be regarded as a feature of the incompetency, but as a comparatively frequent complication. In many cases no abnormal sound is heard at the apex, but when relative insufficiency of the mitral valve has been induced an apical systolic murmur accompanies the diastolic which is heard at the base. This murmur should not be confounded with the interesting bruit sometimes heard at the apex and known as the *Flint murmur.* This is a more or less distinct rumbling sound, described as echoing in quality, usually occurring at the middle of diastole; sometimes it is more immediately presystolic, and heard only at the apex. It is recognizable in about half the cases of aortic incompetency. It is less distinct than the presystolic murmur of mitral stenosis, although it is virtually a murmur of that character. It is attributed to the forcible impact of the regurgitated blood upon the large anterior curtain of the mitral valve, possibly causing it to interfere with the simultaneous entrance of blood from the left auricle. It is not accompanied by the accentuation of the first sound, and it is always associated with the murmur of aortic insufficiency. A double, to-and-fro murmur can sometimes be heard by auscultation over the carotid and femoral arteries.

Diagnosis.—In the ventricular hypertrophy of *chronic nephritis* a murmur with greatest intensity at the base may be heard, but the second sound is distinct and accentuated, and the urinary examination reveals the condition of the kidneys. The hypertrophy is usually moderate. The differentiation can be further established by cryoscopy. The freezing-point of the urine is high, sometimes above the normal limit, —1.30° C., in renal disease, but below —2.20° C. when the heart is affected.

Anemic murmurs are heard at the base, but they are usually softer, of shorter duration, and unaccompanied with hypertrophy. No thrill can be felt, and the arterial pulsations are absent; a venous purring can sometimes be heard in the cervical veins.

The ***prognosis*** depends upon the extent of the insufficiency, the character of its cause, and the presence or absence of myocardial changes and other complications. Cases due to endocarditis are more favorable

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vessels. A musical tone is sometimes heard. The second sound is generally absent; sometimes it is replaced by a murmur of regurgitation when the valvular defect is so great as to cause incompetency. Very similar to this murmur is the bruit caused by hemic conditions or that caused by the passage of the blood over a roughened orifice or calcareous plates in the wall of the aorta near the valve. After the compensation has failed, the murmur becomes softer and less distinct.

Diagnosis.—The condition is not usually difficult of diagnosis, and the only source of error, as a rule, is the adventitious murmur just referred to. This can be excluded by the hypertrophy of the left ventricle and the small, firm pulse. A distinct murmur, especially if musical and heard in this region, is generally due to aortic stenosis.

The ***prognosis*** depends upon the condition of the valve. Uncomplicated stenosis is not incompatible with fair health so long as compensation is maintained, but, associated with regurgitation and after compensation has failed, the consequences are more serious.

TRICUSPID INSUFFICIENCY.

Etiology.—The tricuspid valve is seldom incompetent as a result of disease, and a regurgitation of blood through it is commonly a result of relative insufficiency due to dilatation of the right ventricle following lesions of other valves, or obstruction of the pulmonary circulation in emphysema or interstitial pneumonia.

Symptoms.—A systolic pulsation is transmitted to the veins of the neck as a result of the regurgitation of blood into the auricle with each contraction of the ventricle. This pulsation is distinctly visible, as a rule, in the right jugular, sometimes also in the subclavian and axillary veins. When the valves of the veins remain intact it may amount to only a slight wavy vibration. The pulsation is often transmitted to the liver; the pulsation of the organ can be felt, in bimanual palpation, with each systole; very rarely, it can be seen. A systolic murmur can be heard over the lower sternal region and to the right, sometimes as far as the axillary line, but often over only a very limited area. It is generally soft in quality and variable in pitch. Another marked feature in many cases is an extreme distention of the veins of the upper part of the body when the patient strains or coughs. The pulsation of the veins is distinctly visible during this distention. Percussion shows an increased area of dullness, especially to the right of the sternum. The symptoms belonging strictly to the condition are obscured in most cases by those of the underlying disease. The congestion of the organs is general, however, and that of the kidneys is often a distinct feature of the disease. Anasarca develops toward the close, affecting the face and upper extremities more than it does in other valvular lesions.

Diagnosis.—This is clear in the presence of hypertrophy of the right ventricle, with venous engorgement and pulsation and a systolic murmur heard with greatest intensity in the lower sternal region.

TRICUSPID STENOSIS.

Etiology.—This is a rare lesion except in congenital cases, and these are often associated with other defects that are incompatible with life.

It may, however, be acquired. It then occurs in adult life, and fully 80 per cent of recorded cases have been seen in women. It is seldom the only valvular lesion present; in most cases the mitral or both the mitral and aortic valves are defective. In most cases, too, the tricuspid lesion is a result of one of the other lesions.

Physical Signs.—A presystolic thrill has been observed in some instances. The dullness is somewhat increased, particularly to the right of the sternum. A presystolic murmur is heard at the base of the xiphoid cartilage or just at the right of it. The patient is generally cyanotic, sometimes extremely and constantly so. When the condition is thus extreme, an intense general dropsy often ensues. The prognosis is always exceedingly grave, owing to the impossibility of relief through any such change as compensatory hypertrophy.

PULMONARY VALVE LESIONS.

Functional Murmurs.—A soft blowing murmur is very often heard in auscultation over the second left intercostal space in children and sometimes in adults in ill health, especially when the patient is lying down. It may be heard also in anemia or after slight exertion during convalescence from any of the acute fevers. But it is heard also in some individuals in good health with thin chest-walls, during expiration. It is purely functional and of little significance.

Pulmonary insufficiency is an exceedingly rare condition sometimes resulting from congenital malformation, as when the segments are agglutinated. It may result from endocarditis. It has been suggested also that a "safety valve" leakage sometimes occurs when the pulmonary vessels become engorged. The murmur is diastolic and is heard most distinctly at the second left intercostal space. The right ventricle becomes hypertrophied and dilated. The aortic sounds and the radial pulse remain normal. The bruit is often distinguished with much difficulty from that of aortic insufficiency, except by the absence of the usual results of that lesion.

Pulmonary stenosis is one of the most important congenital deformities of the heart. It may, however, be encountered in adult life as a result of endocarditis or atheroma. When congenital it generally consists of an agglutination of the margins of the valve segments to such an extent as to greatly diminish the size of the orifice. The stenosis is generally compensated for by an incompleteness of the ventricular septum or patency of the foramen Botalli. Tricuspid stenosis is sometimes associated with the lesion. The diagnosis is difficult. The right heart is generally hypertrophied, and a systolic murmur can sometimes be heard in the left intercostal space. It may be transmitted to the right, but never along the great vessels, as is that of aortic stenosis, with which it might otherwise be confused. The pulmonary second sound is feeble or quite inaudible.

Association of Valvular Lesions.—Valvular lesions are generally associated in the following order of frequency: (1) Mitral and aortic lesions; in children mitral and aortic insufficiency are most frequently combined; in adults mitral insufficiency combines with aortic stenosis; (2) mitral and tricuspid lesions; (3) mitral, aortic, and tricuspid;

is of the aortic valves is more frequently
ficiency than with mitral stenosis.

sions.—The prognosis in all valvular lesions
upon the extent to which compensation is
estimated from the degree of dilatation that
haracter of the heart's action. The prognosis
ompensation can be brought about by treat-
favorable than when it is spontaneous, and
ured. Age is also an important factor in prog-
ally poor subjects of valvular lesion, but com-
velops at puberty, and with care life may then
oviding the constitution be vigorous and the
s of rheumatism, influenza, or other infection.
te valvular lesions better than men, probably
e to overexertion or other influence that dis-
The prognosis is impaired by all acute infections,
alcoholism, the arthritic diathesis, chronic pul-
use. The prognosis of the different lesions has
each heading.

lar Lesions.—*Stage of Compensation.*—1. The admin-
medies to a patient whose heart is acting regu-
nsation of the defect, is one of the most serious
ommitted. Nothing is then required but to guard
he influences that are likely to disturb the action
often injudicious to inform him of his condition,
discovered accidentally during an examination. On
discovery may prove to be a valuable one to the
him to avoid excesses that tend to hasten the loss
nder all circumstances the physician should remember
t always signify a valvular disease, and he should be
the correctness of his diagnosis before divulging it.
the patient should also be considered in connection
f the lesion. As a rule, he should be given the assur-
disease of the heart is not necessarily fatal and that
life depends to a great extent upon the manner in
himself. He should be instructed with regard to the
most harmful in his condition. A practical man
ife or engrossed with much business care and worry
old that he has reached the time for rest, especially
from an aortic lesion. A nervous person with a less
often be moved to unnecessary precaution and con-
erest hint that he has a heart lesion. It is not usually
sable to forbid all activity; the patient should rather
ake such moderate outdoor exercise as he can endure
the rhythm of the heart, as indicated by palpitation,
rdial pain. Any exertion or excitement that disturbs
the heart's action must be avoided. The diet need
as a rule, except so far as to avoid overeating and
ndigestible articles likely to cause flatulency. Alcohol
ld, as a rule, be interdicted.

t Compensation.—Loss of compensation is sometimes so

suddenly fatal as to afford no opportunity for treatment. It is generally gradual, however, and may be relieved unless it has been too long disregarded by the patient. The heart must be given rest. This can be accomplished by confining the patient to bed for a week or ten days, thus relieving it of unnecessary work. In some cases this of itself is sufficient to re-establish compensation. It is not always possible, and indeed not always necessary, to secure absolute regularity of action, particularly in mitral lesions. Regularity is more desirable in aortic disease, since irregularity here is generally significant of failing compensation. In severe cases associated with cardiac dilatation, when the dyspnea is urgent and accompanied with cyanosis, venesection affords the promptest relief in cases showing extreme venous engorgement. Purgation acts in a similar manner, but it is slower. The regular action of the bowels is important in all cases.

Medicinal Treatment.—Heart tonics should be employed in most cases to assist in restoring compensation, or to maintain it when restored through rest. They should not be used beyond the quantity required to secure the desired result. Digitalis is universally employed and can be relied upon to maintain its action for many years in some cases. A half-ounce (15.0) of the fresh infusion or ♏x to xx (0.6—1.2) of a good tincture should be given every three or four hours until the heart's action has become full and regular. Then the quantity can generally be reduced to half the original amount or less. When dropsy is present, however, the full dose, if tolerated by the stomach, should be continued until the edema has disappeared, and in cases of this character it is often necessary to maintain the dosage throughout the remainder of the patient's life. It should be employed in all cases of failing compensation of whatever character, but theoretically at least it should be given with greater caution in cases of stenosis than in those of regurgitation. The only ill-effect that is usually observed in the use of digitalis is the production of nausea and vomiting in some cases, a symptom which quickly subsides upon withdrawal of the drug. When persistent vomiting is induced, tr. strophanthus, ♏v to viij (0.3—0.5), may sometimes be employed in its stead, but it does not always fully replace it. Strychnin is often of great benefit in giving strength to the heart muscles and may be employed in connection with the digitalis; occasionally it can be used as a substitute for digitalis. When anemia is a marked feature, as it so often is in aortic incompetency, iron or arsenic should be given in full doses.

Treatment of Special Symptoms.—1. *Dyspnea.*—The chest should be carefully examined in order to determine whether the dyspnea be due to the cardiac incompetency or to hydrothorax or pulmonary edema. When hydrothorax is present, the fluid should be withdrawn by aspiration as often as it becomes excessive. The pulmonary edema may sometimes be relieved by purgation and diuresis, and held in check by full doses of digitalis and strychnin. Cupping the chest may prove beneficial. For the dyspnea and restlessness at night there is no better remedy than morphin, gr. ⅛ (0.008), or codein, gr. ¼ (0.016). Glonoin often affords prompt relief in cases in which the arterial tension is high, but it must generally be given at short intervals and in increasing doses in order to produce more than transitory effects. The par-

oxysmal dyspnea (cardiac asthma) sometimes yields to the compound spirit of sulphuric ether, ʒ ss to j (1.8—3.6), administered in cold water and repeated in an hour if necessary. Potassium bromid is also useful in these cases. Dyspnea due to associated bronchitis or emphysema in elderly persons calls for special treatment of those conditions.

2. *Palpitation and Angina.*—In cases of excessive dilatation, an ice-bag applied to the cardiac region often affords relief to both these symptoms. Tr. aconite may be employed to regulate the heart's action and is sometimes better than digitalis, especially in aortic incompetency. It should be given in doses of gtt. ij or iij every two or three hours. Nitroglycerin may prove beneficial. Potassium iodid, gr. x (0.60) t. i. d., relieves the pain in some cases. Potassium bromid and elixir of ammonium valerianate are also of service; but when the pain is severe, morphin (gr. ¼; 0.016) with atropin (gr. 1-120; 0.0005) should be administered hypodermically.

3. *Edema.*—The patient should be placed upon a dry diet, and an effort made to reduce the edema with hydragogue cathartics. The circulation should be maintained by full doses of digitalis, and the action of the kidneys further stimulated with mild diuretics—potassium bitartrate, citrated caffein or sodium and theobromin salicylate. Calomel in doses of gr. 1-10 (0.006) is an excellent diuretic in cardiac cases, but it must be discontinued as soon as its action becomes apparent. When the edema of the lower extremities becomes extreme, it is better to puncture the skin than to allow it to rupture, although the necessity should be prevented, if possible, by bandaging with flannel. The legs should be bathed with an antiseptic solution before the punctures are made and at regular intervals, morning and evening, thereafter. In hospitals the patient should be isolated in order to protect him from erysipelas or other infection of the wounds.

4. *Insomnia.*—Sleeplessness often calls for special treatment. In some cases the bromids with valerian or camphor induce quiet sleep. Trional in a single dose of gr. xx or xxx (1.30 to 2.0) before retiring may be tried when they fail. Paraldehyd, amylene hydrate, and urethane are also employed, but their action is uncertain. Morphin fails to induce sleep in some cases. The insomnia often subsides with the restoration of compensation.

5. *Hemorrhage* from the nose, lungs, stomach, or uterus requires prompt treatment. Opium is always indicated, but ergot, astringents, and styptics are generally useless. In other respects the treatment is the same as that of hemorrhage from the same sources in other conditions. The warm salt infusion must sometimes be resorted to.

HYPERTROPHY OF THE HEART.

ENLARGEMENT OF THE HEART.

Definition.—An enlargement of the heart due to increased thickness of its walls. The condition may be general, but is usually confined to one or more chambers, more commonly to the ventricles. There may be simple hypertrophy or hypertrophy with dilatation (eccentric hypertrophy); one chamber may be hypertrophied and another dilated. "Con-

centric hypertrophy" is a term now seldom employed to describe thickening of the walls with apparent diminution of capacity, probably due in all cases to post-mortem contraction. Simple hypertrophy is a little more frequently seen in the left ventricle than in the right.

Etiology.—The muscle of the heart, like any other striped muscle, responds to increased exercise by undergoing hypertrophy, becoming larger and stronger. Hypertrophy is in all cases a result of overwork, and it is often favored, no doubt, by overstimulation, especially with alcohol, while working to excess. The immediate causes of hypertrophy of the right and left ventricles are sufficiently different to receive separate consideration:

Hypertrophy of the left ventricle, with or without general enlargement of the heart, results from: (1) Prolonged or habitual muscular exercise, as in athletes. Excessive hypertrophy is seldom due to this cause alone. (2) From such conditions of the heart itself as (*a*) aortic stenosis or insufficiency; (*b*) mitral incompetency; (*c*) pericardial adhesions; (*d*) interstitial myocarditis; (*e*) overactivity or palpitation due to nervous disease, as in exophthalmic goiter, or to toxemia, as in chronic nephritis, gout, or lithemia; (*f*) arteriosclerosis and other conditions producing increased arterial tension or resistance.

Hypertrophy of the right ventricle results from: (*a*) Mitral insufficiency or stenosis, and remotely from aortic lesions; (*b*) lesions of the pulmonary valves, not of frequent occurrence; (*c*) obstruction or partial obliteration of the circulation in the lungs, as in emphysema and interstitial pneumonia; (*d*) pericardial adhesions.

Hypertrophy of the Auricles.—Simple hypertrophy of the auricles perhaps never occurs, but hypertrophy with dilatation is a constant result of valvular lesions or changes in the circulation which increase the intrapulmonary tension. In the left auricle it is due to mitral incompetency or stenosis; in the right, to any of the valvular lesions that retard the flow of blood through the lungs, or, rarely, to tricuspid stenosis.

Morbid Anatomy.—The heart is often increased to double its normal weight. The greatest hypertrophy is usually found in the left ventricle. The walls of the affected chambers may be double their normal thickness; those of the ventricles sometimes attain to treble thickness. The heart appears wider than normal and the apex less pointed. The muscle substance is firm and dense in simple hypertrophy. The enlargement is probably due to hyperplasia (numerical hypertrophy).

Symptoms.—Simple hypertrophy the result of physical exercise, or when it is compensatory, does not usually produce symptoms so long as the heart's action is not disturbed by pathological conditions in other organs, as by indigestion or general ill-health. When, however, the rhythm is disturbed, the abnormal force of the heart's action becomes apparent in the production of precordial uneasiness, headache, flushing of the face, vertigo, tinnitus, and visual disturbances. Pain is unusual. The patient experiences unpleasant sensations, more particularly when he lies on the left side. Arteriosclerosis is often associated with the hypertrophy in cases arising from obstruction of the peripheral circulation and may give rise to symptoms. With disease of the blood-vessels added to the increased force of the heart's action, there is greater

danger of rupture and hemorrhage, particularly into the brain. When compensation fails, a different train of symptoms is added.

Physical Signs.—*Inspection.*—In some cases, especially in children, the precordial region is abnormally prominent. The cardiac impulse is strong and diffused. The apex beat is displaced downward and outward, the extreme limit being the eighth intercostal space and 3 inches (7.5 cm.) beyond the nipple. *Palpation* reveals a strong heaving impulse usually with slow action. A second impulse, an apparent rebound, is sometimes observed.

The pulse in uncomplicated hypertrophy is full and regular, but increased in tension, often throbbing. It may be normal or increased in rapidity, but rapidity and irregularity are generally the first indications of failing compensation.

Percussion.—The dullness is increased in all directions, so that it may extend from the second intercostal space downward on the left sternal margin, and from the right border to one or two inches beyond the left nipple. The apex is more rounded than normal.

Auscultation.—The first sound of the heart in simple hypertrophy is dull and long; it may be reduplicated, especially in the hypertrophy following chronic nephritis. In young subjects it sometimes has a metallic quality. The second sound is also strong and may be reduplicated or metallic in the aortic region. The physical signs accompanying hypertrophy due to valvular disease have been considered in connection with the different forms of these lesions, and need not be repeated.

Diagnosis.—Simple hypertrophy is to be differentiated chiefly from nervous palpitation and pericardial effusion or other conditions which increase the area of dullness.

Nervous palpitation is seen for the most part in exophthalmic goiter, the tobacco heart, neurasthenia, or prolonged overwork. In this condition the impulse is strong, but not heaving, and the action is more rapid than in simple hypertrophy. The area of dullness is not so much enlarged. The first sound is sharp, and the second diminished in force, as a rule.

In *pericardial effusion* the area of dullness is triangular, with the base downward; the heart-sounds are feeble and distant and the action is increased. The pulse is rapid and weak and the patient is acutely ill. Aneurism, mediastinal tumor, pyloric adhesions due to interstitial pneumonia or tuberculosis may produce conditions simulating hypertrophy, but the diagnosis can generally be made from the normal position of the apex, normal sounds often without increased area of dullness, and the regular, normal character of the pulse, except as it may be modified by the other conditions present.

Hypertrophy with dilatation is excluded, but not always with certainty, by the greater force and regularity of action, and to some extent also by the absence of murmurs and the more forcible and distinct second sound, slow full pulse, and entire absence of evidences of pulmonary engorgement.

Error in diagnosis is possible when emphysematous expansion of the lungs obscures the outline of the enlarged heart and renders its recognition more difficult.

Prognosis.—The prognosis is good while the action of the heart

remains normal, or when it is sufficient to maintain compensation. Hypertrophy from active exercise is not incompatible with many years of good health. The prospects in a given case depend upon the cause of the condition and the stage that it has reached. Failure must come, however, and it may be initiated with great suddenness by some intercurrent disease, great fatigue, or mental strain. After compensation has failed, the history of the case is one of dilatation or myocarditis, and the prognosis is correspondingly less favorable.

Treatment.—During the stage of simple hypertrophy with regular action of the heart, no treatment is required further than the avoidance of fatigue, excesses, and all other influences capable of disturbing the condition. After compensation has failed, the treatment is that of dilatation.

DILATATION OF THE HEART.

Definition.—Increase in the size of the heart-chambers, with or without increase in the thickness of their walls. In some cases the walls become abnormally thin.

Etiology.—The two great causes of dilatation are weakness of the walls and increase of internal pressure. These may operate separately or, as is more frequent, together. Dilatation sometimes develops without previous hypertrophy, developing suddenly, as a rule, or it may succeed hypertrophy. It may arise from internal tension which the walls are unable to overcome, or from degenerative changes in the muscle more or less directly due to the hypertrophy. Once established, the dilatation increases through its own impairment of the circulation. The heart-cavities are constantly overfilled, and increased tension is maintained. The nutrition of the muscles is diminished, and the degenerative change is thus promoted. An acute dilatation is often overcome for a time by the compensatory hypertrophy, but, after the compensation has failed, the dilatation increases and often becomes extreme. The causes which have been enumerated as producing hypertrophy, as the prolonged exertion in athletic training, sometimes lead to dilatation with little or no hypertrophy. The dilatation may be acute and terminate fatally. Dilatation sometimes follows degeneration of the heart muscles as a result of the acute infectious diseases, notably typhoid fever, erysipelas, and pneumonia. It is then attributed to the anemic condition or to the action of toxins circulating in the blood. There seems to be an intimate relation in some cases between cardiac dilatation and excessive beer-drinking, especially among the workers in breweries. Dilatation may result from the degenerative changes occurring in endocarditis and pericarditis, sometimes also from the adhesions which result from the latter affection. In advanced interstitial myocarditis, a more localized dilatation occurs at the point that is most markedly sclerotic, generally at the apex of the left ventricle. In some cases of dilatation, especially in those of sudden development, no cause can be discovered, and the condition is often described as idiopathic.

Morbid Anatomy.—The condition of the different heart-chambers is variable. The dilatation is usually associated with hypertrophy of the walls, and affects two or more chambers at the same time. The most extreme dilatation is seen in aortic insufficiency, for all chambers are

then affected. The right ventricle is subject to more extreme dilatation than the left, and the left auricle than the right. The endocardium is generally opaque, and the myocardium is found in various stages of degeneration, for the most part fatty or parenchymatous. Changes of a degenerative nature have been observed also in the ganglia. Dilatation of the veins at the point where they enter the auricle is commonly present.

Symptoms.—Dilatation associated with compensatory hypertrophy may for a long time be unrecognizable by symptoms. Developing slowly, as it usually does, the dilatation may be for a time concealed by the forcible action of the heart. But increasing dilatation means increasing weakness, and the time finally comes when, perhaps as a result of some unusual strain, the hypertrophied walls become unable to properly empty the chambers during systole, and definite symptoms are produced. In the acute dilatation, which is sometimes seen in connection with the acute infectious diseases, the symptoms are often abrupt and severe. They are the same in character, however, whether they are sudden or slow in development, for in both instances they are the manifestations of venous engorgement of the lungs and of the general circulation.

The symptoms on the part of the pulmonary circulation are the same as those that have been described in connection with valvular lesions, notably dyspnea. Bronchitis is generally present, and edema of the lungs is likely to develop as a late or terminal condition. Hydrothorax may ensue in one or both sides.

The symptoms of general venous engorgement appear in different parts of the body. They include engorgement of the vessels of the neck and face, and in severe cases headache, dizziness, tinnitus, visual disturbances, sometimes delirium, and later stupor or coma from edema of the brain. The stomach and intestines respond to the congestion in various disturbances of their functions. The liver, spleen, and kidneys are also involved, and later a general dropsy develops, beginning in the feet, as a rule, and gradually extending upward to finally involve the serous cavities and upper extremities and most noticeable in the dependent portions of the body, as in the loins, when the patient is confined to bed. Pain in the region of the heart is sometimes complained of, and it may radiate to the left shoulder and arm. Attacks of angina are observed in some cases.

Physical Signs.—Inspection and palpation show an increased but diffuse area of pulsation, often so undulatory and feeble that the location of the apex cannot be definitely determined. The direction in which the greatest increase of dullness occurs depends upon the relative dilatation of the right and left sides of the heart, which has been considered under the several valvular lesions. A murmur may not be heard at any time, but, as a rule, if there has not been a valvular defect in the beginning as a causative factor in the production of the dilatation, a relative insufficiency of the mitral or tricuspid valve results from the dilatation, and a bruit is then heard.

Interesting changes of rhythm sometimes occur, the most important of which are the galloping rhythm and embryocardia. In the former the heart-beats resemble the foot-falls of a cantering horse; in the latter

the first sound is so similar to the second that it cannot be readily distinguished, thus resembling the sounds of the fetal heart.

The radial pulse is generally weak and may show greater irregularity than is perceptible in the heart's action, several beats often being lost between those that are perceptible.

Diagnosis.—Enlargement of the heart due to hypertrophy is distinguishable by the strong impulse, distinct apex beat, accentuated second sound, and strong, full, regular pulse, without evidences of venous engorgement.

Pericardial effusion is distinguished by the triangular dullness with the greatest diameter below, often producing an area of dull tympany in the left infrascapular region. Evidences of compression of the left lung are also recognizable in some cases.

Prognosis.—Acute dilatation sometimes proves rapidly fatal, but it is generally recovered from. The dilatation of lost compensation is always unfavorable, but life can be greatly prolonged in most cases by judicious treatment.

Treatment.—In an acute dilatation, life can sometimes be saved through venesection, 25 to 30 ounces of blood being promptly abstracted, as so strongly advocated by Osler. The subsequent treatment, and that of chronic cases, resolves itself into the administration of digitalis, with perhaps one or more of the other measures advocated under the treatment of valvular lesions with loss of compensation. The patient should be made comfortable. He is compelled to sit up in bed. By the use of a suitable back-rest, he may be saved from great suffering due to the extreme anasarca that is so common in those compelled to occupy a chair.

DISEASES OF THE MYOCARDIUM.

MYOCARDITIS.

Definition.—An inflammatory or degenerative disease of the muscular substance of the heart. It may be either acute or chronic, and affects in one group of cases the parenchyma, the fibers, in another group the interstitial connective tissue. The inflammation is sometimes limited to a small area and is then, as a rule, suppurative.

Acute Myocarditis.—***Etiology.***—The disease may occur at any age, but it is more common in men. The principal causes are: (*a*) An infectious principle, probably a toxemia, resulting from the acute infectious diseases, particularly typhoid fever and typhus; (*b*) various autointoxications; (*c*) endocarditis or pericarditis, most frequently occurring in the course of acute rheumatism (rheumatic myocarditis); (*d*) embolism of the branches of the coronary arteries, especially in pyemia or ulcerative endocarditis.

Morbid Anatomy.—The lesions may be studied under three heads corresponding to the general parenchymatous, the interstitial, and the circumscribed forms of the disease. (*a*) In the parenchymatous form the muscle fibers are found in a state of granular degeneration, infiltrated with granular matter, opaque and pale, sometimes showing proliferation of the nuclei. The transverse striations are more or less completely lost. (*b*) In the interstitial form the interfibrillary connective

tissue is infiltrated with small round formative cells as if in the initial stage of hyperplasia. (*c*) The circumscribed form is generally due to the lodgment of septic emboli. The process is therefore limited to one or more, generally to many, small areas of the intermuscular connective tissue, and it is generally suppurative in character. Rupture of the small abscesses thus formed sets up pyemic processes in various parts of the body. In some cases, however, the abscesses become encysted and their contents undergo caseous or calcareous change. Fatal rupture of the heart is sometimes induced. A nonseptic form of circumscribed myocarditis is occasionally met with in which limited areas undergo fatty or hyalin degeneration. The heart becomes irregularly dilated. Cardiac aneurism is one of the possible results of this form of myocarditis.

Symptoms.—The symptoms are indefinite and are not distinctive of the lesions. The heart's action usually becomes feeble and irregular; there may be palpitation and slight distress, with more or less pronounced dyspnea, sometimes cyanosis and cold sweats. The pulse is small, soft, irregular, and increasingly weak with the progress of the disease. The lungs may become congested and produce cough. The urine is diminished in quantity. Delirium sometimes develops. The physical signs are generally those of dilatation, a possible accompaniment of the other pathological changes. The sounds are at first strong and clear, while the heart's action is forcible, but they become more and more indistinct as the degenerative changes become more pronounced. Murmurs are sometimes heard which are attributable to the irregular contraction of the muscle bundles in different parts of the heart-wall. They are probably due also in some instances to the dilatation, degeneration of the papillary muscles, or the pressure of abscesses situated near the valves.

Prognosis.—The diffuse form is almost invariably fatal, and sudden death after slight exertion is not unusual. The circumscribed form may be recovered from, but a guarded prognosis should always be expressed.

Treatment.—The treatment is that of acute endocarditis or pericarditis, with which the affection is often associated. Absolute rest is imperative. Heart tonics are not usually indicated, even when the action is irregular and feeble, and their administration is often dangerous. Small doses of strychnin, gr. 1-60 (0.001) or less, may be used with caution, but digitalis would better be omitted. The nutrition should be carefully sustained.

Chronic Myocarditis.—***Definition.***—A chronic proliferative inflammation of the interstitial connective tissue of the heart muscle.

Etiology.—The disease is more frequently met with in men after middle life. The most frequent cause is the presence of toxic matter in the blood. This may be inferred from the fact that the disease is most common in the subjects of chronic alcoholism, syphilis, rheumatism, gout, diabetes, malaria, chronic nephritis, or chronic poisoning with lead or tobacco. It sometimes results from endocarditis or pericarditis and it has been attributed to traumatism of the chest-wall. In many cases it is associated with disease of the coronary arteries.

Morbid Anatomy.—The typical lesion is an induration of the muscle

due to the increase of connective tissue. This may be general, but it is more frequently limited to one or more definite areas, and commonly to the ventricular septum, the region of the apex, and the papillary muscles. The indurated tissue can be recognized, when of sufficient size, by its firmness and gray color.

Narrowing of the orifices, particularly those of the aorta and pulmonary arteries, is sometimes recognizable, or there may be an incompetency of the valves. Compensatory hypertrophy is often present. The intima of the coronary arteries may be sclerotic as a result of the myocarditis, or, on the contrary, as a previous affection bearing an etiological relation to the myocarditis. Fatty degeneration of the muscle fibers is common, and fragmentation and segmentation are generally observed. In the former condition the muscle fibers have been broken transversely; in the latter they have separated along the cement line.

Symptoms.—These are exceedingly indefinite and rarely so numerous or distinctive as to permit an exact diagnosis. The condition is often discovered at autopsy. Disturbances of rhythm are more or less common. The heart becomes irregular in its action, fast, slow, or unsteady, and feeble. This is accompanied with dyspnea, a sense of weight or oppression in the precordial region, physical and mental debility; and later, attacks of cardiac asthma supervene. There may be sudden severe attacks of pain (angina pectoris), more particularly when the coronary arteries are involved. The disease may exist for a long time without producing serious impairment of health, but it sometimes terminates fatally with the suddenness of apoplexy.

Treatment.—The treatment is principally hygienic. The patient should avoid undue exertion, exposure, and excesses of all kinds. Residence in a mild climate during the winter may prolong life. In other respects the treatment should be that of dilatation and other conditions producing feeble, irregular action of the heart. Strychnin in small doses is often the best tonic. The attacks of angina must be treated with morphin and the other remedies recommended under Angina Pectoris.

DISEASES OF THE CORONARY ARTERIES.

The diseases of the coronary arteries are of importance chiefly on account of their effects upon the heart muscle. The principal accidents to the vessels are: (*a*) They may be blocked by emboli; (*b*) they may be the seat of arteriosclerosis, (*c*) thrombosis, or (*d*) obliterative endarteritis. Sudden blocking of one of the coronary arteries is generally followed by instant death. Incomplete closure, coming on slowly, as in arteriosclerosis or obliterative endarteritis, produces degenerative changes in the muscle, together with various clinical manifestations. The coronary arteries, being end arteries, their occlusion produces infarction, which is generally known as anemic or white infarction, and the subsequent necrosis is termed anemic necrosis.

The anemic infarct is most frequently due to embolism of the anterior artery, and the necrotic areas are therefore situated in the left ventricle and septum. The area has not always the wedge shape of other infarctions. It is usually small, slightly elevated, with irregular margins, yellowish gray or reddish gray color, and a white center. The muscle fibers lose

their striations and become granular. They may preserve their firmness to a considerable extent; or they may undergo fragmentation or a more complete softening, especially in the center of the necrotic mass (myomalacia cordis). When the disease does not prove fatal through rupture of the heart or other accident, the necrotic tissue is replaced by a new growth of fibrous tissue, the process constituting a chronic interstitial myocarditis. Aneurism of the heart is a frequent sequel. When the emboli are septic, the process established is one of suppurative myocarditis.

Symptoms.—The symptoms are often exceedingly obscure. Complete closure of one of the coronary arteries may end life instantly. When both coronary arteries are the seat of arteriosclerosis, sudden death may follow thrombosis with only partial closure of either. Sometimes the sudden fatal termination is due to rupture of the heart-wall at the site of an anemic necrosis. In cases which are not immediately fatal the heart's action becomes feeble and disturbed in rhythm, the pulmonary engorgement producing cough and dyspnea. Attacks of angina pectoris occur, and death often takes place during one of them, sometimes even in the first attack. In other cases the patient suffers repeated seizures during a number of years. When the obstruction of the coronary circulation is but partial, the heart muscle undergoes a slower degeneration and the symptoms are of moderate severity.

FATTY HEART.

The heart becomes fatty either as a result of over-accumulation of adipose tissue (infiltration) or as a result of fatty degeneration.

1. **Fatty Overgrowth.**—The former condition is most frequent after the age of forty and in men. In many cases the fat in simply massed beneath the pericardium, especially around and between the auricles and along the auriculoventricular groove, and the condition is a part of a general obesity. In extreme cases the heart becomes completely covered with a thick layer of fat, which impedes its action, and there is often added to this an infiltration between the muscle bundles to a variable depth, occasionally extending to the entire thickness of the wall. Fatty degeneration of the muscle ensues, and in some instances the adipose tissue has been found to have entirely taken the place of the muscle fibers. Dilatation follows the weakening of the walls. The heart is large, yellow, and flabby. The change is generally more common and more extensive in the right ventricle than elsewhere.

2. **Fatty Degeneration.**—This is a common affection and a prompt result of many conditions which cause an impairment of nutrition. In this relation, it is probably more frequent than we are aware. A moderate degree of fatty degeneration may occur at any time of life: (*a*) In connection with the infectious diseases, especially those of septic character, or in the prolonged febrile affections, as tuberculosis and in cancer. In a more extreme form it is met with as a result (*b*) of the primary anemia, (*c*) in advanced age, (*d*) as a result of poisoning with phosphorus, arsenic, and other drugs, or (*e*) following abnormal conditions in the heart, as chronic pericarditis, hypertrophy, or partial obstruction of the coronary arteries from sclerosis, thrombosis, or embolism. The

degeneration is often associated with fatty overgrowth and not infrequently also with fatty degeneration of other viscera, especially that of the diaphragm. The degeneration may involve all the walls of the heart, or it may be limited to those of one chamber, particularly of the left ventricle, often including the papillary muscles. The heart is greatly enlarged, yellow, flabby, in many cases resembling the condition in fatty infiltration; but in other cases neither the size, the color, nor the consistence is much changed, and the degeneration can be determined only upon microscopic examination. (See Fatty Degeneration, p. 22).

Symptoms.—The history of a case of fatty degeneration is exceedingly variable. An extreme grade of either fatty overgrowth or degeneration may exist for an indefinite time without giving rise to definite disturbances. The heart's action continues full, regular, and strong. Much depends upon the condition of the chambers. So long as there is no dilatation the heart may functionate normally. With dilatation the symptoms must be regarded as due rather to this condition than to the degenerative changes in the walls. There is then more or less pronounced dyspnea upon exertion, and possibly slight faintness or complete syncope, and attacks of angina may occur during the night. Edema often develops in the lower extremities. Sometimes the pulse becomes extremely slow, not more than 40 or even 30 beats in the minute, and along with these symptoms the patient has attacks of cardiac asthma. These sometimes accompany or alternate with attacks of angina pectoris. A peculiar type of breathing known as the Cheyne-Stokes is sometimes observed in extreme cases, but it is more common in arteriosclerosis and uremia. The breathing is irregular. About once a minute the respiratory movement ceases for 15 seconds or more; then it is slowly re-established, and each breath becomes stronger until a full, sometimes snoring respiration is taken and the movements stop, or they may grow gradually more feeble and almost imperceptibly cease.

The ***physical signs*** are those of dilatation. The area of dullness is increased, however, in cases of overgrowth before actual dilatation has occurred. The heart-sounds are feeble, but this is often due, in part at least, to the thickness of the chest-wall. Psychical symptoms are sometimes a distressing feature. Various disturbances of the mind, even maniacal seizures, may come on and persist for weeks or months. More or less complete apoplectic attacks may occur. The fatty arcus senilis, a white line in the cornea, is not of the diagnostic significance it was once supposed to be.

The ***diagnosis*** between the different forms of myocarditis is not usually possible. A fatty heart is to be inferred in the presence of great obesity or when the history of the case otherwise points to it as a probable condition. The heart muscles are usually fatty also in a condition of dilatation following hypertrophy, hence it may be inferred to exist in nearly all cases of long-standing valvular disease.

The ***prognosis*** is always grave. Sudden death is likely to occur at any time, in syncope, as a result of overdistention or rupture of the heart. Temporary improvement sometimes occurs in cases which have not advanced to extensive dilatation, but relapse occurs later in all cases. Complete recovery possibly occurs in cases of slight fatty change

in young subjects after prolonged febrile disease, but it is never possible in the aged or in advanced cases.

Treatment.—When an opportunity is afforded for treatment, it should generally be directed against the failing compensation. When the action is irregular, rapid, or weak, and when dyspnea or edema is to be overcome, digitalis may be employed in carefully regulated doses. When, on the other hand, the heart's action is slow and feeble, stimulants are called for, and digitalis should not be given. Ammonium carbonate or the aromatic spirit, and strychnin in small doses (gr. 1-60; 0.001, or less), should be given, but no especial effort should be made to increase the force of the heart's action.

Great benefit is sometimes obtained from the Oertel or Schott method of treatment, particularly in cases of general obesity induced by overeating and -drinking. The essential features of the Oertel method are: the limitation of fluids and fats, and systematic exercise, especially mountain-climbing, each day showing an increase over the previous day. The Schott, or Nauheim, method consists of warm, carbonated salt-water baths and regular, graduated, resisted exercise. The patient makes voluntary movements of the muscles of the arms, thorax, and abdomen, while the operator resists them. The exercise is made more and more vigorous until a decided improvement of the respiratory capacity and power in particular becomes apparent. Muscles which have been permitted to atrophy from disuse are carefully brought out and strengthened in the exercises. The treatment can be carried out at home with little difficulty and often with excellent results.

Other Degenerations of the Heart.—Most of the other degenerations of the heart are of greater pathological interest than clinical.

Parenchymatous degeneration is often associated with fatty degeneration, or it may precede it. In this relation, or independently, it occurs as a result of endocarditis, pericarditis, acute or chronic infection, or intoxication. The histological appearances are described on page 21.

In brown atrophy, the heart becomes reduced in size and is firmer than normal, and it has a dark, reddish-brown color. The muscle fibers become pigmented, especially about the nuclei, and more or less completely lose their striations. It is usually a condition of old age, but may occur as a result of chronic valvular disease. Amyloid, hyalin, and calcareous degenerations are occasionally met with in the heart muscle. They are described under the head of Degenerations, page 23.

Aneurism of the Heart.—1. Aneurism of a valve is an occasional result of endocarditis. It follows an ulceration or erosion that has extended deeply enough to permit dilatation of the valve-segment without perforation. They are found projecting from the ventricular surface of one of the cusps of the valve, most frequently on the aortic, seldom on the mitral valve. Rupture of the little aneurismal sac sometimes occurs.

2. A saccular bulging of the ventricular wall sometimes occurs at a point where the muscle has become weakened by myocarditis, anemic necrosis, or sclerosis. It has been met with also as a result of the gummatous syphilid of the heart-wall, the cicatrization of a stab-wound, and sometimes apparently as a result of pericardial adhesions. They are almost invariably situated in the wall of the left ventricle

near the apex, and they vary in size from that of a walnut to that of the heart itself. The exterior is usually smooth and firm, like the heart-wall, but the interior is lined with superimposed layers of fibrin. The aneurismal sac sometimes communicates with the ventricle by a very small orifice.

The ***symptoms*** are vague. When the aneurism becomes large it sometimes causes bulging or complete perforation of the anterior chest-wall in the region of the apex. The physical signs are so indefinite that the diagnosis is seldom made during life.

Rupture of the Heart.—This rare accident occurs only in a heart that has previously been weakened by disease. In a majority of cases it has been found due to fatty degeneration, but it sometimes follows endocardial ulceration, anemic necrosis, aneurism or gumma of the ventricular wall. Most patients have passed the sixtieth year of age. The rupture is generally found in the anterior wall of the left ventricle, permitting the escape of the blood into the pericardial sac; but it may perforate any of the heart-walls; and when pericardial adhesions are present the blood may be poured into the mediastinal or pleural cavities. In some cases a rupture of the ventricular septum has been discovered. The rupture generally occurs during some exertion, and the result is almost instantly fatal. In a few instances life has been prolonged for a few hours. When instant death does not occur, the patient is generally prostrated, suffers intense dyspnea, with sighing respiration and sense of suffocation, sharp pain or great oppression in the precordial region. The skin becomes moist and cold, the pulse feeble and fluttering. Vomiting may occur. The expression becomes anxious and there is often a distressing sense of impending death.

New Growths and Parasites of the Heart.—Malignant growths are extremely rare in the heart, but secondary growths are sometimes met with in carcinoma, epithelioma, or sarcoma of the mediastinum or lung. The melanotic sarcoma produces numerous small nodules through metastasis. Nonmalignant tumors are even more rare, but fibromata, myomata, and lymphomata have been found. Cysts occasionally form in the heart muscle in extreme fatty degeneration or as a result of hemorrhage or the breaking down of a gumma. Pyemic abscesses have been referred to under Circumscribed Myocarditis.

The parasites that have been found in the heart are the trichina spiralis, the cysticercus cellulosæ, and the echinococcus cyst. They have not been recognized during life.

Wounds and Foreign Bodies.—The more serious wounds of the heart, due to gunshot, stabs, and crushing, are of greater interest to the surgeon or to the pathologist than to the physician. The heart is often injured without immediately serious result, especially by foreign bodies passing through the wall of the esophagus. Penetrating wounds are by no means always fatal. Pricking the heart has been suggested as a final means of stimulating it to action in cases of asphyxia from drowning, chloroform or illuminating-gas poisoning. The symptoms are those of gradual rupture of the heart, with slow hemorrhage. The severity of the symptoms is not always proportionate to that of the injury, however, for profound syncope may follow a trivial injury which is soon recovered from, and a fatal wound may at first occasion little disturbance.

Foreign bodies entering the heart from the esophagus can rarely be recognized during life. The effect is often limited to the production of a pericarditis, with serous or purulent effusion.

Treatment.—The case should be promptly placed in the hands of a surgeon. In the mean time the patient may be given complete rest, with opium if necessary, and an ice-bag to the precordium. Stimulants must not be administered, but some writers favor the giving of aconite to reduce the force of the heart and, with it, the blood pressure.

NEUROSES OF THE HEART.

Palpitation.—***Definition.***—Irregularity in the frequency or force of the heart's action, which is perceptible to the patient.

Etiology.—Irregularity of action characterizes a great many conditions of the heart, especially valvular lesions in the stage of lost compensation, but in this condition the patient is not, as a rule, conscious of the palpitation, and, since the phenomenon depends upon anatomical lesions, it is not strictly a neurosis. There are cases, too, in which severe palpitation is complained of by the patient when the heart's action is perfectly normal. Like all neuroses, palpitation is generally observed (*a*) in nervous, hysterical, or neurasthenic persons, especially women who are naturally excitable or have been rendered so by fright, work, worry, injury, or disease. (*b*) It is often associated with other neuroses, especially those of the stomach, and it is not uncommonly produced by the upward pressure of a distended or dilated stomach. (*c*) Excessive indulgence in alcohol, tobacco, coffee, or tea is regarded as an exceedingly frequent cause. (*d*) Sexual excesses and ovarian or uterine disease, particularly at puberty or the menopause, often incite it, and it is sometimes complained of at the menstrual periods. (*e*) It is a prominent symptom of exophthalmic goiter.

The irritable heart of young soldiers described by Da Costa was attributed to mental excitement, excessive muscular exertion, and the prevalent diarrhea.

Symptoms.—In addition to the sensation of fluttering felt at the heart, the patient usually experiences a peculiar feeling of fullness as though the heart were being distended, or a contrary sense of vacancy and slight faintness. The action of the heart may be so violent as to become visible, and the arteries may throb. The pulse becomes for the time extremely rapid and the patient may gasp for breath. There is sometimes distinct flushing of the face, sometimes pallor. Gaseous eructations and other nervous or hysterical manifestations commonly accompany the attack. The physical signs are not definite. The sounds may all be normal in character, but are, as a rule, accentuated and metallic in quality. An anemic bruit or a murmur of relative insufficiency is often heard at the base, less frequently at the apex. The attack is usually of short duration, subsiding after a few minutes or at most after a few hours, to recur upon any trivial provocation, especially after excitement or exertion.

The ***prognosis*** is generally good with reference to life, but the affection often proves resistant to treatment, and hypertrophy often remains.

Treatment.—The most important therapeutic element is the removal of the cause. If it be an excessive indulgence in alcohol, tobacco, or other stimulant, it must be overcome; if a disorder of digestion, it should receive appropriate treatment. In hysterical cases the treatment is largely moral. Care should be exercised not to impress the patient too strongly with the gravity of his illness, nor yet to treat it too lightly. All excesses should be avoided, regular exercise should be taken; the mind should be kept free from worry. It is generally better to abandon entirely the use of alcohol, tobacco, and coffee. Tonics may be required in some cases, especially iron, arsenic, and strychnin, when anemia is present, but, as a rule, the administration of drugs should be abstained from as far as possible. All forms of hot baths should be avoided, but a tepid bath may be taken mornings or evenings and followed with rubbing.

ARRHYTHMIA.

Loss of Rhythm.

This is observed in two different degrees. In the lesser only the volume and force of the pulsations are altered, a series of full, strong, pulse-waves alternating with a series of weaker pulsations. In the more pronounced arrhythmia, the irregularity becomes so great that one or more waves are lost before reaching the radial artery at the wrist. This may occur at regular or at irregular intervals. One beat may be lost after every series of three or four, or there may be no regularity in the omissions.

Several forms of irregularity are generally selected for description, their frequency rendering them more or less typical. Among these are: (*a*) *Intermittency*, in which every third, fourth, fifth, or perhaps every tenth beat is omitted. It is not usually a condition of serious import; (*b*) the *bigeminal* or *trigeminal* pulsation, two or three systoles occurring in quick succession, to be followed by an increasing interval. An apparent bigemism is often produced when the impulse of the second systole is lost before it reaches the radial artery, although a faint systole may be heard upon auscultation. This condition is sometimes a result of the administration of digitalis. The purely nervous intermission is to be distinguished from the irregularity due to valve lesions, which is of more serious import. As a neurosis, it is the "nervous trick" referred to by Fothergill, but when due to organic disease it signifies failing compensation.

(*c*) The *paradoxical pulse* indicates weakness, fibrous adhesions about the root of the aorta or a chronic pericarditis. The pulse becomes feeble and usually more rapid during inspiration. It is not necessarily of unfavorable prognosis, and in some cases it is probably not strictly pathological, depending only upon respiration.

(*d*) *Delirium cordis* is the term applied to extreme irregularity of action and force seen when complete loss of compensation occurs in valvular disease or in extreme cases of exophthalmic goiter.

(*e*) The *gallop*, or *cantering rhythm*, is a form of rapidity in which the heart-sounds resemble the foot-falls of a horse in a cantering gait. The irregularity of action can sometimes be recognized on inspection and palpation as well as on auscultation. A third sound is generally to

be recognized owing to a reduplication of one of the normal sounds, the second, as a rule. The condition develops in hypertrophy due to arteriosclerosis, chronic interstitial nephritis, or profound anemia, and sometimes in the myocarditis of the acute infectious diseases.

(*f*) *Embryocardia*, or a fetal-heart rhythm, is acquired through a shortening of the long pause, the first and second sounds at the same time becoming more alike. The sounds are to this extent more like those of the fetal heart. The condition develops in some cases of dilatation and sometimes in connection with the infectious fevers.

Etiology.—The cause of arrhythmia is reflex; the impulse may arise: (*a*) In the central nervous system after hemorrhage, trauma, or psychical disturbance; (*b*) it may be peripheral, when it arises from disorders of the stomach, liver, lungs, or kidneys, or from disturbance of the blood pressure in arteriosclerosis and other obstruction, or from profuse hemorrhage, (*c*) or from toxic irritation, as that of alcohol, coffee, or tobacco. To these causes are generally added the influences of organic diseases within the heart itself, including degenerations, dilatation, and changes in the ganglia. Some writers refer to a physiological arrhythmia embracing cases of irregularity observed in children during sleep, sometimes also in adults, and more commonly in very old persons.

Treatment.—The principal therapeutic indication is the cure of the underlying nervous condition. Drugs should seldom be administered for the immediate condition.

Tachycardia (Rapid Heart, Heart-Hurry).—A rapid action of the heart, even exceeding 100 beats in the minute, is sometimes normal to the individual. When a nervous condition, the rapid action may be constant, or it may occur in paroxysms of variable duration.

Etiology.—The causes are for the most part the same as those of palpitation, but, unlike the latter condition, the patient is often unaware of the rapid action. The direct cause is probably either a stimulation of the sympathetic nerves of the heart or a suspension of pneumogastric control. The condition often follows some great nervous excitement, as in mania, fright, fear, or violent exercise, the rapid action suddenly produced persisting for many days. In women the irritation is often regarded as reflex from the uterus or ovaries, and it is often encountered at the menopause. Toxic cases are recognized as resulting from the action of tobacco, belladonna, digitalis, alcohol, and other drugs. Cases of tachycardia occur also as a result of anatomical lesions, especially in connection with an inflammation, blood-clot, or tumor in the medulla or causing pressure on the pneumogastric nerve, as well as in some cases of myocarditis or sudden dilatation. Martius regards the paroxysmal form as due to sudden, periodical dilatation. The pulse-rate during the paroxysms varies from 100 to more than 200 in the minute. Each attack varies from a few minutes to several hours, and they often recur after intervals of only a few days. The patient may be conscious of the increased rapidity of the heart, or he may experience only a vague feeling of uneasiness in the cardiac region. Dyspnea is not usually present, but the patient may be compelled to sit down or to assume a recumbent posture. The attacks are rarely fatal except in persons of advanced age; they often recur at variable intervals for many years without serious consequences.

Treatment.—The patient should immediately lie down, and, if the rapid action continue, an ice-bag should be placed over the heart. A physician referred to by H. C. Wood obtained immediate relief during many years from drinking ice-water or hot coffee. In severe attacks, morphin should be administered hypodermically. Strophanthus, aconite, and other heart remedies have proved of benefit. Complete recovery is extremely rare, however, and the exciting causes, particularly nervous excitement, should be carefully guarded against.

Bradycardia (Brachycardia, Slow Heart).—Slow action of the heart is sometimes met with as a normal condition; it is occasionally a family peculiarity. It is probably much less frequent as a pure neurosis than in other relations. The former cases are generally associated with other neuroses, as in neurasthenic, hysterical, melancholic, maniacal, paretic, or epileptic subjects, or in persons of extreme age. Attacks sometimes follow intense suffering or the emotions of fright or grief.

(1) A *physiological bradycardia* is recognized as a result of exhaustion, inanition, or as a feature of the puerperal state.

(2) *Pathological bradycardia* is common: (*a*) During convalescence from the acute infections, especially typhoid fever, pneumonia, diphtheria, rheumatism, and meningitis; (*b*) in chronic diseases, as dyspepsia, ulcer, cancer, or dilatation of the stomach, pulmonary emphysema, and diseases of the liver; (*c*) in intoxications by lead, alcohol, coffee, tea, digitalis, aconite, or the autointoxication of diabetes, chronic nephritis, anemia, and diseases attended with jaundice; (*d*) sometimes in diseases affecting the heart, as the degenerations, arteriosclerosis, myocarditis, affections of the coronary arteries, and very rarely in connection with valvular lesions. (*e*) It is occasionally met with in sunstroke and in diseases of the skin or of the sexual organs. A peculiar form of the affection is that described by Adams and Stokes, in which the pulse is constantly slow and the patient suffers occasional attacks of syncope.

Diagnosis.—The condition is readily recognized, but it should not be diagnosticated from the pulse alone. The heart should be examined by auscultation, for in some instances the radial pulse indicates a slowness that is not real, but due to a failure of the impulse to reach the radial arteries.

Treatment is of little avail unless the underlying condition can be discovered and removed. Tonic doses of strychnin are beneficial in some cases.

ANGINA PECTORIS.

Neuralgia of the Heart, Breast Pang, Stenocardia.

Definition.—A paroxysm of intense precordial pain usually extending into the neck and arms and attended in severe cases with a sense of imminent death. It is a symptom of several pathological conditions of the heart and blood-vessels, especially sclerosis of the aorta near its origin or of changes in the coronary arteries; it is probably never an independent disease.

Etiology.—This affection occurs only in adults, and much more fre-

quently in women than in men. It sometimes follows the line of heredity. In a vast majority of cases arteriosclerosis of the coronary arteries or of the aorta is the direct cause; other cases are associated with myocarditis, adherent pericardium, or aortic incompetency, and very exceptionally with a mitral lesion. Diabetes, gout, and syphilis are commonly observed in the antecedent history of the patient, and in recent years influenza has appeared to bear a causal relation to many cases. The exciting cause in most cases is an influence which throws the heart into violent action, as an unusual, sudden exertion, shock, or such emotion as anger or fright. An attack often follows overdistention or sudden gaseous inflation of the stomach, or chilling of the body as by getting out of bed at night.

Theories of Angina.—The exact nature of the disease is not known. Many interesting theories have been offered, but none has been confirmed. The following are the more important of them: (1) That it is a neuralgia of the cardiac nerves induced chiefly by sclerosis of the coronary arteries or by changes in the ganglia within the heart or in the pneumogastric or phrenic nerve; (2) Heberden's theory, that it is a cramp of the heart muscle, now regarded as highly improbable; (3) that it is due to sudden increase of tension within the ventricles due to dilatation (Traube and others); (4) Allan Burns's theory, that it is due to ischemia of the heart muscle, caused by disease or spasm of the coronary arteries.

Symptoms.—The patient is suddenly seized with a most agonizing pain in the heart. The suffering is so intense that he is rendered motionless. He grasps any near object and hangs upon it as if for support, afraid to move and almost afraid to breathe. The sensation is as though the heart were being crushed in a vise, and the pain radiates up into the neck and down the left arm, sometimes into both arms and down the back. Some of the cervical and all of the dorsal areas to the ninth may be included; generally all above the seventh are painful, while in organic disease of the heart and in aortic affections only the upper four are usually involved. There is no pain so intense, no moment so full of anguish, no experience so terrifying. The face becomes pale and ashen; a cold sweat starts, and the patient may sink in a faint from which he does not recover, or death may be instantaneous. It is a remarkable fact that the heart's action may be little disturbed during the attack; its action may be full and regular, but the radial pulse is generally increased in tension and perhaps in force. Sometimes the paroxysm is accompanied with distinct bronchial wheezing and the patient may experience a sensation of suffocation. Flatulent distention of the stomach is an almost constant symptom. The seizure generally lasts only a few seconds or at most a few minutes. It often subsides with an eructation of gas from the stomach, and a large quantity of clear urine is often voided, as after other nervous paroxysms.

A vasomotor angina has been described in which the cardiac pain is preceded by pallor, coldness and cramping of the limbs. The paroxysms are not of the highest grade of severity.

Diagnosis.—In a well-marked case the diagnosis is comparatively easy, but many attacks of moderate severity may occur in which it is

extremely difficult to decide whether the seizure is one of true angina or of pseudoangina, a name applied to a large group of cases not conforming to the description just given. The contrast between the two conditions is sufficiently clear, however. Bearing in mind the clinical picture of true angina—a primary seizure occurring in men over 45, after exertion, excitement, or cold, a paroxysm of short duration, but attended with excruciating pain radiating to the neck and arms, rendering the patient silent, motionless, and breathless, the picture of despair—pseudoangina is characterized by very different features. The patient may be of any age, often a woman with a history of hysteria, the attack often recurring periodically and perhaps spontaneously at night, lasting an hour or two, the pain less severe, and the patient often exhibiting violent hysterical or nervous movements. It is never fatal.

Toxic Angina.—Attacks of severe pain in the region of the heart resembling angina pectoris are frequently met with as a result of excessive indulgence in tobacco, less frequently from coffee- or tea-drinking. The pain is generally paroxysmal or shooting in character, with irregular, intermittent palpitation and real or fancied dyspnea. The patient is usually greatly alarmed, profuse sweating generally occurs, the extremities are cold, and there is often a tendency to syncope with extreme weakness of the circulation, and nausea. The attack usually lasts several hours, and it may recur at increasingly short intervals if the cause be continued.

Prognosis.—True angina pectoris is an exceedingly dangerous affection, the patient often dying during the attack or with equal suddenness and without warning during the interval. Pseudoangina, on the contrary, is never fatal, and the toxic form is rarely so.

Treatment.—Prophylactic treatment is important, but it is unfortunately very limited. Nothing can be done to remove the sclerotic or calcareous condition of the blood-vessels, but the patient may be removed from the usual exciting causes, and he should always be provided with the most efficient remedies for the relief of the paroxysm, especially with the perles of amyl nitrite, each containing 3 or 5 drops, to be crushed in the handkerchief for inhalation.

The treatment of the attack must be prompt. Immediately upon the inception of the pain, the patient should crush a perle, and inhale as deeply and as fully as possible the fumes of the drug. Unfortunately, however, it is not always effective and occasionally rather increases the suffocation. A few drops of chloroform inhaled in the same manner may prove more effective, but in some cases only morphin is capable of affording relief. This too must be given in increasing dosage if often required. Glonoin, ♏j (0.06), hypodermically is sometimes of benefit. Either hot or cold applications to the precordial region may give relief.

In the intervals the most careful attention should be given to the correction of any abnormality in the action of the heart. When the arterial tension is high it may be reduced with aconite or nitroglycerin, in gradually increasing doses, at first repeating the one-minim dose three times during the day. Sodium nitrite has also been recommended. Potassium bromid is of benefit in some cases. It is probably only in syphilitic cases that decided benefit is to be expected from a prolonged course of potassium iodid, so highly recommended by some writers.

The treatment of pseudoangina should be directed to the removal of the cause. Tonics are generally indicated. The existence of a gouty taint should not be disregarded in either form of angina.

CONGENITAL DEFECTS OF THE HEART.

A great majority of the congenital defects of the heart are entirely devoid of clinical interest; some of them are, in fact, incompatible with life. Occlusion of the pulmonary orifice, with patency of the foramen ovale and perhaps deficiency of the ventricular septum, constitutes the only condition likely to call for the attention of the physician.

Symptoms.—Extreme cyanosis is the most striking symptom. The infant appears blue, especially after the exertion of crying. This is generally observed as early as the second week of life. The cyanosis may be general or it may be confined to the extremities. If life be prolonged, the child is generally poorly developed, in part, no doubt, because of its inability to engage in active exercise. The activity of play renders him livid and produces intense dyspnea. The fingers and toes early become clubbed. The surface of the body is always cool. Cough is constantly present. Physical examination reveals enlargement of the heart, and murmurs are heard that do not conform to any recognized lesion of the valves. Increase of the red blood-corpuscles is a prominent feature and sometimes reaches nearly double the normal number in the millimeter.

Prognosis.—"Blue children" rarely live to puberty, but a few have been known to attain their majority. Death may result from the defect or from an intercurrent disease, especially bronchopneumonia.

Treatment.—The child should be protected from cold and other influences liable to increase the bronchial congestion. Venesection may be practiced when paroxysms of cyanosis and dyspnea threaten life. The action of the heart may be to some extent improved by the administration of digitalis or the other remedies usually employed in valvular disease.

DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

An acute inflammation of the intima of the aorta similar in character to acute endocarditis and often associated with it.

Etiology.—Whether an independent affection or associated with endocarditis, the causes are practically the same as those of the latter disease, especially rheumatism and the other acute infectious diseases; alcoholism, lead-poisoning, and other intoxications.

Morbid Anatomy.—The intima may be thickened, hyperemic, and covered with a layer of fibrin, the lesions resembling those of simple endocarditis, or there may be circumscribed destruction of tissue as in malignant endocarditis.

Symptoms.—Pain and dyspnea are the distinctive features. The pain varies from a sense of soreness or tenderness beneath the sternum to a sharp stabbing along the arch of the aorta and reflected to the right shoulder. Palpitation is sometimes observed, and the subclavians may

be felt throbbing above the clavicles. Moderate elevation of temperature usually accompanies the attack. The acute symptoms generally subside in a few days, but the inflammation often passes over into a chronic arteriosclerosis. The differential diagnosis from endocarditis is difficult, resting upon the greater diffusion of the pain and the absence of murmurs indicative of valvular involvement.

The ***prognosis*** is grave, owing to the possibility of embolism or rupture of the aorta.

Treatment.—Absolute rest, with light diet, and an ice-bag over the sternum, are the principal measures of relief. Aconite and opium may be employed for the dyspnea, especially when the heart's action is rapid and irregular. When fever and other symptoms of sepsis arise, the case should be handled with a view to overcoming that condition.

ARTERIOSCLEROSIS.

Arteriocapillary Fibrosis, Atheroma.

Definition.—A degeneration of the coats of the arteries, beginning in the intima, followed by hyperplasia of the connective tissue of all the coats, and terminating in contraction and rigidity, greater or less deformity of the vessel, and diminution of its lumen. The impairment of function is often greatly increased by subsequent calcification.

Etiology.—(1) The disease is rare before the fortieth year. During the declining years of life there is in many individuals an inherent tendency to this hardening of the arteries. (2) Men are more subject to it than women, because they are more generally exposed to the other factors of its production. These are for the most part overwork, worry, excitement, malnutrition, and various intoxications. (3) Notwithstanding these influences, however, there is great difference in individual susceptibility. Some families appear to be predisposed to the affection. The predisposition probably depends in part upon an inherited type of tissue and in part upon similarity in the habits, food, and mode of living. The disease is frequently met with in high-livers, especially those who take little exercise and rapidly accumulate adipose tissue. Excessive consumption of meat is regarded as especially influential by increasing the nitrogenous waste. (4) The chronic intoxications most likely to produce the affection are alcohol, lead, uric acid, and those of syphilis and other diseases. (*a*) Alcohol probably acts by overstimulation of the heart, disturbing the function of the stomach, liver, and kidneys, and thus increasing the production of waste products while impeding their elimination. Uric acid and lead probably act directly upon the tissues of the blood-vessels and to some extent interfere with the peripheral circulation and thus increase arterial tension. (*b*) Syphilis operates chiefly through the formation of gummatous infiltration of the vessel walls, leading directly to sclerosis. Most cases occurring in early life are referable to this influence. (*c*) The other diseases generally regarded as influential are chronic interstitial nephritis, tuberculosis, chronic rhumatism, diabetes, and malaria. They doubtless operate through the production of toxic matter in the blood, or by interfering with its elimination. The most important relation in the latter connection is that of arteriosclerosis with chronic interstitial nephritis. In many

instances the two conditions are, no doubt, a part of the same process, a general arteriosclerosis, affecting the smaller vessels of the entire body; but in another group of cases the sclerotic condition of the kidneys precedes that in the blood-vessels. General neuritis and other sclerotic changes in the nervous system are sometimes associated with arteriosclerosis, but probably as a result of the same influences, and not as causal factors.

Morbid Anatomy.—The lesions may be confined to the aorta, the vessel most frequently affected, or they may be more or less uniformly distributed throughout the arterial system. The other vessels are affected in nearly the following order: The radial, splenic, iliac, femoral, coronary, cerebral, brachial, common carotid, vertebral, and the peripheral branches. The vessels subjected to the greatest strain are generally most markedly affected; those of the alimentary canal, liver, and mesentery are seldom involved. The changes sometimes extend beyond the smaller vessels, into the capillaries, and rarely also into the veins. The lesions generally conform to one or other of two forms designated circumscribed or diffuse.

1. *Circumscribed Arteriosclerosis.*—Definite areas of the intima become thickened and opaque. These are often hemispherical in form, yellowish in color, and they are more frequently situated about the orifices of the branches. Their thickness generally bears a close relation to their diameter and increases with it. Histologically the change begins as an infiltration and degeneration of the middle and external coats of the vessels, especially about the vasa vasorum. This is followed by a thickening of the intima as a compensatory process to fill the slight depression that would otherwise remain, and to compensate for the loss of strength in the vessel-wall. The thickening is followed by hyalin degeneration, however, and the middle tunic is often found in a state of granular disintegration or necrosis (atheromatous abscess). As a result of this weakening of the walls the vessel often becomes greatly dilated, and an aneurism may be formed. In some cases, however, the rapid development of connective tissue prevents dilatation and by subsequent contraction brings about a diminution of the caliber of the vessel.

2. *Diffuse Arteriosclerosis.*—The intima often appears smooth, but it may show nodular prominences, owing to the association of the circumscribed form of the disease. This is usually the condition found in the aorta. On section of the vessel, the walls are found to be greatly thickened, the greatest increase in many cases affecting the intima. Microscopic examination reveals a highly degenerated media, often general in character, and a marked proliferation of the subendothelial connective tissue. The muscular and elastic tissues are sometimes entirely destroyed. Subsequent degeneration and necrosis of the media follow, and calcareous plates are often formed in the larger vessels. The immediate result of the sclerosis is to diminish the elasticity and usually, to a considerable extent, the lumen of the vessel. This increases the resistance to the flow of blood and consequently raises the arterial tension. More work is thrown upon the heart, and the response is a hypertrophy of the left ventricle. The more remote results are, diminished supply of blood to the organs and tissues through the affected vessels, with consequent impairment of their functions, anemia and degeneration, sclerosis or possibly

necrosis. Thrombosis and its results are often produced by arteriosclerosis.

Symptoms.—No single description can be made to include all the clinical manifestations of the disease. In many instances the condition is in fact latent, and pronounced lesions are found after death in persons who had made no complaint leading to their discovery. The manifestations are peculiar to the area affected and the extent of the lesions. When the vessels are extensively or generally involved, an increase of arterial tension is one of the most distinctive features. But the extent of the sclerosis cannot always be estimated in this manner. High tension is recognized for the most part by its effect upon the pulse. The wave is slow in ascent and descent, and the artery remains full between the pulsations. The vessel is firm and so hard that it cannot be fully compressed under the finger, neither is it possible through pressure to entirely overcome the pulse-wave. Sometimes the calcification of the artery becomes so extreme that the pulse-wave becomes imperceptible. The vessel then feels hard and sometimes rough. Hypertrophy of the left ventricle is produced by the increased work thrown upon it. It is generally a simple, pure hypertrophy, without dilatation. The sounds are little disturbed, except that the aortic second sound is accentuated. The aorta becomes markedly dilated in some cases, causing an increase of the area of dullness in the upper sternal region, and the arch may sometimes be felt by pressing down with the finger in the suprasternal notch. The increased pressure may lead to an increase of the quantity of urine voided, with low specific gravity, like that of chronic interstitial nephritis, and the latter condition is, indeed, often present. In many instances, however, the general health of the individual is little, if at all, impaired.

Many other symptoms are occasionally encountered as a result of secondary degeneration of the myocardium or in the region most involved in the arteriosclerosis. Dyspnea, precordial uneasiness, and palpitation are commonly complained of; and when the coronary arteries become involved in the sclerosis, as they frequently do, attacks of angina pectoris supervene. Late in the disease dilatation of the heart may succeed to the hypertrophy, and a train of symptoms is produced which suggests valvular disease with loss of compensation. A murmur may be heard at the apex owing to relative insufficiency of the mitral valve, the arterial tension is reduced, the second aortic sound loses its strong, ringing quality, the urine becomes scant, and serous effusions may occur. The true nature of the disturbance cannot well be determined without a knowledge of the previous condition.

Sclerosis of the arteries of the brain excites a great variety of symptoms. Vertigo, headache, tinnitus, melancholia, insomnia, aphasia, hemiplegia, and monoplegias are all observed, singly or in various combinations, but they cannot be referred with accuracy to definite lesions within the brain, further than that they are associated with the different lesions of arteriosclerosis, including miliary aneurisms and their rupture. When the vessels of the spinal cord are affected, the symptoms conform with more or less exactness to chronic myelitis, multiple sclerosis, locomotor ataxia, syringomyelia, or general paresis. Involvement of the arteries of the extremities leads to numbness, persistent tingling,

coldness or cramps, purpuric eruptions, and sometimes to thrombosis and gangrene. Finally, arteriosclerosis is the essential element in the production of aneurisms in any part of the body.

Diagnosis.—As has been already stated, the disease may be so obscure as to escape recognition during life. In many cases, however, its recognition is possible if search be made for it. In the presence of induration of the radial and temporal arteries, hypertrophy of the heart, and other evidences of increased arterial tension, as shown in the character of the pulse and the accentuation of the aortic second sound, together with other evidences of premature senility, the diagnosis becomes manifest, and it is further confirmed by the discovery of renal changes and ossification of the costal cartilages. The calcification of the arteries can sometimes be distinctly recognized with the aid of the X-ray. After dilatation of the heart has developed, with valvular incompetency, the diagnosis is obscured, unless the hardness of the peripheral arteries can be felt or the presence of calcareous plates can be recognized in the fluoroscope.

Prognosis.—The disease generally progresses slowly and leads ultimately to a fatal termination. The end may come suddenly through rupture of the diseased vessel or the development of thrombosis in the coronary arteries, or it may come with a slowness that is rendered burdensome by the repeated development of gangrene in the extremities, or by gradual wasting and extreme feebleness. Many apparently serious developments, particularly those of the cerebral group, are often partially and occasionally completely recovered from.

Treatment.—The extent to which the progress of the disease can be influenced is uncertain. Good might be accomplished by instruction in the regulation of the diet and avoidance of the causes which induce the disease, to the young members of the families in which a predisposition is known to exist. After the disease has been recognized, the patient should be brought to realize the importance of abstemiousness in food and drink, regular exercise, the avoidance of worry and excitement, strain or heavy lifting, and probably, above all, the importance of maintaining a regular action of the bowels and kidneys, in order to prevent the accumulation of toxic matters in the system. If the history of syphilis is obtained, a vigorous course of treatment should be pursued. Potassium iodid is regarded by some writers as of great benefit in non-syphilitic cases. Large doses are not necessary. For the symptoms due to increased arterial tension, nitroglycerin often proves beneficial. Blood-letting is probably not practiced so often as it should be in cases of this character, for it affords a positive means of warding off, for a time at least, the serious results of extreme distention of the weakened vessels.

ANEURISM.

Definition.—A circumscribed dilatation of an artery. *Classification:* (1) A true aneurism is one in which the wall is formed of one or more of the tunics of the vessel. (2) The so-called false aneurism is a blood-sac formed by the rupture of all the coats of the vessel, and the retention of the blood in the surrounding tissues. (3) An arteriovenous aneurism is formed by the establishment of a communication between

an artery and a vein. When a sac is formed between the artery and vein, it is known as a varicose aneurism; but when the communication is direct and the dilatation is composed principally of the vein, it is called an aneurismal varix. (4) The dissecting aneurism is one in which the dilatation is formed between the coats of the vessel-wall as a result of perforation of the intima. (5) In situation, aneurisms are axial when they involve the entire circumference of the vessel, and peripheral when they are confined to a side of the vessel. (6) In form, they are cylindrical, saccular, fusiform, or circoid, the latter term being applied to the dilatation of an artery and its branches. (7) The term miliary aneurism designates a small, often microscopic dilatation, usually present in large numbers, in the course of a vessel, especially in the brain. (8) External aneurisms are sometimes referred to as surgical, and internal as medical.

Etiology.—Congenital aneurisms are very rarely met with, and they are generally multiple. In adults they usually develop between the ages of 30 and 50, and much more commonly in men. The aorta is more frequently involved than any other vessel, but complete exemption can hardly be claimed for any of the arteries. The frequency of the disease in the aorta is greatest immediately above the heart, and diminishes in an almost constant ratio with the distance from the heart. The same rule applies to its branches.

All cases of aneurism depend upon a natural or acquired weakness of the arterial walls. In a great majority of cases it is a result of: (*a*) Arteriosclerosis with its consequent weakening of the media or of all the tunics. (*b*) The most frequent predisposing cause is syphilis, which is recognizable in about 75 per cent of the cases. Alcohol is doubtless another. (*c*) Sometimes it is attributed to trauma, as a fall upon the back. (*d*) Whittaker emphasizes the importance of the strain upon the vessels occasioned by hard work, in connection with the other influences. Unusually sudden pressure, like that caused in lifting, athletic exercises, or strong emotion, has been regarded as the exciting cause in some cases. (*e*) Aneurism occasionally follows the lodgment of an embolus. The degeneration of the vessel-wall is sometimes directly due to the embolus. (*f*) In a number of cases multiple aneurisms have been traced to the action of pyogenic bacteria derived from a malignant endocarditis or other suppurative focus (mycotic aneurism).

Symptoms.—The clinical manifestations depend so much upon the location and size of the aneurism that few general statements can be made. The distinctive features of a fully developed aneurism in a part accessible to inspection and palpation are those of a tumor with expansile pulsation, a thrill and double murmur, often transmitting the heart-sounds and producing pressure symptoms of various kinds. But these symptoms may one or all be absent. Only the pressure symptoms and the bruit are generally observed in internal aneurisms, until the sac has become so large as to produce erosion of the overlying tissues or to render its pulsation recognizable through them.

Aneurism of the Aorta.—The symptoms vary with the part of the vessel affected and the location of the dilatation with reference to surrounding viscera, whether on the anterior, posterior, or lateral surface of the artery.

Pain is one of the earliest and most constant symptoms in deep-seated aneurisms, often occurring in paroxysms of great severity, like those of angina. It is most severe in rapidly enlarging tumors, and after the pressure becomes so great as to cause erosion of the vertebræ, ribs, or sternum. *Cough* is produced by pressure on the trachea or bronchi, and a bronchitis is often developed, with abundant watery expectoration, becoming purulent and more viscous. *Dyspnea* is common, especially when the transverse portion is affected. It may be due to pressure on the left bronchus, the trachea, or the recurrent laryngeal nerve, usually the left. *Hoarseness* or *aphonia* accompanies the dyspnea, as a rule, when it depends upon this nerve pressure, since it causes spasm or paralysis of the muscles controlling the vocal cord of the affected side. *Hemorrhage* is sometimes induced by the pressure on the bronchus or by the congestion of the lung. A slow oozing of blood preceding for a short time the final rupture of the sac sometimes occasions more or less distinct hemoptysis, or the blood may accumulate in the stomach, to finally induce vomiting and rupture of the aneurism in the effort. When rupture occurs, there is generally a profuse gush of blood, followed by instant death. The rupture often occurs, however, into one of the thoracic cavities or into the esophagus, and there is no visible indication of it. Compression of the esophagus sometimes causes obstruction, and deglutition becomes difficult or impossible. The aneurism has been perforated in attempts to pass the esophageal bougie. The thoracic duct may also be compressed. Pressure on the sympathetic nerves causes dilatation or contraction of one pupil. Pressure on the veins causes engorgement, especially of the head and arms. Pallor of one cheek is sometimes noted.

Physical Signs.—*Inspection.*—Pulsation can generally be detected by close examination in a good light. It is in most cases seen in the right second intercostal space, sometimes in the left, or it may be seen in the suprasternal notch when the transverse portion and the innominate are involved. As the tumor increases in size, bulging becomes manifest. It can be seen best by looking across the chest or downward over the shoulder. The protrusion generally involves the first and second right interspaces and sometimes a part of the sternum, or, if the sac is immediately above the heart, it may cause bulging of the third interspace close to the left of the sternum. Aneurism of the descending portion seldom causes bulging, but it may cause prominence of the second or third left interspace or rarely of the left scapular region.

After erosion has become complete, a pulsating tumor appears, covered only by the skin, which becomes hyperemic and may slough so as to expose the external surface of the sac. The impulse of the heart is often displaced downward as a result of pressure, and may be strong at either the right or left of the sternum. The apex beat is correspondingly low and often beyond the left mammary line.

Palpation.—The pulsation is strong, usually heaving, and expansile. When the tumor has not reached a size to be grasped in the hand, the expansile quality can sometimes be recognized by pasting strips of paper over it and noting their separation with each systolic impulse; or it may become perceptible when one hand is placed over the prominence and the other on the spine. A sharp diastolic shock can sometimes be

felt, especially in aneurism at the root of the aorta. It is synchronous with the closure of the aortic valves, and a valuable sign. A systolic thrill or fremitus is more generally felt over a large dilatation of the aorta than over a saccular one.

Tracheal tugging is a valuable sign when it can be obtained. It is elicited by having the patient sit erect with his mouth closed and the chin elevated almost to the full extent, the head resting against the breast of the examiner, standing behind him. The cricoid cartilage is then grasped between the finger and thumb and gently but steadily drawn upward. If the aorta be dilated, its pulsation can now be distinctly felt, transmitted through the trachea to the fingers.

Percussion.—A deep-seated aneurism produces no perceptible change; but when the tumor reaches the chest-wall or causes bulging, a peculiar flat note and resistance can be recognized that are unlike those of a consolidated portion of lung. The flat area varies with the situation of the tumor; it is on the right side of the sternum in an aneurism of the ascending portion; under the upper part of the sternum and to the left in that of the transverse portion, and usually in the left interscapular and scapular regions in that of the descending portion.

Auscultation.—A murmur is usually heard, but it may be absent even in aneurisms of large size when the layer of fibrin on the interior of the sac is thin or presents a smooth internal surface. It is systolic in time and is transmitted along the blood-vessels so as to be heard in the neck and along the course of the aorta, but with greater intensity immediately over the tumor, corresponding to the area of flatness on percussion. It is often accompanied by an aortic regurgitant bruit, which gives the impression of a double murmur, and it is then a more characteristic sign than when it occurs alone, denoting, as a rule, an aneurism at the root of the aorta. The murmur of regurgitation may be heard when the aneurismal systolic bruit is absent. A systolic bruit can sometimes be heard over the trachea or at the open mouth. It is perhaps due to the expulsion of air from the lung by the expansion of the aneurismal sac.

The Pulse.—Delay of the pulse in the vessels beyond the aneurism is a sign of great value in many cases. A distinct interval can be recognized between the time of the radial pulses, as well as a diminution of the volume of the one on the affected side. Osler observed a complete obliteration of the pulsation in the abdominal aorta and its branches in a case of very large aneurism situated on the descending portion of the thoracic aorta. Ophthalmoscopic examination may reveal strong pulsation of the retinal vessels on the affected side, and little or none on the other side.

Diagnosis.—The recognition of the aneurism becomes difficult when (*a*) the sac is deep-seated and of small size. There may be only a feeling of discomfort within the thorax, but periodical pain radiating to the left shoulder is often of significance, especially if other pressure symptoms, as cough, dyspnea, and bronchitis, be present. (*b*) Aneurism of the ascending portion of the arch has been called the aneurism of physical signs, and that of the transverse portion the aneurism of symptoms; the latter is always more difficult of recognition. Pressure symptoms are generally produced, but they are not sufficiently distinctive

of the affection. In some cases the manifestations are observed only periodically. (*c*) Another class of cases is rendered obscure by the predominance of symptoms on the part of the respiratory system suggesting bronchitis or bronchiectasis. It is sometimes possible in such cases, by laryngoscopic examination, to recognize the compression of the trachea or a beginning paralysis of the vocal cord. The principal affections to be excluded in arriving at a diagnosis are abnormally strong pulsation of the aorta, pulsating empyema, and solid tumors. Violent throbbing of the aorta is generally secondary to aortic insufficiency. The pulsation of an empyema is not expansile and there is no bruit or diastolic shock; the pulsation is generally diffused over the chest. Solid tumors with transmitted pulsation are sometimes differentiated with difficulty, but the pulsation is quite different to the touch, and it is not expansile. The shock is also absent. In deep-seated tumors, however, owing to the similarity of the pressure symptoms, a differential diagnosis may be impossible. The most valuable signs are the absence of murmur, of the ringing aortic second sound, and tracheal tugging. The X-ray has proved a valuable aid to diagnosis in obscure cases.

Prognosis.—There are few diseases of more serious import than thoracic aneurism, and yet spontaneous recovery is possible. Even when complete recovery does not occur, many years of comfort may be secured, but there is ever hanging over the patient the possibility of a sudden rupture of the weakened aortic wall. Death usually ensues from rupture of the sac, but occasionally from other diseases induced by the aneurism or from asthenia.

Treatment.—A large aneurism can be treated only by palliative measures, relieving the suffering with morphin and retarding the ultimate issue by rest and the avoidance of exertion, excitement, and worry. When the aneurism has been discovered early, measures may be taken to promote the coagulation of blood and contraction of the sac. The treatment to be employed is absolute rest of body and mind and restriction of diet. Fluids should be sparingly ingested and all stimulants should be avoided. The Tufnell diet represents the extreme restriction that may be attempted, but it is generally found to be too severe. The patient is allowed, for breakfast and supper, bread and butter, 2 ounces; milk, 2 ounces; and for dinner, meat and bread, each 2 to 3 ounces; and milk or claret, 2 to 4 ounces. The best results are obtained in cases of saccular aneurism. If the heart's action continue rapid or if palpitation occur, aconite may be employed or an ice-bag may be applied over the tumor. Glonoin, gtt. j to ij t. i. d., is often serviceable; and when dyspnea and angina are present amyl nitrite may be very cautiously inhaled. Potassium iodid is the most valuable remedy, however, both for the relief of pain and the reduction of the tumor. It should be given in doses of gr. x to xx (0.60—1.20) t. i. d. The possibility of obtaining benefit from surgical methods should always be considered, although the results in thoracic aneurism have seldom been satisfactory. Electrolysis is safer than such measures as the introduction of wire or hair, and has been followed by excellent results in certain cases.

Aneurism of the Abdominal Aorta.—The dilatation usually affects the portion of the vessel immediately below the diaphragm, near the

celiac axis, and sometimes involving it also. It is more commonly fusiform than saccular.

Symptoms.—A dull, aching pain, reflected to the back and flanks, is generally the principal symptom, but it may be absent. When the tumor extends forward, it may produce prominence of the epigastrium or left hypogastrium; when backward, it erodes the bodies of the vertebræ. Numbness and tingling of the legs are then produced, and paraplegia may follow. Pressure symptoms may be noted, at least vomiting, or the pressure may be exerted upon the intestines, liver, spleen, or kidneys. The femoral pulse is retarded and reduced in volume.

Physical Signs.—Prominence and pulsation may be recognized in one of the regions referred to by both inspection and palpation. The pulsation is expansile, sometimes double, and a thrill may be felt. An area of dullness may be recognizable, and a soft murmur may be heard, but the diastolic shock and systolic murmur are absent.

Diagnosis.—The epigastric pulsation is sometimes so strong in neurasthenic or extremely anemic women as to suggest aneurism. But there is no expansile tumor, and the physical signs are absent. Pulsation may be transmitted through a solid tumor in the abdomen, as in the thorax, but again it lacks the force, the expansile quality, and the thrill. The tumor usually loses its pulsation when the patient is placed in the knee-elbow position, which allows the growth to gravitate away from the aorta.

The ***prognosis*** is unfavorable. Spontaneous recovery has been reported in a few instances, but a vast majority of the cases are fatal through (*a*) rupture, (*b*) complete obliteration of the lumen by clots, (*c*) paraplegia, or (*d*) embolism of the mesenteric artery.

The ***treatment*** is the same as that of thoracic aneurism. Compression of the vessel above the tumor has been practiced, but seldom with benefit and repeatedly with serious or fatal results.

Aneurism of the celiac axis may be associated with aneurism of the abdominal aorta, as stated. It is rarely a primary affection. The hepatic, mesenteric, splenic, and renal arteries, being less subject to sclerosis, are seldom the sites of aneurism, and when affected the condition is not usually diagnosticated during life. They frequently rupture before attaining appreciable size.

DISEASES OF THE MEDIASTINUM.

In this group are included inflammatory processes and tumors of the tissues and glands in the mediastinal spaces, including affections of the thymus gland.

Inflammation.—This may affect either the lymph-glands (lymphadenitis) or the fibrous tissue (mediastinitis); it may be simple, suppurative, or tubercular. Tubercular mediastinitis is generally accompanied with suppuration.

Etiology.—1. *Simple inflammation* arises chiefly from: (*a*) Penetrating wounds and injuries by foreign bodies in the esophagus, (*b*) by extension from inflammatory processes in the bronchi or lungs, especially the bronchitis or bronchopneumonia attending measles or other acute infection, and sometimes (*c*) by extension from the pleura or pericardium.

2. *Suppurative inflammation* arises: (*a*) By extension along the blood-

vessels from the tissues of the neck, including the larynx, trachea, and esophagus, or from the retropharyngeal abscess in children, (*b*) from suppuration in the lungs, thymus or bronchial glands; (*c*) from trauma, as fracture of the sternum, wounds or burns of the neck; (*d*) from perforation of an esophageal ulcer or diverticulum; and (*e*) probably by metastasis in pyemia, erysipelas, typhoid and typhus fevers, pneumonia, or smallpox.

3. *Tubercular mediastinitis* usually results through extension from a tubercular process in the vertebræ or lymph-glands.

Tumors.—The most important of these are cancer and sarcoma. Of the 520 cases of mediastinal disease collated by Hare, 134 were carcinomata, 98 sarcomata, 21 lymphomata, 7 fibromata, 11 dermoid cysts, and 8 hydatid cysts. The most frequent points of origin were the thymus and lymph glands, the pleura and the lung. Men were more frequently affected, and the age was generally between 30 and 40.

Morbid Anatomy.—Simple inflammation is often attended with the throwing out of plastic lymph about the vessels, nerves, and bronchi. This, becoming organized, forms firm adhesions and constricting bands (plastic mediastinitis). Subsequent contraction of the adventitious bands leads to constriction of the blood-vessels and bronchi in some cases. In the suppurative form a greater or less quantity of pus is found in either of the mediastinal spaces, and in many cases it has burrowed from its original source into adjacent structures. One or more of the ordinary pus-forming bacteria may be discovered in it. Ulceration and perforation often occur, more commonly into the esophagus or bronchial tubes, occasionally into the pleura, pericardium, peritoneal cavity, or externally.

Symptoms.—(1) The manifestations of a simple mediastinitis are often concealed by those of the affection to which they owe their origin.

Pressure symptoms are common to all forms of mediastinal disease, though less pronounced in simple inflammation than in abscess or tumor. Prominent among them are: (*a*) Cough, which is usually paroxysmal, resembling pertussis, but continuing indefinitely and probably due to pressure upon the trachea at its bifurcation; or in some cases to pressure on the recurrent laryngeal nerve, when hoarseness is also present. (*b*) Dyspnea, often so extreme as to suggest compression of the vagus, a possible condition, but more probably due to compression of the bronchi. Cyanosis of the face and neck and an audible wheezing respiration usually accompany the cough and dyspnea in severe cases. (*c*) Engorgement of the jugular veins, which is most marked during inspiration, and the pulsus paradoxus of Kussmaul, in which the radial pulse becomes almost imperceptible during inspiration. The apex impulse and sounds are also feeble. (*d*) There is generally a sense of weight, aching, or acute pain beneath the sternum, and the pain sometimes radiates to the back or sides of the chest and to the shoulders. (*e*) More or less complete dysphagia may be present, and a constriction may sometimes be recognized in the passage of the esophageal bougie. (*f*) Other occasional symptoms are epistaxis, vomiting, cardiac palpitation, clubbing of the fingers, weak voice, hoarseness or complete aphonia, ascites, edema of the legs. Systolic bruits are sometimes heard on auscultation over the aorta and pulmonary arteries. Pupillary changes,

particularly inequality, are sometimes occasioned by pressure upon the sympathetic.

(2) In *abscesses* of the mediastinum there is usually added to these symptoms tenderness to pressure over the sternum, a more or less constant elevation of temperature, and other manifestations of sepsis. Other symptoms are produced which are more or less common to tumors in this region. Many obscure symptoms sometimes arise from rupture of the abscess, but the pressure symptoms promptly subside and thus give a suggestion of the nature of the disease.

(3) *Tumors.*—During the growth of a tumor in the mediastinum many of the symptoms that have been enumerated are more or less prominently developed. Yet in some cases the tumor acquires a considerable growth without producing definite disturbances. A distinct pulsation is often transmitted through a solid tumor or tense abscess. Bulging of the sternum and erosion are sometimes observed, or the tumor may cause a prominence above or at the side of the sternum. A cachexia develops in all malignant cases. Percussion elicits dullness over the affected area, which is often most easily recognized posteriorly. Auscultation reveals for the most part an absence of heart and respiratory sounds over the area of dullness, but when the trachea or bronchi are compressed there is usually a prolongation of inspiration and expiration, with high pitched, tubular quality. When a bronchus has been completely closed, there are the usual signs of pulmonary collapse over the area supplied by it. Pleural effusion is often found in the dependent portion of the chest.

Diagnosis.—It is often difficult to determine the exact nature of the disease. Septic manifestations point to the existence of an abscess, and cachexia is no less significant of malignant disease. Sarcoma is more frequently primary than is carcinoma, and it is oftener situated in the anterior mediastinal space. But the differentiation of these growths is often based upon the age of the patient and the presence of a primary growth in some other region.

Aneurism is often excluded with difficulty, for there are no absolutely distinctive signs. As a rule, its growth is much slower than that of cancer or sarcoma, the pain is sharper and more uniformly present and it radiates more commonly to the back, neck, and down the arm. The pulsation is stronger and more expansile than that of an abscess or other tumor, and a distinct diastolic shock can generally be recognized, both on auscultation and palpation. A downward movement of the larynx with the pulsations of the tumor (tracheal tugging) is strongly indicative of aneurism. Finally, a distinct bruit is usually heard, which is more forcible and quite unlike that transmitted through a solid tumor.

The ***prognosis*** is grave in abscess, and invariably fatal in malignant disease.

Treatment.—Abscesses can sometimes be successfully opened and drained after resection of a part of the sternum. The possibility of syphilis should always be borne in mind in obscure cases, and a brief course of treatment employed. When this fails and when the case is beyond the reach of the surgeon, palliative treatment alone remains. Morphin should be freely given in order to render the patient as comfortable as possible.

SECTION V.

Diseases of the Respiratory System.

DISEASES OF THE NOSE.

ACUTE CORYZA.

ACUTE RHINITIS, ACUTE NASAL CATARRH.

Definition.—An acute inflammation of the mucous membrane of the nose.

Etiology.—The disease is believed to be of microbic origin. In many cases a sudden change of temperature, exposure to cold and wet or irritating fumes, is believed to aid infection. The disease attacks individuals of any age; it is most frequent in the autumn and spring. It often appears to be contagious and sometimes assumes epidemic prevalence.

Symptoms.—The attack is often initiated with chilly sensations, and a sense of fullness and pain in the head, repeated sneezing, often with rise of temperature, not usually exceeding 101° F. (38.5° C.), and aching of the limbs and back. The mucous membrane of the nose becomes dry and swollen, and the patient is compelled to breathe through the mouth. There is sometimes a severe aching pain in one or both nostrils. Within a few hours a watery secretion is established which irritates the edges of the nostrils and requires the constant application of the handkerchief. Eczema of the lip is often produced, and nasal or labial herpes may appear. The sense of smell is obtunded, and to some extent that of taste. The lachrymal ducts are involved, and there is a flow of tears. The inflammation, extending to the throat, produces soreness, tinnitus, and partial deafness. The pharynx and larynx are usually inflamed. Sometimes the nasal sinuses are affected and produce the most intense pain in the forehead or face, preventing sleep or mastication. By the second or third day the secretion becomes mucopurulent and more tenacious. Its quantity then diminishes, the swelling subsides, constitutional symptoms disappear, and nasal respiration is re-established. Recovery is complete in a week or ten days. Repeated attacks render the individual more susceptible and may produce a chronic catarrhal condition. Many subacute cases occur.

Diagnosis.—The condition is readily recognized. When, however, the initial symptoms are severe, and particularly if the temperature exceed 101° F., the possibility of its being a prodrome of an acute infection, particularly measles or influenza, should be borne in mind. Nasal catarrh (snuffles) in an infant is highly suggestive of syphilis. A watery discharge from only one nostril may indicate the presence of a foreign body. The ingestion of iodin produces a condition like coryza in a susceptible person.

The ***prognosis*** is favorable except in the extremes of life, when there is danger of extension of the catarrh to the bronchial tubes.

Treatment.—Infants and old persons should be kept indoors and confined to bed during a severe attack. Robust persons seldom need restraint except when fever is present. The patient should then be put to bed and given hot drinks, especially lemonade, and a full dose of Dover's powder at bedtime. Sweating should be encouraged by a heavy covering of blankets. A saline cathartic should be given immediately upon awaking in the morning. Nothing affords greater relief during the daytime than small doses of Dover's powder, gr. j to iij (0.06—0.18), with extract of belladonna, gr. ⅛ to ¼ (0.005—0.01), every two or three hours until the nasal secretion has reached the mucopurulent stage. When the sinuses are involved, hot applications should be employed. A Turkish bath often cuts short an attack.

CHRONIC NASAL CATARRH.

CHRONIC RHINITIS.

There are three forms of chronic nasal catarrh, designated, according to the character of their lesions, simple, hypertrophic, and atrophic.

Etiology.—The disease may result from repeated attacks of coryza, from long subjection to a smoky or dusty atmosphere. Bad hygiene, malnutrition, and deficient clothing favor it, and there is great difference of individual susceptibility. In many persons, however, it is largely due to asymmetry of the chambers from deviation of the septum, or to the presence of adenoids, polypi, or foreign bodies. The disease may be syphilitic or tuberculous in its origin.

1. **Simple Rhinitis.**—The mucous membrane is thick and hyperemic, secreting a large quantity of clear or yellow tenacious mucus which causes obstruction and interference with respiration.

2. **Hypertrophic rhinitis** presents greater swelling of the mucous membrane, particularly over the turbinated bones, and is often associated with adenoid growths in the nasopharynx. There is increased secretion of mucus, generally purulent and tenacious. Mouth-breathing is induced with consequent dryness of the throat, hawking and coughing, and a nasal tone of voice.

3. **Atrophic Rhinitis.**—The mucous membrane of the nasal cavity becomes thin, and the cavity correspondingly larger, but the mucus accumulates and forms thick, hard crusts that sometimes remain in position until ulceration occurs beneath them. There is then an offensive fetid discharge (coryza fetida). The term ozena is also applied to the condition, particularly when the necrosis extends to the bone. Ozena is generally due to tuberculosis or syphilis. The sense of smell is lost, taste is impaired, the hearing becomes defective, and constant noises are often complained of. The disease usually occurs in early life and is more frequent in women.

Treatment.—Hygienic measures are of value, the patient should be instructed with reference to proper ventilation, bathing, and exercise. A change of climate is often advisable in those having a tuberculous tendency. In the hypertrophic and atrophic forms, treatment by a

specialist is generally necessary. Cleanliness of the affected mucous membranes is essential. This can be accomplished by sniffing from the hand or allowing to flow into the nostrils a warm alkaline solution. Seiler's tablets may be used, or a solution may be made with a dram each of salt and bicarbonate of soda in four ounces of warm water. The engorgement of the mucous membrane may be greatly relieved in many instances by frequent spraying with a solution of menthol, ʒ ss (2.0); camphor, gr. xx (1.3); in liquid albolin ℥ ij (60.0).

HAY FEVER.

AUTUMNAL CATARRH, HAY ASTHMA, ROSE COLD.

Definition.—A catarrhal affection of the upper respiratory passages, with asthmatic breathing, generally attributed to irritation of the mucous membranes by vegetable dust or pollen.

Etiology.—The disease generally prevails in August and September, but it is occasionally contracted in the spring. Men are a little more frequently attacked than women. The disease is more common in the United States than in Europe, and more prevalent in cities than in the country. Only certain individuals are affected, and in these there is generally some abnormal condition, as deviation of the nasal septum, polypi, or hypertrophy of the turbinated bones, to account for their susceptibility. Most patients are also distinctly neurotic, and a hereditary tendency often appears. An attack has been induced in a susceptible person by suggestion, with an artificial rose.

Dunbar has recently discovered that the pollen of rye, oats, wheat, rice, corn, and all other forms of grass contains an albuminoid substance which is capable of producing all the symptoms of hay fever in a susceptible person at all times of the year, whether it be applied locally to the nasal mucous membrane or introduced subcutaneously.

Symptoms.—The same individual is generally attacked at the same time, often on the same day, of each year. The onset is announced by persistent sneezing, or the patient may be seized during the night with an asthmatic attack. The condition quickly becomes one of severe coryza, to which are added paroxysms of coughing and more or less frequent asthmatic seizures. The patient is rendered unfit for business, is generally greatly depressed and often melancholy.

Diagnosis.—The diagnosis is generally evident. Asthma of other origin is not attended with coryza or so great mental depression.

Prognosis.—The disease seldom results seriously, but relief from the attack and removal of the tendency are alike difficult.

Treatment.—The attack is relieved in most cases by a visit of six weeks to the mountains. The seashore is better in some cases, and a sea-voyage gives relief to all. When such means are unavailable, medicinal treatment must be applied. Irrigation of the nose with a solution of quinin, gr. j (0.06), to water, ℥ ij (60.0), has been recommended. Spraying the nostrils with a solution of adrenalin hydrochlorid (1 :5000 or less) has been recommended. More is generally to be accomplished by treatment of the patient in the intervals than during the attacks. The nasal chambers should be carefully examined by a specialist, and

abnormal conditions remedied. The neurotic condition calls for the administration of tonics, particularly strychnin, and iron or arsenic. Dunbar believes that he has succeeded in producing a curative serum.

EPISTAXIS.

NOSE-BLEED.

Etiology.—The causes are local and constitutional. (1) Among the former are injury, blows, rubbing, picking, coughing, sneezing, the lodgment of a foreign body, or the presence of neoplasms. The presence of chronic nasal catarrh favors its occurrence.

(2) The principal constitutional causes are: (*a*) Arterial engorgement, so-called plethora, or the hyperemia which sometimes attends the invasion of an acute infection. (*b*) Venous engorgement, particularly when it is due to an advanced valvular heart disease. (*c*) Abnormal states of the blood, as hemophilia, purpura, scurvy, pernicious anemia, or leukemia. (*d*) Sudden reduction of atmospheric pressure, as in ascending to great altitudes. (*e*) Vicarious menstruation and cessation of chronic hemorrhoidal bleeding are possible causes; and (*f*) mental emotion may induce it. The source of the blood is generally a capillary oozing from the septum, floor, or outer wall.

Symptoms.—There is sometimes a prodromal sensation of fullness or throbbing, but the bleeding often starts without warning. Except in abnormal blood-states, it is generally confined to one side. If the bleeding occur at night, the blood may be swallowed during sleep and vomited later. The quantity of blood lost is generally less than an ounce, but in severe hemorrhages it may be so great as to produce syncope. As this condition comes on, the blood ceases to flow. Death rarely results from epistaxis, but the patient may be left in an anemic and debilitated condition, especially if he be the subject of nephritis or heart disease.

Treatment.—Moderate epistaxis is often beneficial, even in the passive congestion of heart disease. When it is necessary to interfere, the bleeding spot should be found, if possible, and subjected to pressure or cauterization. If this cannot be done, ice may be applied to the nose and ice-water sniffed or douched into it. Hot water is equally effective. Astringent solutions may be employed, as alum or zinc (2 to 4 per cent). Solutions of iron or tannin are sometimes effective, but they are exceedingly uncleanly. Pledgets of cotton may be dipped into the solutions or impregnated with the astringent powder and introduced. When these measures are ineffectual, the posterior nares should be plugged, and, this failing, the entire nasal chamber should be firmly packed with pledgets of cotton or gauze threaded on a string. The tampon must not remain longer than 48 hours. Treatment of the constitutional condition should not be overlooked.

NASAL NEUROSES.

This term is applied chiefly to alterations of the sense of smell. These are: (*a*) *Anosmia*, or a more or less complete loss of smell; (*b*) *hyper-*

osmia, or an abnormally acute sense of smell; and (*c*) *parosmia*, in which the sense is altered or perverted. The affections are generally attributed to alterations in the nerve-endings in the Schneiderian membrane that may result from any of the forms of inflammation. Besides these, injury and disease of the fifth nerve may be followed by a loss of the reflexes, so that sneezing is no longer induced by irritation.

DISEASES OF THE LARYNX

ACUTE LARYNGITIS.

Etiology.—The disease may be primary or secondary; primary as a result of atmospheric conditions, or "cold," inhalation of irritating dust or fumes, injury by foreign bodies, or excessive speaking; secondary in connection with the acute infections, catarrh of the nose or throat, or when associated with pulmonary disease.

Symptoms.—There is generally a sense of tickling or pain in the larynx aggravated by the inhalation of cold air and sometimes by swallowing; a cough which may be "croupy," and huskiness of the voice. Dyspnea is often produced in children and may result from associated edema in adults. On examination the mucous membrane is found to be hyperemic, the condition involving the vocal cords, but most pronounced in the aryepiglottic folds. There are usually no constitutional disturbances in an uncomplicated case, except occasionally slight fever in children.

Diagnosis.—The differentiation is generally to be made from spasmodic, diphtheritic, and edematous laryngitis. In spasmodic laryngitis the paroxysm comes on suddenly, and it subsides completely without hoarseness or pain. It is purely a nervous condition. Diphtheritic or membranous laryngitis is generally accompanied with a similar disease of the tonsils or pharynx, and there is a history of possible contagion in most cases. The cervical glands are generally enlarged and the illness is more severe. In edema of the glottis there is greater dyspnea, and examination reveals the condition.

Treatment.—The patient, particularly if a child, and when fever is present, should be confined to bed in a warm room. The air should be kept moist by the evaporation of water, and steam may be inhaled. Hot or cold applications over the larynx are beneficial. The larynx must be given rest, speaking being forbidden. Dover's powder at night and ammonium chlorid and ipecacuanha or squill during the day are the principal remedies. Aconite may be given for the fever.

CHRONIC LARYNGITIS.

Etiology.—Repeated attacks of acute laryngitis, persistent overuse of the voice in the open air, and the inhalation of irritating dust or tobacco smoke are the most frequent causes. Diseases of the nose and pharynx often lead to laryngitis.

Symptoms and Diagnosis.—There is pronounced hoarseness, sometimes amounting to aphonia, sometimes pain and soreness, and more or

less constant tickling and cough. The local signs of inflammation may be slight, yet enough, as a rule, to distinguish it from tuberculosis of the larynx, in which the appearance is one of anemia. Ulceration does not occur, except in late tuberculosis or in syphilis.

Treatment.—The larynx must be given rest; tobacco and alcohol must be abstained from, and other causative agencies removed. Inhalations of steam, sprays of menthol and camphor, afford relief. Silver nitrate and other remedies may be employed, but these and the treatment of the nose and pharynx, which is sometimes necessary, would better be intrusted to a specialist.

EDEMATOUS LARYNGITIS.

EDEMA OF THE GLOTTIS.

Etiology.—(*a*) Any disease attended with general dropsy, especially nephritis and cardiac disease; (*b*) acute infections, especially diphtheria, scarlet fever, smallpox; (*c*) chronic disease of the larynx; (*d*) injury by vapors, hot fluids, or poisons; (*e*) repeated attacks of acute laryngitis, are the most common causes. (*f*) An angioneurotic origin has been referred to.

Symptoms.—A sudden, urgent dyspnea develops, increasing in severity until the face becomes livid, the voice is lost, the heart's action becomes tumultuous, and, if continuing for 24 to 36 hours, sometimes earlier, death may appear imminent. A fatal termination sometimes occurs. Examination reveals the swelling, particularly in the aryepiglottic folds; sometimes it is deeper in the larynx (subglottic). The prognosis is always serious.

Treatment.—If the symptoms be not urgent, cold may be applied externally and pieces of ice may be held in the mouth, but in most cases the edematous swelling should be scarified without delay, the larynx being first sprayed with cocain (4 per cent). If this fail, tracheotomy must be performed.

NEUROSES OF THE LARYNX.

SPASM OF THE LARYNX.

LARYNGISMUS STRIDULUS, SPASMODIC LARYNGITIS.

Two forms of spasm of the larynx are recognized, one a spasm of the adductor muscles without inflammation, usually occurring in early infancy and doubtless a pure neurosis (laryngismus stridulus); the other occurring in later childhood as a result of catarrhal inflammation (spasmodic croup).

1. **Laryngismus Stridulus.**—This form is more frequent in male infants between the ages of six months and two years. The attacks are usually attributed to reflex irritation, particularly from the gastrointestinal tract. They are especially common in rachitic infants and those affected with cerebral or spinal disease. Spasm of the larynx may occur in adults also as a manifestation of hysteria, or as a result of the inhalation of irritating fumes, the lodgment of a foreign body, tubercular or syphilitic ulceration.

Symptoms.—The attack commonly occurs at night. The child awakes struggling for breath. Respiration has ceased. The face is livid, and the struggle may assume the appearance of a convulsion, or a convulsion may actually occur; but in a moment the spasm relaxes, with a long crowing inspiration. The attack may recur several times during the night, and even during the daytime, or at intervals of several days for a week or longer. There is no cough, hoarseness, or other evidence of catarrh. Death has occurred in feeble infants or as a result of cerebral hemorrhage induced by the attack.

Treatment.—The attack is usually too short to require treatment. In the interval, however, search should be made for the cause of the irritation; errors of diet should be corrected, the bowels regulated, and the rachitic or other underlying condition treated. Fresh air, sunshine, exercise, and cold sponging all assist in overcoming the abnormal excitability of the nervous system.

Spasmodic Croup.—This is a more frequent affection and occurs in weakly or robust children between 2 and 6 years of age. The child awakes suddenly during the night, generally after midnight, gasping for breath; or a loud, hoarse cough may be the first indication of the condition. The voice is hoarse and the respirations are sonorous. Cyanosis may be produced, but, as a rule, the paroxysm subsides within a half-hour and the child falls asleep, to awake in the morning entirely free from it, or perhaps still a little hoarse. The attack may be repeated on several succeeding nights. The prognosis is good.

Treatment.—The paroxysm may be relieved by a few inhalations of chloroform, by an emetic, by the inhalation of steam discharged from a convenient vessel under an improvised tent, or by a hot mustard-bath. When the hoarseness continues during the day, a cough sirup containing ammonium chlorid and sirup of ipecacuanha should be prescribed.

Other Neuroses.—Chief among these are hyperesthesia, anesthesia, paresthesia, and hysterical aphonia. True paralysis of the vocal cords is encountered chiefly as a result of diphtheria, the growth of tumors in the larynx or in a situation where they press upon the recurrent laryngeal nerve, as is the case with aneurisms of the arch of the aorta.

Tubercular laryngitis is considered under the head of Tuberculosis (p. 190). Syphilitic laryngitis is referred to under the head of Syphilis (p. 165).

DISEASES OF THE BRONCHI.

ACUTE BRONCHITIS.

Definition.—An acute inflammation affecting the mucous membrane of the bronchial tubes of large and medium size. A similar affection of the smaller tubes, known as capillary bronchitis, is considered under the head of Bronchopneumonia.

Etiology.—The disease is probably of microbic origin. Atmospheric conditions, especially cold and excessive moisture, sudden changes of temperature, and the presence of dust or irritant vapors doubtless exert a predisposing influence. The disease is often caused by direct extension of inflammation or infection from the nose or pharynx. It may be

secondary also to other diseases, particularly to measles, malaria, typhoid fever, and other infections. The predisposing causes are many, especially: (*a*) Age. The disease is more common in early and late life; heredity is often an important factor. (*b*) Habits; indoor, sedentary life without exercise. (*c*) Poverty and privation. (*d*) Occupations which necessitate the breathing of dusty air. (*e*) General health, but particularly the presence of pulmonary disease, as tuberculosis; or a gouty diathesis. (*f*) Climate and season. The disease is much more common in changeable climates and in the winter season.

Morbid Anatomy.—The essential lesions are hyperemia, swelling, and increased secretion, and these are found in the mucous membrane of the trachea as well as in that of the bronchial tubes. The disease is generally a tracheobronchitis. Desquamation of epithelium occurs, and the submucosa becomes to some extent hyperemic and edematous. The bronchial lymph-glands are generally enlarged and hyperemic.

Symptoms.—The onset may be sudden or gradual; it is generally preceded by coryza, except in individuals already affected with pulmonary disease. There is generally a slight chilliness, rarely a rigor, with languor and aching of the limbs and back. Fever follows in the more severe cases, especially in children, but it seldom exceeds 103° F. (39.5° C.), and the pulse is rapid. A dry, harsh, paroxysmal cough develops, and during the paroxysms the patient often experiences a sharp pain behind the sternum and through the chest. Headache may also be present. The cough soon gives rise to a scant, viscid expectoration; this in a few days becomes mucopurulent, then purulent and more abundant. The cough now becomes less painful and the fever subsides. In infants the bronchial secretion is not expectorated and there may be little cough. The disease must be recognized in them through the dyspnea, rapid respiration, and fever, with the physical signs that are always present. In the aged, too, the disease often begins insidiously, with prostration, rapid respiration, and even delirium, with but little cough. At either extreme of life there is great danger of extension to the finer bronchi.

Diagnosis.—The disease is readily recognized upon physical examination, if not by the general symptoms present. In robust individuals of middle age, little is to be observed on inspection. In the infant and old person, however, the respiratory movements become rapid, and, in the former, there may be slight sinking of the intercostal spaces during inspiration. The upper part of the thorax sometimes appears expanded and the lower part depressed. In adults the dyspnea and acceleration of breathing correspond to the degree of fever. The bronchial fremitus can sometimes be felt. Percussion seldom furnishes exact information in a case of simple bronchitis.

On auscultation, numerous râles are heard. In the beginning these are of a dry character, sibilant, or sonorous, but later there are moist, mucous râles. They are heard intermittently, coughing causing them to disappear for a time, as a rule, but they are distinctly audible in all parts of the chest, though with greater distinctness in some regions than in others. Sometimes the disease is confined almost exclusively to one side, especially when it is a complication of tuberculosis. The vesicular murmur is disturbed in rhythm and pitch. Both inspiration and expiration are prolonged, expiration more than inspiration, and

the pause is shortened. The pitch is raised. The sputum is not altogether distinctive in character. After the disease has fully developed, it consists largely of pus, in which alveolar cells are found in greater or less numbers and in different degrees of degeneration.

The features which especially distinguish simple bronchitis from other affections are, the character of the râles and their general distribution. Bronchopneumonia and acute tuberculosis are chiefly to be excluded. In the former, fine moist râles are heard along the margins and at the base of the lungs. The prostration and dyspnea are greater. In acute general tuberculosis, the high temperature, great prostration, night-sweats, and other symptoms rarely fail to distinguish it from acute bronchitis.

Prognosis.—Acute bronchitis, of itself, is seldom a serious disease in middle life, but in the very aged and the infant it should always be so regarded. It may terminate fatally in these patients through asphyxia or exhaustion, and the danger of its extending to the finer tubes and producing a bronchopneumonia is always a great one.

Treatment.—In mild cases in persons of middle age the disease may generally be relieved or greatly modified by the administration of hot lemonade, a hot foot-bath, and a full dose of Dover's powder at the time of retiring for the night. In severe cases, the patient should be kept in bed. A saline cathartic should be given in the morning. A mustard-plaster applied to the chest relieves the pain behind the sternum. The air of the patient's room should be warm and moist. A Turkish bath sometimes proves of the greatest benefit, providing the patient can remain over night in the bath-house; otherwise it is unsafe, on account of the exposure that must follow it. A full dose of quinin (gr. x—xx; 0.60—1.2) at night benefits some cases. A mixture containing ammonium chlorid or potassium acetate (gr. j—ij; 0.06—0.12) and sirup of ipecacuanha (ℳij—v; 0.15—0.3) in each dose, with confinement to bed, is generally all that is required for a child in an uncomplicated case. The compound sirup of squill is often employed. Senega and wild-cherry assist in checking the secretion in the later stage of the disease. An emetic is often effective in clearing out the bronchial tubes when obstructed to such a degree as to cause alarming cyanosis. Inhalations of steam impregnated with the vapor of benzoin, eucalyptus, or turpentine is often beneficial. The vapor may be inhaled through a paper funnel inverted over a pitcher containing a quart of boiling water to which a dram of the medicament has been added. In the extremes of life, attention must be given to the general nutrition of the patient, and stimulants should generally be given in quantity suitable to the age and physical condition.

CHRONIC BRONCHITIS.

Etiology.—Chronic bronchitis may result from repeated attacks of the acute form, but, as a rule, it runs a subacute course from the beginning and is directly attributable to disease of the lungs or other organs. Among diseases of the lungs, the most commonly bearing a causative relation are tuberculosis, emphysema, asthma, chronic interstitial pneumonia, and chronic pleurisy with adhesions. Among diseases of other

organs, valvular heart disease, and nephritis are the most important. The disease is much more frequent in persons past middle life, but it is often encountered in the young. It is often spoken of as the "winter cough" of old people, beginning, as it does, with the first onset of cold weather and continuing until summer returns. It is much more prevalent in cold and changeable climates, and especially near the seacoast.

Morbid Anatomy.—The changes are not constant. In some cases the mucosa and muscular layers are found in a state of atrophy, in some they are thickened and infiltrated. The surface of the mucosa may be granular, smooth, and in places destitute of epithelium, or ulcerated. Bronchiectatic dilatations are common in cases of long standing. Emphysema is always present.

Symptoms.—Cough is a constant symptom. It is generally most troublesome at night, and a prolonged coughing spell is usually induced by the accumulation of mucus after a few hours' sleep. Dyspnea is generally a prominent feature, occurring especially upon exertion, as in climbing a hill or ascending a flight of stairs. It is due either to deficient aëration of the blood or to cardiac weakness. A sense of oppression or of soreness in the chest is usually complained of, which appears to be due either to the strain of coughing or to the exaggerated action of the respiratory muscles. Acute pain is unusual. All the symptoms are subject to frequent changes. Inclement weather, change of temperature, and exposure produce exacerbations. The disease is always worse in winter. For a number of years it may almost wholly subside during the summer, but each year the period of relief becomes shorter until the cough and expectoration become constant. Evening elevation of temperature is frequently observed, especially during the more severe periods of the disease; but in many cases it is so slight as to be overlooked. The sputum is variable, changing from time to time. Sometimes it consists of thick, tenacious mucus, sometimes of almost pure pus; in some cases it is always thin and fluid or frothy. Cases of "dry catarrh" occur also, in which there is little or no expectoration. These and other differences in the character of the sputum have led to the recognition of four forms of chronic bronchitis: (*a*) The common form, which has just been described, (*b*) bronchorrhea, (*c*) putrid bronchitis, and (*d*) dry bronchitis.

Bronchorrhea.—This name is applied to cases in which the bronchial secretion is excessive in quantity. The sputum is generally purulent, rather thin and greenish; sometimes it is almost serous in character, but, on the other hand, it may be tenacious. The entire bronchial mucous membrane is usually affected. Although the condition is not one of bronchiectasis, it is apt to lead to dilatation of the bronchi, and the accumulation of secretion may cause the development of a putrid bronchitis.

Fetid or Putrid Bronchitis.—This is characterized by an abundant expectoration of fetid, mucopurulent, heavy greenish, or thin grayish sputum mixed with frothy mucus, which separates on standing into three layers, the upper consisting of the frothy mucus, the middle of clear serum, and the lower of thick purulent matter often containing the so-called Dittrich's plugs, firm yellow masses as big as peas, composed of granular matter, fat-globules, and fatty acids, with putrid animal

matter and sometimes fungi. Fever is more constant in this form than in simple bronchitis. This form, too, is often associated with bronchiectasis, gangrene or abscess of the lung, or advanced tuberculosis.

Dry catarrh, as already stated, is characterized by a more or less complete absence of expectoration. It is generally associated with emphysema in old persons.

Physical Signs.—The physical signs are nearly the same as those of acute bronchitis. The resonance on percussion is slightly tympanitic. Sonorous and sibilant râles are heard, and mucous râles of every variety are always present, and generally in all regions of the lungs. An occasional extension of the catarrh to the smaller tubes is common, during which times subcrepitant râles can be heard at the base and margins of the lungs.

Treatment.—*Prophylaxis* is of the greatest value. The patient should, if possible, make such changes of occupation or residence as will enable him to avoid the inciting causes of the disease. The climate in this country best suited to the condition is found in southern California, at San Diego, or, better, in the villages in the foot-hills of the mountains near that city or Los Angeles. The southern part of Florida is suitable for a winter's sojourn. Next in importance is the constitutional condition of the patient. If there be an arthritic diathesis, heart or kidney disease, these should receive attention. If the patient be tuberculous, the treatment of that condition overshadows that of the bronchial affection. The digestion must be regulated with especial reference to the prevention of flatulency, a most distressing condition to the patient. The clothing should be warm, but modified to suit changes of temperature. All exposure must be avoided, particularly the respiration of cold air. While the patient should take moderate exercise, he should avoid overexertion and hard work.

The *medicinal treatment* must be suited to the case. Potassium iodid or the sirup of the iodid of iron is more generally beneficial than any other remedy, particularly when the secretion is scant. When the secretion is free, the fluid extract of senega may be added to the solution. Atropin is sometimes of service in bronchorrhea. Among other remedies generally used are ammonium chlorid, sodium benzoate, and other alkalis, ipecacuanha, tolu, tar, creosot, sandalwood, resin of copaiba, compound tincture of benzoin, and terebene. Inhalations of the vapor of turpentine, benzoin, creosot, eucalyptus, or a spray containing one of these or the wine of ipecacuanha are all recommended. In fetid bronchitis, a spray containing carbolic acid (*2* per cent) should be used to destroy the odor.

FIBRINOUS BRONCHITIS.

CROUPOUS, EXUDATIVE, PLASTIC, OR PSEUDOMEMBRANOUS BRONCHITIS.

Definition.—An acute or chronic inflammatory affection of the bronchial mucous membrane characterized by a deposit of plastic matter which becomes detached and is expectorated in the form of a more or less extensive cast of the bronchial tree. A distinction must be made between true fibrinous bronchitis, a comparatively rare disease, and those conditions in which the expectoration of similar molds results from an

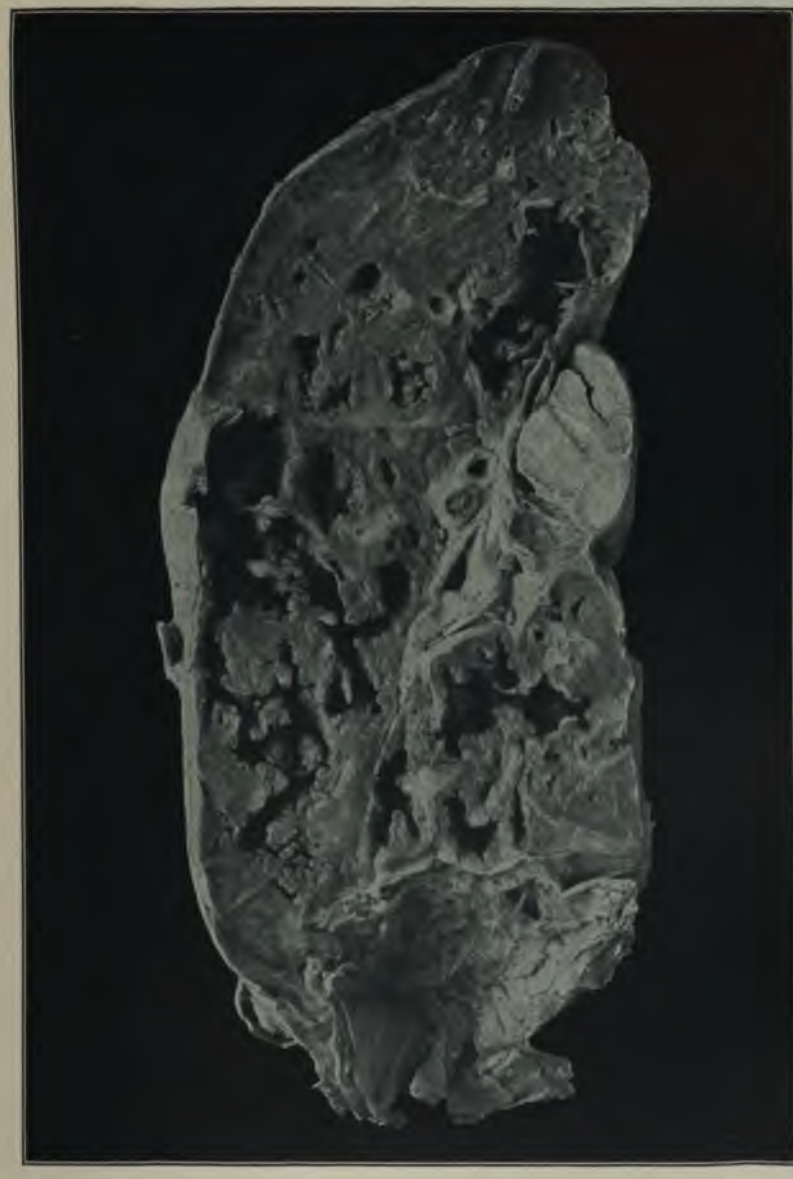
accumulation of clotted blood in hemoptysis, an extension of the diphtheritic membrane, or of the fibrinous exudate in acute pneumonia.

Etiology.—No specific cause is known, but it is probably not the same in all cases. The disease is more common in Europe than in this country and usually occurs during the late springtime. It is not limited to any period of life, but is rare under the tenth or after the fortieth year. It is twice as frequent in men as in women. The patient is generally in an anemic, debilitated condition when attacked, as a result of such diseases as measles, scarlet fever, pneumonia, or typhoid fever, and many are tuberculous or syphilitic. Its occurrence in pregnancy, and its association with such cutaneous affections as herpes and pemphigus, have been repeatedly noted. Various bacteria have been found in the secretions, but none has been identified with the disease.

Morbid Anatomy.—The bronchial mucous membrane has been found hyperemic, the epithelium sometimes intact, sometimes desquamated. The inflammation is more general in the acute form than in the chronic. The casts (Fig. 23), pure white or cream color, sometimes streaked with blood, are firm and elastic and correspond in size to the lumen of the part of the bronchial tree in which they originate. They are probably composed of mucin, although they have been generally regarded as fibrinous.

FIG. 23.—Casts from a case of fibrinous bronchitis. (2-5 natural size.)

Symptoms.—The acute form may have a sudden, severe onset, with high fever, chill, dry cough, dyspnea, and constriction of the chest, but in most cases it begins as a simple acute bronchitis, with cough, scant expectoration of clear mucus, and possibly a slight elevation of temperature. In children it is often preceded by malaise. Much difference has been noted in the severity and abruptness of the initial symptoms in different cases. A chill may mark the transition from the simple to the fibrinous form. The pulse-rate is accelerated, and, with the development of casts, the cough becomes more harassing and paroxysmal and the dyspnea more pronounced. Slight hemorrhage sometimes accompanies or follows their expulsion. Relief follows the removal of the obstruction, but it is transitory, and the paroxysm may recur within a few hours. In severe cases, digestion becomes impaired, nutrition is interfered with, and great nervous irritability may be exhibited. Recovery takes place by a gradual subsidence of the symptoms; the casts no longer appear, the temperature, although high, rapidly declines, appetite and strength return. Fatal cases generally terminate in from three days to two weeks, sometimes suddenly by suffocation.



Bronchiectasis with Chronic Tuberculosis.

The ragged communicating cavities involve a large part of the lung and are bronchiectatic in origin. The bronchial lymph-nodes are enlarged, tuberculous, and caseous. The pleura and interlobar septum are thickened by the formation of dense fibrous tissue.

(*By permission, from "Delafield and Prudden."*)

The chronic form generally follows a more or less protracted bronchial catarrh. Its course is one of exacerbations and remissions. Paroxysms of cough, dyspnea, and constriction occur, to be followed by temporary relief when the casts are expelled, but they recur at longer or shorter intervals for weeks, months, or years. Every grade of severity is seen in the recurrences, and months or years of perfect health may intervene. The temperature rises, if at all, during the exacerbations and does not generally reach so high a degree as it does in the acute form.

The physical signs are variable and depend upon the presence or absence of casts at the time of examination. In acute cases there may be all the evidences of an acute bronchitis; in the intervals of quiescence there may be no adventitious signs. The casts are readily recognized when the sputum is deposited in water, where they unfold.

Treatment.—It is difficult to estimate the results of treatment, since so few cases have come under the care of any one observer. Potassium iodid has been employed more than any other remedy, but its effects have not been uniformly satisfactory. Ammonium chlorid, ipecacuanha, senega, benzoic acid, apomorphin, and other expectorants have been recommended. Emetics may be employed in robust individuals to assist in the expulsion of the casts after they have become detached. Creosot carbonate in 15-drop doses is worthy of a trial.

Obliterative Bronchitis.—Under the term bronchitis obliterans Lange and A. Fränkel have reported three cases in which a plastic exudate in the bronchi, instead of becoming detached, underwent organization and caused a fatal obliteration of the air-spaces in a considerable portion of both lungs. The disease followed the inhalation of highly irritating fumes.

BRONCHIECTASIS.

Definition.—A general or localized dilatation of the bronchial tubes.

Etiology.—The disease is more common in middle life, but it has been found as a congenital condition; men are more frequently affected than women. The direct cause is believed to be a weakness of the walls induced by inflammation involving the muscle, fibrous, and cartilaginous structures, aided by the weight of accumulated secretions and probably by the expansive force of coughing. The disease is generally secondary to: (*a*) Chronic bronchitis and emphysema, (*b*) interstitial pneumonia, bronchopneumonia, or tuberculosis, (*c*) compression of the bronchi by solid tumors or aneurism, (*d*) impaction of a foreign body, or (*e*) contraction of the lung caused by thickening of the pleura. An acute form of the disease has been observed in children; in adults it is generally chronic.

Morbid Anatomy.—The dilatation may be single or multiple and the cavities may be fusiform or saccular; they vary in diameter from a half-inch to three or four inches. (See Plate X.) They may be found in any part of the lungs. The wall of the cavity is usually thin; sometimes it is lined by a thick, indurated membrane, often ulcerated; sometimes the epithelium remains more or less intact. Perforation sometimes occurs. The content of the cavity is a thick, greenish or brown, fetid, mucopurulent matter.

Symptoms.—The symptoms are the same as those of chronic fetid bronchitis, and the disease cannot always be distinguished during life. In the more advanced cases, however, the condition can generally

be recognized through the character of the cough and expectoration. The cough is paroxysmal, usually worse in the morning or after rising from a recumbent posture. An enormous quantity of sputum is brought up, sometimes amounting to more than a pint in 24 hours. After the paroxysm the patient may pass the greater part of the day without cough or expectoration. The sputum is generally rather thin, gray or brownish, and purulent, with a sour or fetid odor. It separates, on standing, into three layers similar to those of the sputum in fetid bronchitis. Microscopic examination reveals pus-cells, crystals of fatty acids, often arranged in bundles, sometimes hematoidin crystals, various bacteria, and, when there is extensive ulceration, elastic fibers. Dyspnea is not always a prominent symptom. Hemorrhage, generally slight, occurs in nearly half the cases. In cases of long standing the patient often becomes cyanotic after exertion, his finger-ends become clubbed and the nails incurved.

The ***physical signs*** depend largely upon the extent of the bronchial dilatation. Retraction of the chest-wall has been noted, but it is due to the contraction of the lung caused by chronic pleurisy or interstitial pneumonia in most cases. The percussion note may be tympanitic over superficial cavities, and amphoric breathing may be heard, especially at the base of the lungs.

Diagnosis.—The disease cannot always be differentiated from chronic bronchitis until the cavity-formation has reached a stage that yields characteristic physical signs. From tuberculosis it is distinguished by its prolonged course and the absence of the tubercle bacillus. A sacculated empyema having a fistulous communication with a bronchus is distinguished with difficulty, but the evacuation of the cavity is not usually so frequent. Cancerous ulceration of the lung and gangrene need seldom be considered, owing to the rapidity of their progress. Actinomycosis of the lung has been mentioned in this connection, but it is rare and can be recognized by the discovery of the fungus.

Prognosis.—The disease usually persists for many years, ultimately tending to a fatal termination, but cases of supposed recovery have been reported.

Treatment.—Little is to be anticipated from internal medication. Creosot in increasing doses, quinin, salol, and turpentine have been employed. Terebene, ℥v to x (0.33—0.66) every four hours, has proved beneficial in some cases. Better results are obtained, however, from the inhalation of sprays of antiseptic substances—eucalyptus, thymol, benzoin, creosot, or carbolic acid. The most satisfactory method of treatment, in many cases, is the intratracheal injection of a solution of menthol 10 parts, guaiacol 2 parts, in olive oil 88 parts. Of this a dram (3.6) is injected twice a day with a special syringe, which renders the use of a laryngoscope unnecessary. Inhalations of vaporized creosot have also been found of great benefit.

BRONCHIAL ASTHMA.

SPASMODIC ASTHMA.

Definition.—A form of dyspnea due to temporary alteration of the condition of the smaller bronchial tubes. The term asthma is often

applied also to the dyspnea, more or less spasmodic in character, which arises from cardiac or renal disease (cardiac or renal asthma).

Etiology.—There are four leading theories of the nature and cause of asthma, namely, that it is due: (1) To a spasm of the bronchial muscles, and probably a neurosis; (2) to a swelling of the bronchial mucous membrane caused by hyperemia, vasomotor nervous influence, or the presence of a toxic irritant in the blood; (3) to an inflammation of the bronchioles, a bronchiolitis exudativa (Curschmann); and (4) that it is due to a reflex spasm of the diaphragm, probably involving also the other respiratory muscles.

The disease is met with in persons of any age, from early infancy to extreme old age, but it commonly begins in early life. It is more frequent in men. There is great difference in individual susceptibility. Many patients are to be regarded as neurotic; there is at least a nervous temperament, an instability of the nervous system. The disease has been known to alternate with epilepsy, and is often accompanied by neuralgia and other nervous affections. The hereditary transmission is often distinct. It is closely allied to hay fever. It is to a great extent a disease of the better classes. It often follows whooping-cough or other acute infectious disease, and in many cases it is associated with disease of the upper respiratory passages, particularly with hypertrophic rhinitis or polyp of the nose. The exciting causes are of the greatest variety and they are generally peculiar to the individual. Asthmatics living in the city are free from it in the country; those residing on the hill are relieved by descending into the valley, and vice versa. The attack is often induced in one person by an odor which does not affect another, as by that of a certain flower or of a certain animal, of feathers or of ipecacuanha. Dust, smoke, and irritating vapors often induce an attack. Nervous impression or emotion, particularly fright, may cause it, and sometimes it appears to depend upon a disturbance in some other organ, especially the stomach, intestines, or genitals. A contraction of the bronchial tubes has been induced experimentally by electric stimulation of the vagus.

Morbid Anatomy.—The disease has no anatomical lesions peculiar to itself, but it is usually associated with those of chronic bronchitis and emphysema.

Symptoms.—The attack may come on suddenly in the midst of the night, or there may be such premonitory symptoms as chilliness, a feeling of oppression in the chest, depression of spirits, indigestion, or some nervous manifestations. The patient is unable to lie down; he often sits at an open window or seeks the open air. Some patients are always able to predict an attack; others can rarely do so. The onset may be gradual or sudden, with a constantly increasing sense of oppression, deepening into the most intense dyspnea. The respiratory efforts become violent. All the accessory muscles are called into action, but only a comparatively small volume of air enters the lungs, and the expiration is equally difficult and much prolonged. It is usually accompanied with wheezing. The face becomes pale, sometimes livid, the expression is anxious. Speech is impossible. If the paroxysm continue, the deficient oxygenation of the blood becomes more apparent; the face becomes cyanotic and moist, the extremities become cold, and, to the

inexperienced, death seems imminent; but, with the deepening of the cyanosis and the approach of unconsciousness, the spasm relaxes and the breathing becomes less labored. The paroxysm often terminates with a fit of coughing and the expectoration of a considerable quantity of mucus. The attack lasts from a few minutes to several hours, sometimes even days, with, perhaps, short intermissions. The relief is not usually complete; the breathing is still laborious, and very often the paroxysm is repeated within a few hours. During the attack, clear, pale urine of low specific gravity is often voided at short intervals and in large quantity. The sputum is characteristic. At first brought up with difficulty, it becomes more abundant, and in the masses of clear mucus can be seen small, round, gelatinous bodies, the perles of Laennec, which, when floated in water and examined with a lens, are found to be formed of spirally arranged mucin casts of the smaller bronchial tubes. They are known also as the Curschmann spirals. Examined with a higher power, some of these are seen to contain, in the center, a thread of clear mucin, while others are merely twisted casts. Numerous leucocytes, mostly eosinophiles, are usually entangled in them, and Charcot-Leyden crystals, octahedra of ethylenimin phosphate, are generally present. In the course of a few days the sputum becomes mucopurulent, and the spirals can no longer be found in it. The eosinophiles are greatly increased in the blood.

The physical signs of asthma are diagnostic. The thorax is fixed, and the respiratory excursions are exceedingly limited. The inspiration is quick and jerking, while the expiration is prolonged to more than double its normal duration. On percussion, the resonance is increased (*i. e.*, tympanitic), especially when emphysema is present. Auscultation reveals dry, sibilant and sonorous, whistling râles on expiration. The vesicular murmur is entirely suppressed, or it is replaced by bronchial breathing.

Prognosis.—Asthma is not fatal and may last for a lifetime. The paroxysms never terminate fatally. Bronchopneumonia may, however, supervene.

Treatment.—The remedies for the paroxysm are many and, for the most part, effective in certain cases, but not in all. The simplest of them consist in the inhalation of the fumes of burning niter-paper, stramonium leaves, tobacco, or cigarettes containing one or more of these substances; or inhalation of chloroform, ether, or amyl-nitrite. The internal administration of chloral or caffein, and the hypodermic administration of morphin, are promptly effective, but such remedies, with the exception of caffein, should be used cautiously on account of the danger of producing a habit. A cup of hot coffee or a hot toddy will arrest the attack in some individuals. Inhalations of oxygen help some patients, but not others. The pneumatic cabinet has been of benefit in some cases, under an increased pressure of from a half to one atmosphere.

During the interval, potassium iodid in doses of gr. v to xx (0.30—1.2) three times a day has proved the most effective remedy in most cases, often completely arresting the disease for a considerable length of time. The diet should be regulated with particular care as to the quantity of food taken in the evening. The patient should dine at noon,

eat a light supper, and should never eat late at night. The quantity of carbohydrates should be limited, especially at the evening meal. But many articles which agree with one patient cause great disturbance in another. Climate exerts a beneficial influence, but in this respect also patients differ. Some do well in the higher altitudes, but most of them, and particularly those having emphysema, generally do better near the seacoast, in an equable climate, like that of southern California or Florida.

DISEASES OF THE LUNGS.

HYPEREMIA.

Hyperemia, or congestion, of the lungs may be either active or passive. Both the parenchyma of the lungs and mucous membrane of the bronchial tubes are generally involved.

1. **Active Hyperemia.**—***Etiology.***—Some writers go so far as to doubt the occurrence of primary active hyperemia of the lung. There can be little doubt, however, that it sometimes occurs: (1) As a result of the inhalation of hot air, illuminating gas, or irritant vapors, and (2) sometimes in individuals whose occupation requires them to enter cold-storage vaults while actively working. (3) It is generally supposed to develop in one portion of the lung when the circulation of another part is interfered with; and it occurs (4) in the beginning of such pulmonary diseases as bronchitis, pneumonia, pleurisy, and tuberculosis. (5) It occasionally results from violent fits of coughing, (6) from too great atmospheric pressure, like that encountered by deep-sea divers and workers in caissons, or (7) from violent action of the heart, as that occasioned by athletic sports or cycling.

Symptoms.—The development of an active hyperemia is generally announced by a chill immediately or a few hours after its onset, with pain in the side, dyspnea, a dry cough, and moderate elevation of temperature (101°—103° F.; 38.3°—39.5° C.). Examination reveals diminished resonance, feeble or bronchial breathing, subcrepitant râles, sometimes over the entire affected lung. Death has resulted from the condition within the first 24 hours, but in most cases complete recovery occurs.

Passive Hyperemia.—This form of congestion is of two kinds, mechanical and hypostatic.

(*a*) *Mechanical hyperemia* or congestion is caused almost exclusively by valvular lesions or dilatation and weakness of the heart which interferes with the normal return of blood from the lungs. It is sometimes induced, however, by the pressure of aneurisms or other tumors. The condition produced in the lung is known as brown induration. The lung becomes distended with blood, its tissues indurated and of a brownish red color. On microscopic examination the capillary vessels are found to be distended, the connective tissue is hyperplastic, and the alveoli contain many desquamated epithelial cells in various stages of degeneration and pigmentation.

Symptoms.—The condition, when well marked, is indicated by dyspnea and the expectoration of sputum containing degenerated and pigmented alveolar cells and possibly free blood, in quantity sufficient to be

evident. Dullness may be found on percussion, and moist râles on auscultation.

(*b*) *Hypostatic Congestion.*—This condition is caused by weakness of the heart's action and favored, in some cases largely induced, by gravitation of the blood to the most dependent portion of the lung as a result of too prolonged lying in the same posture. It is most frequently encountered, therefore, in the continued fevers, notably in typhoid, and more chronic diseases. The posterior parts of the lungs are engorged with blood and become dark, often almost black. The affected portion of the lung may contain so little air that it will sink in water. This condition is often referred to as splenization. A form of hypostatic congestion, usually less pronounced than the foregoing, is met with in some cases of cerebral hemorrhage, especially in aged persons or as a result of cerebral tumors situated near the respiratory center, and sometimes in cases of uremic coma or opium-poisoning.

Symptoms.—The condition is recognized by dullness on percussion, the absence of the vesicular murmur, and the presence of moist râles, moderate dyspnea and cough, sometimes accompanied with blood-stained expectoration, under conditions favorable to its development. The congestion can be made to clear up on the affected side by changing the position of the patient.

Treatment.—In active congestion, great relief may be afforded by a hot bath, by the application of wet or dry cups, a poultice, or mustard over the affected area. In extreme cases, general blood-letting is more certain and prompt. Aspiration of the right auricle has been advised, if the blood does not flow freely from the arm. If blood-letting cannot be resorted to, the tincture of aconite in doses of a half to one drop every 15 minutes for an hour or two may be given for its action on the heart.

In passive congestion the chief indication is the treatment of the cause. Remedies should be applied to strengthen the heart's action and, if possible, to overcome the dilatation. Hypostatic congestion should be treated prophylactically. It should be prevented by proper attention to the posture of the patient. When it has developed, it may be removed by changing the posture, and by careful stimulation of the circulation, preferably with strychnin.

EDEMA OF THE LUNGS.

Definition.—A transudation of serum into the air-cells and alveolar walls of the lungs.

Etiology.—The most prominent cause of transudation is hyperemia, particularly passive hyperemia. The causes of edema are, therefore, practically the same as those of passive congestion, and the most important of them is a feeble action of the heart due to dilatation, degeneration, or chronic pericarditis. Edema occurs also in connection with chronic nephritis, hepatic cirrhosis, profound anemia, cachexias, or any condition in which there is a hydremic condition of the blood; in some cerebral affections and in some cases of acute ascending spinal paralysis. In all such conditions it is often a terminal affection, frequently occurring during the death struggle, a final relaxation of the blood-vessel

walls that permits the escape of serum. A so-called collateral edema occurs in the neighborhood of inflammatory processes, infarcts, new growths, and tubercular formations. An acute angioneurotic edema is also believed to occur, similar to that which affects the larynx and various other parts of the body. It comes on suddenly, often in an individual apparently in good health, except, perhaps, for a slight gastric disturbance, with attacks of gastralgia and vomiting. The cause is supposed to be some irritant in the blood, probably an unoxidizable product of digestion which causes vasomotor paralysis and consequent dilatation of blood-vessels and transudation.

Morbid Anatomy.—When the edema is great, the lung may have a gelatinous appearance; it is heavy, pits on pressure, and, when incised, discharges a large quantity of serum, which is blood-stained when the condition accompanies congestion. The edema may be general, but it is usually most marked at the base and dependent portions of the lungs.

Symptoms.—The symptoms are rapid breathing, audible bubbling or rattling, and dyspnea. There is the same sense of oppression as in asthma. The patient cannot lie down. All the respiratory muscles assist. The expectoration consists of an abundance of watery, frothy, blood-stained serum. Cyanosis often becomes extreme. Edema is usually present in other parts of the body, and the condition may be a part, usually the termination, of a general dropsy. The percussion note is dull, especially over the dependent portions of the lungs, and fine moist râles are exceedingly numerous in all parts of the chest. In secondary edema the temperature is sometimes subnormal, especially when it is the result of chronic nephritis, but in the so-called inflammatory edema there is always fever, and the condition closely resembles one of pneumonia.

Prognosis.—This is always grave, for the edema often proves rapidly fatal, sometimes within an hour. But in chronic cases several attacks of moderate severity are sometimes recovered from. The circumscribed, inflammatory edema is less dangerous.

Treatment.—A severe attack of pulmonary edema calls for prompt treatment. If there is much cyanosis and the condition of the patient will permit, free venesection affords the quickest relief. Dry cups may be applied freely over all parts of the chest, thirty or more at a single application. The object is not to draw blood, but to stimulate absorption. In some cases the patient is benefited by very hot fomentations, turpentine stupes, a poultice, or mustard applied to the chest. The inhalation of oxygen may assist in tiding the patient over. Strychnin should be given hypodermically, gr. 1-40 (0.0016), if the heart's action is feeble. Nitroglyerin (gr. 1-50; 0.0013) assists in equalizing the circulation, and a free purge should be given to aid absorption.

PULMONARY HEMORRHAGE.

Two very different conditions are described under this head; bronchopulmonary hemorrhage or bronchorrhagia, and pulmonary apoplexy or hemorrhagic infarct, sometimes referred to as pneumorrhagia.

1. **Bronchopulmonary hemorrhage** is the form that is usually designated by the term hemoptysis, or the spitting of blood. Although some

writers include in this class of cases hemorrhages from the upper respiratory passages, the term is generally understood to apply only to those in which the blood escapes into the bronchi. Flint restricts the term to the raising of blood, and blood only. The most important conditions in which this occurs are: (*a*) In young persons apparently in good health, a more or less profuse hemoptysis, or a slight expectoration of blood for several days, sometimes occurs without discoverable lesion of the lungs, and is followed for many years by good health and no recurrence of the hemorrhage; (*b*) tuberculosis. This has been considered in the chapter on that disease; (*c*) ulceration of the larynx, trachea, or bronchi. This form is sometimes rapidly fatal from erosion of a branch of the pulmonary artery. (*d*) Pure blood is sometimes expectorated in the primary stage of engorgement in acute pneumonia, in bronchitis, bronchiectasis, emphysema, abscess, gangrene, or cancer, less frequently in sarcoma of the lung. (*e*) Profuse and recurrent hemorrhage sometimes occurs during the course of valvular disease of the heart, more frequently with mitral stenosis than with insufficiency or aortic lesions. (*f*) Aneurism of a branch of the pulmonary artery within the lung usually terminates in a fatal hemoptysis. Aneurism of the arch of the aorta sometimes perforates a bronchus and produces an immediately fatal hemoptysis. But the fatal hemorrhage is sometimes preceded for days or weeks by the expectoration of a small quantity of blood from pressure or erosion, and later from an oozing of blood through the laminæ of fibrin which alone remain. (*g*) Vicarious hemoptysis occasionally replaces menstruation, especially in hysterical and anemic women, or for a time after removal of the ovaries. Hemopytsis after cessation of the menses has been known to continue for several years; but it sometimes indicates the development of tuberculosis, and deception is often practiced by this class of patients. (*h*) Hemoptysis has been observed in connection with the arthritic diathesis in individuals past 50 years of age; (*i*) purpura hemorrhagica and malignant infections; (*j*) parasitic diseases of the lungs, particularly Distomum Westermanni, met with especially in China and Japan.

Exertion, a blow upon the chest, or mental excitement is sometimes the immediate cause of hemoptysis in a person already predisposed to it by pulmonary disease.

Symptoms.—In a majority of cases the hemorrhage comes on suddenly, often at night and during sleep. Sometimes it follows a fit of coughing, strong vocal effort, unusual excitement, or exertion. The first indication of it is usually a welling up into the mouth of the warm, salty fluid. The quantity expectorated varies much with the condition leading to the hemorrhage. Very often the bleeding ceases after an ounce or less has been brought up, or a dram or less may be expectorated at intervals for several days; but in some cases of continued hemoptysis, repeated losses of several ounces occur at short intervals. When an aneurism ruptures into the lung, there is usually a sudden gush that overwhelms the patient. Only a small part of the blood is usually expectorated. In some cases, particularly in those of tuberculous origin, the blood is sometimes poured into a large cavity within the lung, and death occurs from the hemorrhage, without expectoration of blood.

Coughing is generally provoked by the hemoptysis; the patient becomes pale, and the heart's action may be feeble, but this is usually due to the alarm that is naturally occasioned, and not to the loss of blood. After a hemoptysis it is not unusual for the patient to vomit some blood that has been swallowed; sometimes there is blood in the stools for a day or two. The sputum continues to be streaked with blood for a few days after cessation of the hemorrhage.

Diagnosis.—It is not usually difficult to distinguish pulmonary hemorrhage from the other conditions in which blood is expectorated. The statement of the patient that the blood has been coughed up, and not vomited, is generally correct, and the appearance of the blood is quite different. In hemoptysis it has a bright red color and usually contains numerous small air-bubbles which may give it a frothy appearance. Blood from the stomach is generally dark and clotted. If doubt exists, or if the blood be not frothy, it is well to examine the pharynx, after having the patient gargle with water, for blood from the posterior nares may flow back into the throat. Auscultation of the chest reveals moist râles in the affected part of the lung, but it is of little value unless the previous condition be known, and the patient should not be disturbed for examination. Percussion should not be practiced.

Prognosis.—This depends entirely upon the character of the hemoptysis. In a majority of cases the bleeding ceases spontaneously, except when it is from a vessel of considerable size.

Treatment.—Rest is the most important element of treatment. The patient should be placed in a comfortable position, better on the affected side, in order to avoid aspiration of blood into the healthy lung. He should be given all justifiable assurance of recovery, and impressed with the importance of quiet and silence. No remedy is so valuable as opium, for it induces rest, quiets the heart's action, and allays the cough. Morphin may be administered hypodermically (gr. ⅛ to ¼; 0.008—0.016), and followed with heroin (gr. 1-12; 0.005) or codein (gr. ¼; 0.016) every four hours. Aconite is often indicated to quiet and strengthen the heart's action and to reduce the pressure in the pulmonary artery. Digitalis, ergot, styptics, are all more or less positively contraindicated. The application of cold to the chest is favored by some writers, but it is often more annoying to the patient than beneficial. Probably the best method of reducing intra-arterial tension is compression of the brachial and femoral veins by means of an elastic band, or any convenient strap or bandage, passed around the arm and leg and drawn just tightly enough to arrest the venous circulation without compressing the artery. Not more than three extremities should be compressed at the same time, and one tourniquet should be removed every fifteen minutes, and, if necessary, placed upon the remaining limb. This method is often effective in arresting the more profuse hemorrhages which cannot be influenced by medicinal means.

The diet of the patient should be light and nutritious. Stimulants should not be given, unless the patient is in an extreme condition from the loss of blood, and they should then be given hypodermically. A purge is generally indicated; repeated purgation is especially beneficial in cases of continued hemorrhage.

2. **Pulmonary Apoplexy** (Hemorrhagic Infarct of the Lung).—A

condition in which the tissue of a limited portion of the lung is infiltrated and the air-cells more or less completely filled with blood as a result of embolism or thrombosis.

Etiology.—This affection, which is not to be regarded as a hemorrhage in the proper use of the term, results in most cases from the obstruction of a branch of the pulmonary artery with either a thrombus or an embolus. It is usually a sequel of heart disease. The emboli are septic when a result of malignant endocarditis or pyemia, and the extravasation of blood in such cases may be slight. In the chronic forms of heart disease the embolus usually consists of a vegetation from one of the valves, and it is not septic. It may be derived from a remote thrombus, as that of the femoral vein, after typhoid fever.

Morbid Anatomy.—The affected, wedge-shaped area is solidified, dark red in color, and a fibrinous pleurisy develops over its base. The subsequent changes are those peculiar to thrombosis. (See p. 15.) If recovery occur, the tissue is converted into a firm cicatrix. In some instances, caseation or calcification results, or, when septic, the tissue breaks down and forms an abscess or gangrene; general pyemia is possible. One or many infarctions may occur in the same lung.

Symptoms.—A large embolus sometimes causes sudden death before an infarction has had time to develop. On the other hand, the vessel may be so small that its obstruction produces no symptoms. In other cases the patient is seized with a sudden, severe pain in the lung, urgent dyspnea, sometimes a chill and slight elevation of temperature. Examination reveals circumscribed dullness, generally in the region to which the pain is referred, and tubular breathing. Mucus streaked with blood is usually expectorated.

Diagnosis.—The differential diagnosis usually rests between hemorrhagic infarct and pneumonia. Infarction does not occur as a primary disease. In pneumonia the initial chill is more severe, the fever much higher, the lung is more extensively involved, and auscultation reveals the characteristic crepitant râle, or the subcrepitant, over a larger area than is generally affected in infarction.

The ***treatment*** is directed to the relief of pain, weakness, and other symptoms as they arise.

BRONCHOPNEUMONIA.

LOBULAR PNEUMONIA, CAPILLARY BRONCHITIS, CATARRHAL PNEUMONIA.

Definition.—An acute inflammation, probably of infectious origin, affecting the terminal bronchi, air-cells, and interstitial tissue of isolated lobules, or groups of lobules in different parts of the lungs. It usually begins in the mucous membrane of the bronchus and extends to the air-cells.

Etiology.—The disease is regarded by many investigators as an infection, but the specific organism has not been determined. Several bacteria have been more or less regularly found, notably the Bacillus pneumoniæ, the Micrococcus lanceolatus, and the staphylococci and streptococci of suppuration. A mixed infection is present, as a rule. Bronchopneumonia is peculiarly a disease of the extremes of life, affect-

ing most frequently and most seriously the infant and the very aged. It is encountered, however, in middle adult life, particularly as a secondary affection. It may occur as a primary disease, and Holt's statistics indicate that the remarkably high ratio of one case in three is primary, without previous involvement of the bronchi. It is generally secondary to bronchitis.

Primary Bronchopneumonia.—This form of the disease generally occurs in infants and is probably due in most cases to pneumococcus infection. Cases following prolonged inhalation of ether, smoke, or irritant vapors are generally included in this class.

Secondary bronchopneumonia follows bronchitis of the larger tubes and is a common sequel of such affections as measles, pertussis, diphtheria, scarlet fever, influenza, or erysipelas, and it not infrequently follows acute ileocolitis in delicate, improperly fed children, or those suffering with inherited syphilis or tuberculosis. Pulmonary collapse or atelectasis from any cause is almost invariably followed by it. In adults it is often encountered as a result of influenza, variola, emphysema, occasionally in the course of typhoid fever, or as a terminal affection in bronchiectasis, emphysema, chronic bronchitis, asthma, interstitial pneumonia, and tuberculosis. Rickets greatly increases the susceptibility of a child, and long confinement to bed that of an adult.

Aspiration, inhalation, and **deglutition pneumonia** are terms applied to bronchopneumonia developing as a result of the entrance of foreign bodies into the bronchi. This occurs when small particles of food or drink enter the larynx when it is benumbed by paralysis, coma, or anesthesia, or when the epiglottis is ulcerated by syphilis or tuberculosis. Pus or fragments of neoplasms and blood are sometimes aspirated during operations or after the rupture of an abscess in the mouth or pharynx. The exciting cause of the disease is not so much the irritation caused by the foreign substance as the bacteria which are conveyed with it.

J. N. Hall has observed severe cases of bronchitis and bronchopneumonia following inhalation of sulphurous-acid gas, formaldehyd, kerosene, smoke and other vapors.

Morbid Anatomy.—Although the bronchi of all sizes may be found in a state of hyperemia, the pneumonic process is limited to the terminal tubes in small areas. It is strictly lobular in extent, but, owing to the involvement of adjacent lobules, areas of considerable size are often found to be involved, particularly along the margins of the lungs. Sometimes almost an entire lobe is involved. The affected areas do not fully collapse with the rest of the lung as the air escapes; they are not solidified, yet they are firmer and do not crepitate so freely as the surrounding lobules. On section, they appear slightly more prominent, of a brighter red color than the surrounding tissue, which is also hyperemic for a variable distance (3 to 5 mm.). Beyond these regions of inflammation the lung tissue appears normal. The medium and smaller bronchi are filled with mucopurulent matter. The air-cells are more or less filled with serum, which, on microscopic examination, is found to contain numerous leucocytes and desquamated, swollen endothelium. A few red blood-corpuscles are occasionally seen and possibly a trace of fibrin, but not to the extent that they are present in lobar pneumo-

The absence of fibrin and red corpuscles is usually a distinguishing feature. The air-cells nearest the terminal bronchus are the most densely filled with cellular elements. The walls of the alveoli and those of the terminal bronchi appear swollen on account of the distention of the capillaries and infiltration with leucocytes. A compensatory emphysema is generally to be noted in the uninvolved portions of the lungs. The tracheobronchial glands are usually enlarged and inflamed, a fact which explains their frequent infection with tubercle bacilli after the infectious diseases that are attended with bronchitis.

In aspiration or deglutition pneumonia the infiltration is more intense and more liable to become suppurative.

Termination.—Bronchopneumonia terminates: (*a*) In a rapid resolution; (*b*) in caseation, which is generally only a form of tubercular infection; (*c*) in suppuration or gangrene, especially in the deglutition or aspiration form; or (*d*) in a chronic interstitial pneumonia, also more commonly seen in patients who were previously tuberculous.

Symptoms.—The primary form begins in a previously healthy infant with a convulsion, less frequently with a chill, vomiting, prostration, rapid respiration, often reaching 60 in a minute, and elevation of temperature, possibly reaching 104° F. (40° C.). There may be no cough, and infants do not expectorate. The lesions are more definitely localized than in the secondary form. Cerebral symptoms, delirium, photophobia, convulsions, and rigidity, are sometimes so pronounced as to mask the pulmonary affection, unless proper attention is given to the rapid respiration and evidences of dyspnea. The case often terminates with a crisis toward the end of a week, and rapid recovery usually follows. The mortality is slight, except in debilitated infants. The disease is with difficulty differentiated from lobar pneumonia during life.

The secondary form occupies a more positive place in nosology. Following a bronchitis, perhaps during convalescence from measles or other acute infection, the temperature rises, the breathing and pulse become accelerated, and the cough more frequent and severe. The cough is often painful and the infant cries; the respiration is often labored, the lower part of the chest is drawn in by the diaphragm, the alæ of the nose vibrate, and cyanosis often develops. Percussion reveals areas of dullness in some cases, but it is more frequently negative. On auscultation, numerous subcrepitant râles are heard, particularly over the base of the lungs and on either side of the spine. The fever generally reaches 103° or 104° F. (39.5°—40.0° C.), and the skin feels hot and dry. The thirst is urgent, but the child cannot drink, and the infant refuses the breast on account of the rapid respiration and dyspnea. As the disease progresses, often within 24 to 48 hours the dyspnea and cyanosis rapidly increase. The right ventricle is overcome in its effort to maintain the circulation in the lungs and becomes increasingly dilated. The cyanosis rapidly deepens; the child struggles for breath, but finally sinks into unconsciousness, overcome by the accumulation of carbon dioxid in its blood; the breathing becomes less labored, the mucus is more fluid and rattles in its throat, and soon the heart ceases, from paralysis.

When recovery is about to occur, the symptoms gradually ameliorate; the fever subsides, and, usually by the end of a week, convalescence is established. It not infrequently happens, however, that convalescence

is delayed or slow. The child improves, but the cough persists and the emaciation continues. In such cases there may be ultimate recovery, but in some instances a portion of one or both lungs remains permanently collapsed or a chronic interstitial pneumonia is set up; some finally die of exhaustion, and others develop tuberculosis.

Diagnosis.—In primary cases the differentiation generally lies between bronchopneumonia and lobar pneumonia. The former is more frequent in young infants, the latter after the third year. Lobar pneumonia is usually unilateral and confined to a definite region of one lung, which can be determined by the dullness on percussion. Bronchopneumonia affects both lungs, and there is often a general tympanitic note without recognizable dullness anywhere. The cough, pain, and fever are generally more severe in the lobar form; yet in many cases the distinction is extremely difficult.

In secondary cases the diagnosis is much less obscure. The disease follows a bronchitis or an acute infection. The onset is gradual, the fever is more moderate, and the physical signs are more definite. There may be little or no recognizable dullness, but fine moist râles are heard in definite areas over both lungs. Acute tuberculosis, in the beginning, is sometimes differentiated with difficulty, although the temperature generally remains more uniformly high, with periodical sweats, especially at night.

Prognosis.—The result depends largely upon the condition of the patient. The disease is very fatal in the extremes of life. The primary form generally terminates in recovery, but the secondary is always to be feared.

Prophylaxis.—The liability to the development of bronchopneumonia should always be borne in mind in the treatment of the acute infections, particularly measles and whooping-cough. Most important, probably, is the avoidance of exposure to cold, or rather to infection. The patients should be kept warm in flannel gowns, and they should not be released from confinement until all danger has passed. The sick-chamber should not be allowed to become cold during the night. The regular cleansing of the mouth with an antiseptic solution is also important in all diseases which may lead to bronchopneumonia.

Treatment.—The patient should be confined to bed in a room kept at a temperature of 68° F. (20° C.), and the air should be kept moist by the evaporation of water. The treatment is largely symptomatic. The fever should be kept within bounds by the administration of tincture of aconite, ♏j (0.06) every hour or two according to the age and the effect. Cool sponging or the graduated bath serves the same purpose, but is often objected to and condemned as the cause of subsequent accidents. The coal-tar antipyretics should be avoided on account of their depressing effects. Opium should not be used, unless the pain and cough cannot be controlled by any other means. A hot poultice around the chest, although not now in fashion, or the more cleanly cotton jacket which may be pressed out of hot water and made to serve as a poultice, often relieves the pain, and is probably beneficial in other respects. The ice-poultice and cold-water jacket are rarely employed in this country. A simple expectorant consisting of ammonium chlorid, gr. i to ij (0.06—0.12), or the carbonate, gr. ¼ to 1 (0.01—0.06), with

sirup of ipecacuanha, ♏v to x (0.3—0.6) in each dose, in tolu or other sirup, prevents the accumulation of tenacious mucus, providing opium has not been given, and thus renders the cough less annoying. An occasional emetic dose of wine of ipecacuanha may be required to clear the bronchial tubes. The strength of the patient must be maintained with nutritious food, chiefly milk, beef-juice, broths, and egg albumen. The child must be given an abundance of cold water to drink, and brandy (♏x to xv; 0.6—1.0, to an infant) should be given at regular intervals, or strychnin may be administered hypodermically. The bowels should be opened with calomel, gr. 1-10 (0.006) every two hours, until it acts, and kept regular during the illness with magnesium citrate or other laxative. When cyanosis appears, every effort must be made to arouse the patient and induce coughing or vomiting. If the mucus can be removed from the tubes, an apparently hopeless case will sometimes recover.

CHRONIC INTERSTITIAL PNEUMONIA.

SCLEROSIS (CIRRHOSIS) OF THE LUNG, FIBROUS PNEUMONIA, CHRONIC FIBROSIS OF THE LUNG.

Definition.—A chronic inflammation of the interstitial tissue of the lung resulting in proliferation, with subsequent contraction and diminution of air-space. It may be local or diffuse in character.

Etiology.—The disease may be either primary or secondary. Primary cases are generally due to the inhalation of dust. This is described in the following chapter, on Pneumokoniosis. Secondary cases occur in the course of chronic tuberculosis, syphilis, chronic bronchitis, emphysema, or chronic pleurisy, less commonly as a result of bronchopneumonia or lobar pneumonia. These cases are usually classed as examples of the diffuse form.

The local form of the disease is met with as a result of penetrating wounds, the presence of a foreign body, pressure of a tumor or aneurism, or the irritation of healing tubercular nodules. It is commonly met with around bronchiectatic cavities and beneath a thickened pleura.

Morbid Anatomy.—The essential lesion is a firm mass of connective tissue from which lines of similar hyperplastic tissue usually radiate into the surrounding lung substance. The primary hyperplasia may take place around the blood-vessels, the bronchial walls, the interlobular spaces, around the bronchioles, or in the pleura. The resulting conditions are described under two forms, the massive or lobar, and the insular, diffuse, or bronchopneumonic.

(*a*) The massive form is unilateral, affecting a lobe or the entire lung and producing extreme deformity of the chest with approximation or overlapping of the ribs and depression of the shoulder in extreme cases. The heart is drawn toward the affected side, and the opposite lung is emphysematous, while the affected lung is often shrunken into an extremely small mass close to the bodies of the vertebræ. In cases of long standing the tissue has an almost cartilaginous hardness. Tubercular or bronchiectatic cavities are often found in the interior, and within these aneurisms of the pulmonary artery are sometimes found.

(*b*) In the bronchopneumonic form the areas are smaller, less indu-

rated, and usually pigmented. They are found in all parts of the lung, as a rule, but may be confined to the lower lobes.

Symptoms.—The disease is an exceedingly chronic one. The symptoms are most pronounced in the early stage of its development; after it has become fully established, they generally subside to a great extent and the individual continues in fair health indefinitely. There is a chronic cough, and he becomes short of breath upon exertion, as in going upstairs or walking up hill. In many respects the case resembles one of bronchiectasis, especially in the periodical expectoration of large quantities of mucopurulent, sometimes fetid matter. The nutrition generally fails, and the patient then appears tuberculous, especially if hemorrhage occurs, a possible accident in nearly half the cases. But the anemia is not generally so marked. The absence of bacilli is the distinguishing feature.

Physical Signs.—These are exceedingly variable. In extreme cases the affected side is shrunken and immobile, often retracted until the ribs overlap and the opposite side seems to be enlarged. The shoulder is depressed, and the spine has generally a lateral curvature. Percussion of the affected side may reveal flatness or partial dullness, with a tympanitic or amphoric quality over existing cavities. Over the other lung the tone is one of exaggerated resonance (tympanitic resonance). The breath-sounds and voice-sounds depend upon the condition of the contracted lung. The vesicular murmur is generally replaced by a tubular, cavernous, or amphoric breathing in the apex and by moist râles at the base. The disease ultimates fatally from dilatation of the right ventricle and dropsy, or from exhaustion, sometimes from amyloid disease of the viscera, or earlier from hemorrhage.

Diagnosis.—Interstitial pneumonia cannot be mistaken for any other disease except fibroid phthisis, in which the lesions and physical signs are virtually the same, with the additional and distinctive feature, the tubercle bacillus.

Treatment.—Nothing can be done to arrest or counteract the disease. Life may be prolonged by residence in a mild climate, where the liability to bronchitis is least, and by maintaining the nutrition with tonics, the best of which in most cases is codliver oil. When the cough becomes troublesome or the expectoration fetid, the treatment is the same as for chronic or fetid bronchitis.

PNEUMOKONIOSIS.

ANTHRACOSIS, SIDEROSIS, CHALICOSIS, MINER'S LUNG, KNIFE-GRINDERS' PHTHISIS, ETC.

Definition.—A form of fibrous induration of the lung due to the inhalation of particles of dust in various occupations. Anthracosis signifies induration from the inhalation of coal-dust; siderosis, that from the inhalation of metallic dust, especially iron, as from the emery-wheel; chalicosis, that from the inhalation of mineral dust, as in stone-cutting.

Etiology.—The irritant action of the particles of dust upon the connective tissue of the lungs is probably the only etiological factor in

the production of the disease. It does not, however, excite proliferation with the same degree of certainty in all individuals.

Morbid Anatomy.—A large part of the inhaled dust is carried back by the ciliated epithelium of the bronchial tubes and expectorated. A smaller part is picked up by phagocytes, even from the surface of the mucous membrane. Part of these cells then pass out in the sputum, but part of them carry their burden into the lymph-channels and to remote organs, particularly the bronchial glands, liver, and spleen. In individuals who constantly breathe a smoky atmosphere, the surface of the lungs becomes deeply discolored, sometimes jet-black, as a result of the passage of particles of soot into the lymph-spaces and thence into the connective tissue beneath the pleura. The particles probably do not reach the air-cells directly, but they are often found in the alveolar epithelium, having been picked up by these cells while passing through the bronchial tubes. When the quantity of dust that gains access to the bronchi becomes greater than can be carried out or disposed of by the carrier-cells, much of it is stored in the lymph-spaces and connective tissue of the lungs, beneath the pleura, around the bronchi and air-cells. The irritation produced excites proliferation of the tissue and a fibrosis is produced, much like that of chronic interstitial pneumonia, except that the new fibrous tissue is always deeply pigmented. Later, this tissue often undergoes necrosis, and cavities are formed in the lungs. The lesions are bilateral; the bases of the lungs are more extensively affected than other parts, but nodules and cavities are not infrequently found scattered through all parts of both lungs. Chronic bronchitis is a constant accompaniment, and there is generally emphysema. Sometimes the necrotic cavities become tubercular.

Symptoms.—The clinical features of the case are the same as those of chronic bronchitis with emphysema, except that there is a more profuse expectoration of black or otherwise discolored mucopurulent sputum. This discoloration continues for a long time after the patient has abandoned his dusty employment. Examination of the sputum reveals numerous leucocytes, bronchial and occasional alveolar epithelial cells containing dust-particles. The presence or absence of tubercle bacilli depends wholly upon the presence of tubercular infection. Dyspnea is often a prominent symptom, but it depends for the most part upon the emphysema. More or less pronounced asthmatic attacks occur in many cases. The chest often becomes barrel-shaped, as a result of the emphysematous condition.

Treatment.—The case should be treated as one of chronic bronchitis with emphysema. The condition of the lungs cannot be modified by treatment.

EMPHYSEMA.

Definition.—Pulmonary emphysema is a condition of the lungs characterized by distention or dilatation of the infundibula and alveoli, associated, when permanent, with atrophy of their walls. Five more or less distinct forms of the disease are recognized, namely, the compensatory, hypertrophic, atrophic, acute vesicular, and interstitial.

1. **Compensatory Emphysema.**—This condition is produced in one

lung or in a part of either lung whenever some other part is prevented from expanding during respiration. It is compensatory in that it enables the lung, by overexpansion, to fill the space that should be filled by the part whose movement has been arrested. A temporary condition of emphysema is produced in the normal portions of the lungs: (*a*) In the presence of atelectasis or collapse; (*b*) in all the acute conditions attended with consolidation or bronchial obstruction, as in the pneumonias; and (*c*) a more persistent form occurs in connection with pleuritic adhesions, hydrothorax, empyema, pneumothorax, chronic interstitial pneumonia, and tuberculosis. In the purely compensatory condition the alveolar walls are merely distended without undergoing atrophy, and return to their normal condition with the removal of the exciting cause.

2. **Hypertrophic Emphysema.**—The lungs in this condition are much enlarged by the distention of their air-cells. The condition is known also as substantive or idiopathic emphysema, and it is the form that was described by Sir William Jenner as the "large-lunged emphysema," in contradistinction to the atrophic or "small-lunged emphysema."

Etiology.—A hereditary predisposition to the disease, in the form of a congenital defect in the structure or nutrition of the tissue of the lungs, is generally believed to exist, for the disease does not develop in all individuals, or to the same extent, under the same influences. The disease very commonly develops in early life and especially in the children of those affected with it. It is by no means uncommon, however, later in life, and may develop in the aged.

The next important factor in etiology is an increased pressure within the air-cells. This may result from either inspiratory or expiratory force, but it is doubtless more readily and more commonly produced by forcible expiration, such as occurs in pertussis, asthma, chronic coughs, and in playing wind-instruments, glassblowing, and other occupations requiring prolonged acts of blowing. Mechanical causes are believed to be effective only in the presence of a weakened power of resistance in the lung. Dilatation of the heart, a condition usually encountered as a result of emphysema, and alcoholism are regarded by some writers as predisposing causes in some instances. The presence of adenoid vegetations in the nasopharynx is at least a probable cause in a child with inherited predisposition.

Morbid Anatomy.—The chest has a barrel-shape, a condition attributed by some writers to the expansion of the lungs within, by others to the action of the external respiratory muscles. The dilatation of the lungs is generally so great that the pericardium is completely concealed by the overlapping of their anterior margins, when the chest is opened. They do not collapse, but pit on pressure, on account of a complete loss of elasticity. Immediately under the pleura can be seen numerous enlarged air-vesicles, varying in diameter from 1 to 3 or 4 mm. Along the margins and over the inner surfaces, near the heart, there are bullæ, varying in size from that of a pea to that of a hen's egg, which have resulted from a coalescing of several smaller vesicles, fragments of whose walls can be seen in the interior with a suitable lens or in microscopic section. Other histological changes are also to be noted. With the distention of the air-cells the capillary vessels are

stretched longitudinally at the expense of their caliber. As a result, the network becomes imperfect and gradually disappears from the larger sacs. The elastic tissue of the distended air-cells is also lost—some regard it as congenitally defective; and the epithelium is reduced to a delicate squamous layer lining the inner surface of the bullæ. As a result of the destruction of the capillary circulation, increased action is thrown upon the right ventricle. This leads to hypertrophy and in time to dilatation. In the more chronic cases both sides of the heart become hypertrophied, but the right side is most dilated. The pulmonary artery also yields to the increased tension within it and becomes enlarged. Atheromatous changes are commonly found in its walls.

The bronchi also show important changes. The mucous membrane in the larger tubes is often greatly thickened and indurated. The longitudinal elastic fibers sometimes stand out like cords. Around the tubes there is generally a fibrous-tissue hyperplasia, a sclerosis, which leads to dilatation, particularly of the smaller bronchi. Atelectasis is sometimes present, but it is not a frequent result of emphysema. The more remote organs, especially the liver and kidneys, are found in a more or less advanced stage of chronic passive hyperemia.

Symptoms.—Emphysema is an exceedingly chronic disease, and its symptoms from time to time depend largely upon the existence of bronchitis, the extent to which the circulation in the lung is impaired, and the development of complications. There is usually a considerable period, especially in childhood, during which the patient experiences comparatively little discomfort. The muscular system may be well developed and strong, but the body generally becomes emaciated as the disease progresses. The most important symptoms are dyspnea, cyanosis, and bronchitis.

(*a*) *Dyspnea* may be almost constant, but in many cases it is complained of only after a full meal or after exertion. It is at first largely expiratory in character, but later assumes, in many instances, the form of asthmatic seizures, with both inspiratory and expiratory obstruction. A distinct wheezing or rattling rhoncus is often audible to the patient, and it can sometimes be heard at a distance of several feet from him, most markedly with expiration.

(*b*) The *cyanosis* is a common and often a most striking feature of the disease. It accompanies the dyspnea, but is often severe beyond all comparison with that condition. The patient often shows little discomfort; he may be able to walk about when his face is puffy and his lips and finger-tips are blue. So deep cyanosis is not often seen except as a result of anilin-poisoning, congenital heart-lesions, or the most advanced organic cardiac or pulmonary disease, and then only in persons confined to bed.

(*c*) The *bronchitis* is most troublesome in winter, often disappearing almost completely during the summer. It is excited by the slightest exposure and assumes a severity that is unusual in a previously healthy person, intensifying the dyspnea and cyanosis and often inducing severe paroxysms of asthma. With the advance of age it becomes a more dangerous condition and may ultimately lead to a fatal bronchopneumonia.

Physical Signs.—*Inspection.*—The appearance of the chest is so typical

as not to be mistaken. It is the so-called barrel-shaped chest. The thorax is round and deeper than the normal. The anteroposterior diameter may exceed the transverse; the ribs are more horizontal, and the interspaces are widened. The sternum and clavicles are prominent, and fossæ above them are deep. The back is rounded, the shoulders are raised and drawn forward. The respiratory muscles, including all the accessory muscles, are strong and stand out prominently. Their action during active respiration is exaggerated, and the thorax is drawn up as a solid frame; there is little or no expansion. Tranquil respiration is carried on almost entirely by the diaphragm. The expiration is prolonged, and the thorax sinks more slowly than it rises. During inspiration, the upper part of the abdomen often remains fixed, or it may sink, and the suprasternal fossa is usually drawn in. A transverse curve running across the abdomen at the level of the last ribs has been noted. The veins of the neck are distended and usually pulsate. The apex beat of the heart is not visible, but there is usually a strong epigastric pulsation.

Palpation reveals but slight vocal fremitus, the absence of the apex beat, a strong impulse beneath the lower portion of the sternum, and a forcible epigastric pulsation.

Percussion elicits a peculiar type of resonance which is described by different authors as increased resonance, hyper-resonance, or tympanitic resonance. The quality is more or less drumlike and peculiar to the emphysematous chest. It partakes of the tympanitic quality as compared to the normal percussion note, but it is not tympanitic when compared with that of the abdomen. The distention of the lungs is revealed by the obliteration of the cardiac dullness and the lowering of the upper margins of the liver and splenic dullness.

Auscultation.—The vesicular quality of the respiratory murmur is lost. In the absence of bronchitis, the respiration may be almost inaudible, but there is, as a rule, a distinct prolongation of the expiratory murmur, generally accompanied with wheezing and coarse, sonorous, and sibilant râles. When bronchitis is present, the respiratory sounds are replaced by the moist râles belonging to that affection. The heart-sounds can be distinctly heard and may be normal, with the exception of an accentuation of the pulmonary second sound; but in the later stages of the disease there is often a tricuspid regurgitant murmur.

Diagnosis.—The disease cannot be mistaken for any other. Even when the physical signs are masked by those of bronchitis the condition is fully revealed by the appearance of the thorax, the absence of the apex beat, and the disappearance of the usual boundaries of the solid organs.

Prognosis.—Emphysema is incurable; under the most favorable conditions it is slowly progressive, for the elasticity of the lung cannot be restored, and the damage to the pulmonary circulation is a permanent one.

Treatment.—When the disease is encountered early, as in a young child with asthma, a thorough examination should be made of the upper respiratory passages; nasal defects and pharyngeal adenoids or polyps should be removed in the hope of abating the causal influence. After the disease has been developed, nothing can be done, and the

treatment is directed, for the most part, toward the accompanying bronchitis. The emphysematous patient should reside in a warm, dry climate where he can best escape the many influences which excite bronchitis. The diet should be regulated with reference to the prevention of constipation and flatulency. Starches and sugar should be restricted in quantity. Strychnin is a valuable tonic, assisting the weakened heart to perform its function. For extreme cyanosis, oxygen may be inhaled, but there is no better remedy in young, robust patients than free venesection.

3. **Atrophic Emphysema.**—This form of ephysema is purely a senile change, a part of the general wasting which marks the closing years of a long life. It occurs in either sex and is attended with atrophic changes in nearly all other tissues of the body. These patients usually give a history of winter cough, with greater or less difficulty of breathing, for many years. The chest is not expanded, but small, and the obliquity of the ribs is increased. It is only the condition of the lungs that is characteristic of emphysema. In a typical case the air-cells are found to have coalesced into a series of large bullæ. The blood-vessels have undergone atrophy, as in the hypertrophic form of the disease.

4. **Acute Vesicular Emphysema.**—A condition in which the air-cells are acutely distended as a result of strong expiratory efforts in some cases of bronchitis affecting the smaller tubes, bronchopneumonia, cardiac dyspnea, angina pectoris, or asphyxia. The lungs are much enlarged after death. The condition can sometimes be recognized during life by the emphysematous resonance, increase in area, prolonged expiration and loud sibilant râles over all parts of the chest.

5. **Interstitial Emphysema.**—This form of emphysema, which corresponds to surgical emphysema, results from the passage of air into the interstitial tissue of the lungs. It is generally caused by an extreme expiratory effort, as in whooping-cough, bronchopneumonia, convulsions, parturition, defecation, or lifting, a rupture of air-vesicles being produced. The air accumulates in minute bubbles beneath the pleura and in the interlobular spaces. Sometimes it finds its way into the mediastinum and thence into the cellular tissue of the neck. After tracheotomy an interstitial emphysema sometimes develops from the passage of the air down along the trachea to the lungs. Pneumothorax may also result from an interstitial emphysema.

PULMONARY COLLAPSE.

ATELECTASIS.

The term atelectasis signifies the airless condition of the lung before birth, or as a result of the failure to establish respiration after birth. Pulmonary collapse, on the other hand, applies to the collapse of a portion of a lung when a bronchial tube becomes obstructed by a foreign body, a plug of mucus, or from other cause, and to the occasional collapse of a portion of one or both lungs in a feeble, syphilitic child. Collapse may be caused also by the pressure of tumors, hydrothorax, empyema, or pneumothorax. The condition is then known as carnifi-

cation. Bronchopneumonia is a common sequel of partial collapse. A collapsed lung may regain its normal condition, especially when due to hydrothorax, after early removal of the compressing fluid, but later it becomes firmly adherent, and expansion is permanently prevented. The collapsed lung contains little or no air, and has a dark red color from engorgement with blood. The condition is usually a part or sequel of some other condition, and rarely calls for separate treatment. The only measure for its relief, in fact, is the removal of the cause, and this is rarely possible.

ABSCESS OF THE LUNG.

Etiology.—1. Suppuration of the lung results from septic infection after inflammation. It is sometimes a sequel of lobar pneumonia, more frequently of lobular, and exceedingly common after deglutition or aspiration pneumonia. The form of aspiration pneumonia most frequently leading to suppuration is that arising from the entrance of pus or septic matter of any kind during operations upon the nose or throat. Multiple abscesses varying in size from an inch to two inches (2.5—5.0 cm.) in diameter are generally produced, but a solitary abscess is occasionally met with.

2. Embolic or metastatic abscesses are generally a part of a pyemic infection, multiple septic emboli reaching the lungs through the circulation from more or less remote sources, as from a malignant endocarditis, an endophlebitis, or pyonephrosis. The abscesses are, therefore, multiple and may be extremely numerous. They are generally found immediately beneath the pleura. The lodgment of an embolus at first produces a hemorrhagic infarction, but owing to the septic nature of the obstruction the subsequent changes are suppurative. The pleura is at once affected with a septic fibrinous inflammation, and perforation is not uncommon, with the production of pneumothorax.

3. Abscess results also from perforation of the lung from without, the lodgment of foreign bodies, especially bullets, or from the rupture of a subdiaphragmatic or hepatic abscess or echinococcus cyst.

4. Finally, one of the most common types of pulmonary suppuration is that associated with tuberculosis.

Symptoms.—Suppuration of the lung following an inflammatory condition is usually announced by a return of the fever, pain, and dyspnea, with rapid respiration. Later, pus is found in the sputum, or, if the abscess be large, a correspondingly large quantity of pure pus may be expectorated. The pus is often extremely offensive. Multiple pyemic abscesses are often unrecognizable on account of the intense general pyemic condition that is present. The discovery of pus in the sputum, with, perhaps, fragments of elastic tissue, is highly diagnostic of the condition.

Prognosis.—Embolic and aspiration abscesses are almost invariably fatal, but recovery sometimes occurs in those following pneumonia or foreign bodies, after surgical treatment or a long process of spontaneous healing.

Treatment.—Medicinal treatment is practically useless. The quantity and the offensive character of the expectorated pus are sometimes mark-

edly diminished after the administration of calcium sulphid, gr. j (0.06) t. i. d., but when the abscess can be reached the proper treatment is incision and the establishment of drainage.

GANGRENE OF THE LUNG.

Definition.—A localized or diffuse putrefactive necrosis affecting a greater or less portion of the lung.

Etiology.—Gangrene is always a secondary affection; it does not attack previously healthy lung tissue. It arises from the entrance of the bacteria of putrefaction into a tissue already necrotic. It is a common sequence of: (*a*) Aspiration pneumonia; occasionally of (*b*) lobar pneumonia in a previously debilitated subject; more commonly of (*c*) bronchopneumonia, (*d*) bronchiectasis or fetid bronchitis, (*e*) embolism or thrombosis of the pulmonary artery, especially when the embolus is derived from a gangrenous focus, or (*f*) cancer. It sometimes follows (*g*) perforating wounds, (*h*) rupture into a bronchus of an esophageal or other ulcer, (*i*) the perforation of an empyema or hydatid cyst, (*j*) the pressure of an aneurism or other tumor. (*k*) As a result of tuberculosis, it is infrequent only in comparision to the prevalence of the latter disease. (*l*) It sometimes develops during convalescence from fevers of long duration, when the exciting cause cannot be determined. After typhoid fever, it is generally due to the obstruction of a large branch of the pulmonary artery. The disease is most likely to affect elderly persons debilitated by chronic wasting disease, especially diabetes, or by alcoholism, but it sometimes occurs in the young.

Morbid Anatomy.—(*a*) The diffuse form is rare, being occasionally met with after lobar pneumonia, or after the plugging of a large branch of the pulmonary artery. A greater part or the whole of one lung is converted into a dark blue or greenish black, extremely fetid, pultaceous mass, disintegrated at the center and not definitely separated from the surrounding tissue. (*b*) In the circumscribed form, the necrotic tissue is more clearly defined; a distinct sphacelus is sometimes formed. The surrounding tissue is deeply congested, often solidified, and beyond this area the lung is edematous. The original embolus can sometimes be found, and it is not unusual to discover a rupture of a blood-vessel or perforation of the pleura in cases dying from hemorrhage. Bronchitis is a constant accompaniment. Abscesses are not infrequently found also in the brain, liver, or spleen.

Symptoms.—The development of putrefaction upon a previous pulmonary disease is promptly announced by the extremely fetid expectoration. The condition is occasionally encountered post mortem, however, in cases which gave no indication of it during life. The sputum is usually profuse, thin, greenish, containing mucus, pus, elastic tissue, fat-crystals, granular débris, bacteria of putrefaction, and sometimes altered blood. After standing, the sputum separates into three layers, the solid matter sinking to the bottom; a middle greenish fluid, and a supernatant brownish froth. Fragments of necrotic lung tissue are sometimes expectorated, and, when a blood-vessel has been eroded, a profuse hemorrhage occurs. Moderate fever is always present. The patient becomes rapidly emaciated and anemic, but does not, as a rule,

have much pain. The cough is almost constant, sometimes strong and ineffectual, interfering with sleep. Sepsis generally develops, with repeated chills, increased fever, sweats, and delirium. The physical signs are not always distinctive. A severe bronchitis is invariably present, as indicated by the cough, and, when there has been great destruction of the lung, the signs of a cavity may be elicited by percussion and auscultation.

The ***prognosis*** depends upon the previous condition of the patient and the cause and character of the gangrene. Recovery sometimes occurs in a young, previously healthy person after the case has long appeared hopeless, and after extensive destruction of lung tissue, but in the debilitated subject of diabetes or other constitutional disease a fatal result is inevitable.

Treatment.—The patient must be immediately isolated in a well-ventilated apartment, the air of which should be charged with the vapor of carbolic acid, guaiacol, turpentine, or formaldehyd; or one of these disinfectants may be dropped upon a respirator worn by the patient. The strength of the patient must receive especial attention. The food should be liquid and of the most nutritious quality. Whisky and strychnin should be administered freely. The advisability of surgical measures should be considered early when the gangrenous cavity is near the surface. Antiseptic solutions may be injected directly into it, or the cavity may be opened and drained as an abscess, providing the condition of the patient will permit.

NEOPLASMS OF THE LUNG.

1. Benign neoplasms are rare. Fibromata, myxomata, enchondromata, osteomata, and adenomata have been encountered. Dermoid cysts have occasionally been met with.

2. Malignant growths may be either primary or secondary. The primary are extremely rare. Secondary carcinoma, epithelioma, or sarcoma is more common. Cancer reaches the lung by direct growth through the chest-wall, or through the lymph-channels from a primary source in the breast, esophagus, stomach, or liver. It usually appears in the form of multiple small nodules in the pleura of one or both lungs, which increase in size and extend deeper, sometimes giving rise to bronchopneumonia, suppuration, or gangrene. Secondary sarcoma reaches the lung by direct extension from the ribs or other adjacent tissues, or through the blood-vessels from remote parts of the body. It constitutes one of the most frequent locations of secondary growths. When a direct extension, the growth is usually single; when metastatic, there may be a large number of nodules in both lungs. Pleurisy, either malignant, serous, fibrinous, or hemorrhagic, is an almost constant accompaniment of either form of malignant disease. Sarcoma occurs most frequently in early and middle life, but cancer is infrequent under the age of 40. The secondary form is much more commonly met with in women.

Symptoms.—The clinical manifestations of malignant disease are indefinite. In many cases the disease exists for some time without producing recognizable disturbance. Dyspnea is often one of the first

indications of it. Cough is usually present, and it may be painful and ineffectual. There is sometimes a brownish, "prune-juice" expectoration that is regarded as highly diagnostic by some writers. Pain is usually an indication of involvement of the pleura. With the growth of the tumor, the blood-vessels are sometimes compressed in such a way as to induce turgescence of one or both arms and lividity of the face and neck. The heart may be displaced toward the opposite side, and the pneumogastric and recurrent laryngeal nerves are sometimes pressed upon. The dyspnea becomes extreme when the trachea or bronchi are compressed or their walls invaded by the growth. The subclavicular, axillary, and cervical lymph-glands are often enlarged. Auscultation and percussion give little information as to the character of the affection and are of value chiefly after a source of probable metastatic infection has been determined. The development of cachexia is one of the most valuable factors in diagnosis. The duration of the disease after its recognition is generally brief. Death has occurred as early as one or two months after involvement of the lung, and it is seldom delayed longer than six or eight months.

The ***prognosis*** is necessarily fatal.

The ***treatment*** is palliative, directed to the relief of pain and the support of strength. Morphin should not be withheld.

PARASITIC DISEASES OF THE LUNG.

Echinococcus of the lung is considered on page 286.

Actinomycosis of the Lung.—A disease caused by the growth within the lung of the actinomycosis, or ray fungus. This may result either from inhalation of the fungus, or from direct extension of the disease from the jaw and neck. Three more or less distinct forms are recognized, as noted on page 235, one affecting particularly the bronchi another producing bronchopneumonia, and a third resembling tuberculosis. The inflammatory process that is set up is usually modified by the accompanying pyogenic bacteria. With manifestations like those of an intense bronchitis or tuberculosis, the fibrous tissue of the lungs and pleura is proliferated, and pus-pockets are often formed within the new tissue. As the disease progresses, erosion of the ribs often occurs and the skin may finally be perforated. In some cases the perforation occurs through the diaphragm, and the pus burrows into the liver or other abdominal organ, or it may pass down the psoas or iliacus muscle. The disease was no doubt confounded with tuberculosis until within the past few years. Septic manifestations are common, and a fatal pyemia is the usual termination. The extension of the disease outward and the perforation of the skin reveal the diagnosis, since the fungus can be found in the discharges. This also distinguishes the disease from a syphilitic or tuberculous abscess with fistulous opening. The disease may last for months or years, during which time metastatic infections are liable to occur, with the production of abscesses in the abdominal organs, heart, and brain. The patient finally succumbs to exhaustion.

Treatment.—The treatment which promises most is the administra-

tion of large doses of potassium iodid. Arsenic or iron may be advantageously combined with it. The declining strength of the patient calls for the most nutritious food and the free administration of alcohol or strychnin.

DISEASES OF THE PLEURA.

ACUTE PLEURISY.

Pleurisy is classified: (*a*) Etiologically as primary or secondary; (*b*) anatomically, as plastic or adhesive (dry), and pleurisy with effusion; (*c*) in its course, as acute and chronic; (*d*) in the character of the exudate, as fibrinous, serofibrinous, purulent, and hemorrhagic, to which is sometimes added the so-called chylous pleurisy. (*e*) In addition to these, such terms as diaphragmatic, encysted, interlobular, and tubercular are often employed to describe differences in the location or origin of the process.

Fibrinous Pleurisy.—*Etiology.*—The disease may be primary or secondary in origin. (1) *Primary pleurisy* is often attributed to cold, but such micro-organisms as the bacillus tuberculosis, pneumococcus, and streptococci are often found, and are then looked upon as the exciting causes of the inflammation.

(2) *Secondary pleurisy* generally occurs in connection with acute inflammatory affections of the lung. It is constantly present in acute pneumonia and it is generally associated with abscess, gangrene, hemorrhagic infarction, and tuberculosis. It sometimes results from extension of inflammation from the pericardium or other adjacent structures. It is generally encountered in penetrating wounds, malignant or other disease of the wall of the thorax, and caries of the vertebræ.

Morbid Anatomy.—The lesions are generally unilateral. The pleura is at first hyperemic and edematous. The surface is opaque and dry, and a layer of fibrin of variable thickness, uniform and smooth, or with a granular surface, or superimposed layers of fibrin, are formed upon the surface. Within the meshes of fibrin are leucocytes and often a few red blood-corpuscles; a small quantity of serum is also exuded. Beginning on either surface, more commonly on the pulmonary, the process usually extends to the opposing layer. In the course of a few days, as a rule, the exudate becomes absorbed and adhesions are generally formed between the two layers of the pleura. These are often permanent, particularly when the apex has been the seat of the disease.

Symptoms.—The disease often begins with a sharp stitch in the side, which is aggravated by deep breathing and coughing. The pain is generally referred to the lower portion of the chest, but it may be confined to the apex region. A dry cough is usually present. In the more severe cases there may be an initial chill, with slight elevation of temperature. Tenderness is sometimes elicited by pressure over the affected region. The only physical sign belonging to the disease is a dry friction sound, a rubbing to and fro with the movements of inspiration and expiration, which gives the impression of being immediately under the ear. It is usually compared to the creaking of new leather. Sometimes there is a fine dry, crackling sound that can hardly be distinguished from the crepitant râle of lobar pneumonia, but it is not

always confined to the end of inspiration, as in that disease. Many other râles are sometimes heard as a result of associated inflammatory conditions in the lung. The disease often occurs at longer or shorter intervals, especially when it is tuberculous in origin.

Serofibrinous Pleurisy.—This is by far the most frequent form of the disease; it is the form commonly known as pleurisy with effusion.

Etiology.—The causes are the same as those of fibrinous pleurisy. Bacteria are believed to play even a more important part in its production, and a great many cases are tuberculous. The disease sometimes occurs as a terminal affection in hepatic cirrhosis, chronic nephritis, and cancer, but it has been repeatedly found to be tubercular in these cases, and a dropsical effusion is more commonly formed. Some writers have gone so far as to attribute all cases of primary pleurisy with effuson to tubercular infection. But the pneumococcus and streptococcus are without doubt the exciting factors in some cases, and the typhoid bacillus, Friedlander's bacillus, and the diphtheria bacillus have been found in some instances. The tubercular exudate is generally sterile, but that from the streptococcus or pneumococcus is prone to become purulent.

Morbid Anatomy.—The membrane is inflamed and covered with a layer of fibrin, as in the adhesive form of the disease, but the serous exudate is much more profuse, so that an accumulation amounting to from one to four quarts (liters) is found in the pleural cavity. This is usually a clear or slightly turbid, straw-colored serum, containing flocculi of fibrin, which sometimes settle to the dependent part of the sac. It has a specific gravity of 1.010 to 1.015, and faintly alkaline reaction. It is highly albuminous and usually contains, in addition to fibrin, a great number of leucocytes and degenerated epithelial cells, sometimes a few red blood-corpuscles. It rarely coagulates spontaneously. Sugar, uric acid, and cholesterin are sometimes found in it. The lung of the affected side is compressed to the extent required to accommodate the fluid. In extreme cases the entire lung is pushed back against the upper posterior wall of the thorax and completely collapsed. In such cases the mediastinum with the heart is pushed over a variable distance to the opposite side. When the left side is greatly distended with fluid, the apex of the heart may reach the middle line of the sternum.

Symptoms.—The disease sometimes develops abruptly with a chill, severe pain in the side, elevation of temperature, and a dry cough, suggesting acute pneumonia, but, as a rule, it is less severe in all its features. A great many cases, on the other hand, begin so insidiously, with little disturbance beyond a gradually increasing dyspnea, that the condition is not recognized until a large quantity of serum has accumulated. Dyspnea is usually an early symptom. It is at first due to the pain and in a measure to the fever, but later to the compression of the lung by the fluid accumulation. When, however, the accumulation forms very slowly, there is often but little evidence of dyspnea except on exertion. The pain, at first sharp and severe, is generally referred to the affected area, but sometimes to the back or abdomen. The fever seldom exceeds 103°F. (39.5°C.) and it is often intermittent in character. It may terminate at the end of a week, or it may persist for several weeks. The surface temperature is higher on the affected side.

Physical Signs.—*Inspection.*—The respiratory movements of the affected side are restricted to a degree that corresponds to the quantity of effusion. When this is extreme, it even causes that side of the chest to appear as if it were in a constant state of inspiratory expansion. The diameter is greater than that of the unaffected side, but the difference is less than it appears. The intercostal furrows are obliterated and may rarely become slightly prominent. The cardiac impulse is obliterated or displaced. In a right-sized effusion, the apex-beat may be displaced beyond the left nipple, even into the axilla and raised to the level of the fourth interspace. In left-sided effusion, the apex beat is generally concealed behind the sternum. The pulsation of the right side of the heart may be seen at the right of the sternum and perhaps as high as the third or fourth interspace.

Palpation.—The affected side is almost immobile, showing little or no expansion during full inspiration. The interspaces feel prominent. The tactile fremitus is diminished when the accumulation is moderate, and obliterated when it is excessive.

Mensuration.—Differential measurements show a difference of from a half-inch to more than an inch (1—2.5 mm.) between the two sides when at rest. The measurements may be about equal in full expansion.

Percussion.—The percussion note over the fluid is flat; over the compressed lung it is tympanitic, the tone fading away into complete flatness as the lung becomes more and more compressed. Several points should be carefully studied in this connection: (1) Percussion over fluid gives a very different sensation to the fingers than that obtained by percussion over solidified lung tissue.

(2) The upper boundary of the pleuritic fluid, when the patient is in the erect posture, does not follow a horizontal line, but a curve known as the Ellis or S-line of flatness. When the quantity of fluid is moderate, the lowest point is behind, near the spine. From that point it advances upward and forward in an S-curve to the axillary region, and thence declines in a straight line to the sternum. When the quantity of fluid is extreme, the upper margin behind is concave; it may reach the clavicle in front and extend beyond the sternum of the healthy side. In order to determine with exactness the upper boundary of the fluid, it is necessary to percuss with a light, quick stroke.

(3) A peculiar tympanitic note, known as Skoda's resonance, is often heard on percussion in the infraclavicular region, and sometimes in the back just above the upper margin of the fluid.

(4) The upper margin of the fluid in cases of moderate accumulation is found to change when the position of the patient is changed. If the upper margin of the fluid be marked in the axillary region when the patient is in the erect posture, the previously dull area will be found to be resonant when he lies upon the unaffected side.

Auscultation.—In the beginning, when the exudation is but slight, a friction sound is heard with the respiratory movements, a dry crackling, as in the fibrinous form of the disease; but as soon as the pleural surfaces have been separated by the fluid accumulation, the friction disappears. When the accumulation is sufficient to compress the lung, the respiratory sounds become less distinct and apparently distant. With the filling of the chest, the sounds undergo many changes; sometimes

there is tubular breathing with distinct inspiration and expiration, sometimes only a short, puffing expiratory sound; sometimes there is a metallic quality like the amphoric breathing heard over a cavity. Numerous râles may be heard as adventitious sounds, when tuberculosis, bronchitis, or other disease is associated with the pleurisy. The vocal sounds are also modified, absent, or intensified. Much depends upon the quantity of fluid, the position of the patient, and the presence or absence of adhesions. Bronchophony is not infrequently heard, and occasionally there is a more or less typical egophony. Baccelli affirms that the whispered voice can be heard through a serous effusion, but not through a purulent one, but exceptions to this rule have been repeatedly noted. The heart-sounds may be normal, but a systolic murmur is sometimes heard over a displaced heart, and a pleuropericardial friction sound is not unusual.

As the fluid undergoes resorption, the respiratory sounds return. When the roughened pleural surfaces again come into contact, there is usually produced a friction sound not unlike that of the inflammatory stage, sometimes a creaking or crackling, sometimes much like fine râles, but generally described as a redux friction. These sounds often persist for months, or recur at intervals even for years, especially when there is a tubercular background. The heart-sounds also return to their normal position with the disappearance of the fluid.

The duration and course of an acute serofibrinous pleursy are very indefinite. As in the adhesive form, all evidence of disease may subside within a week or ten days; a moderate exudate is sometimes absorbed within two or three days. When, however, the fluid has become excessive, compressing the lung, it is more apt to prove persistent, and in the tubercular form it often undergoes little change in quantity for many months. While the natural tendency of the serofibrinous exudate is toward absorption, there is always a liability to the development of suppuration. Spontaneous evacuation through the lung or chest-wall occasionally occurs, but less frequently than in the suppurative form. Sudden death has occurred in cases of long standing, usually in syncope, following some slight exertion or a sudden change of position. The exact cause of the accident cannot always be determined, but it has been attributed, as a rule, to embolism or thrombosis of the heart or pulmonary artery, or to a supposed twist of the great vessels.

PURULENT PLEURISY.

EMPYEMA.

Etiology.—(*a*) In a majority of instances purulent pleurisy follows the serofibrinous form of the disease, but it is often primarily purulent in children. Although it is probably of bacterial origin in most cases, the pus is often found to be sterile, and no satisfactory explanation of its occurrence can be given at the time. (*b*) It often develops after the acute infections, particularly scarlet fever, pyemia, sometimes after dysentery, and it may be apparently purulent from the beginning. The same is true of its occurrence with typhoid fever, but it occurs less frequently in that connection. An important relation often exists between

the disease and pneumonia, the purulent accumulation developing either during the pneumonic attack or in convalescence. Aspiration, done with proper precautions for the prevention of septic infection, has probably no influence in the conversion of a serous into a purulent effusion. In tuberculous cases, however, the needle-wound may be sufficient to produce infection under the most careful supervision, the infectious matter being derived from within. (*c*) The rupture of a tubercular cavity in the lung, and the extension of malignant disease from the lung, esophagus, or thoracic wall, excites a purulent pleurisy in some cases. (*d*) The disease may be established also as a result of injury, a penetrating wound, or the fracture of a rib.

The micro-organisms most frequently found in the exudate are the streptococcus, staphylococcus, especially in pyemic cases; the pneumococcus, usually indicating a favorable termination; the micrococcus lanceolatus; and the tubercle bacillus. Most cases of sterile purulent exudation are tubercular. The leptothrix pulmonus has been found in putrid exudates, and psorosperms have been discovered in a few cases.

Morbid Anatomy.—The fluid found in the pleural cavity after death varies from a slightly turbid, seropurulent, flocculent liquid to a thick, creamy pus. In pneumococcous cases it is usually thick and creamy and has only a faint, sweetish odor. In cases associated with gangrene it may be more fluid and has an extremely fetid odor. The pleura is much thickened and is often eroded, sometimes perforated, in one or more places. The lung may be much compressed, as in a serofibrinous accumulation.

Symptoms.—A purulent pleurisy often develops so insidiously that it can be regarded as of rather long standing when discovered. Symptoms of sepsis sometimes precede its recognition; they are seldom entirely absent. Sometimes, on the other hand, the onset is abrupt. The transition from a serous to a purulent effusion is marked in some cases by a rigor with rapid and pronounced elevation of temperature and profound prostration, often accompanied with severe pain in the side, which is aggravated by deep breathing. Cough is generally present, but it is by no means constant. Dyspnea is more uniformly present, but it may also be comparatively slight or entirely absent, except as a result of exertion. The patient sometimes sinks into a typhoid state soon after the development of pus, and more certainly after the exudate has become putrid. The course of the disease in the more severe cases is marked chiefly by manifestations of sepsis, with repeated chills, irregular or intermittent fever, profuse sweating, and finally delirium. Leucocytosis is present and often reaches a high grade. Peptonuria is observed in most cases, and indicanuria is more or less constant.

Physical Signs.—All the signs characteristic of serofibrinous pleurisy are found in the purulent form of the disease. In addition to these, however, certain peculiarities may be noted. (*a*) The distention of the chest often reaches a more extreme degree, especially in children, and a bulging of the intercostal spaces is more frequently observed. (*b*) The heart becomes even more widely displaced, and the liver and spleen are more distinctly depressed. (*c*) The subcutaneous veins are often distended over the affected side, and the chest-wall may become edematous. (*d*) Fluctuation has been noted, but generally as a result of a

beginning process of spontaneous evacuation and only over the region of "pointing." (*e*) The vocal sounds, sometimes audible over a serous accumulation (Baccelli's sign), are not transmitted through the purulent exudate. (*f*) In some cases a peculiar pulsation can be detected which is synchronous with the heart-beats (pulsating pleurisy). It is due probably to nothing more than the forcible action of the heart and the weakened resistance of the chest-walls.

The natural tendency of an empyema is to become chronic and, ultimately, to a fatal termination. Spontaneous recovery has been observed, however, in a few instances, either through absorption of the fluid or after spontaneous evacuation through the lung or chest-wall. Evacuation through the lung, if too rapid, may terminate fatally, by suffocation, the large quantity of pus rapidly filling the lung. It is only in cases in which the pus seems to filter through an area of softened lung tissue that this accident is prevented. Spontaneous evacuation through the chest-wall (empyema necessitatis) usually occurs at some point in the anterior wall between the third and sixth interspaces. More than one opening may occur, and there is sometimes a fistulous tract of considerable length in the thoracic wall. Ultimate recovery is sometimes observed after a chronic discharge of many years' duration. There is always danger in these cases, however, from the possible production of amyloid disease. Perforation may occur also into the esophagus, stomach, pericardium, or peritoneum. Cases have been observed in which the pus passed down along the spine and psoas muscle to the iliac fossa, producing a condition resembling a psoas or lumbar abscess.

Special Forms of Pleurisy.—1. **Tubercular Pleurisy.**—Many writers regard all cases of pleurisy with effusion as of tubercular origin. The clinical manifestations are the same as have been described, and need not therefore be repeated. From the standpoint of tuberculosis the condition has been considered in the chapter on Tuberculosis.

2. **Hemorrhagic Pleurisy.**—This term has been applied to cases in which the serofibrinous exudate contains blood in sufficient quantity to give it a reddish color. The condition is distinct from that already described under the head of Hematothorax. Hemorrhagic pleurisy is encountered, for the most part, in: (*a*) Tubercular pleurisy, from the rupture of newly formed blood-vessels in the exudate; it may occur, however, in chronic tuberculosis; (*b*) in cancer of the pleura; (*c*) in asthenic conditions of the system, resulting from cancer, chronic nephritis, hepatic cirrhosis, and sometimes in the malignant types of infectious disease. (*d*) Cases of hemorrhagic pleurisy are sometimes met with in persons previously healthy and in whom its occurrence cannot be explained. It should not be forgotten that a clear serous effusion may become contaminated with blood as a result of the injury of a small vessel in aspiration.

3. **Encysted Pleurisy.**—The pleuritic effusion is sometimes circumscribed by adhesions to such an extent as to separate it from the general pleural cavity, or two or more pockets, loculi, may be formed. These may be separated or they may communicate through small openings. The pockets may be found in any part of the pleural cavity, as on the sides, or they may be confined to the diaphragmatic surface. The diagnosis is often difficult, but a friction sound is sometimes heard,

and the aspirator needle withdraws fluid from the area of dullness. The condition is more frequent in connection with a purulent than with a serous effusion.

4. **Diaphragmatic Pleurisy.**—This term is applied to cases in which the inflammation is limited, in part at least, to the diaphragmatic pleura. It may be adhesive or serofibrinous. The effusion is not great in quantity, as a rule. Pain is the most prominent feature; it is generally severe and confined to the affected region and limits the respiratory movements. It sometimes suggests angina pectoris, or it may be referred to the epigastrium. There is generally tenderness to pressure over the diaphragm, especially over its insertion to the tenth rib and extending from its anterior extremity to the sternum and xiphoid cartilage.

5. **Interlobular Pleurisy.**—Inflammation of the interlobular pleura usually accompanies pleurisy of other regions. It is more frequent on the right than on the left side, and most marked near the root of the lungs, between the upper and middle lobes. As a result of it, the surfaces are closely agglutinated, but in some instances a pocket is formed. The exudate may be serous or purulent, and tubercles are commonly to be found. Spontaneous evacuation of the encysted fluid sometimes occurs through perforation of a bronchus, and the resulting purulent expectoration may be the first symptom to indicate the presence of a suppurative process within the chest. In some cases, however, there is a history of previous attacks of pleurisy.

Diagnosis of Pleurisy.—An adhesive pleurisy is generally recognized without difficulty by its symptoms and physical signs. In pleurisy with extensive effusion, too, the diagnosis is generally simple. The presence of fluid and its character can be immediately demonstrated with the hypodermic needle. When, however, the quantity of fluid is moderate, many of the physical signs are sometimes wanting or they may be of such a character as to simulate other affections, especially lobar pneumonia, simple hydrothorax, or excessive pericardial effusion. Echinococcus cyst, subphrenic abscess, and tumors of the base of the lung may be excluded in some cases.

Pneumonia is generally characterized by a greater severity of the subjective manifestations; the sudden onset with chill, the high temperature, greater prostration, together with the physical signs, showing bronchial breathing, an increase rather than diminution of tactile and vocal fremitus. In pneumonia, too, the boundary of dullness is usually highest posteriorly, corresponding to the limit of the interlobular fissure, while in pleurisy it is lowest at the spine.

Hydrothorax is a dropsical accumulation of fluid within the chest. It is generally bilateral, is not attended with the characteristic stitch in the side, but develops insidiously, as a rule, in the course of a chronic disease of the heart or kidneys, or in hepatic cirrhosis, cancer, or other affections which lead to dropsy.

Pericardial Effusion.—It is only when the pericardial accumulation is enormous that it can be mistaken for a pleuritic effusion. There is a sense of distress rather than of pain in the precordial region, and the dyspnea is extreme. Percussion reveals a circumscribed area of flatness corresponding in outline to the distended pericardial sac. The heart-

sounds are not displaced to the right, but they are often nearly or quite inaudible, and the pulse is usually feeble.

Hydatid cyst, tumors of the liver, and upward displacement of the liver cause dullness simulating that of a right-sided pleuritic effusion, often reaching the fourth rib in front and embarrassing respiration. But in these conditions the upper margin of dullness does not follow the curving line of pleuritic effusion.

Abscess of the lung may lead to confusion when the aspirator needle has been thrust into the pus-sac. As a rule, however, the symptoms are so different, the sudden development of dyspnea with rapid breathing, and purulent expectoration, that empyema is not suggested.

Treatment.—1. *Pain.*—In the initial stage of pleurisy of either form, the pain may be so severe as to call for the hypodermic administration of morphin. It may, however, yield to the application of heat, cold, or counter-irritation. Much benefit is derived from the application of dry cups or leeches over the affected region; and the application of a band or adhesive strip around the chest tightly enough to restrict the respiratory movements is of great comfort to the patient. A mercurial or saline purge should generally be given at the onset.

2. *The Effusion.*—When a serous effusion has formed, it is well to confine the patient to bed for a few days and to favor the resorption of the fluid by limiting the ingestion of fluids and confining the diet to "dry" food, providing there be no fever. Saline cathartics should be given in concentrated form. Counter-irritation with mustard, turpentine, or iodin seems to hasten absorption in some cases. A hot vapor bath acts well, and free diaphoresis produced by pilocarpin is often followed by rapid absorption of a moderate effusion. Diuretics are sometimes of service.

Paracentesis.—Aspiration should be resorted to: (*a*) As soon as it becomes apparent that these methods of treatment will not prove effective, (*b*) whenever the accumulation of fluid is so rapid as to cause marked dyspnea or (*c*) when the effusion becomes considerable and persists for several days, although dyspnea be not a prominent symptom. The operation is so simple that there are practically no contraindications, for the fever often subsides after the withdrawal of the fluid. It must be performed, however, under the strictest measures of antisepsis, including disinfection of the skin and of the operator's hands with mercuric-chlorid solution and thorough cleansing and boiling of the needle. The instrument should always be tested and its action should be thoroughly understood before beginning the operation. The point selected on the left side is usually either the seventh intercostal space in the axillary line or the eighth immediately below the angle of the scapula, and on the right side the sixth interspace in the axillary line. The arm of the patient is brought forward and the hand allowed to rest on the opposite shoulder, to widen the intercostal spaces, and the needle is thrust in close to the upper margin of the rib in order to avoid the intercostal artery. The skin should be drawn up a little before inserting the needle, so that the wound will be drawn down over the rib after the needle is withdrawn. Ethyl chlorid may be employed to deaden the sensibility of the skin, but it is not usually required in adults, since it is not necessary to use a large needle. The fluid should be withdrawn

slowly. The quantity to be withdrawn depends to some extent upon the quantity of the effusion, the age and physical condition of the patient. Some writers advocate the withdrawal of all the fluid obtainable, while others limit it to a quart (liter) or even a pint (500 c.c.). The withdrawal of a small quantity sometimes stimulates the absorption of the remainder. The operation should generally be repeated at intervals of a few days, as long as the effusion continues to reaccumulate. Accidents rarely occur in paracentesis. A sharp pain is generally complained of after a variable quantity of fluid has been withdrawn, and it may persist until it becomes evident that the aspiration should be discontinued. A pneumothorax has been produced in a few instances, and a cutaneous emphysema is even more common. An albuminous expectoration has been observed after the tapping, and it has been accompanied with rapidly fatal dyspnea in a few instances. Syncope or slight faintness is not uncommon, and epileptic seizures have been observed in a few instances.

3. *Empyema.*—The treatment of empyema is strictly surgical. With the determination of the purulent character of the fluid, the utility of aspiration ceases, and an early surgical interference becomes imperative. The operation consists of making a free incision through the chest-wall, usually with exsection of a portion of one or more ribs, and the establishment of free drainage. Irrigation is sometimes necessary, especially when the pus is found to be fetid. The operation is so simple that it is applicable to all cases of empyema, regardless of the condition of the patient, and it affords the surest means of relief. Cases in which the pneumococcus is found, and cases affecting children, should not be excluded, for, although spontaneous recovery has been known to occur in them, complete recovery is more rapid and the subsequent expansion of the lung more certain after operation.

CHRONIC PLEURISY.

Chronic pleurisy is of two forms, distinguished by the presence or absence of effusion, and known as (*a*) the adhesive or dry, and (*b*) chronic pleurisy with effusion.

Etiology.—The disease may follow either form of acute pleurisy, but it often begins insidiously as a subacute or chronic process. The influences which favor its development are the same as those of acute pleurisy—cold, wet, tuberculosis, and other conditions producing moderate inflammation of the pleura.

Morbid Anatomy.—(*a*) *Chronic Adhesive Pleurisy.*—As in the acute form of the disease the lesions are usually confined to one side, but both pleuræ are sometimes affected. The changes are usually most pronounced near the base of the lung, unless they are tubercular in origin, when they are more commonly found at the apex and often on both sides. The pleura is much thickened and firm, but the apparent thickness is much increased, especially after pleurisy with effusion, by the superimposed layers of fibrin, which become organized and firmly adherent. It is often impossible to separate the layers, and the entire thickness often amounts to more than an inch. Contraction of this new connective tissue causes shrinking and deformity of the affected side of the

thorax in old cases. Calcareous degeneration sometimes occurs. The underlying lung may be found in a state of carnification when compressed by fluid, and bronchiectatic cavities are not infrequently found in it. In some cases a process of sclerosis extends from the thickened pleura to a variable distance into the substance of the lung, involving especially the interlobular connective tissue. Villous projections from the surface have also been observed.

(*b*) *Chronic Pleurisy with Effusion.*—The pleura is much thickened and, as a rule, covered with a variable thickness of more or less completely organized fibrin. In other respects the condition is much the same as that of the acute form of the disease. In many cases a greater or less number of pockets are found, as in encysted pleurisy. In purulent cases of long standing the pus may be thick and calcareous.

Symptoms.—In a majority of cases the patient suffers from periodical attacks of acute pleurisy, with stitch in the side or a sense of dragging in the lower part of the chest. The general health may be but little impaired, and many cases are recognized only through their objective signs. The expansion of the chest may be much restricted on the affected side, and the respiratory and vocal sounds diminished or suppressed. In other cases there are marked dullness and suppression of these sounds without much impairment of respiratory action. In cases of the adhesive form, after the fibrous tissue has contracted, a corresponding depression of the chest-wall is produced. The respiratory and vocal sounds are diminished and distant, on account of the thickening of the pleura. Dyspnœa is usually brought on by exertion, and the patient becomes weak and emaciated as the disease progresses.

Diagnosis.—The chronic adhesive form is often difficult of recognition. Its presence may be inferred, however, from the dullness, diminished vocal resonance, and particularly from the resistance offered by the thickened, indurated pleura to the passage of the aspirator needle. When effusion is present, it can be demonstrated by aspiration. The hypodermic needle is generally too short to penetrate beyond the thick layers of the pleura.

Prognosis.—The disease is not of itself fatal, but it renders the patient more liable to other pulmonary affections—bronchiectasis, bronchitis, and bronchopneumonia.

Treatment.—When the pain is severe, the case should be treated as one of acute pleurisy. The contraction of the chest in the later stages of the disease may be overcome to some extent by systematic exercise of the respiratory muscles and by so-called respiratory gymnastics, the patient practising full inspiration and forced expiration through a tube or other device for regulating the resistance. The general condition of the patient should be looked after. Tonics, nutritious food, and fresh air are indicated.

HYDROTHORAX.

Definition.—A noninflammatory accumulation of serous fluid in the pleural cavity.

Etiology.—The condition is always secondary to some other affection; it is a symptom, not a disease, and occurs for the most part in connection with general dropsy in the course of cardiac or renal disease or

affections causing a hydremic condition of the blood. The accumulation generally occurs in both sides, but it may be unilateral, and is often more copious in one cavity than in the other. It sometimes develops as a result of the pressure of intrathoracic or mediastinal neoplasms. As a terminal affection it is often met with in pulmonary diseases and others terminating in cardiac failure. The accumulation is often rapid, but seldom so extreme as that of serous pleurisy. There is no pain or fever, as a rule. The lung becomes compressed, and a corresponding dyspnea is produced.

The physical signs are the same as those of a serofibrinous pleurisy, but the heart is rarely displaced. The diagnosis is readily established by the introduction of the hypodermic needle. The prognosis is that of the causal condition.

Treatment.—It is only when the dyspnea becomes urgent that treatment must be directed to the hydrothorax. The fluid may be withdrawn by aspiration, but it rapidly reaccumulates. The treatment of the underlying condition is of more importance, embracing the various measures for the removal of dropsical effusions in cardiac and renal diseases.

PNEUMOTHORAX, HYDROPNEUMOTHORAX, PYOPNEUMOTHORAX.

Pneumothorax, air in the pleural cavity, is an exceedingly rare condition. Hydropneumothorax, in which a serous effusion occupies a part of the cavity, and pyopneumothorax, in which pus is present, are more frequently met with.

Etiology.—The condition is more frequent in adult males and is generally a result of: (*a*) Perforating wounds, whether accidental or surgical. It sometimes follows the exploratory puncture of a hypodermic needle and more certainly incisions for the evacuation of pus, or a spontaneous evacuation of an empyema, pulmonary abscess, or hydatid cyst through the chest-wall. (*b*) Perforation of the lung. This sometimes occurs in a healthy lung as a result of violent straining, as in pertussis, parturition, or defecation, or blows upon the chest; rarely without cause and with the lung at rest. In such cases the air is sometimes absorbed, but, as a rule, pleuritic inflammation and exudation are excited by it. In a majority of cases, however, the perforation of the lung is a result of disease, as the caseous softening and rupture of a tubercular focus, septic bronchopneumonia, or gangrene, rarely from the breaking down of a hemorrhagic infarct. The perforation may occur also from the pleural side in an old empyema. (*c*) Perforation sometimes occurs through the diaphragm in connection with gastric, esophageal, or intestinal ulceration, especially that due to malignant disease. In these conditions, intestinal gases are found instead of air. (*d*) The condition has resulted also from the passage of air down along the trachea after tracheotomy. (*e*) Gas is very rarely developed within a pleural exudate as a result of the action of bacteria, notably the bacillus aerogenes capsulatus, and a hydropneumothorax is developed.

The air enters, or, as some writers express it, is drawn into, the pleural cavity when a perforation occurs, on account of the elasticity of

lung, which causes it to collapse. This elasticity normally creates a condition of negative pressure, and the collapse continues until this pressure has been equalized by the entering air. Owing to a similar elasticity of the opposite lung and the abdominal viscera, the affected pleural sac becomes distended, the heart is displaced to the opposite side, and the diaphragm is depressed. Sometimes when the abnormal opening becomes closed, the internal pressure grows even greater than the atmospheric, and the displacements are correspondingly increased. The most pronounced displacements occur, however, when the opening is of a valvular form, which permits the entrance of air, but prevents its exit.

Morbid Anatomy.—Post-mortem examination reveals, in addition to the accumulated air and fluid and the resulting displacements and compression of the lung, a more or less pronounced hyperemia of the pleura. The abnormal opening is often discovered with difficulty owing to its small size.

Symptoms.—When the disease develops in connection with advanced pulmonary disease, and usually when it is the result of incision for the evacuation of an empyema, comparatively little disturbance is produced by the entrance of air into the pleural cavity. As soon as the internal pressure has become equal to the external after incision, the air passes in and out, producing a hissing sound, with the respiratory movements. Latent cases are sometimes discovered at autopsy. When, however, it develops after a sudden perforation of the lung or chest-wall, a sharp pain is generally complained of with intense dyspnea, cough, cyanosis, and rapid respiration, often amounting to 50 or more in the minute. The pulse becomes rapid and feeble, and the patient may sink into a collapse, with cold perspiration and subnormal temperature. Death occasionally results from shock. If the condition lasts for a few days, and particularly if pyothorax be also present, fever of a hectic type generally develops as a result of the pleuritic inflammation which is excited. The hand of the affected side has been found edematous in exceptional cases.

Physical Signs.—*Inspection.*—The affected side is greatly distended and immobile; and the opposite side is similarly, though less, affected.

Palpation.—The tactile fremitus is diminished over the upper, air-containing portion of the chest, and nearly or quite absent over the lower portion when fluid is present. The apex beat of the heart is indistinct and displaced toward the opposite side.

Percussion.—The resonance may be high-pitched, ringing, tympanitic in quality, amphoric, or of a low pitch approaching closely to dullness. Dullness has been noted, in fact, in some instances. The lower portion of the chest generally yields a flat note owing to the presence of fluid. This dullness changes, however, with the position of the patient, as in a case of moderate pleuritic effusion, and usually to a more marked degree. The cracked-pot sound can be obtained in some cases in which there is a large external opening.

Auscultation.—Over the compressed lung the respiratory sounds are feeble and distant or quite inaudible. Moist râles are often heard, and the metallic tinkle of Laennec is sometimes present. Succussion may sometimes be obtained by shaking the patient from side to side, and it is sometimes audible a short distance from the chest. The voice-sounds

are high-pitched and metallic, but usually faint. One of the most valuable signs is that known as the coin test, in which a metallic sound produced by striking a coin placed upon the anterior chest-wall with another coin is transmitted with increased intensity to the ear placed upon the posterior wall.

Diagnosis.—Pneumothorax is readily recognized by its physical signs, especially when succussion can be obtained. When, however, the percussion note is dull, the condition is easily mistaken for one of ordinary effusion. Error may arise also in the presence of a dilated stomach, diaphragmatic hernia, subphrenic abscess containing air; and the encapsulated form must be distinguished from a large lung cavity.

In *pleuritic effusion* tubular breathing is heard above the water-line, while in pneumothorax the respiratory sound is usually absent; succussion, the metallic tinkle, and the coin tests are absent.

In a greatly *dilated stomach*, succussion can be produced, and there is hyper-resonance of tympanitic quality, but this is confined to the abdomen and lower part of the thorax, and the respiratory and vocal signs are unaffected.

Diaphragmatic hernia is either congenital or a result of injury. In it the tympanitic note is confined to the lower zone of the chest. Borborygmi are generally heard.

Subphrenic abscess containing air is rare. It is confined to the right side and can be mistaken only for an encysted pyopneumothorax. Pulmonary symptoms are absent; the heart is not displaced; there is usually a history of preceding gastric or intestinal ulceration, and epigastric tenderness is found on palpation. The pus flows freely when the aspirator needle is inserted.

Prognosis.—The result depends greatly upon the cause of the condition. Previously healthy individuals often recover, but chronic tuberculous patients generally succumb within from a few days to two weeks.

Treatment.—The case is to be managed for the most part as one of pleurisy with effusion. When dyspnea develops, the fluid should be withdrawn by aspiration, or, if the accumulation be purulent, a permanent opening should be made for its evacuation. Respiratory gymnastics should be practiced when there is a chance for recovery.

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improper feeding, malnutrition, anemia, and the infectious diseases, particularly tuberculosis.

Symptoms.—Vesicles and ulcers occur, singly or in clusters of a dozen or more, and they may coalesce, especially when they accompany an infectious disease. The area is surrounded by a zone of catarrhal stomatitis. The ulcers are so sensitive and painful that the child often refuses to nurse and becomes restless and fretful. When the pharynx is involved, deglutition is painful. The secretion of the mouth is increased, and driveling is produced. The breath may be offensive, but not fetid. There are usually no constitutional symptoms beyond those of the accompanying disease.

Treatment.—Each ulcer should be touched with silver nitrate fused upon a probe, after a careful application of cocain (2 per cent). Application of a solution of borax in glycerin and water, potassium chlorate, or hydrogen peroxid is beneficial. The constitutional condition should be treated. The disease generally subsides promptly upon recovery from the causal affection.

The *aphthæ of Bednar* are small ulcers on the hard palate, caused by the irritation of the artificial nipple or injury inflicted by the nurse's fingers. They are not of serious import.

Parasitic Stomatitis (Thrush, Mycotic Stomatitis).—An affection caused by the growth of the saccharomyces albicans upon the surface of the mucous membrane of the mouth.

Etiology.—The mycelial filaments form a dense meshwork among the epithelial cells, thus producing pearly white patches that coalesce as they grow and form a membrane of variable extent. The fungus does not grow upon a healthy surface, but follows a catarrhal stomatitis, especially when this is a result of the acid fermentation of food. Uncleanliness of the nursing-bottle and nipple is a common source of infection. The disease often becomes epidemic in foundling asylums. Although more prevalent in infants, it is sometimes encountered in adults, particularly in the late stages of tuberculosis, cancer, diabetes, and other cachectic conditions.

Symptoms.—The fungus generally appears first on the tongue, from which it extends to the lips, cheeks, hard palate, and often to the tonsils and pharynx. It has been found also in the esophagus, stomach, cecum, in the respiratory passages, and in a few instances in the brain and blood-vessels. The mouth is abnormally dry. The distinctive feature of the condition is the fact that the membranous formation can be scraped off without difficulty, and leaves a surface that is intact or, at most, slightly eroded. There are no distinct ulcers, as in aphthous stomatitis.

Treatment.—The disease may be prevented by proper attention to the cleansing of the mouth and teeth. The constitutional treatment is more important than the local, for the growth often proves stubborn in the mouths of feeble or cachectic patients. The disease generally yields promptly, however, to proper feeding, with proper care of the mouth, the nipple of the mother, or the regular disinfection of the nursing-bottle and its nipple. The fungus should be removed as fast as it forms, and the surfaces bathed with an alkaline solution. Small doses of calomel are generally specific when the growth extends into the esophagus and stomach, and are doubtless capable of preventing such extension.

Ulcerative Stomatitis (Fetid or Putrid Sore Mouth).—This affection

is a destructive ulceration of the gums met with in children after the first dentition; occasionally, also, in adults. It is probably contagious and sometimes becomes epidemic in institutions for children or in camps and prisons.

Etiology.—Its origin is probably microbic, but the specific germ is not known. It is favored by improper food, dampness, faulty sanitation, sudden changes of temperature, but more particularly by lack of cleanliness in the care of the mouth and teeth. Attempts have been made to identify it with the foot-and-mouth disease of cattle and with contagious impetigo, but the relationship has not been demonstrated. Chronic poisoning with mercury, lead, arsenic, copper, phosphorus, and iodin, and the presence of catarrhal stomatitis, favor its development. It often accompanies or follows the acute infections, and is common in tuberculous, rachitic, or syphilitic children. An accumulation of tartar assists in its production.

Symptoms.—The disease begins at the margin of the gum, and extends in all directions often passing across to the contiguous surface of the lip or cheek. The gum is at first swollen, intensely red, and bleeds readily. It becomes everted and detached from the teeth. Ulcers soon form along the edges, covered with a grayish white, firmly adherent membrane. Owing to the destructive process, the teeth appear elongated and in severe cases they often become loosened. The periosteum may become involved, and a greater or less portion of the alveolar process may be detached; actual necrosis is unusual. An intense catarrhal stomatitis spreads over the tongue, lips, and cheeks, but ulcers are seldom formed. The saliva is enormously increased in quantity; the breath is rendered fetid by the necrotic tissue, and mastication is extremely painful. Fever, anemia, and emaciation develop rapidly, and death sometimes occurs in feeble children. In older children, however, there may be no constitutional reaction. The lymph-glands below the jaw are usually swollen.

Treatment.—Potassium chlorate is a specific in all cases. It should be given to a child in doses of gr. ij to x (0.13—0.65) three times daily in aqueous solution without sirup. The same solution may be employed as a wash, but it is painful in the early stage of ulceration. Potassium permanganate solution (1:500) diminishes the fetor. The application of silver nitrate promotes healing. The action of potassium chlorate on a young infant should be watched, and the remedy should be discontinued immediately upon evidence of renal irritation, the first symptom of which is usually albuminuria or persistent drowsiness.

Gangrenous Stomatitis (Noma, Cancrum Oris).—A rapidly progressing gangrenous process beginning on the inner surface of one cheek or on the gum, and resulting in extensive destruction of tissues.

Etiology.—The disease is probably due to a micro-organism, and several bacteria have been described as the specific cause. The diphtheria bacillus, or one identical with it, has been found by several investigators in a considerable number of successive cases. The disease ordinarily occurs between the ages of 2 and 15 and more frequently in girls. It usually follows an acute infection, more than half the cases having occurred after measles, some after diphtheria, and several after ulcerative stomatitis, but it may arise as a primary affection under extremely poor sanitary conditions.

Symptoms.—The affection generally begins as a small papule or vesicle on the mucous membrane of the cheek, and, before it is discovered, it has usually developed into a gangrenous ulcer which spreads with rapidity. In some cases it is preceded by a catarrhal stomatitis. The surrounding tissues become extremely hyperemic. The necrotic process frequently extends to the lips and gums, rarely involving also the tongue, and within 48 hours the cheek is often perforated by it. The vessels in the hyperemic zone become thrombotic, and the adjacent lymph-glands become large and soft. In extreme cases, unless death occurs early, the entire side of the face becomes involved, the entire cheek, the eye, nose, soft palate, and frontal bone; sometimes the ear and tissue back of it and the maxillæ are destroyed. Metastasis to the lungs may occur, with the production of fatal bronchopneumonia, infarction, abscess, or gangrene. The odor is extremely fetid and characteristic. Salivation is produced and fragments of necrotic tissue are discharged, or they may be swallowed and produce nausea and diarrhea. Albuminuria is commonly developed. The constitutional disturbances are severe. The child becomes prostrated at once, and the temperature rapidly rises to 104° F. (40° C.); the pulse is rapid and weak. The process is not usually attended with much pain. The mortality is fully 90 per cent; death occurs in 7 to 14 days, sometimes much earlier. Recovery is sometimes spontaneous or follows active treatment.

Treatment.—As soon as the primary lesion has been discovered, it should be thoroughly destroyed, under anesthesia, with the electric or Paquelin cautery or fuming nitric acid. The mouth should then be cleansed at least every two hours with a solution of hydrogen peroxid followed with a solution of potassium permanganate (1:500). The wound should be dressed with iodoform or other antiseptic gauze. Liquid nourishment and stimulants (whisky or brandy) must be administered freely and at short intervals, by the rectum if necessary. Strychnin may be administered hypodermically.

Membranous Stomatitis (Diphtheritic or Croupous Stomatitis).—This form of stomatitis is characterized by the formation of a false membrane. It may be caused by the Klebs-Löffler bacillus, or by the corrosive action of strong acids or alkalis, heat or cold, or gonorrheal or syphilitic infection. It is differentiated from mycotic stomatitis by the firmness with which the membrane adheres to the mucous membrane, especially when of true diphtheritic character. The treatment is that of diphtheria, except that the antitoxin is not used when the bacillus is absent.

Syphilitic Stomatitis. (See Symptoms of Syphilis, page 164.)

Mercurial Stomatitis (Ptyalism).—An inflammation of the mouth, tongue, and salivary glands, caused by mercury. A similar inflammation is occasionally produced by other drugs, as iodin and jaborandi.

Etiology.—The disease may be produced by any form of mercury introduced into the system in any manner whatever. It rarely occurs as a result of the treatment of syphilis, and it is now seldom seen except in persons abnormally susceptible to the action of the drug, or as a result of the continued use of calomel as a diuretic. It may, however, follow a single dose of but a few grains.

Symptoms.—The first manifestation is generally a metallic taste; then the gums become red, swollen, and painful (gingivitis), and the flow of

saliva is greatly increased. The tongue is sometimes affected. The breath is foul. Ulcerative stomatitis is sometimes produced. In extreme cases the teeth become loose or completely detached. Mastication is painful or impossible. The patient becomes pale, emaciated, and sometimes feverish. Albuminuria may be produced.

Treatment.—The administration of mercury should be immediately suspended. A solution of potassium chlorate should be used to rinse the mouth, and in severe cases it should be administered internally; or the permanganate may be used as a rinse and the chlorate internally. A saline purge should be given, and the patient should drink freely of water to promote diuresis. Sweating is beneficial. Atropin, gr. 1-100 (0.0006), diminishes the flow of saliva.

La Perleche.—An affection consisting of the formation of painful fissures at the angles of the mouth. The disease occurred epidemically among children in France in 1886 and was communicated through the drinking-cups. The name was suggested by the constant licking of the fissures. The treatment consists in the application of astringent solutions, especially alum and cupric sulphate.

Riga's Disease.—A grayish membrane forms over the frenum of the tongue, accompanied with much induration. Ulcers may form beneath it. It usually appears with the irruption of the first teeth. The affection occurred as an epidemic in Italy. The treatment is that of catarrhal stomatitis.

Ludwig's Angina (Cellulitis of the Neck).—An acute, suppurative inflammation beginning in the floor of the mouth and extending to the cellular tissues of the front of the neck. It is due to streptococcus infection, but occurs most frequently in a secondary relation to the infections, particularly scarlatina or diphtheria. It may originate in the adjacent glands, after trauma. Mastication, deglutition, and articulation become painful. Edema of the glottis may develop. Abscess or extensive sloughing is usual. Symptoms of septic infection are common. The treatment is surgical, consisting of the evacuation of the pus and removal of sloughs.

DISEASES OF THE TONGUE.

GLOSSITIS.

1. **Acute Glossitis.**—An acute inflammation of the tongue caused by burns, corrosives, injury by foreign bodies or carious teeth, or the sting of insects. It is not a common affection. The tongue rapidly becomes swollen, red, and painful, and may protrude from the mouth. The soft palate and epiglottis may also become edematous, and the sublingual glands are generally enlarged. Fever, headache, and languor are usual symptoms. Recovery occurs, as a rule, after a week. The treatment consists in the application of alkaline solutions. When the swelling is not too great, fragments of ice should be held in the mouth. A calomel purge is beneficial.

2. **Chronic Glossitis.**—This may result from repeated attacks of acute glossitis, or it may develop gradually as a result of the irritation of

tobacco, alcohol, or carious teeth. It often accompanies impaired digestion. The tongue becomes large, inflamed, and painful, and in places denuded of its papillæ. The treatment requires the removal of the cause and the application of a 2 per cent silver-nitrate solution. Tonics are generally indicated.

3. **Glossitis Desiccans.**—This is a chronic disease of unknown origin, in which the surface of the tongue becomes denuded, fissured, and extremely sensitive. Its appearance is ragged and uneven. The treatment is the same as that of chronic glossitis.

4. **Geographical Tongue** (Lingual Psoriasis, Eczema of the Tongue).—The tongue becomes denuded of its surface epithelium in patches which spread in all directions while the central portion heals. A narrow white line sometimes borders the patch and increases its resemblance to the outlines of a map. A sense of burning and itching is usually produced. The affection sometimes becomes chronic and often relapses after apparent cure. Its cause is not known; it occurs at any time of life. The treatment is that of chronic stomatitis.

5. **Leukoplakia Buccalis** (Smoker's Tongue, Buccal Psoriasis, Ichthyosis Linguæ).—A disease of unknown origin consisting of the formation of irregular, unsymmetrical, smooth white patches which do not ulcerate. The epithelial layer often becomes greatly thickened and the papillæ may be hypertrophied, producing warty prominences (lingual corns). It is often met with in excessive smokers, but may be independent of such irritation. It is probably not related to syphilis. It is painful and often persistent. The patches must generally be removed with the curette, cautery, or chromic acid. Ravitch recommends the applying of a 15 per cent solution of silver nitrate or 5 per cent chromic acid, to be followed by the negative galvanic current for 10 or 15 minutes each day. The indication for radical treatment is the liability of the papillomatous formation to become epitheliomatous.

6. **Macroglossia,** a congenital enlargement of the tongue due either to hypertrophy of muscle or the presence of a lymphangioma. The tongue may become deeply fissured and painful. A similar enlargement is sometimes associated with acromegaly, myxedema, and cretinism. The treatment of the congenital disease is surgical and consists in the removal of wedge-shaped pieces of the tongue.

7. **Hemiglossitis.**—This name is applied to a vesicular eruption that is sometimes seen on the side of the tongue and inner surface of the cheek, apparently bearing a relation to the terminal filaments of the trigeminal nerve and thought to be of the same nature as herpes zoster.

8. **Epithelioma of the Tongue.**—This presents a persistent, gradually enlarging ulceration, with indurated edges, along the sides or at the base of the tongue. Good results are obtained from treatment with the X-ray. This failing, the new growth must be excised.

DISEASES OF THE SALIVARY GLANDS.

1. **Supersecretion** (Ptyalism).—An excessive secretion of saliva, sometimes amounting to 5 quarts (liters) in 24 hours; the specific gravity may rise to 1.030 or higher. The causes are many. Nearly all the inflammatory conditions of the mouth are attended with salivation; it

may be produced by reflex irritation of the nerves governing the secretion. It occurs at times in connection with mental and nervous disease, hydrophobia, or the acute fevers. It has been observed during gestation and in connection with disease of the pancreas. It is more frequently, however, a result of mercurial poisoning; it may be induced also by iodin, copper, gold, silver, arsenic, or lead and by the vegetable drugs, jaborandi (pilocarpin), muscarin, and tobacco. The condition generally subsides upon removal of the cause; in nervous diseases it may prove persistent. The local treatment is that of mercurial stomatitis.

2. **Xerostomia** (Aptyalism, Dry Mouth).—An arrest of the salivary secretion. This disease is rare and is generally encountered in neurotic women. The tongue becomes dry and red, sometimes fissured; the mucous membrane is smooth and glazed; mastication, deglutition, and articulation become difficult, and the food may collect along the gums in a hardened mass. A similar dryness often occurs in diabetes. Pilocarpin may stimulate the secretion, but the galvanic current has proved more successful.

3. **Inflammation of the Salivary Glands.**

(*a*) *Specific Parotitis.* (See Mumps, page 144.)

(*b*) *Symptomatic Parotitis* (Secondary Parotitis, Parotid Bubo).—An acute swelling of the parotid gland, usually a result of septic infection reaching the gland through the duct or through the blood. It is seen especially in connection with or as a sequence of the infectious diseases, particularly typhoid fever, typhus, sometimes in pneumonia, tuberculosis, pyemia, or syphilis. It has been observed also in connection with abdominal, pelvic, or genitourinary disease or injury, and in a few cases it has been attributed to the influence of menstruation, pregnancy, or the introduction of a pessary. Gowers saw it in a case of fatal peripheral neuritis. The treatment consists in efforts to prevent suppuration through the application of ice, leeches, iodin, mercurial or guaiacol ointment. An incision should be made immediately upon the development of pus, and a poultice may then be applied.

(*c*) *Chronic Parotitis.*—A chronic enlargement of the parotid gland has been observed as a result of mumps, mercurial or lead intoxication, inflammation of the throat, and in the course of chronic nephritis.

(*d*) *Gaseous distention* of the gland and duct occurs in glassblowers and cornet-players; suppuration may be produced. The distention can sometimes be relieved by catheterization of the duct.

DISEASES OF THE PHARYNX.

1. **Circulatory Disturbances.**—(*a*) *Hyperemia* is commonly associated with acute and chronic diseases of the throat. It is a constant condition in smokers. Passive congestion is seen also as a result of obstruction of the circulation in the vena cava by aneurism, neoplasm, or valvular disease of the heart. It generally subsides upon removal of the cause. A 2 per cent solution of silver nitrate may be applied.

(*b*) *Anemia* is observed chiefly in connection with general anemic conditions, as after hemorrhage and in chlorosis.

(*c*) *Ulcers* sometimes result from chronic pharyngitis, but much more frequently from tuberculosis, syphilis, the general debility of long-stand-

ing disease, as lupus, cancer, or nephritis, or from septic infection contracted in hospital or the dissecting-room. The treatment consists in the improvement of the general condition and the application of silver nitrate.

(*d*) *Edema of the pharynx and uvula* occurs in quinsy, chronic nephritis, profound anemias, and other debilitated conditions. The enlargement of the uvula may interfere with deglutition and respiration, especially when there is congenital elongation. The serum may be evacuated by puncture or by snipping off the tip of the uvula when elongated. Hot gargles should be employed. Treatment of the general condition is even more important in many cases than local treatment.

(*e*) *Hemorrhage of the pharynx* is sometimes associated with that of other mucous membranes. The blood may be retained and form a hematoma. Vomiting of blood (hematemesis) may occur after a large quantity has been swallowed. The condition may be mistaken also for hemoptysis, and for this reason the pharynx should always be examined in a case of moderate spitting of blood. The blood may come from granulations in the nasopharynx, and can then be seen trickling down the posterior wall. It can generally be arrested by the application of astringents or the peroxid of hydrogen.

2. **Neuroses of the Pharnyx.**—These occur especially as a result of neurotic conditions, bulbar paralysis, hydrophobia, tetanus, or of varicosity of the veins of the throat, reflex irritation of an enlarged pharyngeal tonsil, or growths in the posterior nares. They are manifested for the most part as hyperesthesia, anesthesia, paresthesia (altered or unnatural sensation); less frequently as spasms, neuralgia or paralysis, all of which conditions interfere with deglutition and sometimes impair the sense of hearing. The treatment is usually that of the underlying condition.

3. **Acute Pharyngitis** (Sore Throat, Simple or Catarrhal Angina).—An acute inflammation of the pharynx, generally involving also the uvula and tonsils.

Etiology.—It may result from cold or the inhalation of hot or irritating vapors, but it is frequently a part of a general nasopharyngeal catarrh. It is doubtless due to the action of bacteria in some cases, especially when it is associated with the acute infections. Along with inflammation of the tonsils, it often precedes an attack of rheumatism or other acute disease. It may be induced by vomiting, and is thus associated with gastric catarrh.

Symptoms.—It may set in with a slight chill and fever, and a burning soreness of the throat. This is sometimes followed with stiffness of the neck, glandular enlargement, tinnitus, an irritable cough, and perhaps hoarseness, from extension to the larynx. Swallowing and speaking become difficult. The pharynx appears red and swollen and is usually coated with viscid mucus.

Treatment.—Gargling with hot milk or tea, sprays containing sodium bicarbonate or menthol, and lozenges containing alkalis afford great relief. The neck may be rubbed with volatile liniment or camphorated oil, and covered with flannel. A purge is often beneficial, and laxatives should be a routine treatment with most patients in order to ward off recurrences.

4. **Chronic Pharyngitis** (Chronic Ulcerative or Granular Pharyngitis, Chronic Angina, Clergyman's Sore Throat).—A chronic inflammation of the mucous membrane of the pharynx, involving to a variable extent the other structures of the throat.

Etiology.—The disease may follow repeated attacks of the acute form. In children it is associated with chronic hypertrophy of the tonsils and the presence of adenoids. It is induced by mouth-breathing, and it may be an extension of a catarrhal process from the nose. It is a common result of alcoholism and the excessive use of tobacco, but is often induced by voice-strain in lecturers, clergymen, and street-criers. It is probably more frequent in the rheumatic and gouty and in those predisposed to tuberculosis, as well as in the subjects of chronic diseases of the heart or lungs.

Symptoms.—There is usually a sense of dryness or of irritation of the throat, that is most severe in the morning, and accompanied with cough or hawking, huskiness of the voice, and pain upon swallowing. The secretion is often abundant, and the enlarged follicles can be distinctly seen as prominences on the wall of the pharynx, but later the follicles may shrivel and the pharynx become dry. A few dilated capillaries usually traverse the posterior wall. The mucous membrane appears relaxed and the uvula may be elongated and swollen. Chronic disease of the sphenoid occasionally produces a purulent discharge that flows down the wall of the pharynx.

Treatment.—Removal of the cause is important, and the general condition of the patient must be improved. The enlarged follicles should be touched separately with silver nitrate or the electric cautery. The use of astringent gargles, sprays, and pastilles is beneficial, but in most cases the condition proves extremely resistant to all treatment. Chronic atrophic pharyngitis results, the mucous membrane becoming shrunken and pale, and the patient is greatly annoyed by the formation of crusts. Partial deafness often results from involvement of the orifices of the Eustachian tube.

5. **Retropharyngeal Abscess.**—This affection may occur in healthy infants or young children as a result of septic infection which cannot always be accounted for, or it may follow the acute infections. As a result of caries of the bodies of the vertebræ it may assume a chronic or recurrent form. The swelling may be so great as to interfere with swallowing and even to threaten asphyxia. The sound of the voice is altered. The neck may become greatly swollen. The wall of the pharynx is intensely red, and the abscess can generally be felt with the finger.

The ***prognosis*** is grave, on account of the danger of suffocation, pneumonia from the aspiration of pus when rupture occurs during sleep, septicemia, edema of the glottis, or perforation of a blood-vessel, the trachea, or esophagus.

Treatment.—An early incision should be made when the abscess can be reached through the mouth. In some cases the pus burrows and must be evacuated through the neck.

6. **Acute Infectious Phlegmon.**—This name has been given to a rare, malignant inflammation which begins in the side of the pharynx and rapidly passes to suppuration. The pus burrows down the neck and may reach the mediastinum. The side of the neck becomes intensely

swollen, red, and tender, deglutition painful, and respiration may be obstructed. Severe constitutional symptoms of a septic character usually develop, and death may be a matter of but a few days. The treatment is purely surgical and consists in the evacuation of the pus when it can be reached.

DISEASES OF THE TONSILS.

ACUTE TONSILITIS.

CATARRHAL, FOLLICULAR, LACUNAR, OR ULCERATIVE TONSILITIS, OR AMYGDALITIS.

Definition.—An acute inflammation of the mucous membrane of the tonsils, accompanied with soreness of the throat and more or less systemic disturbance.

Etiology.—The disease is ordinarily a part of a general pharyngitis. It is more frequent in the spring and affects particularly children and young adults. It often appears to be infectious, attacking simultaneously several members of the same family, and it often recurs in the same individual at the same time each year. Cold and poor hygiene are doubtless influential in its production. Some writers regard it as related to rheumatism, for an attack of tonsilitis not infrequently precedes the onset of rheumatism. It is a common symptom of the acute exanthemata and may be associated with or follow an attack of indigestion.

Morbid Anatomy.—Several forms of the disease are recognized, the most important of which are: (*a*) The superficial, in which only the mucous membrane of the surface of the tonsils is involved, and (*b*) the lacunar or follicular, in which the mucous membrane of the crypts is also affected. The tonsils are enlarged and intensely hyperemic, and the follicles not infrequently become filled with a fetid, cheesy material composed of epithelium and micrococci. The exudation from several follicles sometimes blends into a uniform coating resembling the false membrane of diphtheria. Small vesicles like those of herpes have been observed on the tonsil in a few instances (herpetic tonsilitis); (*c*) a suppurative form, in which the tissues are more deeply involved and the inflammatory process rapidly goes on to suppuration.

Symptoms.—The affection may begin with or without constitutional manifestations. There is often a slight chill and fever, with pain in the back and limbs. The temperature often reaches 104° or 105° F. (40.0°–40.5° C.) in children. The throat is sore, and swallowing is painful. Fluids may be regurgitated into the nose on account of the swelling of the uvula. The tongue is furred, the tonsils much enlarged, and the crypts filled with a white or yellowish exudate. The voice is nasal, and articulation may be difficult and painful. The inflammation may extend to the middle ear and impair the hearing. The disease does not usually last longer than a week. Albuminuria is sometimes present, and endocarditis and pericarditis have been observed; but the discovery of a systolic apex murmur does not necessarily indicate an endocarditis in a feverish child. Paralysis does not follow an acute tonsilitis unless it is of diphtheritic character.

Diagnosis.—The disease is to be differentiated particularly from diphtheria. In the latter disease the membranous formation has a grayish

color, it is more uniformly spread over the surface of the tonsil, and is more firmly adherent, leaving a bleeding surface when forcibly removed, and it is not usually confined to the tonsils. Fever is not generally present at the beginning of the disease. The presence of the diphtheria bacillus establishes the diagnosis.

Treatment.—As a prophylactic measure the child should be isolated and confined to bed, at least until the presence of diphtheria has been excluded. The fever, headache, and joint or muscular pains are relieved by phenacetin or sodium salicylate. Aconite is highly recommended in the lacunar form. The diet should be liquid until the difficulty in swallowing subsides. Cold milk, egg-nog, and ice-cream are the most acceptable food. Hot gargles of sodium bicarbonate or borax with thymol, or astringents in glycerin and water, hot tea or milk, generally afford temporary relief. A folded flannel dipped in ice water should be applied to the neck at night and covered with oil-silk. In the suppurative form, hot applications are better. An incision should be made as soon as fluctuation can be detected. This is usually done with a curved bistoury guarded nearly to the point with a strip of adhesive plaster, the incision being made downward and parallel to the anterior pillars, avoiding the carotid region. Very rarely the tonsil reaches an extent that renders suffocation imminent before suppuration has occurred. The necessity of tracheotomy as the only means of saving life in such cases should be borne in mind.

CHRONIC TONSILITIS.

Chronic Nasopharyngeal Obstruction, Mouth-Breathing, Aprosexia.

Definition.—A chronic hypertrophy of the tonsils and of the pharyngeal adenoid tissue.

Etiology.—The condition generally begins about the third or fourth year, but may be congenital. It is a little more frequent in boys. An inherited predisposition is often apparent, and it is favored by bad hygiene and poor food. It often follows diphtheria, scarlet fever, or measles. Repeated attacks of acute tonsilitis produce permanent enlargement in some cases.

Morbid Anatomy.—The enlargement of the tonsils is due to an increase of all their constituents. In many cases the hypertrophy of the lymphoid tissue predominates, while in others, especially cases of long standing, the stroma is greatly increased and the glands become quite firm. The tonsils are usually about equally affected.

The enlargement of the adenoid tissue, the so-called pharyngeal tonsil, is sometimes of a papillomatous character in the more chronic cases.

Symptoms.—The most prominent symptom is obstruction of respiration, due, in great measure, to the presence of the adenoids. The disease develops gradually. The child becomes restless at night and sleeps with the head thrown back and the mouth open. The obstruction causes loud snoring, and in severe cases the child often awakes in a fright as though at the point of suffocation. Next the child acquires the habit of keeping the mouth open during the day, and the face becomes dull and expressionless, the voice nasal and indistinct, especially in the pronunciation of the sounds *b*, *p*, *m*, and *n*. The hearing often becomes defective. The

secretion of mucus is increased and the breath becomes foul. Small cheesy, foul-smelling masses from the crypts are often brought up by coughing or hawking. Taste and smell are also affected in many cases.

One of the most important results of the obstruction of the breathing is the production of deformities of the chest, especially the pigeon-breast, barrel-chest, and funnel-chest. While it is probable that the deformity is more readily produced in a rachitic child, it may be independent of the latter disease. The greatest prominence of the sternum is usually in the upper part. The barrel-chest is associated especially with asthma and emphysema. Among the more remote results are habit chorea of the face, dreams, enuresis, forgetfulness, and inaptitude for study. Headache is a common complaint, and the children are especially susceptible to cold and other forms of infection.

Diagnosis.—There is no difficulty in recognizing the condition in a well-marked case; examination of the throat reveals the enlargement of the tonsils. The adenoid vegetations may be seen through the throat mirror or they can be felt with the finger.

Treatment.—An attempt may be made to reduce the enlargement by the local application of astringents, as glycerite of tannin, or, better, the compound solution of iodin in glycerin, applied with a stiff brush, but in most cases it is better to remove the tonsils.

The treatment of the adenoids is of greater importance than that of the tonsils. Their removal can be readily accomplished under anesthesia, with the finger-nail, or, more esthetically, with a curette. The hemorrhage is usually slight, but, if persistent, yields to astringents or a spray of adrenalin solution (1:1000). It is sometimes necessary after the operation to apply a bandage or chin-strap at night, in order to overcome the habit of mouth-breathing. The child should have the benefit of fresh air, sunshine, and good food.

Enlargement of the Lingual Tonsils.—Enlargement of the so-called lingual tonsils, a group of closed follicles at the root of the tongue, produces the sensation of a foreign body lodged in the throat, causing repeated swallowing and hawking. It is usually associated with pharyngitis, and the treatment is the same.

DISEASES OF THE ESOPHAGUS.

ACUTE ESOPHAGITIS.

Etiology.—The inflammation may be (*a*) an extension of disease in the pharynx or in the stomach; (*b*) it may be produced by mechanical or chemical irritation, (*c*) the passage or lodgment of foreign bodies, or (*d*) the swallowing of corrosives. It frequently follows the infections, typhoid fever, diphtheria, smallpox, or pneumonia. In some instances the cause cannot be discovered.

Morbid Anatomy.—The inflammatory process may be confined to the mucous membrance or it may extend deeply into the underlying tissues. It may be simple or catarrhal, membranous or diphtheritic, suppurative or phlegmonous, or gangrenous. The epithelial coat is lost, the follicles enlarged; erosions are often present, and ulcers of considerable depth, rarely perforating the entire wall, are sometimes formed as a

result of corrosive poisons. The membranous form may be due to diphtheria or the thrush fungus, and an interesting form is described in which casts of a greater or less portion of the tube are ejected similar to those of the bronchi in fibrinous bronchitis.

Symptoms.—The chief symptom is pain upon swallowing, a burning sensation which may last for hours after the taking of food. A spasm is sometimes produced, with regurgitation of food. As the disease advances, pus and blood may be regurgitated. Thirst becomes urgent, and emaciation results from the inability to take nourishment. A more or less constant pain beneath the sternum is usually complained of. On the other hand, there may be extensive disease of the esophagus, with ulceration, unattended with any symptoms of prominence.

Treatment.—The diet must be entirely fluid, or rectal feeding may be employed. Bismuth subcarbonate, gr. xv (1.0), with sodium bicarbonate, gr. v (0.3), may be given, suspended in mucilage or placed dry upon the tongue. Small fragments of ice and, as the inflammation subsides, demulcent drinks may be given.

CHRONIC ESOPHAGITIS.

This form may result from the acute or it may be produced by chronic alcoholism, irritating food, the lodgment of a foreign body, stricture, varix, cancer, or other tumor. The mucous membrane becomes greatly thickened, ulcers develop, and polyps may form. There may be pain on swallowing, and regurgitation of food, sometimes coated with mucus. The treatment consists in the removal of the cause and the administration of the remedies for acute esophagitis.

Ulcer is usually encountered in connection with acute esophagitis, cancer, or other neoplasms. It has been seen also after typhoid fever and in peptic ulcer of the cardiac orifice of the stomach. The condition is rarely recognized during life. The treatment is the same as that of peptic ulcer.

STRICTURE OF THE ESOPHAGUS.

Etiology.—Stricture, or stenosis, is produced by organic changes in the wall of the tube, by abnormal internal conditions, or through pressure from without. Congenital narrowing has been observed. Cicatricial contraction results from corrosive, tubercular, syphilitic, diphtheritic, or smallpox ulceration, and peptic ulcer at the cardia. The lumen of the tube may be closed by a polyp, cancer, or other neoplastic growth. The chief sources of external pressure are tumors in the neck or mediastinum, enlarged lymph-glands, aneurism, and pericardial effusion.

Morbid Anatomy.—The stenosis may occur in any part of the tube, generally near the upper or lower extremity, and rarely involves its entire length. It is usually single. A diverticulum may be formed, or the muscular coat above the constriction becomes greatly hypertrophied and the tube dilated.

Symptoms.—A gradually increasing difficulty in swallowing is usually observed. The food seems to lodge on its way to the stomach, and for a time it must be assisted with a swallow of water. As the stenosis

becomes more complete, the food can no longer be forced down, and regurgitation occurs—immediately when the stricture is in the upper part, sometimes not for several hours when it is near the cardiac extremity. The ejected matter has an alkaline reaction, unless changed by the formation of fatty acids. It shows no indication of gastric digestion. Auscultation may be of service in locating the stricture, or the esophageal bougie or stomach-tube may be employed for this purpose. The use of the esophagoscope is a more recent method. The X-ray has been successfully employed also, after giving the patient a large dose of bismuth subnitrate in order to produce a shadow. The utmost care must be exercised in passing a bougie or tube in a case of long standing, for the wall of an aneurism or a cancerous mass may be punctured, even when the instrument is most skillfully used.

Treatment.—The patient must be nourished by rectal alimentation. Attempts may be made to dilate the stricture with graduated sounds, and in the cicatricial form this may sometimes prove successful. Electrolysis has been used with benefit. Esophagotomy or gastrostomy may be resorted to in extreme cases. No attempt should be made, as a rule, to dilate the carcinomatous stricture.

CANCER OF THE ESOPHAGUS.

Etiology.—The disease is generally primary, and it affects most frequently men between 40 and 60 years of age. It is more common in alcoholic subjects, and may follow injury by a foreign body or chronic gastritis from any cause. The type of cancer is generally the epithelioma.

Morbid Anatomy.—Some writers place the point of greatest frequency in the upper third, others at the lower extremity, of the tube; the growth is sometimes found at the point of crossing the left bronchus. An annular mass from one to two inches in length is often formed. It may involve only the mucous membrane or the entire thickness of the wall. Ulceration and perforation often result, with evacuation of the contents into the trachea, bronchus, mediastinum, or pericardium. Secondary growths develop in the neighboring lymph-glands.

Symptoms.—Gradually increasing dysphagia is complained of, and a complete stenosis finally develops. The food, when regurgitated, is often coated with bloody mucus after ulceration has occurred, or pure blood may be brought up. Fragments of the cancer are sometimes found. Periodical attacks of sharp pain are not uncommon. Laryngeal or bronchial cough is produced by pressure. In other cases there are no symptoms except gradual emaciation and the development of a cachexia.

Diagnosis.—The history of the case usually excludes spasm, stricture, and foreign bodies, for sudden occlusion does not occur. The emaciation, pain, and cachexia, in a man past middle life, with regurgitation of blood, should arouse suspicion of the disease. The tumor may be located by the careful passage of the stomach-tube.

Prognosis.—The disease is invariably fatal. Death occurs from asthenia or from perforation of the cancerous ulcer.

Treatment.—This is purely palliative in most cases. The patient can generally be nourished with liquids, milk, predigested beef, egg-nog,

broths, and gruels until a late stage has been reached, when rectal feeding must be resorted to. Pain requires the administration of morphin. The patient's life may often be prolonged by an early gastrostomy.

NEUROSES OF THE ESOPHAGUS.

Spasm of the Esophagus.—This affection is generally met with in neurotic individuals, in connection with hysteria, hypochondriasis, epilepsy, or chorea, and sometimes in hydrophobia. It has been observed during pregnancy and in connection with ovarian or uterine disease. It may follow a choking fit or such emotional excitement as anger or fright. It is sometimes a result of gastric irritation or a reflex influence from the respiratory passages, and it sometimes occurs in persons debilitated by neurasthenia, tuberculosis, or other chronic disease.

Symptoms.—The spasm generally develops suddenly. It may affect any part, but more usually the upper or lower extremity of the tube. The food is regurgitated, and for a time all efforts to overcome the obstruction are futile. Hiccough, pain, palpitation, and a sense of contriction generally accompany the attack. The spasm is sometimes caused only by certain articles of food, and fluids can generally be swallowed. The attacks recur at variable intervals, sometimes daily, sometimes not for weeks; and the duration of each attack is equally indefinite, sometimes lasting for only a few moments, sometimes for days at a time, until dilatation is finally produced. The bougie may pass without difficulty. The diagnosis is established by anesthetizing the patient, when the spasm will be found to have completely subsided. The affection is not usually serious, but fatal cases have been recorded.

Treatment.—The treatment is that of the causative condition. The passage of the bougie often cures a hysterical case. The valerianates, asafetida, and the bromids are usually administered. The patient should be persuaded to swallow food in the presence of the physician, in order to overcome his fear of spasm.

Globus Hystericus.—This name is given to the sensation of a lump rising in the throat and threatening suffocation. It is a hysterical manifestation and is generally accompanied with repeated efforts at swallowing, and dyspnea. It is promptly relieved by remedies which quiet the hysterical seizure, as the bromids and the valerianates.

Hyperesthesia occurs in the same class of nervous, hypochondriacal, or neurasthenic patients. It is indicated by a sense of burning or soreness in swallowing, without evidence of inflammation. It may precede or accompany spasm.

Anesthesia occurs either as a hysterical manifestation or in connection with paralysis. It is indicated, not so much by an absence of sensation, as by the slow passage of food.

Paralysis is a rare condition. It may be the result of central disease, as bulbar paralysis, or of peripheral neuritis from diphtheria toxemia or metallic poisoning. It may occur in a hysterical person. It has been attributed also to myositis and adhesions. The food is regurgitated, sometimes after long intervals. Swallowing provokes coughing, and there is always danger of the aspiration of particles of food into the lungs. The patient rapidly becomes emaciated. The treatment consists in sup-

plying nourishment through the stomach-tube, and the application of a weak faradic current, while the constitution is built up with bitter tonics, especially strychnin.

Dilatation.—This is almost never primary, but is usually a result of stricture, and affects the part of the esophagus immediately above the constriction. Hypertrophy of the wall accompanies the dilatation. The food is regurgitated, sometimes after a considerable quantity has been swallowed, and respiration may be interfered with.

Diverticula.—Saccular dilatations are of two kinds, those due to pressure (pulsion) and those due to traction. The former are usually found on the posterior wall at the junction of the pharynx and esophagus. A small dilatation is gradually increased by the pressure of food within it until a distinct pouch has been formed. Traction diverticula are generally situated on the anterior wall near the bifurcation of the trachea. They are generally produced by the contraction of cicatricial bands resulting from a previous inflammation of the lymph-glands.

Foreign Bodies.—Such foreign bodies as fishbones, pins, buttons, coins, pieces of bone, false teeth, and numerous other articles sometimes lodge in the esophagus. When of a harmless character the foreign body may be carried on into the stomach with such food as bread or potatoes swallowed in large mouthfuls. Or the patient may drink a glassful of milk, and 20 minutes later take an emetic. The foreign body may be dislodged by the coagulated milk. Another method is to have the patient swallow a mass of tangled thread. An hour or two later the foreign body can sometimes be withdrawn with the thread.

Rupture of the esophagus, when not a result of cancer or ulcer, is generally produced by violent vomiting. It is most frequently encountered in intoxicated persons. It is always fatal.

Varix.—The veins at the lower end of the esophagus sometimes become dilated as a result of hepatic cirrhosis or valvular disease of the heart; chronic esophagitis is induced, and a fatal hemorrhage often follows rupture of the vessels.

Hemorrhage occurs also in connection with ulcers and cancer or from the injury inflicted in the passage of a foreign body. Profuse hemorrhage follows the rupture of an aneurism into the esophagus. It sometimes occurs also in purpura and pernicious anemia.

DISEASES OF THE STOMACH.

The stomach is so situated that its cardiac orifice normally lies behind the seventh left costal cartilage, one inch from the sternum and four inches from the surface. The pyloric orifice is less than three inches to the right of the cardiac when the viscus is empty, but it lies behind the left lobe of the liver when distended. The fundus rises to the level of the fifth rib. The line of the greater curvature varies, but it seldom sinks below midway between the xiphoid cartilage and the umbilicus in a healthy person. The blood supply of the stomach is received from the three branches of the celiac axis, and the residual blood is returned to the splenic and superior mesenteric veins. The nerve supply consists of the terminal branches of the two pneumogastrics, conveying impulses from the central system, and of branches from the solar plexus of the sympathetic system. The normal capacity of the stomach is from 1,500 to 1,700 c. c. (3—3½ pints). The gastric secretion consists of the true secretion of the peptic glands and mucus from the columnar cells found on the surface and in the mouths of the glands. The secretion proper contains, in addition to inorganic salts, hydrochloric acid and two enzymes, pep-

sin, which acts upon proteids, and rennin, which has the power of coagulating the casein of milk. It is normally almost colorless, of acid reaction and characteristic odor. Its specific gravity is usually between 1.002 and 1.003. The acidity is chiefly, if not wholly, due to hydrochloric acid, and amounts to 0.2, rarely 0.3 per cent.

EXAMINATION OF THE STOMACH.

The examination of the stomach includes the application of the usual methods of physical diagnosis—inspection, palpation, percussion, and auscultation—and the chemical, microscopic, and bacteriologic examination of its contents. Many mechanical devices have recently been introduced which render it possible to illuminate the interior of the stomach, determine its size, and outline its form with considerable accuracy.

Inspection.—It is only when the stomach is distended that its outline becomes distinguishable by inspection. Peristaltic movements can sometimes be recognized, and they may be induced in some individuals by applying heat or cold to the abdominal wall or by tapping with the finger. The stomach may be artificially inflated with air or carbonic-acid gas. For this purpose, the stomach-tube is introduced, and air is slowly pumped in with a small hand-pump or a Politzer bag. Distention with gas may be effected by administering alternately small quantities of an acid and an alkaline solution, as the two parts of a Seidlitz powder, until the desired degree of distention has been obtained. The latter method must be used with caution, in order not to produce serious overdistention, and the stomach-tube should be at hand to relieve such a condition. Neither method should be employed in a case in which cancer or ulcer is suspected. The stomach may be illuminated by means of Einhorn's electric bulb introduced at the end of an esophageal sound, thus revealing its size and any inequality of its surface due to neoplasms. The X-ray may also be employed after filling the stomach with a solution of bismuth subnitrate.

Palpation.—This method is employed chiefly for the purpose of determining the presence of a tumor. The palm of the hand should be slowly but forcibly pressed upon the surface and, by having the patient relax the abdominal muscles, the neoplasm can often be felt, especially during expiration. A more certain method is to examine the patient in the knee-elbow position, with the thighs well drawn up. In many cases the size, location, outline, and firmness of the tumor may be determined, as well as the presence of tenderness, pulsation, or fluctuation.

Percussion is of value chiefly in determining the size of the stomach and its relations to other organs, particularly when it is inflated. The presence of food or fluid alters the result, especially along the lower margin. Tumors may also be recognized. The normal percussion note is tympanitic, of rather high pitch, with more of a metallic quality than that of the colon; but it differs with the degree of distention. Auscultatory percussion is often more accurate. In order to accurately determine the size of the organ, percussion should be practiced first with the stomach empty, then inflated with gas, and finally distended with water. It is well also to examine the patient both in the recumbent and in the standing posture.

Auscultation is of comparatively little value except in connection with percussion. Succussion may be produced by shaking the patient or by

his voluntary abdominal movements, but it affords only an indefinite idea of enlargement. The promptness with which fluid reaches the stomach may also be determined by auscultation (deglutition murmur). This should normally occur six seconds after the act of deglutition, but it may be indefinitely delayed when a tumor is present at the cardiac orifice.

Examination of the Stomach-Contents. (For methods see p. 719.)

ACUTE GASTRITIS.

SIMPLE GASTRITIS, GASTRIC CATARRH, ACUTE DYSPEPSIA.

Definition.—An acute inflammation of the mucous membrane of the stomach.

Etiology.—The disease is exceedingly frequent in all classes of people and at all ages, but especially in childhood. Many individuals are predisposed to it, and the "weak stomach" is not infrequently inherited. The idiosyncrasy may exist only toward certain articles of food. The gouty tendency, chronic valvular heart-lesions, and hepatic cirrhosis predispose to it. The same is true of many nervous affections, mental and physical fatigue and exhaustion, which favor its occurrence by impairing the gastric secretion. The disease is often present at the beginning of an acute infection. Insufficient clothing and bad hygienic surroundings favor it, especially in children.

The immediate cause in most instances is a local irritation, which is generally produced by errors in diet. The food may be too great in quantity, irritating or indigestible in quality, or taken at too short intervals. When the food is retained too long in the stomach, as when the quantity is too great, it is liable to undergo fermentation, with the production of lactic and fatty acids, and these may be the direct cause of the inflammation. Or the food, especially beef, fish, or milk, may have undergone partial decomposition before being ingested. Excessive indulgence in alcohol, very hot or very cold drinks, are also common causes.

Morbid Anatomy.—The mucous membrane becomes red, swollen, and in places eroded, and it is covered with mucus. The submucosa is edematous, and small hemorrhages may occur in it. The cells of the gastric tubules undergo cloudy swelling, and there is often a small-celled infiltration between the tubules. The inflammation is most marked in the pyloric region, and it may extend into the duodenum.

Symptoms.—The disease may occur as a mild, afebrile attack, or it may assume a severe, febrile form. In mild cases there is usually a sense of discomfort, with flatulent distention of the abdomen, followed by thirst, headache, depression, dizziness, eructations, nausea, and vomiting. The tongue is coated and there may be an increased flow of saliva. There is a disagreeable taste, and the breath becomes offensive. Temporary relief is afforded by vomiting. The vomited matter consists of the food last eaten, and it shows little change, although it may have remained in the stomach for several hours. In children, diarrhea, with colicky pains, commonly follows; in adults, constipation is more common. Recovery usually takes place in twenty-four hours.

The febrile form may set in with a chill and rise of temperature to

102° or 104° F. (39.0°—40.0° C.). The affection has been called gastric fever. Lebert described a special, infectious form which occurred epidemically. The tongue is broad, pale, and covered with a heavy coat. The breath is foul. Thirst becomes extreme, and the appetite is lost. The abdomen is distended, and there is often tenderness in the epigastrium. Vomiting is almost always present, and it may be persistent, even water being ejected. At first only food is brought up, then a bile-stained fluid containing much mucus. HCl is absent, and lactic and fatty acids are often abundant. Constipation is generally present in the beginning, but it may give place to diarrhea. An eruption of herpes occasionally appears on the lips. The urine is concentrated, the urates increased, and indican is sometimes present. The disease ordinarily subsides after from three to five days, but it may pass into a subacute or chronic form.

Diagnosis.—Mild cases are recognizable without difficulty. The severe attack may, however, prove to be an initial symptom of one of the infectious diseases, especially in young children. When there is high fever and much headache, the presence of meningitis is often suggested. Excessive pain may arouse suspicion of biliary colic, but the pain is not over the gall-bladder. The short duration of the attack or, on the other hand, the appearance of other symptoms soon removes all uncertainty. Typhoid fever may be excluded by the sudden onset and rapid rise of temperature, without epistaxis, bronchitis, or other prodromes.

Treatment.—Mild cases generally recover in a day without treatment. It is well, however, to administer a mild purge, calomel or castor oil, to remove decomposed food that may remain in the intestine. In severe cases the vomiting should not be too speedily arrested. If vomiting does not occur, it should be induced by the administration of warm salt water or ipecacuanha, or by apomorphin hypodermically administered. The patient should abstain from food as much as possible for a day or two. The eructation and nausea may be relieved with the aromatic spirit of ammonia, or sodium bicarbonate and bismuth, each gr. v (0.3), with a drop of oil of anise or peppermint in each powder. If a free diarrhea does not develop, a dose of calomel (gr. iij—v; 0.2—0.3) should be given in the evening, and a Seidlitz powder or calcined magnesia in the morning.

PHLEGMONOUS OR SUPPURATIVE GASTRITIS.

This is a rare form of gastritis in which a suppurative inflammation occurs in the submucous and muscular coats of the stomach.

Etiology.—Idiopathic cases have been observed, but, as a rule, the disease is a complication of one of the septic infections, notably puerperal sepsis or pyemia. The suppurative process may be diffused throughout the wall of the stomach, or it may be localized. In the former condition the pus is discharged either into the stomach or into the peritoneal cavity; in the latter, abscesses are formed.

Symptoms.—The case has the appearance of an extremely severe, acute gastritis, with the addition of septic manifestations. Frequent chills and hyperpyrexia are commonly present, with a rapid, feeble pulse, abdominal tenderness, and diarrhea. The diagnosis is seldom made during life, but the localized abscess can possibly be recognized by physical

examination, and in the diffused form pus may be found in the vomited matter. The patient generally passes into a delirium, or a coma from which he does not arouse, and death is an event of but a few days. The disease occasionally assumes a more chronic course.

Treatment.—Nothing can be done to arrest the disease. Morphin should be given at short intervals to relieve the suffering.

TOXIC GASTRITIS.

A severe inflammation of the stomach, often attended with great destruction of tissues, as a result of the ingestion of irritating substances, especially the corrosive acids and alkalis, or arsenic, antimony, phosphorus, alcohol, and other noncorrosive poisons.

Morbid Anatomy.—The corrosive poisons produce a charred appearance of the mucous membrane, with complete destruction of vitality, over a greater or less area. When the quantity of poison taken has been large, immediate perforation of the stomach-wall may be produced. If death do not occur within a few hours, an intense hyperemia develops around the necrotic areas, and hemorrhages and transudation of serum and round cells occur in the submucosa. Granulation may begin, yet perforation may occur after several days, producing a rapidly fatal general peritonitis.

The noncorrosive poisons produce hyperemia with hemorrhages and cellular infiltration into the submucosa, with fatty degeneration of the gland-cells. Healing is effected by cicatrization, and stricture or hour-glass contraction is occasionally produced as a late result. Atrophy of the stomach and ulcers are possible results.

Symptoms.—There is generally intense pain and burning in the epigastrium; it may extend from the mouth to the stomach, and there is constant vomiting or retching. Blood may be vomited, and later fragments of the slough may be ejected. The abdomen becomes distended, tender, and painful. The urine is scant and albuminous or bloody. Petechiæ sometimes appear in the skin. Collapse occurs in most cases immediately after the poison has been ingested; the pulse is then feeble and the surface is bathed in a cold sweat. Convulsions often supervene, and death may occur within a few hours, or only after several days. Recovery is possible when the quantity of poison has been moderate or when treatment has been promptly instituted. The diagnosis is not usually difficult, for the characteristic burns are generally to be found in the mouth, or the poison may be recognized in the vomited matter.

Treatment.—In poisoning with acids, magnesia should be immediately given with milk, egg-albumen, flour paste, or oil. In alkali-poisoning, lemon-juice, vinegar, a dilute mineral acid, or cream of tartar should be given. When the case is seen early, the stomach should be thoroughly irrigated. Morphin should then be administered for the pain.

Membranous Gastritis.—This is a rare form of gastric disease which has generally been met with in diphtheria, typhoid or typhus fever, variola, or pneumonia. A diffused or circumscribed membranous exudate is formed in which are found the Klebs-Löffler bacilli or the pyogenic micro-organisms. Portions of the false membrane are sometimes vomited; otherwise the condition is seldom recognized during life.

Mycotic and Parasitic Gastritis.—Fungi, especially the saccharomyces and sarcinæ, in one case that of favus; anthrax bacilli; trichinæ, and the larvæ of insects have been recognized as causes of acute gastritis in a few cases. Tuberculosis and syphilis rarely attack the stomach.

CHRONIC GASTRITIS.

CHRONIC GASTRIC CATARRH, CHRONIC OR FLATULENT DYSPEPSIA, ATROPHY OF THE STOMACH.

Definition.—A chronic inflammation of the stomach characterized by changes in the gastric juice, increased secretion of mucus, weakening of muscular power, and the symptoms of chronic dyspepsia (Pepper).

Etiology.—*Local Influences.*—In a majority of cases the disease can be traced to repeated attacks of acute indigestion or to a more or less constant irritation of the stomach by improper food. The food may be indigestible in character or it may be rendered so by improper cooking. Many cases are to be attributed to the constant use of fried food, hot bread, pie, confections, or too highly seasoned articles. Either the fat or the carbohydrates may be in too great quantity. Eating at unseasonable hours, or at too short or irregular intervals, and eating too rapidly, without proper mastication, are among the more frequent causes. The excessive use of coffee or tea or drinking ice-water during or after meals may gradually induce the disease. A chronic gastric catarrh almost always accompanies cancer, ulcer, and dilatation. It may be produced also by obstruction of the portal or general venous circulation as a result of cirrhosis of the liver, valvular disease of the heart, and chronic interstitial disease of the lungs, or the pressure of tumors or abscesses. Other local causes are the prolonged use of irritating drugs and nostrums, as the mineral acids, arsenic, or "bitters"; the drinking of liquors before meals, and the excessive use of tobacco. To what extent the disease may be caused by the adulteration of food has not yet been determined.

General Influences.—An important part in the production of the disease is often played by anemia, chlorosis, gout, tuberculosis, nephritis, diabetes, uterine disease, and many other affections.

Morbid Anatomy.—The lesions are generally studied under two heads, those of simple chronic gastritis, and those of an interstitial or sclerotic character; but the two forms usually represent an earlier and later stage in the same pathological process.

Simple Chronic Gastritis.—The stomach is usually much enlarged and all of its layers may be thickened. The mucous membrane is pale and covered with a heavy coating of tenacious mucus. The veins stand out prominently in cases associated with retarded circulation, and there may be ecchymoses or small hemorrhages into the mucosa or submucosa, especially in the pyloric region, where the disease is usually most pronounced. In a large class of cases, often described separately, the most pronounced feature is the large quantity of tenacious mucus which covers the mucous membrane, especially in the vicinity of the pylorus. The mucous membrane in this region also appears mammillated or wrinkled in many instances, on account of the increase of connective tissue and

beginning contraction. The condition has been described as gastritis polyposa, and by French writers as the état mamelonné.

On microscopic examination, the glands appear enlarged and saccular; sometimes they are converted into little cysts as a result of obstruction of their mouths with desquamated, degenerated epithelium. The gland-cells are usually granular from fatty degeneration, or they may be atrophied so that the principal and parietal cells can no longer be distinguished. The tubules are often widely separated by the new connective tissue. Ewald describes a condition in which the inflammation extends down to the base of the glands, and the cells are often in different stages of mucoid degeneration, but he was able to demonstrate these changes only in specimens which had been placed in alcohol while they were still warm.

Interstitial or Sclerotic Form.—This form is usually a late result of the catarrhal process, and its most distinctive feature is atrophy. The atrophy, however, may affect the stomach as a viscus or only its walls. In general atrophy the organ sometimes becomes so small that it will hold only an ounce or two of fluid, while its walls may be greatly thickened. It is often referred to as cirrhosis, or, better, sclerosis ventriculi. In the other condition, the stomach as a whole may be much dilated, but the walls become extremely thin. In the former, the wall of the stomach may be an inch thick; in the latter, it may be less than an eighth of an inch. In extreme cases the mucous membrane is often so atrophic that it is impossible to find a vestige of gland tissue in it, but cysts formed from the tubules may remain. The mucous surface is smooth and firm, almost cicatricial in appearance. The muscular coat may be hypertrophied, or it may be largely replaced by connective tissue. There may be associated with the lesions of the stomach a proliferative peritonitis, perihepatitis, and perisplenitis. Ascites is often present.

Symptoms.—Symptoms more or less closely conforming to those of acute gastritis usually occur at longer or shorter intervals in the chronic form, and there are often abnormal sensations in the throat, and motor phenomena which produce rumbling noises, eructations, and possibly vomiting. The ingestion of a small quantity of food produces a feeling of satiety, fullness, and pain, or even disgust and nausea; and violent eructations may continue for several hours afterward (flatulent dyspepsia). The gas may be odorless or highly offensive. At a later period distress is experienced when the stomach is empty as well as when it is full, and eating no longer produces satiety. When the stomach is greatly reduced in size, the food may be too rapidly carried into the intestine, and intestinal catarrh may be set up. The tongue is generally heavily coated, but the tip and edges may remain intensely red. A catarrhal or aphthous stomatitis is sometimes observed. Thirst often becomes most annoying, and the patient craves sour or highly seasoned and indigestible articles of food. The salivary and pharyngeal secretions are much increased, and these in turn aggravate the nausea. The so-called stomach cough, which is not infrequently present, is probably due to the catarrhal condition of the pharynx. Morning nausea and vomiting or retching are common symptoms, especially in old topers. Very often only bile-stained mucus is brought up. Vomiting may occur also with regularity either immediately after meals or several hours later; yet in some cases

it is absent, although the patient may desire it as a means of relief. Acid eructations (pyrosis) frequently accompany the belching. The acid fluid thus brought up may be in part retained in the lower end of the esophagus and cause pain in the region of the heart, known as cardialgia or heartburn.

Digestion becomes slow, and there is often little or no indication of digestion when vomiting occurs several hours after a meal or if the contents are withdrawn as late as six or seven hours after the ingestion of food. Abnormal fermentation is generally found to have set in, one result of which is a sour, disagreeable odor. Chemical examination reveals little or no HCl, but a greater or less quantity of lactic, butyric, and acetic acids. When the atrophy of the mucous membrane has become extreme, the hydrochloric acid, pepsin, and rennet ferment may all be absent. Absorption is also much delayed in most cases, so that potassium iodid does not appear in the saliva for a half-hour or longer after it is taken into the stomach, or twice the length of time ordinarily required. The abdomen often becomes much distended with gas, producing pain and dyspnea. Severe pain in the abdomen is not common, but more or less frequent attacks of colicky pains are experienced, and there is a more or less constant sense of uneasiness. Constipation usually prevails. Headache and vertigo are common, and the patient often becomes morose or melancholic. The pulse is generally small; it may be slow, irregular, or intermittent from the irregular action of the heart that is often present. The motor function of the stomach may not be disturbed.

In the extreme atrophic form, the symptoms are variable, and, from their severity, the rapid emaciation and anemia, carcinoma is often suspected, a diagnosis which is apparently supported by the discovery of a firm mass in the region of the pylorus. But the mass is due either to the hypertrophy of the muscular coat or to the lamellated condition of the mucous membrane, and the patient long outlives the limit of cancer. In some cases the features correspond more closely to those of pernicious anemia. The urine is usually of dark color, high specific gravity, and contains a large quantity of urates and phosphates and often calcium oxalate.

Diagnosis.—Ulcer, cancer, dilatation, and the neuroses must be excluded before a diagnosis of chronic gastritis can be arrived at. The first three of these affections are ordinarily accompanied with gastritis, however, and for this reason the presence or absence of cancer in particular cannot be determined in some cases until the patient has been under observation for several months and the effects of treatment have been observed. Cancer is characterized by a much more rapid progress, with emaciation, weakness, and cachexia, features which are seldom so pronounced in an independent case of chronic gastritis.

Treatment.—*Dietetic.*—In a case of moderate severity, before extreme atrophy has developed, it is often unnecessary to rigidly restrict the diet. It is sometimes sufficient to prohibit the use of certain articles. The patient generally knows better than the physician what kinds of food he can most easily digest, but he generally requires the positive direction of the physician to enable him to control his appetite. Persons present themselves, also, who have already reduced their dietary beyond reason, and these are often benefited by a more liberal allowance. In other

cases, a cure can often be effected by nothing more than proper regulation of the food. Advice must generally be given as to the necessity of eating at regular intervals, the thorough mastication of the food, and the limitation of its quantity. Hasty eating and overeating are the two main factors in the production of chronic dyspepsia in this country, and the third element in many cases is the habit of immediately rushing back to business after a hasty meal. Both mental and physical exertion should be postponed, if possible, for an hour after a full meal. Many dyspepsias are benefited by a change of the time of the principal meal to an hour at which it can be leisurely eaten and followed by an hour's rest. Idiosyncrasy must always be regarded, however, and the form of food which is theoretically best for the patient may prove less suitable than articles which should not agree. A vast deal depends also upon the proper cooking of the food, and the physician should be capable of giving instruction in this department of domestic economy. Articles fried in grease are generally unfit for a healthy stomach, much more so for one that is inflamed. In many instances it is necessary to give explicit written direction in regard to the food to be eaten, and that to be avoided. In other cases, doing this only converts despondency into despair by impressing the patient too strongly with the seriousness of his affliction.

Albuminoids generally agree best with the stomach. Those which are most easily assimilated should be chosen for a severe case. It is often advisable to begin the treatment by placing the patient on an absolute milk diet; but there are many individuals who cannot take milk. Sometimes the objectionable feature can be removed by the addition of lime-water, Vichy or other alkaline carbonated water, and a pinch of salt. From two to two and a half quarts of milk should be consumed daily, but it should be taken in quantities of a half-pint or less every two or three hours. Skimmed milk agrees better than whole milk in some cases, and some patients can drink buttermilk who cannot take sweet milk, but they usually tire of it more readily. While the patient is on the milk diet the stools should be regularly examined in order to avoid giving more of it than can be assimilated. After a few days on this diet, especially if hunger is developed by it, a soft-cooked egg, beef-juice, scraped raw or rare broiled beef, and a piece of toast or zwiebach may be added to the diet list. If it is deemed advisable to continue the liquid diet, broths, bouillon, clear soups, junket, and gruels may be employed. Farinaceous food should generally be prohibited until the patient has shown marked improvement. It is especially contraindicated in cases of dilatation, for it then remains so long in the stomach that fermentation occurs and intestinal catarrh results. Hot bread and pastry should be permanently forbidden. White bread may generally be eaten in small quantity if it is not too fresh, or better after it has been toasted. Only a small quantity of butter should generally be eaten. When acid eructations are produced, the bread should be temporarily discontinued. The same is equally true of potatoes, although a mealy baked potato is sometimes well borne. Graham or brown bread or that made from the whole wheat agrees with some individuals better than the white. Sugar should be taken sparingly, as a rule.

The behavior of the stomach toward fruits and green vegetables is

very uncertain, and the permission of such articles must be based upon the experience of the individual. Cooked ripe fruit can generally be eaten, and a baked apple is an agreeable addition to the exclusive milk diet. Young peas and beans and stewed onions may prove digestible, but cabbage, cauliflower, corn, strawberries, peaches, bananas, and many other fruits and vegetables must, as a rule, be forbidden. Fat can seldom be eaten; veal, pork, and the meat of any animal just after it has been killed are difficult of digestion. In some cases a diet consisting almost exclusively of beef, roast or broiled, but always tender and rare and freed from its coarse fiber, with an occasional roast of mutton or broiled chops, proves the most satisfactory. In severe cases, after atrophy of the mucous membrane, peptonized beef preparations and peptonized milk may be used with advantage. When solid food is eaten, it is better to limit the quantity of fluids. A small quantity of soup may be taken at the beginning of a meal, but little or no fluid should be drunk with the food. Very hot or very cold drinks, tea, coffee, and alcoholic beverages should generally be forbidden. Finally, the patient should never allow himself to fill his stomach; it is much better to stop before the appetite has been fully assuaged.

Hygienic.—A very important factor in the treatment of most cases is the relief of the morbid introspection which is generally present, with its resultant despondency and melancholy. When the condition is very pronounced, it is often better to have a change of scene, a sojourn in the mountains or a sea-voyage in the summer, or a visit to the Southern resorts in the winter. It is often better to send the patient to one of the watering-places where the dietetic treatment can be carried out. Under any circumstances he should take systematic exercise including walking, horseback riding, and outdoor games. He should always be with cheerful companions who are capable of holding his attention away from himself.

Medicinal.—The objects to be attained by the administration of drugs are: (1) To supply the chemical elements of the gastric secretion which are absent; (2) to restore the secretory and motor power; (3) to prevent abnormal fermentation; and (4) to relieve special symptoms. To meet the first indication, dilute hydrochloric acid is usually administered in doses of ℥xv to xx (1.0—1.2) in two or three ounces of water immediately after meals. It should be taken through a tube in order to protect the teeth, and the dose may be repeated a few hours later or whenever a feeling of discomfort arises. Pepper advised the administration of quinin (gr. j; 0.06) and strychnin (gr. 1-60; 0.001) with the acid. A few grains of pepsin may be added with benefit in some cases, but it is probably seldom necessary, and much of the pepsin that is dispensed is worthless. In cases of extreme atrophy of the mucous membrane, when the administration of the acid fails to stimulate the secretion of pepsin, an active preparation should be administered. The action of hydrochloric acid is not uniformly beneficial. Pancreatin in doses of gr. v to x (0.30—0.60), with an equal quantity of sodium bicarbonate, is often of great value when given a half-hour after each meal, especially in mucous and atrophic cases. Ptyalin and diastase or a good malt extract are recommended by some writers for the same class of cases.

To increase the secretory and motor power of the stomach, the general condition of the patient must be treated, especially in the presence

of anemia or malnutrition, or when the condition is due to venous obstruction, as in connection with valvular disease of the heart or hepatic cirrhosis. Lavage is usually the most successful method of local treatment. (For the method, see page 719.) It should be performed with a large quantity of lukewarm water, either plain or containing 1 per cent of common salt or 5 per cent of sodium bicarbonate. The alkaline solution is especially indicated when much mucus is present. A 3 per cent solution of boric acid or a very dilute carbolic acid solution may be employed when there is much fermentation. The irrigation should generally be continued until the clear water returns. One treatment each day is usually sufficient, or one in two days if the patient be weak. It is best done in the morning, when the stomach is empty, but when there is much distress and flatulency during the night it may be practiced just before retiring. The relief is so great that many patients are restrained with difficulty from abusing the practice. In other cases, however, the fear of the tube is so great that the method cannot be satisfactorily employed, or the patient positively and persistently refuses to submit to it. In such cases the same object may often be attained, with almost as much benefit, from the administration of sodium bicarbonate, gr. xx to xxx (1.3—2.0) in a half-pint (250) or more of warm water, twenty minutes before each meal. In this way the mucus is dissolved, and the effect is much the same as that of lavage. The subsequent secretion of HCl is believed to be increased.

This secretion is sometimes improved by an increase of the amount of salt in the food. The bitter tonics are also useful in some cases, especially to stimulate appetite, but they sometimes prove irritating. Strychnin or the compound tincture of gentian with nux vomica may be prescribed.

One of the most valuable remedies is the nitrate of silver. It is sometimes applied in solution through the tube, but, as a rule, it is administered in pills containing also the extract of belladonna and perhaps nux vomica. It should be given when the stomach is empty, as a half-hour before mealtime. A record of the quantity administered should always be kept, in order to avoid the production of argyria. The first indication of this condition is a dark line on the gums.

Electricity has proved beneficial in some cases. A mild faradic current is applied through Einhorn's electrode after the stomach has been moderately distended with water. Many other remedies have been recommended—among them, creosot, carbolic acid, magnesia, animal charcoal, bismuth subgallate, salicylic acid, chloroform, and the essential oils—for the relief of acid fermentation and flatulency. For nausea and vomiting, the dilute hydrocyanic acid, three drops; serium oxalate, camphorated tincture of opium or cocain, gr. ⅛ (0.008), may be given. The regular action of the bowels must be secured by laxatives.

DILATATION OF THE STOMACH.

GASTRECTASIA, GASTRECTASIS.

Definition.—An acute or chronic enlargement of the stomach, with relaxation and weakness of its walls. The term *megastria* is applied to the

condition in which the stomach is abnormally large, but still capable of discharging its contents into the duodenum.

Etiology.—Acute dilatation is rare, in this country at least, and usually results from a too hasty ingestion of an enormous quantity of food or drink; occasionally from the rapid evolution of gas. The sudden dilatation produces a paralytic condition of the walls, which sometimes proves fatal.

Chronic dilatation may occur at any time of life, but it is more frequent in men of middle age, especially in beer-drinkers. It is not uncommon, also, in rachitic children. The principal causes are narrowing or obstruction of the pylorus or duodenum, and deficiency of muscular power.

(*a*) Narrowing of the pylorus is seen in the rare congenital stricture and as a result of cancer, the cicatrization following ulcer, toxic or phlegmonous gastritis. It is produced also by the nonmalignant hyperplastic thickening of the pylorus in chronic gastritis. Among the other recognized causes are pressure from without, by tumors, rarely by a floating kidney; or obstruction from within, as by polypi. A sharp bend or twist caused by adhesions to the liver or gall-bladder or by the dragging weight of a distended stomach (volvulus of the stomach) has been observed in a few instances. Obstruction by foreign bodies, as balls of hair, or coins, has been the cause in some cases.

(*b*) Deficiency of muscular power may result from habitual overfilling of the stomach with food and drink or from atony of the muscle due to malnutrition in the course of such diseases as chronic gastritis, anemia, tuberculosis, or cancer, or following nervous exhaustion or an acute infection, as typhoid fever. It may follow degeneration of the muscle occasioned by amyloid disease, constipation, or peritonitis; and hernia has been named as a cause which might operate through restraining the movements of the stomach.

Morbid Anatomy.—The degree of dilatation is exceedingly variable. Extreme cases have been reported in which the stomach held from 10 to 16 pounds of fluid. When the dilatation is moderate, there may be compensatory hypertrophy of its walls; but when it is extreme or of long duration, the walls become thin through stretching and atrophy. The greatest dilatation is found in the fundus, and the greater curvature sometimes sinks two or three inches below the umbilicus. Gastroptosia, or downward displacement of the entire stomach, may accompany the dilatation, and the right kidney is sometimes displaced downward. The mucous membrane is usually hyperemic and, on microscopic examination, shows atrophy of the secretory glands, as it does in chronic gastritis.

Symptoms.—The clinical manifestations vary greatly with the severity of the disease and the nature of its cause. Symptoms are ordinarily present which resemble closely those of an aggravated chronic gastritis. There is a feeling of fullness or distress in the stomach, with epigastric tenderness, especially after eating. Large quantities of gas and sour fluid are eructated. The appetite is variable, sometimes ravenous; the tongue is heavily coated and the breath is usually foul. The most nearly pathognomonic symptom, however, is periodic vomiting. This may occur several times a day in the beginning, but as the dilatation increases it becomes less frequent until it occurs only at intervals of two or three days, and an enormous quantity of food, liquid, and gas is brought up.

As much as a gallon (4 liters) is sometimes vomited at one time. The vomited matter is generally frothy, from abnormal fermentation, and it has a characteristically sour odor. It separates, on standing, into three layers. The lowest contains the food, the middle a dark gray turbid fluid, and the upper a brownish froth. HCl is often absent, but it may be present in normal, increased, or diminished quantity. Lactic, butyric, and acetic acids and various gases, especially hydrogen sulphid and marsh gas, are present. Different molds and bacteria, the yeast fungus and sarcina ventriculi, are found on microscopic examination. Constipation results from the obstruction to the passage of food and fluid into the intestine; and anemia and emaciation, with marked dryness of the skin, commonly follow. When the stomach finally becomes unable to contract with sufficient force to discharge its contents through vomiting, the patient's discomfort is extreme. The abdomen becomes so much distended that he cannot lie down with comfort, and the nausea and gaseous eructations become almost constant. The absorption of the products of decomposition gives rise to such nervous phenomena as numbness, vertigo, and insomnia; tetany and epileptiform spasms may be induced. Fat-necrosis has been reported in at least one instance, probably as a result of pressure on the pancreas.

Physical Examination.—*Inspection.*—The outline of the dilated stomach is often distinctly visible, especially after artificial distention. The greatest prominence is in the left hypochondrium, and below the umbilicus when the patient is standing, but the epigastrium, right hypochondrium, and umbilical regions are prominent. The peristaltic movement from left to right can sometimes be observed, and rarely there is a reversed peristalsis.

Palpation.—A tumor-like thickening in the region of the pylorus can often be felt in the nonmalignant as well as in cancerous cases. The sensation obtained from pressure on the stomach is peculiar and not unlike that of palpating an air-cushion. A splashing sound which can be heard at some distance (clapotage) may often be produced through bimanual examination or by shaking the patient. It is significant of dilatation only when it can be elicited several hours after a meal. The gurgling of gas passing through the pylorus can sometimes be felt.

Percussion.—The outline of the stomach can be accurately mapped out by this method, but the examination should be repeated with the patient in different positions, in order to avoid error in the lower boundary. This can be more definitely made out with the patient lying on his back. The percussion note is tympanitic and rather high-pitched when the stomach is distended with gas or air, but flat when distended with water. The latter method of distention is of little value for determining the outline.

Auscultation is of limited service. The succussion sound may be readily determined in most cases, but it is not of much value. A fine sizzling sound can sometimes be heard, which is believed to be due to the evolution of gas in the decomposition of the food, since the same sound can be heard after the administration of an effervescent powder. When the stomach is distended with gas, the heart-sounds are transmitted with unusual distinctness and they often have a metallic quality. Auscultatory percussion also affords an accurate means of determining the outline.

Diagnosis.—This is usually quite simple. It is based upon: (*a*) The periodical vomiting, (*b*) the enormous quantity ejected, (*c*) the gaseous eructations, and (*d*) the enlarged outlines of the distended stomach. The condition cannot well be mistaken for any other, although some remarkable blunders have been recorded. In *gastroptosia* the stomach is displaced downward and may be somewhat enlarged, but the food does not stagnate in it, and vomiting is not present. In *megastria* the enlargement in not strictly pathological, since the stomach is capable of performing its normal function.

Prognosis.—The prognosis is hopeless in a case due to cancer, and very unfavorable in other forms of stenosis. When the condition is due to overdistention with food and drink or to chronic gastritis, and providing that it is not extreme, great benefit can be obtained from treatment, but it is seldom possible to control the patient's habits outside of a hospital. The results of surgical methods in nonmalignant stenosis have been brilliant in some cases.

Treatment.—This comprises relief from the source of distention through removal of the fermenting stomach-contents. The best means of accomplishing this is lavage. The second object is to increase the muscular power of the organ, and the third to select the most suitable diet for the patient.

(*a*) *Lavage* is not only the most useful method, but it has its greatest field of usefulness in this condition. By it the weight of the accumulating food is removed from the stomach, and the expansive force of the gases developed in it is taken away. At the same time the mucous membrane is cleansed and disinfected, and any ptomains or toxins that may have been formed are gotten rid of. (For the method of lavage, see page 719.) The stomach should generally be washed out once a day, but in extreme cases it may be done twice. It is customary to use lukewarm water or an alkaline or antiseptic solution. The reduction of size obtained in a few weeks is sometimes remarkable. The patient can generally be taught to use the tube without assistance.

(*b*) To increase the muscular power, strychnin is the best remedy. A tablet containing gr. 1-30 (0.002) should be given three or four times daily. Iron and ergot are also of benefit in some cases. The faradic current may be applied through the Einhorn electrode introduced into the stomach, the opposite pole being applied to the epigastrium. E. G. Marshall has obtained better results by applying a large sponge electrode to the epigastrium, and a smaller one, connected to the same pole by means of a V-shaped cord, to the neck, over the course of the pneumogastric nerve. The wearing of an elastic abdominal band is a source of relief from the dragging weight.

(*c*) *Diet.*—The important indication is to administer the food in a concentrated form, in order not to produce distention or downward traction. The food must be of a character that will not produce gaseous dilatation. It should be taken in small quantities at short intervals. In the beginning, it should consist principally of raw or rare broiled or roast beef and other tender meats; soft eggs with a little toasted bread or zwiebach. Very little fluid should be taken; the milk diet is contraindicated. Water may be drunk an hour before each meal, or in the morning upon rising, and before retiring at night, but not with the meal.

After the size of the stomach has been considerably reduced by treatment, thoroughly cooked vegetables may be carefully added to the diet-list. In cases of stenosis that are known to be nonmalignant, and in those that are doubtful, the resort to surgical means of diagnosis and treatment should not be too long delayed, for a condition has not infrequently been revealed upon the post-mortem table that might have been remedied. The principal methods will be found in the works on surgery, under the heads of Loreta's method of digital dilatation of the pylorus, gastroenterostomy, and pylorectomy.

PEPTIC ULCER.

SIMPLE, ROUND, PERFORATING, OR RODENT ULCER OF THE STOMACH.

Definition.—A round or oval, usually single, sharply defined loss of tissue caused by the digestive action of the gastric juice on a portion of the mucous membrane of the stomach or of the duodenum whose nutrition has been impaired. The process of its formation is one of necrosis rather than of ulceration.

Etiology.—Two factors are generally regarded as operative in the production of the so-called ulcer; first an impairment of the nutrition of a small portion of the mucous membrane, second the action upon this area of a superacid gastric juice, favored, perhaps, by an alteration in the composition of the blood. The disturbance of nutrition is probably due to such vascular change as the plugging of a small blood-vessel, through thrombosis or embolism, or to a diminished circulation in the vessels. The interference may be caused by mechanical or thermal irritation of the mucous membrane, as by blows or pressure upon the epigastrium or by hot food. The embolus may originate in a diseased heart, or it may consist of bacteria which have gained entrance to the circulation. And, since duodenal ulcer frequently follows burns, it has been suggested that an embolus may be caused by such injury. The superacidity of the gastric fluid is regarded by some writers as a result of the ulcer. If not active in its production, it doubtless retards its healing. Cystic dilatation of Brunner's glands has been suggested also as a cause of the duodenal ulcer. It is probable that more than one of these causes is operative in most cases. It is also highly probable that the true cause of many cases remains to be discovered.

Age and Sex.—Gastric ulcer is about twice as common in women as in men. In women it generally occurs between the ages of 20 and 30, in men between 30 and 40. It is not infrequent in children and old persons, and it has been found in the fetus and new-born infant. It is doubtless more common than is usually recognized. Heredity is thought to be a causative influence in some instances. Ulcer of the duodenum is more frequent in men.

Occupation is an important factor, since the disease is very frequent among servant girls and cooks, probably as a result of improper food and consequent anemia; and in shoemakers and tailors, as a result of pressure over the stomach. The history of such injury as a blow is often obtained.

Anemia and *chlorosis* are recognized as frequent causes, operating,

perhaps, by reducing the acidity of the blood, or in some other way impairing the power of the mucous membrane to resist the action of the gastric juice. Such affections as tuberculosis, syphilis, disease of the heart or liver, and arteriosclerosis have been regarded as favoring the production of gastric ulcer.

Morbid Anatomy.—The ulcer is usually single, but a large number (in one case 34) have been repeatedly found. Several ulcers may coalesce to form an irregular destruction of tissue. They may occur in any region, but in three-fourths of the cases they have been found near the pylorus, generally on the posterior wall near the lesser curvature. In the duodenum they are much less frequent than in the stomach, and they are always found above the biliary papilla. Their diameter is usually about ¼ inch (6 mm.), but they may be as large as four or five inches (10.0—12.5 cm.). The appearance of a recent ulcer is as though the tissue had been removed with a punch, so uniform and clean-cut are its edges. The shape is that of a truncated cone, the apex of which may rest upon the submucous, muscular, or peritoneal coat. In very acute cases, especially when located in the anterior wall, the peritoneum may be perforated, with fatal general peritonitis as the inevitable result. In some instances a sinus is formed which communicates with the colon, pleura, pericardium, even with the left ventricle of the heart. Localized abscesses sometimes result, and when air penetrates these a condition known as subphrenic pyopneumothorax is produced. In very chronic ulcers the clean-cut appearance may be lost. A striking feature is the absence of inflammatory reaction in the vicinity of the ulcer.

The scars of former ulcers are not infrequently discovered, and a recent ulcer is often found in close proximity to an old cicatrix. Deformities of various kinds may be produced by the contraction of the cicatrices of large or numerous ulcers. Hour-glass contraction is occasionally observed, but a much more common result is the narrowing of the pylorus, which may lead to dilatation. Deformity may result also from the adhesions formed between the peritoneum immediately over an ulcer and a neighboring organ, as the liver or spleen. These adhesions, however, often prevent perforation into the peritoneal cavity. A gastrocutaneous fistula is a rare, though relatively fortunate, result of perforation. It usually opens near the umbilicus. Emphysema of the subcutaneous cellular tissue has been observed as a result of perforation. The erosion of a blood-vessel in the stomach-wall produces hemorrhage, a not uncommon symptom. The hepatic and splenic arteries and the portal vein have also been opened.

Symptoms.—The early symptoms are usually those of indigestion, with discomfort after eating and gaseous or acid eructations. The appetite sometimes remains normal, but nausea frequently develops, and vomiting may occur. The patient rapidly becomes anemic, and the dyspeptic symptoms gradually or suddenly increase until the discomfort becomes pain. This is usually limited to the region of the ulcer. It may be an almost constant, gnawing sensation, but it is generally sharp. It is aggravated by eating and relieved by vomiting, sometimes also by a change of position which allows the contents of the stomach to gravitate away from the ulcer. Pressure on the painful spot elicits tenderness, but it is often found to afford relief, and the patient bends over a chair

or lies with a pillow under his abdomen. The pain commonly radiates to the back, sometimes also to the sides. It is generally felt in the back at a point a little to the left of the tenth dorsal vertebra. It may be continuous for weeks, but occasionally ceases for a variable interval. Attacks of intense gastralgia sometimes occur independently of the local condition. This is generally at a point an inch or two below the ensiform cartilage and a little to the right of the median line.

Vomiting sometimes occurs without much nausea, either immediately after meals or at irregular intervals of several days. The vomitus is usually highly acid, the free HCl sometimes reaching 0.5 per cent. The ferments are not generally altered in amount.

Hemorrhage occurs in nearly or quite half the cases. It may be slight, but it is generally profuse and may induce syncope or convulsions. Unaltered blood, bright red and fluid, is brought up in these cases. Hemoptysis is, indeed, one of the most characteristic symptoms. A free, even fatal, hemorrhage sometimes results from a small superficial ulcer or a scarcely recognizable erosion. After the blood has remained in the stomach for a short time, it becomes altered and mingled with the food. Blood from either a gastric or a duodenal ulcer can generally be found in the stools after a hematemesis, and sometimes in cases which have not been attended with hematemesis. The blood appears in the stools as black, tarlike matter. The stools should always be examined in a case of suspected ulcer.

Perforation occurs in about 6 per cent of cases. It may follow undue pressure or the ingestion of food. It is more common in women. Its occurrence is announced by a sudden severe pain which is generally confined to the epigastrium, but may radiate over the abdomen or be referred to another region. Collapse quickly follows; the abdomen becomes distended and tender, the pulse small and rapid. The Hippocratic facies, shallow respiration, and other manifestations of peritonitis rapidly develop. The evidences of perforation are occasionally the first indication of the existence of the ulcer.

Complications.—The most important of these are pylephlebitis, with abscess of the liver; chronic peritonitis; and suppurative parotitis. Dilatation of the stomach follows narrowing of the pylorus from cicatrization, and sclerosis of the walls is not uncommon.

Diagnosis.—The cardinal symptoms are the peculiar localized sensation of discomfort or of pain and tenderness, vomiting, especially hematemesis accompanied with pronounced anemia. Ulcer is to be differentiated from gastralgia, hyperchlorhydria, acute gastritis, and cancer, and the hemorrhages from hematemesis of other character.

Gastralgia is a neuralgic pain which is not limited to a definite area, and it is, as a rule, neither relieved nor intensified by the taking of food. The patient does not become anemic; digestion may be normal between the attacks; vomiting seldom occurs; hematemesis is absent; and if there is tenderness, it is diffuse. [illegible] may be present.

In *hyperchlorhydria* the pain is diffuse and it usually occurs two or three hours after the ingestion of food.

Acute gastritis is generally accompanied with fever and evidences of toxemia. Blood is absent from the vomited matter, or present in only trifling amount. The [illegible] more than a week.

Carcinoma almost always occurs in individuals past 40. The pain is irregular, sometimes absent; tenderness may not develop until late. Vomiting occurs irregularly or it is absent; hematemesis is not profuse, and the blood usually has the coffee-grounds appearance. A tumor can generally be felt, and the patient acquires a cachectic appearance. The free HCl is usually diminished and sometimes absent.

The *gastric crises* of locomotor ataxia sometimes simulate the paroxysms of pain from ulcer before the more characteristic symptoms of the disease have developed. They are usually associated, however, with the lightning pains, ocular symptoms, and absence of the patellar reflex.

Chlorosis.—Hematemesis sometimes occurs in chlorotic girls, which cannot always be attributed to an ulcer, on account of the rapidity of the recovery which follows. It is believed to be due to simple vascular engorgement of the gastric mucous membrane. The diagnosis is difficult, but the localized pain and tenderness of ulcer are generally absent, and there may have been no previous indigestion.

Gall-stone colic can generally be recognized by the location and character of the pain, its sudden onset and sudden termination, as well as by the enlargement and tenderness of the liver, sometimes accompanied with distention of the gall-bladder and jaundice.

The *hematemesis due to cirrhosis* of the liver accompanies the characteristic symptoms of that disease. The liver is small, and jaundice and ascites are usually present.

The differentiation between gastric and duodenal ulcer is often impossible. The latter location of the ulcer may be suspected, however, when a sudden intestinal hemorrhage (melena) takes place in a previously healthy person, or when there is a history of pain in the right hypochondrium two or three hours after meals.

Prognosis.—Recovery usually follows appropriate treatment, but recurrence is common. It is never safe to give a favorable prognosis, for the course of the disease is exceedingly uncertain. The danger of perforation is always to be regarded, and the hemorrhages sometimes prove fatal. A chronic ulcer may terminate unfavorably through inanition and exhaustion.

Treatment.—The first indication is to give the stomach complete rest. This may be accomplished in severe cases by confining the patient to bed and resorting to rectal alimentation for at least the first week. In ordinary cases, however, it is sufficient to give easily digestible, unirritating food in small quantities at regular intervals. It is generally best to give only milk at first; 4 ounces every two hours. It may be plain or peptonized, and buttermilk often agrees better than sweet milk. Beef-juice or peptonized beef, and egg albumen, may be allowed after a few days, and the diet should be restricted in most cases to such articles as these during the first month. It may then be extended so as to include rare beef, the white meat of chicken, poached eggs, toast, and well-cooked farinaceous articles.

Pain is rarely so intense as to require the administration of morphin. Relief can generally be obtained from alkalis, as sodium bicarbonate, the compound spirit of sulphuric ether, camphor-water, or a few drops of chloroform.

When vomiting is persistent, it may be necessary to adopt rectal ali-

mentation. Mustard may be applied to the epigastrium, or a few quick strokes may be made with the cautery. The vomiting is sometimes checked by bismuth, with or without opium, by dilute hydrocyanic acid, chloroform, or by sipping a carbonated water or champagne. Lavage with a warm alkaline solution has been recommended, but it should be performed with the utmost care. The action of the bowels must generally be regulated by either a saline laxative or enemata. Thirst is best relieved by enemata of salt water. The powder of Stockton and Jones often proves an excellent remedy for the pain, vomiting, and constipation. Each powder contains: Of cerium oxalate, gr. ij (0.13); light magnesium carbonate, gr. x (0.65); and bismuth subcarbonate, gr. xx (1.30). It should be administered from three to six times a day. A grain or two of reduced iron should be added to each powder, for the anemia.

For the healing of the ulcer, large doses of bismuth subcarbonate, gr. xxx to lx (2.0—4.0) three times a day, are of unquestionable benefit. The nitrate of silver in doses of gr. ¼ (0.016), combined with opium, was highly recommended by Pepper.

When hemorrhage occurs, the patient should be immediately placed under the influence of morphin, given hypodermically at such intervals as will insure complete rest for several days. Ergotin, gr. ij (0.13), may also be administered hypodermically. Astringent remedies per os are useless. In extreme cases the subcutaneous injection of saline solution should be resorted to. During convalescence iron should be administered freely in a nonirritating form. The reduced iron is probably best. Surgery has accomplished excellent results in a few instances after perforation or persistent hemorrhage. The source of the bleeding cannot always be discovered, but ligature of the artery in the stomach-wall which supplies the region has resulted in arrest of the hemorrhage and healing of the ulcer.

CANCER OF THE STOMACH.

Etiology.—*Sex and Age.*—The sexes are about equally affected. More than half the cases occur between the ages of 40 and 60; the disease is rare under 30, but cases have been observed in children, and a few congenital cases have been reported. Next to the uterus, the stomach is the most frequent location of cancer.

Race.—The disease is much more frequent in the white race than among negroes.

Heredity is regarded as a strong predisposing factor, but in many cases no such influence can be traced.

Previous Disease and Habits.—That previous irritation of the stomach is influential is inferred from the frequent location of the growth in the pyloric region as well as from the frequent history of long-standing catarrh before its onset. In about 6 per cent of cases there is a more or less definite history of ulcer, or of injury to the region of the stomach. A considerable proportion of cancer patients have been addicted to free indulgence in alcohol. But in general no great importance can be attached to previous disease, habits, occupation, or station in life.

Bacteria.—Cancer is regarded by many authorities as a specific infection, but the microbe has not been identified. It is possible that some

one of the micro-organisms recently demonstrated in the growth, and cultivated with more or less success, will prove to be the cause of the disease. Cancer of the stomach is usually primary, but it is sometimes secondary to cancer of one of the adjacent organs.

Morbid Anatomy.—The different types of cancer are found in the following order of frequency: (1) Cylinder-celled epithelioma, (2) encephaloid, (3) scirrhous, (4) colloid. The epithelioma is much the most common. The soft encephaloid or medullary and the colloid are the most rapid in growth and invasion of tissues, and the hard scirrhous is least so. The epithelioma and scirrhus are usually found as comparatively small masses, while the encephaloid and colloid frequently invade almost the entire wall of the stomach. The growth most commonly originates near the pylorus, next in the lesser curvature, then at the cardia, the posterior wall, the greater curvature, and finally in the fundus. Multiple tumors are occasionally encountered.

The stomach is dilated as a result of cancer at the pylorus, and much contracted when the growth is located at the cardiac orifice. The esophagus is much dilated above a tumor at the cardia. In some cases of pyloric scirrhus, however, the stomach is contracted, although marked stenosis may have been produced. The stomach may be displaced downward by its weight, and it may drag with it the surrounding viscera. In some cases the tumor is remarkably movable, while in others adhesions are formed between the stomach and colon, liver, or anterior abdominal wall. Metastatic growths are often found in the lymph-glands, liver, gall-bladder, peritoneum, omentum, intestine, pancreas, spleen, pleura, and lungs, or elsewhere. Small subcutaneous cancers are sometimes found in the epigastric and hypogastric regions.

Symptoms.—There is great diversity in the symptoms of different cases. There may be no manifestations by which the disease can be recognized until a comparatively late period. The history that is generally obtained is that of indigestion during several months, increasing in severity, and attended with anemia and emaciation. The disease is sometimes discovered post mortem in those dying from other causes. In another class of cases the symptoms appear comparatively early and are characteristic. The disturbances caused by the metastatic growths, especially in the liver, are occasionally more prominent than those of the primary disease.

The early symptoms in an ordinary case are loss of appetite, impaired digestion, pain, nausea, and vomiting. Later there are loss of weight and strength, anemia, emaciation, cachexia, and finally prostration, and death from toxemia or exhaustion. The early symptoms are not typical; they indicate only a disturbance of the function of the stomach. The loss of appetite is an early symptom, and it is one of the most constant. The tongue becomes heavily coated and dry. Nausea and distress soon develop after meals; then the feeling of oppression in the stomach becomes more constant, and it is aggravated into a distinct pain by the ingestion of food. Eructations become a prominent feature, and then occasional vomiting.

Vomiting.—In some cases neither nausea nor vomiting is present throughout the disease, but in others they are extremely troublesome symptoms, especially in the last stages. Vomiting is more frequently

present when the cancer is situated at the pylorus, and more frequently absent when it is at a distance from this region. It is observed, however, early or late, in about four-fifths of all cases. It often bears no relation to the taking of food. The vomitus may consist only of food and mucus, or it may contain adventitious matter, particularly blood. It has a sour odor and in some cases becomes distinctly fetid or feculent, especially as a result of the separation of gangrenous sloughs from the tumor. The food may show but little digestion, after having remained in the stomach many hours.

Hemorrhage.—Free hematemesis is rare. The blood is sometimes so small in quantity that it can be discovered only by microscopic or chemical examination. Altered corpuscles can often be recognized; hemin crystals may be obtained, or the guaiacum test may be applied. In other cases the blood appears as coffee-ground matter, which is regarded as highly pathognomonic of cancer. Large ulcerating cancers are most liable to be attended with free hemorrhage; scirrhus often runs its course without it.

Pain is a prominent symptom in about three-fourths of the cases and often occurs early. It is usually confined to the epigastrium, but may be referred to the shoulders, sides, or back. It is generally of a burning, gnawing, or dragging character; distinct cardialgia rarely occurs. It is generally constant, but increased by ingestion of food. Tenderness is usually elicited by pressure over the region affected, sometimes also over the back between the fifth and twelfth dorsal vertebræ.

Anemia and *cachexia* are often early symptoms and almost invariably present. The number of red corpuscles often sinks below 3,000,000, occasionally below 2,000,000, and the hemoglobin may fall below 50 per cent. The anemia is one of the chief elements in the production of cachexia, but, in addition, the skin acquires a pale yellow tint, often associated with brownish discoloration of the face, neck, and backs of the hands or other regions. The skin appears firm and inelastic, sometimes slightly edematous. When the anemia is extreme, there is often edema of the lower extremities and sometimes a more general dropsy.

Emaciation often begins early, but in a large proportion of cases there is little loss of weight until a late period of the disease. The degree of emaciation is often remarkable, the body being literally reduced to "skin and bones." The decline of strength usually keeps pace with the loss of flesh, but a remarkable degree of vigor is sometimes retained to the end.

Fever is not a prominent symptom, but there is usually some elevation of temperature during the course of the disease. It may not occur until late, and may never exceed 101° F. (38.5° C.), but toward the end it often rises to 103° F. (39.5° C.) or higher. When suppuration occurs at any time, in the growth or near it, fever is commonly produced. The pulse becomes weak. Thrombosis of one of the femoral veins, rarely of other vessels, has been encountered.

The *urine* often remains unchanged. Indican may be present, however, and sometimes a small quantity of albumin. Aceton, pepton, and glucose are occasionally found in it. Constipation prevails in most cases, rarely diarrhea. Blood may be occasionally found in the stools.

The tumor can usually be recognized at some time during the course of the disease when it is situated at the pylorus; less frequently when at

the cardia or lesser curvature. The diffuse carcinoma seldom produces a prominence that can be felt. It is usually recognized by palpating deeply into the epigastrium just to the right of the median line. It is at first slightly movable, sometimes freely so, but later it may become adherent. It may be firm and smooth or nodular. As it enlarges it usually transmits the pulsations of the aorta with distinctness. Sometimes it drags down the stomach and other viscera to a lower position in the abdomen. In most cases, a pyloric cancer causes obstruction, consequent increase of the peristaltic movement, and, later, dilatation of the stomach. Occasionally, however, the pyloric orifice is held open, permitting a regurgitation of bile into the stomach.

The *motor efficiency* of the organ is impaired in nearly all cases, except those involving the cardia or in the presence of small tumors of the fundus. It is most reduced when a large portion of the stomach-wall has been invaded by the growth. When the tumor is located at the cardia, the stomach generally becomes contracted, and in other respects the symptoms are the same as those of cancer of the esophagus.

Physical Examination.—*Inspection.*—From this we learn the general condition of the patient as to nutrition; his color, whether anemic or cachectic, and the presence of abnormal pigmentation. The abdomen usually appears prominent, and the lower intercostal spaces may be widened. Small subcutaneous nodules in the epigastric or umbilical region are often of diagnostic value. When the tumor is large it may protrude slightly, and the transmitted aortic pulsation may become visible. Exaggerated peristaltic movements are often seen comparatively early. They are more readily recognized after the stomach has been inflated with air, but this method should not be practiced in a case of extensive involvement or after hemorrhage has occurred.

Palpation affords more positive means of recognizing the tumor. In some cases it can be recognized by examining the patient in a recumbent posture; in other cases by placing him in the knee-elbow position. Sometimes the tumor can be felt only during inspiration. The mobility of the growth is often of diagnostic value; not only the extent to which it can be displaced with the hand, but also the extent to which it moves during respiration and with peristalsis or with inflation. Tumors of the pylorus are occasionally extremely movable, so that they can be displaced into either hypochondriac region or drawn down to the umbilicus. Through palpation, also, the escape of gas through the narrowed pylorus can be discerned, as in other forms of stenosis. *Percussion* is seldom of much value, and auscultation reveals nothing characteristic.

Examination of the Stomach-Contents.—(For methods see p. 719.) HCl is not invariably absent, but its persistent absence is regarded as highly pathognomonic of cancer, especially that of the pyloric region. It serves also to distinguish cancer of the stomach from that of adjacent organs. Lactic acid is often, but not invariably, found. Its constant presence is regarded as of greater diagnostic importance than the absence of HCl. The rennet, or milk-curdling ferment, is generally reduced in quantity. On microscopic examination, various micro-organisms are ordinarily found, among them the Boas-Oppler bacillus, a long, non-motile rod, which is supposed to be operative in the production of lactic acid. Yeast fungi are usually found, and sarcinæ may be present.

The course of the disease seldom exceeds a year or eighteen months, and cases are occasionally reported which apparently run their course in three or four months.

Complications.—Such more or less direct results as dilatation or perforation, and such pressure symptoms as occlusion of the bile-ducts or blood-vessels, and the development of metastatic growths are often referred to as complications. The metastases are most commonly encountered in the liver, lymph-glands, omentum, mesentery, pancreas, occasionally in the spleen, lungs, pleura, axillary glands, the pelvic organs, or other parts.

Diagnosis—The early recognition of cancer of the stomach is often extremely difficult, especially its differentiation from chronic gastritis and ulcer. It is to be suspected especially when it develops pronounced symptoms of gastritis in a person who has always been healthy, and more particularly when anemia and cachexia become apparent and when, in an elderly person, the indigestion is accompanied with pain. The methods of examination already referred to may render the recognition of the disease possible. When a tumor is discovered, it strongly supports the diagnosis. The diseases from which cancer is to be particularly differentiated are chronic gastritis, ulcer, and the severe primary anemias.

In *chronic gastritis* there is usually a history of long-continued indigestion, without tumor or cachexia, and the blood-changes are less pronounced. Lactic acid is not generally found after a test-meal.

Ulcer is to be distinguished especially by the presence of hyperchlorhydria, the gastralgic attacks, and the profuse hemorrhages. It usually occurs in younger subjects.

Anemia.—Grave anemia is often suggested by the appearance, especially when the digestive disturbances are of only moderate severity and when tumor is absent. In countries where profound anemia is a common result of animal parasites, the differentiation may be especially difficult, until examination of the feces is made. In pernicious anemia the blood-count shows a more profound diminution of the red corpuscles than is observed in cancer. In cases of doubt, an exploratory incision is to be recommended. In many cases, without such examination the case must remain in doubt until the development of characteristic symptoms, possibly several months after the first examination.

Prognosis.—The disease is invariably fatal. The only exceptions are the few cases in which early removal of the tumor has more or less permanently arrested the disease.

Treatment.—The most important question is, Can anything be done by surgical measures? If not, the treatment is wholly palliative. The management of the digestive disturbances is that of chronic gastritis, and perhaps that of dilatation. When HCl is absent or greatly deficient, it should be supplied by the administration of the dilute acid. When stenosis occurs, the patient may be nourished by the rectum, but usually only for a very short time. Previous to this the most easily digestible and most nourishing food must be given, as finely chopped meats, soft vegetables, salads, eggs, rice, custards, and the like. When the tumor is at the cardia, the diet must be entirely liquid in most cases. Much benefit and prolongation of life have followed in some instances the introduction of the gastric canula. Milk is often the best food. When

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Peptic ulcer and cancer;

passive congestion due to obstruction of the portal circulation, as hepatic cirrhosis, pressure of a tumor, thrombosis of the portal vein, chronic valvular disease of the heart, and fibrosis or emphysema of the lungs; (*c*) enlargement of the spleen. (*d*) Traumatic hemorrhage also occurs as a result of blows and wounds or the introduction of the stomach-tube or sounds, and it may follow the action of corrosive poisons, phosphorus, or alcohol, or the injury occasioned by a foreign body. (*e*) A toxic cause is sometimes recognized, when it occurs in connection with such acute infections as measles, smallpox, yellow fever, pernicious malaria, and dengue, or in acute yellow atrophy of the liver.

2. The general conditions causing it are (*a*) chlorosis, pernicious anemia, leukemia, purpura, scurvy, and hemophilia. (*b*) Vicarious menstruation from the stomach has been observed. (*c*) Hemorrhage of the stomach alone or in connection with the bleeding of other mucous membranes occasionally occurs in the new-born infant. (*d*) Hematemesis is sometimes feigned by hysterical women who vomit blood or colored fluids that have been previously swallowed.

Morbid Anatomy.—In the absence of such definite lesions as ulcer, cancer, corrosion, or other injury, the source of the blood may be determined with difficulty, if at all, since it often comes from numerous small erosions which are not recognizable after death, or from the rupture of a miliary aneurism or a small vein in the submucosa which occasions so slight lesions of the surface as not to be discovered on careful examination. When death has resulted from the hemorrhage, the mucous membrane participates in the general anemic condition and appears extremely pale.

Symptoms.—The blood may be retained in the stomach, even in cases of fatal hemorrhage. The quantity vomited in other cases may vary from two or three ounces to as many pints or even more. Fatal hemorrhage most frequently results from ulcer or the rupture of varicose esophageal veins in cirrhosis; death may ensue from a single hemorrhage, but more commonly as a result of repeated hemorrhages during several days. Free hematemesis often brings up bright red arterial blood, but if the blood has been retained in the stomach for a short time it assumes the character of coffee-grounds or chocolate-colored masses, which become bright red on the surface after exposure to the air. The blood may be mixed with food, mucus, or pus. Melena is also a common symptom. Such other symptoms of hemorrhage are observed as pallor, restlessness, rapid feeble pulse, accelerated respiration, cold perspiration, subnormal temperature, syncope, or convulsions.

Diagnosis.—When a large quantity of blood is retained in the stomach, the fact that a hemorrhage has occurred can generally be recognized by the pallor, cardiac weakness, rapid respiration, and other indications already referred to. The percussion note over the stomach is flat. The statement of the patient that blood has been vomited cannot always be accepted, for deception is sometimes practiced by the hysterical, and errors have occurred from the staining of the stomach contents with fruit-juices or red wine or from the black discoloration caused by bismuth or iron. Such stains can generally be recognized by mere ocular inspection, but microscopic or chemical examination is sometimes necessary. The most important distinction is usually to be made be-

tween hematemesis and hemoptysis. In *hematemesis* there is usually a history of gastric, hepatic, or splenic disorder, an acute infection, toxic or mechanical injury, and the blood is brought up by vomiting in a more or less changed condition and often mixed with food, mucus, or gastric juice, which gives an acid reaction. In *hemoptysis* there is the history of cough or other evidence of pulmonary or cardiac disease; the blood is raised by coughing and it is bright red and frothy, of alkaline reaction, rarely clotted, but sometimes mixed with mucus or pus. Vomiting may accompany the hemorrhage, and hence the presence of stomach-contents cannot be regarded as evidence against hemoptysis. Auscultation generally reveals numerous moist râles or coarse bubbling in one of the lungs. Following hematemesis, tarry stools commonly occur, and after hemoptysis the sputum continues to be blood-stained for several days.

Prognosis.—The hematemesis of ulcer or cancer is not commonly fatal; that from cirrhosis of the liver, splenic enlargement, the rupture of an aneurism, or penetrating wounds is generally so.

For the treatment of hematemesis see that of gastric ulcer.

NEUROSES OF THE STOMACH.

NERVOUS DYSPEPSIA.

Definition.—This term has been applied to a group of functional disorders which occur without discoverable anatomical lesions. They are in nature either secretory, motor, or sensory.

Etiology.—Underlying all cases of true neurosis there is a disordered state of the nervous system. This may be inherited or acquired. It often happens, however, that the gastric symptoms are so prominent as for a time to overshadow the general nervous condition. The inherited form often shows itself in different members of the same family. The acquired neuroses result from nervous excitement, worry, overwork, especially in a confining occupation, and are favored by neglect of hygiene, irregular or hurried meals, and loss of rest. Eyestrain, especially that due to astigmatism and errors of refraction, has been adduced as a cause. The neuroses sometimes develop in the course of the acute infections or as a sequel to them. The gastric crises of locomotor ataxia are regarded as a neurosis. More than one form of neurosis is often present in the same case, and the severity of the condition varies within broad limits.

1. NEUROSES OF SECRETION.

(*a*) **Supersecretion or Superacidity.**—This is a rather rare condition, but is sometimes seen in connection with neurasthenia, locomotor ataxia, and other nervous affections. The entire quantity of the gastric juice is increased, its acidity remaining normal or undergoing increase. In most cases the condition is constant, but it may be periodic, lasting for several days at a time.

Symptoms.—The periodic attack usually sets in with a gnawing sensation in the stomach and headache. Vomiting soon follows, with the ejection of a large quantity of watery fluid, which is so highly acid in most instances that it irritates the throat and leaves it raw and sore.

The secretion of the fluid is remarkably rapid. One of the principal results of constant superacidity is spasm of the pylorus from the irritation produced by the highly acid juices constantly bathing it. Dilatation commonly follows. There is a sense of weight and oppression in the epigastrium, and the digestion is impaired. Vomiting at night or early in the morning is a characteristic feature in many cases.

(*b*) **Hyperchlorhydria** (Superacidity, Acid Dyspepsia).—In this condition the percentage of HCl is increased during digestion. The condition is generally seen in young neurotic girls and often in connection with chlorosis.

Symptoms.—Pain of a burning character, or a sensation of weight and pressure, is the most common and most characteristic symptom. It occurs, as a rule, from one to three hours after the ingestion of food. Acid eructations generally occur, and sometimes vomiting. There is also tenderness in the pit of the stomach, as in ulcer, a disease in which superacidity is commonly present, but not as a neurosis. The patient is often aroused from sleep by the distress or by the sensation of hunger, which is almost constantly present. Temporary relief is generally afforded by vomiting or the ingestion of food, particularly by animal food.

Treatment.—In superacidity or hyperchlorhydria the patient should be placed on an exclusively milk diet for the first few days. Then beef, fish, eggs, and dry toast may be added to the list. Alcoholic drinks should be excluded, and the use of tobacco would better be abandoned. Relief from the pain and eructations is afforded by sodium bicarbonate, magnesium carbonate, and other alkalis. They should not be administered, however, until the distress is felt. The compound spirit of sulphuric ether and chloroform-water give temporary relief. Atropin has been employed to reduce the quantity of secretion.

(*c*) **Hypochlorhydria** (Subacidity).—In this condition the quantity of HCl is reduced below 0.14 per cent. It is generally believed to result from deficient innervation.

Symptoms.—The symptoms are those of chronic gastritis, without, however, an arrest of the secretion of pepsin and rennin or the excessive secretion of mucus.

Treatment.—The normal secretion can generally be restored by the administration of bitter tonics, as the compound tincture of gentian, with the tinctures of nux vomica and capsicum, immediately after meals. Dilute hydrochloric acid is also beneficial. Some cases recover more promptly on the administration of an alkaline solution, as sodium bicarbonate, gr. xx (1.30) in a half-pint of hot water twenty minutes before each meal, and the dilute hydrochloric acid, gtt. xx in water immediately after the meal. The diet should consist chiefly of farinaceous food, as well-cooked cereals, and very little meat.

(*d*) **Achylia Gastrica** (Nervous Anacidity).—A condition in which the gastric secretion is permanently absent. As a true neurosis it is usually due to some reflex irritation, as that of eyestrain in asymmetrical astigmatism. The term is commonly applied, however, to conditions in which the gastric juice fails of secretion on account of atrophy of the peptic glands, as in some cases of chronic gastritis, a condition in which it is not truly a neurosis, but depends upon anatomical lesions. The

absence of HCl in cancer and sometimes in tabes is regarded as a neurosis.

Symptoms.—In many cases there are no subjective symptoms, and the condition may be first recognized on chemical examination of the stomach-contents. In other cases there is dilatation with its accompanying symptoms, or in the absence of dilatation there may be flatulency, eructation, hiccough, gastralgia, nausea, and vomiting. Intestinal indigestion may also be present, with diarrhea, anemia, and nervousness. When the motor activity of the stomach remains normal and the intestinal digestion active, the condition may persist for years without greatly impairing the health or producing emaciation.

Diagnosis.—After the usual test-breakfast, the stomach-contents show an entire absence of HCl, the ferments and proteoses. The salivary digestion is sometimes found to have progressed in the stomach. The administration of HCl with the test-meal of meat is not followed by evidence of digestion, since no pepsin is secreted.

Treatment.—The diet should consist chiefly of vegetables and starchy food. They should be well cooked and thoroughly masticated, or artificially divided into small fragments. Well-hashed sweetbreads, raw oysters, and chicken may be occasionally allowed in small quantities. A large quantity of food should be eaten in order to maintain the nourishment of the patient, since he depends solely upon intestinal digestion. The bitter tonics and hydrochloric acid may be of benefit. An active pepsin may also be given. Lavage with a weak salt solution, followed by faradization, has been recommended.

2. NEUROSES OF MOTION.

(*a*) **Nervous Eructations.**—As a pure neurosis, eructation, or the belching of air, is common in hysterical and neurasthenic women and children. An entire family is sometimes affected through imitation. The eructations generally occur in rapid succession and produce a loud noise. They are usually maintained by the involuntary swallowing of air, which has been attributed to a spasm of the esophagus. The condition is commonly associated with the other neuroses and with many pathological conditions of the stomach, especially conditions in which the stomach does not discharge its contents regularly into the duodenum. A spasm of the pylorus and patulousness of the cardia are supposed to exist in some cases.

(*b*) **Pneumatosis** is a condition in which the stomach becomes distended with gas, which, owing to weakness of its muscular coat, it is not able to discharge. The gas is usually derived from the abnormal fermentation of food or it may be regurgitated from the intestine. It usually consists of carbon dioxid, nitrogen, and hydrogen; but other gases may be present.

Treatment of Eructation and Pneumatosis.—Remedies are usually administered with a view to hastening the expulsion of the gas as well as to prevent its formation. Turpentine, aromatic spirit of ammonia, chloroform-water, sodium bicarbonate, magnesia, and spirit of peppermint are chief among the former class; dilute hydrochloric acid and tincture of nux vomica among the latter. The diet should be for a time

restricted to easily digested articles. Pneumatosis may be temporarily relieved by the application of turpentine stupes.

(*c*) **Pyrosis** (Regurgitation, Heartburn).—This affection occurs much less frequently as a pure neurosis than as a result of chronic gastritis, superacidity, or dilatation of the stomach, or obstruction of the esophagus. Treatment is directed to the general nervous state or the underlying pathological condition.

(*d*) **Rumination (merycismus)** is a rare form of regurgitation in which the food is voluntarily raised into the mouth and subjected to a second mastication, as is customary with some of the lower animals. It is usually seen in hysterical, idiotic, or epileptic subjects. It may be associated with achylia gastrica, as in the case recorded by Einhorn, or with other neuroses. The treatment is that of the nervous condition, together with moral suasion.

(*e*) **Nervous vomiting** sometimes occurs in hysterical or neurasthenic persons, independently of pathological lesion or abnormal gastric contents. The source of the nervous impulse may be found in a displaced uterus, prolapsed kidney, or localized inflammation; and it may result from injury. It is seen also in connection with nervous diseases, notably locomotor ataxia. It is commonly observed in hysterical women, or it may follow nervous strain or mental emotion. The initial vomiting of the acute infectious diseases is probably of this nature. The vomiting is not usually preceded by nausea or accompanied with gagging or much apparent effort. Pallor and faintness may be present or feigned at the beginning of the attack. The attacks recur at regular intervals and sometimes persist almost without interruption for several days.

(*f*) **Supermotility (hyperkinesis)** is an increased peristaltic activity of the stomach, the chief manifestation of which is a premature discharge of the contents into the intestine. Although it is believed to be primary and of nervous origin in some cases, it is generally a sequence of supersecretion or superacidity. No definite symptoms are produced.

(*g*) **Peristaltic unrest** is a name given by Kussmaul to a not infrequent form of supermotility manifested in loud gurgling or rumbling noises (borborygmi). The peristalsis is usually excited by taking food, but may be induced by the emotions. The patient can often feel the peristaltic movements. In some cases the intestine participates in the increased activity, and occasionally the peristalsis is reversed.

(*h*) **Spasm of the Cardia.**—This sometimes results from such slight irritation as a cold drink or from the passage of a sound in a hysterical person. It may be periodic in occurrence, or it may persist for several days. Pneumatosis sometimes accompanies the tonic spasm.

(*i*) **Relaxation of the cardia** is occasionally met with in connection with regurgitation, eructation, and rumination.

(*j*) **Spasm of the pylorus** may be caused by reflex influences or it may result from superacidity, ulcer, or gastric catarrh. When persistent, it occasions retention of food, fermentation, and dilitation of the stomach.

(*k*) **Relaxation of the pylorus,** or pyloric incontinence, permits the too early escape of the gastric contents into the intestine. The food is sometimes passed into the duodenum immediately after entering the stomach.

(*f*) **Atony of the Stomach.**—This condition, which signifies a relaxed or enfeebled condition of the muscular coat of the stomach-wall, occurs as a neurosis in debilitated or neurasthenic conditions of the general system. It is more frequently a result of organic disease, produced by habitual overdistention with food or drink, or a general wasting of the tissues of the body from chronic disease. It sometimes occurs also in connection with the acute infections, especially typhoid fever. The symptoms are generally those of dilatation, especially eructations and a feeling of distention and weight.

Treatment of Motor Neuroses.—In addition to the general treatment to be considered, little is usually required. When the activity is too great, the diet must be of the mildest kind, free from irritating or stimulating properties; but deficient motility calls for highly seasoned food and preferably a meat diet, with the administration of bitter tonics. The spasm and persistent vomiting sometimes require careful treatment, abstinence from food for a day, followed by the administration of small quantities at short intervals and the treatment recommended for the vomiting in gastric ulcer. The treatment of atony is practically the same as that for dilatation of the stomach.

3. NEUROSES OF SENSATION.

(*a*) **Hyperesthesia.**—In this condition the patient complains of more or less distress in the stomach, with a burning pain in the epigastrium after the ingestion of food or drink. In all other respects the function of the stomach is normally performed. A colored fluid taken as medicine may not occasion discomfort. The affection is met with in the same class of neurasthenic or hysterical individuals, mostly women, who are the subjects of the other neuroses. It may follow a violent emotion, as fright or anger, shock, or a severe illness, as the influenza or an infectious fever. The gastric irritability may be so extreme that the patient becomes greatly emaciated.

(*b*) **Gastralgia,** or gastrodynia, may occur as a pure neurosis, independent of organic disease or the ingestion of food, and often seizing the patient at night. In other cases it is associated with other neurotic manifestations. They sometimes accompany menstrual disorders, possibly at puberty, but more particularly at the menopause. It is sometimes associated with superacidity or supersecretion and may occur in neurasthenic men. Malaria is regarded by some writers as a possible cause. The gastric crises occurring in the course of nervous diseases, notably in locomotor ataxia, belong to this group of affections.

Symptoms.—The pain is often excruciating. It is generally most severe in the epigastrium, but often radiates to the shoulders, sides, and back, resembling the pain of ulcer. It is usually independent of the ingestion of food, and often recurs at more or less definite intervals, frequently awakening the patient from sleep. Tenderness may be elicited on deep pressure, but moderate pressure affords relief. Eructations, restlessness, and other nervous or hysterical manifestations commonly accompany the attack. Vomiting seldom occurs, and the ingestion of food may give relief, as in hyperchlorhydria.

Diagnosis.—The diagnosis is often difficult, especially at the first ex-

amination of a patient not previously known. The seizure must be differentiated from gastric ulcer and cancer, bilious and renal colic, and angina pectoris. *Ulcer* is excluded by the greater periodicity of the attacks, perhaps by the absence of anemia. Hematemesis is not present; the pain radiates over a wider area, and the taking of food relieves, rather than aggravates, it. *Cancer* usually occurs at a more advanced age, the pain does not radiate so widely, and the symptoms are not so constant. The presence of a cachexia favors cancer. In *hepatic colic* the pain is even more extreme, and it is confined to the region of the gall-bladder, where tenderness is also found. The presence of jaundice and clay-colored stools is highly significant. In *renal colic* the pain is lower in the abdomen and radiates along the ureter to the bladder or thigh. The urine is highly acid and often contains sand or gravel. In *angina pectoris* the pain is in the precordial region, often radiating to the left arm. There is marked dyspnea with a sense of suffocation and impending death.

Treatment.—If the attack comes on soon after the ingestion of a full meal, an emetic may afford relief; if during the night, it calls for the application of hot fomentations, the hot-water bottle, or a mustard-leaf, and internally chloroform, ℥xv (1.0), or the compound spirit of sulphuric ether. Morphin must sometimes be given hypodermically, but it should be avoided if possible, for this class of patients are especially susceptible to the habit. Atropin should be combined with it. It is sometimes necessary to keep the patient on a milk diet for several weeks, with the administration of the general remedies for the neurotic condition in order to overcome the tendency to the attacks.

(*c*) **Anorexia,** or the absence of appetite, is associated with so many conditions, as a neurosis or otherwise, that it requires no special consideration. It is a common result of febrile disease, but it is also met with in hysteria and neurasthenia. The administration of the bitter tinctures, or strychnin and arsenic, is generally beneficial.

(*d*) **Bulimia, hyperorexia,** and **polyphagia** are terms applied to a ravenous appetite or constant sense of hunger; and **akoria** signifies an absence of the sense of satiety after a full meal. The conditions may be constant, but usually occur periodically. They are seen especially in connection with the psychoses, sometimes in hysteria, epilepsy, idiocy, cerebral tumors, and occasionally in exophthalmic goiter. They sometimes occur during pregnancy, and may be associated with superacidity. Hunger may continue after a full meal, or the patient may be awakened during the night with a sense of faintness from hunger. A similar exaggeration of appetite often accompanies convalescence from prolonged illness, especially typhoid fever, and it is a common symptom of early diabetes.

(*e*) **Parorexia** signifies a perverted appetite. It is more commonly met with in the insane, sometimes in the hysterical, occasionally in pregnant women. Such articles as chalk, slate-pencils, clay, salt, and baking-soda are commonly craved. This condition is sometimes called pica. When the patient loathes ordinary food and craves highly seasoned articles, pickles and spices, it is sometimes spoken of as malacia.

General Treatment of Gastric Neuroses.—The treatment of the general physical and mental condition of the patient is more important than

that of the gastric disturbance itself. The occupation and habits may be at fault. Many cases are observed in individuals occupied so many hours during the day that they have no time for rest and recuperation; other cases can be attributed to a too ardent devotion to society. In either condition a change to outdoor exercise, athletic sports, and games often produces immediate improvement. A vacation of a month or two in the country, or a sea-voyage, is all that is required. Complete rest of mind, with bodily exercise, is generally best. The diet should be regulated according to the special condition as already noted under the different forms of neuroses. In the conditions that lead to dilatation or atony of the gastric walls, stimulating food and bitter tonics should be prescribed. The nitrate of silver is often useful, and lavage may prove beneficial. The anemia which is often present calls for iron, arsenic, and nutritious, easily digestible food, with exercise in the open air. Constipation is common and should be met with laxatives. When intestinal indigestion accompanies the gastric, as it often does, it may be necessary to regulate the diet still more rigidly and to administer intestinal antiseptics. Cases in which insomnia proves troublesome require the bromids or a narcotic, as trional, gr. xv (1.0), at night. The Weir Mitchell treatment—confining the patient to bed on a graduated milk diet, with massage, for a month or six weeks—is often a most successful method in hysterical cases.

DISEASES OF THE INTESTINES.

ACUTE CATARRHAL ENTERITIS.

DIARRHEA, ACUTE INTESTINAL CATARRH, ILEOCOLITIS.

Definition.—An acute catarrhal inflammation involving a greater or less portion of the intestinal tract. Although the disease is perhaps limited in some instances to a single region of the intestine, such distinctions as duodenitis, jejunitis, and ileitis, or other anatomical classification is not practicable.

Etiology.—The disease is a very common one, affecting individuals of any age, but particularly infants and children. It may be primary in character, or it may occur secondarily in the course of other affections. A tendency to recurrence develops as a result of repeated attacks. Personal idiosyncrasy is also an important factor, and in many persons certain articles of food invariably produce diarrhea. 1. The most frequent causes of *primary* enteritis are: (*a*) Errors in diet; the eating of food that is improper in quality or excessive in quantity. Unripe and overripe fruit, decomposed meats, sour milk, or food containing organic or mineral poisons, especially canned meats and fruits, are common sources of offense.

(*b*) Impure drinking-water is an occasional cause of epidemic outbreaks. Mere change of drinking-water necessitated in travel often produces diarrhea.

(*c*) Toxic substances. The free or prolonged use of such drugs as arsenic, acids, alkalis, mercury, iodids, antimony, or colchicum and other vegetable preparations may induce the disease.

(*d*) Alterations in the chemical composition of the intestinal secretion, as through the absence of the pancreatic juice, an increase or decrease in the quantity of bile. Arrest of the pancreatic secretion usually produces fatty diarrhea.

(*e*) A common cause, affecting especially certain individuals, is chilling of the surface of the body, particularly of the abdomen, through change of weather, exposure to cold and wet, or sleeping in a draft.

(*f*) A nervous origin may be traced in many cases, especially when the attack follows fright or other strong emotion; such attacks are occasionally met with in students under the strain of an impending examination. (See Nervous Diarrhea, p. 508.)

2. The usual causes of *secondary* enteritis are: (*a*) Conditions of the stomach which cause the passage of undigested or fermenting food into the intestine.

(*b*) Various acute infections, as typhoid fever, malaria, dysentery, cholera, tuberculosis, and sepsis, affecting the bowel through structural changes or the production of toxins.

(*c*) The extension of inflammation to the bowel from adjacent structures as in peritonitis of different forms, or from a focus of ulceration, malignant disease, hernia, or other strangulation.

(*d*) Disturbance of the circulation such as results from chronic disease of the heart or lungs, but more particularly of the portal circulation in hepatic cirrhosis, or through the pressure of tumors on the portal or mesenteric veins.

(*e*) A secondary diarrhea sometimes follows extensive burns. Diarrhea is often produced also by influences which merely hasten the peristalsis.

Morbid Anatomy.—The intestine during life, as occasionally seen at abdominal section, appears red and swollen, and the secretion of mucus is increased; but the evidences of hyperemia usually disappear after death. The solitary and agminated follicles are often enlarged, and they may show a variable degree of ulceration, particularly in children. Desquamation of the surface epithelium is seen, but probably as a post-mortem change.

Symptoms.—Diarrhea is the most characteristic symptom, yet it is not invariably present. The onset of the disease is frequently announced within an hour or two after the ingestion of improper food. The individual may be suddenly aroused from sleep by an intense colic or tenesmus, sometimes accompanied with cramping of the abdominal or leg muscles. Gurgling or rumbling noises (borborygmi) are heard in the abdomen. The pain recurs at short intervals until there is an imperative desire to evacuate the bowel. When the pain is severe and when the irritation affects also the stomach, nausea and vomiting precede or accompany the diarrhea. The abdomen is generally tympanitic. The pain is often so intense that the patient bends over, presses a pillow against the abdomen, or rolls from side to side in efforts to obtain relief. It is usually intermittent and relieved by the evacuation of the bowel. Thirst is extreme, and the gratification of it induces or prolongs the vomiting. Fever is uncommon, but there may be a slight elevation of temperature, particularly in children. With profuse diarrhea the temperature may become slightly subnormal. The stools vary greatly in fre-

quency, appearance, and consistence. They are sometimes thin as water and contain mucus or undigested food. When food appears in them, the condition is called lienteric diarrhea. The attack usually lasts only a day or two, but it may be prolonged for a week or longer.

Diagnosis.—It is important, with reference to treatment, to determine whether the disease is situated in the large or small intestine. When the *small bowel* is alone affected, the diarrhea is not usually so profuse, but the pain is more persistent and of a colicky character; there may be gurgling, but seldom borborygmi. The stools are more watery, yellowish, green or grayish in color, and often lienteric. They do not, as a rule, contain mucus. When the *large bowel* alone is affected, pain may be absent, or it precedes and accompanies the dejection. The occurrence of tenesmus indicates an involvement of the lower portion of the bowel. The stools are of uniform consistence and they usually contain mucus, either in flakes or in large masses. Involvement of the *duodenum* is to be inferred only when the attack follows an acute gastritis or when jaundice develops as a result of the extension of the inflammation into the common bile-duct.

Cholera nostras is distinguished by the more profuse watery discharges and greater prostration which attend it.

Prognosis.—Primary cases usually recover promptly, but secondary cases, especially when due to tuberculosis or cachectic conditions, generally pass into a chronic form.

Treatment.—Rest is all that is required in most cases. The patient should remain in bed and abstain from food or take only milk with lime-water or carbonated water in small quantities for 24 to 48 hours. If the pains are extremely severe, a hypodermic dose of morphin may be given. It is not well to check the diarrhea until the intestine has been thoroughly evacuated. If only a few small dejections have taken place, calomel, gr. iij (0.2), castor oil, ℥ j (30.0), or a bottle of effervescent magnesium citrate should be given. This may be followed with a few doses of bismuth, gr. x (0.65), and opium, gr. ¼ (0.016), or the camphorated tincture, ʒ j (3.5). The thirst should be relieved with chipped ice.

The secondary diarrhea is often difficult of treatment, especially when it is due to tuberculosis or other chronic disease. (See Tuberculosis of the Intestine.) When the disease is confined to the large bowel, a copious enema checks it immediately in most cases. Opiates are not usually required, but, for the relief of pain, starch-water, ℥ ij (60.0), containing tr. opii, ♏xx (1.2), may be injected into the rectum.

CHRONIC CATARRHAL ENTERITIS.

CHRONIC DIARRHEA, CHRONIC INTESTINAL CATARRH, CHRONIC COLITIS.

Definition.—Chronic catarrhal inflammation of a greater or less portion of the small and large intestine.

Etiology.—The disease often results from repeated attacks or from a single prolonged attack of the acute form of enteritis; all the causes which are operative in that form are, therefore, indirectly the causes of this. In most cases, however, the disease is directly due to other chronic disease, as tuberculosis, malaria, cirrhosis or passive congestion of the

liver. Tubercular or cancerous ulceration of the bowel is attended with chronic diarrhea. The disease sometimes develops also in the course of chronic nephritis, diabetes, or gout. Damp dwellings and prolonged exposure, with insufficient nourishment, as in camps and prisons, are common causes.

Morbid Anatomy.—When the disease has lasted only a comparatively short time, the changes may be slight, but later there are usually found hyperemia, ecchymosis, thickening of the mucous membrane, and very often a more or less extensive ulceration affecting especially the follicles. The intestine may be uniformly or irregularly dilated or contracted, and fecal accumulations are sometimes found. The great thickening of the mucous membrane sometimes produces stenosis; and the same result may follow the cicatrization of ulcers. The mucous membrane has usually a reddish brown or grayish color; occasionally it is black, or the pigment may be deposited in the tops of the villi, around the circumference or in the center of the follicles. When in the last of these locations, the mucous membrane has the so-called shaved-beard appearance. The lesions are usually most pronounced in the lower portion of the ileum and in the colon. The increased activity of the secreting glands, together with the ulceration, produces an increased quantity of fluid in the intestine, which is usually of a thinner consistence than normal and often serous, sanguinolent, or purulent in character.

Symptoms.—Diarrhea is the important symptom, but it frequently alternates with constipation. As a rule, from one to six or eight thin dejections occur in 24 hours; frequently they occur only in the morning. They may be large or small in quantity, semisolid or watery, sometimes containing undigested food and varying in color from a pale yellow, green, or white to reddish brown or black. Pain and borborygmi, with abdominal distention, tenderness, and flatulence, are frequent accompaniments. If the stomach be also involved, there are anorexia, pyrosis, a disagreeable taste, and the tongue is heavily coated or red, glazed, and fissured. The patient often becomes extremely emaciated, marasmic, weak, and hypochondriacal or melancholic. Fever usually develops toward the close of a fatal case.

Diagnosis.—The recognition of the disease is easy, but the determination of the part of the bowel that is affected may be quite difficult, in part because the disease is seldom limited to a single region. Affection of the duodenum is probable when there is jaundice, and when the stools are fatty or have a clay color. In affection of the *jejunum* and *ileum* there is usually pain at the umbilicus a few hours after taking food, but pain may be entirely absent; the diagnosis is never sure. *Colitis* is characterized by large stools and severe pain, often just before a movement. Catarrh of the rectum (*proctitis*) is generally indicated by frequent painful dejections and a constant desire for evacuation. Ulceration is indicated by extreme localized tenderness and the discharge of pus or blood or of portions of the mucous membrane.

Prognosis.—The disease is persistent, and it often proves fatal in children or aged and debilitated subjects. When not promptly treated, it may last for months or years. Death usually results from exhaustion, intercurrent bronchitis, pneumonia, peritonitis, thrombosis of the cerebral sinuses, or other disease. Relapse is common after apparent recovery.

Treatment.—Dietetic and hygienic measures are all-important. In many cases an absolute milk diet, preferably predigested, for a few weeks is advisable. This may be followed with eggs and articles made with milk, eggs, and corn-starch, rare beef, junket, toast, and rice; but an excess of starch must be avoided. Fats, sweet fruits, pastry, and alcoholic beverages should be avoided. Light exercise in the open air and an abundance of sunshine, with rest from mental worriment, are important. These patients should be warmly clad, and a flannel abdominal band is often beneficial. Pancreatin, gr. v (0.32), should be given with the meals, and salol, gr. v to x (0.32—0.65), or bismuth subgallate, gr. xx (1.30), two or three hours after. Calomel or castor oil should be occasionally administered to cleanse the bowel, especially when there are intervals of constipation. Silver nitrate, gr. ¼, in keratin-coated pills, is often beneficial in persistent cases. When the disease is located in the descending colon, a daily irrigation with a warm salt or alum solution (1 per cent) or silver-nitrate solution (0.5 per cent) is often curative. Codliver oil, iron, and arsenic may be given for the anemia and emaciation. Opium should be avoided except in a hopeless case.

CHOLERA MORBUS.

CHOLERA NOSTRAS.

Definition.—An acute catarrh of the stomach and intestines in adults, presenting symptoms resembling in severity those of Asiatic cholera.

Etiology.—The disease is probably due to a specific microbe which has not yet been isolated. It occurs especially during the late summer, when the days are excessively hot and the nights cold. It affects commonly young adults, though it may attack persons of any age. It is generally attributed to errors in diet of the same character as cause acute catarrhal enteritis, but epidemic outbreaks are not unusual.

Morbid Anatomy.—The lesions are those of acute gastritis combined with those of acute enteritis; a catarrh of the entire intestinal tube. The walls are swollen and edematous, the follicles enlarged and abnormally active. In severe cases the appearances are much the same as in cholera, except that the comma bacillus is not found.

Symptoms.—The onset is usually sudden, but in some instances it is preceded by malaise, loss of appetite, and nausea. The attack frequently begins at night with severe abdominal pain or tension, nausea, and repeated vomiting. After the food has been ejected, the vomit consists of thin, serous, often bile-stained fluid. Purging develops either with it or shortly afterward. The stools are at first feculent, but later become watery, almost odorless and colorless, and often assume the typical rice-water appearance. Both the vomiting and purging are often violent and almost incessant. Severe abdominal pains follow each spell of vomiting and purging, and cramps and twitching of the muscles of the calves often occur. There is intense thirst, scant, sometimes albuminous urine or anuria. The skin becomes cold, cyanotic, and clammy; the emaciation is often so rapid that within a day or two the eyes become sunken, the cheeks hollow, and the nose pinched. The patient appears to shrivel and waste away as in true cholera. All these changes, includ-

ing the muscular cramps, are attributed to the withdrawal of water from the blood and tissues. Fever is seldom present and it rarely exceeds a degree or two. In severe cases the patient sinks into a collapse; the pulse becomes extremely feeble and rapid, the voice weak, and the respiration sighing, but the mind often remains clear. Death may occur within the first forty-eight hours, or after three or four days, from exhaustion. In most cases, however, the attack lasts only one or two days, then subsides, leaving the patient greatly prostrated, but recovery is complete within a week or two.

Diagnosis.—In the absence of Asiatic cholera, the disease is readily recognized through the severity of its symptoms. During a cholera epidemic, however, cases of this character are often mistaken for that disease. They are to be excluded only by the absence of the cholera spirillum from the stools.

Prognosis.—The mortality is trifling in previously healthy persons. In the very young or very aged, and in those debilitated by previous disease, however, it is often fatal.

Treatment.—Morphin should be promptly administered hypodermically, and hot applications should be made to the abdomen and extremities. A mixture containing morphin, chloroform, ether, and spirit of peppermint ("chlorodin") is much employed internally. Stimulants, especially brandy and strychnin, are indicated. The thirst may be relieved with fragments of ice, and by the injection of salt solution into the bowel or subcutaneously. Milk with lime-water should be the only diet for several days.

ENTERITIS IN CHILDREN.

ACUTE GASTROENTERITIS, SUMMER DIARRHEA, ACUTE DYSPEPTIC DIARRHEA.

Etiology.—This is the common form of summer diarrhea and is particularly frequent in bottle-fed infants. It is especially liable to occur in those raised at the breast, about the time of weaning. It is often seen also at the time of the irruption of the teeth, although it is denied by some writers that this normal process can be in any degree responsible for a pathological one. As a rule, the disease follows directly upon a palpable error in the feeding of the infant, as that of nursing too much or too often, or the mother's milk may be unfit on account of ill-health, intemperance, anger, or other emotions. Bottle-fed infants are often given food that is unsuitable for their age and digestive power, or the food is given too often, in too great quantity, at an improper temperature, or after it has soured in the bottle. All these influences and many more contribute to the production of the disease. A feeble constitution, rickets, faulty hygiene, including overcrowding, lack of air and sunshine, filth, exposure to cold and wet, and neglect, exert an important influence in many cases. In older children the attack often follows the eating of unripe fruit.

In many cases, if not in all, the disease is directly due to the presence of bacteria or their chemical products in the food. The studies of Messrs. Duval and Bassett in 1902 indicate that the disease is probably due in most cases to the Bacillus dysenteriæ of Shiga. This organism

found in the stools of more than forty cases, in large numbers in the acute and less numerously in mild or chronic cases. They found also that the blood of the infants affected with the disease possesses an agglutinative power over this bacillus.

Morbid Anatomy. The lesions found after death are limited to hyperemia with swelling and increased secretion; ulceration does not occur.

Symptoms. The attack may be ushered in with vomiting or diarrhea. In some cases the infant appears restless or gives other evidences of pain for a few hours before the onset, and convulsions occasionally occur. The dejections become rapidly more frequent until six or eight, often twice this number, may occur in twenty-four hours. They are usually large, pasty, and offensive; sometimes they are frothy and sour-smelling. They generally contain undigested milk, and, after the first day or two, they become green. The tongue is coated and dry, and the infant suffers from thirst. There is not usually much fever, but in severe cases the temperature may rise to 104° or 105° F. (40°—40.5° C.). The cases differ in severity to a very great extent. The disease sometimes passes into an ileocolitis of greater or less duration.

Diagnosis. The affection is differentiated from *cholera infantum* chiefly by the character of the stools, which have not the serous, watery quality of those of the latter disease. From *ileocolitis* it is distinguished by the absence of mucus and blood from the dejections.

The *prognosis* is usually favorable except in feeble infants or those debilitated through neglect, improper food, or previous disease. The attack lasts from three to seven days, but relapses are common.

Treatment. The promptest relief is often afforded by thorough irrigation of the stomach, or of both stomach and bowel, with lukewarm water. The most important item in other respects is the adoption of a suitable diet. In young bottle-fed infants this is sometimes difficult. Pure cow's milk, properly diluted for the age, is safest, unless a suitable wet-nurse can be obtained. When the stools are offensive, albuminous food should be withheld, and carbohydrates in the form of dextrin may be added to the milk; when the stools are frothy and sour, showing fermentation, the carbohydrates must be withheld, and nitrogenous food, as koumyss, or albumen water may be given. The limiting of the food to albumen water, with a teaspoonful of brandy to each eight ounces, is often necessary for a few days. As soon as the diarrhea subsides, milk and broth may be allowed. Great care must be exercised in the administration of water, since an excess keeps up the vomiting, and thirst is [illegible]

The hygienic management of the case is equally important. The patient should be kept in a cool, well-ventilated room or removed from the close apartments of the tenement-house, where most cases occur. It should be kept constantly in the open air, day and night, except perhaps during the heat of the afternoon, but it must not be carried in [illegible] If circumstances permit, it should be taken to the country.

In the beginning a purgative dose of calomel or castor oil is indicated, and this, with regulation of the diet, is generally [illegible] required. The diarrhea usually ceases spontaneously in twenty-four hours; otherwise, or if it be severe, bismuth in large [illegible] every three hours, with camphorated tincture of

opium according to age, may be given. Intestinal antiseptics, particularly salol, resorcin, naphthalin, and sodium salicylate, are recommended by some writers.

CHOLERA INFANTUM.

Definition.—A severe, acute gastrointestinal catarrh, probably of bacterial origin, and characterized by a profuse serous diarrhea with extreme prostration and rapid loss of weight.

Etiology.—The disease is not a frequent one, and there can be little doubt that it is due to the action of micro-organisms, if not to a specific microbe. The investigations of Duval and Bennett in this connection will doubtless prove of much value. The disease commonly occurs in the same class of improperly fed, poorly nourished infants and under the same faulty hygienic conditions as have been referred to in the etiology of gastroenteritis. It seldom develops in healthy infants under normal conditions.

Symptoms.—In most cases the actual onset of the disease is preceded by a short attack of gastroenteritis or ileocolitis, with pain and restlessness. In other cases there is sudden prostration, with gradual rise of temperature for a few hours, during which time the infant may give evidence of nausea and profound illness. Vomiting and purging then set in with violence, and recur at short intervals. After the stomach contents have been discharged, the vomitus consists of serum and mucus, which soon becomes tinged with bile. All food and drink are generally immediately ejected. The stools are large and watery; at first pale green or yellow and offensive, they soon lose their color and odor and consist almost entirely of serum. They may recur at intervals of a half-hour or less. They are at first acid in reaction, but later often become alkaline. The microscope reveals in them much epithelium and numerous bacteria. The child becomes rapidly emaciated, pale, and bloodless in appearance, the anterior fontanel is depressed, and the abdomen is retracted. The tongue is at first coated, but it becomes red and dry, and the thirst is extreme. The urine is scant and not infrequently suppressed. The rectal temperature is elevated, but the surface is often cold. The infant ceases to cry, through weakness, or its efforts are inaudible. Collapse usually develops early. The restlessness of the early stage gives way to a semicomatose condition, in which the child lies with half-open eyes, breathing rapidly, often irregularly, with little remaining evidence of life. The Cheyne-Stokes respiration sometimes develops, and there may be retraction of the head as in hydrocephalus ("hydrencephaloid"). Convulsions sometimes occur, or a condition of tetany may develop. The temperature often becomes very high shortly before death, sometimes reaching 106° or 108° F. (41°—42° C.). When recovery is about to occur, the vomiting ceases and the stools become less frequent, finally acquiring their normal consistence and color. Convalescence is usually slow.

Diagnosis.—The diagnosis is not difficult, but the distinction between cholera infantum and gastroenteritis is perhaps too often neglected. The distinctive features of the former disease are the constant vomiting, profuse serous dejections, thirst, high fever, profound prostration, and

collapse. It is the severest and most rapidly fatal form of intestinal disease in infants.

Prognosis.—Fully two-thirds of the cases are fatal, regardless of the condition of the patient or the treatment adopted. The disease is naturally most fatal in debilitated, unhealthy children, but a great deal depends upon the severity of the attack. Death or recovery may occur in the first two or three days, and the initial symptoms generally reveal the prognosis.

Treatment.—Prompt measures are demanded. Excellent results are often obtained from thorough irrigation of the stomach and large bowel. Since nothing can be retained in the stomach, hypodermic medication should be resorted to. Morphin, gr. 1-100 (0.0006), and atropin, gr. 1-800 (0.00008), may be given to an infant, and repeated in an hour or two if vomiting and purging do not cease. The result is often remarkable, but the action of the morphin must be carefully watched until the tolerance of the little patient has been determined. It must not be administered in a case in which the purging has ceased, when the child is in a stupor, or when there is evidence of cerebral irritation. As soon as the irritability of the stomach has been relieved, mercury with chalk, gr. ss (0.03), and Dover's powder, gr. 1-10 (0.006), or bismuth salicylate may be given, but in many cases it is better to refrain from any medication. The temperature should be reduced by cool sponging or the graduated bath. Ice-water enemata or irrigation of the large bowel reduces the temperature and relieves the thirst. The subcutaneous injection of the saline solution, as practiced in cholera, is often of great benefit in replacing the water abstracted by the disease. Iced brandy or champagne in quantities of 10 or 15 drops every hour should alone be given internally. If vomiting be persistent, brandy, ether, or spirit of camphor may be administered hypodermically. When the surface of the body is cold and when the temperature is subnormal, warm mustard-baths should be given, and hot-water bottles applied to the extremities. After the severe symptoms have been allayed, a more liberal diet, beginning with albumen-water, should be given, and the subsequent management is the same as that of acute gastroenteritis.

ACUTE ENTEROCOLITIS.

ACUTE FOLLICULAR COLITIS, FOLLICULAR DYSENTERY.

Definition. An acute catarrhal, often ulcerative inflammation of the ileum and colon of infants.

Etiology. The disease occurs especially during the hot summer months and in the debilitated children of the poor. It is peculiarly troublesome in the "second summer" of infancy. All the causes which lead to gastroenteritis and cholera infantum are operative in its production, and it is often a sequel to one of these diseases or of one of the acute infections, as diphtheria, scarlet fever, or measles. The resemblance of the disease to acute dysentery, and the recent discovery of the bacillus of the latter disease as a probable cause of gastroenteritis, suggest the possible identity of the two affections, except with reference to the location of their lesions.

Morbid Anatomy.—A catarrhal inflammation, with the characteristic changes already described under Catarrhal Enteritis, is usually found with greatest severity in the colon, and, in addition to these, more or less extensive follicular ulceration.

Symptoms.—Most cases develop as attacks of acute gastroenteritis, and gradually or suddenly merge into enterocolitis. The transition is indicated by an aggravation of the symptoms. Fever usually develops; the stools become smaller and more frequent, and they contain mucus and blood. Convulsions sometimes occur; and in severe cases the dejections become reduced to only blood-stained mucus, generally having a very offensive odor. Tympanites and tenderness, with tenesmus and the passage of much gas, are present in most cases. Death may occur within a few days from toxemia, with convulsions or coma, or, after a week or two, from exhaustion. Other cases pass into a chronic condition, but final recovery is the rule, except in feeble or ill-nourished children.

Treatment.—A few doses of calomel, gr. 1-10 (0.006), or a purgative dose of castor oil should be administered in order to cleanse the upper bowel. The large intestine should be irrigated, once a day or oftener, with warm salt solution through a long elastic catheter introduced as far as possible. The griping pains may be relieved with warm applications to the abdomen, turpentine stupes, hot fomentations, or a spice poultice, and the tenesmus by injecting into the rectum one or two drops of laudanum in a half-ounce of starch-water. The diet for the first day or two should be restricted to albumen-water with a little brandy, then to cream or milk with lime-water and beef-juice. In other respects the treatment is that of acute gastroenteritis.

CELIAC DISEASE.

DIARRHEA ALBA, DIARRHEA CHYLOSA.

This name was applied by Gee to a subacute diarrhea of children between one and five years of age, in which there are large, frothy, pasty, whitish stools. The cause is not known. Intestinal ulceration is sometimes found. The child becomes pale and emaciated, the skin dry, and the abdomen distended, but soft. Fever is generally present, but vomiting is unusual. The disease is very fatal, death occurring after several weeks, from marasmus.

SPRUE OR PSILOSIS.

This is described by Manson as a disease characterized by "irregularly alternating periods of exacerbation and of comparative quiescence; by an inflamed, bare, and eroded condition of the mucous membrane of the tongue and mouth; by flatulent dyspepsia; by pale, copious, and generally loose, frothy, fermenting stools; by wasting and anemia; and by a tendency to relapse. It may occur primarily or it may supervene on other affections of the bowels." It is of slow progress and tends to terminate in atrophy of the intestinal mucous membrane, that usually proves fatal. Musgrave, on the other hand, regards it as only a second-

ary condition, which, like the typhoid state, develops in connection with other diseases, since he failed in careful microscopic examinations to find any additional etiological feature in any of the cases observed by him at Manila.

DIPHTHERITIC ENTERITIS.

PSEUDOMEMBRANOUS OR CROUPOUS ENTERITIS OR COLITIS.

Definition.—An inflammation of the small or large intestine characterized by the formation of fibrinous psuedomembrane within or upon the mucosa.

Etiology.—The disease most frequently appears: (*a*) In connection with the acute infections, as typhoid fever, pneumonia, pyemia, cholera, scarlet fever, or tuberculosis; (*b*) as a result of toxic influences, as from lead, arsenic, or mercury; or (*c*) as a terminal affection in chronic nephritis, cancer, hepatic cirrhosis, or other cachectic conditions. The thrush fungus has also been met with in the colon.

Morbid Anatomy.—The anatomical lesions are not uniform in character. In some cases patches of variable size and thickness are found upon the surface of the colon or involving the entire thickness of its mucous membrane. It is sometimes confined to the cecum. The small intestine is generally markedly involved in toxic cases. In some instances the membrane has a grayish white color like that of true diphtheria, but sometimes it has more the appearance of thick crusts. In yet another group of cases the disease affects especially the solitary follicles, which are enlarged and sometimes suppurating or ulcerated and covered with the diphtheritic formation.

Symptoms.—The symptoms are those of a more or less severe enterocolitis, with griping pain, frequent dejections, prostration, sometimes collapse, varying much with the cause of the condition. In toxic cases the stools are often mucopurulent and contain blood. Tormina and tenesmus are frequently present, as in true dysentery. There are no distinctive symptoms, however, except the discharge of fragments of the membrane, and the diagnosis is often impossible during life.

The ***treatment*** is symptomatic, except so far as it applies to the causative condition.

PHLEGMONOUS ENTERITIS.

Definition.—A localized or diffuse suppurative inflammation of the mucous membrane of the intestine.

Etiology.—The disease is extremely rare as a primary affection. It is most frequently observed after strangulation or intussusception, or as a result of pyemia or of malignant disease of the bowel. The primary cases are supposed to be due to the presence of the colon bacillus. The disease sometimes accompanies phlegmonous gastritis. The principal symptoms are pain, tympanites, tenderness, with constipation and fever. Septic peritonitis, strongly suggested by these symptoms, is generally developed. The disease usually terminates fatally within a few days. The condition is seldom diagnosticated, but is usually confounded with

septic peritonitis or one of the conditions from which it arises. The relief of pain, and other measures for the comfort of the patient, are the only therapeutic indications.

ULCERATIVE ENTERITIS.

The duodenal, typhoid, syphilitic, and tubercular ulcers of the intestine have already been considered. Several other forms may be briefly referred to.

Simple Ulcerative Colitis.—This affection, although of rather doubtful pathology, is worthy of study on account of its common confusion with dysentery. It is not infrequently encountered in men past middle life, especially in those who have suffered much from digestive disorders. Some of the cases that have been described under this head, however, were perhaps cases of either amebic dysentery or of phlegmonous enteritis. The anatomical changes are not constant. In some cases the bowel is much dilated and the mucosa greatly thickened and extensively ulcerated; but in others the lumen of the bowel is diminished. Polypoid projections are sometimes seen on the margins of the ulcers. The patient generally suffers from a lienteric diarrhea or alternating constipation and diarrhea, with frequent dysenteric stools, a gradual loss of strength, and sallowness of the skin amounting to cachexia. Perforation of the bowel frequently occurs, or the condition may pass into a chronic stage of indefinite duration.

Follicular or Catarrhal Ulcers.—Sharply defined ulcers limited to the follicles of either the small or the large intestine, but particularly the colon, are met with, especially in the enterocolitis of children or the chronic dysentery of adults. They are sometimes found at autopsy, also, in connection with other diseases where their presence had not been recognized.

Stercoral ulcers are peculiar to cases of long-standing constipation, being formed in the sacculi of the dilated colon as a result of the pressure and irritation of the hardened fecal masses, some of which often contain calcareous matter.

Traumatic ulcers result from the irritation caused by the lodgment of foreign bodies or from the entrance of corrosive poisons.

Perforative ulcers are produced by the extension of inflammation from without, as by the pressure of a retroverted uterus, new growths, the rupture of an abscess or of a gastric ulcer, or from disease of the appendix, pancreas, or peritoneum.

Solitary ulcer occasionally develops in the cecum or colon and may pass to perforation. It resembles the peptic ulcer of the stomach.

Amyloid, scorbutic, purpuric, and leukemic ulcers of the intestine have been described in connection with the several diseases indicated by these titles.

Symptoms.—The symptoms of ulcer of the intestine vary with the location and extent of the ulceration. Diarrhea is a common symptom, especially of ulcers in the colon, sigmoid flexure, or rectum. Extensive ulceration may exist, particularly in the small intestine, without the production of diarrhea. Morning diarrhea, often limited to two passages in close succession, is highly characteristic of rectal ulcer. The passage

of pus, shreds of tissue, and blood, is the distinctive feature of most cases; but pus alone is not characteristic, unless, perhaps, in the form of small sago-like masses or plugs corresponding in size to the follicles. The blood from an ulcer in the sigmoid flexure or rectum may be bright red and fluid, like that from hemorrhoids, which must be excluded. That from the upper bowel is usually dark and tarry, and it is sometimes mingled with feces. The most profuse hemorrhage occurs as a result of typhoid ulceration or the duodenal ulcer. Pain is not common and it is not distinctive, but in some instances there are localized pain and tenderness directly over the ulcer and due to peritoneal inflammation. Perforation may occur, and it is generally followed by a rapidly fatal, suppurative peritonitis. A localized abscess is sometimes produced.

The ***treatment*** is that of chronic catarrhal enteritis.

HEMORRHAGE OF THE INTESTINE.

Intestinal hemorrhage is a symptom common to a large number of conditions. The bleeding may occur in any part of the intestine, and blood from other sources not infrequently passes through the bowel. It may thus originate in the upper alimentary or respiratory tract or in the stomach, or it may enter the intestine through the rupture of an abscess or an aneurism.

Etiology.—The causes of true intestinal hemorrhage may be general or local in character. 1. The general causes include such blood-states as are ordinarily accompanied with hemorrhage of the mucous membranes, as purpura, pernicious anemia, leukemia, scurvy, and such profound intoxications as are seen in smallpox, yellow fever, malaria, and bubonic plague. It has been noted also as a rare form of vicarious menstruation.

2. The local causes are: (*a*) Hemorrhoids, probably the most frequent source of hemorrhage from the bowel; (*b*) anal fissure or fistula and intestinal polypi; (*c*) all the different forms of ulcer noted under Ulcerative Enteritis; (*d*) hyperemia, especially the passive congestion from cirrhosis of the liver or valvular disease of the heart; (*e*) embolism or thrombosis of the mesenteric vessels, the result of malignant endocarditis, pyemia, or other infection; (*f*) trauma from perforating wounds, blows upon the abdomen, or the passage of foreign bodies; (*g*) neoplasms; (*h*) corrosive poisons; (*i*) intestinal parasites, especially the ankylostomum duodenale.

Symptoms.—The blood may be mingled with the feces or it may pass independently. As previously noted, it is bright red and fluid when from the lower bowel, but dark and tarry when from a higher source, owing to the action upon it of intestinal juices and gases.

Treatment.—The treatment must be directed to the cause of the hemorrhage. This will be considered under the various diseased conditions that have been named as causes.

HEMORRHAGIC INFARCTION OF THE INTESTINE.

This is a rare condition usually caused by embolism of the superior or inferior mesenteric arteries or their larger branches. The remote

cause is generally a disease of the heart, especially malignant endocarditis. The extent of the infarction and the severity of the consequent symptoms depend upon the size of the vessel obstructed. The wall of the intestine becomes intensely edematous, and the mucosa is covered with blood and mucus. Necrosis usually follows, and localized peritonitis may be developed.

The condition is manifested by the sudden development of severe intestinal pain, free hemorrhage from the bowel, tympanites, fall of temperature, and collapse. In some instances the blood is retained in the bowel and there may be obstinate constipation. The recognition of the cardiac condition to which the infarction owes its origin is generally the key to the diagnosis, but the exclusion of hemorrhagic pancreatitis is often extremely difficult or impossible. The disease is generally, if not always, fatal. The treatment is palliative, morphin being given for the pain and ergot for the hemorrhage. Cold applications may afford relief.

AMYLOID DISEASE OF THE INTESTINE.

Amyloid degeneration affects the intestine, along with other structures as a result of prolonged suppuration, especially in tuberculous or syphilitic subjects and when the suppuration involves bone. Both the small and large intestines are generally involved, but the degeneration is most pronounced in the lower ileum and upper colon. The process begins in the smaller blood-vessels and extends to the mucosa, which becomes pale and thickened. In advanced cases the other layers of the intestine may also be involved, and ulceration of the mucosa is common. The principal symptom is a profuse watery diarrhea, without pain. Hemorrhage of the intestine occasionally occurs late in the disease. The recognition of the condition is possible, as a rule, only through the association of other amyloid disease and the peculiar waxy appearance of the patient, together with the discovery of the suppurative focus back of the trouble. Intestinal amyloid disease is generally a late manifestation and one that signifies an early fatal termination. The treatment is purely symptomatic, directed to the relief of the diarrhea or hemorrhage and the support of the patient's strength.

APPENDICITIS.

The appendix vermiformis is an offshoot from the cecum, which ceases to undergo further development at an early period of fetal life and remains a "wormlike process," functionless so far as we know. Its usual length is about 3 inches (8 cm.), but it has been found less than 1 inch and more than 9 inches (23 cm.) in length. Its diameter is about ¼ inch (0.5 cm.), but in this also it varies. It is provided with a mucous membrane rich in lymphatic tissue, with numerous follicles of Lieberkuehn. It is partly surrounded by peritoneum. On account of its glandular structure it has been called the "abdominal tonsil" by Ransohoff and others. It has also a mesoappendix, slightly shorter than itself and bearing the same relation to it as the mesocolon bears to the colon. The distal end may be found in any position with reference to the cecum. Most frequently it points inward from the left border of the cecum, crossing the psoas muscle. It is often behind the cecum and may hang down toward or into the true pelvis. The lumen of the appendix communicates with the intestine through an orifice that is sometimes as small as a pinhole, sometimes large enough to admit a No. 7 English sound.

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plete the canal is often closed at the
e appendix becomes thickened to the
tuting a cyst, the contents of which
. Or the distention may be due to
ntaining desquamated epithelium, leu-
débris. The lumen is sometimes divided
ough the formation of cicatricial bands.
pass on to suppuration and perforation.
distention is not great, the obliterative
rigidity of the thickened wall. Repeated
anifestations are a common result. In
peritoneal covering may become involved,
n nor perforation has occurred. It is then
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on may result from the presence of concre-
pus-forming micro-organisms, and sometimes
the typhoid or tubercle bacillus. Actinomy-
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e catarrhal. Single or multiple ulcers may be
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at any part of the tube. As the inflammatory
e, an adhesive peritonitis is developed, uniting
op of the intestine, the bladder, right kidney,
parietes, or any surface with which it may come in
ndix is of unusual length, it may become adherent
the gall-bladder, or other remote structure. But
may thus be prevented for a time, a suppurative
developed. In a large percentage of cases the
lete and permit the escape of the contents into the
ith production of a general peritonitis. In some
l abscess is formed; its most frequent location is
umbilicus and the anterior superior spinous proc-
ation of smaller abscesses is over the psoas muscle
en the ileum and cecum. They may be found, how-
e region of the umbilicus, near the promontory of the
in the pelvis. When the perforation has occurred
ppendix not covered with peritoneum, an extraperito-

neal abscess is formed and the peritoneum may not become affected. An occasional result of the adhesive inflammation is the constriction or bending of a loop of the intestine in such a manner as to produce temporary or permanent obstruction.

Necrotic Type.—This is usually an advanced stage or consequence of one of the preceding types. It is perhaps primary in some instances. It may affect the entire appendix or only a limited portion, a single small area or several. Perforation more commonly follows the limited necrosis. In either case, a severe localized or general peritonitis is the usual result. The localized gangrene with consequent perforation is more frequently found at the base, close to the cecum, but it may occur at the tip or at any point in the wall. The appendix often sloughs off, and is then found as a highly necrotic mass in the abscess cavity. When still adherent it may be dark red, black, or greenish, corresponding to the degree of necrosis. In all cases of this type, micro-organisms are found in great numbers in the extremely fetid pus. It is more probable that the pus-forming streptococci and staphylococci are the active agents, particularly the streptococci in the more virulent cases, but in a large number of cases the Bacillus coli communis has been the only organism found. This fact has been explained by Welch, however, as due to the ability of this bacillus to outlive the other organisms in the presence of inflammation.

Remote Effects of Perforation.—These are due, for the most part, to abscess-formation or the burrowing of pus and its erosive action. When the perforation is extraperitoneal, a retroperitoneal abscess is formed. The pus may pass beneath the iliac fascia and even perforate the skin in the region of Poupart's ligament, or it may pass beneath the ligament. It sometimes travels along the psoas muscle, and may reach the hip joint, the scrotum, or, by passing through the obturator foramen, form as abscess in the gluteal region. It sometimes burrows upward to form a perinephric abscess, or still further to erode the liver or perforate the peritoneum in this region. It has also passed through the diaphragm and pleura into the lung. More common avenues of perforation, especially for the intraperitoneal abscesses, is into some portion of the intestine, the urinary or gall-bladder, vagina, or rectum; and in a few instances the pus has penetrated a hernial sac or found an external exit through the abdominal wall.

An occasional result of perforation is the erosion of a blood-vessel, with severe or fatal hemorrhage. The internal iliac artery, portal vein, and smaller vessels in the walls of the intestines or other viscera have been perforated. Phlebitis of the mesenteric vein and abscess of the liver are occasional results.

Symptoms.—There is scarcely another disease in which the symptoms are so diverse or in which they may be so little significant of the real pathological condition as this. It is better, therefore, not to attempt a classification of them into types, but to study the disease in its entirety, for the mildest onset often precedes a rapidly fatal course, and the severest cases may subside with surprising rapidity. In some cases symptoms are absent, or they are so mild as to attract little attention. Many cases begin gradually with manifestations which may be regarded as prodromal. These are generally characterized by colicky pains and

tenderness more or less confined to the right iliac fossa, with malaise, loss of appetite, constipation or diarrhea, sometimes also with nausea and vomiting. With or without these early manifestations, however, there is ordinarily a sudden paroxysm of severe pain. This is followed with fever, gastrointestinal disturbances (constipation, nausea, and vomiting), and tenderness or pain on pressure over the region of the appendix.

Pain.—The pain is usually violent and begins suddenly, without obvious cause, or it may follow one of the recognized causes of the disease, an error in diet, a blow, strain, or jar. It may amount only to a sense of discomfort, but it is often sharp and agonizing. It is generally persistent, but subject to paroxysmal exacerbations. It is often referred at first to the umbilical, epigastric, or hypogastric region, or it may be diffused over the abdomen, but it becomes localized in most cases within twenty-four to forty-eight hours, in the right iliac fossa. Extremely sharp pain usually denotes an involvement of the peritoneum with great danger of perforation, or that this has already occurred. On the other hand, no positive deduction can be made from the character of the pain, for, although severe, it is sometimes transitory, and sometimes, it is thought, significant only of appendicular colic, occasioned by violent peristaltic action in attempts to expel mucus from the interior of the appendix.

Fever.—Elevation of temperature is one of the most significant symptoms, since it indicates an inflammatory process as the source of the pain. It not only serves to exclude appendicular colic, an afebrile condition, but it is highly indicative of the severity of the inflammation or the presence of suppuration. It usually develops within twenty-four hours after the onset; it may be preceded by chilly sensations, but seldom by a distinct rigor. In a mild case it may never exceed 101° F. (38.5° C.), except in children, when it is usually higher. In severe cases it frequently rises rapidly to 103° or 104° F. (39.5°—40° C.). It generally pursues an irregular course. But fever fails to prove a trustworthy sign in many cases and can never be implicitly relied upon, for even in the presence of abscess it may be absent, and in some of the severest cases, when general peritonitis is present from the beginning, the temperature is subnormal. The pulse is usually accelerated in ratio to the temperature. The respiration is often superficial or irregular on account of the pain.

Gastrointestinal Disturbances.—The tongue is coated, though usually moist. The appetite is lost, and thirst is generally excessive. Nausea and vomiting are more uniformly present in severe perforative cases; they may be absent in the milder types. Obstinate constipation is the rule after the onset. Hiccough is often an annoying symptom. Great irritability of the bladder is often complained of. The urine is scant, often albuminous, and indican is generally present.

Physical Examination.—*Inspection.*—The facial expression, general condition and attitude, of the patient are of much value in arriving at a diagnosis. As the Hippocratic or abdominal fascies indicates extensive involvement of the peritoneum, so its absence may signify the reverse. In cases having a mild beginning, the patient may continue at his vocation for a day or two, but in walking he assumes a slight stoop

and leans to the right. In most instances, however, he at once takes to his bed. Here he lies on his back or possibly on the right side, with the right knee drawn up. There is usually nothing peculiar in the appearance of the abdomen unless the disease has progressed unfavorably for a few days. After abscess-formation or perforation it becomes distended, and the right side may be slightly the more prominent.

Three valuable signs are elicited by palpation and percussion, namely, rigidity, tenderness, and dullness.

Rigidity.—Abnormal resistance to pressure over the right iliac fossa is usually an early sign. It is due chiefly to rigidity of the right rectus muscle, which is unmistakable when compared with the normal tension of the left. In from twenty-four to forty-eight hours, sometimes earlier, a distinct swelling can often be felt in the region of the cecum. It is sometimes concealed, however, by the abdominal rigidity or by the distention of the intestine, unless the patient be anesthetized.

Tenderness.—There is usually from the beginning great tenderness or acute pain on pressure in the right iliac fossa. In a majority of cases the most acutely sensitive spot is found at "McBurney's point." This is situated on a line drawn from the umbilicus to the right anterior superior spinous process, where it intersects the outer margin of the right rectus muscle. The pressure should be made with the tip of one finger pressed deeply and firmly into the abdominal wall. In some cases, owing, perhaps, to an unusual position of the appendix, the greatest tenderness may be found in another location, or it may be diffused over a wider area. Rectal or vaginal palpation is sometimes of value in such cases, for a characteristic point of tenderness, the swollen appendix, or more certainly an abscess of large size, may be felt. Fluctuation can sometimes be obtained by palpation of an abscess of considerable size.

Dullness.—Percussion elicits a dull tympanitic note over the region when there is tumefaction of considerable extent.

While all these signs are of value when well marked, the absence of any one or more of them does not preclude the presence of appendiceal disease. They are often less distinctly recognizable or altogether absent after perforation has occurred, although there may be extensive burrowing of pus.

Blood-Count.—Leucocytosis is usually present, especially after suppuration has occurred, when it may exceed 50,000.

Course.—Mild cases, in which pus is absent or so small in quantity that it can be absorbed, usually ameliorate after three or four days. The pain subsides, the fever declines, the constipation yields; all the symptoms abate, and recovery is complete in from ten to twenty days. Sometimes the recovery is less rapid, and slight fever persists during a week or two. Recovery occurs, however, in a majority of all cases. It is sometimes permanent, but too often it is of only short duration. Recurrence is to be anticipated, particularly in cases in which induration or tumefaction remains in the region of the appendix.

Chronic Appendicitis.—This term is sometimes given to cases in which the induration fails to subside with the other symptoms. The patient usually suffers from a more or less constant uneasiness in the ileac region, or occasional attacks of pain, with or without other symptoms.

Recurrent Appendicitis.—In another large class of cases an apparently complete recovery takes place and the induration subsides, but in the course of three or four months, often much earlier, a relapse occurs, accompanied with all the symptoms of the original attack. This may also be recovered from, and another relapse may follow, and thus the disease may run on for several years. Ultimate recovery sometimes occurs in these cases, probably as a result of obliterative inflammation or from the evacuation of a pus cavity into the bowel, but any of the attacks may prove fatal, and the condition is an exceedingly dangerous one.

Cases that are characterized by a sudden, violent onset usually correspond to the suppurative or ulcerative type of the disease. The initial symptoms in many cases do not correspond to the actual beginning of the disease. The inflammatory process may have been going on for an indefinite time, and the sudden pain, tenderness, and fever indicate the beginning of suppuration, the rupture of the distended appendix, or possibly the giving way of an abscess that has formed insidiously. Although the symptoms may ameliorate after three or four days, as in a mild case, the improvement is generally of short duration. The fever, as a rule, assumes a remittent course from the beginning, and after a few days the symptoms of sepsis become clearly marked. Death may result from septicemia, pyemia, or pyelophlebitis, without rupture of the appendix, but it is more frequently a result of general peritonitis, which may be produced through the action of bacteria, either before or after rupture.

Peritonitis, as just stated, may result from infection without rupture of the appendix, but it is more commonly a result of that accident. The pus is sometimes shut off from the general peritoneum by adhesions, producing a localized abscess. The abscess may rupture later and set up a general peritonitis. In many instances the general peritoneal involvement has been established before the appearance of acute symptoms, and herein lies the greatest danger of the disease. The initial sudden, sharp pain often means the onset of peritonitis and the termination of a previously unrecognized appendicitis. It is then usually followed within a few hours by an extension of the pain and tenderness more or less generally over the abdomen, with distention, tympanites, and rigidity of both sides. The pulse becomes rapid and feeble, the respiration correspondingly fast, often irregular or stertorous, the voice weak, and the face anxious and pinched. The tongue is dry and the bowels are constipated and vomiting persistent, while the urine becomes scant or suppressed. The temperature is variable, sometimes not over 100° F. (37.5° C.), sometimes over 105° F. (40.5° C.). Death is inevitable.

Diagnosis.—A sudden attack of violent pain in the right iliac fossa, in a person who was previously healthy, especially in one under 30 years of age, and yet more positively if associated with abdominal rigidity, tenderness on pressure in this region, vomiting, constipation or diarrhea, is almost invariably due to appendicitis, for this is the most common of all inflammatory diseases of the abdomen in early life. The diagnosis becomes difficult only when some of the symptoms are absent or when unusual manifestations appear, as when the pain is referred to a distant region. The greatest cause of error is, no doubt, too great

haste in reaching a conclusion. To avoid this, the complete history of the case should be carefully obtained and carefully studied; then a thorough examination should be made with a view to excluding all possible sources of error. Many affections enter into the consideration.

1. *Colic.*—Severe intestinal colic may for a time cause confusion, but the absence of distinctive signs in the appendix region is generally sufficient. Diarrhea is more common than constipation. In *hepatic colic* the conditions usually found in the right iliac fossa are absent; the pain generally radiates toward the right shoulder and back; the tenderness is confined to the region of the liver and gall-bladder if calculi are present, and jaundice commonly appears. Gall-stones may be found in the feces. *Renal colic* is excluded by careful palpation of the abdomen and examination of the urine. Pain radiating to the bladder and penis or scrotum is more common than in appendicitis, and gravel is frequently passed. Dietl's crises, due to movable kidney, are relieved by restoration of the organ to its proper position. Gastrointestinal disturbances are less common. Lead colic is less likely to be mistaken for appendicitis than the reverse, for without careful examination the pain of the latter condition may be referred to lead colic when it occurs in a subject of lead-intoxication.

2. *Perforation of Ulcers.*—The symptoms arising from the perforation of gastric, duodenal, or typhoid ulcer may be mistaken for appendicitis, but can usually be excluded by the history of the case and the absence of the local signs of the latter disease.

3. *Intestinal obstruction*, intussusception, fecal impaction, internal strangulation, and other obstructive conditions can generally be excluded, but not always without difficulty. Fecal impaction of the cecum is of slower onset, the pain is moderate at first and of a colicky character, the tumor is usually large and hard, sometimes doughy, and there is less tenderness; the right rectus is not generally so tense. Bloody mucous evacuations usually accompany intussusception. Fecal vomiting is common to nearly all obstructions. The tympanites develop rapidly and may be confined to the upper part of the abdomen. Pericecal abscess cannot be differentiated from that due to appendicitis without incision, but the differentiation is unimportant, since both conditions call for operative treatment.

4. *Psoas Abscess.*—Fluctuation can be more uniformly obtained, and examination of the spine reveals the source of the pus in most cases.

5. *Renal Disease.*—The pain of pyonephrosis, perinephritic abscess, or tumor of the right kidney is often excluded with difficulty. Examination of the urine may reveal the condition, but in perinephric abscess the differentiation cannot be made without exploratory incision.

6. *Hemorrhagic pancreatitis* may be mistaken for appendicitis, in part on account of its rarity, but the pain is usually different in character and location.

7. *Female Disorders.*—The colicky pain of the menstrual period is sometimes a source of error. A neuralgia of the right ovary is more likely to cause difficulty in diagnosis. In both conditions, however, the absence of tumefaction or rigidity of the rectus, with the history of the case before the attack and during the few days succeeding it, generally reveals the true condition. Pyosalpinx, pelvic hematocele, and pelvic

peritonitis can generally be recognized on careful examination, but the differentiation from a ruptured appendix is extremely difficult except with a clear history of the case. Extrauterine pregnancy may rarely be a source of confusion. The signs of pregnancy are usually to be found in the breast; and the location of the tumor, pain, and tenderness, seldom corresponds to that of the appendix.

8. *Typhoid fever* does not occasion confusion when the history of the case is obtained. The iliac pain is seldom so severe at an early period of the disease; the tumor is not present; the rectus muscle is not so rigid, and the fever is higher and more regular in its course. The presence of leucocytosis speaks for appendicitis, the Widal reaction for typhoid fever.

9. *Enteralgia*, mucous colitis, and other painful affections occurring in neurasthenic, hysterical, or hypochondriacal persons often closely simulate appendicitis on account of the strong conviction in the mind of the patient that the disease is present. In an individual who is familiar with the symptoms of the disease the picture may be so accurately drawn that unless the most careful physical examination be made without regard to the subjective manifestations, the most skillful diagnostician may be misled.

Prognosis.—A large majority, estimated at 80 to 90 per cent of all cases, recover spontaneously or under treatment. Nevertheless, a favorable prognosis should not be too early pronounced, for the most favorable condition may be converted into the most unfavorable within a very few hours. The outlook becomes less hopeful, as a rule, with each succeeding attack of the disease, not only because each attack brings the patient into greater danger of perforation, but because a condition is ultimately reached which is exceedingly unpropitious for operative measures. No deduction can be safely drawn from the experience of the patient, the physician, or, indeed, from medical and surgical statistics. Every case has peculiarities of its own and it must be regarded as fully liable to the most unfavorable results. We have no means of recognizing the obliterative appendicitis, although it may be assumed to have been present when the disease finally ceases spontaneously after a series of attacks.

Treatment.—The patient should be strictly confined to bed and made as comfortable as possible. For the relief of the pain an ice-bag should be placed over the region of the appendix. Opium should be avoided if possible, for it often masks the real condition, but in extreme cases morphin must be given hypodermically in doses only sufficient to render the suffering bearable. Full doses of sodium salicylate often afford relief without producing profound insensibility to pain and tenderness. Relief sometimes follows a copious enema of warm soap-suds, but the internal administration of laxatives is objected to by most authors. The diet should be exclusively liquid.

The most important question to be determined is the advisability of resorting to surgical measures. It is only in a case that runs a mild course from the start, and shows distinct improvement by the third or fourth day, that this question can be decided in the negative. The necessity of an operation should be urged upon the patient: 1. If he has passed through one or two previous attacks; 2, in every case of severe

onset with violent pains; 3, in every case in which a tumor can be recognized; 4, whenever a mild case shows a sudden increase of severity, with rise of temperature, severe pain, or the development of a tumor; 5, in every case in which there is evidence that suppuration or perforation has occurred, providing that the patient's condition admits of immediate operation. In all cases of uncertainty, the surgeon should be called without avoidable delay, and, when the indications are distinct, the operation should be performed without the delay of an hour, for the mortality after early operation is inconsiderable, and the chances of recovery rapidly diminish with each day of procrastination.

INTESTINAL OBSTRUCTION.

ILEUS, OBSTIPATION.

1. **Strangulation** (Constriction of the Bowel, Intra-Abdominal Hernia).—The exhaustive investigations of intestinal obstruction by Fitz have given us the best analysis of the condition. Strangulation constitutes about 35 per cent of all cases of obstruction. It may be partial or complete. It is much more frequent in males, and nearly half the cases occur between the ages of 15 and 30. The small intestine is involved in nearly 90 per cent of cases and usually in the lower part of the abdomen; often in the right iliac fossa (67 per cent).

Etiology.—The causes in the order of their frequency are: bands, cords, slits, and fissures in the omentum and mesentery, diaphragmatic hernia, and peritoneal pouches. Rare forms of strangulation are the duodenojejunal hernia of Treitz, in which a loop of the intestine slips into the duodenojejunal fossa; and the hernia of the omental bursa, in which the loop passes through the foramen of Winslow. The condition producing the strangulation, unless a congenital defect, is generally a consequence of previous peritonitis. This is particularly the cause of the formation of adhesive bands between the intestine and the abdominal wall, as after a surgical operation, between loops of intestine or between persistent vitelline remains, as a prolongation of Meckel's diverticulum or obliterated vitelline blood-vessels and other abdominal viscera. In the same manner the tip of the vermiform appendix may become attached and cause constriction of a coil of the intestine, which slips through the unnatural opening.

2. **Intussusception** (Invagination).—In this condition, which constitutes over 30 per cent of the cases of obstruction, a constricted portion of the intestine is forced into a relaxed portion immediately below it. Nothnagel believes that the lower bowel is drawn up over the constricted upper portion. It is a condition peculiar to early life, a majority of cases occurring in males before the tenth year and fully one-third in the first year.

Etiology.—In a majority of cases diarrhea or habitual constipation precedes the invagination, but in some cases no more definite cause can be assigned than irregular or excessive peristaltic action. An invagination sometimes occurs at the time of death, but it can be distinguished at autopsy by the absence of adhesions or other evidence of inflammation.

Morbid Anatomy.—As a result of intussusception, a cylindrical tumor

is produced, which varies in length from a few inches to a foot or more. In extreme cases the ileocecal valve has been found in the rectum. The intussusception consists of three layers of intestine. The outer, known as the intussuscipiens, or receiving layer, is continuous with the bowel below, and the innermost, or entering layer, with the bowel above. The middle or returning layer joins the two. The mesentery attached to the entering layer is also drawn in, and as a result the opening at the lower extremity of the invaginated part has the appearance of a slit. The invaginated portion has a dark red or purplish color due to congestion, and the veins are distended with blood. Hemorrhages are commonly found within or upon the walls. The peritoneal surfaces of the entering and returning layer, lying in contact with each other, show the changes of acute peritonitis, being covered with fibrin and more or less firmly united by adhesions. In some cases the invaginated portion becomes separated and is discharged as a slough. Union may then take place between the upper and lower portion of the bowel at the mouth of the invagination. Fibrous stricture is apt to form, but complete recovery has occurred in many cases.

3. **Twists (Volvulus) and Knots.**—These constitute 14 per cent of all cases. A majority (68 per cent) of twists are encountered in men, and about one-third of them between the ages of 30 and 40. The large intestine is involved in nearly 90 per cent of cases, most commonly the sigmoid flexure, next the cecum. The condition is favored by an unusual length of the mesentery, elongation of the intestine by hernia, the traction of adhesions, or an accumulation of feces. Rarely a loop of the intestine is twisted about another portion. The bowel may be twisted on its long axis a half-turn, a whole turn, or more, complete strangulation being produced. The intestine below the constriction is distended and deeply congested. A fatal peritonitis is usually developed. Knots are extremely rare.

4. **Stricture and Tumors.**—Stricture of the intestine sometimes exists at birth, or more commonly the canal is completely obliterated in a part of its length, as is usually the case in imperforate anus or when the duodenum is separated from the stomach. Acquired strictures are generally a result of the cicatricial healing of ulcers. These may be stercoral, syphilitic, or tubercular, or they may result from a localized peritonitis, the repair of an intussusception, or very rarely from dysentery or typhoid fever. Tumors cause obstruction either through obliterating the lumen of the intestine when within it, or by compressing or drawing upon the bowel from without. Cancer is the most frequent cause, and it is generally located in the large bowel, very often in the sigmoid flexure or rectum. It is more common in women after the fortieth year. Papilloma, fibroma, adenoma, and lipoma occasionally cause occlusion. Pelvic abscess may compress the bowel. An accumulation of feces in one portion of the bowel, as in the sigmoid flexure, sometimes closes an adjacent loop of the intestine by compression.

5. **Abnormal Contents.**—The most common cause of obstruction by foreign bodies is an accumulation of gall-stones. A majority of the patients are women, all are adults, and six-sevenths are over 50. The next most common cause is impaction of feces. This may occur in either sex and at any period of life, often in young children. Enteroliths not

infrequently occur. These generally have as a nucleus some undigested substance, as hair, thread, fragments of bone, or the pits or husks of fruit, and an external coating of calcium or magnesium phosphate. They are often as large as a hen's egg. Foreign bodies of every description may be swallowed and pass to the intestine, or they may be introduced into the rectum and produce obstruction. The most common of these are coins, nails, stones, pins, buttons, and artificial teeth; but spoons, forks, and other large articles have been found.

Symptoms.—(*a*) *Acute Obstruction.*—The usual symptoms of acute obstruction are constipation, abdominal pain, tympanites, and tumor. At the beginning of the obstruction, several loose dejections often occur, but a complete stoppage follows, often so complete that neither fluid nor gas can pass it. Pain in the abdomen is an early symptom and often develops suddenly. It is at first colicky, but soon becomes intense and continuous. In intussusception the pain is more gradual in onset and it may have the character of tenesmus. Localized tenderness may be present, but it is not characteristic. Vomiting is a constant symptom and it generally follows immediately after the initial pain. It may be preceded by eructations of gas. The contents of the stomach are first brought up, then a greenish, bile-stained fluid, and finally, by the third day, a feculent, brownish liquid (stercoraceous vomiting). The solid contents of the large intestine are probably never carried up, but the fluid contents may pass the ileocecal valve and appear in the vomit. There are frequent efforts at the evacuation of the bowel, with the discharge of only a little blood-stained mucus. Tympanites and abdominal distention become extreme when the large bowel is obstructed. They are less pronounced in intussusception or when the obstruction affects the upper part of the small intestine. A palpable tumor is more characteristic of intussusception than of other forms of obstruction. The tumor may be felt in the rectum or through the abdominal wall, often in both locations, and, as a rule, during the first two or three days of the obstruction. It has the form of an elongated cylinder or sausage-like mass. When it reaches the rectum, a peculiar relaxation of the anus is often observed.

Constitutional symptoms are generally well marked. There may be slight fever after the first day of strangulation, but collapse is common, and the temperature may then be subnormal. When peritonitis develops, the temperature generally rises, the pulse becomes rapid and feeble, there is incessant thirst, and the tongue becomes parched. The urine is of high color and scant when vomiting is excessive; it may be suppressed when the obstruction is in the upper bowel. It often contains albumin and indican. Hiccough is sometimes a troublesome symptom.

(*b*) *Chronic Obstruction.*—Constipation of long duration is a constant symptom in this condition. When the obstruction is due to fecal accumulation, the dejections usually become less and less frequent for a period of several weeks, possibly for months. The obstructing mass is sometimes channeled in such a manner as to permit a part of the contents of the bowel above to pass through, and the patient may thus have evacuations at regular intervals. The bowel may become extensively eroded or ulcerated, and a fatal perforation or peritonitis may occur without complete obstruction. Sometimes an evacuation does not occur

once in a week, especially in old persons, and yet little discomfort is experienced. There may be frequent mucous discharges and attacks of nausea and vomiting. Finally the abdomen becomes much distended and severe pain develops. Feculent vomiting ensues as the obstruction becomes complete. The hardened mass of feces may be felt through the rectum or abdominal wall as a large, slightly movable tumor. When the obstruction is due to stricture or tumor, the pain corresponds to the location of the obstruction. Anemia and emaciation are common. The case generally terminates fatally with the symptoms of acute obstruction, but of more than the usual duration. Death may, however, result from exhaustion, without complete arrest of alvine evacuations.

Diagnosis.—An early diagnosis of the condition is important. It is necessary to take into consideration the situation of the obstruction, its nature, and the exclusion of other conditions which lead to error. The situation of obstruction is revealed, as a rule, by the history of the case, inspection and palpation of the abdomen, and examination of the rectum. Inspection may reveal the part obstructed through the character of the distention and the location of peristaltic movements when visible. When the obstruction is low in the large bowel, the colon may stand out prominently and a tumor may sometimes be felt, but the entire abdomen is often distended. Feculent vomiting is absent, at least until late. With the obstruction in the region of the ileocecal valve, the distention is greatest in the umbilical region, as a rule, and the feculent vomiting appears early. The folds of the small intestine may be thrown into ladder-like prominences by the increased peristalsis. When the duodenum or jejunum is obstructed, the distention is usually confined to the upper part of the abdomen, the urine is suppressed, fecal vomiting does not occur, but collapse develops early. In obstruction involving the large bowel, digital examination of the rectum may reveal it. Examination through the vagina is often useful. When these methods fail, the bowel should be distended with warm water, with the aid of an anesthetic if necessary, the patient lying on his back or right side with the hips well elevated. The water should be allowed to flow in slowly, especially after the first or second day, from a fountain syringe at a height of not more than three feet, for the bowel may be ruptured by too great pressure. The quantity of fluid that can be introduced sometimes reveals the situation of the obstruction. The adult colon should hold six quarts, the rectum three pints. The capacity of the infant colon is about three pints. Inflation with air is sometimes practiced, but it is a less satisfactory method.

The nature of the obstruction is usually more difficult to determine than its location. The statistics already given are of much service in this respect. The character and location of the pain and the presence or absence of fever are of little diagnostic importance. A majority of cases are due to strangulation or intussusception. The former is a condition of adult life, the latter of childhood. In strangulation the history is important with reference to former attacks of peritonitis or a laparotomy; a tumor is seldom present. Intussusception is characterized particularly by tenesmus and frequent small, bloody, mucous dejections. The sausage-shaped tumor is usually felt in the region of the transverse colon. Acute obstruction of the large intestine is generally due to

intussusception, volvulus, a tumor, or stricture. The first of these is practically eliminated after childhood. Volvulus can seldom be diagnosticated, but its frequent location at the sigmoid flexure should be remembered. Tumors may be recognized by rectal examination or abdominal palpation. Stricture is of slow formation, giving a history of increasing constipation for a week or more. Impaction of feces is more common in old persons, and the mass can be felt in the rectum or along the course of the colon. Its shape can generally be altered by external pressure. Obstruction by gall-stones is usually indicated by a history of repeated attacks of gall-stone colic. Vomiting occurs early and jaundice is sometimes observed.

Hernia.—Careful examination should always be made to exclude possible hernial strangulation, even when no external signs exist.

Appendicitis sometimes simulates obstruction, but it is generally recognized by the intense localized pain and tenderness, with fever.

Peritonitis is characterized by great abdominal tenderness, an elevation of temperature, but tumor and feculent vomiting are absent.

Blows on the abdomen and prolonged laparotomy are sometimes followed by obstinate constipation, but the other symptoms of obstruction are absent and the history of the case explains it.

Persistent constipation occurring in connection with floating kidney, renal or hepatic colic, and other conditions, especially when tympanites also develops, may arouse suspicion of obstruction, but the history of the case, the character of the abdominal distention, the absence of tumor, and the action of large enemata usually remove all uncertainty.

Prognosis.—This depends largely upon the character of the obstruction and the promptness of the treatment. Obstruction from strangulation is usually fatal unless an early resort to surgery is had. Relief sometimes occurs spontaneously or follows treatment. Intussusception generally proves fatal from the third to the fifth day, but recovery is sometimes secured. Obstruction by gall-stones or fecal accumulation is much less fatal.

Treatment.—*General.*—Purgatives must never be administered. The vomiting and pain may be greatly relieved by lavage of the stomach. When the suffering is intense, however, morphin should not be withheld, although it is claimed that it obscures the diagnosis. It sometimes renders a thorough examination less difficult, because less painful. The tympanites may be reduced by turpentine stupes. All food should be withheld, except as it can be administered by the rectum.

Special Treatment.—The treatment of nearly all cases of acute obstruction is surgical, but, to be of benefit, the operation must be made within the first three days, on the first or second day if possible. When the diagnosis cannot be determined so early, an exploratory incision is generally indicated. Intussusception can sometimes be overcome on the first day without operation, through the injection of a large quantity of water or olive oil. The patient should be anesthetized, and his body held in an inverted position. The colon is then filled, and the reduction of the invagination may be assisted by kneading the abdomen or by shaking the patient violently. The method may be repeated if necessary, but after the first day it is not devoid of danger, and the case should be submitted to the surgeon.

Chronic obstruction, before it has become complete, may be treated by irrigation and the administration of the mildest laxatives. When it has become complete, it should be treated as an acute case, and an operation may be required. Enterectomy or the establishment of an artificial anus may be found necessary.

CONSTIPATION.

COSTIVENESS.

Definition.—Prolonged retention of feces, or the habitually difficult or infrequent evacuation of the bowels.

Etiology.—In many cases there appears to be a constitutional proneness to constipation, and an entire family is often thus affected. It is probably a result of a similarity in habits of life and disregard of hygienic and dietetic rules acquired in childhood, and not a result of hereditary influences.

Age and Sex.—Constipation may occur at any age, but it is especially frequent after middle life, when the vital functions become sluggish and muscular exercise is neglected. It is not uncommon in infants, even from birth. Women are much more subject to it than men, probably to a great extent on account of the greater capacity of the pelvis, which permits distention of the rectum without discomfort. It is often caused by retroversion of the uterus and tumors within the pelvis. Repeated pregnancy and the menopause favor its development.

Habits.—Sedentary habits and mental application induce constipation largely through inducing neglect of the natural calls for evacuation. Neglect of physical exercise removes one of the influences which maintain the flow of bile and increase the peristaltic movements of the intestine. Railroad travel often induces constipation.

Diseases.—Any condition of ill health is liable to produce constipation. It is generally associated with anemia, often with neurasthenia, hysteria (nervous constipation), chronic disease of the heart, stomach, intestines, or liver. Stricture of the esophagus or at the pylorus induces it by preventing the passage of the food into the intestines. Central nervous and mental diseases, especially insanity, chronic myelitis, and destructive lesions of the cord, are commonly attended with obstinate constipation. The condition prevails in the acute fevers, except those which affect directly the intestinal tract, as cholera and typhoid fever.

Diet is one of the most important factors. Food which leaves too little or too much residue, improperly prepared or insufficiently masticated food, particular articles, as cheese, nuts, raw vegetables, and certain beverages, as milk, tea, and some of the sour wines, induce constipation to a greater extent in some individuals than in others. The drinking of an insufficient quantity of water is a common cause. The loss of fluid by profuse sweating in hot weather, and lactation, are regarded as influential in many cases. Diabetics are usually constipated.

Symptoms.—Constipation often exists for a long time without producing other abnormal manifestations than the condition itself, but sooner or later in most cases definite symptoms arise. These have been attributed by some writers to copremia, the absorption of poisonous matter

from the retained feces. Different individuals are affected quite differently. Some experience much discomfort from constipation of a day's duration, while others are not at all inconvenienced by retention for a week, and complain of illness only on the days on which the bowels move. The most constant symptoms are headache, lassitude, physical and mental debility, and inaptitude for work. Hypochondriasis, hysteria, melancholia, seminal emissions, enuresis of children, and many other disorders have been attributed to constipation. The appetite is generally lost, the tongue becomes heavily coated, the breath foul, and the patient suffers from a sense of abdominal weight and distention. Periodic attacks of slight fever are not uncommon. Neuralgia is often complained of, especially that of the sacral nerves due to the pressure of the fecal accumulation in the sigmoid flexure. In women the distention of the rectum is often a cause of painful menstruation. Hemorrhoids are often induced by the pressure of the hemorrhoidal veins; ulcers, by the pressure and infection of the intestinal mucosa; and fissures, by the passage of the hardened masses. Attacks of cramps and abdominal distention usually occur at variable intervals, and diarrhea not infrequently alternates with the constipation, especially when a hardened fecal accumulation becomes channeled in such a manner as to permit the escape of the contents of the upper bowel. The patient often acquires a sallow, muddy complexion, and acne or eczematous eruptions may appear.

Constipation in infants is often due to improper food, milk that is too rich in casein or deficient in fat. Failure to give the infant an occasional drink of water is a common cause of it. Sometimes, no doubt, it is a result of feeble digestive power. It has been caused in some instances by congenital stricture, a constricting band or other structural defects. The condition is common and often very difficult of relief. The principal symptoms are colic, abdominal distention, and sometimes vomiting.

Prognosis.—This depends chiefly upon the cause and duration of the affection and the physical condition of the patient. Serious results are generally due to gross neglect on the part of the patient. Constipation of infants usually disappears immediately upon the commencement of a mixed diet.

Treatment.—*General.*—Constipation seldom develops in those who have acquired the habit of evacuating the bowels at a fixed hour every day. The importance of this habit is not sufficiently recognized. And there is no more important measure for the cure of constipation. The patient should retire to the closet at a stated time, even when there is no desire. He should sit and wait, without straining, for probably ten minutes. If a spontaneous movement does not then occur, an enema of cold water, a weak salt solution, or soap-suds may be employed. Glycerin, ʒ j in a pint of water or in a suppository, is more active. Individuals of sedentary habits should resort to systematic exercise, walking in the open air, or moderate horseback or bicycle riding. Those with pendulous or relaxed abdomens should wear an abdominal band, and practice calisthenics with especial reference to the strengthening of the abdominal muscles, swinging the arms upward and bending to touch the floor. If a gymnasium is accessible, they should use the overhead pulleys and the pumping apparatus. Massage of the abdomen is useful in most

cases, and the "cannon-ball," weighing 5 or 6 pounds, may be rolled over the abdomen, following the course of the colon.

Dietetic.—The diet must be regulated to suit the individual case. Persons whose food has been too coarse should modify it so as to avoid such articles. In many persons coarse food, as Graham or brown bread and oatmeal, act as laxatives. Fruit, especially an orange or an apple, before breakfast, and such vegetables as lettuce, spinach, onions, and tomatoes are beneficial in many cases. Salads containing much oil are wholesome. Molasses and honey are laxative, and some persons can regulate the bowels by eating a piece of taffy every day. An important element of treatment in some cases is the regulating of the time of meals and the taking of sufficient time for thorough mastication of the food. The patient should learn to drink plenty of water. A glass of cold water immediately before retiring and on rising in the morning is often beneficial. Hot water is more serviceable in some cases if taken morning and evening or before each meal. Strong coffee, beer, cider, and carbonated waters are laxative to some persons.

Medicinal.—Drugs should be avoided if possible. When they are deemed necessary, a small dose of a saline laxative, sodium or magnesium sulphate or sodium phosphate, should be given in the morning before breakfast, or the fluid extract of cascara sagrada, 3 ss to j (1.8—3.6), or the compound licorice powder, 3 j (3.8), at night. The 3-grain cascara pill is usually preferred to the bitter fluid extract. Many other drugs, singly or combined, especially aloes, colocynth, rhubarb, and podophyllin, are employed, and the addition of the extract of belladonna, gr. 1-12 (0.005), and nux vomica, gr. ¼ (0.016), to the prescription is recommended.

Constipation in infants can often be overcome by giving an occasional drink of water, allowing the infant to suck a few drams from a linen rag, by administering two or three drams of cream in water before each nursing time, or by adding it to the artificial food. Barley-water or oatmeal-water acts well in some cases. A small glycerin or soap suppository is generally efficient for moving the bowels, or a small injection of cold water may be employed. For older children the effervescent magnesium citrate solution is generally agreeable, but there is no better laxative than castor oil. Children old enough to eat fruit seldom require drugs.

HEMORRHOIDS.

PILES, EMERODS.

Definition.—A varicose condition of the external hemorrhoidal veins, producing painful swellings just within or around the external margin of the anus. When the swelling affects the veins beneath the mucous membrane within the external sphincter, the protrusions are known as internal hemorrhoids; when those beneath the skin, they are external hemorrhoids.

Etiology.—Hemorrhoids occur most frequently in middle and advanced life; they are rare before puberty. Both sexes are affected, but men more frequently than women. The common cause is venous stasis. This may be due to a local condition, especially to the pressure of accumu-

lated feces in habitual constipation, to stricture of the rectum, to tumors of the rectum, prostate, uterus, or ovaries, or to more remote obstruction as that of the portal vein in cirrhosis of the liver, or general venous stasis in the chronic dilatation of valvular disease of the heart. Pregnancy often induces them. Excessive indulgence in alcohol, and, more remotely, all the influences which lead to constipation, favor the development of hemorrhoids.

Symptoms.—These vary with the character and severity of the disease. Internal piles often exist without causing inconvenience and may not be recognized until an erosion and bleeding occur in the passage of a hardened mass. In many cases, however, the hemorrhoidal mass is extruded with every act of defecation. Free bleeding often occurs, and this, in severe cases, produces anemia. Rarely, hemorrhage takes place independently of defecation. A considerable quantity of blood may be lost externally, or it may be retained within the rectum and discharged with the stool without the patient's knowledge. Such cases may be recognized only in a search for the cause of an obscure anemia. As a rule, however, only a small quantity of blood is lost, perhaps only enough to streak the fecal mass as it passes. A sense of fullness, itching, burning, or pain often accompanies severe cases, especially during and after defecation. In the worst cases the pain may be reflected to the loins or it may radiate down the thighs and legs to the soles of the feet. In cases of long standing an anesthetic condition of the anus is sometimes produced which renders the patient unable to recognize the completion of defecation, or there may be a constant desire for evacuation. Vesical irritation is sometimes an aggravating symptom. Constipation is usually kept up through the patient's dread of defecation. No little distress is often occasioned by the inability to retain the hemorrhoidal mass within the sphincter; the slightest exertion, even walking, a cough, or a sneeze, will sometimes cause it to protrude and possibly to bleed. Various reflex symptoms, as hypochondriasis and melancholia, are more or less directly a result of the condition in some cases.

External hemorrhoids cause inconvenience more than suffering, except when they become eroded through friction.

Prognosis.—Serious results are seldom produced, but when the condition is attended with profuse hemorrhage, the patient's health may be greatly impaired and a coexistent disease may be aggravated.

Treatment.—The curative treatment of internal hemorrhoids is surgical, and every case should be submitted to the surgeon unless the condition of the patient precludes the administration of an anesthetic. Such cases are often encountered by the physician. The indications are, to overcome the constipation and to relieve symptoms as they arise. Relief of the irritation is generally afforded by suppositories containing opium extract and powdered nutgalls with iodoform or ichthyol. A condition of comfort almost amounting to cure may then be obtained from the habitual use of an enema of cold water, or very hot water immediately before defecation. This should be done at a fixed hour every day. The best time in many cases is just before retiring, since protrusion of the hemorrhoids may otherwise follow. The quantity of water should not exceed a pint, as a rule; just enough to relieve venous engorgement and stimulate peristalsis. It should not flow from a height of more than

three feet, for rectal dilatation may be induced by too large a quantity or too great pressure. The patient must sometimes be taught to reduce the protrusion. This is generally done without difficulty, after bathing it with cold water, by pressing it upward with the fingers while making an expulsive effort. When, however, the piles become strangulated, cocain or general anesthesia is sometimes necessary if permissible. External piles are often cured by an ointment of gallic acid, gr. x in an ounce of vaselin. Incision and evacuation of the clot are better.

ENTEROPTOSIS.

GLENARD'S DISEASE.

Definition.—An abnormal descent of the intestines in the abdominal cavity, usually associated with prolapse of the other viscera (visceroptosis). The terms used to describe the descent of the individual organs are: Gastroptosis, descent of the stomach; splenoptosis, descent of the spleen; coloptosis, descent of the colon. Displacement of the liver is very rare.

Etiology.—The condition may be due to congenital laxity of mesenteric attachment, but it is more common in young women, especially in anemic neurasthenics; and in another class of cases, it is due to a removal of the support of the abdominal wall as a result of constipation, pregnancy, ascites, or ovarian cyst.

Symptoms.—In some cases there is little or no disturbance, while in others the patient experiences a constant abdominal discomfort. Constipation and digestive disorders are present, and these may lead to emaciation, debility, and melancholia. The transverse colon can sometimes be felt just above the pelvis, and the acute bending of it may occasion more or less complete obstruction. Its location can readily be determined by artificial inflation.

Treatment.—This is directed to (1) the relief of constipation, (2) support of the abdomen by a properly adjusted abdominal bandage, and (3) the general condition of the patient, particularly the relief of the neurasthenic state. When the abdominal walls are much relaxed, massage and calisthenics are advantageous.

DILATATION OF THE COLON.

Etiology.—The causes are (1) increased pressure, distention, from within the bowel, (2) diminished resistance on the part of the intestinal walls, and (3) obstruction. Congenital dilatation is also recognized (Hirschsprung's disease).

1. The increased pressure from within may be produced by either gaseous or solid contents. The dilatation is at first temporary, but, often repeated, it leads to permanent enlargement.

2. The diminished resistance on the part of the abdominal wall may result from (*a*) the acute distention, (*b*) a paretic condition of the muscular coat which may have originated in a general enfeeblement of the system through anemia and malnutrition, or (*c*) a prolonged use of cathartics.

3. Obstruction is generally due to (*a*) congenital narrowing of the lumen, (*b*) acquired stricture, (*c*) foreign bodies, (*d*) impaction of feces or gall-stones, (*e*) incomplete twist, especially at the sigmoid flexure, or (*f*) pressure from without, by tumors or displaced organs.

Morbid Anatomy.—The colon is sometimes enormously dilated and its wall may be extremely thin. In less pronounced cases, the walls may appear normal or the muscular layer may be hypertrophied. In the case of the "balloon man" recorded by Formad, the colon was from 15 to 30 inches in circumference and with its contents weighed 47 pounds.

Symptoms.—These are more prominent when the dilatation is acute; when the dilatation is gradual, it may occasion comparatively little discomfort. In a severe case cardiac palpitation and dyspnea or fatal embarrassment of the heart and lungs may result from the upward pressure. Obstinate constipation is the rule, and in cases caused by obstruction frequent spells of vomiting occur. Percussion reveals an increased area of colon tympanites, particularly after artificial distention.

Diagnosis.—This is determined by the history of the case and a careful examination as to the cause of the abdominal distention. The condition is to be differentiated, as a rule, from gaseous distention of the peritoneal cavity due to perforation of typhoid, gastric, or other ulcers. Such perforation, however, is announced by sudden acute pain and collapse. The tympanites extends over the area of normal hepatic dullness. Peritonitis is quickly developed, with elevation of temperature and diffuse tenderness.

Treatment.—1. Acute gaseous distention can often be relieved by the passage of the rectal tube, giving vent to the gas. Turpentine stupes are beneficial. 2. When due to fecal accumulation, enemata containing ox-gall, and restriction of diet, especially the exclusion of starchy food, may overcome the condition. Laxatives should be regularly administered, and antifermentatives, salol, bismuth subgallate, or betanaphthol, may prevent a recurrence. 3. When anemia and malnutrition are present, the administration of iron and strychnin is indicated, and abdominal massage may prove beneficial. 4. Cases due to obstruction often require surgical treatment—the making of an artificial anus or excision of a portion of the bowel.

NEUROSES OF THE INTESTINE.

NERVOUS DIARRHEA.

Definition.—A functional motor disturbance of the intestine, producing diarrhea.

Etiology.—The condition is encountered in either sex and at any age, but it is more common in nervous or hysterical women at the menopause or in connection with disease of the generative organs. It is not infrequent, however, in young women. Anemia, malnutrition, and disordered gastric digestion are predisposing causes. Back of the disorder there is very frequently a strong emotion, as of grief, hope, or fear. Disappointment, bereavement, fright, anger, and pain induce acute attacks, which may prove persistent. The affection is sometimes observed in connection with nervous affections, as exophthalmic goiter and locomotor ataxia.

In the latter disease it sometimes assumes the form of persistent crises. Cases in which diarrhea follows the eating of certain articles of food, harmless to other people, are probably of this nature.

Symptoms.—The only symptom in many cases is diarrhea. This is often limited to two or three watery, pasty, or scybalous passages in the morning. In other cases the ingestion of food is immediately followed by an imperative demand for evacuation. Intestinal rumbling or gurgling is often present and a cause of embarrassment to the patient. The affection often runs an intermittent course, improvement being broken by the occurrence of any nervous irritation or worry.

Diagnosis.—This is based on the history, the character of the diarrhea, and the nervous condition of the patient. It is to be differentiated chiefly from acute enteritis. In the latter affection, the attacks often occur at night, they are attended with pain and numerous evacuations, often with vomiting, and usually follow a definite error in diet.

Treatment.—All treatment is useless which fails to remove the cause. On this account a change of scene, removal from the cause of worry, and diversion from sorrow are more important than drugs. Relief of the neurasthenic condition, by whatever means, is promptly followed by arrest of the diarrhea. Astringents are seldom beneficial, and opiates should not be employed. The bromids, ammonium valerianate, or asafetida is beneficial in some cases.

ENTERALGIA.

COLIC, INTESTINAL NEURALGIA, INTESTINAL CRAMPS, ENTEROSPASM, ENTERODYNIA.

Definition.—A disturbance of the sensory filaments of the intestinal nerves, producing sharp pain, often accompanied with localized spasm of the muscular coat of the intestine.

Etiology.—The condition occurs at any age, very frequently in infancy and childhood, and it is more common in women.

1. *Predisposing Influences.*—As in gastralgia, there is often a constitutional disorder back of it, sometimes apparently a hereditary predisposition; a neurotic temperament, improper hygiene, poor health, chronic disease or gastric indigestion, business care and worry, or mental strain.

2. The *exciting causes* are: Irritating intestinal contents, toxemia, or reflex excitation.

(*a*) In the infant, the meconium, if too long retained, may cause irritation (colica meconialis); in the adult the food may be coarse and irritating in quality, or decomposed, or the chyme may lack gastric digestion. Unripe fruit, cold and acid drinks and food, are common causes. Retained scybalous masses, foreign bodies, or an accumulation of gas may cause colic through pressure or stretching of the intestinal wall.

(*b*) The blood may contain bacterial toxins which are irritating, as in cholera and malarial cachexia, or such poisons as uric acid, lead, copper, or arsenic.

(*c*) The reflex causes are many. They include organic disease of the brain or cord and the crises of locomotor ataxia, hypochondriasis, and

hysteria. Chilling of the surface of the body produces enteralgia in some persons.

Symptoms.—Pain is the principal symptom. This is usually referred to the umbilical region, from which it may radiate. Sometimes it begins in several locations at the same time. It is generally periodical and it may be a dull aching or of a sharp, lancinating character, usually with increasing intensity. Tenderness may be complained of, but pressure may give relief, and the patient often lies on the stomach or with the knees drawn up, or bends over a chair. The abdomen is either tympanitic or retracted. The peristaltic movements of the bowel may be visible. Rumbling noises often accompany the attack. In severe cases the body is bathed in a profuse sweat and the face becomes pale. Nausea is sometimes complained of; vomiting is unusual. The pulse is generally tense, but slow. Reflex symptoms may be observed, as palpitation, dyspnea, hiccough, rectal or vesical tenesmus, strangury, priapism, vertigo, syncope, cramps of the voluntary muscles, occasionally convulsions. The attack may last only a few minutes, or it may continue for hours or days, finally ceasing suddenly or gradually.

Diagnosis.—The condition is to be differentiated: (1) From peritonitis by the absence of fever and marked abdominal tenderness; (2) from appendicitis by the absence of tenderness at McBurney's point, rigidity of the right rectus, fever, and induration; (3) from intestinal obstruction by the absence of localized tenderness, obstinate constipation, and stercoral vomiting; (4) hepatic and renal colic by the different character and different location of pain. (5) Rheumatism of the abdominal walls is rare; the pain is superficial and aggravated by pressure or movement. (6) In lumboabdominal neuralgia the pain is unilateral and there are generally characteristic tender points.

The ***prognosis*** is generally favorable, but relapses usually occur, unless the cause can be removed.

Treatment.—This is directed to the relief of pain and to the removal of the cause.

In severe cases, morphin must be injected hypodermically. Mild cases are generally relieved by the administration of spirit of peppermint or compound spirit of sulphuric ether, with camphorated tincture of opium, chloroform or tincture of ginger, capsicum or camphor, or various combinations of these remedies. Hot poultices, fomentations, turpentine stupes, and the hot-water bag are serviceable in the intervals.

Removal of the cause embraces: (*a*) Relief of constipation by enemata and laxatives; (*b*) exclusion of irritating articles from the diet; (*c*) remedies to assist digestion, when any of these causes are present. (*d*) When a tendency to neuralgia is recognized, arsenic or quinin should be employed. (*e*) A gouty, rheumatic, neurotic taint and the various diseases named as predisposing causes must be treated.

MUCOUS COLITIS.

MEMBRANOUS COLITIS, MUCOUS COLIC, MUCOUS OR TUBULAR DIARRHEA.

Definition.—A chronic secretory neurosis of the intestine characterized by the discharge of mucous shreds or long tubular mucous casts of the mucosa of the colon.

Etiology.—This rather rare affection may occur at any period of life, from childhood to old age, but is more prevalent in women, particularly neurotic, hysterical, or neurasthenic subjects and those debilitated by organic nervous disease. The attack is commonly induced by mental emotion or the eating of improper food.

Morbid Anatomy.—No anatomical lesions are present. The mucous shreds and casts, when detached, leave the surface of the mucosa in a normal condition. The casts consist of mucin, not fibrin.

Symptoms.—The disease is generally marked by periodical attacks of enteralgia accompanied with abdominal tenderness most marked at the splenic flexure of the colon, and tenderness, during or after which the characteristic shreds or casts are discharged. These may accompany defecation or pass independently of it. Pain and tenesmus often occur during the course of the disease, without the discharge of casts. Disordered digestion sometimes precedes the attack for a few days. The pain may be severe, and pronounced nervous manifestations, hysterical in nature, may accompany the attack or develop upon the discovery of the casts. Constipation is usually present. Slight hemorrhage rarely accompanies the extrusion of the casts. Fever is absent. Emaciation results from long-continued colitis. The attack may last from a day to a week or longer, and the disease may persist, with variable intervals, for many years.

Diagnosis.—The casts should be carefully examined, microscopically if necessary, in order to exclude fragments of tapeworm or undigested remnants of food, as the skin of sausage, husks of various vegetables, or the pulp of orange or other fruit. The differentiation from the other painful affections of the abdomen is the same as that of enteralgia.

Prognosis.—Complete cure can sometimes be secured through improvement of the general health, but the disease is a stubborn one. Death has occurred during the attack.

Treatment.—The treatment is for the most part that of the underlying condition. The painful attack should be relieved, if possible, with the carminatives and local applications recommended for enteralgia. Morphin should not be given, for a habit is readily acquired by these patients. Constipation is to be relieved and the diet so regulated as to avoid irritation.

Intestinal Sand (Sable Intestinale).—It occasionally happens that large quantities of material resembling sand or gravel pass from the bowels. The sandlike particles generally consist of vegetable sclerenchymatous matter, sometimes of the seeds of such fruit as raspberries or blackberries. Very rarely biliary sand is discharged. True intestinal sand, consisting of the carbonates and phosphates of calcium and magnesium, has been observed. C. H. Bedford, England, reports a case of this character, associated with colitis and constipation, in an extremely gouty woman of 44 years.

DISEASES OF THE MESENTERY.

The mesentery is seldom the seat of primary disease. The secondary affections are considered in connection with the diseases that bear a causal relation to them. (1) Hemorrhage is rare and usually associated

with hemorrhagic pancreatitis. (2) Embolism and thrombosis of the mesenteric arteries are occasionally encountered. (See Hemorrhagic Infarction of the Intestine.) (3) The mesenteric artery is one of the least frequent sites of aneurism. (4) The mesenteric glands are enlarged in typhoid fever, tuberculosis, syphilis, and occasionally in Hodgkin's disease and other affections. (5) Malignant growths, hydatid, chylous, and other cysts sometimes occur.

DISEASES OF THE LIVER.

ANOMALIES OF FORM AND POSITION.

Malformation may be congenital or acquired. (1) Congenital malformation is rare. The only examples of it are seen in livers showing (*a*) disproportion in the size of the lobes, or (*b*) lobulation, which is generally a result of hereditary syphilis.

(2) Acquired malformation results from a great variety of influences, as: (*a*) The corset or lacing liver of women. It is characterized by a transverse groove running across the right lobe in a position corresponding to the lower margin of the ribs. In extreme cases the furrow is narrow and deep and the compressed portion is transformed into fibrous tissue with hardly a vestige of hepatic structure. The blood-vessels are to a great extent obliterated. The lower margin of the organ may rest between the umbilicus and crest of the pubis. Occasionally the liver has a pyramidal shape, with the apex downward. (*b*) Deformity of the vertebræ or ribs, and tumors of the surrounding organs or structures, frequently cause an alteration of the shape of the liver. (*c*) The alterations of size and form due to disease will be referred to in their proper relations.

Symptoms may be absent. In some cases there is a sensation of dragging or pressure. The part of the liver below the constriction becomes inflamed, swollen, and painful. Vomiting, prostration and jaundice occasionally occur, especially after unusually tight lacing. The prominent lower portion of the liver may be mistaken for a neoplasm, amyloid disease, or passive hyperemia.

Malposition.—The liver may be displaced upward, downward, or laterally, and the displacement may be congenital or acquired.

(1) *Congenital displacement* is met with: (*a*) In the rare condition of transposition of the viscera, when it occupies a position on the left side corresponding to its normal position on the right. (*b*) The organ may be found in a hernia of the diaphragm or abnominal wall. There may be no interference with its function in these cases. (*c*) The so-called suspensory ligament may be of unusual length, permitting descent or lateral movement. In extreme cases the organ lies in the epigastric region or sinks to the lower part of the abdominal cavity.

(2) *Acquired Displacement.*—(*a*) The liver may be raised by ascites, abdominal tumor, or intestinal distention. (*b*) It may be lowered by pleuritic effusion, emphysema, or an intrathoracic tumor of large size, rarely by extensive pericardial effusion or subphrenic abscess.

Symptoms.—Tension and dragging are the usual symptoms. There may be occasional attacks of pain, which is often referred to the right shoulder.

Diagnosis.—The condition is apt to be confounded with various neoplasms of the stomach, ovary, uterus, kidneys, or with hydronephrosis or pyonephrosis. The diagnosis is generally based upon the absence of hepatic dullness in the usual place, but this sign may mislead when cirrhosis or fatty degeneration is present.

Treatment.—The liver can generally be replaced without much difficulty. A suitable bandage should then be worn in order to prevent recurrence of the displacement.

DISTURBANCES OF THE HEPATIC CIRCULATION.

Anemia.—It is assumed that anemia of the liver accompanies the general deficiency of blood after profuse hemorrhage and in the primary anemias. The liver is found to be almost bloodless after death in these conditions as well as in amyloid disease, fatty degeneration, and other conditions. There are no clinical manifestations, however, through which the condition can be recognized.

Hyperemia of the liver is a condition common to many diseases. It may be either active or passive.

1. *Active Hyperemia* (Active Congestion).—(*a*) A physiological hyperemia of the liver is believed to occur after every full meal, owing to the increased activity of the portal circulation, especially if alcohol be ingested. In either case the condition is transitory, but in the gormand or drunkard it leads to permanent changes, especially to cirrhosis. (*b*) Other causes of the hyperemia are the toxins of disease, especially those of malaria, dysentery, typhoid or typhus fever, erysipelas and yellow fever, exposure to cold, amenorrhea, and the suppression of habitual hemorrhoidal bleeding, and (*c*) the toxic products of intra-intestinal fermentation and such intoxications as accompany gout and diabetes.

The ***symptoms*** are generally due to associated catarrh of the stomach, duodenum, or bile-ducts. There may be a sense of fullness or pain, slight enlargement, and tenderness of the liver; less commonly, slight jaundice, enlargement of the spleen, and a bilious diarrhea.

Treatment.—For the local condition a calomel or saline purge is indicated. Hot or cold applications to the hepatic region relieve the abnormal sensations. Beyond this the treatment is that of the underlying disease.

2. *Passive Hyperemia* (Chronic Congestion of the Liver, Nutmeg Liver).—***Etiology.***—This condition results from obstruction of the flow of blood from the liver. (*a*) The common seat of obstruction is in the heart. Any form of uncompensated valvular disease may excite it, but diseases of the right heart act more directly. It may result also (*b*) from obstruction of the pulmonary circulation as in emphysema, chronic interstitial pneumonia, or from deformity of the spine or extensive pleuritic effusion; (*c*) from obstruction of the ascending vena cava or the hepatic vein when compressed by an aneurism or other tumor situated anywhere in its course. (*d*) The condition has rarely been caused by valves or other projections within the veins or by the presence of constricting bands.

Morbid Anatomy.—The liver is uniformly enlarged, of a dark red or

[illegible] In a case of [illegible] slightly, if at all, [illegible] connective tissue [illegible] dilatation [illegible] of the fibrous [illegible] and fatty [illegible] The spleen is [illegible] and the [illegible] normal, and the [illegible]

Symptoms.—[illegible] until late in [illegible] sensation of weight and fullness [illegible] disturbances [illegible] Jaundice is occasionally [illegible] pigments [illegible] Ascites and dilated veins are common, [illegible] of the hepatic congestion. [illegible] enlargement of the liver.

Treatment.—[illegible] directed to the affection causing [illegible] sometimes be necessary to [illegible] through the methods [illegible]

[illegible]

Hemorrhage.—Hemorrhage into the substance of the liver may result from a trauma [illegible] such diseases as purpura, [illegible] Rarely there may be a single hemorrhage, usually [illegible] numerous small hemorrhages. When rupture of one of the portal [illegible] so great a quantity of blood [illegible] fatal syncope.

Embolism and Thrombosis of the Portal Vein.—(1) *Embolism.*—The [illegible] of the portal vein may be obstructed by emboli originating in the [illegible] Infarction is not general, [illegible] owing to the free anastomosis with the hepatic artery. In the event of septic embolic suppuration is to be feared.

(2) *Thrombosis.*—Thrombi seldom form in the portal vein. They are occasionally met with, however, as a result of (1) Cirrhosis; (2) cancer; (3) sclerosis with roughening of the vessel-wall; (4) local inflammation following trauma, abscess, or foreign bodies; (5) slowing of the circulation by the pressure of a tumor, enlarged glands, abscess, impacted calculi, or proliferative peritonitis.

Morbid Anatomy. The thrombus undergoes the usual changes, except that organization is unusual. In rare instances, the collateral circulation becomes complete and the portal vein is reduced to a fibrous cord (*pylephlebitis adhesiva*). Infarction is rare. In parietal thrombosis the circulation may be gradually restored.

Symptoms. The symptoms are generally slight or entirely absent. When the obstruction is complete, the manifestations are the same in character as those due to obstruction in hepatic cirrhosis. The treatment is also the same.

Suppurative Pylephlebitis (Suppurative Inflammation of the Portal Vein).—***Etiology.***—Septic emboli originate from processes of suppuration in the peritoneal cavity, most frequently from an appendiceal focus, an intestinal or gastric ulcer, or suppuration within the pelvis. It sometimes follows infection of the umbilicus in the new-born infant, or it may be a part of a general pyemia.

Symptoms.—(*a*) In many cases the condition leads to the formation of one or many distinct abscesses. (See Abscess of the Liver.) (*b*) In other cases the suppurative process remains confined to the portal vein. The liver is enlarged, tender, and often painful. In other respects the symptoms are those of pyemia—irregular fever, occasional chills and sweats with enlargement of the spleen, headache, loss of appetite, scant urine, jaundice, and diarrhea. The disease usually lasts from one to four weeks, invariably terminating fatally, generally in delirium or coma. The diagnosis is often extremely difficult in the absence of distinct abscess-formation.

Stenosis of the Portal Vein.—Extensive obliteration of the branches of the portal vein is a common result of cirrhosis and syphilis. Stenosis may result also from the presence of thrombi or the pressure of tumors.

Symptoms may be absent, if the stenosis form slowly and permit the establishment of compensatory circulation. When it is due to thrombosis, edema and ascites may suddenly develop. The diagnosis is difficult in either condition.

Affections of the Hepatic Artery and Vein.—These include dilatations and obliterations, aneurism, embolism, thrombosis, and other changes, for the most part due to secondary inflammation, pressure by tumors, adventitious connective tissue in cirrhosis, and the various lesions of syphilis. They are all of greater interest to the pathologist than to the physician, since they are seldom recognizable during life.

Hepatalgia.—An independent neuralgia of the liver is believed to occur in neurasthenic cases, or in connection with neuralgia of other regions. It is thought at times to be due to malarial infection, and similar pain accompanies abscess, neoplasms, active or passive hyperemia. The differentiation of many cases from the colic of gall-stones is extremely difficult, if not impossible. The treatment is usually directed against the cause of the affection; but temporary relief may be afforded by hot applications and counter-irritants.

ACUTE HEPATITIS.

ACUTE YELLOW ATROPHY, ACUTE PARENCHYMATOUS HEPATITIS, MALIGNANT JAUNDICE, ICTERUS GRAVIS.

Definition.—An acute disease of the liver characterized by a rapid destruction of the parenchyma cells, and consequent atrophy and softening of the organ, with jaundice, hemorrhages, and grave cerebral manifestations.

Etiology.—1. The disease is rarely primary. It has usually been observed in the third decade of life, but occasionally in infants. 2. The secondary form is not at all frequent. It has been seen with apparently greater frequency in some localities than in others, and groups of cases occasionally occur in the same locality within a comparatively

short time, giving them the appearance of an endemic. The disease is more common in women. Pregnancy, puerperal and typhoid fevers, diphtheria, septicemia, and malaria are regarded as predisposing causes. A microbic origin has been suggested. Acute phosphorus-poisoning produces a rapid fatty atrophy of the liver, but it is not identical with this disease. The same statement doubtless applies to the fatty necrosis which is a terminal stage of cirrhosis and obstructive jaundice. Some writers assert that an acute yellow atrophy may follow a debauch or strong mental emotion.

Morbid Anatomy.—The external surface of the body is extremely jaundiced. The degree of emaciation is variable. The liver is much reduced in size, and, as a rule, so flaccid that it sinks back against the posterior wall of the abdomen as soon as air has been admitted. In very rare cases it has been found enlarged. Its external color is a dirty yellow. On section it may be uniformly yellow, but there are often dark red patches which correspond to areas from which all the fat has been absorbed. After exposure to the air, crystals of leucin and tyrosin are formed on the surface. Microscopic examination reveals an extreme fatty degeneration, with almost complete destruction of the cellular elements. A variable degree of cellular infiltration is to be seen in the interstitial connective tissue, and here and there clusters of cells that are regarded as new-formed bile capillaries. Crystals of leucin and tyrosin are present in varying abundance. The heart muscle shows fatty change, the spleen is enlarged, the kidney epithelium is degenerated, and the entire alimentary tract shows catarrhal changes. The pleural and pericardial fluids are often increased.

Symptoms.—1. The disease usually begins as a gastrointestinal catarrh, with loss of appetite, eructations, nausea, vomiting, constipation or irregularity of evacuation, headache, and general prostration. In a few days jaundice begins to appear.

2. At an indefinite time, from three or four days to two or three weeks from the beginning, the symptoms suddenly become intensified. (*a*) The jaundice deepens, and vomiting becomes constant. The vomit at first consists of bile-stained stomach-contents, later containing blood. (*b*) The headache becomes intense, nervousness develops, and the patient may finally pass into a maniacal delirium. (*c*) Muscular twitchings or general convulsions are sometimes observed. This stage is often accompanied with (*d*) hemorrhages into the skin (ecchymoses), from the nose, stomach, bowels, kidneys. (*e*) The temperature is variable; fever may be absent until shortly before death, when hyperpyrexia often develops. An afebrile course is sometimes observed, and a subnormal temperature may be present at the end. (*f*) During the severe stage, the size of the liver rapidly diminishes and the hepatic region becomes tender and often intensely painful. (*g*) The spleen is usually enlarged. (*h*) The urine becomes scant, deeply bile-stained, and sometimes albuminous. The urea is greatly reduced or entirely absent. Leucin, tyrosin, and other abnormal products of less importance, as creatinin, and sarcolactic acid, are usually found in it. Constipation is generally present in the latter part of the disease, the stools being clay-colored from absence of bile, unless they be darkened by the presence of altered blood.

Diagnosis.—The recognition of the disease in the initial stage is seldom possible. When, however, the acute stage develops with typical symptoms, especially if in a pregnant woman, there is seldom room for doubt. The conditions suggested are generally limited to hypertrophic cirrhosis and acute phosphorus-poisoning. *Hypertrophic cirrhosis* affords a different history; the area of hepatic dullness is greatly increased and the urea is generally increased. The last is a more valuable symptom than the absence of leucin and tyrosin. *Acute phosphorus-poisoning* is generally recognized by the history of the ingestion of the poison, the sudden onset, with nausea, vomiting, and pain in the liver, the appearance of jaundice on the second or third day; in some cases, by phosphorescence of the vomit and stools, and the absence of leucin and tyrosin from the urine. The symptoms ameliorate after the appearance of jaundice, and hematemesis develops toward the close of the disease.

Prognosis.—The primary form is regarded as inevitably fatal. Recoveries from the secondary form are occasionally reported.

Treatment.—All measures should be directed to the comfort of the patient. There is no curative treatment. For the vomiting, cracked ice, dram doses of paregoric, and other remedies should be employed. For the nervousness or delirium, an ice-bag to the head and the administration of hydrobromic acid, chloral, or camphor are beneficial. In extreme cases, hypodermic administration of morphin should not be withheld. The strength of the patient should be maintained by the most nutritious diet and free stimulation with brandy and strychnin.

CIRRHOSES OF THE LIVER.

Definition.—A chronic proliferative inflammation of the interstitial tissue of the liver which results in atrophy of the parenchyma and the production of circulatory, gastrointestinal, and other more remote disturbances. There are two principal forms of the disease, the atrophic and the hypertrophic.

ATROPHIC CIRRHOSIS.

SCLEROSIS OF THE LIVER, INTERSTITIAL HEPATITIS, FIBROUS HEPATITIS, HOBNAIL LIVER, GIN-DRINKER'S LIVER, ALCOHOLIC CIRRHOSIS.

Etiology.—1. *Sex and Age.*—The disease is commonly seen in men from 40 to 60 years of age, but it has been met with in women, and in children from 4 to 12 years old. In children, however, it is usually a manifestation of inherited syphilis or a result of one of the acute infections, especially scarlet fever, typhoid fever, intermittent fever, dysentery, or tuberculosis, influences which are regarded also as occasional causes in adults.

2. The *exciting cause* is an irritation of the connective tissue. (*a*) In a considerable majority of all cases, the irritation is produced by alcohol carried to the liver in excessive quantities through the portal circulation. (*b*) Some other influence is believed by some writers to be operative in most cases, on account of the fact that the disease is by no means universal, even among habitual drunkards. It is believed, on the one hand, that liquor containing a large percentage of fusil oil

is more active in producing the disease. The disease is more certain to develop in those who drink undiluted whisky, brandy, gin, or rum when the stomach is empty, than in those who take their "eye-openers" and "night-caps," with a nip before meals for an appetite. Some few have attributed it to a greater extent to the drinking of strong wines. In not a few cases there is a history of former syphilitic infection, and this is, no doubt, a potent element in etiology. (*c*) The free use of spices and even of coffee has been regarded as the cause of the disease. (*d*) Poisoning with ptomains is now regarded as an important factor by some investigators. Autointoxication due to the indigestion and abnormal fermentation excited by alcoholism is thought to hasten the interstitial inflammation. (*d*) Obstruction of the bile-ducts is believed to cause a form of cirrhosis in some instances. (*e*) The disease has been produced experimentally in a fairly typical form through the action of lead, silver, arsenic, antimony, phosphorus, butyric, valerianic, and other organic acids, croton oil, and carbolic acid. Dead tubercle bacilli and the toxins of several bacteria have been thus employed, administered with the food, injected into the liver or into the blood. (*f*) Syphilis of itself produces a form of cirrhosis which will be considered separately. The fibrous induration which forms about foreign bodies, abscesses, and other inflammatory products, and the sclerosis which is sometimes seen as a result of chronic passive hyperemia, are not proper examples of the disease under consideration.

Morbid Anatomy.—Although the disease is recognized clinically as a progressive one, most authors describe the conditions found after death as belonging to one or other of two types, which are doubtless different stages of the same process. These are known as the fibrous or atrophic and the fatty forms. The essential feature in each is the excessive formation of fibrous tissue in the interstitial spaces.

(*a*) *Atrophic Cirrhosis.*—In this form the new connective tissue has undergone marked contraction at the expense of the parenchyma cells, reducing the size of the lobules, and consequently that of the entire organ. Many of the acini are entirely destroyed, many remain as tufts of comparatively few cells. The liver is greatly diminished, even to less than half its normal dimensions and weight, and it is much firmer than normal. The surface is roughened by numerous depressions, between which are the "hobnail" prominences. The left lobe is sometimes converted into a narrow, hard, fibrous appendage without a vestige of normal liver tissue in it. The color is a tawny yellow, sometimes lighter, sometimes darker. Another result of the fibrous-tissue contraction is the compression and obliteration of the radicles of the portal vein and of the smaller bile-ducts. Thrombophlebitis is sometimes set up.

(*b*) *Fatty Cirrhosis.*—In this there is an extreme fatty degeneration, sometimes confined to the outer zone of the lobule, sometimes affecting all parts of it to such an extent that scarcely a trace of liver-cells remains. The pathological process has generally been regarded as beginning in the fibrous interstitial tissue and affecting the parenchyma secondarily, but it has been suggested by Weigert and others that the disease begins in the parenchyma. The size of the organ may not be so markedly reduced as in the fibrous form, but there is pronounced obstruction of the portal circulation in extreme cases.

Results of Portal Obstruction.—1. Anastomotic communications are established between the branches of the portal vein and those of the vena cava. Almost every conceivable avenue of communication is opened up in some cases. The most frequent are: (*a*) Dilatation of the veins in the suspensory and round ligaments. In the latter a large vessel is sometimes found which is regarded by some as a reopening of the obliterated umbilical vein, and there may be associated with it a coil of dilated vessels around the umbilicus known as the caput Medusæ. (*b*) A free communication is established between the two systems through the gastric and inferior esophageal veins; (*c*) through the inferior mesenteric and hemorrhoidal veins; (*d*) through the retroperitoneal plexus of veins, reaching the kidneys and other organs; (*e*) through the epigastric and internal mammary veins.

2. Another result of portal obstruction, although possibly influenced in some instances by the development of endophlebitis, is an accumulation of fluid in the peritoneal cavity. The peritoneum becomes opaque, and the quantity of fluid poured out is sometimes enormous. Anasarca is also commonly present.

3. The spleen is almost invariably enlarged; in part, perhaps, as a result of the general venous tension; possibly, as has been suggested, as a result of autoinfection. The kidneys are generally hyperemic.

A common result of the obstruction of the bile-ducts, in the late stage of the disease, is the jaundice. Rows of cells suggestive of the formation of new ducts are frequently seen in the microscopic sections.

Symptoms.—1. The early manifestations of the disease are indefinite. They consist, for the most part, of digestive disturbances, anorexia, morning nausea or vomiting, constipation or diarrhea, with epigastric weight and hepatic tenderness, symptoms which may be attributed with equal propriety to the gastric and intestinal catarrh consequent upon the direct action of the alcohol.

2. The more important symptoms of cirrhosis proper are due to portal obstruction. (*a*) The gastrointestinal disturbances become more pronounced and more constant, the appetite capricious, the action of the bowels irregular, and other evidences of a severe gastrointestinal catarrh develop. (*b*) Hemorrhages of the stomach or intestines occur in many cases, generally in an advanced stage of the disease, but sometimes early. When the bleeding is confined to the stomach, the blood is vomited; a fatal hematemesis may take place. Hemorrhage in the intestine leads to melena, or tarry stools. Much blood is often lost in the course of the repeated hemorrhages, for they may recur during the course of a year or more. (*c*) Repeated epistaxis occurs in some cases. (*d*) Hemorrhoids are generally mentioned, but they are by no means uniformly present. (*e*) *Edema.*—Ascites does not usually develop until the liver has undergone considerable atrophy, but it is one of the most constant and characteristic manifestations of the disease. Edema of the ankles or of the genitals is often the first symptom to attract the attention of the patient, but the ascites always precedes it. The quantity of fluid accumulated is often enormous; as much as 20 quarts (liters) may be present. After tapping has been performed, the fluid generally reaccumulates at intervals of a few weeks during the remainder of life. Toward the close, the edema often reaches the pleural and peri-

cardial cavities, and death may be due directly to edema of the lungs. General dropsy is unusual. (*f*) A more or less general dilatation of the veins over the surface of the body is sometimes observed, and cutaneous hemorrhages may occur. All these manifestations may be prevented by an early development of compensatory circulation, or they may subside after its development. (*g*) The spleen is to a greater or less extent enlarged in all cases, and this adds to the abdominal distention. (*h*) Jaundice generally appears late in the disease, but sometimes comparatively early. It is ordinarily slight and may recur at intervals.

3. Toxic symptoms are apt to develop at any time during the course of the disease. They generally assume the form of a mild delirium. Their exciting cause is not definitely known. They often develop after weeks of abstinence from alcohol. Uremia and cholemia are generally cited as the probable causes, but there is often no evidence of a retention of either urea or bile.

4. Fever is not a common symptom, but it occurs at times in some cases, particularly toward the close of the disease and after tapping.

5. With the progress of the disease the patient becomes more and more emaciated. The face becomes sallow, often slightly icteric; the conjunctivæ especially show the discoloration. The abdomen becomes large and contrasts strongly with the wasted extremities.

6. Physical examination reveals slight enlargement of the liver, possibly tenderness, in the beginning, but later the area of dullness is greatly diminished. The roughened surface can occasionally be felt. The spleen can generally be recognized on palpation. The ascites yields characteristic dullness and fluctuation.

7. The urine is generally reduced in quantity, always deeply colored, of high specific gravity, and often contains albumin. The urea is usually diminished, probably on account of deficient production in the liver.

Diagnosis.—The disease is not usually recognizable until the sclerosis has become well established. The development of ascites after a prolonged gastrointestinal catarrh in an individual long addicted to excessive indulgence in alcohol, especially if there be slight jaundice, the hepatic facies, and emaciation, is generally conclusive evidence of cirrhosis. The only disease to be excluded in most cases is carcinoma. Chronic or tubercular peritonitis must sometimes be considered.

Carcinoma is generally attended with greater anemia, fully as marked emaciation, and a cachexia that is quite different from the icteric tinge. A family predisposition can often be traced. The liver is generally enlarged and distinct; umbilicated nodules can often be felt in an advanced case. The differentiation is often difficult in a case of primary carcinoma affecting an alcoholic subject.

Chronic or tubercular peritonitis is generally attended with elevation of temperature. Tuberculosis of other organs may be found. The skin is pale and anemic; jaundice is not present. The spleen is not, as a rule, enlarged.

Prognosis.—The disease is incurable, and, as a rule, it runs a rapidly fatal course, except in those rare instances in which the drink habit is broken off. When this is done, the patient may improve, with the development of anastomotic circulation and the relief of abdominal tension through tapping. But the improvement is generally only temporary,

and a fatal termination may be anticipated within a year at most after the occurrence of a hemorrhage or the development of ascites.

Treatment.—All possible sources of irritation to the liver should be promptly removed. The ingestion of alcohol must be stopped, and the diet should be so regulated as to exclude all irritating substances, even coffee. The milk diet is probably the best, at least until the gastric irritation has subsided and the more active hepatic hyperemia has had time to abate. The bitter tonics should be employed to stimulate the appetite, especially gentian and quassia, and dilute hydrochloric or nitrohydrochloric acid should be given to aid digestion. The latter acid was formerly thought to have specific action.

An attempt to diminish the ascitic accumulation may be made through the administration of diuretics, especially potassium bitartrate or acetate, or squill, with digitalis in suitable cases. But it is generally found necessary to resort to the hydrogogue purgatives, as compound jalap powder, every morning, or eleterin once or twice a week. Carefully administered Turkish or Russian baths are often of great benefit in strong patients.

Tapping becomes necessary in most cases. It does not require the skill of a surgeon, but it should be performed under careful antisepsis with a large aspirator needle or a trochar introduced in the median line about midway between the umbilicus and the pubis, the patient sitting, and after evacuation of the bladder. A more extensive surgical treatment has been employed in a few cases, the abdomen being opened, and the surface of the liver and peritoneum thoroughly scrubbed in order to induce adhesive inflammation.

Hemorrhage is best controlled by cold applications to the abdomen and the administration of opium in full doses. Ergot is employed by some physicians; astringents are useless.

Potassium iodid may be employed when there is a history of syphilis, or experimentally in one who denies such infection, but it is not of benefit in simple alcoholic cirrhosis, even when it affects a syphilitic subject. Its effect in syphilitic cirrhosis is quite marked, especially in the early part of the disease.

A return to alcoholic stimulation may become necessary late in the course of the disease, simply as a means of prolonging the patient's existence.

HYPERTROPHIC CIRRHOSIS.

Hanot's Disease.

Definition.—A rare form of hepatic sclerosis attended with marked increase in the size of the liver.

Etiology.—The disease is generally observed in men during early adult life, but it sometimes attacks women or children. The exciting cause is not known. There is no evidence that it is to any extent caused by alcoholism. In one form of the disease (biliary cirrhosis), it is believed to originate from an obstruction of the bile-ducts with calculi.

Morbid Anatomy.—The liver is uniformly enlarged, its surface is smooth or slightly granular, and the section shows a tawny, cirrhotic color. Histologically, the appearances are the same as those of atrophic cirrhosis, except that the hyperplastic connective tissue shows no tendency

to contract. A new growth of bile capillaries is believed to take place, especially in the biliary form. The liver cells sometimes appear hypertrophied; but fatty degeneration and destruction of the cells do not occur, as in the atrophic form. The spleen is enlarged and the abdomen is distended, but there is no ascites.

Symptoms.—1. Enlargement of the liver is often the first manifestation of the disease. Either lobe or the entire organ may become prominent. The lower margin, sometimes found below the level of the umbilicus, is sharp and firm. The surface feels smooth. The gall-bladder is not enlarged. 2. Jaundice often appears early and persists throughout the long course of the disease. It is at first slight, but later may become extreme. A febrile jaundice (icterus gravis), with delirium, may develop at any time during the course of the case. The temperature runs up rapidly, even to 108° F. (42.2° C.), and death may ensue, in delirium or coma.

3. Paroxysms of pain and tenderness in the region of the liver sometimes occur, with nausea and vomiting. They may be the first symptoms.

4. Hemorrhages are a prominent feature from an early period. There may be bleeding from the nose, gums, stomach, or intestines; or a purpura may develop.

5. Other features are: (*a*) A chronic course, sometimes lasting 5 or 10 years; (*b*) an enlargement of the spleen; (*c*) the absence of ascites; (*d*) urticaria and other eruptions, occasionally noted; (*e*) the urine contains bile pigment and the stools are dark; they do not show the clay color of obstructive jaundice.

Diagnosis.—This is based on: (*a*) The chronic enlargement of the liver and spleen; (*b*) the persistent jaundice; (*c*) a tendency to hemorrhages; and (*d*) the absence of ascites. The affection is to be differentiated especially from amyloid disease, sometimes from abscess or carcinoma of the liver, or echinococcus cyst.

Amyloid disease follows a history of suppuration, tuberculosis, or syphilis; the skin is waxy, seldom jaundiced. The liver is hard, and other organs are affected. *Abscess* usually follows dysentery; its course is febrile and of shorter duration. The hepatic enlargement soon becomes sensitive, and fluctuation develops. *Carcinoma* is differentiated under Atrophic Cirrhosis. In *echinococcus cysts* the enlargement is cystic, and hydatid purring may be felt. The aspirator withdraws characteristic fluid.

Treatment.—The treatment, like that of atrophic cirrhosis, is chiefly symptomatic, and at best has little influence on the course of the disease.

Syphilitic Cirrhosis.—The syphilitic diseases of the liver are considered in the general chapter on Syphilis (p. 164).

PERIHEPATITIS.

CAPSULAR CIRRHOSIS.

An acute and a chronic form of perihepatitis are recognized.

1. **Acute Perihepatitis** (Subphrenic or Subdiaphragmatic Pyopneumothorax, Subphrenic Abscess).—A fibrinous or suppurative inflam-

mation involving the contiguous surfaces of the hepatic and diaphragmatic peritoneum.

Etiology.—The disease is rarely primary except when the result of injury. The principal causes of fibrinous perihepatitis are: (*a*) Extension of inflammation from a diaphragmatic pleurisy or empyema; (*b*) abscess or cyst of the liver, gall-bladder, right kidney, or contiguous parts. The suppurative form may arise from any of these causes, but is more commonly a result of the perforation of gastric, duodenal, or other intestinal ulcers, particularly of appendiceal perforation. It occasionally develops in pneumonia or malignant disease.

Morbid Anatomy.—The conditions are the same as those found in a fibrinous or suppurative peritonitis. A localized abscess is usually formed by the growth of adhesions. When a perforation has caused the condition, the pus is mingled with gas (subphrenic pyopneumothorax). Bile pigment and fatty acids may be found in the pus.

Symptoms.—The most prominent of these are: (*a*) Local pain and tenderness, increased by deep respiration; (*b*) fever, especially in the suppurative form, when chills and other indications of sepsis are commonly present; (*c*) dyspnea, as a result of the pressure of the subphrenic abscess. (*d*) Examination reveals generally a downward displacement of the liver, and marked prominence of the abdominal wall above; the skin may be inflamed and edematous; percussion reveals tympanites or flatness corresponding to the presence or absence of gas. A friction fremitus and crepitation are sometimes felt and heard over the region. (*e*) Rupture of the abscess may pour the contents into the pleural or general peritoneal cavity, rarely outward through the skin. The disease is differentiated from empyema of the right side chiefly by the abdominal character of the early symptoms, by the downward displacement of the liver, the absence of cardiac displacement, and other indications of empyema. Pus may be withdrawn by introducing a trochar at the seventh or eighth intercostal space. The flow is strong and is not arrested by inspiration as it is in pleuritic effusion (Pfuhl's sign).

Prognosis.—Acute fibrinous cases usually terminate favorably; the suppurative form, unless arrested by early surgical procedure, is exceedingly fatal. Recovery occasionally follows the spontaneous evacuation of the pus.

The ***treatment*** is the same as that of localized peritonitis, with the addition of surgical measures for the suppurative form.

2. **Chronic Perihepatitis** (Chronic Hepatic Capsulitis, Capsular Cirrhosis, Glissonian Cirrhosis).—A chronic inflammation of the serous covering of the liver, resulting in marked thickening and contraction, sometimes producing deformity or atrophy of the organ, with or without sclerosis of its interstitial tissue.

Etiology.—The disease is peculiar to adult life and is usually accompanied with perisplenitis, chronic interstitial nephritis, and chronic proliferative peritonitis or possibly chronic mediastinitis. Syphilis is regarded by Anders as a frequent cause, and tight-lacing has been mentioned. Its origin is generally obscure unless traceable to inflammation in adjacent tissues.

Morbid Anatomy.—The capsule is much thickened. The liver is greatly reduced in size as a result of compression.

Symptoms.—These are usually vague. Persistent or recurrent ascites, with reduction of the liver outlines, pain, and tenderness, associated with the manifestations of chronic interstitial nephritis, are the most frequent indications of the disease.

The treatment is the same as that of atrophic cirrhosis.

ABSCESS OF THE LIVER.

SUPPURATIVE HEPATITIS.

Definition.—A suppurative inflammation affecting the substance of the liver.

Etiology.—1. The *tropical abscess*, usually single, is generally met with in adult males. It is especially common in India and other tropical countries; less so in our Southern States, and cases occur occasionally in the North. (*a*) Idiopathic cases are recognized, cases, at least, in which there is no history of previous disease. (*b*) The disease is much more frequently a sequel of dysentery. (*c*) The ameba coli, streptococci, and staphylococci are found in the pus, and the ameba especially is regarded as a cause of the disease, since it is to be found in the stools, even independently of dysentery. Overindulgence in liquor, particularly by European residents of the tropics, is thought to be influential in some cases, possibly by developing duodenal and biliary catarrh.

2. *Embolic abscesses* are not frequent. They develop: (*a*) Generally as a result of infection through the portal vein, in connection with appendicitis, typhoid fever, dysentery, or the various intestinal ulcers; (*b*) less frequently through the hepatic artery, as from an ulcerative endocarditis; (*c*) rarely, perhaps, through the vena cava and hepatic vein, from a gangrenous focus in the lungs; (*d*) the disease is often a part of a general pyemia.

3. The *traumatic abscess* usually results from direct injury of the liver; occasionally it follows injury of the head.

4. *Suppurative colangitis*, inflammation of the bile-passages, due to the lodgment of gall-stones, to tuberculosis, or an extension of duodenal inflammation, is an occasional cause, especially of multiple abscesses.

5. *Foreign bodies*, as needles, pins, or fishbones, and such parasites as round worms, echinococci, or the distoma, acting as foreign bodies, sometimes produce abscesses.

Morbid Anatomy.—As a rule, there is a single large abscess; occasionally there are several of considerable size or numerous small ones (in the pyemic form).

1. The *solitary* or *tropical* abscess is usually single. It may have a capacity of five or six quarts (liters), excavating more than half the entire liver, and is then generally confined to the right lobe. It is only in abscesses of long standing that there is a distinct limiting wall. In those of more recent development the inner surface is necrotic, ragged; and beneath this there is a transition from necrotic to inflamed liver tissue. The pus is of different colors, from white to brown or green, often peculiar in odor and possibly containing amebæ, staphylococci, streptococci, or colon bacilli. It is sometimes sterile. Rupture of the abscess is not uncommonly discovered. The pus may be found to have

perforated the right pleural cavity, the peritoneal cavity, the bile-ducts, gall-bladder or colon, rarely even the pericardium or vena cava.

2. *Multiple* or *pyemic* abscesses may be extremely numerous, but a single abscess of pyemic origin is occasionally met with. The pus varies in appearance as in other abscesses and is often bile-stained. When an obstructive colangitis is present, gall-stones are usually found, and extensive suppurative inflammation may extend through almost the entire system of bile-ducts and the gall-bladder. Suppuration about foreign bodies and parasites is often quite extensive, but a distinct abscess is usually formed.

Symptoms.—1. *The Solitary Abscess.*—The severity of the symptoms varies in different cases. Death sometimes occurs from the rupture of an abscess, especially in the tropics, before the condition has been recognized. In most cases, however, the condition is recognized through local pain and tenderness, enlargement of the liver, and elevation of temperature, with other indications of sepsis.

(*a*) The *pain*, usually of a dull aching character, may be confined to the hepatic region, but is generally referred to the back and shoulder. The *tenderness* is usually greatest at the margin of the ribs in the mammary line. A dragging sensation is sometimes complained of when the patient lies upon the left side.

(*b*) The *enlargement* is confined in most cases to the right lobe and is greatest above and to the right, so that the line of dullness in the axillary line is often as high as the fifth interspace, and it may rise posteriorly nearly to the angle of the scapula. The prominence may be distinctly visible. Palpation often reveals decided tenderness, especially when deep pressure is made. Fluctuation is sometimes detected. After adhesions have formed between the peritoneal surfaces over the abscess, the skin becomes red and edematous, as "pointing" progresses.

(*c*) The *fever* is usually of an intermittent type, or it may be irregular. It is sometimes accompanied with chills, sweating, and other indications of sepsis. The case often resembles one or other of the forms of malaria, or it may suggest a cachectic condition. In the more chronic cases fever may be absent, but the appearance of the patient becomes highly distinctive. The face is pale, generally sallow, the skin is usually slightly icteric, seldom deeply so. Constipation may be present, but diarrhea is a more usual condition; nausea, vomiting, and other indications of digestive disturbance are common. Ascites sometimes develops. Pressure symptoms, especially dyspnea and cough, commonly result from the elevation of the diaphragm, and a suppurative pleurisy is sometimes induced without actual rupture of the abscess. When rupture occurs, the pus is discharged in any of the directions already referred to, especially into the pleural or peritoneal cavity or one of the hollow viscera. The case may terminate fatally from the results of perforation or from profound septicemia. The duration of the disease, when not relieved through surgical measures, is exceedingly indefinite, varying from one or two months to as many years.

2. *Pyemic Abscesses.*—Although the liver may be extensively involved, the condition may escape notice, especially when it is a part of a general septic infection. The symptoms, aside from slight pain, localized tender-

ness, and some enlargement of the organ, and perhaps an icteric tinge of the skin, are those of septicemia or pyemia. The condition can rarely, if ever, be distinguished from that due to a suppurative pylephlebitis.

Diagnosis.—The diseases most frequently to be excluded are malaria, empyema, pylephlebitis from impaction of calculi, and other affections causing enlargement of the liver.

Malaria is most likely to be suspected in tropical and other malarious regions. It is readily excluded, however, by the absence of plasmodia or free pigment, and the presence of a decided leucocytosis. The fever resists quinin. In malaria there is usually a history of previous attacks, and the temperature curve is more uniform. If chills occur, they are at more definite intervals than in sepsis. The spleen becomes enlarged, and the color of the skin is usually darker than that accompanying abscess.

Empyema of the right side gives us a history of pleuritic pain, possibly of injury to the chest or the rupture of a tuberculous or emphysematous cavity, the side of the thorax becomes distended, the intercostal spaces obliterated, the lung is displaced upward, the heart is pushed to the left, and the fluid level changes with change of position. The aspirator needle reveals pus above the diaphragm, not below it. When, however, a hepatic abscess ruptures into the pleural cavity, an empyema is set up with a history of previous subdiaphragmatic symptoms.

Pylephlebitis occasions symptoms not readily distinguishable from abscess, and the suppurative form sometimes leads to the formation of localized abscess. The attacks of pain, jaundice, fever, chills, and sweats which characterize the disease are accompanied with clay-colored stools, and calculi are often found in them.

Other conditions occasionally to be differentiated are carcinoma, hypertrophic cirrhosis, and hydatid cyst. Abscess in the abdominal wall must often be excluded. In all cases, the diagnosis should be confirmed by means of the aspirator needle.

Prognosis.—Hepatic abscess is in itself inevitably fatal, except in those rare cases in which spontaneous evacuation is followed by recovery. Cases of single large abscess amenable to surgical treatment frequently recover, but, although the cavity can be evacuated, complete recovery is often long delayed or never attained. Multiple pyemic abscesses are almost necessarily fatal, but the hepatic conditions may contribute but little to the fatal issue.

Treatment.—Aside from surgical measures for the evacuation and obliteration of the abscess cavity, the treatment is wholly symptomatic. The nutrition and strength of the patient are to be supported, and suffering relieved. In many cases the treatment resolves itself into that of the septic condition. (See Treatment of Septicemia and Pyemia.)

FATTY LIVER.

The liver is subject to both fatty infiltration and fatty degeneration. (See pp. 22 and 23.)

Etiology.—The condition results from: (*a*) Obesity, a condition in which the liver serves as one of the storehouses for redundant fat; (*b*)

from the prolonged action of alcoholic or malt liquors, or such poisons as phosphorus, arsenic, or chloroform, conditions in which the degeneration is probably due to diminished supply of oxygen to the tissues; and (*c*) from acute infections, as, perhaps, in acute yellow atrophy, profound anemia, or the cachetic states associated with tuberculosis, cancer, chronic dysentery, or other chronic wasting disease.

Morbid Anatomy.—The organ may be normal or atrophic, but it is generally enlarged. The surface is smooth, the color a pale yellow, and the consistence is diminished.

Symptoms.—There are sometimes no manifestations by which the disease can be recognized during life. When the liver is found to be much enlarged in connection with obesity or during the course of the chronic cachectic diseases, a fatty degeneration is probable.

Diagnosis.—The fatty liver is to be distinguished from the *amyloid* chiefly by the absence of suppuration as a cause. As both affections occur in connection with tuberculosis, the differentiation is generally reduced to the comparative firmness or softness of the organ, which can be recognized best along the lower margin, and the presence of amyloid disease in other parts. From *hypertrophic cirrhosis* fatty liver can be distinguished by the absence of jaundice and other evidences of portal and biliary obstruction, taken in connection with the history of the case. From *carcinoma* it is distinguished by the uniform smoothness of the surface and the absence of nodules, profound emaciation, pain, and cachexia. From *leukemia* it is distinguished through examination of the blood.

Treatment.—The treatment is that of the condition upon which the fatty infiltration or degeneration depends. It is seldom that the condition of the liver need be made the object of special treatment.

AMYLOID LIVER.

WAXY OR LARDACEOUS LIVER.

Etiology.—For the causes of amyloid disease see page 23.

Morbid Anatomy.—The liver becomes enormously enlarged and exceedingly firm, the surface smooth, color lighter than normal, and the cut surface anemic. An atrophied liver in a state of amyloid degeneration has been described. The usual reactions of amyloid tissue to iodin and the anilin stains can be obtained.

Symptoms and Diagnosis.—Few or no disturbances are occasioned by even advanced amyloid disease of the liver. The lower margin may be felt, firm and sharp, as low in the abdomen as the umbilicus, sometimes even at the pelvic brim, and the surface is everywhere smooth. The spleen is usually enlarged on account of the same disease in it, and the diagnosis rests upon the history of suppuration, with, perhaps, syphilis or tuberculosis in the background, the evidences of the disease elsewhere, and the peculiar waxy pallor of the skin. The blood should be examined in order to exclude the enlargement due to leukemia.

Prognosis.—The disease is incurable, but the result, as it affects life, depends more upon the condition of other organs, especially that of the intestines and kidneys.

Treatment.—There is no treatment for amyloid disease, and, so far as the liver is concerned, there is little call for medication. It is doubtful if treatment of the underlying condition is capable of arresting to any extent the progress of the disease.

CANCER OF THE LIVER.

Etiology.—Cancer of the liver is primary in less than 5 per cent of cases. It is usually secondary to the same type of cancer in the stomach, pancreas, gall-bladder, rectum, or other organ. The most important of the recognized causes are: (*a*) *Age.*—More than half the cases occur between the 40th and 60th year, and few before 30, but cases have been reported in children. (*b*) *Sex.*—Men are somewhat more frequently affected than women, notwithstanding the fact that the disease is often secondary to cancer of the uterus and breast. (*c*) *Heredity.*—This is regarded by some authors as a probable cause in from 15 to 20 per cent of cases, but it is doubted by others. (*d*) *Trauma.*—In some cases there is a history of direct injury; in others the passage of gall-stones has been regarded as an important factor in the production of the disease in either the liver or gall-bladder.

Morbid Anatomy.—Histologically, the growth generally belongs to the alveolar or trabecular type, rarely to the cylindromata, and corresponds, as a rule, to that of the primary neoplasm.

1. Of the primary cancer three modes of growth have been recognized: (*a*) The massive, one or a few large masses of uniform structure, occupying a large part of the liver substance and causing decided enlargement of the organ. It is generally white or gray and firm, except at the center of the medullary variety. (*b*) The nodular, in which liver tissue may be almost completely replaced by the numerous cancer nodules of different sizes. The primary nodule can generally be recognized by its greater size and possibly by degeneration at the center. The other nodules are secondary to it, as a rule. (*c*) Cancer with cirrhosis, a rare form in which the entire liver is infiltrated with small, yellowish gray cancer nodules usually not more than 0.5 cm. in diameter and surrounded by fibrous tissue. The liver is little, if at all, enlarged, and its surface is roughened, much as in cirrhosis.

2. Secondary cancer nodules are generally multiple and so large as to be felt on palpation, and to cause great enlargement of the liver. In color and consistence they resemble the primary nodules, but more frequently show secondary degeneration, especially hyalin change, in the center, and they may be umbilicated.

Symptoms.—The primary disease may exist for a considerable time without the production of symptoms. Cachexia may, in fact, be the first indication of the disease, or this may be preceded by pain and jaundice, with occasional elevation of temperature.

The symptoms of the secondary form are generally preceded by those on the part of the organ primarily affected, especially when this is in the stomach, gall-bladder, rectum, or other part of the alimentary canal. The usual manifestations are: (*a*) Emaciation and anemia, which are prominent features of either form. The red corpuscles may be fewer than 2,500,000, and leucocytosis may be present or not. The tongue

usually becomes coated, dry, and flat. The heart's action is feeble. (*b*) Evidences of obstruction of the portal circulation develop. The superficial veins become distended, ascites and edema of the lower extremities are often observed. (*c*) Jaundice appears in about half the cases as a result of compression of the bile-passages. The skin sometimes becomes mottled with a brownish discoloration; sometimes the discoloration is uniform and suggestive of Addison's disease, but the color is generally a deeper yellow. (*d*) Cachexia develops sooner or later in all cases. The skin then has a peculiar and characteristically glossy pallor. (*e*) Fever usually appears late in the disease, if at all. It may be irregular, intermittent, and only moderate, but sometimes toward the close it runs up to 104° or 105° F. (40.0°—40.5°C.). In some instances it is of a septic origin and type, due to suppuration. Occasionally the temperature becomes subnormal near the end of life. (*f*) Examination of the abdomen shows enlargement, particularly in the upper portion, except in the sclerotic form of cancer, when enlargement may not occur. The lower margin of the organ can be distinctly felt several inches below the margin of the ribs, and the nodules can be readily palpated in many cases; on simple inspection, they are sometimes recognizable through the abdominal wall, moving with the diaphragm. The liver is usually sensitive. The duration of the disease from the time of its recognition is generally from three to eighteen months, rarely longer.

Diagnosis.—The cardinal symptoms are an enlarged, nodular, sensitive liver, with pain, progressive emaciation and anemia, jaundice, loss of strength, and cachexia. The chief difficulty lies in the exclusion of fatty and amyloid liver, hypertrophic cirrhosis, syphilis, and echinococcus.

Fatty and *amyloid* liver are eliminated chiefly by the pain, marked jaundice, cachexia, more rapid emaciation, and the nodular surface.

Hypertrophic cirrhosis is attended with no pain, little emaciation, uniform, less rapid enlargement of the liver, and the spleen is simultaneously enlarged.

Syphilitic gummata may simulate cancer nodules on palpation, but the age of the patient, the absence of cachexia, and the results of a judicious test with potassium iodid establish their identity.

Echinococcus is of less rapid growth, the nodules are softer, and there is no cachexia. The fluid withdrawn by aspiration establishes the diagnosis.

It is sometimes difficult to distinguish cancer of the liver from that of neighboring organs, and the differentiation of the sclerotic form of the disease may be impossible.

Treatment.—Measures are to be directed to the support of strength and relief of suffering. There is no curative treatment.

OTHER TUMORS OF THE LIVER.

Sarcoma rarely attacks the liver primarily, and it is by no means common as a secondary growth. Nearly all the varieties have been encountered secondarily, especially the myxo-, lympho-, and melanosarcomata. The last is a part of a universal dissemination following the primary growth in the choroid or skin.

Angioma is a not infrequent tumor of the liver, but it is of no clinical interest. Adenomata and cysts are occasionally met with. The latter are usually congenital and associated with cystic disease of the kidneys.

PARASITES OF THE LIVER.

Echinococcus, or hydatid cyst, the most frequent of these, is considered on page 285. Other parasites that have been more or less repeatedly found in the liver are the lumbricoid worms, the cysticercus cellulosæ, pentastomum denticulatum, the distoma, and the psorosperms.

DISEASES OF THE BILE-PASSAGES AND GALL-BLADDER.

JAUNDICE.

ICTERUS.

Definition.—A condition in which the bile pigment becomes disseminated throughout the body, staining the skin, mucous membranes, and many other tissues, and the secretions. Although a symptom, rather than a disease, the condition usually receives separate consideration. Two forms are recognized, (1) the obstructive and the (2) nonobstructive or toxemic.

Etiology.—1. *Obstructive jaundice* is produced by any influence within or outside of the liver, which causes nearly or quite complete closure of the bile-ducts. The most important forms of obstruction are: (*a*) Impaction of foreign bodies, especially of gall-stones, concretions, or parasites; (*b*) catarrhal or suppurative inflammation within the ducts or at the orifice of the common duct, or strictures resulting from such inflammation; (*c*) tumors within the ducts or causing compression of them. Such growths include neoplasms not only of the gall-bladder, duodenum, colon, pancreas, and other contiguous structures, but occasionally those of the kidneys, uterus, or ovaries, and aneurisms of the aorta, hepatic or mesenteric arteries. (*d*) Fecal impaction is a possible cause.

2. *Toxemic jaundice* is the form that is met with particularly in the acute infections, as in malaria, yellow fever, acute yellow atrophy, typhus, relapsing fever, Weil's disease, sometimes in typhoid fever, scarlatina, probably also in some cases of phosphorus, arsenic, ptomain, and other chemical poisoning.

Symptoms.—**Obstructive Jaundice.**—1. *The Skin and Conjunctiva.*—(*a*) The color of the skin varies with the intensity and duration of the condition, and the quantity of bile pigment deposited in it. All shades from a pale lemon yellow to a greenish brown or black are encountered in different cases. They are usually compared to lemon, orange, saffron, or olive. The color is seen to advantage by stretching the skin across the back of the hand. It frequently makes its first appearance in the conjunctivæ. (*b*) Intense pruritus accompanies the jaundice, the itching being most severe at night. This may precede the visible manifestations. It is sometimes confined to certain regions, as the palms and soles or between the fingers and toes. (*c*) Sweating is commonly observed, and

it also is sometimes confined to certain regions, as the palms, the axillæ, or the abdomen. (*d*) Urticaria, herpes, xanthelasma, furunculosis, and carbuncles sometimes develop, and hemorrhages into either the skin or mucous membranes are occasionally observed.

2. *The Secretions.*—(*a*) The sweat, saliva, sometimes the bronchial mucus, and rarely the tears are stained to such an extent that they discolor white linen. (*b*) The urine always shows the presence of bile pigment on chemical test, if not in its color, even before the discoloration of the tissues can be discerned. When the jaundice is intense or of long standing, albumin is generally present, and the microscope reveals bile-stained casts.

3. The *feces* are of a pale slate or gray color, owing to the absence of bile pigment. Constipation and flatulency usually exist, and the stools are pasty and offensive; diarrhea may occasionally supervene. Gastric digestion may remain normal.

4. *Pulse and Respiration.*—The pulse is generally slow; it sometimes sinks to 40 or even 20 in the minute. The respiration is generally normal; it may be reduced in ratio to the pulse.

5. The *blood* is often normal in simple catarrhal jaundice, except with reference to its corpuscles. A peculiar enlargement of the red cells and other, unimportant, changes have been observed. The plasma is stained by the bile pigment.

6. *Hemorrhages* sometimes occur into the skin or from the mucous membranes in the more malignant cases.

7. *Cerebral Symptoms.*—Headache and vertigo are common, and the patient often becomes irritable, or he may become morose and melancholy, with mental dullness, somnolence, or restlessness and insomnia. A fatal stupor sometimes supervenes, the patient dying in coma or convulsions. Such symptoms are due to intoxication of unknown character, sometimes spoken of as cholemia. The vision is sometimes disturbed. There may be an inability to see distinctly in full daylight (nyctalopia), or the opposite condition (hemeralopia); very rarely objects appear yellow (xanthopsia). The duration of icterus is exceedingly variable. Acute and chronic cases occur. The former usually recover within two or three weeks; the latter may last for several years, with or without intermissions. Death results apparently from either exhaustion or toxemia.

Diagnosis.—Jaundice is generally self-evident, except in the dark-skinned races. From the discoloration of Addison's disease it can be distinguished by the presence of bile pigments in the urine.

The diagnosis of the cause is important. (*a*) When the gall-bladder is distended and the feces are colorless, the obstruction is usually situated at the duodenal end of the common duct. (*b*) Colorless feces without distention of the gall-bladder indicate obstruction of the hepatic duct. When neither of these conditions is present, the obstruction usually affects only a part of the bile-ducts within the liver. (*c*) Attacks of severe pain in the region of the gall-bladder generally point to gall-stones as the cause of the obstruction. (*d*) When fever is present, it is, as a rule, due to catarrhal inflammation involving the smaller ducts within the liver. (*e*) A moderate, chronic jaundice is usually due to cirrhosis, malignant disease, or chronic passive hyperemia. The first

of these conditions is rendered probable by the coexistence of ascites, the second by enlargement and a nodular surface of the liver, and the third by a valvular lesion of the heart.

Prognosis.—This depends entirely upon the cause of the condition. Catarrhal jaundice usually subsides within a few weeks; persistent, deep, and increasing icterus is more generally associated with a fatal disease.

Treatment.—The treatment is that of acute or chronic angiocholitis and the other affections causing the condition.

Toxemic Jaundice.—The symptoms of this condition are not usually so pronounced as those of the obstructive form. In the acute infections, as malaria, the skin acquires only a pale tinge; the feces are bile-stained, and the urine may contain little or no bile pigment, although it is deeply colored with other pigments. In the more malignant forms, as in acute yellow atrophy, the color becomes intense. The constitutional disturbances are profound, being often manifested by high temperature, delirium, convulsions, black vomit, suppression of urine and cutaneous hemorrhages as seen particularly in yellow fever.

The ***treatment*** is that of the disease of which the jaundice is a symptom.

ICTERUS NEONATORUM.

Definition.—"Jaundice of the new-born infant." Two forms of the condition are recognized, a mild form, sometimes referred to as physiological icterus, which is never fatal; and a severe, generally fatal, form.

Etiology.—Various theories have been offered in explanation of the condition. The mild form is generally attributed to stasis of the bile within the tubules as a result of its abnormal concentration, feeble respiration and circulation, or from compression of the bile capillaries by distended portal vessels. It has been referred to the rapid destruction of blood-corpuscles during the first days after birth and to other conditions not yet demonstrated.

The causes generally discovered in the severe form are a congenital occlusion of the common bile-duct, congenital syphilitic cirrhosis, or pylephlebitis due to septic infection through the umbilical vein.

Symptoms.—Mild cases generally begin within the first three days, present the usual symptoms of jaundice—discoloration of the skin, scleræ and urine and chalky feces—and subside within a week or two. Severe cases present, in addition to these manifestations, an elevation of temperature and sometimes the symptoms of severe sepsis—fever, vomiting, and profound prostration. Persistent hemorrhage from the cord sometimes occurs. These cases are rapidly fatal.

Treatment of the mild cases is unnecessary; treatment of the severe cases is futile.

ANGIOCHOLITIS.

CHOLANGITIS.

Definition.—Inflammation of the bile-ducts. The disease may be simple catarrhal, infectious, suppurative, or ulcerative. Diphtheritic or croupous and hemorrhagic inflammations have been described, but they cannot be differentiated clinically.

CATARRHAL ANGIOCHOLITIS.

This form of the disease may be either acute or chronic.

1. **Acute Catarrhal Angiocholitis.**—An acute inflammation of the common bile-duct, causing obstruction and consequent icterus.

Etiology.—(*a*) The disease is most frequent in young persons, but it may occur at any age. It is generally, if not always, an extension of inflammation from the duodenum. This in turn results from a catarrhal condition of the stomach, arising from the ingestion of irritating substances taken as food or drink, or from irritant medicines or poisons. By some writers it is regarded as invariably of infectious nature. (*b*) It is sometimes attributed to exposure to cold, and it may often be traced to the active or passive congestion of cirrhosis, or disease of the heart or kidneys. (*c*) It sometimes complicates the acute infections, particularly pneumonia, typhoid fever, or malaria. (*d*) It may occur as an epidemic, or (*e*) follow emotional disturbances.

Morbid Anatomy.—The inflammation is generally limited to the duodenal end of the common duct, particularly to the portion within the intestine (portio intestinalis); it is thought in some cases to extend back to the intrahepatic or to the cystic ducts and gall-bladder, but, as the disease is not a fatal one, post-mortem observations have been too few to establish the truth of the supposition. The duct is usually obstructed with a plug of tenacious, bile-stained mucus, sometimes limited to the intestinal portion, without definite evidences of the extent of the inflammation that existed before death. The liver is usually enlarged and the gall-bladder distended. Gall-stones are sometimes present, but this condition is considered separately in the succeeding section.

Symptoms.—Jaundice is usually the first symptom indicative of the condition. It may appear as the first evidence of illness, or it may be preceded for a week or longer by more or less severe gastrointestinal disorder, gastrointestinal catarrh, or catarrhal enteritis, with discomfort, pain, and tenderness in the region of the liver, the pain often radiating to the back and shoulder or to the limbs. Constitutional symptoms are generally present and may assume considerable severity. Nausea and vomiting, headache, malaise, and moderate fever are often complained of. The usual signs of obstructive jaundice also appear, as the discoloration of the skin, tissues, and urine, with clay-colored stools, perhaps with restlessness and pruritus, but the skin is not so intensely bronzed as in chronic obstruction. The pulse and respiration may be reduced in rate. Slight enlargement of the liver and spleen can generally be made out, but the gall-bladder cannot usually be felt. The duration of the disease is from two weeks to three months, in most cases about a month.

Diagnosis.—The recognition of this disease is not difficult in a young person. When, however, in middle life, the jaundice persists beyond the usual four or six weeks, a fear of a more serious affection, as cancer or gall-stones, is aroused. Such affections are excluded, for the most part, through the absence of pain and other diagnostic symptoms.

Prognosis.—Simple angiocholitis is never fatal. The first indication of recovery is a return of color to the feces.

Treatment.—In mild cases little treatment is required further than the

regulation of the bowels and restriction of the diet until the catarrhal condition has had time to subside. Small laxative doses of calomel may be given every second or third day in the beginning, and followed by a saline laxative, as sodium phosphate, on the succeeding morning. Purgation is to be avoided. Large quantities of water should be drunk, preferably alkaline mineral waters or water containing a little sodium bicarbonate. Or the salicylate may be given in small doses and the water taken pure. Daily irrigation of the large bowel with cold water has been recommended. The diet should be limited in quantity, and such articles as will throw the least work upon the liver—meat, fats, and sweets, being excluded. It is necessary to confine the patient to bed only when fever is present or when the pulse-rate is greatly reduced.

2. **Chronic Catarrhal Angiocholitis** (Hepatic Fever, Chronic Cholangitis).—***Etiology.***—The common, if not the exclusive, cause of this form of the disease is obstruction of the common bile-duct, whether by gall-stones, stricture, neoplasm, or pressure, causes which have been referred to elsewhere under the heads of "jaundice" and "gall-stones." It sometimes, perhaps, follows repeated attacks of acute angiocholitis.

Morbid Anatomy.—The walls of the common duct are thickened. Its mucous membrane may be little altered or it may show the usual changes of chronic inflammation. The duct is distended to a degree corresponding to the extent of the obstruction. When the obstruction is complete, all the bile-passages and the gall-bladder are greatly distended and filled with a clear, usually sterile mucus. When the obstruction is but partial, the mucus is turbid and bile-stained. In the majority of cases, gall-stones are found in the gall-bladder or common duct or in both; but in some cases, although gall-stones have been passed during life, they are not found within the passages.

Symptoms.—The usual symptoms of obstruction are present, particularly icterus, staining of the urine and other secretions and colorless feces, but a more distinctive feature is the periodical occurrence of the intermittent type of fever, which has received the name of hepatic intermittent fever. This is characterized by an attack of chill, fever, and sweating, with prostration, sometimes pain, lasting for a few days and recurring at intervals of a few weeks or months. The cause is not definitely known, but it is thought to be infection or aggravation of the previous inflammation by the passage of calculi. The attack is not necessarily indicative of suppuration.

3. **Suppurative and Ulcerative Angiocholitis.**—A diffuse, suppurative inflammation of the larger and smaller bile-ducts usually associated with similar disease of the gall-bladder.

Etiology.—The direct cause is infection. The mucous membrane is rendered liable to infection through direct injury, particularly through the extension of inflammation from an adjacent focus, as from a pylephlebitis, an abscess, or malignant disease in the liver. Extension from the gall-bladder is not common, but it may occur. Many cases have followed typhoid fever or pneumonia, and the pneumococcus, typhoid, and colon bacilli, streptococci, and staphylococci have been found in the pus.

Morbid Anatomy.—All the ducts are thickened and distended with pus and bile, sometimes mingled with mucus and blood. Here and there the walls show saccular dilatations, which are virtually abscess cavities,

and the mucous membrane is often ulcerated or necrotic, especially in cases arising from gall-stones. The gall-bladder is usually affected, its walls thickened and distended with pus, ulcerated and adherent to surrounding viscera, sometimes perforated.

Symptoms.—The symptoms are those of profound septic inflammation with marked enlargement and sensitiveness of the liver and gall-bladder. Pain may or may not be a prominent feature, and jaundice, although constant, may be slight or profound. Leucocytosis is usually present. The patient becomes weak and emaciated.

ACUTE INFECTIOUS CHOLECYSTITIS.

Definition.—An acute inflammation of the gall-bladder due to the entrance of bacteria.

Etiology.—The specific cause is bacterial invasion. Infection is favored by the presence of gall-stones or of a foreign body, but it may occur independently of all such local conditions. In some instances the disease has been regarded as an extension of inflammation from the common duct or elsewhere in the vicinity, and it often follows an acute infection, particularly pneumonia or typhoid fever.

Morbid Anatomy.—There are three types of the disease, the catarrhal, the suppurative, and the phlegmonous, and the condition found after death corresponds to one of these types, usually to the suppurative or phegmonous. The gall-bladder is distended and its walls are tense. There may be adhesions to other organs, localized abscesses or perforation with discharge of the contents, and with the production, in the more violent cases, of a general peritonitis. The contents of the gall-bladder are mucopurulent, purulent, or hemorrhagic, often foul in odor and containing various bacteria. The pneumococcus, typhoid bacillus, streptococci, and staphylococci are commonly found. The cystic duct is usually closed, whether or not calculi be present.

Symptoms.—The symptoms are not distinctive. There is usually pain in the region of the liver, sometimes it is in the epigastrium or elsewhere in the abdomen, even at a point suggesting appendicitis. Fever develops, the pulse becomes rapid, nausea and vomiting follow, and the abdomen becomes distended and tender, the tenderness finally becoming localized, in most cases, in the region of the distended gall-bladder. Jaundice develops when gall-stones are present, but not, as a rule, when they are absent. The gall-bladder cannot always be felt. More or less complete obstruction of the bowel sometimes occurs, even gas failing to pass in extreme cases, probably as a result of adhesions between the gall-bladder and bowel.

The ***diagnosis*** is often extremely difficult on account of the uncertainty of the symptoms, particularly the uncertain location of the pain and tenderness which may lead to the erroneous diagnosis of appendicitis or intestinal obstruction. The diagnosis of the type of the inflammation is impossible. The presence of jaundice is of value, but it is unfortunately absent in many cases. When vague symptoms of the character just described follow an acute infection, particularly typhoid fever or pneumonia, the possibility of an acute cholecystitis should be borne in mind.

Prognosis.—The disease is exceedingly fatal, unless it be early relieved through surgical measures.

Treatment.—The treatment is entirely surgical.

CHOLELITHIASIS.

GALL-STONES, BILIARY CALCULI, OR CONCRETIONS.

Definition.—A condition in which concretions, "gall-stones," are formed within the bile-ducts or gall-bladder.

Etiology.—More than half the cases occur in persons above 40 years of age, rarely in those under 25, but congenital cases have been recorded. Fully 75 per cent of cases occur in women, 90 per cent of whom have borne children. Chief among the predisposing causes are conditions believed to favor the stagnation of bile, overindulgence in food, sedentary or indolent habits, occupations which require a leaning attitude, constipation, corset-wearing, enteroptosis, and nephroptosis. The immediate formation of gall-stones consists in the deposit of cholesterin, lime, or other salts from the bile upon a nucleus. The nucleus is generally composed of epithelial débris and micro-organisms, occasionally of bile-salts; rarely it is a foreign body. It is probable that the micro-organisms are the important element in most cases, for gall-stones have been experimentally produced by inoculation of the gall-bladder of animals with attenuated cultures of the colon and typhoid bacilli. These organisms are repeatedly found in the gall-bladder and they are capable of living there for a great length of time. The so-called lithogenous catarrh, which has long been regarded as the essential factor in the production of concretions, is induced by the bacteria. It is believed to so modify the mucous membrane of the gall-bladder and ducts that they secrete, as shown by Naunyn, both cholesterin and calcium, the most important ingredients of the calculi.

Physical Properties of Gall-Stones.—A single gall-stone sometimes attains an enormous size, as much as five inches in length and distending the gall-bladder. As a rule, however, there are several smaller concretions more or less completely filling the viscus. More than a thousand calculi may be found in it, and the bile-ducts within the liver sometimes contain enormous numbers of them. When a moderate number of stones of medium size are contained in the gall-bladder, they usually have four or more facets and often resemble beech-nuts, or they may be irregular or rough like mulberries. They may be so minute as to be rightly designated sand. In composition the larger stones often consist chiefly of cholesterin, some containing 98 per cent, in an amorphous or crystalline form. When cut, the crystalline stones show both radiating and concentric striations. Others, particularly the smaller ones, contain the salts of lime, magnesia, bile acids, fatty acids, and sometimes traces of iron and copper. Those containing much cholesterin are lustrous and on section are of almost pure white color; others present various colors, often beautiful striations, and have usually a hard external crust of a yellow, gray, brownish, or almost black color. The nucleus is generally visible. Calculi generally lodge at the duodenal end of the common duct, frequently in the intestinal portion, sometimes in the cystic duct or at the orifice of the gall-bladder.

Symptoms.—One or many calculi may remain in the gall-bladder indefinitely without giving rise to symptoms. It is estimated that fully 25 per cent of all women over 60 are the subjects of gall-stone, yet only a small minority of them suffer. The more serious results are, therefore, to be regarded as in the nature of accidents. These are generally considered under three heads: (1) Biliary colic, a result of the passage of a stone through the ducts; (2) chronic obstruction, a result of permanent plugging of the cystic or hepatic duct; and (3) remote effects, notably ulceration, perforation, or the formation of fistulæ.

1. **Biliary Colic.**—The passage of a gall-stone through the cystic or hepatic duct is not invariably painful, but in a majority of cases that come under observation it is extremely so. The attack often follows a sudden jar, a fall or jump, but it may occur spontaneously at night. The patient is suddenly seized with an excruciating, lancinating pain in the right hypogastrium, epigastrium, or lower thoracic region, which often radiates to the right shoulder. It is so intense that he rolls from side to side or assumes cramped-up positions in efforts to obtain relief. The pain is due to the passage of the stone through the cystic duct, the inflammation occasioned by it, or to the distention of the gall-bladder by the pent-up fluid. It is at first paroxysmal, but may become continuous. The attack, after lasting from a few hours to a week or longer, ceases suddenly. Intervals of complete cessation of pain are not unusual. Chills, with fever and vomiting, usually occur soon after the onset. The vomit may contain bile if the obstruction be not complete. The patient sometimes becomes greatly prostrated or sinks into a collapse, with pinched features, cold extremities, cyanosis, cold sweating, and restlessness, even under morphin. Hiccough may develop and convulsions may occur. Jaundice generally appears if the attack lasts more than a day or two. The abdomen becomes distended and sensitive, particularly in the region of the liver and gall-bladder. The stools are pale, and the urine often contains, in addition to bile, albumin or blood. Any of these symptoms may be intermittent. The course of the disease is indefinite. It may terminate in a few hours or days and permanently with the first attack, or it may recur at various intervals for years. The results are equally uncertain. Impaction may occur, with the production of chronic jaundice, if the impaction is in the common duct. There is then great danger of angiocholitis and its results, perforation, with fatal syncope or subsequent fatal peritonitis. Disturbance of a previously diseased heart is sometimes the immediate cause of death.

Diagnosis.—The sudden onset of intense pain, followed by vomiting and prostration, with tenderness in the region of the gall-bladder, are generally sufficient for a diagnosis, particularly when jaundice develops. The affections to be excluded are gastralgia, gastric or duodenal ulcer, nephritic colic, and appendicitis.

Gastralgia is generally accompanied with such evidences of a disordered stomach as flatulency or superacidity, and temporary relief is afforded by pressure or eructation.

Gastric and *duodenal ulcers* are attended with hematemesis or melena, the former immediately, the latter two or three hours, after a meal.

The pain of *nephritic colic* radiates to the groin or thigh, and the urine generally contains blood. A calculus may be passed.

Appendicitis.—It is only when the appendix occupies a high position that it may cause confusion. A differentiation may then be impossible without incision, unless jaundice appear or stones can be found in the stools.

2. **Chronic Obstruction.**—The obstruction may occur in the cystic or common duct, and the symptoms differ accordingly.

Chronic obstruction of the cystic duct leads to: (*a*) Dilatation of the gall-bladder. For a time the contents consist of mucus or mucopurulent fluid and bile, but later they become clear, of low specific gravity and alkaline or neutral reaction. Albumin also is usually found. The condition has been called dropsy of the gall-bladder (hydrops vesicæ felleæ). More than a quart of fluid may be present. The outline of the viscus and its connection with the liver can generally be recognized by palpation and percussion, sometimes on inspection. Jaundice is often absent. A peculiar gall-stone crepitus can sometimes be felt through the thin, relaxed abdominal wall, if the bladder be not too tense.

(*b*) Acute cholecystitis sometimes develops, and it may become suppurative, although it is generally simple in the beginning. Perforation may follow. When the gall-bladder is distended with pus the condition is termed an empyema of the gall-bladder.

(*c*) Calcification of the gall-bladder is an occasional, late result of distention. The walls become infiltrated with lime-salts and the mucous membrane incrusted. The term ossification has been improperly applied to the condition.

(*d*) Atrophy sometimes follows, the fluid disappearing, and the walls shrinking into a small nodule, often containing a small calculus.

Obstruction of the common duct may be complete or partial. It may be occasioned by the lodgment of one or many stones in any part of the duct. A peculiar form is that in which one stone, lodged in the intestinal portion of the common duct, or higher up, acts as a ball-valve. With complete obstruction, the hepatic ducts become greatly distended and the intense jaundice persists. The fluid within them may become clear and remain sterile. The symptoms are often intermittent, with recurrent attacks of jaundice, and the stools becoming alternately light and dark. Symptoms may be absent; the liver and gall-bladder may remain nearly or quite normal in size. An infectious cholangitis is often set up, however, which is recognizable by the usual symptoms, particularly by the hepatic enlargement and tenderness, with fever. Osler attributes the repeated attacks of so-called hepatic intermittent fever, sometimes recurring for years without evidence of suppuration, to the action of the ball-valve calculus. Suppurative cholangitis may, however, supervene.

3. **Remote Effects of Gall-Stones.**—The most important of these are the development of biliary fistulæ and intestinal obstruction. (*a*) Fistulous communication has been established between the gall-bladder and the hepatic duct, the peritoneal cavity, or a cavity formed in the liver. In several instances the portal vein was opened. Communications with parts of the gastrointestinal tract are common, particularly with the duodenum or colon, seldom with the stomach. Gall-stones have been found in the urinary bladder as a result of fistulous communication, and they have been coughed up through openings into the lung. They have escaped, also, through cutaneous fistulæ.

(*b*) Intestinal obstruction by gall-stones is not an extremely infrequent accident. A large number of stones are often passed without its occurrence, and in a majority of cases the stones causing obstruction have been lodged in a diverticulum or in the appendix.

Treatment of Gall-Stones.—An attack of hepatic colic calls for the prompt administration of morphin hypodermically in doses of ¼ grain (0.016), repeated with caution. A few inhalations of chloroform may be administered until the morphin has taken effect. Hot fomentations should be applied freely. The remedies most in favor for their supposed action in preventing gall-stone formation or in dissolving them when formed are sodium salicylate and olive oil, and Durande's mixture of ether and turpentine. The sodium salicylate should be given in the quantity of gr. xxx or more daily, and olive oil in tablespoonful doses, or more, three times a day. Both may be coincidently employed with excellent results. Fatty concretions are often formed from the oil and may be mistaken for gall-stones. The itching is to some extent relieved by dusting the skin with starch-powder containing camphor, or by inunction with carbolated vaselin. The patient should take outdoor exercise and restrict his diet largely to fresh vegetables and fruits, excluding meats and fats. The advisability of surgical measures should not be overlooked in the case of a person whose age and strength justify an operation.

CANCER OF THE GALL-BLADDER AND BILE-DUCTS.

Etiology.—The disease may be primary or secondary. Both are peculiar to the cancer age. The former affects the sexes about equally; the latter is about three times more frequent in women. The presence of gall-stones is thought to greatly favor the development of the disease.

Morbid Anatomy.—The growth begins in the fundus of the gall-bladder or in the common bile-duct, usually near its junction with the duodenum.

Symptoms.—Pain and tenderness in the region of the gall-bladder, followed by jaundice and the formation of a tumor, with the development of a cachexia, are the usual symptoms. Distention of the gall-bladder, and jaundice, are more certain when the duct is affected.

Diagnosis.—The chief elements in the diagnosis are the presence of a solid tumor, jaundice, and cachexia. The diagnosis is not difficult, as a rule, when a primary growth can be discovered in some other region.

Treatment.—Unless the case can be treated surgically, only palliative measures can be employed.

DISEASES OF THE PANCREAS.

HEMORRHAGE OF THE PANCREAS.

Etiology.—Pancreatic hemorrhage has usually occurred in individuals above 40 and more frequently in men. Very little is positively known of the cause. The accidents and conditions generally inducing it are: (*a*) Blows and penetrating wounds; (*b*) acute pancreatitis; (*c*) chronic passive congestion from cardiac valvular disease or portal obstruction;

(*d*) abnormal blood-states, as in purpura, scurvy, pernicious anemia, or the acute infections; (*e*) diseases of the blood-vessels, as arteriosclerosis, embolism, or aneurism; and (*f*) fat-necrosis, cystic degeneration, or cancer of the gland.

Morbid Anatomy.—The hemorrhage may be circumscribed and the degenerative changes causing or resulting from it may be confined to one or more areas, but in many cases the entire gland is more or less completely destroyed or degenerated, and the degeneration may even affect adjacent tissues.

Symptoms.—The individual is usually seized suddenly with the most intense pain in the region of the pancreas. This is followed by marked prostration or complete collapse. The temperature becomes subnormal, the pulse feeble; the body is bathed in a cold sweat, and great restlessness develops. Nausea and vomiting usually accompany the seizure. Tenderness is generally found in the epigastrium or just below it. Death often occurs in syncope or coma, as a result of pressure and reflex paralysis of the heart, rather than from the loss of blood. If death be delayed for a few days, fever develops and the abdomen becomes tympanitic; the bowels are usually constipated.

Diagnosis.—The recognition of the disease is based upon the suddenness of the onset and the location of the pain and tenderness. From the collapse following the perforation of a gastric or intestinal ulcer it is generally differentiated by the previous healthy condition of the patient. The diagnosis is not always possible, and the condition has repeatedly been discovered post mortem.

Prognosis.—The prognosis is extremely unfavorable, but recovery is believed to have occurred.

Treatment.—Morphin and hot fomentations or an ice-bag are required for the relief of the pain. The advisability of surgical treatment should be immediately determined.

ACUTE PANCREATITIS.

1. **Acute Hemorrhagic Pancreatitis.**—This form of the disease is not clearly distinguished from hemorrhage of the pancreas, and it is often impossible in a given case to determine whether the inflammation or the hemorrhage was the initial lesion.

Etiology.—The disease is generally encountered in adult males, often in those addicted to alcoholism. The immediate cause is probably an extension of inflammation from the duodenum, occasionally, perhaps, the passage of bile into the pancreatic duct, since the disease has been experimentally developed in animals in this manner.

Morbid Anatomy.—The gland is generally somewhat enlarged. The interlobular tissues are infiltrated with blood, and fat-necrosis is commonly present. An accumulation of inflammatory products is found around the acini. Fat-necrosis is usually found also in the omentum and mesentery, sometimes in other regions.

The ***symptoms*** are the same as those of pancreatic hemorrhage. These two conditions therefore cannot be differentiated clinically.

2. **Acute Suppurative Pancreatitis** (Pancreatic Abscess).—***Etiology.***—Two-thirds of the cases recorded have been in men. The cause is not

known. That it is due to infection is probable, and that it follows upon an inflammatory condition is at least possible.

Symptoms.—There are in most cases attacks of severe pain and vomiting, followed by fever and perhaps delirium. A tumor may develop in the region. When the condition has lasted for some time, fatty diarrhea, glycosuria, and icterus may appear, possibly with indications of sepsis.

The ***treatment*** is surgical. One case diagnosticated by Thayer and operated upon by Finney recovered, but the diagnosis has rarely been made.

3. **Gangrenous Pancreatitis.**—The entire pancreas or only a portion of it may undergo necrosis. The cause is not definitely known, except that the condition sometimes follows injury, hemorrhage, hemorrhagic pancreatitis, suppuration, or gastric ulcer. The entire pancreas has been found separated from its attachments and lying in an abscess cavity or in the omental cavity, and in two instances the gland has been discharged per rectum. Recovery followed its discharge in these cases, but in most instances the disease has proved fatal in from two to three weeks.

CHRONIC PANCREATITIS.

Etiology.—The disease may follow one or more attacks of the acute form of pancreatitis, but it is generally a result of obstruction of the duct by calculi, infection by micro-organisms, or the extension of inflammation along the pancreatic duct from the duodenum. Inflammation of contiguous structures, especially that due to gall-stones, alcoholism, or syphilis, has been regarded as the cause in some cases.

Morbid Anatomy.—The pathological change is a sclerosis, a proliferation of the interstitial connective tissue, with corresponding destruction of the glandular structure. Small cysts are sometimes formed through compression of the pancreatic duct by the proliferated tissue. Interstitial hemorrhage or the pigmentation resulting from it is sometimes found. Suppurative pancreatitis may also become chronic and result in the formation of a single large abscess or several small ones. These may subsequently undergo caseation or calcification.

Symptoms.—The symptoms are chiefly those of gastric and intestinal indigestion, with occasional attacks of epigastric pain, fever, and unaccountable prostration. Jaundice may occur, and ascites has been observed. Lipuria and fatty stools, with undigested muscle fiber, may also be present, and, if the destruction of the gland be extensive, glycosuria may develop. The emaciation may be extreme, and the indurated gland may be felt through the abdominal wall. The urine usually reacts to the phenylhydrazin test.

FAT-NECROSIS.

This peculiar change is liable to affect the pancreas and the adipose tissue of various regions in connection with any of the diseases of the gland, particularly with the hemorrhagic or gangrenous forms of pancreatitis. The necrotic tissue is circumscribed and appears as small yellowish or white areas. In the pancreas these are seen between the lobules. In the adipose tissues they are commonly found in the omen-

tum and abdominal fat, but they may be found in the fat of almost any part of the body. The cause of the necrosis is not known. It has been attributed to almost every possible accident or pathological change, as obstruction of the duct, deficient circulation, the action of steapsin, the fat-splitting ferment, and very naturally to the action of various bacteria. In some instances the pancreas has shown no recognizable disease. The recent investigations in regard to the islands of Langerhans, referred to under the Etiology of Diabetes, may lead to a better explanation of this disease.

PANCREATIC CYST.

Etiology.—This rare disease has been observed in both males and females, chiefly in early adult or middle life, but congenital cases have been recorded. The principal causes are: (*a*) Injury, as by blows or continued pressure; (*b*) extension of inflammation from the duodenum; (*c*) obstruction of the duct by calculi or occlusion by pressure (the retention cysts of Körte); (*d*) the presence of tumors, including the growth of cystic adenomata; or (*e*) sclerosis of the gland.

Morbid Anatomy.—The location of the cyst is variable. In most instances it pushes the stomach upward and the colon downward, and it may reach the abdominal wall between them. It may, however, be found above the stomach or below the colon. Again, it may project into the region of the left kidney, when it develops from the tail of the pancreas. It varies greatly in size, sometimes containing only a few ounces, sometimes several quarts, of fluid. Communication with the duct is occasionally established in some cases, and the size of the tumor varies with the escape of fluid and closure of the opening. The contents of the cyst may be mingled with blood, or clotted blood may be found in it, especially in cysts of traumatic origin.

Symptoms.—The discovery of a tumor in the upper portion of the abdomen may be the first indication of a cyst. As a rule, the patient experiences occasional attacks of sudden, severe pain, radiating from the epigastrium downward or to either side, often to the left shoulder. These may occur spontaneously or after an error of diet, and they are often accompanied with vomiting, constipation, or diarrhea, perhaps with bloody dejections and jaundice. Fatty stools are rare, but the passage of undigested meat has been repeatedly observed. After the tumor has attained a size that can be recognized, the symptoms generally become more severe and the nutrition of the patient rapidly fails. Dyspnea may be produced by the pressure of its growth. Glycosuria has been observed, and, rarely, salivation.

Diagnosis.—This is usually based upon the symptoms described, but particularly upon the presence of a semicircular tumor in the median line or on either side of it in the upper abdomen. Sometimes the tumor becomes extremely large, invading almost the entire cavity of the abdomen. It does not move with respiration, as a rule. Fluid aspirated from it is generally dark brown, of alkaline reaction, and has a specific gravity between 1.010 and 1.020 and it contains fat-granules, the débris of cells, and sometimes cholesterin; but the most valuable diagnostic feature is the fact that the fluid will digest fibrin and albumin. All

three pancreatic ferments are sometimes present, but the others are not distinctive.

Prognosis.—The prognosis is favorable under proper surgical treatment. This consists in opening and draining the cyst under proper precautions.

TUMORS OF THE PANCREAS.

Cancer, although the most frequent form of pancreatic neoplasm, is rare. Sarcoma, adenoma, and lymphoma are extremely rare. Syphilitic gummata are sometimes observed. The cancer is generally of the alveolar type. The symptoms are not distinctive, hence the diagnosis is often difficult. There are generally paroxysms of pain, nausea and vomiting, sometimes jaundice, with rapid emaciation and the development of cachexia. The tumor may often be felt, but its location is determined with difficulty. Dilatation of the stomach sometimes results from compression of the pylorus. Fatty stools, glycosuria, and salivation may be present, and ascites sometimes develops. The treatment is purely symptomatic, unless surgical relief is attempted. Extirpation has been successfully performed in a few instances.

PANCREATIC CALCULI.

Etiology.—Calculi rarely form within the pancreatic duct. They have usually been attributed to (*a*) chronic inflammation of the gland or duct, (*b*) altered secretion, or (*c*) the action of bacteria.

Morbid Anatomy.—The calculi are usually numerous, small, round or irregular, smooth or rough, white masses consisting chiefly of calcium carbonate and phosphate and varying in size from 1 mm. to 2.5 cm. in diameter. As a result of the obstruction occasioned by them, the duct becomes dilated and a greater or less portion of the gland may become inflamed, with the production of chronic induration and possible atrophy. Cysts are often formed, and carcinoma has been attributed to the irritation occasioned by them.

Symptoms.—These are little more definite than evidences of possible pancreatic disease. Occasional attacks of pain in the region may occur; fatty stools, glycosuria, and emaciation are common symptoms. A positive diagnosis is rarely possible. The ***treatment*** is surgical or purely symptomatic, if the case does not admit of surgical treatment.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum. It may be either primary or secondary in character, general or local in extent.

Etiology.—1. **Primary Peritonitis.**—This form of the disease is exceedingly rare, if we retain the old idea that it originates spontaneously, idiopathically, or from no more definite cause than exposure and cold. It is sometimes thought to be of rheumatic origin. Such cases are doubtless instances of cryptogenic infection. That cases occur primarily,

in the sense that they are preceded by no other specific disease, ther is little doubt.

2. **Secondary Peritonitis.**—This form may arise: (*a*) From the exter sion of inflammation from the stomach, intestines, gall-bladder, genito urinary or other organs, especially after perforation has occurred; (*b* from the passage of bacteria through the intestinal wall, without the es tablishment of an opening; or (*c*) from injury.

Peritonitis due to perforation is the most common form, followin the rupture of an ulcer of the stomach, bowel, or gall-bladder, or tha of an abscess anywhere within the abdominal cavity. It has followe the rupture of an apparently normal Graäfian follicle (Osler). Peri tonitis due to extension of inflammation is generally associated with ulceration, suppurative inflammation, or cancer of the stomach, intestine, liver, spleen, or other organs and tissues having a peritoneal investure, particularly disease of the appendix, Fallopian tubes, or ovaries. The passage of bacteria through the bowel wall is by no means infrequent. No single species is required for the production of this disease, but many are doubtless capable of exciting it, and in many instances two or more kinds are present. Among the most frequent are streptococci, staphylococci, and the colon bacillus, especially in connection with fecal impaction and strangulation. The ameba coli has been found in cases arising from dysentery, and the gonococcus in cases from tubal disease. The pneumococcus, Bacillus proteus, pyocyaneus, aërogenes capsulatus, and many others have been observed, and less frequently the typhoid and anthrax bacilli. Acute peritonitis often follows the acute infections, particularly typhoid fever, scarlatina, diphtheria, cerebrospinal meningitis, and pneumonia, and it occasionally complicates rheumatism, pleurisy, chronic nephritis, septicemia, and tuberculosis. Tubercular peritonitis has been considered under the general subject of Tuberculosis. Septic pleurisy and pericarditis often extend to the peritoneum, but it is due to the passage of bacteria, and not to an extension of the inflammation. Septic peritonitis sometimes occurs in infants as a result of infection of the cord.

Injury as a cause of peritonitis includes blows, penetrating wounds, and infection during surgical operations, for all such cases are due to infection following the injury or exposure of the peritoneum.

Morbid Anatomy.—The inflammation may be of any of the varieties usually met with in the serous membranes, especially fibrinous, serofibrinous, hemorrhagic, purulent, and gangrenous. (See Inflammation, p. 29.) When the inflammation involves the entire peritoneum, it constitutes a general peritonitis; when circumscribed, possibly shut off from the general peritoneum by adhesions of its own surfaces, it is a local peritonitis. The character of the inflammation depends upon the infectious agents that are present, largely upon the ability of the microorganisms to produce suppuration. Pus-formers may, however, be found in a simple fibrinous inflammation of recent origin.

Symptoms. —*Local.*—Pain is usually the first and most prominent manifestation of the disease. It is constant, increasing in severity, and generally localized or most intense at the point of origin of the inflammation. When the inflammation originates in the rupture of a gastric ulcer, the pain is in the epigastrium, or even more frequently it is re-

ferred to the back or shoulders. If it originates from disease of the appendix, the pain is greatest in that region. It is aggravated by pressure or by movement, even by respiration. On this account the patient lies with his knees drawn up, and his breathing is shallow. Vomiting is an early and often a persistent symptom, and the bowels are usually constipated. Sometimes diarrhea develops and vomiting may be absent, but eructations are usually present in such cases. The tongue, although moist and coated in the beginning, generally becomes dry, brown, and perhaps fissured. Micturition becomes frequent and painful when the vesical peritoneum is involved. Retention may follow from paralysis of the muscular coat of the bladder, or it may be caused by the administration of morphin. The urine generally contains a trace of albumin and rather large quantities of indican. In cases in which the sensibilities are obtunded, notably in typhoid fever, the pain and tenderness may be readily overlooked on account of the psychical condition. The abdomen almost always becomes distended, soon after the onset of the disease, and the distention often becomes extreme, particularly when the abdominal muscles are poorly developed. These muscles, when strong, often become firmly contracted on the side of the pain, and may, for a time at least, produce a retraction of the abdominal wall. The abdominal distention is chiefly due to the accumulation of gas in the relaxed bowel, but in some cases in part to the accumulation of serum in the peritoneal cavity.

General Symptoms.—In the beginning, especially in perforative and septic cases, the paroxysm is usually accompanied with chilly sensations or a distinct rigor. The temperature often rises rapidly to 104° or 105° F. (40.0°—40.5° C.) and the pulse becomes rapid, small, and wiry, often reaching 100 or 120 and later 140 or 150. The respiration is also accelerated, possibly to from 30 to 50, and shallow. The elevation of the diaphragm and the rapid, weak action of the heart both contribute to the embarrassment of respiration. The temperature curve becomes more moderate after the onset, and its general features are those belonging to a septic infection. The evidences of suffering soon become apparent; the face becomes pinched, moist, and shrunken, often deeply cyanotic, brownish, lead-colored, or livid, and the expression is anxious unless the sensorium is obtunded by the underlying condition. This so-called Hippocratic face is highly indicative of the disease. Restlessness usually develops and there may be mild, muttering delirium or stupor. A persistent hiccough sometimes develops. In asthenic cases the prostration may amount to collapse; the pulse then becomes running and extremely feeble, and the extremities cold and livid. The clinical picture of peritonitis is often greatly modified, however, by the features of the primary disease.

Physical examination reveals the abdominal distention, the abdomen often appearing widened or the rigidity of the muscles producing extreme hardness, even concavity. Palpation reveals also extreme tenderness, and a friction fremitus is sometimes perceptible, but not so uniformly as in chronic peritonitis. With the development of ascites, fluctuation is elicited. Owing to the elevation of the diaphragm, the apex beat of the heart is displaced upward and toward the left. Percussion yields a high-pitched tympanitic note above the water-line and flatness in the

dependent portions. After perforation of the stomach or intestine, the liver dullness is often absent, but the same effect is occasionally produced by the crowding of intestinal coils between the liver and the abdominal wall. The upper limit of hepatic dullness is also displaced upward. The spleen may be unrecognizable. In some cases the areas of dullness are circumscribed owing to the formation of pouches. Such pouches may occur in general peritonitis, although more characteristic of the localized disease.

The course of the disease is usually rapidly fatal, terminating in from 36 to 48 hours in the worst cases, but occasionally running a course of 8 or 10 days. The average duration is from 3 to 5 days. The approach of death is generally signalized by a more or less rapid failure of the heart's action and the deepening of the stupor. The breathing becomes shallow, the lividity of the skin becomes more intense and the surface colder, but the rectal temperature becomes higher. Sometimes the end is instantaneous, probably through cessation of the heart's action.

Diagnosis.—The chilliness, abdominal tenderness, pain, and distention, with vomiting, fever, prostration, and Hippocratic facies, generally establish the diagnosis. But some symptoms may be lacking. The previous history, with the discovery of a possible cause, whether in the abdomen or pelvis, may throw needed light upon the condition. The diseases which are most likely to cause error are acute enterocolitis, obstruction of the bowel, embolism of the superior mesenteric artery, and the so-called hysterical peritonitis.

In *acute enterocolitis* the pain is more colicky and paroxysmal and the diarrhea is more severe. There is seldom rigidity of the abdomen or persistent elevation of the knees.

Intestinal obstruction often presents symptoms so like those of peritonitis that the diagnosis is extremely difficult. The chief reliance is then to be placed upon the history of the case.

Embolism of the superior mesenteric artery causes sudden, severe pain, vomiting, and collapse, which cannot for a time be differentiated. The rectal temperature would not, however, be elevated. Rupture of abdominal aneurism, although generally instantly fatal, has been suggested as a possible source of error.

Hysterical peritonitis can generally be differentiated through the presence of other hysterical manifestations. There is the usual exaggeration of symptoms and a cessation of them when the attention has been diverted.

LOCALIZED PERITONITIS.

Of this there are three principal forms, corresponding to the location and origin of the disease, namely, subphrenic, appendicular, and pelvic. (1) The first of these, the subphrenic abscess, has been described under the head of Perihepatitis (p. 522).

(2) *Appendicular Peritonitis.*—This is the most frequent form of peritonitis in males and is due to inflammation about the appendix. Its location corresponds to the varying locations of the appendix. The localization of the inflammation is due to the early formation of adhesions between the layers of the peritoneum, which completely shut out

a limited portion from the general cavity. Complete healing with obliteration of the appendix sometimes results from it.

(3) *Pelvic peritonitis* results from inflammation about the uterus and more particularly about the Fallopian tubes. The disease is generally septic, gonorrheal, or tubercular in character. Here, too, the process may be completely shut off from the general peritoneum, and recovery is possible, but not infrequently the arrest is but temporary, an abscess is perhaps formed which ultimately ruptures into the general peritoneal cavity, or a tuberculous infection may extend in the usual manner from the localized focus to the general cavity.

Prognosis of Peritonitis.—Acute general peritonitis is an exceedingly dangerous disease. Its course depends chiefly upon the cause, and the character of the exudate. When it has arisen from the perforation of a gastric or intestinal ulcer or from the rupture of an abscess, it is almost inevitably fatal. That due to puerperal sepsis or induced abortion is also fatal, as a rule. In other forms of localized peritonitis, the prognosis is less unfavorable.

Treatment.—The severity of the pain demands the hypodermic administration of morphin, gr. ¼ to ⅓ (0.01—0.02), at short enough intervals to keep the patient comfortable, but with caution. The fact that patients with peritonitis require large doses of opium is not true of the hypodermic administration of morphin. The patient should be propped with pillows in a position that is most comfortable, the knees being well drawn up. Hot fomentations or turpentine stupes applied to the abdomen at short intervals are soothing. In some instances, cloths wrung out of ice-water are more agreeable. Hot-water bottles, ice-bags, and poultices are generally objected to on account of their weight. The application of twenty or thirty leeches is sometimes beneficial.

The administration of laxatives in peritonitis was strongly advocated a few years ago, as a reversal, perhaps, from a too free resort to opium. At the present time there are probably few who would withhold morphin during the painful period, yet there are some who recommend the administration of saline laxatives in concentrated solution, believing that it favors the exosmosis of serum from the blood-vessels of the intestine, and through it the removal of collateral edema. The increased peristalsis is believed to prevent the danger of peritoneal adhesions. Much success has been claimed for the treatment, but, as Osler frankly remarks, the reports of cases do not always convince one that peritonitis actually existed. As in other inflammatory conditions, rest is essential. The saline treatment may be resorted to after operation, but never in cases due to perforation. Vomiting is sometimes arrested by the morphin; otherwise it may yield to a total suspension of all food and drink. Sometimes the giving of small fragments of ice or sipping hot water or hot brandy will check it. The meteorism is often relieved by lavage of the stomach and rectal injections of cold water. The passage of the rectal tube sometimes gives exit to a large quantity of gas. The diet should consist chiefly of milk, either hot or cold, plain or with the addition of lime-water or a carbonated water. Peptonized milk is preferred by some writers. If it cannot be retained, rectal alimentation may be resorted to. Whisky and strychnin should be given freely to support the strength.

The possibility of surgical interference should be considered at the very beginning, and a surgeon should generally be consulted. In all cases of localized peritonitis, and particularly when the inflammation is spreading, the surgical treatment offers greater hope for recovery than the medical, which is, after all, only palliative.

CHRONIC PERITONITIS.

This may be either local or general in extent, adhesive, proliferative, or hemorrhagic in character. The disease may be the outcome of the acute form, but it is more frequently tubercular or cancerous.

(1) **Local Adhesive Peritonitis.**—This is a common form, occurring particularly after appendicitis, and very often found over the surface of the spleen and liver after localized acute inflammation. These organs are firmly bound to the overlying structures—the liver and spleen to the diaphragm, the appendix to the nearest structures, and the coils of the intestine are usually firmly matted or connected by fibrous bands. Strangulation of the intestine sometimes results from the passage of a loop through the abnormal opening thus formed.

(2) **General Adhesive Peritonitis.**—This form may result from an acute attack, but it often develops in connection with such diseases as hepatic cirrhosis or chronic passive congestion. The peritoneum becomes thickened, adhesions form between all contiguous surfaces, until the entire sac may be obliterated and the intestinal coils completely matted together. There are sometimes great thickening and deformity of the omentum and mesentery. The liver and spleen are covered with a thick layer of fibrin, and a friction fremitus may sometimes be felt or the surface may become completely united with the diaphragm and other contiguous structures. Any part of the cavity that is not entirely obliterated is generally filled with serum, which may be clear, but is generally turbid with fibrin, cellular débris, or occasionally with pus.

(3) **Proliferative Peritonitis.**—The peritoneum becomes gradually thickened, but the surfaces are not uniformly adherent. Moderate effusion is generally present, occasionally a pronounced ascites. The thickening is not uniform. The omentum is occasionally rolled into a hard mass extending across the abdomen between the stomach and colon. In some cases the liver and spleen are covered by a thick mass of almost cartilaginous firmness, and a similar condition may be encountered in other regions. Small nodular masses grossly resembling tubercles are sometimes found. The mesentery may be much shortened by the contraction of the new tissue. The gastrohepatic omentum may be contracted and the portal vein compressed, the liver, spleen, and other organs reduced in size, and the walls of the intestine so contorted that the mucous membrane is thrown into large folds. The peritoneal cavity may be divided into compartments as in tubercular peritonitis, with a variable quantity of fluid in each. This form of the disease is met with especially in alcoholic subjects, often in connection with hepatic cirrhosis. The friction fremitus is sometimes felt in this form, as it may be, at times, in all others.

(4) **Chronic Hemorrhagic Peritonitis.**—In this unusual form, described by Virchow, the peritoneum is here and there covered with a layer of

new connective tissue possessing large vessels from which an extravasation of blood occasionally occurs and forms a hemorrhagic layer. Such alternating layers are met with particularly in the pelvis. The blood-stained effusions sometimes found within the cavity in tubercular and cancerous peritonitis are not indicative of this form of peritonitis.

Symptoms.—Chronic peritonitis frequently presents few or no symptoms, following so insidiously upon an acute attack that recovery is thought to have taken place. Pain and tenderness are generally present, but there may be little or no distention. Fever may occasionally develop. All the symptoms are intermittent in their appearance. The action of the bowels is irregular. The patient becomes anemic, emaciated, and feeble. The diagnosis is difficult and the condition is often overlooked during life.

Treatment.—Little can be accomplished through medicinal treatment further than to support the strength and allay the pain. Benefit has been claimed for inunctions of ichthyol or mercurial ointments.

CANCER OF THE PERITONEUM.

Cancer rarely attacks the peritoneum primarily, but may reach it directly or through metastasis from a primary focus in some adjacent part of the gastrointestinal canal. Any form of the disease may be encountered, but the encephaloid, scirrhous, and colloid are the more common. One or more large masses may be developed, or there may be numerous small nodules. The disease occurs only after the usual age limit, and is more frequent in women than in men.

Symptoms.—The manifestations of the disease are those of local, or, more frequently, of chronic peritonitis. The diagnosis can rarely be made unless the primary focus be recognized, for it is often only after tapping that it is possible to feel the nodules that are present. It is not always possible then to differentiate the nodules from those of tuberculosis. The presence of a large number of tumors, however, favors the diagnosis of cancer. In colloid cancer, the peritoneal cavity may be found distended with a thick, gelatinous colloid matter instead of serum.

The ***prognosis*** is always unfavorable, and the treatment is wholly palliative.

ASCITES.

ABDOMINAL DROPSY, HYDROPERITONEUM.

Definition.—An accumulation of serous fluid within the peritoneal cavity. It is a symptom common to many diseases.

Etiology.—Ascites is met with in either sex and at any time of life, so many are the conditions that may give rise to it. It may be due to either local or general causes.

(1) *Local Causes.*—The most important of these are: (*a*) Chronic inflammation of the peritoneum, which may be simple, tubercular, or cancerous; (*b*) obstruction of the portal circulation, as in cirrhosis, cancer, or other diseases of the liver, chronic passive congestion due to heart disease or chronic pulmonary disease, or thrombosis; or through the pressure or growth of tumors, peritoneal contractions or adhesions.

The pressure of tumors or aneurism upon the lymph-vessels, hepatic vein, or inferior vena cava is also a possible cause; (*c*) the growth of solid tumors within the abdomen, less frequently an enlargement of the spleen, as that due to malaria.

(2) *General Causes.*—These are: (*a*) Conditions which lead to general dropsy, as valvular heart disease, chronic emphysema, or so-called fibroid phthisis; (*b*) altered conditions of the blood which favor the transudation of serum, as in chronic malaria, chronic nephritis, cancer, amyloid disease, or syphilis. (*c*) Two other forms of ascites should be mentioned. The first is due to the escape of fluid from the lacteal vessels as a result of injury, ulceration, or other disease, compression of the thoracic duct or thrombosis of the left subclavian near its entrance, or to the presence of filaria, and known as chylous ascites. The other is due to fatty cellular degeneration with the production of so-called adipose ascites, a rare form generally associated with cancer or tuberculosis.

Symptoms.—The first symptom is usually a gradually increasing distention of the abdomen. This is often overlooked by the patient, however, until other evidence of dropsy appears. Sometimes there is a sense of fullness and weight, and as the accumulation increases, respiration is interfered with. Gastrointestinal disturbances and alterations of the quantity and character of the urine are often observed, but they are generally referable to the condition producing the ascites.

Physical Examination.—*Inspection.*—The abdomen is prominent, often flattened at the sides. When the distention is great, the skin becomes stretched and glazed, and lineæ albicantes may be produced. The superficial veins are distended, those of the abdomen becoming continuous with dilated branches of the external mammary, and the circulation may be found to be reversed, passing from below upward, as can be demonstrated by pressure. Around the navel there is sometimes a varicose wreath known as the caput Medusæ. The umbilicus may protrude, rarely it appears to be obliterated. The respiratory movements are almost entirely thoracic.

Palpation reveals fluctuation when the accumulation amounts to several liters. This is brought out by placing one hand upon the side of the abdomen, and striking a sharp, quick blow with the fingers of the other hand at a corresponding point on the opposite side.

Percussion elicits dullness over the fluid, which gravitates to the most dependent region in the different positions that the patient may occupy, and a tympanitic note over the intestines, which float above the fluid. When the patient stands, the dullness is in the lower part, rising a variable distance above the brim of the pelvis; when he is in the recumbent posture, it is along the flanks. A small quantity of fluid can sometimes be recognized by placing the patient in the knee-elbow position and percussing over the most dependent area.

Diagnosis.—The recognition of ascites is not difficult when the accumulation of fluid is sufficient to cause distention, dullness, and fluctuation. Less than a quart (liter) of fluid cannot be detected with certainty. The principal condition to be excluded is ovarian cyst, but pancreatic cyst, echinococcus cyst, pregnancy, and a distended bladder are possible sources of error.

Ovarian cyst is generally preceded by normal health or by nothing more serious than dysmenorrhea. The distention is at first unilateral, and it usually remains asymmetrical until the tumor becomes extremely large. The fluctuation is limited to a definite area, and does not change with change of position. Vaginal examination reveals uterine displacement; sometimes the cyst can be felt. The fluid accumulates after aspiration much more slowly than does that of ascites. The ascitic fluid is usually of a clear, pale straw-color, and has a specific gravity of from 1.010 to 1.015, while that of ovarian cyst is turbid and 1.020 or over. The ovarian fluid is often highly albuminous, and does not coagulate spontaneously, as the ascitic fluid may do. Cholesterin may be found in the ovarian, but not in the ascitic fluid. Echinococcus and pancreatic cysts are readily excluded by the character of their fluid. The echinococcus fluid contains hooklets and fragments of the chitinous membrane, that of the pancreatic cyst the digestive ferments. The distended bladder should not cause error, for it is immediately relieved on catheterization. In pregnancy, the fetal heart, the movements, and other signs are sufficient for a diagnosis, if proper examination be made, regardless of the quantity of amniotic fluid, the only source of error.

Prognosis.—The presence of ascites is always of serious import, for it often indicates the approach of the fatal termination of the underlying disease; yet ascites has lasted from ten to twenty years. All depends upon the gravity of the causative disease.

Treatment.—The treatment of ascites embraces the application of therapeutic and other agencies for the removal of the fluid. The general management of the patient and measures to obtain a permanent cure belong to the treatment of the disease causing the condition. It sometimes happens, particularly in cirrhosis of the liver, that a systematic withdrawal of the fluid greatly favors the production of changes in the circulation, which, for a time at least, check the formation of the effusion.

Hydragogue cathartics are usually first resorted to, especially when the condition depends upon disease of the heart or kidneys. A dram (4.0) of compound jalap powder should be administered on alternate mornings. Potassium bitartrate in large doses is to be preferred in many cases. It may be conveniently added to lemonade to be drunk during the day. Magnesium sulphate in large doses and concentrated solution an hour before breakfast often acts well. Diuretics may be administered with benefit when they are not contraindicated by the condition of the patient. Repeated small doses of calomel, alone or with digitalis, may prove effective, particularly, however, in cases of heart disease. Tapping often becomes necessary, and should not be too long delayed, especially in hepatic cirrhosis.

SECTION VII.

Diseases of the Kidneys.

ANOMALIES OF FORM AND POSITION.

Anomalies of form and position are thus classified by Newman: A. Displacements without mobility. (1) Congenital displacement; (2) congenital displacement with deformity; (3) acquired displacement.—B. Malformations. (1) Variations in number: (*a*) Supernumerary kidney, single kidney, congenital absence of one kidney, atrophy of one kidney, absence of both kidneys. (2) Variations in form and size: (*a*) Congenital variation in form, lobulation, etc.; (*b*) hypertrophy of one kidney, fusion of two kidneys, horseshoe kidney, sigmoid kidney, cystic kidney.—C. Variations in pelvis, ureters, and blood-vessels. It is scarcely that these anomalies assume clinical importance.

MOVABLE KIDNEY.

WANDERING KIDNEY, PALPABLE KIDNEY, NEPHROPTOSIS.

Definition. A condition of abnormal mobility of the kidney, much [illegible] sometimes affecting both kidneys.

Etiology. The condition is rarely congenital. The acquired form is [illegible] in women. In some instances it is due, in part at [illegible] congenital looseness or to a relaxation of peri- [illegible] unusual length of the renal artery. The con- [illegible] with enteroptosis. An important factor in [illegible] the relaxation of the abdominal walls by [illegible] favors its production. Trauma, as [illegible] influential in some instances.

[illegible] may exist without producing symp- [illegible] accidental discovery during physical examination [illegible] from the post-mortem table. In some cases, [illegible] intermittent disturbances occur which may be [illegible] local and reflex symptoms.

A sense of dragging weight or pressure in the [illegible] intercostal neuralgia is often complained of, [illegible] Severe pain or reflex symptoms sometimes [illegible] sudden change in the position of the affected

[illegible] reflex manifestations may be confined to [illegible] may be general. They are intermittent, [illegible] influenced by any influence which aggravates [illegible] a sudden change of the location of the [illegible] disturbances are common. Nausea,

vomiting, indigestion, and constipation can often be distinctly traced to the renal displacement; but such conditions as gastric dilatation and icterus are probably no more than coincident conditions. Cardiac palpitation, anasarca, intestinal obstruction, and functional disorders of the uterus have been attributed to the pressure of a dislocated kidney.

Dietl's crises are almost distinctive. They consist of sudden, sometimes periodical, attacks of sharp abdominal pain, with chill, fever, nausea, vomiting, and collapse, probably due, as Dietl thought, to compression or twisting of the ureter. They may be induced also by overindulgence in food or strong drink. During the attack the urine usually becomes highly colored and charged with uric acid and oxalates, sometimes containing also blood and pus. Albuminuria or hemoglobinuria may be present. The displaced kidney becomes swollen and tender. The compression of the ureter sometimes leads to permanent or intermittent hydronephrosis.

The general reflex symptoms usually assume the form of hysteria, neurasthenia, or simple nervousness, with anxiety and melancholia, especially pronounced for a time after the discovery of the tumor.

Physical Examination.—The patient should be placed on the back, with the head low and the abdominal walls thoroughly relaxed. Bimanual palpation is then practiced with the left hand over the lumbar region behind the last ribs, and the right over the hypochondriac region. The kidney, if sufficiently displaced, can be felt, as a firm, globular body, just below the margin of the liver. Sometimes it is possible to feel the lower edge of the kidney only when the patient takes a full inspiration. This is called a *palpable* kidney. If the entire organ can be felt and the finger passed above the upper margin of it during inspiration, it is known as the *movable* kidney. If the organ can be depressed below the level of the umbilicus, it is designated a *floating* kidney. To this class belong those rare instances in which the kidney sinks into the pelvis.

Diagnosis.—Few conditions are likely to be confounded with this after careful, thorough examination. A floating kidney is often momentarily suggested by a movable cancer of the pylorus, fibromata, or secondary carcinomata of the omentum or intestine, fecal impaction, tumors of the gall-bladder or ovary, and movable spleen. But the peculiar kidney shape, with notched edges, can seldom be mistaken, and still more significant is the peculiar feeling of nausea that is induced by pressure upon it.

Treatment.—It is often advisable to withhold from the patient the nature of the condition, especially when the displacement is so slight as to be classed with the palpable or movable kidneys, and when it produces no serious disturbance. When, however, painful crises occur, more or less radical measures may become necessary. Sometimes the organ can be replaced by taxis, and nothing more is necessary than rest in bed until the pain has subsided. Morphin must sometimes be administered. After a paroxysm the patient should avoid jolts and jars, lifting and other possible causes of the crises. It is often necessary to treat the neurasthenic condition of the patient rather than the renal condition. Relief often follows a decided gain of adipose tissue. Surgical measures are required in extreme cases. The kidney capsule may be stitched to

the abdominal wall (nephrorrhaphy), but the result is not always permanent. Extirpation of the kidney is a more serious operation, often successful, sometimes fatal, and the loss of one kidney in itself is not always free from injurious effects.

HYPEREMIA OF THE KIDNEY.

Hyperemia may be active or passive, acute or chronic. ***Etiology.***—**Active hyperemia** is always present in the early stage of acute parenchymatous nephritis, and it is not always possible to distinguish clearly between a simple hyperemia and that of Bright's disease. The former condition is frequently induced, however, by: (*a*) The toxemia of the acute infectious diseases, probably the most frequent cause, or (*b*) such irritant drugs as turpentine, cantharides, copaiba, carbolic acid, potassium chlorate, phosphorus, arsenic, and alcohol. (*c*) It is supposed to occur in one kidney after sudden arrest of the function of the other.

Morbid Anatomy.—The kidney is large, of dark red color, the capsule is tense, but not adherent, and blood flows from the cut surface. The columnar epithelium is cloudy and desquamating, as in acute nephritis.

Symptoms.—The urine may be increased and of low specific gravity, or diminished and of higher specific gravity. Albumin and oxalates, sometimes blood, are found in it. The treatment consists in removal of the cause if recognized, rest in bed and milk diet for a few days. When the hyperemia is extreme, and especially if strangury or anuria develop, the hot pack and dry cups to the region of the kidneys are beneficial. Internal medication is not usually necessary, and diuretics may prove injurious.

Passive Hyperemia.—***Etiology.***—This condition is induced (*a*) By any influence which retards the flow of blood through the ascending vena cava, as chronic valvular disease of the heart, emphysema, interstitial pneumonia, myocarditis, obliterative pericarditis, or arteriosclerosis; (*b*) by the secondary collateral obstruction in hepatic cirrhosis; and (*c*) by compression of the renal veins by tumors, the gravid uterus, or ascitic fluid.

Morbid Anatomy.—The kidney is large, often intensely congested, the cortex is deep red and the pyramids purple. The capsule becomes more or less adherent, and the cut surface bleeds freely. The substance is firm, especially in the heart-kidney, owing to hyperplasia of the connective tissue, intertubular cellular infiltration, and not infrequently thickening of the glomerular capsule. The condition cannot always be differentiated from diffuse nephritis, into which it usually merges.

Symptoms.—These are for the most part confined to the concentration of the urine, diminution of quantity, with increase of color and solid ingredients. Albumin and casts are present and sometimes blood-corpuscles. Uremic symptoms may develop in severe cardiac cases. Edema of the lower extremities, dyspnea, and gastrointestinal disturbances often accompany the condition, but they are attributable to the cardiac incompetency.

Treatment.—The treatment must be directed in most instances to the cardiac condition. Digitalis and nitroglycerin may be indicated, the

latter especially to stimulate the renal circulation. Hot applications or dry cups over the kidneys are sometimes of benefit. When extreme congestion, threatening life, results from pregnancy, it is sometimes necessary to induce labor. The diet should be liquid, chiefly milk. Diuretics should be employed with caution, if at all.

ANOMALIES OF SECRETION.

Anuria.—Total suppression of the urine occurs under a variety of causes. (1) It may be encountered in the new-born infant as a result of absence of the kidneys, a condition incompatible with life of more than a few days' duration; (2) it occurs as a symptom of severe acute nephritis; (3) as a result of the obstruction of both ureters with calculi, usually occurring in men; (4) as a result of toxemia in the various acute infectious diseases, especially cholera, yellow fever, or the irritant diuretics referred to as causes of hyperemia of the kidneys; (5) from extensive disease or excision of a single kidney; or (6) as a result of the collapse following injury, surgical operations, or the passage of the catheter. It sometimes occurs also as a manifestation of hysteria, but retention and deception are more common in this connection.

Symptoms.—In some cases there is a remarkable absence of symptoms. Convulsions occur in comparatively few cases, and the other manifestations of uremia are equally rare. The patient may survive from one to two weeks, and one case is reported in which death did not occur for nineteen days.

Treatment depends upon the cause. Mechanical obstruction calls for prompt surgical measures. In nonobstructive cases, hot applications or dry cups to the loins, free purgation, and sweating induced by hot baths or pilocarpin may prove effective in re-establishing the secretion. Pure water should be drunk freely. Rectal irrigations with a large quantity of hot normal saline solution has been employed with excellent result.

Albuminuria.—The presence of albumin in the urine was formerly regarded as a pathognomonic sign of Bright's disease—a name which for many years included all forms of nephritis. It is now recognized as a symptom common to many affections, not of the kidneys alone, but of the blood, blood-pressure, the general system, or the nervous system. These are conveniently grouped under the two heads of Albuminuria without Definite Lesions of the Kidney, and Albuminuria with Definite Lesions of the Kidney.

Albuminuria without Definite Kidney Lesions.—(1) *Functional Albuminuria.*—The term physiological albuminuria has also been applied to this form, but rather unfortunately, since it is not probable that albumin ever escapes into the urine independently of some defect in the integrity of the epithelium of the glomerulus or of the tubules, with perhaps two exceptions. These are, first, the slight trace of nucleoalbumin that is almost constantly recognizable in normal urine with the more delicate tests; and, second, the elimination of egg-albumen after it has been ingested in large quantity.

Different names have been applied to albuminuria appearing under different conditions. (*a*) *Cyclic*, intermittent, and paroxysmal albumi-

nuria are terms applied to cases in which the presence of albumin is intermittent, often following a regular daily course, disappearing during sleep, increasing with activity, or appearing in a cycle of twelve to thirty-six hours' duration. It is more frequently observed in boys, often, according to Teissier, the offspring of gouty or arthritic parents. In some instances it seems to be related to the ingestion of food. The quantity of albumin is seldom more than a trace, but it may be abundant. Hyalin casts are occasionally passed, but as a rule albumin is the only abnormal ingredient. The specific gravity may be increased, and the color high. Mucin and the proteids of semen, the prostatic secretion, or a leucorrheal discharge should be carefully excluded.

(*b*) The *albuminuria of adolescence* is closely related to the cyclic, if not identical with it. It has been looked upon in some instances as a persistence of an embryonic type, since it has been shown that the embryonic kidney secretes albuminous urine up to the time of birth.

(2) *Febrile albuminuria* has been regarded as a result of the pyrexia, but in most cases it is doubtless due to the action of the toxins of the febrile disease. It is especially frequent in connection with diphtheria, typhoid fever, pneumonia, and malaria. The quantity of albumin is usually slight, but the urine is concentrated, of high specific gravity and color, and the urates are increased. Casts are frequently found. A transient cloudy swelling of the renal epithelium is probably always present, but complete recovery usually occurs spontaneously soon after the toxic matter has been eliminated from the blood.

(3) *Hemic albuminuria* depends upon changes in the blood which favor the transudation of albumin. It is seen in profound anemia, pregnancy, syphilis, purpura, scurvy, or as a result of poisoning with metallic salts, turpentine, carbolic acid, and the irritant diuretics, and sometimes after ether or chloroform narcosis.

(4) *Neurotic albuminuria* is observed after convulsions, epilepsy, or tetanus, sometimes in exophthalmic goiter, cerebral hemorrhage, or injury of the brain.

Albuminuria with Definite Kidney Lesions.—Every form of inflammatory or degenerative disease of the kidneys is more or less constantly accompanied with albuminuria. The presence of a large quantity (it seldom exceeds 3 per cent by weight) is usually indicative of a correspondingly grave lesion; but the presence of casts, particularly of fatty casts, is of greater diagnostic significance. In the chronic interstitial form, however, albumin is rarely abundant, and may be temporarily absent.

Tests.—The tests for albumin and other ingredients of the urine will be found upon page 727 *et seq.*

Prognosis.—The prognosis of albuminuria depends upon its cause. Much can be inferred also from its persistency. Febrile and cyclic albuminurias are generally transitory, but they cannot be regarded as trivial when they become persistent. Age is an important factor in prognosis. Albuminuria of moderate degree is comparatively common in advanced age, increasing in frequency after the fortieth year, and although it denotes decline and probably renal degeneration, it is not necessarily of serious moment. The presence of casts in connection with this form of albuminuria has been variously estimated. F. C. Shat-

tuck regards the presence of a small quantity of albumin with small hyalin and granular casts in those past fifty years as often of little or no practical importance. Its presence should, nevertheless, arouse greater care to relieve the kidneys from excessive work in the elimination of effete matter.

Hemoglobinuria.—Hemoglobin, the red coloring matter of the blood, may be found in the urine in either of its three forms, but it is generally in the form of methemoglobin, especially when the urine is acid. It is due to a dissolution of the red blood-corpuscles (hemolysis) and may be toxic or paroxysmal.

(1) *Toxic hemoglobinuria* sometimes occurs: (*a*) In the more virulent infections, as yellow fever, typhus, malignant scarlet fever, malaria, sometimes in syphilis; (*b*) as a result of large doses of potassium chlorate or poisoning with pyrogallic acid, carbolic acid, arseniureted hydrogen, carbon monoxid, muscarin, turpentine, and other drugs; or (*c*) such blood-states as those of pernicious anemia, leukemia, purpura, scurvy, or after burns or the transfusion of blood from a different animal. It has been attributed to the action of quinin in the malarial patient, or to the direct action of cold, as in soldiers sleeping upon the ground. Epidemic hemoglobinuria is occasionally observed among the new-born.

(2) *Paroxysmal hemoglobinuria* occurs chiefly in adult males at variable intervals, often after exposure to cold or overexertion. It sometimes occurs in connection with Raynaud's disease. Malarial hemoglobinuria, although toxic, may be periodic.

Symptoms.—The attack frequently comes on suddenly without recognizable cause, or it may be preceded by a chill and fever and accompanied with vomiting, diarrhea, and pain in the lumbar region. Jaundice is sometimes present. Two or three paroxysms sometimes occur in a day, with intervals in which clear urine is voided. The essential feature is the presence of methemoglobin, without corpuscles or coagula.

The ***prognosis*** is favorable, except in connection with the more malignant diseases, or in acute poisoning.

Treatment.—Rest is essential. Dry cups or other counter-irritation over the kidneys sometimes arrests an attack. Blisters should not be applied. Hot drinks are of benefit; stimulants must be avoided. Ergotin administered hypodermically is sometimes effective, and amyl nitrite is said to arrest or cut short an attack. Quinin should be given without hesitation in malarial cases.

Hematuria signifies the presence of blood in the urine. This occurs in certain general diseases and in local renal conditions.

(1) The general diseases are: (*a*) Those causing profound alteration of the blood, as pernicious anemia, leukemia, purpura, scurvy, malaria, and occasionally other acute infections; (*b*) those causing renal congestion, as the late stages of valvular heart disease, interstitial pneumonia, or the hepatic cirrhoses.

(2) *Local Conditions.*—(*a*) It may originate in the kidney from intense acute nephritis, renal infarction, calculus, tuberculosis, pyelitis, parasites, or injury, including injury of a floating kidney; (*b*) from injury, calculus, or disease of a ureter, the bladder, or urethra, particularly from malignant or tubercular growths or ulceration, gonorrhea, the rupture of a dilated vein, or parasites. The term endemic hema-

turia is applied to a form which occurs in tropical countries as a result of the filaria sanguinis or distoma hematobium.

Diagnosis.—Blood in the urine is recognized by the color, ranging from a smoky hue to a dark brown or black, often by the presence of clots, and by the reactions of albumin and the discovery of corpuscles on microscopic examination. Blood-casts are found in renal hematuria.

Hematoporphyrinuria.—Hematoporphyrin, iron-free hematin, sometimes appears in the urine in connection with several diseases, especially tuberculosis, leprosy, pneumonia, pleurisy, and hepatic cirrhosis, or after prolonged use of sulphonal or trional. The urine is dark and concentrated.

Albumosuria occurs during many febrile conditions, in chronic suppuration and in connection with neoplasms or syphilis of bone, especially in the myelomata. The urine is often turbid. The precipitate formed by the addition of nitric acid is dissolved by heat, and again thrown down upon cooling. The reaction is often referred to as the Bence Jones reaction.

Globulin appears in the urine generally, if not exclusively, in connection with albumin, and it often exceeds the latter in quantity. Its significance is the same as that of albumin, except that it is more exclusively present in acute parenchymatous nephritis and amyloid degeneration of the kidneys.

Chyluria.—This symptom has been referred to in connection with the filaria sanguinis, but it occasionally occurs independently of the presence of parasites. The urine has a milky appearance due to the admixture of fat-globules, or it may be pink from the presence of blood. Spontaneous coagulation sometimes occurs upon cooling. Nonparasitic chyluria is generally attributed to an accidental communication between a lymph-vessel and some part of the urinary tract.

Pyuria.—Pus is found in the urine in variable quantity in connection with all suppurative affections of the urinary passages. It may also enter the urine from other sources, as by the rupture of pelvic or other abscesses into the urinary passages. The conditions with which it is generally associated are: (*a*) Pyelitis, pyonephritis, pyonephrosis, and cystitis; (*b*) gonorrheal urethritis, leucorrhea, and prostatic abscess; and (*c*) rupture of a pelvic, perityphlitic, or perinephric abscess into the passages.

Lithuria.—Uric acid is found in excess, usually in the form of sodium or ammonium urates, chiefly in connection with gout or in the conditions described as lithemia. The urates are deposited in the form of crystals or as amorphous, "brick-dust" sediment; but it is only when the deposit is excessive that it is of importance, since a small deposit is normal after the urine becomes cold. An increased elimination of uric acid constantly follows excessive ingestion of proteid food. Quite a number of morbid conditions involving every vital system have been attributed to lithemia, and all are grouped by many writers under the head of the lithemic diathesis. There is, however, very little positive knowledge of the pathology of the conditions. Excessive eating and drinking, with deficient muscular exercise, are essential factors in the production of gout, a closely allied affection. Deficient oxidation is probably the important

element in the faulty metabolism of both affections. In gout, the urates are deposited about the joints, while in lithemia they are eliminated in excessive quantity. High arterial tension, with a tendency to degenerative changes in various tissues, is also common to both conditions.

Phosphaturia.—An increased elimination of phosphates occurs in extensive destructive lesions of bone, as rickets, osteomalacia, and tuberculosis, in acute yellow atrophy and cirrhosis of the liver, pernicious anemia, leukemia, and in diseases affecting the nerve-centers. It sometimes occurs in flatulent dyspepsia or simply as a result of the ingestion of food rich in phosphates, and cases are sometimes met with in which an extremely large quantity is more or less constantly discharged without recognizable cause further than a decomposition of the urine within the bladder. The phosphates are usually deposited in the form of calcium or magnesium phosphates, or of ammonium-magnesium phosphates, the so-called triple phosphates. The term phosphatic diabetes has been applied to an excessive elimination of phosphates with polyuria, abnormal appetite, emaciation, furunculosis, and other symptoms resembling diabetes. Sugar has sometimes been found in the urine of these cases. The phosphates may be deposited from the urine as a result of decomposition due to cystitis or other causes, when they are not excessive in quantity.

Oxaluria.—A small quantity of oxalic acid is normally an ingredient of the urine in the form of calcium oxalate, being a derivative of the acid entering the system in fruit and vegetables. An excessive elimination occurs for the most part as a result of the ingestion of such articles as tomatoes, turnips, onions, apples, and pears. It not infrequently occurs also in the presence of gastric or intestinal indigestion, particularly when free hydrochloric acid is absent from the gastric juice. Rarely an excessive formation of the calcium-oxalate crystals within the urinary passages leads to the formation of calculi. Acid fermentation of mucus in the bladder is believed to produce oxaluria in some cases. Various nervous manifestations, especially hypochondriasis, neurasthenia, and less pronounced mental depression or languor are sometimes associated with oxaluria. Albuminuria is sometimes induced apparently by the irritation of the urinary passages by the minute crystals.

Cystinuria is a rare condition of the urine, usually associated with jaundice due to hepatic disease. A family tendency to the condition has been observed. Its chief importance lies in the fact that cystin sometimes forms the nucleus of a calculus.

Indicanuria.—Indican or potassium indoxylsulphate is recognized in the urine only upon the addition of strong acid, by which it is oxidized into indigo. It is regarded as a derivative of indol formed in the intestine by bacterial decomposition of proteid, especially in cases of obstruction, chronic peritonitis, wasting diseases, or carcinoma.

Melanuria is usually associated with melanotic sarcoma. The urine acquires a dark color from the presence of pigment in solution or in granular form. In some instances, before melanin appears, the urine is found to acquire a dark color after standing, from the presence of melanogen.

Alkaptonuria.—This condition, in which alkapton is found in the urine, may be of long duration, occurring at longer or shorter intervals for

many years, and at any period of life. Several members of a family may be affected. It is increased by meat diet, and in some cases of tuberculosis. The reaction has been attributed by different writers to uroleucinic, uroxanthic, or homogentisinic acid. Some regard it as due to intestinal decomposition in some manner affecting the tyrosin normally present, since it has been found that the administration of tyrosin to the alkapton patient increases the quantity eliminated. The administration of intestinal antiseptics does not arrest the excretion.

Lipuria, in which molecular or crystalline fat appears in the urine, may result from the excessive ingestion of fat, as codliver oil, and it may occur in such affections as diabetes, chronic diffuse nephritis, chronic suppuration, fat-embolism from fracture of bone, chyluria, and phosphorus-poisoning. The urine becomes milky in extreme cases.

Lipaciduria is a term applied to a condition in which the urine contains fatty acids, acetic, butyric, formic, or propionic. It has been observed after the eating of a large quantity of fat, and sometimes in connection with nephritis.

Diaceturia signifies the elimination of ethyldiacetic acid in the urine. It is generally observed in the acute infectious diseases or in diabetes. In the latter connection it frequently indicates the approach of coma. Oxybutyric acid sometimes accompanies the diacetic acid in diabetes.

Pneumaturia denotes the passage of gas from the urethra after the discharge of urine. It is due to the decomposition of the urine by gas-forming bacteria or to the entrance of gas from the bowel through a fistula. Air may enter also during catheterization or cystoscopic examination, to escape at the next urination. The diagnosis can be established by catheterizing the patient with the end of the catheter held under the surface of water.

UREMIA.

Definition.—A form of autointoxication which generally occurs in acute or chronic nephritis or in conditions attended with anuria. The nature of the toxic substance is not known; urea is no longer regarded as the sole cause of the condition. Several theories are entertained in regard to the nature and cause of the condition.

Theories of Uremia.—(1) The most commonly accepted view is that the intoxication is due to the retention of one or probably several nitrogenous *excrementitious substances.* The relation of urea, uric acid, and the urates to the condition is no longer looked upon as an important one. Other members of the same group are probably of greater activity. The blood-serum becomes highly toxic during the paroxysms; all are not agreed, however, as to whether this toxicity is due to the retention of poisons normally present or to the production of new toxic matter.

(2) *Brown-Séquard's theory* refers the toxemia to an undemonstrated internal secretion of the kidney, similar to that of the thyroid gland or suprarenal bodies.

(3) *Traube's theory* attributes the symptoms to edema of the brain.

(4) Delafield regards the motor symptoms as due to contraction of the arteries by some other influence than toxemia.

Symptoms.—Uremia may be acute or chronic. To these forms is added by some authors a third, a latent form, supposed to exist in cases of anuria.

Acute Uremia.—The onset may be sudden and severe or gradual, with symptoms of the same character, but of less severity. The attack may develop in the course of any form of nephritis, but more particularly in an acute nephritis due to an infectious disease. The prominent symptoms are headache, vomiting, dyspnea, convulsions, and delirium or coma. (*a*) The *headache* is generally occipital, extending to the neck, and intensely severe. It is often attended with vertigo and deafness. (*b*) *Vomiting* is often the first manifestation and sometimes the only one. Its chief characteristic is its persistency, occasionally leading to death from exhaustion. It may be preceded by intense nausea and accompanied with profuse diarrhea. In some instances the diarrhea develops without vomiting. A membranous colitis is sometimes found after death. (*c*) *Dyspnea* may be constant or paroxysmal, and occurs more particularly during the night. The patient must sit up as in asthma, and this often adds to his suffering by aggravating the edema of the lower extremities, when this is present. Restlessness is often developed. The lungs may be free from adventitious signs, but there is great danger of pulmonary edema, especially in the parenchymatous forms of nephritis. Cheyne-Stokes respiration is often observed, and the patient may become deeply cyanotic, the extremities blue and cold during the temporary cessation of breathing. (*d*) *Fever* is present in some cases, but it is usually slight until immediately before death, when the temperature may rapidly rise. The pulse is variable, usually full and bounding, but later becoming feeble and threadlike. It is not usually very rapid. (*e*) *Convulsions* sometimes develop toward the close. For days before the appearance of a distinct convulsion, however, there is often a more or less constant twitching of the muscles of the face and fingers. The convulsions are epileptic in character, either local or general, usually unattended with outcry. The patient generally becomes comatose before the seizure, and remains in this state between the attacks, which may recur as often as every hour or two. Hemiplegia or monoplegia may develop before or during the convulsion, and blindness or deafness often remains for a short period in cases that recover. General edema of the brain is often the only morbid condition found after death, aside from the kidney lesions. (*f*) *Delirium* develops in some cases. It may be mild and muttering, or it may assume the form of violent, even suicidal mania. (*g*) *Coma* with stertorous breathing may precede or follow the delirium. The condition is usually rapidly fatal; it may, however, subside into a chronic state or the uremic manifestations may entirely subside. Acute attacks not infrequently supervene upon the chronic form of the condition.

Chronic Uremia.—A chronic uremic condition is more commonly associated with arteriosclerosis or chronic interstitial nephritis than with the parenchymatous forms of kidney disease. The symptoms are the same in character, but less violent than those belonging to the acute form. Acute exacerbations are not unusual during the course of chronic uremia. The headache is persistent, usually occipital, sometimes frontal. The dyspnea is more or less constant, and the Cheyne-Stokes breathing

may develop during sleep. Insomnia is often a troublesome symptom, however, independently of other conditions. Gastrointestinal disturbances occur periodically. A persistent catarrhal or mycotic stomatitis often develops, with swelling of the tongue and gums, and intensely foul breath. The tongue is heavily coated, brown and dry. Nausea, vomiting, and diarrhea are more or less frequently present. Cramping in the leg muscles and dryness of the skin, with pruritus, are often exceedingly annoying complications. These manifestations frequently persist for years, becoming at times more pronounced or developing into an acute uremia with delirium or hallucinations, and finally terminating in coma or convulsions, or by the development of a technical infection of the endocardium, or more frequently of one of the serous membranes, the pericardium, pleura, peritoneum, or meninges.

Diagnosis.—The early symptoms of uremia are readily recognized, as a rule, upon thorough examination. Routine examination of the urine is the best safeguard against error. The urine should invariably be examined chemically and microscopically in a case of persistent headache, nausea, vomiting, diarrhea, and even when persistent muscular cramps or supposed neuralgic pains are complained of. The diagnosis is most difficult when the case is seen for the first time during coma. The conditions most likely to enter into the differentiation are diabetic or alcoholic coma, unconsciousness due to cerebral hemorrhage or opium-poisoning. When the comatose condition becomes protracted, it may simulate the stupor of typhoid fever, miliary tuberculosis, meningitis, or other infectious disease.

Diabetic coma comes on suddenly, without premonitory symptoms, and it is not accompanied with convulsive manifestations; the odor of the breath is sweetish or fruitlike, not fetid, and the arterial tension is usually normal and not increased. The bladder generally contains a considerable quantity of urine rich in sugar.

Cerebral hemorrhage is characterized by sudden onset, complete hemiplegia, irregular pupils, and conjugate deviation of the eyes. The breath has no distinctive odor, the arteries are usually atheromatous, the urine may be normal, and the reflexes are exaggerated on the affected side. A distinct convulsion is unusual. A brain-tumor sometimes causes confusion, but, as a rule, the comatose condition is preceded by a considerable period during which more or less typical localizing symptoms are observed. Meningitis may be excluded with difficulty in some cases. As a rule, however, it is not accompanied with persistent vomiting, and constipation is the rule. The neck is retracted, and the pain occasioned by forcibly moving the head or extremities may be indicated by the patient, even in coma.

In *alcoholic coma* the onset is gradual and the patient can be partially aroused; the breath has a strong odor of alcohol; the pupils are usually dilated. The urine may be normal. Vomiting and muttering are not uncommon, but convulsions seldom occur.

In *opium-poisoning* the onset is slow; the pupils are extremely contracted; the respiration is slow, sometimes less than ten in the minute, and often irregular. The skin is cold and pale or cyanotic. The heart's action becomes feeble. There is usually little urine in the bladder, but its reactions are normal, except for the presence of morphin. If a liquid

preparation of opium has been drunk, its odor may be detected on the breath.

A form of coma which is probably closely related to uremia is occasionally encountered as a result of prolonged violent muscular exertion. The urine may be albuminous, but the nature of the condition is revealed by the history, as when it develops upon a long bicycle tour. The unconsciousness due to heat prostration can generally be recognized from the history as well as by the high temperature.

Prognosis.—The prognosis is exceedingly unfavorable, especially in advanced arteriosclerosis or chronic interstitial nephritis, and when the uremia occurs in an alcoholic subject. Recovery may take place, however, in apparently extremely unfavorable cases.

The ***treatment*** is considered in connection with the treatment of chronic interstitial nephritis.

ACUTE NEPHRITIS.

ACUTE DIFFUSE, PARENCHYMATOUS, EXUDATIVE, DESQUAMATIVE, CROUPOUS, OR CATARRHAL NEPHRITIS; ACUTE BRIGHT'S DISEASE.

Definition.—An acute inflammation involving to a variable extent both the parenchyma and interstitial structures of the kidneys.

Etiology.—The disease may occur at any period of life, but it is somewhat more frequent in the young than after middle-life. Men are more commonly affected, probably because of greater exposure; among children, both sexes are affected. Alcoholism increases susceptibility, if in no other way, by increasing exposure.

The exciting causes are: (1) *Influences acting upon the skin*, especially cold and wet. Exposure probably also increases the susceptibility of the patient to the action of toxic matter upon the kidneys. Chronic skin diseases, burns, and other injuries may be followed by an acute nephritis.

(2) *Biological Toxic Agents.*—The specific poisons of the acute infectious diseases, especially of scarlet fever, but to a less extent of the other exanthemata, diphtheria, typhoid fever, malaria, yellow fever, and dysentery. Syphilis and tuberculosis often bear a more or less close etiological relation. The disease may develop in connection with purpura and other blood-states, septicemia, rheumatism, or erysipelas.

(3) *Chemical toxic agents*, turpentine, cantharides, carbolic acid, potassium chlorate, possibly lead, arsenic, and phosphorus, may induce the disease.

(4) *Pregnancy.*—This usually develops in primipara toward the close of gestation. Whether it is due to interference with the circulation through pressure, an altered blood-state, or the presence of unrecognized toxic substances, is not known. Acute nephritis may develop after surgical operations on the kidneys, and it is often a complication of chronic nephritis due to increase of the inflammation.

Morbid Anatomy.—The post-mortem appearances are not constant. In a majority of cases the kidneys are normal in size, or slightly en-

larged, and intensely hyperemic. Both organs are equally affected. The capsule strips off normally, but the denuded surface is mottled, the larger hyperemic areas including normal or anemic patches. The cut surface bleeds. In some cases, however, all evidences of hyperemia are absent and the kidneys may be abnormally pale. The medullary portion is generally more deeply congested than the cortical. In some instances the glomeruli are prominent and congested, in others they are pale. On microscopic examination the lesions may be found almost exclusively confined to the tufts and the convoluted tubules. The changes may involve chiefly the capillaries or the epithelium. In the former, there is more or less extensive obstruction by cells and thrombi; in the latter there are cloudy swelling, proliferation, and desquamation of the epithelium. Hyalin and fatty degeneration are seen in the cells of the convoluted tubules, especially in toxic cases. Hyalin degeneration sometimes affects also the contents of the blood-vessels and tubules after obstruction. In severe cases an exudation of serum with a greater or less number of red and white blood-cells is found between the tubules (acute exudative nephritis of Delafield). The changes in other organs are seen chiefly in the serous membranes. All the serous cavities as a rule contain an increased quantity of fluid. A form of acute nephritis usually met with in children has been described by Councilman, in which the cells resemble plasma-cells, and are possibly derived from other sources, as the spleen and bone-marrow, and are brought to the kidneys in the blood.

Symptoms.—The symptoms develop suddenly or gradually. (*a*) A sudden onset is common after exposure to cold or as a result of the rapid accumulation of toxic matter. It is usually marked by a chill with moderate fever, drowsiness, pain in the loins, and prostration. The urine is diminished in quantity, dark, of high specific gravity, and contains much albumin, hyalin, epithelial, and blood-casts, often blood-corpuscles. Edema of the face and extremities is usually developed early, and in severe cases, headache, nausea, vomiting, and delirium or coma set in as uremia approaches. (*b*) A gradual onset is often observed after a febrile disease. The patient becomes anemic, the eyelids puffy, and other manifestations of dropsy ensue. The urine becomes scanty, dark, and concentrated, and albumin and casts are found in it. Headache, nausea, and vomiting may follow, and the bowels are irregular. Muscular weakness is complained of, and dyspnea follows slight exertion. Fever is not generally present, but the skin is dry. The symptoms may subside, or they may at any time give place to a sudden uremic explosion, with more or less rapidly fatal termination. The disease occasionally passes into a chronic form.

Special Symptoms.—The urine may be reduced to four or five ounces in twenty-four hours, the specific gravity increased to 1.025, 1.030, or higher; the color is dark, often turbid, and it may be smoky from admixture of blood. The albumin in it rarely exceeds 1 per cent. The urea, chlorids, and phosphates are diminished. The casts appear early in many cases, and persist after the albumin has ceased to be found. Epithelial cells in a state of fatty degeneration, leucocytes, and granular detritus are usually present, and especially abundant in scarlatinal cases.

Edema appears first in the eyelids, ankles, and hands, and, when the

patient is confined to bed, the dependent portions of the body, as the loins and side of the face, become extremely puffed. A little later the edema becomes more general. The loose skin of the penis and scrotum is often greatly distended. In extreme cases the eyes are nearly closed, and the skin over the extremities pits deeply upon pressure. Finally, the lungs may become edematous, and the brain may also be affected. Edema of the glottis occasionally supervenes. Gastrointestinal symptoms are prominent in some cases, or they may be absent until the system reaches the degree of intoxication which characterizes uremia.

Nervous Symptoms.—Headache, somnolency or insomnia, and delirium are manifestations of intense intoxication bordering on uremia, but they are not uncommon in children, and may completely subside with the infection upon which the disease depends.

The *blood* is hydremic. Its specific gravity may be reduced a half, and the serum albumin 20 per cent. The red corpuscles are diminished, but leucocytosis is not present. The arterial tension is high, and the second sound at the base of the heart is intensified.

Diagnosis.—The disease is readily recognized in well-marked cases, yet the condition may be obscure. The importance of routine examination of the urine is again apparent. The urine should be occasionally investigated in all cases of acute infectious disease, and during the later months of pregnancy. If this were always done, fewer cases of unsuspected convulsions or coma would be encountered.

In *febrile albuminuria*, violent manifestations are seldom met with, and the urinary changes are less pronounced. The albumin may not exceed a trace, and blood is seldom present.

The symptoms of an *acute nephritis* occurring during the course of chronic nephritis can be recognized, as a rule, by the coexistence of cardiac hypertrophy, great arterial tension, possibly retinitis. The anemia is more pronounced. Blood-casts are not found in the urine, but epithelial and fatty casts may be present, the latter more particularly in advanced cases.

The ***prognosis*** depends upon the cause of the condition, and to some extent upon the age of the patient. Scarlatinal nephritis in very young children is often fatal. In older persons, and when due to exposure, recovery is the rule. Extreme dropsy, convulsions, coma, great cardiac weakness, and marked suppression of the urine are bad indications. Recovery is usually well established within a month, if at all.

Treatment.—The success of treatment depends for the most part upon the extent to which the kidneys can be given rest. To this end they should be as far as possible relieved of their function by the adoption of a diet which yields the least waste, the cessation of muscular exercise, confinement to bed, and the substitution of the action of the skin and bowels for that of the kidneys. The best diet is milk, and in addition to it as much water as can be drunk should be taken, both for its diuretic properties, and for the purpose of diluting the toxic matter, which must be carried off through the kidneys. Pure buttermilk, gruels, chicken broth, lemonade, and mineral water may also be taken. An excellent drink is made by the addition of a dram of cream of tartar and the juice of a lemon, with sufficient sugar to a pint of water. A pitcher containing this solution may be kept at the bedside.

The action of the skin is best stimulated by heat. The patient should be kept as warm as possible, and urged to submit to more than an agreeable degree of warmth. A flannel gown should be worn. The room should be kept at an even temperature, and well ventilated. Hot-air or hot-water baths should be administered daily or as often as the strength of the patient will permit. Bathing must be practiced, however, with the utmost precautions against subsequent chilling. The patient must be removed from the bath directly to the bed, wrapped in a blanket and well covered in order to promote sweating for at least an hour. The hot-air bath is often the safest and requires no exertion on the part of the patient. It may be administered by passing hot air from an ordinary lamp through an improvised tube (or rain-spout) with a funnel-like opening into the bed, after having raised the clothing upon hoops arched over the patient. Diuretics should not be given until the acute manifestations have disappeared. The administration of digitalis and sodium-theobromin salicylate (diuretin) acts beneficially in some cases. The more irritating diuretics should not be employed under any circumstances, and many cases recover completely without medication. Pilocarpin is objected to by many writers on account of its depressing effects. Yet in many cases its action is remarkable and entirely free from depression. It should always be used with caution. The first dose to an adult should not exceed gr. 1-12 (0.005) hypodermically. Afterward the quantity may be doubled. A child of ten years should not receive more than gr. 1-24 (0.002) as an initial dose. The bowels should be kept freely active. In case the edema fails to subside after repeated sweating, a robust patient should receive a saline purge every morning or every second morning. Magnesium sulphate in concentrated solution or the compound jalap powder is generally most reliable. The effervescent magnesium-citrate solution is more pleasant for children. When, however, the edema is not urgent, purgation is not necessary. The skin must sometimes be punctured in order to prevent its rupture from overdistention with fluid. It should be done only when it is unavoidable, since under the most careful precaution an opportunity is afforded for infection. In practice, however, such infection is not common. The simple puncture of the skin with a sterilized needle after disinfection is usually sufficient, but a Southey or other tube may be permitted to remain.

Special symptoms sometimes require treatment. When the vomiting is persistent, the free ingestion of liquids must be discontinued. Chipped ice relieves the thirst and often checks the vomiting. The required fluid may be introduced through the rectum. Prolonged irrigation of the large bowel through the long rectal tube is an excellent means also of stimulating the action of the kidneys. Dilute hydrocyanic acid, creosot, or carbolic acid often allays the vomiting. The anemia should be counteracted with iron. The only means of combating the albuminuria is by the relief of the renal engorgement through the measures already suggested, and by restoration of the blood to its normal condition. High arterial tension, particularly when it is associated with muscular twitchings, is often a premonitory indication of uremia. It can sometimes be reduced with a few doses of glonoin. Digitalis should be avoided in this condition.

During convalescence the diet must be gradually added to. Farinacious food should be allowed for a considerable time before nitrogenous articles are added to the fare. The patient should not be allowed to leave the house, until after full recovery, unless the weather is warm.

CHRONIC NEPHRITIS.

The pathological process in all forms of chronic nephritis is a diffuse one, affecting both the parenchyma (*i.e.*, the glomeruli and the epithelium) and the interstitial connective tissue. When the later stages of the disease are taken into consideration, two very different conditions must be recognized. These are designated by Delafield, from the pathological standpoint, (*a*) the chronic diffuse nephritis with exudation, and (*b*) chronic productive diffuse nephritis without exudation. The former is known also as the large or small white kidney, and the latter as the granular, sclerotic, or contracted kidney. Clinically the two forms are designated as chronic parenchymatous nephritis, and chronic interstitial nephritis.

CHRONIC PARENCHYMATOUS NEPHRITIS.

CHRONIC DIFFUSE OR DESQUAMATIVE NEPHRITIS, CHRONIC TUBAL NEPHRITIS, CHRONIC BRIGHT'S DISEASE.

Etiology.—The frequency of the disease is greatest in young adult males. In children it usually develops from the acute nephritis of scarlatinal origin. The disease may follow the acute form induced by cold, pregnancy, or other influences. Among infections, malaria and septicemia are important. In many cases the excessive drinking of beer and stronger alcoholic beverages induces the disease; syphilis and tuberculosis sometimes appear in its etiology. It may follow chronic suppuration, but amyloid degeneration is more common in this relation. In a great many cases, if not in a majority, the disease begins insidiously without definite recognizable cause.

Morbid Anatomy.—Between the large white kidney and the small white kidney, which may be taken as the two principal types, there are many varieties. The former is more common. In it the organ is much enlarged, the capsule is thin and nonadherent. The surface is pale, except for the distention of the stellate veins. The cortex is much thickened and pale, with occasional opaque areas. Histologically the changes consist for the most part in fatty degeneration of the epithelium and glomeruli. The glomeruli are large and the capillaries show hyalin degeneration, the epithelium cloudy swelling or hyalin degeneration. The convoluted tubules are filled with casts largely made up of desquamated fatty epithelium. The interstitial tissue is not apparently altered. In the extreme large white kidney there is a great excess of fat above that which is usually seen in kidneys that are of moderate size, and the condition has been regarded as a fatty degeneration of the kidney.

As has been already stated, the disease is always diffuse, and the small white kidney represents the extreme degree of interstitial proliferation that is seen in a strictly parenchymatous form of the disease. In size it is usually about normal or slightly larger, rarely smaller. The

connective tissue is increased, the capsule thickened and moderately adherent. The capsules of the glomeruli and the intertubular tissue are increased. The smaller size of the kidney is due to the contraction of this new tissue. It is often a later stage of the condition represented by the large white kidney, but it may develop independently. The changes in the parenchyma of the two forms are the same.

Sometimes the cut surface appears mottled, owing to different stages of degeneration in different parts. A hemorrhagic nephritis is also recognized, in which small hemorrhages have occurred into the tubules of the cortex and between them. The changes in other organs are of minor importance. Cardiac hypertrophy is occasionally present, but it is more commonly associated with the interstitial nephritis.

Symptoms.—The disease generally begins so insidiously that it is for a time unrecognized. When it follows the acute form, the patient may appear to have improved, but the anemia persists and the urine is still dark, deficient in quantity, and albuminous. When it develops independently, the anemia is often the earliest symptom, and this may be disregarded until puffiness of the eyelids or of the ankles or hands attracts attention to it. Even when the blood-count does not show marked anemia, the pallor is often extreme and the face assumes a peculiar yellow hue in many cases. Before the edema has become persistent the patient usually suffers from dyspnea. As the disease progresses, the dropsy increases. It is always more marked in the morning; the eyelids, extremities, and dependent portions of the body are most affected. Later, a tendency to the involvement of the serous membranes becomes manifest, and ascites, hydrothorax, or hydropericardium may develop. Acute exacerbations sometimes occur in which all the symptoms of acute nephritis are present. The heart becomes hypertrophied chiefly in cases in which the small white kidney is found—cases approaching nearest to the interstitial form of nephritis. In these, the arterial tension is high.

Gastrointestinal symptoms are frequent and often persistent. Vomiting often becomes uncontrollable during acute paroxysms of the disease, and a troublesome diarrhea is common at such times. The analysis of the urine is the key to the diagnosis. The quantity is reduced, the color is high, the specific gravity is usually above 1.025, and the albumin often exceeds 1 per cent by weight, the coagulum after boiling representing a third to a half of the volume in the test-tube. After centrifugal precipitation, the microscope reveals numerous large and small hyalin, epithelial, granular, and fatty casts, epithelium from the tubules in a state of fatty degeneration, many leucocytes, and often a few red corpuscles. To a certain extent these features vary with the condition of the kidneys. As the interstitial tissue becomes more involved and the size of the kidneys is reduced, the quantity of urine is somewhat increased, the specific gravity lower, and the percentage of albumin reduced; the dropsy may also subside to some extent. It is not always possible, however, to diagnosticate the condition of the kidney by these signs. The other symptoms are the same in character as those of acute nephritis. Drowsiness and headache are often prominent features; delirium, restlessness, and convulsive manifestations, tremors and muscular cramps are more commonly met with in the interstitial form.

Prognosis.—The prognosis is always unfavorable. The possibility of recovery rapidly vanishes. It is only rarely in children, as a rule, that the disease subsides after a year's duration. Death may be the result of uremia, edema of the lungs or inflammation of the serous membranes.

Treatment.—The treatment of special symptoms is based upon that of the same conditions in acute nephritis. The nearer a milk diet is maintained, the less will be the work thrown upon the kidneys. Nitrogenous food should be eaten sparingly, a small portion of meat being allowed at only one meal each day. Water should be drunk freely and the saline diuretics, especially potassium bitartrate and acetate, may be employed. Digitalis is the best diuretic in many cases, and the sodium-theobromin salicylate administered with it adds greatly to its action. Iron is indicated for the anemia; its administration should be regulated by the condition of the blood rather than by the appearance of the patient. When properly administered, a marked improvement is often observed, yet the effect is rarely permanent.

The patient must lead a quiet, temperate life, free from excitement or excesses of any kind. If his circumstances permit, he should reside in a moderate, uniform climate, like that of the extreme southern district of California.

CHRONIC INTERSTITIAL NEPHRITIS.

SCLEROSIS OF THE KIDNEYS, GRANULAR KIDNEY, CONTRACTED KIDNEY, GOUTY KIDNEY, RENAL CIRRHOSIS.

Etiology.—(*a*) In some instances the sclerotic kidney is a late result of chronic diffuse nephritis characterized in its early history by prominent symptoms on the part of the parenchyma; the parenchymatous nephritis passes over into the interstitial form. (*b*) In another group of cases it is a primary affection, and in a third (*c*) it is a manifestation of an arteriosclerosis.

a. In the first group it is a further development of the sclerotic process seen in the small white kidney, a further hyperplasia of the connective tissue and consequent induration.

b. The primary form cannot always be accounted for. It is more frequently observed in men past middle life, and more commonly in persons of a gouty tendency, and those who have been indulgent of their appetites. It is induced probably more commonly as a result of over-eating than as a result of excessive drink. The excessive eating of meat is regarded as especially influential. Lack of exercise and sluggishness of the bowels, with consequent increase of effete matter to be eliminated by the kidneys, is an exceedingly important element in the production of a large proportion of cases. Activity of the mind as well as activity of the body has been regarded as operative in some cases, but like alcoholism these influences are not usually alone in their action. Syphilis appears in the history of many cases. Some authors regard the increased work thrown upon the liver by over-eating and- drinking as the more remote cause, through the production of substances which produce irritation of the kidneys in passing through them. Gout, chronic lead-poisoning, and chronic rheumatism are looked upon as causes in some cases.

c. In the arteriosclerotic form the exciting cause does not differ materially from that in the independent form, but the result is often much less pronounced in the kidneys, a thickening and hardening of the smaller arteries of the entire body often taking place.

Morbid Anatomy.—Sclerotic kidneys are small, each weighing less than an ounce in extreme cases. The surface is rough and granular, the color dark red, rarely pale, the capsule is thickened and adherent so that it cannot be stripped off without laceration of the kidney substance. Many cysts of various sizes are often found immediately under the capsule. The tissue is firm, and the knife meets with much resistance in making a section. The cortex is more markedly atrophic than the pyramidal portion. The essential histological change is hyperplasia of the connective tissue. This affects to a variable degree all the parts of the kidney, but is more pronounced in the cortical portion. In the pyramids the new connective tissue is more uniformly distributed, though less abundant. Bowman's capsule is often supplemented by a thick layer of dense connective tissue, and a similar thickening of the adventitia of the blood-vessels is often seen. Degenerative changes also occur in the parenchyma; by some writers this is regarded as the primary change, by others as secondary to the sclerosis. Hyalin degeneration and cloudy swelling are more or less general in the tufts and in the capillary walls, as well as between the loops. As a result of these changes, and to a great extent probably as a result of pressure, the glomeruli are often extremely small, and many may be entirely destroyed. An occasional tuft in a comparatively normal condition is seen, however. The epithelium of the renal tubules shows more or less general cloudiness or a well-marked fatty or hyalin degeneration. Many of the tubules are filled with masses of granular detritus often in the form of epithelial and other casts. Dilatation amounting to the formation of cysts is not uncommon. Pigmentation of the interstitial tissue is sometimes met with as a result of hemorrhages.

The heart generally shows hypertrophy, especially in the left ventricle. This may be secondary to the renal condition alone or to the general arteriosclerosis.

Symptoms.—Not only the invasion, but the entire course of the disease may be so obscure as to escape recognition until near its close. In a majority of cases it is rather a complication than the disease itself that first attracts attention, and many cases are first recognized at the autopsy. The patient experiences no illness which he regards of sufficient importance to require the services of a physician. In a considerable group of cases, however, he becomes anemic, is sleepless and restless. Headache and dyspnea are complained of, the digestion becomes impaired, the tongue coated, and the bowels irregular. The quantity of urine voided is greatly increased, and the patient must generally arise at night to urinate. The color of the urine is light and the specific gravity is low, often below 1.010, or even 1.005. A trace of albumin is present, constantly or at variable intervals. The solid constituents are greatly reduced in ratio to the quantity, but the total quantity eliminated in each twenty-four hours is often about normal until comparatively late in the disease. Hyalin casts can generally be found, and, as the disease advances, granular and fatty casts become more

abundant. Toward the termination of most cases, when, apparently, the remaining unaffected glomeruli become involved, the albumin increases in quantity, and the specific gravity becomes higher. In the kidney of the arteriosclerosis patient, however, the quantity, weight, and color of the urine may remain nearly normal throughout, the quantity of albumin is generally greater, and casts may be found more numerous.

The heart is hypertrophied, the arterial tension is high, the impulse is strong, and the aortic second sound is accentuated, often with a metallic ring. The pulse is small and hard. The skin is generally dry; perspiration can seldom be detected; edema rarely develops, except as a complication due to cardiac loss of compensation. Eczema often appears, and pruritus is common. Muscular cramps or twitchings are often observed. As the disease advances, various disturbances of the nervous system are of frequent occurrence, as disorders of sight and hearing, and retinal hemorrhages. Diffuse retinitis or diplopia is sometimes the first manifestation of the disease. The headache often assumes the form of migraine. Delirium may develop, and convulsions sometimes supervene shortly before death; meningeal or cerebral hemorrhage occurs in some cases. The cases which merge into a parenchymatous type toward the close are generally marked by stupor and coma. Edema of the glottis may develop suddenly in advanced cases. Many cases terminate in uremia, others with chronic bronchitis, pneumonia, or edema of the lungs.

Diagnosis.—The disease should always be suspected when an individual above forty presents a history of headache and insomnia, with increased excretion of urine, and when upon examination the pulse is found to be small and hard, the heart hypertrophied, and the second sound accentuated, the urine of low specific gravity and color, with a trace of albumin and a few casts. Polyuria alone in one past middle life is a suspicious feature, and should always lead to repeated examinations of the urine for albumin and casts. The urine should be let stand for an hour or two in a conical glass, then poured out, and only the last 15 c.c., which contain the sediment, placed in the tube of the centrifuge. It is well to examine specimens voided two hours after a meal as well as those of the morning and evening.

Prognosis.—The prospect of prolonged life is better than in the chronic parenchymatous nephritis, but there is always great danger of a uremic seizure in a pronounced case. Severe, persistent headache or vomiting, diarrhea, uremia, cardiac dilatation or loss of compensation, the development of serous effusions, and the appearance of the urinary changes indicative of more complete parenchymatous involvement are unfavorable. The disease is incurable, and the possibility of spontaneous recovery is inconceivable.

Treatment.—The treatment is purely palliative. As long as the kidneys are not unusually taxed by the elimination of toxic matter, there is no indication for the administration of diuretics, diaphoretics, or laxatives; but a large quantity of water should be consumed in order to compensate for the increased elimination, the skin should be kept active by frequent warm baths followed by friction with a coarse towel, and the bowels should be kept normally active. The patient should wear flannels and dress warmly. If possible, he should reside in a warm

equable climate, like that of southern California, during the entire year, or at least during the winter. Moderate exercise is beneficial, but fatigue of body and mind, all worry, and excesses of every kind, especially in eating and drinking, must be avoided. Alcohol and tobacco should be abandoned. Drugs are of little value, except for the relief of symptoms as they arise or to maintain the permeability of the kidneys when there are indications of obstruction. The last indication is met by the administration of pure water or one of the alkaline mineral waters.

A moderate increase of arterial tension is necessary to maintain the circulation through the inelastic arteries, and it is only when it becomes excessive that drugs are required for its reduction. For a time a proper balance can be maintained by the administration of saline laxatives or an occasional purgative dose of calomel, hot baths, sweating, and restriction of diet. When, however, the tension becomes persistently too high, and especially if there be evidence of venous engorgement, glonoin must be administered, beginning with gtt. j t. i. d., and gradually increasing as it is found necessary. The maximum dose is very different in different cases, and from 10 to 15 drops must often be given. The quantity should not be increased if flushing of the face is produced. The remedy should be discontinued for a few days after periods of two or three weeks. Iron is indicated in most advanced cases, for the anemia. The tincture of the chlorid is often the best preparation, and a temporary improvement usually follows its administration. When the heart begins to fail, and especially when dilatation supervenes, digitalis must be given in moderate doses. Strychnin can often be used, to maintain the heart's action, with as much advantage as digitalis.

Treatment of Uremia.—The first indication is the elimination of the poisons from the blood. A saline purge should, therefore, be immediately administered, and, while its effect is waited for, the patient should be given a hot bath in order to produce profuse sweating. Unless the heart be too feeble, the sweating may be increased by the administration of pilocarpin, and the effect prolonged by covering heavily with blankets. When there is great restlessness, delirium, or urgent dyspnea, morphin should be carefully given. The arterial tension must sometimes be reduced with glonoin. Irrigation of the rectum with water at a temperature of 120° to 150° F., after the method of Grandin, has yielded good results. If these methods fail and the patient be robust, great benefit may sometimes be obtained from the abstraction of 12 to 18 ounces of blood from the arm.

AMYLOID KIDNEY.

WAXY OR LARDACEOUS DEGENERATION OF THE KIDNEYS.

Amyloid degeneration of the kidneys develops as an independent affection, or in connection with one of the forms of chronic diffuse nephritis.

Etiology.—The condition usually develops simultaneously in the kidneys, liver, spleen, and other organs of the body. It is caused in most cases by prolonged suppuration, more particularly by that accompanying tuberculosis or syphilis. Suppuration affecting bone or the pleura is particularly liable to induce it. Sometimes it develops in cachectic

states without known suppuration. It has been referred to malaria, gout, lead-poisoning, leukemia, and chronic endocarditis in some cases.

Morbid Anatomy.—The kidneys are generally large, firm, and pale. The cortex shows the greatest thickening, and on section has a glistening (lardaceous) appearance. The glomeruli are first affected and become more than normally distinct. The pyramids are usually deep red in color. The amyloid matter is beautifully demonstrated by penciling the cut surface with a dilute solution of tincture of iodin, and immediately washing away the excess with water. This gives it a mahogany color. Late in the disease, the infiltration usually affects the tubules as well as the glomeruli. On microscopic examination the changes of parenchymatous or interstitial nephritis are found in all advanced cases.

Symptoms.—There are seldom any manifestations by which the amyloid disease of the kidneys can be recognized independently of the associated changes in other organs. The urine is increased in quantity, of low specific gravity, and colorless. Albumin is usually found in large quantity, sometimes, however, only as a trace, or it may be absent. Globulin is generally present in considerable quantity, and the urates are deficient. Hyalin and fatty or granular casts are usually found. The casts occasionally show the distinctive reaction to iodin and the anilin dyes. The patient becomes extremely anemic and develops a peculiar cachexia, and edema often develops toward the close. Such symptom as increased arterial tension or uremia may develop as a result of the associated interstitial or parenchymatous nephritis. A colliquative diarrhea, due to the accompanying amyloid infiltration of the intestine, frequently hastens the fatal issue.

Diagnosis.—The amyloid kidney is diagnosticated, as a rule, more from the history and the recognition of the associated affections than by its own symptoms. An increased quantity of pale albuminous urine of low specific gravity, accompanied with enlargement of the liver and spleen in a tuberculous or syphilitic patient, especially after suppuration, is an almost invariable indication of amyloid disease of the kidneys.

Prognosis.—This is always unfavorable. The disease runs a very variable course, however, sometimes continuing several years, sometimes terminating fatally within a few months. Much depends upon the extent to which other organs, especially the intestines, are involved.

Treatment.—Syphilitic cases sometimes run a slower course under treatment, and the removal of suppurative processes, by improving the general condition of the patient, enables him to longer combat the disease; but there is no means at our command by which it can be arrested.

PYELITIS.

PYELONEPHRITIS, PYONEPHROSIS, SURGICAL KIDNEY.

Definition.—An inflammation of the pelvis of the kidney, usually suppurative in character, rarely catarrhal.

Etiology.—The catarrhal form is generally attributed to the toxins of the infectious diseases, chemical irritants like turpentine and balsams, or the passage of renal calculi. The suppurative form is caused

by the direct action of the pyogenic bacteria, which, as a rule, reach the kidney from the lower urinary passages, as when gonorrhea, stricture, prostatic abscess, or other suppurative affection of the urethra, bladder, or contiguous part is present. This form is usually designated pyelonephritis. When the suppuration originates in the kidney as a suppurative interstitial nephritis, the infectious agent has reached the kidney through the blood or lymph circulation. This form is commonly associated with tuberculosis, pyemia from infected wounds, malignant endocarditis, or other focus of suppuration. It is sometimes due to the twisting of the ureter of a displaced kidney or the growth of a neoplasm, hydatids, or the ova of parasites, sometimes to the lodgment of a calculus, although calculus-formation is probably more frequently a result of the suppuration.

Morbid Anatomy.—When the suppurative process has continued long, the pelvis of the kidney is much enlarged, thickened, and indurated. The ureter is similarly altered for a variable distance in most cases. Pus is generally present. The inflammation extends also to the kidney, the calyces become large, and in extreme cases the kidney structure is destroyed, leaving a large pus cavity (pyonephrosis). In tubercular cases, particularly, the pus often forms a large case ousmass, impregnated with lime-salts.

Symptoms.—The catarrhal form may be recognizable only by such symptoms as pain and tenderness over the kidneys, with an increased flow of pale, turbid urine, of low specific gravity. Vesical irritation or cystitis may be produced, and fever commonly develops, especially in children, if it be not previously present.

In the suppurative form, chills, high fever, and sweating, with pain and tenderness, are often early symptoms. In some cases, however, these manifestations do not appear until later. When the disease becomes pronounced, a distinct tumor-like prominence often develops over the affected kidney, and the tenderness becomes extreme. The pain often radiates toward the umbilicus or downward toward the testicles. The urine becomes laden with pus, albumin, and desquamated, degenerated epithelium and blood-cells. The pus is subject to marked variation, occasionally appearing in enormous quantity as a result of the rupture of an inclosed pocket or removal of an obstruction in the ureter. When the renal tubules become involved, casts may be discovered, if not destroyed by the bacteria always present; the quantity of albumin is also increased. Fragments of disintegrated kidney-tissue are sometimes found. The quantity of urine voided is much reduced, but the specific gravity remains low, often below 1.010. It may be acid or alkaline, the reaction depending upon the character of the infecting micro-organisms.

The general health of the patient becomes greatly impaired. He is anemic, and ultimately becomes cachectic. There are exceptions to this rule, however, cases in which the suppuration continues more or less constantly for years, especially in tuberculous individuals, without much impairment of health. Some cases develop sepsis, and the patient passes into a typhoid state, which may terminate fatally. A perinephric abscess may be produced by the rupture of an abscess in the kidney.

Diagnosis.—It is generally possible to determine which kidney is af-

fected, by the location of the pain and tenderness. Catheterization of the ureter, when it can be done, affords a more positive means in doubtful cases, and cryoscopy, determining the freezing-point of the urine from each kidney separately, offers the most accurate means of determining the organ affected and the extent to which its function is destroyed.

The differentiation of the tubercular from the nontubercular pyelitis depends upon the discovery of the tubercle bacillus in the pus. Repeated, careful examination is usually necessary, often supplemented with intraperitoneal inoculation of animals. The presence of calculi can often be recognized with the fluoroscope.

Perinephric abscess is excluded by the history of the case, the presence of a definite tumor, and the absence of edematous swelling; albuminuria is more constant in pyelitis.

Cystitis is attended with more pain in the bladder and frequent micturition, the urine is less albuminous, though often of higher specific gravity, and there is no tumor, pain, or tenderness in the lumbar region. The diagnosis cannot be made from the character of the epithelium, since the transitional type is found both in the bladder and renal pelvis. When the urine can be obtained directly from the ureters, the presence of pus in it definitely establishes the suppuration at a higher point. Acid pus almost positively indicates pyelitis.

Prognosis.—Catarrhal cases usually recover; suppurative cases run a prolonged and finally unfavorable course.

Treatment.—Life can often be greatly prolonged by surgical measures. When operation is not required, the condition of the patient may be greatly improved by the administration of alkalis, especially sodium benzoate or salicylate and the alkaline mineral waters in large quantity. The oil of sandalwood in capsules containing ♏x (0.60); methylene blue, gr. iij to v (0.18—0.30); and urotropin, gr. v to x (0.30—0.60), three times daily, are all beneficial. Urotropin often controls the suppuration for a long time, but permanent arrest of it cannot be secured. The patient should avoid exposure to cold and wet, he should be warmly clad, and he should restrict his diet to food which taxes the kidneys least.

HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calices of one or both kidneys, with atrophy of the parenchyma, due to obstruction and the retention of the urinary secretion.

Etiology.—The condition is sometimes congenital and due to obstruction of the ureter or urethra, as when an abnormally long ureter becomes twisted or contains a valve. It is sometimes caused by the pressure of a tumor, or by disease of the prostate or urethra, and it may result from the displacement of a floating kidney, with tortion of the ureter.

Morbid Anatomy.—Various degrees of dilatation and atrophy are met with, from an enlargement of the pelvis, with little change in the kidney proper, to the conversion of the entire organ into a smooth-walled cavity free from suppuration and bounded by a thin layer of the cortex, which,

upon microscopic examination, may be found almost normal in structure. The condition is usually unilateral, except when the obstruction is situated in the urethra or in a single ureter, a condition observed in a few instances. The most extreme dilatation occurs in cases of prolonged partial obstruction, and the kidney sometimes resembles an enormous cyst.

Symptoms.—Bilateral, congenital cases usually terminate fatally within a week. Unilateral cases sometimes do not produce symptoms, unless the obstruction be suddenly developed, until the tumor has attained a comparatively large size. Fluctuation can often be obtained over a very large tumor, yet many cases pass unrecognized, especially those arising from the pressure of a tumor. Intermittent cases have been observed in which the tumor periodically disappears, with the discharge of a large quantity of fluid. This is especially the case in floating kidney and may continue for many years.

Diagnosis.—In an infant the condition is differentiated with difficulty from a congenital sarcoma. Aspiration and the withdrawal of a large quantity of clear or cloudy fluid of low specific gravity, containing albumin and the salts of urine, indicate hydronephrosis; the withdrawal of blood indicates sarcoma.

Ovarian tumor can be differentiated by its greater mobility, its more superficial situation, and the signs obtained through vaginal examination and aspiration.

Pyonephrosis is differentiated by the purulent character of the urine, greater pain and tenderness, and the more or less constant elevation of temperature, with other septic symptoms in many cases.

Prognosis.—Hydronephrosis may exist in one kidney for many years without serious impairment of health. The greatest danger to be anticipated is rupture of the cyst into the peritoneal cavity or the development of suppuration.

Treatment.—Little can be done, unless through surgical measures. If the obstruction can be removed, further destruction of the kidney is prevented, but an operation is seldom possible until the destruction is far advanced.

NEPHROLITHIASIS.

STONE IN THE KIDNEY, RENAL CALCULUS, GRAVEL.

Definition.—The formation of concretions within the kidney. The concretions vary in size from microscopic particles to stones the size of a bean and include agglutinated masses capable of filling a greater part of the renal pelvis (coral calculi). (See, also, Renal Infarcts, page 18.) The principal varieties of stone are the uric-acid, calcium-oxalate, and the phosphatic. The first of these is recognized by its smooth surface, its hardness, and red-brown color. The oxalate stone is hard and white. Both these are occasionally incrusted with urates or phosphates, the result being a comparatively soft calculus with an exceedingly hard nucleus. The calcium-phosphate stones are grayish, soft stones composed of calcium phosphate and the triple ammonium-magnesium phosphate. In addition to these forms, concretions are rarely met with which

are composed of calcium carbonate, cystin, xanthin, fibrin, fatty or saponaceous matter (urostealith), or indigo.

Etiology.—Calculi develop at all ages, even in the intrauterine life. A family tendency is often traceable, especially in gouty subjects. Men are more frequently affected than women. Improper food, as well as overindulgence in eating and drink, is regarded as influential. In the aged the calculus-formation is usually referred to an excess of uric acid. The exciting cause is not definitely understood. A nucleus is no doubt essential. In some cases this consists of bacteria, the ova of parasites, a blood-clot, or tube-casts. A highly acid urine, due to excess of uric acid and a low percentage of salts, is believed to favor their development.

Symptoms.—A large coral calculus may exist in the kidney for years without producing disturbances, and the most intense suffering is often induced by the passage of a stone the szie of a pea. The pain is induced by the passage of the calculus through the ureter, and it becomes most severe when the stone becomes lodged. These attacks of so-called renal colic often occur periodically for many years. The stone remains indefinitely in the renal pelvis until dislodged. This often follows a blow, a jolt or jar. In some cases the passage of a single stone is the only manifestation of the disease that is ever experienced. The renal colic is usually set up immediately upon the entrance of the calculus into the ureter. The pain is sharp and lancinating; beginning in the region of the kidney it radiates downward along the ureter to the testicle and the inner side of the thigh. The testicle is retracted. Strangury is developed and nausea and vomiting usually follow, sometimes accompanied with chills, fever, cold sweating, and great prostration. The pain may radiate to the back or chest. The attack lasts from an hour to a day, and ceases spontaneously when the stone finally drops into the bladder. There is then a copious flow of urine containing albumin and casts and sometimes tinged with blood. A feeling of soreness remains for a day or two. The calculus frequently passes on through the urethra, occasioning more or less pain, or it may remain in the bladder and become the nucleus of a larger vesical calculus.

When the stone remains in the kidney, becoming too large to enter the ureter, and when there are many calculi in the renal pelvis, more or less characteristic symptoms are produced. A dull aching pain and weight are felt, or periodic attacks of more intense suffering occur. The pain is not always confined to the affected side, but may be reflected to the other kidney or even be confined to it. Hematuria is not common, but it may occur in these cases. Pyuria, with or without other symptoms of pyelitis, may accompany the condition. Reflex manifestations occasionally develop, as gastric disturbances, and headache approaching the character of migraine. A renal intermittent fever, similar to the intermittent fever of gall-stones, has been described in cases associated with pyuria.

Diagnosis.—The pain sometimes resembles that of gall-stones, but in that condition there is generally slight jaundice, clay-colored stools, inlargement of the gall-bladder, bile in the urine; the pain radiates to the umbilicus or shoulder, and there is no retraction of the testicle.

Intestinal colic is differentiated by the abdominal distention, diarrhea,

borborygmi, and the absence of urinary changes or tenderness in the region of the kidney.

A *vesical calculus* may occasion pain in the kidneys, which is generally bilateral; tenesmus is present, the urine is alkaline and contains much mucus. The X-ray sometimes affords the most positive means of differentiation.

Prognosis.—The prognosis is always grave and at any time corresponds to the extent to which the kidney is damaged.

Treatment.—The treatment of a large or impacted stone is purely surgical. There is no other means of removing a stone once formed. The further growth of a uric-acid stone may possibly be retarded by keeping the urine alkaline through the administration of alkalis, and that of the phosphatic calculus by maintaining its acidity with benzoic acid, gr. v (0.3) in capsules three times a day. Piperazine has not proved satisfactory, although it is capable of dissolving calculi outside of the body. It may be tried in the daily quantity of gr. xv (1.0) dissolved in a quart of water. A large quantity of water should always be drunk by these patients, and pure water is better than that laden with mineral salts to be eliminated through the kidneys, unless they are required for the regulation of the reaction of the urine. The diet should be regulated with a view to maintaining the desired reaction.

Renal colic requires the administration of morphin hypodermically, and in some cases the inhalation of a little chloroform must be given until the morphin takes effect. The complete relaxation of narcosis favors the passage of the stone. Hot fomentations or poultices to the lumbar region assist in the relief of pain, and a full hot bath is more effective. Hot lemonade or other hot drinks should be given.

PERINEPHRIC ABSCESS.

PERIRENAL ABSCESS.

Definition.—Suppurative inflammation of the connective tissue about the kidney.

Etiology.—The disease is usually secondary to suppurative inflammation in or near the kidney or to the rupture of an abscess in the kidney or appendix, caries of the vertebræ, or empyema. It sometimes results from trauma, falls, blows, or wounds, and it may follow the acute infectious diseases of childhood.

Morbid Anatomy.—The formation of a distinct, localized abscess is exceptional. As a rule, the pus surrounds the kidney and burrows along the psoas muscle toward the groin or upward to the lung, sometimes penetrating one of the abdominal viscera. The tissues about the kidney are found in a greatly altered and indurated condition.

Symptoms.—There are usually great pain and tenderness over the affected kidney, with swelling and edema of the overlying tissues. The pain is often referred to the hip, or it may radiate downward and the testicle may be retracted. The patient lies with the affected thigh drawn up, and in walking leans toward the opposite side, bending forward. Chills, fever, and other indications of sepsis generally develop unless the pus is early evacuated.

Diagnosis.—The distinctive points are the diffused swelling and the edema in the lumbar region, the absence of pyuria in most cases, and the withdrawal of pus by aspiration.

Treatment.—The treatment consists in opening the abscess early and maintaining thorough drainage.

CYSTIC KIDNEY.

Several varieties of cysts occur in the kidney. (*a*) Congenital cysts are usually numerous in both kidneys, varying in size up to an inch in diameter and enlarging the kidneys to such an extent that they may together weigh five or six pounds. The cysts contain a clear or cloudy fluid, sometimes dark in color, in which albumin and triple phosphates, blood-crystals, cholesterin, and fat-crystals may be found. Their cause is not known. Death usually occurs before or soon after birth, but an apparently congenital cystic condition has been found in aged persons. (*b*) Dermoid cysts have been found in the kidney. (*c*) A general cystic condition affecting the kidneys, liver, and spleen has been observed. (*d*) The retention cysts resulting from the dilatation of obstructed tubules have been referred to under Chronic Interstitial Nephritis.

Symptoms.—Small cysts are usually associated with the symptoms of chronic interstitial nephritis, sometimes with the addition of hematuria. Large cysts are generally recognizable both by their size and by manifestations on the part of the kidneys. The largest may occupy the greater part of the abdominal cavity without occasioning great disturbance. Pressure symptoms are not infrequently excited. One or more firm, smooth tumors may be felt in the region of the kidney or lower and nearer the abdominal wall. Fluctuation can sometimes be elicited. Some cases pass unrecognized, however, until uremia is suddenly developed, and some are discovered only after death.

Treatment.—Nothing can, as a rule, be accomplished by treatment. Removal of the affected kidney is rarely justifiable.

TUMORS OF THE KIDNEY.

Both benign and malignant growths are met with in the kidney. The former are uncommon and seldom occasion much disturbance. They include fibromata, adenomata, lymphadenomata, lipomata, and angiomata. Papillomata are sometimes found in the renal pelvis. Of the malignant growths, sarcoma is more frequent than carcinoma. Either may be primary or secondary. Rhabdomyomata, alveolar sarcomata containing striped muscle-fibers, are occasionally met with. The encephaloid cancer is the most common form.

Symptoms.—A gradually enlarging tumor is recognized in the region of the kidney, becoming more prominent anteriorly as it enlarges and usually pushing the colon before it. The growth is rapid, the tumor hard. Pressure symptoms may supervene and the adjacent organs may be invaded by the growth. Pain is variable and may be absent, or sharp and radiating. Hematuria is frequently developed. The patient becomes emaciated in most cases, and the usual cachexia develops.

Diagnosis.—The malignant tumors are recognized by their rapid growth, the greater pain, and more frequent hematuria. Fragments of the tumor may rarely be found in the urine. Sarcoma often develops in early childhood, carcinoma seldom before the thirtieth year.

Enlargement of the retroperitoneal lymph-glands in children cannot always be differentiated, but the urinary changes are usually absent, unless the ureter be compressed. An enlarged spleen is distinguished by its distinct, notched margin, descending with inspiration; the colon generally lies behind it. Tumors of the liver are, as a rule, higher, causing protrusion of the lower ribs; the lower margin of the liver can generally be recognized above the renal tumor.

Tubercular and syphilitic growths are recognized by their history and the presence of the disease in other parts.

Treatment.—Benign growths seldom require treatment; malignant tumors can sometimes be successfully removed.

SECTION VIII.

Constitutional Diseases.

ARTHRITIS DEFORMANS.

OSTEOARTHRITIS, CHRONIC RHEUMATIC ARTHRITIS, RHEUMATOID ARTHRITIS.

Definition.—A chronic, progressive disease of the joints, affecting chiefly the articular cartilages, bones, and synovial membranes, and producing loss of function and great deformity.

Etiology.—The disease may occur at any time of life, but its frequency increases from 35 to 55, and rapidly declines after that period. It is rare in children.

Sex.—It is much more frequent in women than in men, commencing in most cases during or after the menopause and somewhat oftener in those who have been sterile. Earlier in life it sometimes follows rapid childbearing or uterine disease.

Heredity plays a doubtful part, but there is often an arthritic diathesis in the family, a predisposition to rheumatism, gout, or arthritis deformans. The daughters of gouty fathers are supposed to be more susceptible. The disease is not, however, related to either rheumatismor gout.

Hygienic Influences.—Exposure to cold and wet is less active than in the production of rheumatism, but it may aggravate the condition of the patient. Bad hygienic surroundings, insufficient or improper food, and residence in damp quarters are important factors in many cases. Mental and nervous depression, worry and care, anemia, malnutrition, and the excessive use of amylaceous or saccharine food are recognized as exciting causes and as capable of producing exacerbations. A dissolute life, sexual exhaustion, and veneral disease, especially gonorrhea, are mentioned as causes. A tuberculous taint has been repeatedly observed, and influenza and other acute infections prepare the patient for the disease. Finally, injury is sometimes thought to contribute to its production. There are two principal theories with regard to the immediate cause of the disease.

1. *Nervous Origin.*—The disease is thought to be of nervous origin. This theory is supported: (*a*) By the symmetrical distribution of the joint lesions; (*b*) by the similarity of these lesions to those occurring in locomotor ataxia, syringomyelia, and other affections of the spinal cord; (*c*) by the frequent occurrence in the course of the disease of nutritive changes (dystrophies) of the skin, nails, muscles, and bones; and (*d*) by the evident importance of shock, worry, grief, and mental exhaustion in the etiology of some cases. The exact nature of the nerve-changes has not been fully determined. Ord attributed the disease to lesions in the trophic centers of the cord and to peripheral

irritation. They have been attributed also to the absorption of toxic substances from without and to autointoxication of a form that is especially associated with dilatation of the stomach (Bouchard).

2. *Infectious Origin.*—This theory is supported: (*a*) By the fact that micro-organisms have been found in the tissues and fluid of the joints. (*b*) The disease sometimes begins with an acute onset. (*c*) It frequently follows an acute infection; and (*d*) enlargement of the spleen and lymph-glands has been noted in some cases.

Morbid Anatomy.—The lesions are usually symmetrical and involve primarily the articular cartilages, synovial membranes, and the bones. Later, changes occur in the capsular and other ligaments, in the periosteum and muscles, and to complete the picture we must include the changes in the nervous system, especially neuritis and atrophy. In the articular cartilages the change begins in the center, the part farthest removed from the blood circulation. The cartilage becomes fibrillated and softened and is removed by friction and absorption, exposing the underlying bone. Around this a process of new-formation takes place, as in caries, and nodular masses are formed which ossify and constitute the chief element in the production of deformity and the limitation of motion. A bony ring is sometimes formed. As a result of either process the end of the bone appears to be enlarged. As a result of friction the surface of the bone becomes hardened (osteosclerosis) and acquires an ivory-like polish (eburnation). A rarefying ostitis occurs at the same time in the spongy portion, and the articular face often becomes grooved and deformed. True bony ankylosis occurs only in the spinal column.

The synovial membranes become highly vascular, thickened, and their fringes elongated. Cartilaginous nodules are sometimes formed in them which become detached and lie loose in great numbers in the joint cavity. The synovial fluid is at first increased, but later the joint becomes "dry." The bursæ in the vicinity of the joints are often distended with fluid, forming cysts. The muscles are atrophied and have a brownish color. The ligaments, periosteum, and tendons often undergo thickening.

Symptoms.—There are three principal types of the disease, known as the multiple progressive and monarticular forms and Heberden's nodosities. Clinically, these forms have few features in common.

1. **The Multiple Progressive Type.**—This may be subdivided into an acute and a chronic form. The *acute form* is rare after the age of 40. The joints are generally enlarged from the beginning, but in the most acute cases the pain is out of proportion to the swelling. The skin is not usually reddened. The small joints, especially of the fingers and toes, are symmetrically enlarged. The disease does not migrate, but continues in the joints originally affected, while others become involved. The patient appears anemic, and headache, anorexia, and malaise are sometimes complained of. There is seldom elevation of temperature above 102° F. (39.0° C.).

The *chronic form* is more frequent. It is of insidious onset. It often begins in a single joint of a finger or toe, then passes to the corresponding articulation of the opposite side, and afterward to others, until all the joints of the body have been involved. The joints are swollen, painful, and tender. Neuralgic pains often accompany those of the joints and are attributed to atrophic degeneration of the nerve-roots.

2. **Monarticular Type.**—The disease is not always confined to a single joint, as the name signifies. A single, large articulation is generally more severely affected than any other. This form is more frequent in men and after the fiftieth year, attacking especially the knee, shoulder, elbow, and hip. When located in the hip, it constitutes the disease known as morbus coxæ senilis. It generally confines itself to the joint first affected, but that of the opposite side may become to a less degree involved. The vertebræ are often affected (spondylitis deformans), the entire column sometimes becoming fixed and motionless through bony ankylosis. Motion of the affected joints often produces creaking or grating sounds. The muscles atrophy. The deformity may be greatly added to by the accumulation of fluid in the bursæ.

3. **Heberden's Nodosities.**—These are small exostoses, "little hard knobs," seldom larger than peas, which form on either side of the distal joints of the fingers. The characteristic destruction of cartilage and eburnation occur in the joint proper. The disease follows the intermittent course of the other forms, with occasional attacks of pain and swelling. The nodosities are most frequently seen in women between 30 and 40. They are generally the only manifestation of the disease, but they may accompany the monarticular form.

Arthritis Deformans in Children.—The disease is not frequent in children. Koplik, in 1896, found only 18 cases recorded. Schüller holds the very plausible theory that all cases that have been described as occurring in children are examples of polyarthritis chronica villosa, a disease recently described by him. The pathological conditions of the joints undoubtedly conform more closely to that disease than to arthritis deformans, for the articular cartilages show no destructive changes, and the clinical manifestations are much at variance from those found in adults.

As generally described, the disease begins in children with acute symptoms; fever, sometimes a chill, swelling, stiffness, and tenderness of the joints. It is more frequent in girls. The enlargement is due rather to thickening of the soft parts than of the bone. The children generally lack physical development. Enlargement of the spleen and lymph-glands has been noted by Still.

Appearance of the Joints.—The deformity of the joints is quite characteristic. The fingers are turned toward the ulnar side, strongly flexed, and they generally overlap one another. The distal joints may be turned toward the radius. The joints are generally firmly locked. The feet are strongly extended, and the joints are often more rigid than those of the fingers. The deformity of the larger joints usually consists of a widening of the articulation by osteophytic growths. The greatest prominence is on a level with the articular surface, as a rule. As a result of changes in the hip and knee, the legs are drawn up in most cases, rarely firmly extended.

Diagnosis.—The disease is to be differentiated from subacute and chronic rheumatism, gonorrheal rheumatism, gout, progressive muscular atrophy, Charcot's disease, coxa vara, and polyarthritis chronica villosa.

From *rheumatism* the distinction is often difficult in the early stages of the disease. Involvement of the smaller articulations and the stationary character of the disease should arouse suspicion, even in acute cases, and especially when the patient is a woman over 45. *Chronic rheumatism*

usually involves fewer joints, is more likely to be unilateral, and the joints do not creak.

Gonorrheal rheumatism may produce similar joint enlargement, but the osteophytic formations are absent.

Chronic gout is distinguished by its affecting only a single joint, it is more painful, tophi form about the affected articulation, and arterio-scleroses are common. There is an inherited tendency.

Progressive muscular atrophy is free from joint enlargement.

Charcot's disease is distinguishable by the presence of the characteristic symptoms of locomotor ataxia, and the osteophytic formation is not so great.

Coxa Vara.—Maydl affirms that a differentiation often cannot be made until the joint has been cut down upon and examined, but coxa vara occurs only in young subjects at or near puberty.

Polyarthritis Chronica Villosa.—This disease occurs most frequently in women before the menopause, sometimes in children. The lesions are confined to the synovial membrane and may continue for a decade without causing destruction of cartilage. Pain is a prominent symptom and occurs independently of motion.

Prognosis.—The disease is incurable, yet it is not directly dangerous to life. Its progress usually becomes slower and less painful as it advances, and the patient, although bedridden on account of weakness and deformity, may pass his later years in comparative comfort. But the confinement to bed often induces the development of other affections, notably bronchopneumonia, which may hasten the end.

Treatment.—The treatment is for the most part hygienic and dietetic. Whenever possible, the patient should reside in a warm, equable climate; he should at least live in dry, healthful quarters. Unfortunately, poverty often prevents such measures. Mere change of air and scenery is often beneficial. Every precaution must be taken against exposure and chilling of the body. The diet should be liberal, including an abundance of both nitrogenous and carbohydrate food. Codliver oil, alone or with malt, is beneficial.

Internal medication is of no benefit, except in the early stage or during acute exacerbations. The sirup of the iodid of iron is indicated for the anemia. The salicylates relieve the pain of an acute exacerbation. Moderate exercise should be taken, just short of fatigue. The joints should not be given complete rest so long as it can be avoided.

The hot-air treatment has been much employed of late, with decided benefit in some cases. It consists in placing the affected limb, well wrapped in a cylinder constructed for the purpose, and raising the temperature within the cylinder to 250° or 300° F. (120° to 150° C.) for from half an hour to an hour, repeating the treatment twice or three times a week. The benefit is, no doubt, to be attributed to a more or less permanent dilatation of the blood-vessels of the joint. Massage is of benefit in restoring the nutrition to the muscles.

CHRONIC RHEUMATISM.

Definition.—A chronic affection of the joints, of insidious development, slow progress, and producing painful thickening and contraction of the

fibrous structures of the articulation, that result in great impairment of motion and more or less deformity.

Etiology.—The disease usually appears after the fortieth year, and a little more frequently in women. A hereditary predisposition is sometimes apparent. Poverty, hard labor, exposure to cold and wet, and traumatism are often important factors in its production. It is usually a primary condition, rarely following acute articular rheumatism and only occasionally the subacute.

Morbid Anatomy.—The disease progresses so slowly that there is at no stage any very active pathological process. The synovial membrane is usually thickened and injected. The same condition is often found in the capsular and other ligaments and in the tendon sheaths about the articulation. The muscles become atrophied from disuse, and there is sometimes a neuritis of the peripheral nerves. The articular surfaces are either unchanged or there is a slight superficial erosion of the cartilages. The synovial fluid may be normal, diminished, or slightly increased in quantity.

Symptoms.—The most prominent symptoms are pain and stiffness of the joints, more or less constant in character. The pain often becomes greater toward evening, and the stiffness persists in the morning until the joints have been "limbered up" by exercise. There may be slight tenderness and moderate swelling, seldom any redness. The motion of the affected joints becomes more and more restricted until the condition becomes practically one of ankylosis. Acute exacerbations occasionally develop, with slight elevation of temperature. The disease generally affects several joints, but in some instances it is confined to one or more of the larger articulations, as the shoulder, knee, or hip. It may attack only the small joints, especially in those who work with the fingers. It sometimes passes from one location to another, but it is, as a rule, stationary, and it is not infrequently unilateral. Neuralgic pains are often added to those of rheumatism. The general health may remain good, but the patient usually acquires an anemic appearance. Dyspepsia and emaciation develop, largely, no doubt, as a result of the general debility and loss of rest. Cardiac valvular lesions of a sclerotic nature are found as a senile change, but not as a result of the rheumatism.

Diagnosis.—The slow progress of the disease and the absence of all acute manifestations serve to distinguish the affection from other joint diseases. The prognosis is unfavorable with regard to cure, but the disease is not fatal. Treatment may afford comfortable quiescence for many years.

Treatment.—The treatment should be directed to the general condition of the patient and the relief of suffering. Improvement of digestion and nutrition is important. Internal medication is often of little benefit. Potassium iodid, guaiacum, and sarsaparilla are sometimes useful. The salicylates are useless except for the relief of acute exacerbations. Cod-liver oil and tonics should be employed to improve the general nutrition. Residence in a dry, warm climate often arrests the progress of the disease. Local applications of ointments containing camphor, menthol, or alkalis, and embrocations or poultices, often afford relief. Cold applications are sometimes better. The hot-air treatment has recently been

employed with great benefit. Supplemented with massage and passive motion, it has restored to usefulness many deformed and stiffened joints.

MUSCULAR RHEUMATISM.

MYALGIA.

Definition.—A painful affection of various voluntary muscles and of the fasciæ and periosteum, to which they are attached. Special names are usually applied to it when the muscles of certain regions are involved, as torticollis, lumbago, pleurodynia, cephalodynia, etc.

Etiology.—The disease attacks individuals of any age, but acute cases are more frequent in children and young adults. Men are more commonly affected on account of greater exposure. The rheumatic or gouty diathesis and a previous attack favor its development. Exposure to cold or wet, especially when the body is overheated, or exposure of a part, as the neck, to a cold draft often induces an attack. Strains, bruises, and overaction of a set of muscles may induce it, or a myositis presenting the same manifestations.

Pathology.—The true nature of the disease is not known, since it never proves fatal. Many authors regard it as a neuralgia of the sensory nerves in the muscles, and not as an affection of the muscle tissue, but there is some evidence of its being a myositis with involvement of the fasciæ and periosteum, since these conditions have been seen in cases of acute articular rheumatism in which the muscles were simultaneously affected. In chronic cases there is a round-celled infiltration of the muscles, with proliferation of nuclei and hyperplasia of the connective tissue.

Symptoms.—Pain is the most important feature. It may be severe and paroxysmal or it may have the character of a constant aching. It is often lessened by pressure. It may last for only a few days or for several weeks, and recurrences are common; it may become chronic. Fever is observed in not more than a third of the cases, and it seldom exceeds 102°·F. (38.8° C.). The principal varieties of the disease are:

(1) **Lumbago,** affecting the muscles of the lumbar region and their tendinous attachments. It is probably the most frequent form. It often attacks the individual without warning, the first indication being an excruciating pain in the loins upon attempting to rise from a sitting posture. When in bed the patient often cannot change his position.

(2) **Torticollis,** wry-neck, or stiff-neck, affecting the anterolateral muscles of one side, less frequently those of the back of the neck. The head is held in a characteristic position and cannot be rotated.

(3) **Pleurodynia,** usually involving the intercostal muscles of one side, generally the left, less frequently the pectorals and serratus magnus. The pain is rendered extreme by coughing or sneezing, and ordinary respiration is painful.

(4) **Cephalodynia,** affecting the muscles of the head. The terms scapulodynia, omodynia, and dorsodynia are occasionally employed when the

shoulder or upper part of the back is affected. The muscles of the abdomen and extremities may be involved.

Diagnosis.—The condition is recognized by its location, the increase of pain upon motion, and the absence of constitutional disturbance. Pleurodynia may be confounded with intercostal neuralgia or pleurisy, but is generally distinguished by the local tenderness and the absence of tender points along the nerve-trunks, as well as by the constant character of the pain.

Treatment.—Rest and the application of heat, or of ointments containing menthol and camphor or salicylic acid, and counter-irritation are the best methods of treatment. In extreme cases, morphin hypodermically may be required, but it should be avoided if possible. The Paquelin cautery may be advantageously applied with a few quick strokes. In torticollis, a hot poultice or turpentine stupes often afford relief. In lumbago, the application of a hot iron over a few thicknesses of flannel, a hypodermic injection of distilled water, or the hot-water bottle often affords relief. Acupuncture, thrusting long, sterilized needles into the muscles of the back and allowing them to remain for five or ten minutes, is highly recommended, but often fails. In pleurodynia, strapping the chest is one of the best measures. The constant current is sometimes beneficial in all forms of the disease. A Turkish bath frequently cuts short an attack. In chronic cases, potassium iodid, guaiacum, sulphur, quinin, nux vomica, and arsenic should be employed in succession, if necessary. A gouty subject should restrict his diet, and drink freely of alkaline mineral waters.

GOUT.

PODAGRA.

Definition.—A perversion of nutrition producing recurrent attacks of arthritis, deposits of sodium biurate in and about the joints, and various constitutional disturbances.

Etiology.—Neither the nature of gout nor its cause has been satisfactorily determined. The disease is attributed to faulty metabolism, especially to a defective oxidation of proteids, with deficient elimination of waste-products, particularly the urates. The blood, it is asserted, contains an abnormally large quantity of uric acid, probably in the form of a biurate, which is deposited as tophi, little chalky masses, about the joints, and occasionally in other localities. The disease is generally described as belonging peculiarly to England, a little less exclusively to France, Germany, and Holland, but it is by no means rare in the United States. It is apparently becoming more common; it is at least more frequently recognized than it was a few decades ago. The more important of the recognized causes are:

(*a*) *Age.*—It is a disease of advanced life, but primary attacks generally occur before 50; very rarely before 20 in the presence of a strong hereditary tendency.

(*b*) *Sex.*—Men are much more frequently attacked, chiefly on account of their addiction to habits which induce it; yet goutiness is not rare in women.

(*c*) *Occupation.*—It is rather a lack of occupation that induces the disease. Idleness and neglect of exercise are largely operative in its production.

(*d*) *Heredity* is recognized as one of the most important factors, but it is not commonly traceable in this country. In England the disease can be traced to the parents or grandparents in more than 50 per cent of the cases.

(*e*) *Alcohol* is the most important factor, especially the free indulgence in fermented liquors. The disease is more frequently found, therefore, to follow the free use of wine and malt liquors, or the heavy beers and ales of England and Germany, than whisky or the light beers of America.

(*f*) *Food*, especially excessive eating, without proper exercise, is next in importance to alcohol. Gouty dyspepsia is common, but many cases occur in individuals with vigorous digestion. Rich, highly seasoned food is commonly referred to as the cause, but gout frequently occurs among the poor, as a result of defective nutrition, bad hygiene and intemperance ("poor man's gout").

(*g*) *Toxic Agents.*—Lead favors the production of gout. The gouty deposits occur not only as a result of chronic lead-poisoning, but, as Garrod has shown, very commonly among painters and workers in lead. Haig attributes an unfavorable influence also to opium, cocain, strychnin, mercury, iodids, nitrites, some of the sulphates, hypophosphites, lithia, acids, and several other agents. The acute form of the disease occurs a little more frequently in the late fall and early spring.

Pathogenesis.—Our knowledge of the origin of the disease is purely speculative. The most important theories are the uric-acid theory of Garrod, Ebstein's theory of nutritive disturbance, and Cullen's theory of nervous influence. All are based upon the assumption that the condition is intimately related to the presence of an excess of uric acid in the system.

(1) *Uric-Acid Theory.*—This attributes the disease to the presence of an excess of uric acid in the blood and tissues, and its deposit in the tissues to a deficient alkalinity of the blood-plasma and other fluids which normally hold it in solution. In an acute paroxysm this accumulation of uric acid is shown by a gradual diminution of the quantity eliminated by the kidneys for several days before and during the attack. The inflammation is believed to result from a sudden deposit of urates in crystalline form in the tissues of the joints. Some writers believe that there is increased formation of uric acid as well as deficient elimination. Ebstein concludes that the acid may be formed in unusual places, as in the muscles and in the bone-marrow. Kolisch thinks that the kidneys normally form uric acid, and that the disease develops only when the function of these organs is impaired. And, since he has found that the xanthin bases are also increased in gout, he attributes the functional impairment of the kidneys to their action. Garrod, accepting the theory of uric-acid formation in the kidneys, holds that when uric acid is found in the blood it is as a result of its absorption from the kidneys. It has been found, in support of Kolisch's theory, that the injection of xanthin and hypoxanthin into the blood is followed by struc-

shoulder or upper part of the back is affected. The muscles of the abdomen and extremities may be involved.

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Treatment.—Rest and the application of heat, or of ointments containing menthol and camphor or salicylic acid, and counter-irritation are the best methods of treatment. In extreme cases, morphin hypodermically may be required, but it should be avoided if possible. The Paquelin cautery may be advantageously applied with a few quick strokes. In torticollis, a hot poultice or turpentine stupes often afford relief. In lumbago, the application of a hot iron over a few thicknesses of flannel, a hypodermic injection of distilled water, or the hot-water bottle often affords relief. Acupuncture, thrusting long, sterilized needles into the muscles of the back and allowing them to remain for five or ten minutes, is highly recommended, but often fails. In pleurodynia, strapping the chest is one of the best measures. The constant current is sometimes beneficial in all forms of the disease. A Turkish bath frequently cuts short an attack. In chronic cases, potassium iodid, guaiacum, sulphur, quinin, nux vomica, and arsenic should be employed in succession, if necessary. A gouty subject should restrict his diet, and drink freely of alkaline mineral waters.

GOUT.

PODAGRA.

Definition.—A perversion of nutrition producing recurrent attacks of arthritis, deposits of sodium biurate in and about the joints, and various constitutional disturbances.

Etiology.—Neither the nature of gout nor its cause has been satisfactorily determined. The disease is attributed to faulty metabolism, especially to a defective oxidation of proteids, with deficient elimination of waste-products, particularly the urates. The blood, it is asserted, contains an abnormally large quantity of uric acid, probably in the form of a biurate, which is deposited as tophi, little chalky masses, about the joints, and occasionally in other localities. The disease is generally described as belonging peculiarly to England, a little less exclusively to France, Germany, and Holland, but it is by no means rare in the United States. It is apparently becoming more common; it is at least more frequently recognized than it was a few decades ago. The more important of the recognized causes are:

(*a*) *Age.*—It is a disease of advanced life, but primary attacks generally occur before 50; very rarely before 20 in the presence of a strong hereditary tendency.

(*b*) *Sex.*—Men are much more frequently attacked, chiefly on account of their addiction to habits which induce it; yet goutiness is not rare in women.

duces an appearance which Duckworth has compared to splashes of white paint. The synovial membrane sometimes contains the white splotches, but its fringes escape. The synovial fluid in the larger articulations sometimes becomes thickened and may contain tufts of crystals. The fibrocartilage and ligaments are later involved, and there are generally the distinct masses of deposit known as tophi or chalk-stones. The tissues covering these masses frequently become eroded, and the tophi finally protrude through the skin. Ulceration of the surrounding skin and sometimes necrosis ensue. The articulations most frequently affected are the first joint of the great toe, then the ankles, knees, and the small joints of the hands and fingers. The joints of the upper extremity escape in many cases. Tophi are commonly found in the cartilage of the ear, at the margin of the helix, less frequently in the cartilages of the nose, eyelids, and larynx. Rarely they are met with also in the substance of the muscles, in the sclera, or in the cerebral and spinal meninges.

Lesions are more or less regularly found in other localities, notably in the kidneys and blood-vessels. The renal changes correspond to those of interstitial nephritis, with the addition of the so-called uric-acid infarcts both within the tubules and in the epithelial cells and interstitial tissue. The deposits are found especially in the region of the papillæ and on the bases of the pyramids. In the blood-vessels various changes are seen, but most frequently an arteriosclerosis, sometimes a hypertrophy of the muscular coat or an atheromatous deposit. Hypertrophy of the heart usually accompanies the change. Chalky concretions in the valves have been described.

Symptoms.—The clinical manifestations are generally described under the three heads of acute gout, retrocedent gout, chronic gout, and goutiness or irregular gout.

(1) **Acute Gout.**—Theacute attack usually begins with premonitory indigestion, restlessness, headache, and often melancholia, with occasional twinges of pain in the joints of the hands or feet. The urine becomes scant, dark, and strongly acid, and shows a deposit of urates on cooling. It may contain traces of albumin or sugar (gouty diabetes). The uric and phosphoric acid ingredients are generally diminished shortly before and during the attack, but much depends upon diet. A chill sometimes occurs. As a rule, however, the patient is awakened in the early morning with an intense pain in the distal joint of the right big toe, possibly in the left. The pain increases during the next two or three nights; it may subside to a great extent during the day. Such paroxysms last six or eight days; they may be prolonged by the involvement of additional joints. It is described as a burning, throbbing, lancinating pain that seems to wedge the bones apart or to press them together as in a vise. At first the veins about the joint become distended, then the skin becomes uniformly swollen, red, and glazed; the slightest motion, a touch, or the weight of the bedclothing causes intense pain. Fever is often present, reaching 102° or 103° F. (38.9°—39.5° C.). The inflammation subsides gradually; suppuration does not occur. Desquamation of the skin over the affected joint is sometimes observed. Recurrence is common; some patients have three or four attacks every year.

(2) **Retrocedent Gout.**—This term has long been applied to such phenomena as violent gastralgia, precordial distress, dyspnea, vomiting, and collapse when they occur at a time when the acute symptoms of gout are subsiding. They sometimes follow the application of cold to the affected joints. Fatal pericarditis, apoplexy, and uremic coma are sometimes included in this class of manifestations.

(3) **Chronic Gout.**—This is the outcome of repeated acute attacks. Its distinctive feature is the formation of tophi. These are seen especially at the sides of the joints, then in the ligaments and other structures, until marked deformity and immobility or ankylosis are produced. They are seen first in the hands and feet, later, perhaps, in the elbows and knees, the tendons, particularly on the dorsum of the hands, in the bursæ, and elsewhere. After ulceration of the skin they become visible.

Indigestion is a prominent symptom in most cases, with flatulence, acid eructations, and constipation. Irritability, moroseness, and mental depression are often observed, but not in all cases. The disease has always been notable for its prevalence among men of prominence and scholarly attainments. As Sydenham expressed it, "More wise men than fools are victims of the affection." Many conditions described under the head of Goutiness are more or less uniformly present, and uremia, inflammation of the serous membranes or meninges not infrequently develop as terminal affections.

(4) **Goutiness or Irregular Gout.**—These terms, as well as gouty or lithemic diathesis, are applied to ill-defined groups of symptoms which occur in the members of gouty families. They are often the only manifestations of acquired gout. Acute attacks are often absent. Unfortunately, there is a tendency to attribute to the diathesis every disturbance which occurs in an individual bearing the inherited taint, whether it affects the joints, skin, nervous system, or other parts. Prominent among these affections are:

(*a*) *Cutaneous Eruptions.*—Urticaria is common in early life; chronic eczema in later life. Burning and itching of the feet at night are regarded as gouty indications.

(*b*) *Digestive Disorders.*—Flatulence, hyperacidity, with pyrosis and "biliousness," with coated tongue, fetid breath, and constipation, gingivitis, tonsilitis, enlargement of the uvula, congestion of the liver, hemorrhoids, headache, colic, neuralgia, and intestinal catarrh, are encountered in some instances.

(*c*) *Respiratory Disorders.*—There is often a tendency to catarrh, producing attacks of coryza, pharyngitis, laryngitis, or bronchitis. Emphysema and asthma are common, and uric-acid crystals have been found in the sputum.

(*d*) *Circulatory System.*—Arteriosclerosis is a common change. Owing to the high blood-tension, changes are produced, not only in the vessels, but in the heart and kidneys. The right ventricle is hypertrophied, but later yields to dilatation and becomes feeble in action. Dropsy then ensues. Aneurism may be developed or a cerebral vessel may rupture, and thrombosis of the coronary arteries is often a cause of death.

(*e*) *Urinary System.*—Nephritis may develop early or late. "Showers" of uric acid occur, large quantities of sand or gravel being passed. The small quantity of sugar often found sometimes increases into a true

diabetic condition. Calcium-oxalate crystals are sometimes found in the urine. Urethritis is readily induced in gouty subjects; some writers believe that it may develop spontaneously after an attack.

(*f*) *Eye Affections.*—Iritis, glaucoma, and gouty lesions in the retina or its vessels and the optic nerve, keratitis and panophthalmitis, have all been attributed to the gouty condition.

Diagnosis.—Acute gout is recognized by its usually attacking the smaller articulations in the first instance. The swelling does not wander, but continues while other joints become affected. There are also less fever and sweating than in acute rheumatism. The habits of the individual and the condition of his mucous membranes are of value. The chronic form is usually made obvious by the presence of tophi in the region of the joints or in the ears, the history of the diathesis, previous attacks, and other evidences of the disease.

Treatment.—*Hygienic.*—Gouty persons and those predisposed to the disease should abstain from fermented liquors; they should also avoid overeating, and take regular outdoor exercise. They should favor the elimination of urea through the skin by frequent bathing. Robust individuals should take a cold bath every morning, followed by vigorous rubbing, and an occasional Turkish bath; those in feeble health, a warm bath before retiring. They should wear warm clothing and guard against sudden changes of temperature. Removal from a humid atmosphere to a higher, dryer climate is often beneficial.

Dietetic.—Some writers recommend an exclusively vegetable diet, others a mixed one; some advise the use of fruit, others forbid it. In the acute stages the food should be largely liquid; milk, buttermilk or koumiss, broths, junket, and gruels. Large quantities of water should be drunk, and pure water is doubtless better than water containing lithia or other solids which must be eliminated by the kidneys. Alkaline waters afford relief from the hyperacidity of the stomach, and have the advantage of appealing to the fancy of the patient. Much benefit may be derived from a prolonged visit to mineral springs. Farinaceous food and fresh vegetables are generally allowed, with the exception of strawberries, cherries, and bananas; but sweets are to be avoided. Hot bread and articles made of Indian corn are not to be eaten. Table salt should be eaten sparingly. Fats are allowed by Ebstein.

Medicinal.—The acute inflammation of the joints is greatly relieved by bathing them in hot water, then applying an ointment containing menthol, or chloroform liniment. A mixture containing one part each of guaiacol and oil of wintergreen and two parts of olive oil is soothing to the pain. The joints should be thickly wrapped in flannel. The hot-air treatment affords at least temporary relief.

The internal treatment should be begun with the administration of a mercurial purge; even when diarrhea is present, small doses of calomel should be given. The wine or tincture of colchicum should then be administered in doses of ♏xv to xxx (1.2—1.8), usually in combination with potassium, sodium, or lithium citrate, bicarbonate, or salicylate, gr. xv (1.0), every four hours until the pain has been relieved. The doses of colchicum should then be reduced to ♏x (0.6). The action of this drug is cumulative and should, therefore, be watched. Personal idiosyncrasy often prevents its use. If used too freely it is apt to produce

vomiting, epigastric pain, diarrhea, or renal irritation. Morphin is sometimes necessary for the relief of suffering, but it should not be given until colchicum has failed, since its use should be avoided in all chronic diseases. A few doses of phenacetin or lactophenin in the beginning will often afford relief until the colchicum has had time to act.

RICKETS.

RACHITIS.

Definition.—A disease of infancy characterized by defective nutrition, with its most pronounced manifestations in the growing bones.

Etiology.—The disease is more common in Europe than in America, but it is by no means infrequent among the children of the poor in our large cities. Congenital cases have been recorded, but the disease seldom becomes apparent before the second year, or until the child has begun to crawl and stand. Male and female children are equally affected. Tardy rickets has been described, developing as late as the ninth to the twelfth year, but it is at least quite rare. Improper food, as the milk of a pregnant mother; bad hygiene, including lack of light and ventilation, are important factors in its production. But it is occasionally met with among the children of the wealthy, especially in those fed upon condensed milk and other artificial foods deficient in animal fats and proteid. Defective assimilation of lime-salts doubtless plays a part in its production. The disease is probably independent of syphilis, but may be modified by it.

Morbid Anatomy.—The lesions are found especially in the bones. On account of a deficient deposit of lime, sometimes as a result of the absorption of already formed bony tissue, the bones remain or become soft and unnaturally flexible. The changes are best studied in the long bones. The periosteum, cartilage, and often the bone itself, in the early stages of the disease, are hyperemic. This condition is in itself regarded as sufficient to explain the other changes, since it has been shown by Kassowitz, that hyperemia prevents the deposit of lime-salts and at the same time disturbs the nutrition of the bone previously formed. The periosteum may strip off readily, but it frequently brings spiculæ of bone with it, and the underlying shaft is usually soft and porous. Instead of the two narrow parallel lines which normally represent the zone of proliferation between the shaft and the epiphyses, there are two rather thick bands with bulging, serrated edges. There is a superabundant proliferation of cartilage-cells, and the remaining matrix often becomes fibrillar. Unnatural areas of defective ossification are also seen. In the flat bones of the cranium, these centers are often large and prominent, producing the condition known as craniotabes. In the atrophied parts the bone becomes so flexible that it can be depressed with the fingers, to which it gives the sensation of bending parchment (parchment-crackling). The liver and spleen are usually larger than normal, and the systemic arteries and lymph-glands are often enlarged. After recovery has occurred, the bones have their normal firmness, but a part of the deformity generally persists throughout life.

Symptoms.—The development of the disease is generally insidious, and

it is too often overlooked until distinct deformities have taken place. The rachitic child generally suffers early from indigestion or distinct gastro-intestinal catarrh. It is especially susceptible to bronchitis and other affections of the respiratory organs. It is usually pale, often emaciated and weak. It is fretful, peevish, and restless at night; it often rolls its head until the back of it becomes denuded of hair. It sweats profusely. It has trouble with the irruption of its teeth, which may be delayed, irregular, or slow. The child often cries with pain when it is lifted from its bed. There is often slight fever, and the enlargement of the spleen can be recognized early in most cases.

The *head* generally appears large and has a comparatively square shape (caput quadratum), owing to the prominence of the thickened frontal and parietal eminences. The fontanels remain open until the second or third year, and their edges are extremely thin and flexible. The lower jaw often appears angular. The jaws are, in fact, poorly developed. The skin is thin, and the veins stand out like blue cords. A systolic murmur can often be heard by auscultation over the anterior fontanel or parietal region, but it is not peculiar to rickets.

The Thorax.—The changes in the thorax develop early and are quite characteristic. Along either side there is a row of beadlike prominences (the rosary of rickets), due to the swelling at the junction of the cartilages and the ribs. The sides of the chest, along the line of attachment of the diaphragm, is often drawn in, and there is an evident sinking of the chest-wall during inspiration, particularly when the child is suffering from bronchitis. In most cases the upper portion of the thorax also appears depressed laterally, as though by the hands of the mother in lifting the child. The sternum becomes prominent, especially in its lower portion, sometimes to the extreme degree known as pigeon or chicken breast. A posterior curvature of the spine is often seen also, and the vertebral processes are prominent. The clavicles are often deformed, and there may be partial fractures, especially at the insertion of the steno-mastoid muscle.

The abdomen is prominent, partly as a result of the enlargement of the liver and spleen, but chiefly on account of intestinal distention. Deformity of the pelvis is usually present, and later in the life of a woman it may interfere with parturition.

The extremities show a distinct enlargement of the epiphyses and the lower limbs; the tibiæ especially often show characteristic curvatures, either anteriorly, posteriorly, or laterally. The femur may also become bent in extreme cases of bow-leg or knock-knee, producing a waddling gait. The upper extremities are less commonly affected, but the humerus may become bent as a result of crawling. Sharp bends (green-stick fractures) are often produced by injuries. The deformities in nearly all cases correspond to the manner in which the weight of the body has been supported (carpopedal spasms).

Rachitic children are particularly liable to nervous disturbances, especially to laryngismus stridulus and convulsions. Tetany sometimes develops, especially in the arms and hands, occasionally also in the lower extremities.

The disease almost invariably runs a chronic course unless treatment is instituted at an early stage. Improvement may be recognized by the

gradual closure of the fontanels, increase in the length of the bones, and improvement in the strength of the patient. Many of the deformities, especially those of the thorax and pelvis, usually persist, and dwarfism is a not unusual result. Acute rickets (infantile scurvy) is described on page 308.

Diagnosis.—Early recognition of the disease is important. Persistent restlessness, peevishness, and tossing of the head, abdominal distention, irregular or delayed dentition, should arouse suspicion of a rachitic condition before osseous deformity becomes apparent. The student should disabuse his mind of the idea, too often expressed, that the disease is a rare one or that it is found only among the poor. After deformities have developed, the diagnosis is apparent.

Prognosis.—The disease is not of itself fatal, but by favoring the development of respiratory disorders and lessening the power of resistance to the acute infections it contributes largely to the mortality of early childhood.

Treatment.—The treatment in many instances should begin before the birth of the infant. If conception occur during lactation, the child should be taken from the breast, both for its own sake and for that of the fetus. The general health of the mother should be looked after. If the child must be taken from the breast, and a wet-nurse cannot be obtained, the safest diet is properly diluted cow's milk, to which beef-juice, egg albumen, barley-water, or oatmeal gruel may be added as its age increases. The child must be bathed daily, and the brine bath is especially recommended. It should also be kept in the open air and sunshine as much as possible. It should not be allowed to attempt to walk so long as the bones are in an abnormal condition. Extreme cases should be kept in bed and handled as little as possible.

Medicinal Treatment.—In mild cases, before the disease has become advanced, improvement often begins promptly after the addition of salt to the food, as much as is consistent with palatability. Lime-water, calcium phosphate, and other remedies supposed to furnish lime to the tissues have been recommended, but they are probably not assimilated. Phosphorus is the most highly esteemed remedy. It should be administered in the dose of 1-120 grain (0.0005) three times daily, in codliver oil. Rubbing the skin with the oil is thought to act beneficially, particularly when there is marked soreness. The sirup of the iodid of iron is also useful in many cases. Orthopedic treatment often becomes necessary for the relief of the deformities.

DIABETES.

Definition.—A condition in which, owing to an inability of the system to consume it, sugar accumulates in the blood and is excreted in the urine.

Etiology.—The blood normally contains a small quantity of sugar, but under ordinary circumstances it is not excreted in appreciable quantity by the kidneys. There is attributed to the blood also the power of destroying a considerable quantity of sugar through, as some writers believe, a glycolytic ferment contained in the leucocytes. When, however, the quantity in the blood exceeds a certain limit—a condition known as

hyperglycemia—the excess is carried off in the urine. In order to be regarded as diabetes the glycosuria must be continuous for a period of several weeks or longer. This feature alone serves to distinguish diabetes from a transitory glycosuria arising from a great variety of causes.

The exact nature and specific cause of diabetes are alike unknown. It seems probable, however, that recent investigations have approached very near to the revelation of them. It has been long known that a transitory glycosuria may result from: (*a*) Profound narcosis of ether, alcohol, opium, or other drugs; (*b*) from coma of whatever origin; (*c*) from poisoning with carbon dioxid, amyl nitrite, mercury, strychnin; (*d*) from hysteria, neurasthenia, epilepsy, the traumatic neuroses; and (*e*) from chlorosis, exophthalmic goiter, or the acute infectious diseases. The administration of phloridzin, a glucosid found in the bark and roots of apple and cherry trees, also produces marked glycosuria, but probably of a different kind, since its action is known to be exerted upon the renal epithelium. There is a marked difference also in the capacity of different individuals to consume sugar. In some persons the ingestion of seven ounces (200.0) or less of grape-sugar into an empty stomach produces glycosuria, a condition known as an alimentary glycosuria.

Diabetes proper may probably depend upon any one of several pathological conditions, the most important of which are believed to be located in the liver, nervous system, or pancreas; possibly the suprarenal bodies are implicated in some cases. The theory that the disease may arise from a perversion of the glycogenic function of the liver-cells is an old one, supported by the common discovery of pathological conditions in the organ as well as by the fact that the liver is the chief factory and storehouse of sugar. Some writers believe also that the condition may be a result of disturbed metabolism in the tissues generally, or of trophic disturbances. The conditions of the nervous system most frequently associated with the disease are the results of injury, tumors, or the so-called neuroses. Conditions causing irritation of the floor of the fourth ventricle have been longer and probably oftener recorded than others, but they are not essential, since tumors in other regions and inflammation of the meninges may produce glycosuria.

In the further study of pathogenesis, two facts stand out prominently, namely, (1) that there is in the body-fluids of the diabetic a ferment which is capable of inducing glycosuria, and (2) that in 50 per cent of all cases lesions can be found in the pancreas. The first of these propositions has been established by repeated experiments in which the injection of diabetic urine into dogs produced glycosuria, even after the sugar had been removed from it by fermentation. The same result has been obtained also by injection of the contents of the intestine of a diabetic person under the skin of a rabbit or into the intestine of a dog.

The Pancreas and Adrenals.—Complete removal of the pancreas, or complete destruction of it through disease, is immediately followed by permanent glycosuria, with the production also of aceton, oxybutyric acid, and other substances peculiar to diabetes. It has been found, however, that if as little as one-fifth of the gland remains, glycosuria is not produced, even though the communication with the intestine be cut off. Or, if a small portion of the pancreas be previously transplanted to another part of the body, the remainder of the organ can be removed

without causing glycosuria. Recent investigations by several European experimenters, and by Opie and Steele in this country, have further shown that in all cases of pancreatic diabetes lesions can be found in the islands of Langerhans, groups of cells abundantly supplied with blood, but not in any way connected with the pancreatic duct. This secretion is therefore an internal one, and it is entirely different from the pancreatic juice. These cells are found in a state of hyalin or granular degeneration in many cases of diabetes. More recently Herter and Richards have shown that, after the injection, into the small animals, of dried suprarenal extract, glucose invariably appears in the urine. They found that the effect was especially pronounced when the adrenalin was injected into the peritoneal cavity or applied directly to the pancreas. In the latter instance, the solution produced, not the usual blanching, but intense hyperemia and engorgement. In this respect the effect is similar to that produced by the application of solutions of potassium cyanid or other substances capable of reducing the power of oxidation. After fatal doses of adrenalin, the cells composing the islands of Langerhans were found to be in a state of granular degeneration. Herter concludes from these facts that it is probably an interference with the internal oxidizing power of the cells in the islands of Langerhans that is responsible in large part for the production of diabetes. Although all the experiments referred to have not been confirmed, they strongly indicate the probability that the action of adrenalin upon the islands of Langerhans is at least one of the causes of their degeneration and consequently of diabetes. Flexner's experiments seem to confirm the suspicion that the regurgitation of acid fluid from the intestine into the pancreatic duct may cause a destructive inflammation of the gland.

There is some evidence that the disease may be communicated, and consequently its infectious nature has been suggested. In a little more than 1 per cent of a large number of cases the disease was observed in both husband and wife, and it has been affirmed that a systematic investigation would show glycosuria in 6 to 8 per cent of the apparently healthy marital partners. The discovery of the disease in several members of the same household, not related, has a bearing on this question, although it is argued also that these individuals become affected because they are subjected to the same diet and other influences, and not to a specific infection.

Predisposing Influences.—(1) The disease may occur at any time of life, but it is more frequent after 30. A large proportion of adult cases occur after 50. Men are a little oftener affected than women. (2) Heredity is an important factor. The disease has repeatedly been observed in successive generations and among brothers and sisters. In many instances, also, it has been met with in families of nervous temperament. It is much more frequent among the affluent than among the poor. (3) The Hebrews are particularly susceptible, and in some of the large cities the Irish rank next. (4) Obesity favors its development (lipogenic diabetes). Strümpell calls attention to its frequency among obese beer-drinkers. (5) The disease is more common in cities than in the country, and in Europe than in America. (6) It has occasionally followed the infectious diseases, as influenza, scarlatina, typhoid fever, cholera, or syphilis. Tuberculosis is often associated with it, but it usu-

ally plays the part of a terminal affection. Trousseau believed that an inherited tuberculous tendency increases the susceptibility to diabetes.

The nervous influences which are believed to lead to the affection are many; among them close application to business, or other nervous strain, shock, worry, fright, and injury or disease of the brain or cord. Conditions which lower the blood pressure as well as those which increase the rapidity of the capillary circulation, particularly vasomotor paralysis, have been regarded as the cause of permanent glycosuria.

Morbid Anatomy.—Aside from the lesions of tuberculosis and nephritis which are commonly found after death, the pathological changes are few. The body is extremely emaciated; abnormal areas of pigmentation are occasionally found in the skin. The blood contains an excess of glucose, the quantity often amounting to 0.4 or 0.45 per cent instead of the normal 0.15 per cent. Numerous fat-granules are usually seen in the plasma. The polynuclear leucocytes are especially rich in glucose. The heart is sometimes hypertrophied; endocarditis is unusual, but arteriosclerosis is common. The lungs are tuberculous in many cases; bronchopneumonia or chronic interstitial pneumonia is found in others. The liver is often fatty or cirrhotic and pigmented; it is sometimes enlarged, notwithstanding the sclerosis. The stomach is frequently dilated. The kidneys are generally hyperemic and often sclerotic. The lesions found in the nervous system are not uniform. In many cases there have been tumors or cysts, once a cysticercus, pressing on the floor of the fourth ventricle, but often in other localities. Perivascular changes and inflammations of the meninges have been described. The important changes found in the pancreas have been referred to under Etiology. The conditions leading to the degeneration of the islands of Langerhans have probably not all been recognized. The changes may be inflammatory, degenerative, atrophic, or neoplastic.

Symptoms.—The invasion of the disease is usually so insidious as to render the precise time of its beginning indefinite. The patient becomes languid and weak, and he rapidly loses flesh. Headache, nervous depression, insomnia, and neuralgia are often complained of. The appetite becomes voracious (bulimia) and the thirst almost unquenchable. Disturbances of digestion are not uncommon, as nausea, eructations, and constipation, but in many cases the digestion is remarkably good. The mouth becomes dry from deficiency of saliva; the tongue becomes red and glazed; aphthous stomatitis often develops late in the disease. In many instances the thirst and polyuria are the first symptoms to attract the attention of the patient. Three to four quarts (liters) are voided in 24 hours in the beginning, but in severe cases it may rapidly increase to 12 or 15 quarts. The skin is dry and harsh, and sweating seldom occurs except when tuberculosis is also present. The temperature may be subnormal except under the same condition. The pulse is rapid and its tension is high. The emaciation and loss of strength keep pace with the progress of the disease, but cases are occasionally met with in which a comparatively large quantity of urine rich in sugar is voided for years without loss of weight or recognizable impairment of health. In most cases the emaciation corresponds to the quantity of urine that is voided. As a rule, the disease progresses with a rapidity that is inversely proportionate to the age of the patient. It is particularly rapid and fatal in

young children, but it often lasts for many years in the aged. Nearly all young patients die in a profound coma; but older persons usually succumb to one of the complications. To this rule, also, there are exceptions. In some of the more rapid cases, polyuria is not marked, but there has been an evident defect in the assimilation of albuminoids and fats as revealed by examination of the feces and urine.

Special Symptoms.—(1) *The Urine.*—The quantity, as already stated, varies from 6 or 8 to 30 or 40 pints in 24 hours. It has usually a pale straw color and a high specific gravity, ranging from 1.025 to 1.050 or even higher. It has a sweetish odor and acid reaction. The quantity of sugar varies from 1 or 2 to 10 per cent. Ten to twenty ounces may be excreted in a day, and, exceptionally, as much as two pounds. (For sugar tests see page 731.) The diagnosis should not be based upon a single examination of the urine, but only upon repeated analyses during a period of several weeks. The urea, and more particularly the phosphates, are often greatly increased. Glycogen and aceton are often present and β-oxybutyric acid may be found after coma develops. Albumin is not uncommonly present, and finely emulsified fats may be found. Pneumaturia, or gaseous urine, sometimes results from fermentation within the bladder.

(2) *The Skin.*—Owing, no doubt, to the presence of sugar in the perspiration, the pus-formers find the skin a good medium for their growth; consequently wounds rarely heal without suppuration, furunculosis is common, and carbuncles are liable to develop; gangrene and sloughs readily form. Eczema is often observed, and it is particularly significant of the disease when it involves the genitalia.

(3) *Respiratory System.*—Acute tuberculosis, gangrene of the lung, lobar and bronchopneumonia are frequently terminal complications. Fat-emboli have been found in a few instances. The breath has often the sweetish odor of aceton, not unlike that of chloroform.

(4) *Circulatory System.*—The chief affection is arteriosclerosis, which may be manifested in many ways, often in the form of an interstitial nephritis, sometimes as a myocarditis, or by the production of cerebral hemorrhage, edema and, later in some cases, gangrene of the extremities.

(5) *Nervous System.*—Coma is especially frequent in young patients. Occasionally it is the first symptom to arouse suspicion of the disease. In other cases it is preceded by indigestion, nausea, vomiting, or one of the respiratory lesions accompanied with great dyspnea; or it may develop suddenly and with little or no premonition. It may last four or five days, or it may terminate fatally within a few hours. It is attributed to the presence of some toxic substance in the blood, possibly β-oxybutyric acid. Neuritis is comparatively common, appearing as a sciatic, trigeminal, intercostal, or other form of neuralgia, as muscular pain or cramp, facial paralysis, hemiplegia, hyperesthesia, or paresthesia of small areas, the latter being occasionally the seat of pain. Herpes zoster sometimes occurs. The knee-jerk is occasionally lost late in the disease, and there may be steppage gait, but the posterior columns of the cord are rarely or never affected unless locomotor ataxia develop in the patient. Atrophy of the optic nerve has been observed. Perforating ulcer of the foot is occasionally encountered. Lesions of the central

nervous system are less frequent, although severe headache is not unusual, and the patient often becomes morose or hypochondriacal, and general paralysis may occur. The sexual power is often lost; conception rarely occurs, and abortion is apt to follow.

(6) *Organs of Special Sense.*—Cataract is not uncommon, and it is especially rapid in its development among young persons. Retinitis, atrophy of the optic nerve, paralysis of accommodation, or sudden amaurosis is liable to occur.

Diagnosis.—Diabetes is to be distinguished from transient glycosuria and simple polyuria; and the diabetic coma is to be differentiated from that of uremia and alcohol. The distinction from transient glycosuria is practically one of time; but in most cases of the latter condition there is less rapid emaciation, the urine has a lower specific gravity and contains less sugar.

In *polyuria* the specific gravity is usually below 1.010, and sugar is not present.

In *alcoholic coma* there is usually other evidence of alcoholism; the patient can be aroused to attempt the answer of questions; the condition passes off in a few hours. The urine has a dark color and lower specific gravity, and it contains no sugar, or at most a mere trace.

In *uremic coma*, dropsy is generally present, the urine is highly albuminous and contains casts, but no sugar; the bladder may be almost empty.

Deception has been practiced, as recorded by Osler, through the introduction of cane-sugar or glucose into the urine.

Bremer and Williamson have each proposed a blood-test, which may be of value in the diagnosis of obscure cases. (See p. 718.)

Prognosis.—Recovery from true diabetes is extremely rare. Intermittent glycosuria, which is probably often mistaken for diabetes, is, on the other hand, very amenable to treatment. In patients under middle age, the prognosis is exceedingly grave, while in older persons the disease usually runs a slow and milder course, more amenable to treatment. The severity of a case may be estimated from the response to the removal of all carbohydrates from the food, as by putting the patient upon a milk diet for a few days. If the elimination of sugar continues without marked reduction, the case may be regarded as a grave one.

Treatment.—*Dietetic.*—Theoretically the patient should abstain entirely from carbohydrates, since the glycosuria depends to a great extent upon the quantity of these ingredients in the food, but practically this is next to impossible in most cases, and, if too rigidly insisted upon, it is apt to destroy the appetite, and lead to an occasional refraction of the rules with highly injurious consequences. It is probably better, as Thompson advises, to allow a small portion of bread, from two to four ounces daily, preferably toasted, for the craving for bread generally proves stronger than for any other article of food. Potatoes may occasionally be substituted for the bread, since they contain a smaller proportion of starch. In other respects the carbohydrates should be excluded, bearing in mind, however, the apothegm of Von Noorden, "Under all circumstances, the diet in diabetes must be so ordered that the strength of the patient may be thereby maintained and as far as possible increased." When it is found that the patient is not holding his own

on a restricted diet, some change should be made, and the effect of any particular diet should be carefully estimated through repeated analyses of the urine. A number of diabetic flours are offered in the market, but few of them are reliable, and some of them are largely adulterated with wheat-starch.

On account of the ravenous appetite of the patient it is often less difficult to institute the diabetic diet by degrees, causing a daily reduction of the quantity of carbohydrates, and at the same time giving a substitute, unless the urgency of the case demands a prompt change. Sugar should be the first article enjoined, and in its place the patient may use saccharin tablets. There is no better diet, perhaps, than one consisting largely of fats. The patient should consume two ounces or more of butter daily, and as much cream as his digestion will tolerate. Other sources of fat are, beef, bacon, smoked sausage, and ox tongue, cream cheese, mackerel, salmon, eels, and the free use of mayonnaise or other dressings prepared with olive oil. Bone-marrow is tasteful to many persons. One or more of these articles should be included in the dietary of each meal. The patient may eat also beef, veal, pork, venison, and the meat of domestic or wild fowl and birds, also the heart, sweetbreads, brain, and kidneys, nearly all parts, in fact, except the liver; but the meats must not be breaded. Oysters, lobsters, crabs, and shrimps may be taken. Among vegetables, those which grow above the ground are generally allowable, as lettuce, celery, cauliflower, asparagus, tomatoes, onions, cabbage, cucumbers, and watercress. Sour fruits may generally be eaten, especially sour oranges, apples, lemons, cherries, currants, pears, plums, strawberries, raspberries. Among liquids the patient may take clear soups, especially bouillon, turtle, and oxtail; coffee, tea, chocolate, cocoa, with cream, but sweetened with saccharin; whole milk, buttermilk, plain and carbonated alkaline mineral waters.

The list of articles to be avoided usually includes bread and all farinaceous preparations, potatoes and other vegetables that grow below the surface, and such beverages as beer, sweet and sparkling wines, and all that contain sirup. Confections are of course to be avoided.

Hygienic Treatment.—The patient should guard against overwork, nervous strain and worry, and he should take more than ordinary care to avoid exposure to cold. He should take moderate exercise daily. At the same time the skin should be kept in good condition by frequent bathing, either warm or cold, according to the reaction after the bath. A cold bath in the morning is the best if well borne; the warm bath is better at night.

Medicinal Treatment.—Opium has long held first place among remedies, and fortunately it is usually well borne and less liable to develop the habit than in a normal individual. It is better, however, not to inform the patient that he is taking it. It should be given in the form of pills containing gr. ss (0.032), two or three times a day, or codein may be given in the same dose, since it is less constipating. The dose should be gradually increased until 8 or 10 grains are taken daily, or amelioration of the symptoms has been obtained. It should be gradually withdrawn, after the elimination of sugar has nearly or entirely ceased. Good results have been obtained from the use of the arsenite of bromin in doses of ♏iij to v (0.18—0.30), and more recently from a solution of the

bromid of arsenic and gold in gradually increasing doses from gtt. iij to xv. Many other drugs have been employed with alleged benefit, especially the salicylates, creosot, iodoform, arsenic, nitroglycerin, jambul, and lactic acid. A glycerin extract of the fresh or dried pancreas, and trypsin, have been employed on the erroneous assumption that they supplied the internal secretion that is wanting, but little benefit has been claimed. Strychnin is an excellent tonic, and ergot may be combined with it as a vasomotor stimulant when needed. Constipation should be guarded against, since it increases the liability to coma. Should the digestion fail, the bitter tonics and a dilute mineral acid should be administered. Codliver oil may be given to supply the needed fat. A two or three grain pill of asafetida (0.15—0.20) has been recommended for the relief of the feeling of insatiety and epigastric gnawing. The pruritus and eczema are treated by bathing the skin with a boric-acid or sodium-hyposulphite solution, and applying an ichthyol or other ointment.

The coma is usually fatal, and little can be done to delay the result. Inhalation of oxygen has been thought of benefit, and large doses of sodium-bicarbonate, have been recommended to reduce the acid intoxication. Subcutaneous or intravenous injection of physiological salt-solution should be tried, since it has proved beneficial, to the extent of temporarily restoring consciousness in a few instances.

DIABETES INSIPIDUS.

Definition.—A chronic condition in which anexcessive quantity of normal urine is voided daily by a person who in other respects is in good health.

Etiology.—The cause is unknown. From analogy the disease is generally regarded as of nervous origin. It sometimes follows emotional excitement, concussion, or other injury of the brain, as well as trauma of the trunk and extremities, or such acute infectious diseases as typhoid fever, malaria, or cerebrospinal meningitis. Again, it has been attributed to congenital syphilis and malnutrition. In some instances the condition has followed sunstroke or the drinking of a large quantity of water on a hot day. It is to be distinguished, however, from the excessive flow of urine which is due to excessive drinking in polydipsia, a condition characterized by excessive thirst and often a hysterical manifestation.

The disease is a rare one. It occurs most frequently in young boys, sometimes in girls; it may develop in middle life, seldom later. It sometimes appears to be inherited, and congenital cases have been observed.

Morbid Anatomy.—There are no essential lesions. Various lesions of the nervous system have been found. The kidneys are sometimes enlarged and congested. Dilatation of the renal pelvis and ureters and hypertrophy of the bladder are sometimes present. Death has usually been the result of an independent affection, as tuberculosis.

Symptoms.—The condition develops gradually in the absence of a definite cause, otherwise abruptly. The essential symptom is the marked increase in the volume of urine. As much as 8 or 10 quarts (liters) are often excreted in 24 hours, and cases have been observed in which the quantity reached three or four times this limit. The specific gravity

usually ranges from 1.001 to 1.004, and the color is extremely pale. The total solid constituents may remain normal. Sometimes there is slight excess of urea, and inosite, phosphoric acid, sulphuric acid, creatinin, and very rarely a mere trace of albumin or sugar have been noted. The thirst is extreme, and the dryness of the tongue and skin resembles that of diabetes mellitus, but furunculosis is rare. Salivation has been noted. The appetite is generally good, and the general health may be undisturbed for many years. In cases due to a definite cause, however, there may be decline with emaciation, languor, feebleness, and sometimes insomnia. Diminution of the urinary secretion sometimes follows the development of an intercurrent malady. Recovery is extremely rare, but death is usually a result of another disease, as tuberculosis, pneumonia, or cancer.

Diagnosis.—The condition is to be distinguished from the polyuria of diabetes mellitus, hysteria, and interstitial nephritis. From the first of these it is readily distinguished by the low specific gravity of the urine and the absence of sugar; from hysterical polyuria, by its permanent character and the absence of hysterical manifestations; from that of interstitial nephritis, by the absence of albumin and casts or other evidence of ill health.

Treatment.—It is useless to restrict the diet or to limit the quantity of fluid consumed, except so far as the thirst can be relieved by chipped ice instead of water. Opium has been employed, but it is not curative. Valerian, in doses of 5 grains (0.30) of the powdered root, gradually increased to 20 grains (1.30), three times a day, has proved of benefit. Ergot, the salicylates, arsenic, strychnin, bromids, carbolic acid, atropin, and galvanization of the cervical spine have all been recommended. If a cause for the condition can be discovered, it should be treated. Congenital syphilis may thus call for specific treatment.

OBESITY.

Definition.—A condition of disordered nutrition characterized by a greatly increased development of adipose tissue.

Etiology.—The proximate cause is generally regarded as deficient oxidation. The condition may be to a great extent inherited, but it is seldom transmitted to all members of a family. By some writers it is thought to be related to the uric-acid diathesis, diabetes, and other forms of perverted nutrition. It is more apt to develop after middle life, but it is not infrequent in children. The principal causes that lead to it are excess of food and drink, especially of starches, sugar, and malt liquors, with deficient exercise, yet many fleshy persons are remarkably abstemious, and some are overcome with fat in the midst of an active life.

Morbid Anatomy.—The heart is usually large and infiltrated with fat, the right side dilated and the left hypertrophied; or there may be atrophy of the muscular structure of the entire organ. The lungs are usually, small the liver large and fatty; the stomach is large and the muscular coat well developed; the intestines are often dilated; the spleen, kidneys, and lymph-glands are usually small, and the pancreas hypertrophied. The blood often contains a greatly increased quantity of fat.

Symptoms.—The appearance is too well known to require description, except for the fact that the individual may be either ruddy or pale and anemic. All the functions of the body may be carried on normally, but there are usually interruptions, particularly of digestion. The bodily activity is impaired; the mind may be sluggish and dull, or bright and active. Obesity is generally progressive, except when it begins in early life; it may then subside at puberty. More important is the tendency to disease, and the diminished power of resistance which it entails. Death may occur by syncope from extreme fatty degeneration of the heart, from apoplexy due to the rupture of an atheromatous artery in the brain, from acute pulmonary congestion, rupture of the heart, angina pectoris, or uremia.

Treatment.—The general indications are to reduce the quantity of carbohydrates ingested and the allowance of fluid; alcohol should be forbidden. The change should not be too suddenly made, or carried to the extent of reducing the patient's strength. There are several methods of regulating the diet, chiefly by limiting the quantity of fluid and excluding certain articles of food. Banting's method consists in reducing the quantity of all kinds of food to an extent that can seldom be enforced. It permits only from 21 to 27 ounces of solids in a day, of which 13 to 16 ounces consist of animal food and only 2 ounces of bread. Sugar and other starches are strictly excluded. Ebstein restricts the same articles, but allows fats, because they produce satiety and diminish thirst, a fact observed by Hippocrates. Oertel strongly objects to the free allowance of fat and adopts a diet consisting of lean beef, veal, or mutton, and eggs, with green vegetables, and a limited quantity of fats and carbohydrates, including 4 to 6 ounces of bread daily. The quantity of fluid he limits to 6 oz. of tea, coffee, or milk, morning and evening, 12 oz. of wine, and 8 to 16 ounces of water in 24 hours. A most important part of his treatment, however, consists in systematic forced exercise, particularly mountain-climbing. The Weir Mitchell treatment confines the patient to bed for a month or six weeks on a regulated milk diet, with massage and the Swedish movement.

Hot baths, massage, and active exercise, including much walking, may be employed as adjuncts to any of the other methods which do not include them. Among drugs, the most satisfactory, perhaps, is the thyroid extract in doses of gr. v (0.32) t. i. d., but it fails in many cases.

Adiposis Dolorosa.—An affection of middle age, characterized by an irregular, symmetrical deposit of fatty masses in various regions of the body, preceded or attended with pain. The disease was first described by Dercum. Large, often pendulous, nodular, encapsulated masses of reddish fat are formed. Nerve fibers run over the nodules. Paresthesias sometimes develop. The nature of the disease is not definitely known, but atrophic changes in the thyroid gland, and interstitial neuritis, have been observed in cases, and improvement has followed the administration of thyroid extract.

SECTION IX.

Intoxications and Miscellaneous Diseases.

ALCOHOLISM.

INEBRIETY, DRUNKENNESS.

Definition.—An acute or chronic intoxication due to excessive indulgence in alcoholic beverages.

Etiology.—1. While the immediate cause of acute alcoholism is over-indulgence, there are many influences which predispose or lead to it. Among these are the example or invitation of companions, the desire to meet the demands of society, to cope with a rival, to relieve fatigue, anxiety, melancholy, grief, or pain. In most instances the intoxication is accidental, for the individual seldom starts with the intention to become drunken. 2. Chronic alcoholism is doubtless largely due to an inherited neurotic taint or instability of the nervous system. Not infrequently a more or less continuous line of inebriety is associated through several successive generations with occasional cases of hysteria, epilepsy, or insanity. The influence of example is also strong; but it often happens that the remembrance of a drunken parent stimulates the children to abstinence. The inherited tendency may crop out, however, in the third generation. The use of alcohol as a medicine in acute diseases has seldom begotten a fondness for it, but the physician should be guarded in advising its use as a tonic. Other predisposing causes are occupations requiring the handling of liquors, overwork, idleness, and other forms of debauchery.

Symptoms.—(1) **Acute Alcoholism.**—The first effect of the ingestion of a large quantity of alcohol is usually shown in an increased rapidity and force of the circulation. The face becomes flushed, later, perhaps, cyanotic; the pulse full and bounding, and the respiration deep and sometimes irregular. Nervous phenomena soon follow. There is at first stimulation of the centers of the cortex and cerebellum. The ideas flow rapidly, but later they become confused, and finally there is a complete demoralization both of common and of special sense. Natural peculiarities of disposition are exaggerated, and the individual becomes obtrusive in his friendship or quarrelsome to a degree. Muscular incoordination soon supervenes, then relaxation, and finally narcosis. While in this state, the drunken person is unconscious and to a great extent anesthetic; but he can almost always be aroused to the point of muttering answers to questions. The pupils may be either dilated or contracted, they are seldom unequal. The temperature is reduced, sometimes to several degrees below normal, even to 90°, 85° F. (29.5° C.), or

less; the respiration may become stertorous. The breath has the strong odor of alcohol. Muscular twitchings are not uncommon, but convulsions seldom occur, except in the chronic drunkard or after the ingestion of an enormous quantity of alcohol. Under such circumstances the convulsions may be fatal. A homicidal mania is sometimes induced. The term dipsomania is applied to the habit of indulging in an occasional spree, especially by one strongly predisposed to inebriety.

Diagnosis.—The diagnosis is seldom difficult, but serious errors are the more frequent on that account. The alcoholic coma is to be differentiated from that due to apoplexy, uremia, diabetes, epilepsy, opium, and other poisons. In most cases the diagnosis is best established by examination of the stomach-contents. The odor of the breath and condition of the pupils are alike untrustworthy, since these forms of coma frequently occur in alcoholic subjects.

Apoplectic coma is more profound, the pupils are more constantly unequal, and the hemiplegic relaxation of the muscles of one side and deviation of the tongue may be recognizable. *Uremic coma.*—An edematous face, contracted pupils, muscular twitchings, and convulsions are common, the coma is profound, and, unless the individual has indulged in alcohol, the odor of the breath is ammoniacal. The urine is albuminous, and contains casts. *Diabetic coma* is deep; the breath may be sweetish, the urine contains sugar. *Epileptic coma* follows a seizure the character of which can usually be recognized. *Opium narcosis* is characterized by extremely slow, interrupted respiration, close contraction of the pupils, feeble pulse and great muscular relaxation. *Other drugs*—absinth, chloral, ether, chloroform—and poisonous gases are generally recognizable by their odor upon the breath of the patient; the drug may be discovered in the stomach-contents or in the urine.

(2) **Chronic Alcoholism.**—This condition follows either constant or periodic excess, but more rapidly the former. The effects are seen for the most part in the gastrointestinal and nervous systems. The pathological changes are chiefly of a sclerotic character, and affect especially the liver and the peripheral nerves (alcoholis neuritis). To what extent the central nervous system may be involved has not been fully determined.

Digestive System.—Chronic gastritis is one of the most common results of excessive alcoholic indulgence. This is manifested by indigestion, nausea, gastric distress, vomiting, especially in the morning, anorexia, perverted appetite, furred tongue, and foul breath. Constipation usually accompanies it. Hepatic cirrhosis is induced in a variable proportion of cases, but especially in those who habitually take undiluted whisky into an empty stomach.

Symptoms.—*Nervous System.*—The manifestations may be either functional or organic in character; but the transition from functional disturbance to structural change is an insidious one. Among the functional symptoms are tremors of the hands and tongue, dullness of intellect, apathy, forgetfulness, disregard of duty, irritability of temper, often slovenliness, and sometimes general immorality and degradation. Periodical hallucinations may occur. Epilepsy and various forms of insanity, especially paralytic dementia, are generally regarded as possible results of chronic alcoholism.

The facies of the toper is generally characteristic. His eyes are watery, the conjunctivæ congested, the nose and cheeks are reddened by the dilatation of superficial veins, producing acne rosacea, the countenance becomes dull, and the speech slow and indistinct.

Circulatory System.—The heart of the chronic drunkard is not infrequently dilated, and a more or less general arteriosclerosis is almost uniformly present at a late stage of the disease. The extent to which the kidneys are affected by alcohol is variously estimated. It is not unusual, however, to find them normal. Formad has described an enlargement, especially in the transverse diameter, peculiar to excessive drinkers of beer.

Delirium Tremens (*Mania à Potu*).—This affection is generally only an acute disturbance occurring during the course of chronic alcoholism, but it may supervene upon a debauch or occur shortly after the cessation of a long-continued excess. Rarely it develops from a single spree, and then, as a rule, in one given to excess. Again, it is sometimes induced in an alcoholic subject after weeks of abstinence, by the receipt of an injury, a surgical operation, or an attack of illness; abstinence from food and mental distress are often operative factors.

Symptoms.—The attack usually begins with restlessness, insomnia, fear, and suspicion. Hallucinations of sight and hearing soon supervene. Rats, mice, and snakes, often of brilliant colors, appear upon the wall or crawl over the bed. The patient is often busily engaged in some imaginary employment; angels or demons are often his advisers or tormentors. In an unguarded moment he often tries to escape from his persecutors. Muscular tremors, especially of the hands and tongue, are constant features. The patient often sinks into a typhoid state, with elevation of temperature, seldom above 102° or 103° F. (39.5° C.). The pulse is rapid and soft, and the tongue becomes heavily coated. The symptoms subside after a few days, or the strength gradually declines and death ensues from failure of the circulation.

Diagnosis.—The condition is usually readily recognized when the history of indulgence is known. It is important, however, to make a thorough examination, particularly of the lungs, in order to exclude the presence of pneumonia, especially in the apex. Meningitis is not infrequently suggested by the condition. Erysipelas is often accompanied with delirium like that of alcoholism.

Treatment.—Sleep is generally a specific in acute cases. After a debauch, sleep generally comes spontaneously. In delirium tremens, however, it must be induced by the administration of drugs. Chloroform may be cautiously administered in a violent case. Chloral is safer, and should be combined with the bromids, gr. xv. (1.0) of the former and gr. xxx (2.0) of the latter, every two hours. Small doses of apomorphin (gr. 1-40; 0.0016) every hour often quiet the patient. Hyoscin hydrobromate, gr. 1-100 (0.0006) hypodermically, is perhaps better. Morphin is much employed, but it is often useless and never free from danger. Two or three doses of ¼ grain (0.016) should be the limit.

When the case is seen early, lavage of the stomach is indicated, unless vomiting has occurred. Milk and broths, given at short intervals, should constitute the diet. It is sometimes necessary to administer alcohol to support the heart for a few days, and strychnin should gener-

ally be given. When the temperature is low, the hot pack and hot bottles should be applied. For the relief of the gastric irritability and headache, usually following a debauch, the aromatic spirit of ammonia should be given in half-dram (2.0) doses. Blood-letting is recommended in sthenic cases following the ingestion of a large quantity of alcohol.

The treatment of chronic alcoholism is exceedingly unsatisfactory. As a rule, relapse occurs sooner or later. In a few cases, when there is a strong desire on the part of the patient to reform, prolonged residence in a sanitarium is effectual; but in the absence of determination and more than ordinary will-power, treatment is useless. The "drugging" of the patient's liquor with apomorphin or tartar emetic is occasionally successful in producing a temporary disgust for drink. The hypodermic administration of small doses of atropin, apomorphin, and strychnin, but more particularly of hyoscin hydrobromate, is said to have a similar effect. When circumstances will permit, a permanent removal to new scenes, and a careful selection of new associates, or continued travel in the companionship of persons capable of giving moral support to the patient's feeble determination, are sometimes productive of good results.

MORPHINISM.

MORPHIA HABIT, MORPHINOMANIA, OPIUM HABIT.

Definition.—A chronic intoxication with morphin or one of the other derivatives of opium.

Etiology.—In a majority of cases, in this country at least, the habit is acquired through the prolonged use of the drug for the relief of pain or insomnia, or to quiet alcoholic nervousness. It is most readily acquired from hypodermic administration. The habit is prevalent to a surprising degree among physicians and druggists, and a majority of the other habitués are women. It is very rarely deliberately developed for the supposed pleasure of it, but in the nether-world it is often adopted simply as an additional mode of dissipation. Morphin is taken hypodermically, laudanum and paregoric are drunk, and occasionally opium is smoked in the same manner as in the Orient. The same difference of susceptibility is observed as in alcoholism. Some persons give up the drug without difficulty after using it constantly as a medicine for many months, while others develop a craving for it almost from the beginning. Those who inherit an alcoholic tendency are the surest victims.

Symptoms.—For a short time the drug produces a feeling of exhilaration, a pleasant freedom from worry and care; but this is soon lost, and an increased indulgence is essential to even moderate comfort. As the effect of a dose begins to wear off, a feeling of weakness and mental depression, often accompanied with gastric distress and nausea, comes over the victim, and unless another dose is taken, he becomes nervous, irritable, cold, and tremulous. The continued use of it develops an appearance which is characteristic. There is progressive emaciation; the face becomes sallow, often wrinkled and prematurely aged. The pupils are contracted to the size of a pin-point when under the influence, or widely dilated, irregular, and changeable when deprived, of the drug. Itching, especially of the nose, is commonly a symptom. The tongue is

dry, and the lips must be frequently moistened; the speech becomes slow and drawling, and old habitués are not infrequently overcome with drowsiness, even in the midst of conversation. Sleep is often disturbed. The muscles twitch, and the limbs sometimes assume positions suggestive of catalepsy. Profound hysteria or neurasthenia is, in fact, often developed in women. Chills sometimes occur, and the tremor and excitement occasioned by deprivation of the drug may amount almost to mania. The quantity required by different habitués is not the same. Some never exceed 5 or 6 grains a day, while others rapidly increase the dose to 20, even 40, or more grains. In some instances, as in alcoholism, a moderate quantity is taken continuously, and a large dose is indulged in occasionally. The patient's statements can seldom be relied upon in regard to the quantity taken, for in most cases they become utterly untruthful. The duration of the habit is also variable. In Oriental countries the drug has apparently little effect upon the health, and it is often tolerated for many years. In other instances a fatal decline of strength is early induced by it.

Treatment.—The physician in general practice is seldom justified in attempting to cure the habit. What can be accomplished with safety and almost certainty in a sanitarium is extremely difficult and often dangerous elsewhere. The patient must be removed from the possibility of securing a supply of the drug. The method usually employed is the gradual withdrawal of the morphin. The doses must be given at exact intervals, about four a day, and each day less. The greatest difficulty is experienced in the final withdrawal. Atropin in sufficient doses, to produce extreme dryness of the mouth and throat and other physiological effects, is an aid at this time. During the treatment the patient should receive the most nourishing food at regular intervals, and of a character depending upon the condition of the digestion. The aching pains, sleeplessness, and general nervousness that usually occur toward the end of treatment are best relieved by hot baths and massage. Trional may be required at night in doses of 20 or 30 grains (1.30—2.0). A new treatment has been advocated by Lott of Texas, and supported by Hare and others. It consists in the administration of large doses of hyoscin hydrobromate, even gr. ¼ (0.015) in each twenty-four hours, and the immediate withdrawal of the morphin. The patient often develops alarming symptoms, but recovers without a craving for the drug. Pettey has shown that the treatment is extremely dangerous in some cases, and that in another group it is efficient in much smaller dosage than recommended by Lott, providing the intestine be thoroughly evacuated by free purgation before its administration is begun. The treatment has not yet been extensively employed. After recovery the patient should remain for several months away from home. A change of residence is often advantageous in removing old suggestions of the habit.

COCAIN HABIT.

The cocain habit is becoming prevalent, especially among the negroes and lowest class of whites. It is most frequently taken in the form of snuff, sometimes hypodermically. Before its dangers had been recognized,

many individuals acquired the habit from the use of sprays, ointments, and solutions for the nose, throat, or eye. The effect of a large dose is often maddening, but prostration ensues, and the individual lies for several hours in an unconscious state. Hallucinations of sight and hearing are commonly induced. The pupils are dilated, nystagmus is common. The pulse is rapid and feeble. The continued use of the drug produces the utmost depravity. The appearance is not always distinctive. The inflamed and often ulcerated condition of the nose, the blackness of the tongue and teeth, the anemic appearance, and restlessness of the eyes will generally suggest the use of the drug.

Treatment.—The management of the case is practically the same as that of the morphin habitué.

CHLORAL HABIT.

This habit is acquired in much the same manner as that of morphin. It is less common than either morphinism or cocainism.

The effect of the drug is less exhilarating, and the ultimate effect is profound depression, anemia, and tremor of the hands. The patient is nervous, irritable, morose, and may finally become demented. Indigestion and diarrhea are common, the breath is fetid, and the tongue heavily coated. Erythema and other cutaneous eruptions are common, the general integument is dry and blanched. As in other habits, the moral sense is obtunded.

The treatment consists in the withdrawal of the drug either gradually or at once, and the administration of bromids in large doses, hyoscin, and tonics, particularly strychnin and iron. The treatment is more easily accomplished in an institution for the treatment of inebriety.

LEAD-POISONING.

PLUMBISM, SATURNISM.

Etiology.—The disease may be produced by the slow intoxication of the system with lead. The disease occurs most frequently among artisans—those handling lead in any form, from the smelter to the painter and glazier. Miners are seldom affected. The lead may be absorbed through the respiratory passages, the digestive tract, or the skin. In the smelting of the ore, the grinding of white lead, and the mixing of paint, the poisoning arises probably both from the inhalation and swallowing of the dust or fumes. Among painters, glaziers, plumbers, and the like, it is largely a matter of carelessness in eating with unwashed hands. Poisoning sometimes results from drinking water, wine, or cider which has passed through new lead pipes or that has been stored in lead-lined tanks. Women are often very susceptible to the poison, and have been affected through the use of cosmetics, hair-dyes, false teeth, or by biting lead-dyed silk thread.

Morbid Anatomy.—The lead becomes deposited more or less generally in the soft tissues of the body, but especially in the muscles, nerves, and mucous membranes. Slow elimination takes place through the skin, kidneys, liver, and salivary glands. The muscles become pale, atrophied.

and sometimes indurated with fibrous tissue. Parenchymatous neuritis is also found most markedly in the peripheral ends of the nerves, and the nerve-endings in the muscles are degenerated. Sclerosis of the arteries, liver, and kidneys is found in advanced cases.

Symptoms.—The manifestations of lead-intoxication usually follow long exposure, but in some instances they have developed after exposure of only a few weeks, or even of only a few days' duration. Rapid poisoning is more common as a result of the inhalation of the fumes of smelting-furnaces, the dust from the grinders and mixers, or that from sand-papering in paint-shops. The symptoms may be either acute or chronic in character.

Acute Symptoms.—Cases are occasionally encountered in which the violence of the poisoning resembles that caused by the taking of a large dose of one of the soluble salts of lead, intense pain in the abdomen, vomiting, and diarrhea. As a rule, however, the more rapid intoxication is shown by a rapidly developing anemia, peripheral neuritis, sometimes accompanied with convulsions and delirium. Severe gastrointestinal symptoms are equally common. Obstinate constipation develops, and the patient is suddenly seized with a violent cramp in the abdomen (painters' colic). The wall of the abdomen is usually retracted, and there is a feeling as though the intestine was being twisted into a knot beneath the umbilicus. The paroxysm may continue almost constant for several hours, or it may be intermittent. In the intervals there are moderate pain and tenderness. Vomiting sometimes occurs. The temperature may be subnormal. The urine is usually scant and albuminous. Such attacks may recur at intervals for months and years, especially when the patient continues to work in lead. Death may, however, occur within the first two weeks, rarely even in the first attack, especially in an individual who has been overwhelmed by a short exposure to lead. Acute lesions of the central nervous system are not common, but hemiplegia has been attributed to an exposure of only three days.

Chronic Symptoms.—The most typical symptoms are those of a chronic character, the most distinctive of which are anemia, paralyses, the deposit of lead in the gums, and encephalopathies.

(1) *Anemia*, or the saturnine cachexia, is characterized by emaciation, deep pallor, sometimes a yellowish hue, and dryness of the skin. The blood-count shows a decrease of the red corpuscles seldom reaching 50 per cent, with corresponding reduction of the hemoglobin and a granular degeneration of the erythrocytes.

(2) A *blue line* in the gums, which, when present, is one of the most valuable diagnostic signs. It is due to the formation of lead sulphid, and is best seen along the margin of the lower gum as an indigo-blue line, which cannot be removed by cleansing. It usually forms early, and may persist indefinitely or it may shortly disappear.

(3) *Lead-Palsy.*—Several forms of lead-paralysis occur as a result of a peripheral neuritis. The most common is: (*a*) That known as wrist-drop, or the antibrachial type. When the arms are extended, the hands and fingers droop and cannot be raised through the action of the extensor muscles. It is due to affection of the musculospiral nerve. Less frequent forms are: (*b*) The brachial, in which the scapulohumeral

is involved, producing paralysis of the deltoid, biceps, brachialis anticus, and rarely of the pectorals. It may follow wrist-drop, but is occasionally a primary affection; (*c*) The Aran-Duchenne form, which may closely resemble poliomyelitis anterior chronica, affecting the small muscles of the hands, and producing marked atrophy, especially of the thenar and hypothenar eminences. In some instances the muscular atrophy is the primary change; (*d*) the peroneal form, affecting the muscles of the lower extremities, especially the lateral peroneals and extensors of the big toe. The steppage gait is produced. (*e*) A rare form in which the adductors of the larynx are involved.

Cramps sometimes occur in the affected muscles or in the flexed joints (lead-arthralgia), and tremors, increased by muscular effort, are not unusual. Sensation may not be altered. Rarely there is a general paralysis which slowly or rapidly extends to all the muscles of the extremities, resembling an ascending spinal paralysis. The diaphragm may be involved, with fatal result. A febrile form also has been recognized. The electrical reaction of degeneration is usually present.

(4) *Cerebral Symptoms* (Lead-Encephalopathy).—These may be purely functional or they may depend upon structural lesions, particularly endarteritis of the cerebral vessels. Manifestations of a hysterical nature are common in women; convulsions may occur, or epilepsy may develop. Acute delirium with hallucinations may occur independently or alternating with convulsions.

(5) *Arteriosclerosis* is frequently produced, especially in the kidneys, and hypertrophy of the heart may follow it.

(6) *Saturnine gout* is occasionally observed, especially in England. It is believed that the presence of lead favors the deposit of urates in the tissue of the joints.

Diagnosis.—The history of the case seldom leaves doubt in the diagnosis. Lead-colic is to be distinguished from that of volvulus or appendicitis, and from renal and hepatic colic. This is usually not difficult, on account of the retraction of the abdomen and the peculiar sense of constriction at the umbilicus, the absence of tumor or fecal vomiting and the subnormal temperature. The pain is not of the sharp, cutting character of hepatic and renal colic, and it is confined to the umbilical region, as a rule. Alcoholic neuritis is distinguished from that due to lead by the presence of sensory disturbances and the more usual affection of the lower extremities.

Prognosis.—This is favorable in a majority of cases. When the symptoms develop with violence after short exposure, it is often less favorable than in the more chronic cases. Atrophy of the muscles and the reaction of degeneration are unfavorable indications. The cerebral disturbances sometimes become permanent. Persistent treatment of the paralysis is often followed by surprisingly good results.

Treatment.—Prophylactic measures should be adopted by all workers in lead. Respirators are in a measure beneficial to those working in the smelting-works and where lead is ground or mixed. The greatest care should be taken in the cleansing of the hands, including the nails. The colic requires the hypodermic injection of morphin and the application of hot stupes. The constipation should be overcome by repeated dram doses of magnesium sulphate, which serves also to render the lead in-

soluble. The elimination of the lead from the tissues is favored by potassium iodid in doses of gr. v to x (0.30—0.60). It should not be given in the more violent cases, or until it is probable that all of the metal has been removed from the intestine. The action of the kidneys should be maintained by the drinking of a large quantity of water. For the paralysis, galvanic and faradic electricity may be employed, with massage of the muscles. Iron and strychnin are also indicated for the anemia and to restore muscular tone. The effect of the strychnin is more pronounced and more rapid when the drug is injected into the paralyzed muscles.

ARSENICAL POISONING.

Etiology.—The poison may enter the system either through ingestion or through inhalation. Poisoning is not infrequently developed among artisans in the manufacture of glazes and colors for paper and other fabrics. The red and green colors of wallpaper, artificial flowers, carpets, and draperies are the most likely to contain arsenic. Through the action of moisture or certain molds the poison may be liberated in the air. Poisoning has been contracted also through the sorting of playing-cards and other glazed paper, curing skins by the taxidermist, and in the manufacture of stained glass. Paris green is a frequent source of poisoning to farmers, who use it as an insect-poison. A case is occasionally met with in which the prolonged use of arsenic as a medicine has produced toxic effects. There is a great difference in individual susceptibility. The arsenic habit is sometimes contracted, especially by the Austrian peasants, who take as much as eight grains daily without serious effects.

Morbid Anatomy.—A degenerative peripheral neuritis is commonly found, associated with a similar change in the anterior horns of the spinal cord. A granular degeneration of the viscera is often produced, especially in the liver and kidneys.

Symptoms.—Edema of the eyelids, and conjunctivitis with headache, vertigo, attacks of nausea, mental depression or hysteria, are the symptoms which usually first attract attention. Anemia, with more or less emaciation, is a constant symptom. The mucous membranes of the nose and throat are generally dry or inflamed, especially if the poison has entered with the respired air. The skin is dry, the hair falls out, and there may be pigmentation or bronzing, eczema, herpes, or urticaria. The arsenic may be found in the secretions, especially in the urine. Albumin and casts are also present, and sometimes blood-corpuscles. Arsenical paralysis is sometimes developed; it is a progressive and painful neuritis affecting in the beginning the extensors and peronei muscles of the legs and foot, and sometimes involving later the arms. Tremors and contractures of the muscles and the steppage gait are usually produced.

Diagnosis.—Lead-neuritis is distinguished by the history, the blue line in the gums, and generally by the primary affection of the arms. Alcoholic neuritis can generally be recognized by the history and appearance of the patient; the face is flushed, not pale, and generally an acne rosacea appears.

Treatment.—The source of the poisoning must be removed; the occupation of the patient changed, perhaps. In early cases this is soon followed by recovery. The patient should drink freely of water in order to favor the elimination of the poison. Further than that the treatment is symptomatic. Galvanism and massage are beneficial for the neuralgia.

FOOD-POISONING.

BROMATOTOXISMUS.

Ptomain-Poisoning.—This is, perhaps, the most common form of food-poisoning encountered in our country. The nature of these poisonous alkaloids has been described on page 35. They may be formed in the food before it has entered the body, or afterward in the intestine. Some ptomains are extremely toxic, others are harmless. The poison is to some extent destroyed by cooking.

Meat-poisoning (kreotoxismus) is produced by eating pork, sausage of various kinds (botulismus or allantiasis), and occasionally from beef, veal, or mutton. Although the poisonous ingredient is in most instances due to a form of decomposition, it is not known to be identical with any of the ptomains. Its nature is, in fact, not known. Such poisoning has repeatedly followed the eating of canned meat, but in many such cases the poisoning has been attributed to a metallic poison. The meat of wild animals and birds is sometimes rendered poisonous by their food.

Symptoms.—The symptoms usually develop within six hours after the poison has been ingested. When due to a ptomain formed within the body they may be delayed as long as forty-eight hours or more. The first indication is often a chilliness or pronounced rigor, followed by nausea, vomiting, griping pains in the abdomen, with vertigo, extreme prostration, cold perspiration, and great thirst. Diarrhea is generally present and may be severe. Fever, 101° to 103° F. (38°—39.5° C.), is usual and may last for several days. The pulse becomes rapid and feeble. Dyspnea may be urgent. An intense pain is sometimes felt in the back between the shoulders. The tongue is heavily coated, often red at the tip and edges. The stools are offensive. Cramps in the muscles, twitching of the face-muscles, prickling and tingling of the skin, and psychical disturbances or stupor characterize some cases. Collapse usually attended with collapse, like that of cholera, with coldness of the surface and rapid shrinking of the tissues.

Milk-poisoning (Galactotoxismus).—This is due to the development of bacteria in milk, and it is most frequently encountered in summer. Milk, ice-cream, and cheese are often poisonous from the presence of the tyrotoxicon of Vaughan, or even more frequently from the presence of other poisons not yet identified. The symptoms are those of acute gastroenteritis, and do not materially differ from ptomain and meat-poisoning.

Fish-poisoning (Ichthyotoxismus) **and Shellfish**—(*a*) Two varieties are recognized. One is due to a poison secreted by the fish, especially the sturgeon of Russia, the barb of the Mediterranean, and the fugu of Japan. The other is produced by the bac-

teria of putrefaction. The symptoms are those of intense disturbance of the gastrointestinal tract, nausea, vomiting and diarrhea, sometimes accompanied with profound nervous prostration and weakness. Death sometimes occurs within a few hours.

(*b*) **Mussel-Poisoning** (Mytilotoxismus).—This form of intoxication is attributed by Brieger to a ptomain found chiefly in the liver of the mussel; cooking does not destroy it. The symptoms are acute and often fatal. They are almost solely due to the effect of the poison on the nervous system, and consist of numbness, extreme weakness, with rapid, feeble pulse, dilated pupils, subnormal temperature, and finally collapse. Vomiting and diarrhea occur in some cases. Owing to idiosyncrasy, some persons are poisoned by eating oysters or clams. In such cases there is usually gastrointestinal disturbance, and often an eruption of erythema or urticaria.

The ***treatment*** of all these conditions is alike. Vomiting and diarrhea are to be encouraged, until it becomes evident that they no longer serve a beneficial purpose in removing the poison. In case they do not occur, the stomach should be washed out or an emetic administered, followed by a purge. The further treatment is symptomatic. Stimulants are generally required for the prostration and feebleness, morphin to arrest the diarrhea and quiet the nervous manifestations.

5. **Grain-Poisoning** (Sitotoxismus).—This was formerly of frequent occurrence in some parts of Europe, but it is almost unknown in the United States. It may occur under several forms:

(*a*) *Ergotism* is due to the use of meal or flour made from spurred grain, a condition produced by the fungus claviceps purpurea. Two forms of ergotism are recognized, one gangrenous and attributed to the action of sphacelinic acid, the other nervous and attributed to cornutin. In the gangrenous form the necrosis affects the fingers and toes, occasionally the ears and nose. In the nervous form the patient complains for a week or two of headache and weakness, and is then seized with severe cramps and contractures of the muscles. The arms are flexed and the legs and toes are extended. The spasms recur at variable intervals and sometimes last for several hours, or even many days, at a time. Delirium or melancholia often develops, and the condition may pass into dementia. Convulsions are not uncommon. A sclerosis of the posterior columns of the spinal cord, not unlike that of locomotor ataxia, is sometimes produced. Preceding the development of the more pronounced symptoms in either form of the disease, the patient often experiences a sensation of itching or tingling in various parts of the body, especially in the fingers and toes.

(*b*) *Lathyrism* (Lupinosis).—A form of poisoning produced through the adulteration of flour with the chick-pea vetch (Lathyrus sativus). It has occurred for the most part in France, Italy, Algiers, and India. The symptoms are much the same as those of the convulsive form of ergotism.

(*c*) *Pellagra* (maïdismus), poisoning caused by the eating of fermented, unripe maize or Indian corn. It is seldom seen in the United States. The symptoms are indigestion, weakness, insomnia, sometimes salivation and diarrhea, with an erythematous eruption, followed by dryness of the skin, sometimes with desquamation or the development

of furuncles. In the more severe cases pronounced nervous and mental disturbances may be developed, as a gradual paralysis of the legs, melancholia, or a suicidal mania. The disease may last several months and end in recovery; or, through progressive emaciation and debility, it may terminate fatally.

The *treatment* consists in a change of diet and the employment of tonics. The addition of salt to the meal is said to counteract the poison.

Beriberi has been attributed to poisoning with fermented rice. (See p. 98.)

Mushroom-Poisoning.—Two forms of poisoning result from the eating of poisonous fungi, one acting upon the gastrointestinal canal, the other on the nervous system. The gastrointestinal form is characterized by violent vomiting and purging, intense thirst, prostration sometimes amounting to collapse, and a peculiar sense of constriction of the throat. Fever is sometimes present. In the nervous cases the poisoning is usually of a narcotic character. The individual is first seized with a vertigo, confusion of vision, and muscular weakness. Rigidity and spasms may supervene. He then becomes drowsy, and often passes into a comatose state, from which he cannot be aroused. Recovery is the rule, but many fatal cases have been recorded. The treatment is the same as that of ptomain-poisoning.

SUNSTROKE.

INSOLATION, HEATSTROKE, THERMIC FEVER, SIRIASIS, COUP DE SOLEIL.

Definition.—A condition produced by exposure to excessive atmospheric temperature.

Etiology.—Any influence which reduces the power of resisting the influence of heat may be a predisposing cause of sunstroke. Prominent among such influences are fatigue, privation, loss of rest, overeating, and, above all, indulgence in alcohol. A previous attack increases the susceptibility. A heavy moist atmosphere favors the action of the high temperature. The direct cause may be exposure to the sun's rays or exposure to high atmospheric temperature without the action of the sun's rays. The former gives us one, the latter two forms of prostration.

1. **Sunstroke** proper usually occurs in individuals who are engaged at hard labor in direct sunshine, as farmers, masons, bricklayers, and other outdoor laborers. The cases are most frequently seen in considerable numbers when a hot wave passes over the country, and when the intense heat is accompanied with a high degree of atmospheric humidity.

2. **Heatstroke,** or thermic fever, is produced by a high temperature without the direct action of the sun's rays. It attacks especially those who are confined in close apartments, as engineers, firemen, molders, glass-blowers, laundresses, and cooks. Its occurrence is much more frequent, however, among those who drink freely of beer and whisky. While sunstroke occurs only in the daytime, thermic fever may occur at any time of day or night.

3. **Heat-Exhaustion.**—This form of prostration occurs under the same conditions as the preceding, but the results are often different, a subnormal temperature and collapse being not unusual.

Morbid Anatomy.—Rigor mortis develops unusually early and putrefactive changes begin almost immediately. Ecchymoses and extravasations of blood are found in the skin and serous membranes. The left ventricle is usually firmly contracted and the right dilated. Intense venous engorgement is found in the cerebrum and in the cerebrospinal meninges, sometimes also in the lungs and other viscera. The conjunctivæ are congested. Degenerative changes may be found also in the liver and kidneys. The blood is fluid and dark, and the corpuscles show no tendency to form rouleaux. Van Gieson found the neurons of the entire neural axis in a state of parenchymatous degeneration similar to that produced by autointoxication, and it has been suggested that the condition is one of autointoxication superinduced by excessive heat. The suggestion has also been made that it is an acute infection. The toxicity of the urine is increased, and the sweat and blood-serum become toxic to animals.

Symptoms.—The onset is generally sudden; occasionally, however, there are such premonitions as dizziness and pain or fullness in the head. The symptoms are usually classified to correspond to types of prostration; but they differ for the most part in severity, and in the presence or absence of fever. In mild cases the prostration may be complete, yet short of unconsciousness, accompanied with headache, vertigo, and sometimes a transitory delirium. The temperature rises to 101° or 102° F. (38.0°—39.0° C.) for a few hours, but later subsides and may become subnormal. The skin is often cold and may be moist. The pulse is accelerated, but feeble. Nausea and vomiting may occur. Recovery within a few days is the rule.

In severe cases the prostration is more extreme. The individual falls as though he had received a blow upon the head, and death is sometimes almost instantaneous, or it may be delayed for a few hours. This is generally observed in cases of true sunstroke, and it occurs in those who are compelled to disregard the premonitory indications of its approach. It is the form which overtakes the soldier on a forced march. Sometimes the attack is a little less sudden. The individual then becomes dizzy, weak, nauseated; there is a feeling of constriction of the head or an intense headache. The vision becomes colored (chromatopsia) or indistinct. A person in this condition sometimes wanders for several hours in a state of subconsciousness. Finally unconsciousness supervenes and he sinks into a profound coma. The temperature rapidly rises and often reaches 108°, 110° F. (42°—43° C.), or higher, hence the name thermal fever. The pulse is rapid and tense, or slow, soft, and feeble. The respiration is labored, often sighing, or it may become stertorous. The skin is generally dry and intensely hot. Sudden arrest of the perspiration is often a premonitory symptom. Temporary dilatation of the pupils is sometimes observed, but it is usually followed by extreme contraction. There is, as a rule, complete relaxation, but jerking of the muscles or convulsions may occur. In fatal cases the pulse becomes more and more feeble, the respiration quick and irregular, or of a Cheyne-Stokes character, and death occurs from failure of both circulation and respiration. Recovery is usually foretold by a return of consciousness, restoration of normal respiration and circulation, and a subsidence of the other symptoms. For a long time, however, some-

times for many years, the individual remains highly sensitive to heat, and, unless exposure be avoided, a repetition of the attack is apt to occur.

A continued thermic fever, Florida fever or country fever, often mistaken in the South and in tropical countries for malaria or typhoid fever, has been described by Guiteras.

Diagnosis.—The condition is readily recognized, as a rule, since the circumstances under which the prostration occurs and the appearance of the patient are distinctive.

Prognosis.—The mortality of severe cases is great. All depends upon the promptness with which treatment can be instituted and the energy with which it is carried out, providing the patient is robust and not too deeply alcoholic. The recovery is often incomplete, however, and the patient is left for a long time in a vulnerable condition with reference to heat, and his mental and physical strength are often much impaired. Insanity is occasionally a sequel.

Treatment.—The best prophylactic measure is temperance in all things during the torrid season. In mild cases little is required for the attack further than rest in a sheltered place, sponging with cold water, and fanning, if there be no breeze. Stimulants should be given if there is great weakness, and strychnin and aromatic spirit of ammonia are better than alcohol.

In severe cases the first indication is to reduce the temperature. The patient should be immediately placed in a bathtub containing ice-water and pieces of ice; he should be literally packed in ice. When this is impossible, the entire body should be rubbed with ice. An enema of ice-water will assist in reducing the fever. Patients in a moribund state are often saved by this method. In the country, until ice can be secured, the patient may be stripped in a sheltered place and sprinkled freely with water from a sprinkling-can or poured from buckets. Antipyretics may be employed in such cases, but their action is depressing and too slow to be relied upon.

DISEASES OF THE MUSCLES.

MYOSITIS.

Definition.—Inflammation of the muscles. Muscular rheumatism, or rheumatic myositis, is considered elsewhere. A form of myositis occurs in trichinosis, and suppurative myositis is observed in pyemia and in localized abscess-formations. Two forms remain for consideration, namely, infectious myositis, or poliomyositis, and myositis ossificans progressiva.

INFECTIOUS MYOSITIS.

Etiology.—Although the disease has been regarded as infectious, no specific micro-organism has been identified. Wagner has suggested that some cases are to be regarded as instances of acute progressive muscular atrophy.

Morbid Anatomy.—The muscles become swollen, firm, and friable, owing to the infiltration of small round cells and the proliferation of

the connective tissue. Hyalin and fatty degeneration are generally observed.

Symptoms.—The swelling and firmness of the muscles are readily recognized, and the surrounding tissues become edematous. Fever and enlargement of the spleen are usually present. Pain is produced by motion, and paresthesia may be detected. Dyspnea is produced when the muscles of respiration, especially the diaphragm (diaphragmitis), are affected, and deglutition may be interfered with.

Diagnosis.—Trichinosis is excluded by the absence of trichina in a fragment of muscle removed for examination. The presence or absence of eosinophilia has not been sufficiently established in either condition to be regarded as a positive indication.

Prognosis.—The disease usually terminates fatally after two or three months, from interference with respiration or the development of pneumonia. A purulent infiltration of the muscle, with consequent sepsis, has been observed.

Treatment is only palliative.

MYOSITIS OSSIFICANS PROGRESSIVA.

This is an exceedingly rare disease in which the muscles are ultimately converted into bony tissue. It is more frequent in boys about the age of puberty. The process usually begins in the neck and back, but other muscles are occasionally involved, including the heart. The muscles become swollen as in acute myositis. The skin becomes reddened and a slight fever develops. After the acute symptoms subside, the muscles remain indurated and the process of ossification gradually progresses. Motion is restricted from the first, and complete pseudo-ankylosis follows.

Prognosis.—The disease lasts several years and finally terminates fatally. No method of treatment has been found of any benefit.

MYOTONIA CONGENITA.

Thomsen's Disease.

Definition.—A congenital condition in which a tonic spasm of the muscles follows voluntary motion.

Etiology.—The disease is rare in this country, less so in Europe. It is hereditary and usually follows the male line of descent. The cause is not known.

Morbid Anatomy.—The muscle nuclei are greatly increased both in size and number, and the transverse striations are obscured by a finely granular degeneration. The intermuscular connective tissue is normal or proliferated. Changes in the terminal nerve-plates have not been fully demonstrated.

Symptoms.—The disease is first manifested in infancy. The muscles appear large, but their strength is deficient. A voluntary effort, a sudden change of temperature, or violent emotion induces a tonic contraction which relaxes but slowly, and interferes with the more delicate movements. The muscles of the face, eyes, larynx, and heart are not usually involved, but the diaphragm may be affected. The reflexes and

sensations remain normal. Brief galvanic or faradic stimulation produces normal reaction, but the maximum is reached tardily and the relaxation is slow. Contraction is excited only by closure of the circuit, not by opening it (Erb's myotonic reaction). Tapping upon the muscle also excites unusual contraction.

The disease is incurable, but it does not materially impair health. No treatment for it is known.

PARAMYOCLONUS MULTIPLEX.

This is an affection of unknown pathology in which clonic contractions occur constantly or in paroxysms, chiefly in the muscles of the extremities. By some writers it is regarded as a nervous affection allied to chorea or hysteria. A hereditary transmission has been noted in some cases. It is sometimes associated with degeneracy. It has been observed most frequently in males after violent emotional disturbances, as fright, or after prolonged exertion or great strain. In the more violent paroxysms the movements may be extremely rapid, and a tremor sometimes persists in the intervals. It is sometimes impossible to keep the patient in bed. The condition is not influenced by treatment, except in hysterical cases, or as the movements may be quieted by morphin and other narcotics.

SECTION X.

Diseases of the Nervous System.

DISEASES OF THE NERVES.

NEURITIS.

Definition.—Inflammation of a nerve. Neuritis may be localized, affecting a single nerve, or general, involving many nerves. General neuritis is called also multiple neuritis and polyneuritis.

Etiology.—The causes of *localized neuritis* are: (*a*) Traumatism, as by blows, wounds, laceration by fractures or dislocations, injury by the hypodermic needle or the fluid injected, prolonged pressure by muscle, tumor, or clamps used in operations; (*b*) cold, affecting particularly the facial or sciatic nerve; (*c*) extension of inflammation from contiguous parts; (*d*) toxins in the blood, or the unknown influences of such diseases as syphilis and leprosy.

Multiple neuritis is induced by: (*a*) The toxins of many o the acute infectious diseases which have been referred to; (*b*) the metallic poisons, lead, arsenic, mercury, or phosphorus, and such organic poisons as alcohol, ether, carbon bisulphid, ergot, anilin, and illuminating gas; (*c*) the undetermined intoxicant of beriberi; (*d*) cachectic conditions as in tuberculosis, cancer, marasmus, or anemia; and (*e*) overexertion or prolonged exposure to cold.

Morbid Anatomy.—The lesions may be interstitial or parenchymatous. In the former the inflammation may be limited to the perineural connective tissue, or it may extend into the nerve and induce a cellular infiltration between the bundles and a proliferation of the nuclei of the neurilemma. The nerve-fibers may be unaffected, or they may be destroyed by subsequent sclerosis.

In the parenchymatous form the changes are like those in a nerve-fiber which has been cut off from its neuron cell. The axis-cylinder and myalin become fragmented, and a granular fatty degeneration completes their destruction. The fat is then absorbed, leaving the neurilemma empty, but still showing proliferated nuclei. The muscles supplied by the affected nerve atrophy from loss of innervation, but a new fiber sometimes grows along the periphery and restores the communication.

Symptoms.—1. **Localized Neuritis.**—The symptoms are generally local. Heat, redness, and swelling of the skin are commonly observed over the affected nerve, which is extremely sensitive to pressure. An intense aching or lancinating pain is felt along the course of the nerve and the parts supplied by it. Localized sweating, an eruption of herpes,

muscular twitching, joint effusion, numbness, formication, impairment of tactile sense, and particularly a progressive loss of motion are commonly developed. Atrophy of the paralyzed muscles and contractures ensue; the skin, especially of the fingers, becomes glossy, and the nails brittle (trophic disturbances). The neuritis may extend upward from the peripheral to the larger nerve-trunks, especially in traumatic cases (ascending or migratory neuritis).

Diagnosis.—The pain is constant and intensified by pressure—not relieved as in neuralgia. The condition is further distinguished by paresthesia and a reaction of degeneration. The persistence of the reflexes and absence of incoordination excludes the peripheral pains arising from disease of the cord. The duration of the disease varies from a few days to several months. Recovery is not always complete.

2. **Multiple Neuritis.**—The attack begins abruptly, or after a short period of muscular weakness, with numbness or tingling. The symptoms may be acute or subacute.

(*a*) *Acute Febrile Multiple Neuritis.*—There is often a chill and rapid rise of temperature to 104° F. (40° C.), headache, vomiting, and muscular pains, rapidly followed by paralysis beginning in the feet or arms. Hyperesthesia and pain of variable severity accompany the paralysis. When the pneumogastric is involved, tachycardia and labored breathing are produced. More or less complete recovery is possible, after weeks or months, but the disease is often fatal.

(*b*) *Subacute* cases are marked by numbness and tingling, cramping pain, and great tenderness, with less rapid paralysis of the muscles. Fever may be present or absent. The reflexes are more or less completely lost. The hyperesthesia subsides and is followed by localized anesthesia. The condition generally becomes chronic, the paralysis persisting in the form of wrist-drop or foot-drop, especially in lead and alcoholic cases. The muscles later atrophy and become soft, and contractures develop. Slight reaction to the galvanic current without reaction to the faradic is characteristic. Trophic and vasomotor disturbances are common. The cranial nerves, particularly the optic, are sometimes involved in severe cases. In the alcoholic form, neuralgic pains, sensory disturbances, delirium, or convulsions often occur and may pass into confusional insanity. The sphincters may be temporarily paralyzed. Recovery is quite common, even in the worst cases, the symptoms gradually subsiding; but the steppage gait usually persists for several months. In walking, the foot is raised and swung forward in order to prevent the toes from striking the ground. Recurrence is not uncommon in alcoholic cases, and occasional in those due to lead.

Diagnosis.—The combination of wrist-drop and foot-drop, or the latter condition alone, is highly characteristic of multiple neuritis. These symptoms, in the absence of inco-ordination, exclude locomotor ataxia. *Diffuse myelitis* is more febrile. The cutaneous anesthesia and muscular atrophy are more pronounced and more rapid; trophic disturbances and sphincter paralysis more common, and a girdle pain may be present. *Acute ascending paralysis* is attended with less atrophy and sensory disturbance, and the electrical reactions persist.

Treatment.—The patient should be given complete rest. The affected

should be bandaged on splints in such a manner as to permit applications of chloroform liniment, poultices, or lead and laudanum to relieve the pain. In alcoholic cases the stimulant should be withdrawn gradually, as a rule. Care must be taken to prevent the formation of bedsores. Later, massage and the interrupted galvanic current are of great benefit. The contractures must be overcome by passive motion and extension. Strychnin and small doses of arsenic assist in the restoration of the muscles. The results of persistent treatment are sometimes remarkable.

NEUROMATA.

The tumors which affect the nerve-fibers are divided into two groups, true neuromata and false neuromata. The former are composed of nerve-fibers, rarely of ganglionic cells; the latter of fibrous, gummatous, malignant, or gliomatous tissue. They may result from injury in surgical operations, from diathetic disease, as leprosy, or they may rarely be hereditary. Their favorite seats are in the skin, the subcutaneous tissue, the amputation stump, or along the course of the nerve. Multiple fibroneuromata of the skin are known as *fibroma moluscum*, those growing on the terminal filaments of the sensory nerves as *tubercula dolorosa*.

The ***symptoms***, in addition to the presence of small nodules, are pain and tenderness relieved by pressure on the proximal side, paresthesia, or anesthesia. Constant twitching is sometimes present. In some cases subjective symptoms are absent. The treatment is surgical. Excision is usually followed by complete relief, except in the amputation neuromata, which are liable to recur.

DISEASES OF THE CRANIAL NERVES.

The cranial nerves are subject to both functional and organic disturbances. In the former there is no anatomical alteration, the condition often being associated with hysteria, neurasthenia, or other neurotic affections; there may be a reflex irritation, or the nerve may be compressed by inflammatory exudates or tumors. The organic lesions include neuritis and degenerations, which may be situated at the peripheral termination, along the tract, or in the cerebral centers. The central lesion may be syphilitic, inflammatory, hemorrhagic, or septic; it may be due to meningitis of either form, caries, or fracture of the skull-bones. The result is a temporary or permanent increase, diminution, or loss of function.

I. OLFACTORY NERVE AND TRACT.

The first nerve may be affected in its nasal origin, the bulb, tract, or central nuclei. The result is a disturbance of the sense of smell. The chief causes of peripheral disturbances are acute or chronic rhinitis and ozena; the causes of deeper-seated disease are meningitis, especially tubercular; tumors of the hippocampus; caries of the ethmoid, or abscess

in the frontal lobe. The functional disturbances are **anosmia, hyperosmia,** or **parosmia.**

Anosmia is a loss of the sense of smell. This may be purely functional as in hysteria; it is temporary in acute rhinitis and when due to polypi, and more permanent in chronic rhinitis or destructive disease of the nerve-tract. It sometimes results from blows upon the head, arrest of nasal secretion in paralysis of the fifth nerve, and rarely from atrophy in locomotor ataxia. It is occasionally due to congenital lack of development.

Hyperosmia is an abnormal acuteness of the sense of smell, usually associated with hysteria or neurasthenia. Parosmia is a perverted sense of smell, observed in the same class of patients or in the insane, and sometimes as an aura in epilepsy.

Treatment.—The treatment of these conditions is directed to the removal of the cause, but, even when this can be accomplished, the result is not always satisfactory.

II. OPTIC NERVE AND TRACT.

Disease of the optic nerve and tract produces visual disturbance, partial or complete blindness. Retinitis and neuroretinitis are of greatest importance to the general practitioner, from the fact that their recognition is often a valuable factor in the diagnosis of chronic nephritis, especially the interstitial form. They are met with also in profound anemia, leukemia, purpura, and syphilis. The changes in the retina are, for the most part, hemorrhagic or sclerotic. The hemorrhages are found in the layer of nerve-fibers and often follow the course of the blood-vessels. The retina may be much swollen and the disk obscured. White opacities are commonly seen, which are due to fatty degeneration of the retina, extravasation of leucocytes, or fibrous induration.

Optic neuritis, papillitis, or choked disk, is also encountered in chronic nephritis, even more frequently than neuroretinitis, as a result of pressure by a tumor within the cranium, or in connection with simple or tubercular meningitis. The condition is recognized from the peculiar appearance of the disk. The edges are opaque and striated and the center congested, both appearances increasing as the disease progresses. The depression disappears with the swelling, and hemorrhages often take place. The congestion may subside in the less severe cases, but atrophy of the nerve often ensues.

Atrophy of the optic nerve is generally due to pressure neuritis associated with tumors, but it may be a congenital condition. It sometimes occurs in diabetes, locomotor ataxia, acute infectious diseases, or other toxic conditions. The field of vision is reduced, the color sense impaired, and total blindness may ensue.

Functional disturbances of vision arise from many causes: (*a*) Partial or complete blindness (amaurosis) sometimes occurs in hysteria and other neurotic states, or as a result of violent emotional excitement. (*b*) Toxic amaurosis, usually of a few days' duration, sometimes follows poisoning by lead, alcohol, quinin, or tobacco. (*c*) Amblyopia (dimness of vision) is a more common result of excessive use of tobacco. It is progressive, and, if the habit is persisted in, frequently leads to

organic change with atrophy of the disk. A central dark spot (scotoma) to tests with red and green is always found. (*d*) Night-blindness (nyctalopia), in which objects visible by day cannot be seen in a dim light, and the opposite condition, hemeralopia, in which objects cannot be distinctly seen in a strong light, are among the curiosities of visual disturbance. (*e*) Retinal hyperesthesia is a rare condition usually seen in hysterical women. (*f*) Photophobia is a common condition in the initial stages of the acute infections and may be met with in neurotic states. (*g*) Hemianopia, in which one-half of the visual field is obscured, may be functional, associated with hysteria or migraine; but in some cases it is organic and due to lesions in the optic chiasm, or anywhere between this and the cortical center. It is sometimes due to the pressure of a tumor in the cerebral cortex. The outer half of both fields is usually affected (temporal hemianopia) in lesions of the central portion of both chiasms; the inner portion (nasal hemianopia) is affected in lesions of the lateral region of both chiasms. Lateral hemianopia is produced by lesions between the chiasm and lateral geniculate body, lesions of the central fibers of the nerve between the geniculate bodies and cortex, and by lesions of the cuneus. Lesions of the angular gyrus and injuries of the brain in its vicinity may produce hemianopia, crossed amblyopia, or mind-blindness, in which things are seen, but cannot be named.

Treatment is beneficial only when the cause can be removed, as in tobacco, amblyopia, some cases of injury or tumor, and in cases due to syphilis, malaria, anemia, or nephritis.

III. OCULOMOTOR NERVE.

Lesions of this nerve may be situated in the nucleus of origin, or along the course of the nerve. Either paralysis or spasm is produced. In character the lesion may be a neuritis, irritation from the pressure of a gumma or other tumor, or meningitis. The central lesions are usually associated with those of other ocular nerves.

Paralysis due to a central lesion is generally associated with paralysis of other muscles, producing general ophthalmoplegia. When not associated, there is generally a neuritis or other lesion in the course of the nerve, as sometimes occurs in diphtheria or locomotor ataxia, or in meningitis, gumma, or aneurism. Complete paralysis of this nerve includes all the muscles of the eye, except the superior oblique and external rectus, producing ptosis, divergent strabismus with double vision (diplopia). Partial paralyses involve only the levator palpebræ and superior rectus, the ciliary muscles (cycloplegia), or the iris (iridoplegia). Iridoplegia may be manifested in three ways. (*a*) The pupil fails to contract with accommodation for a near object. (*b*) There is loss of the iris-reflex. If a bright light is flashed into the eye while the individual is looking at a distant, dark object, the iris fails to contract. This loss of reflex without loss of accommodation-contraction is known as the Argyll Robertson pupil, an important element in the diagnosis of locomotor ataxia. (*c*) Loss of the skin-reflex, in which the pupil fails to dilate when the skin of the neck is pinched.

A periodically recurrent complete oculomotor paralysis is sometimes observed. Anisocoria, or inequality of the pupils, is sometimes a feature of tabes or paresis, but it may occur in healthy persons.

Spasm of the muscles supplied by the third nerve, particularly of the internal rectus and levator palpebræ, is sometimes met with in meningitis and hysteria. A rhythmical involuntary clonic spasm of the eye, known as nystagmus, is not infrequently seen in meningitis and other brain-diseases in children. It is sometimes met with in adults, particularly in albinos, independently of nerve-disease.

IV. FOURTH NERVE.

This nerve is subject to lesions of the same character as the preceding. Paralysis impairs the downward motion of the eye, and is manifested by strabismus and double vision when the patient looks downward.

V. FIFTH OR TRIGEMINUS NERVE.

Neuritis is not frequent in this nerve. Its intracranial lesions are more commonly due to meningitis, sclerosis, hemorrhage, or tumor of the bones. It is sometimes due to injury by caries or fracture of the skull. Its extracranial portion may be involved in tumors or injury. The results are paralysis, and sensory or trophic disturbances.

(*a*) **Motor Portion.**—The paralysis affects the temporal and masseter muscles, interfering with mastication. The jaw deviates toward the affected side when opened, owing to weakness of the pterygoid muscle.

Spasm of the muscles of mastication, trismus, or lock-jaw, may be tonic or clonic, and a common symptom in general convulsions. It sometimes follows reflex-irritation in the mouth or teeth, caries of the jaw, or exposure to cold. It also follows organic disease near the motor nucleus of the nerve. The tonic form of the spasm is a distinguishing feature of tetanus, is occasionally seen in tetany, and may complicate hysteria. Clonic spasm, producing chattering of the teeth, is usually seen in hysteria or chorea, but occasionally as an independent affection.

(*b*) **Sensory Portion.**—Lesions of this portion produce tingling or anesthesia of the parts supplied, the half of the face, side of the head, and the mucous membranes of the lips, tongue, hard and soft palate, nose, and conjunctivæ. Arrest of the nasal secretion affects the sense of smell; the sense of taste may also be modified, and trophic changes not infrequently ensue in the affected parts. Opacities and ulcers of the cornea are common, persistent and painful eruptions of herpes often appear on the face, the teeth become loose and may drop out. The supraorbital reflex is usually absent.

Gustatory Disturbances.—The sense of taste is generally, though not always, lost in the anterior two-thirds of the tongue when this nerve is paralyzed.

Treatment consists in removing the cause when this is possible. Mor-

phin may be required for the pain, and local applications are of benefit, especially when the condition is due to neuritis of the superficial portion of the nerve. Faradization and massage of the affected muscles are beneficial. Syphilitic cases require specific treatment.

VI. SIXTH NERVE (NERVUS ABDUCENS).

This nerve is subject to the same affections as the other motor nerves of the eye, especially in connection with syphilis, meningitis, and tabes. The result is paralysis of the external rectus muscle, producing internal strabismus, with inability to rotate the eye outward, and diplopia when the patient looks toward the affected side. When the lesion is in the nucleus, the opposite eye cannot be turned inward, and both eyes deviate from the side of the lesion.

Ophthalmoplegia.—This term signifies a chronic, progressive paralysis of the ocular muscles. The condition is a rare one, occurring in two forms, affecting the external or the internal muscles of the eyeball. Its usual cause is nuclear degeneration, but it sometimes arises from the pressure of a tumor or the exudate in basilar meningitis. Optic-nerve atrophy and cerebral symptoms may be associated with it.

Treatment of Ocular Paralyses.—Recovery sometimes occurs spontaneously after cessation of the cause or when this can be removed. The most successful treatment, as in all nervous diseases, is obtained in syphilitic cases. Conditions associated with locomotor ataxia resist treatment. When the onset is acute, local hot applications are sometimes beneficial. Tonics, especially arsenic and strychnin, are sometimes useful.

VII. FACIAL NERVE.

Paralysis (Facial Paralysis, Bell's Paralysis).—Facial paralysis may arise from: (*a*) Lesions of the cortex, including fibers in the corona radiata or internal capsule, in connection with cerebral hemorrhage or softening, tumors, abscesses, or chronic inflammation; (*b*) the effect of the toxins of infectious diseases, particularly of diphtheria; (*c*) lesions in the nerve, most frequently caused by exposure of the face to an intensely cold wind; injury at its point of emergence by meningitis, gummata and other tumors, or fracture of the base, and lesions in the Fallopian canal from suppuration of the middle ear or caries. The nerve is sometimes severed in surgical operations or compressed by the obstetric forceps in delivery, causing, as a rule, only temporary paralysis.

Symptoms.—Facial paralysis is more frequent in children or young adults, rarely congenital. In peripheral cases, especially after exposure to cold, the onset is sudden, and all the branches of the nerve are involved. The patient experiences a sense of numbness or tingling on the affected side and is unable to produce voluntary movement of it. The eyelids remain open, and the lips are drawn slightly toward the other side. The tears escape from the eyes and the saliva from the mouth. Difficulty is experienced in drinking, mastication, pronouncing the labials, and in expectorating. The patient cannot purse the mouth as in whis-

tling. The reflex and normal electrical reaction are lost. The skin is smooth and the forehead cannot be wrinkled. The skin may be edematous, and an eruption of herpes sometimes develops at the angles of the eyes and on the lips. The paralysis is best demonstrated by causing the patient to laugh. Absence of the supraorbital reflex serves to differentiate a peripheral facial paralysis from one of central origin.

Facial diplegia, or paralysis of both sides, is a rare form beautifully illustrated by Thompson, usually due to lesions at the base of the brain, in the pons, or to simultaneous lesions in both nerves, as in diphtheria or middle-ear disease.

When facial paralysis is associated with hemiplegia, the reflexes persist, and cerebral symptoms are usually present. The paralysis is most marked in the lower portion of the face. When the lesion is in the Fallopian canal, there are deviation of the uvula, impairment of the sense of taste (probably due to injury to the nerve of Wrisberg), and evidence of middle-ear disease. Paralysis of central origin is always accompanied by involvement of other cranial nerves. Paralysis of the stapedius muscle is indicated by increased sensitiveness to musical sounds, often amounting to pain.

Prognosis.—The prognosis is good, except in cases due to destruction of the nerve by injury or suppuration. The recovery is sometimes slow and not always complete. Contractures often develop with the restoration of motion.

Treatment.—Cases due to cold are often benefited by the application of poultices or hot fomentations. Stronger counter-irritation may be made with blisters or the thermocautery over the mastoid. If due to middle-ear disease, this must be given drainage and proper treatment. When syphilis is suspected, potassium iodid and mercury should be prescribed. Calcium sulphid is indicated in all cases associated with suppuration, unless syphilitic. Later, faradization and massage of the muscles are of great service.

Spasm may affect any or all of the muscles innervated by the facial nerve. It may be primary or secondary, sometimes, perhaps, reflex, and may affect one or both sides. When organic, the lesion is generally central. Various muscles or groups of muscles are intermittently thrown into contraction, under excitement, fatigue, or emotion. The orbicularis and the muscles in its vicinity are most commonly affected. Closely allied to this is the habit spasm or convulsive tic (tic convulsif) of children, sometimes persisting through later life.

The treatment consists first in the removal of any recognized irritation. A painful spot sometimes exists and should be made the seat for the application of blisters, the thermocautery, or methyl-chlorid spray.

VIII. AUDITORY NERVE.

Central lesions are rare. The cochlear branch is more frequently affected in its course than the vestibular. Either branch may, however, be the seat of inflammation or neuritis, the latter especially in diphtheria and cerebrospinal meningitis, or it may be involved in tumors, hemorrhage, or fracture at the base. A primary degeneration sometimes occurs in locomotor ataxia.

The *cochlear portion* may be affected in its cortical center, producing word-deafness, in which words are not comprehended, although heard; or the central auditory path may be affected, producing deafness. In most cases affecting the hearing the lesion is an extension of inflammation from the middle ear. Hyperesthesia (hyperacusis), or abnormally acute hearing, sometimes results, but partial or complete deafness is more common. The nerve is often affected also in the acute infectious diseases, or by the loud vibration in machine shops. Functional disturbances occur in hysteria and other nervous conditions. Hallucinations of hearing frequently arise in insanity and paresis.

Tinnitus is the term employed to embrace a large group of abnormal subjective sound-perceptions, as ringing, crackling, buzzing, and roaring. These may result from disease of the ear, obstruction of the Eustachian tubes, the pressure of wax on the tympanum, injury, or they may be associated with anemia, cardiac hypertrophy, or increased arterial tension due to other causes. Tinnitus sometimes occurs as an aura in epilepsy.

The *vestibular portion* is rarely affected. The distinguishing symptoms are vertigo, nystagmus, and a disturbance of the equilibrium of the head, due to loss of co-ordination.

Auditory vertigo, or Ménière's disease (labyrinthine vertigo), is a peculiar form of vertigo, associated with noises in the ears, vomiting, rarely with loss of consciousness, and followed by gradual loss of hearing in some cases. It has been attributed to inflammatory disease or hemorrhage of the labyrinth, and to organic changes in the auditory nerve, but little is known of its pathology. The symptoms occur periodically at intervals varying from a day to several months. The attack comes on suddenly, sometimes accompanied with other nervous disturbances.

Treatment.—In hysterical cases the bromids are beneficial; in neurasthenics, rest and tonics, with light exercise; in increased arterial tension, nitroglycerin; in syphilitics, specific treatment. Quinin and the salicylates improve some cases. The correction of errors of refraction sometimes relieves a vertigo attributed to auditory disease. The treatment of Ménière's disease is very unsatisfactory.

IX. GLOSSOPHARYNGEAL NERVE.

Primary diseases are rare. The nucleus is probably involved in cases of bulbar paralysis, and the trunk may be compressed by tumors or the exudate in meningitis. The sense of taste may be impaired in the posterior part of the tongue and palate, but it is not lost. The upper part of the pharynx becomes anesthetic and the middle portion paralyzed, rendering deglutition difficult.

X. PNEUMOGASTRIC (VAGUS) NERVE.

The nucleus, the intracranial or extracranial branches may be affected in the same manner as the other cranial nerves. The results are exceedingly various, owing to the wide distribution of the branches.

Pharyngeal Branches.—Paralysis may follow diphtheria or accompany bulbar paralysis, causing difficulty of deglutition, particularly when only one side is involved. The food may pass into the larynx, or it may be regurgitated into the posterior nares. Spasm occurs also in hysteria or under emotional disturbance (globus hystericus), and in both true and false hydrophobia.

Laryngeal Branches.—Affections of these branches produce paralysis of the vocal cords or of the adductor or abductor muscles, with stridulous respiration, cough, and hoarseness or huskiness of the voice, or complete aphonia and dyspnea. These may result from diphtheritic paralysis, pressure of a thoracic aneurism, mediastinal tumor, or goiter upon the recurrent laryngeal branch. Functional disturbances are common in hysteria, especially adductor paralysis, with complete aphonia. Spasm occurs in laryngismus stridulus, in the crises of locomotor ataxia, and in hysteria.

Pulmonary Branches.—We have no positive knowledge regarding the affections of these branches. That bronchial asthma is due to central lesions of this nerve in some cases is an old theory, lacking support. Disturbance of respiration of vagus origin is probably always accompanied with cardiac disturbance.

Cardiac Branches.—Motor, sensory, and trophic disturbances of the heart are believed to result from disease or irritation of these fibers. Bradycardia and tachycardia are examples of motor disturbances of this character. The nerve may also be involved in a multiple neuritis, and it is commonly affected by the toxins of the infectious diseases. The respiration is generally increased in rate when the action of the heart is accelerated. The sensations of fluttering and faintness associated with irregularity of rhythm are transmitted to the brain, it is believed, by the pneumogastric nerve. (See Cardiac Neuroses.) The more profound lesions of the cardiac branches produce fatty degeneration of the myocardium.

Esophageal and Gastric Branches.—Paralysis or spasm of the esophagus and various disturbances of gastric sensation, as pain, hunger, satiety, and the motor disturbances described under Neuroses of the Stomach, are attributable to changes in these branches.

XI. SPINAL ACCESSORY NERVE.

Paralysis may result from involvement of the nuclei or of the cord. The nuclei of the internal portion are involved especially in bulbar paralysis, those of the external portion in progressive muscular atrophy. Paralysis may result also from diseases of the spinal cord or meninges affecting the cervical portion, caries of the vertebræ, and injury. The manifestations are atrophy of the sternomastoid muscle of the affected side, with diminished power of rotating the head toward the opposite side, and partial paralysis of the trapesius, interfering with the lifting of the arm and the internal rotation of the angle of the scapula. The shoulder droops, and the supraclavicular depression is increased. Bilateral paralysis of the sternomastoid in progressive muscular atrophy permits the head to fall backward. When the trapezii are affected, however, it falls forward.

Treatment.—Cases due to central lesion are seldom benefited by treatment. Disturbance due to pressure is sometimes relieved, especially if due to syphilitic formations.

Spasm (wry-neck, torticollis) may be congenital or acquired. The former is a stationary, fixed contraction; the latter may be tonic or clonic.

Congenital wry-neck is due to lack of muscular development or to injury during delivery, and is probably never of nervous origin. It affects the right side, as a rule, and often passes unnoticed for several years after birth. It is usually associated with facial asymmetry. The mastoid muscle is much shortened and hard. The trapesius is affected in rare instances.

Acquired torticollis may also arise from other than nervous influences, as from rheumatism (myositis), adenitis, caries, or neoplasms. The true spasmodic form occurs without recognizable lesions and in association with vascular and trophic disturbances.

A nodding spasm of the muscles of this group is occasionally seen in children and hysterical women.

Symptoms.—The head is rotated upward and away from the side of the lesion. The spasm, at first clonic, may become tonic. The facial nerve or the brachial plexus may also be involved, producing a combined spasm of the muscles supplied. In clonic cases the muscles become painful and sensitive, and in tonic cases much suffering is sometimes induced by the strong contraction. The muscles involved become large and firm.

Prognosis.—Recovery is possible, but it is unusual. The disease becomes chronic, or, if relieved, it recurs.

Treatment.—This is often unsatisfactory. The bromids, hyoscyamus, and the galvanic current have yielded good results in some cases. Recurrence is the rule. Surgical measures have been employed.

XII. HYPOGLOSSAL NERVE.

This nerve also is subject to nuclear lesions, usually bilateral, due to degeneration, especially in bulbar paralysis or locomotor ataxia; injury, or pressure by tumors; meningitis, gout, or lead-intoxication. In its course it may be affected by tumors, disease of the skull or cicatrices. Cortical lesions of the nerve are usually associated with hemiplegia. In either form, the tongue is affected, and speech is interfered with. In nuclear lesions the tongue undergoes atrophy, but in cortical disease this is unusual and the electrical reactions are retained.

Symptoms.—*Paralysis.*—This is usually unilateral, and the tongue, when protruded, deviates toward the affected side, except when the lesion is within the medulla, when it deviates toward the sound side. In peripheral lesions it is protruded toward the affected side. Speech is not greatly interfered with. Paralysis of the larynx and of the palate muscles of the affected side is associated with it. When a complete bilateral paralysis occurs, the tongue lies motionless on the floor of the mouth. Speech, mastication, and deglutition are difficult, but the senses of taste and touch are not interfered with. The tongue atrophies, the electrical reaction is lost, and a fibrillary tremor develops.

Spasm may affect one or both sides of the tongue, but it is rare, and, as a rule, associated with epilepsy, chorea, hysteria, or facial spasm. Stuttering is sometimes due to it. Cases have been observed in which a paroxysmal clonic spasm causes the tongue to be rapidly thrust out and in, even during sleep. Recovery is usual in all forms.

DISEASES OF THE SPINAL NERVES.

CERVICAL PLEXUS.

This plexus and its branches may be involved in neuralgia, paralysis, or spasm. Occipitocervical neuralgia results from exposure to cold, injury, especially that from carrying burdens on the neck, caries of the vertebræ, or tumors. The pain is situated in the back of the head, the neck and ear. Tender points are found between the mastoid and spine, above the parietal eminence, and between the sternomastoid and trapesius.

Phrenic Nerve Disease.—*Paralysis* may be due to a lesion in its origin or course, rarely to neuritis after diphtheria or lead-poisoning. It sometimes follows ascending myelitis. Sudden paralysis causes dyspnea and cyanosis; less rapidly developed, it is characterized by dyspnea on exertion, inability to draw a full breath and thoracic breathing due to paralysis of one or both halves of the diaphragm. The abdominal wall protrudes in expiration instead of with inspiration. The chief dangers lie in the development of bronchitis, hypostatic congestion, or edema of the lungs. The diagnosis is often difficult, and the condition may be confounded with the fixation of the diaphragm due to inflammation in its vicinity, as in pleurisy, peritonitis, or abscess.

Treatment is not successful, as a rule, except when the condition is due to neuritis.

Spasm.—The best examples of this are seen in torticollis, already described, and in hiccough.

Hiccough is caused by a sudden, intermittent contraction of the diaphragm, with sudden closure of the glottis. The afferent impression may be peripheral or central; the efferent impulse is through the phrenic nerve, accompanied, however, with an impulse through the recurrent laryngeal branch of the vagus. With reference to cause, the condition may be (*a*) irritative, as when the spasm is excited by swallowing hot food, disease of the lower extremity of the esophagus, gastric or intestinal indigestion; (*b*) inflammatory, occurring in gastritis, peritonitis, appendicitis, hernia, internal strangulation, or typhoid fever; (*c*) organic, in which the condition accompanies cancer of the stomach or intestinal tumor, gout, diabetes, nephritis, or other constitutional disease. These cases are the most persistent; (*d*) neurotic, occurring in hysteria, cerebral tumor, epilepsy, emotional disturbance, shock, or reflex from a peripheral irritation. The attacks vary in duration from a few hours to several weeks or months.

The prognosis is unfavorable only in cases complicating the more serious diseases.

Treatment: In the less severe cases many of the popular remedies are effective, as a drink of cold water, swallowing ice, salt, vinegar, sugar, or brandy, a sudden fright, or the induction of sneezing. Firm pressure in the epigastrium, a tight bandage

around the lower thorax, massage of the abdomen, an emetic or lavage of the stomach, will relieve some of the more severe cases. The cold pack is often promptly curative. Of internal remedies there are many, but they must be tried in succession in the worst cases and none is infallible. Of these the best are: Cocain, gr. 1-6 (0.01); spirit of chloroform, ʒ ss (1.8); codein, gr. ss (0.03), asafetida, and the bromids. Morphin may be employed hypodermically in the worst cases, but its effect is usually only transitory. Pilocarpin, nitroglycerin, apomorphin, and inhalations of chloroform or amyl nitrit have been curative in some cases. Pressure over the phrenic nerve and galvanism have been recommended, but it is not probable that the electric current can be made to reach the nerve.

BRACHIAL PLEXUS.

The nerve-trunks, before entering the plexus, may be compressed in the supraclavicular region by tumors, enlarged glands or aneurism, or injured by blows or inflammation. Neuritis is rare, but it may occur as an ascending inflammation from the peripheral branches. The most common lesion of the plexus is injury produced by subcoracoid dislocation of the humerus. Blows upon the shoulder and the use of an improper crutch are occasional causes of complete paralysis of the arm. The plexus of the infant may be injured by traction with the finger or hook in the axilla during delivery, that of the adult by methods for the reduction of shoulder dislocation. The result in all cases is paralysis of the muscles supplied by the branches of the plexus. The loss of power may be preceded by numbness, pain, or formication. The paralysis may develop after reduction of the dislocation and may persist notwithstanding the removal of the cause. The entire plexus or only one or more of its branches may be affected. The results are different with the affection of each branch. In lesions of the upper and middle portions of the plexus, the upper arm is paralyzed, and the ability to flex the arm is lost. Lesions of the last two cervical and first dorsal paralyze the hand and abolish extension of the forearm.

Lesions of the Nerves of the Arm.—(*a*) *The Long Thoracic.*—Paralysis of this nerve affects the serratus magnus. The angle and posterior border of the scapula stand out from the chest, especially when the arm is drawn forward and the movement of the shoulder is restricted. Neuralgia is often present from involvement of the sensory filaments.

(*b*) *Circumflex Nerve.*—The deltoid and teres minor are involved. The arm cannot be raised, sensation is impaired, and the deltoid atrophies.

(*c*) *Musculospiral.*—This is affected in wrist-drop from any cause, as lead, arsenic, or injury by pressure. Extension of the forearm, wrist, and fingers is lost. Anesthesia or formication may be present.

(*d*) *Musculocutaneous.*—In this, flexion of the forearm is lost, and sensation may be affected.

(*e*) *Ulnar.*—Flexion of the ulnar side of the wrist, ring and little fingers is impaired, the thumb cannot be adducted or the first phalanges flexed. In old cases the *main en griffe*, or claw-hand, is produced. Sensation is lost in two and a half fingers on the dorsal side, and in one and a half on the palmar side.

(*f*) *Median.*—The median nerve is seldom involved alone. The wrist cannot be flexed toward the ulnar side. Abduction of the thumb and flexion of the second phalanges and the distal phalanges of the first and second fingers are lost. The sensation is lost in the parts affected, and the thumb muscles atrophy.

LUMBAR PLEXUS.

Paralysis or spasm may affect the parts supplied, as a result of inflammation, psoas abscess, injury, caries of the vertebræ, enlarged glands, tumors, obturator hernia, fecal impaction, or hip-joint disease. Flexion of the thigh and extension of the leg are impaired, and pain or anesthesia may be produced. In spasm, the thigh is drawn up over the abdomen. When the obturator nerve is injured, as in parturition, the adduction of the thigh is impaired and the inner side is anesthetic. When the anterior crural is involved, as in a wound or dislocation of the hip, the extension of the knee is impaired, the inner side and front of the thigh and the inner side of the leg are anesthetic. Gluteal paralysis weakens the abduction of the thigh.

SACRAL PLEXUS.

This plexus is subject to lesions of the same character as those of the lumbar plexus. Neuritis is, however, more common, usually extending upward from the sciatic nerve. In paralysis the muscles supplied by the sciatic are affected. The flexors of the leg and all the muscles below the knee are involved. The outer half of the leg and the dorsum of the foot are anesthetic. The lesser sciatic nerve is seldom affected. Such involvement is indicated by paralysis of the gluteus maximus, producing inability to rise from a sitting posture without difficulty, and an area of anesthesia in the middle portion of the back of the thigh.

SCIATICA.

The greater sciatic nerve may be the seat of neuralgia or neuritis. The affection is more common in men, but women are not exempt. It is somewhat more frequent after the fortieth year, and many patients are the subjects of gout or rheumatism. It often follows exposure to cold and wet, and is therefore more prevalent during the winter and spring. Sometimes it follows an unusual strain, as in lifting. It may arise from the pressure of a tumor, impacted feces, the fetal head, or a hip-joint lesion. The pathological lesions in the nerve are a perineuritis and an interstitial neuritis, most pronounced at the notch and middle of the thigh, but sometimes extending upward to the plexus or to the cord.

Symptoms.—Pain predominates. It usually develops gradually and increases in intensity. Sometimes, however, the onset is sudden and sharp. At first confined to the middle part of the back of the thigh, the pain rapidly extends downward to the entire distribution of the nerve, along the entire leg and over the dorsum of the foot. It is usually a constant aching, burning, or boring, but sometimes paroxysmal. The leg is partially flexed; the patient, in attempting to walk, rests the weight on the toes. Extension and motion increase the suffering. The external temperature is reduced and there may be a subjective sense of coldness.

A herpetic eruption sometimes appears; formication is common, anesthesia of the skin unusual. The tender spots characteristic of neuralgia are generally found at the notch, middle of the back of the thigh, popliteal space, middle of the calf, sometimes back of the external maleolus, and on the dorsum of the foot. In protracted cases, the muscles atrophy, but the reaction of degeneration is not usually present.

Diagnosis.—Neuralgia is distinguished from neuritis by its occurring in younger patients, as a rule, its shorter duration, and the absence of atrophy. Pain due to the pressure of a tumor can be excluded by thorough examination of the pelvis. In lumbago, the pain is more confined to the loins, and there is tenderness on deep pressure. Hip-joint disease is recognized through the pain occasioned by motion of the joint. The pains of locomotor ataxia are bilateral; loss of the patellar reflex and other symptoms of the disease are present. The Achilles tendon-reflex (plantar flexion of the ankle) is weakened or obliterated in cases of sciatic neuritis.

Prognosis.—Ordinary cases recover within four or six weeks. Much depends upon the season and weather. Many cases persist until midsummer.

Treatment.—Rest is the most important factor. The patient should be kept in bed, in severe cases, with the leg extended on a splint. Hot water, poultices, the cautery, mustard, and blisters afford relief for a time. The same is true of ointments and liniments containing menthol, camphor, chloroform, or aconite. Deep injections of cocain or of distilled water, and acupuncture, have often proved beneficial. Chloroform and ether have been injected into the region of the nerve with benefit, but there is danger of producing abscess or aggravating the neuritis. Morphin should not be employed if it can be avoided, and then without the knowledge of the patient. After the case has persisted for a time, and especially if atrophy has begun, massage and galvanism are valuable agents. Nerve-stretching was formerly much resorted to, but the relief thus afforded does not, as a rule, prove permanent. Rheumatic patients are often benefited by the salicylates, gouty patients by colchicum. Removal to a warm climate is advisable in intractable cases.

Coccygodynia is a neuralgia of the posterior sacral branches usually due to injury, especially to fracture or dislocation of the coccyx. Palliative treatment consists in the introduction of anodyne suppositories, but in most cases removal of the coccyx must be resorted to.

DISEASES OF THE SPINAL CORD AND MENINGES.

SPINAL PACHYMENINGITIS.

Definition.—Inflammation of the dura mater of the spinal cord. Either the outer or inner layer may be affected, the latter more frequently. External and internal pachymeningitis are thus recognized. Inflammation of the connective tissue between the dura and the bony canal is known as peripachymeningitis.

Etiology.—External pachymeningitis is produced by injury, often accompanied with hemorrhage, tumors, but especially by tubercular or

syphilitic caries or abscess of the bone. It may be acute or chronic and affects an area corresponding to one or several vertebræ.

Internal pachymeningitis is usually an extension from the external or of inflammation in the vicinity. An independent, chronic, hemorrhagic form occurs, however, in alcoholic or paretic subjects, after convulsions in epilepsy or tetanus, or in connection with the hemorrhagic forms of the acute infections. It involves especially the cervical portion.

Morbid Anatomy.—In the external form, the dura is thick and firm. It may be adherent or covered with a caseous layer. When hemorrhage has occurred, the blood generally lies between the membrane and the spinal canal. It is sometimes referred to as spinal apoplexy. In the internal form the dura is also much thickened. In the hemorrhagic form there may be engorgement of blood-vessels, punctate hemorrhages, or large extravasations when the hemorrhage has resulted from the perforation of a vessel. A firm-walled blood-cyst is sometimes found filled with disorganized blood and pigment, in old cases.

Symptoms.—These are often indefinite, but they usually suggest myelitis. The manifestations are about the same in all forms. (1) There is intense neuralgic pain along the nerves whose roots are compressed, especially those of the arm and neck. Hyperesthesia, numbness, and tingling are usually present. Spasm of the muscles of the neck may occur. (2) Later, the hyperesthesia may give place to anesthesia, and the muscular contractions to paralysis. Atrophy of the muscles of the neck and arm and shoulder ensues, and, when the lesion is high enough to involve the musculospiral, there is wrist-drop. (3) Degenerative changes in the cord follow, in the worst cases, producing spastic paralyses with exaggeration of the reflexes and anesthesia, but without atrophy of the muscles. Bedsores and paralysis of the bladder are sometimes produced.

Diagnosis.—Difficulty may arise in the exclusion of amyotrophic lateral sclerosis, syringomyelia, and compression by tumors. From the first of these it is distinguished by the severe pain in the arm and neck; from the second, by the absence of such sensory changes as alteration of the temperature-perception. Tumors cannot always be excluded, for the pathological conditions are often identical.

Prognosis.—The disease is chronic, sometimes lasting from one to several years. Recovery is possible, but a fatal termination is to be expected from exhaustion, septic infection, or an intercurrent disease.

Treatment.—Counter-irritation with blisters or the cautery is sometimes of benefit. The ice-bag and internal administration of ergot reduce the inflammation. Potassium iodid is often of service.

SPINAL LEPTOMENINGITIS.

Definition.—An acute or chronic inflammation of the pia mater of the spinal cord.

ACUTE LEPTOMENINGITIS.

Etiology.—The disease most frequently occurs in a secondary relation to an acute infection, especially cerebrospinal meningitis or tuberculosis; or as an extension of the inflammation from a pachymeningitis or my-

elitis; rarely, perhaps, from exposure to cold. It may arise from injury of the spine.

Morbid Anatomy.—The lesions are those peculiar to an inflamed serous membrane, hyperemia with opacity, and a serofibrinous or purulent exudate, most marked on the posterior portion. The cord is occasionally involved in a meningomyelitis.

Symptoms.—The symptoms, with the exception of a wider range of severity, are the same as those of cerebrospinal meningitis (p. 112) and tubercular meningitis (p. 180). The differentiation from either of these conditions rests upon a recognition of the secondary nature of the disease and its dependence upon a previous infection, inflammation, or injury.

Treatment.—The treatment is that of cerebrospinal meningitis.

CHRONIC LEPTOMENINGITIS.

Etiology.—The chronic form of the disease usually develops independently of the acute, except in infants, and as a result of disease of the cord, syphilis, tuberculosis, or injury.

Morbid Anatomy.—The pia mater is opaque, usually much thickened, adherent, and often pigmented. The arachnoid is infiltrated with serum, and gummatous or tubercular lesions may be found.

Symptoms.—These are usually limited to rigidity of the neck, with sensory disturbances. Paralyses are developed when the nerve-roots are involved.

Treatment.—Potassium iodid should always be administered in the hope that the disease is syphilitic. Sirup of the iodid of iron is better in children and tuberculous cases. Hot and cold applications and douches are often beneficial.

AFFECTIONS OF THE BLOOD-VESSELS AND CIRCULATION OF THE CORD.

Hyperemia.—The occurrence of hyperemia, or congestion, of the spinal cord is largely hypothetical, for it is rarely, if ever, discovered after death except in connection with myelitis. When seen, it is usually confined to definite regions of the gray matter, which has generally a pale reddish color. There are no symptoms by which its presence can be diagnosticated.

Anemia.—Little is known of spinal anemia. In the most profound general anemias, chlorosis and leukemia, there are rarely any manifestations on the part of the spinal cord. There is no positive basis for attributing neurasthenia to anemia of the cord. When, however, a profound anemia is suddenly produced by the loss of a large quantity of blood, as in hemorrhage from the stomach or uterus, loss of sphincter control or complete paraplegia is not infrequently developed.

Embolism and Thrombosis.—Embolism of the vessels of the cord is rare, but may follow endocarditis. Thrombosis is more commonly met with as a result of endarteritis in either acute or chronic affections of the cord.

Arteriosclerosis develops in the spinal cord under the same conditions as elsewhere, particularly as a result of syphilis or as a senile change. The intima of the vessel is much thickened at the expense of its lumen. Miliary aneurisms are much less frequently produced than in the brain.

Hemorrhage into the Cord (Hematomyelia).—***Etiology.***—Hemorrhage into the substance of the cord is most frequently due to traumatism; it may, however, follow exposure to cold, overexertion, excessive coitus, and such affections of the cord as myelitis, syringomyelia, and tumors.

Morbid Anatomy.—A considerable portion of the cord is sometimes involved. The blood may be fluid or clotted, confined to the gray matter or it may extend to the meninges, after producing laceration of the cord substance. In cases of long standing there sometimes remains only a pigmented area from which the fluid portion of the blood has been absorbed.

Symptoms.—The symptoms are much the same as those due to the pressure caused by hemorrhage into the meninges, and at a later period resemble myelitis. Paraplegia or paralysis of all the members is produced in some cases. The diagnosis generally rests upon the suddenness of the onset, and the restriction of the symptoms to the cord.

The ***prognosis*** is nearly always fatal. In the few cases that recover, more or less permanent paralyses are generally left.

The ***treatment*** is the same as that of hemorrhage into the meninges.

CAISSON DISEASE.

DIVER'S PARALYSIS, COMPRESSED-AIR DISEASE.

Definition.—A condition usually characterized by paraplegia or general paralysis, which is induced in caisson and tunnel workers, rarely in divers, by too suddenly returning to the surface.

Etiology.—A pressure of three atmospheres (45 pounds to the square inch) or more is usually maintained in caissons and deep tunnels in order to exclude water. Caisson disease is produced by coming suddenly from a pressure of more than three atmospheres to that of the external air. It does not occur so long as the workman is under the pressure or after leaving a pressure of less than 45 pounds. Several explanations have been given for the symptoms, the most plausible being that they are due to the liberation of gases which have been retained in the blood on account of the high pressure. The muscular activity under pressure probably has its influence in some cases.

Morbid Anatomy.—Lesions are not always discovered in fatal cases. Punctate hemorrhages are sometimes found in the thoracic portion of the cord, and in some instances a condition resembling lacerations, and fissures, as if due to the liberation of gas, or the lesions of myelitis, may be revealed. Corresponding lesions are not found in the brain.

Symptoms.—The manifestations of the disease may appear immediately upon leaving the caisson or three or four hours after. Such premonitory symptoms as headache, giddiness, and neuralgic pains sometimes precede the attack for several days. In mild cases the only symptoms are agonizing pains in the knees, which subside within a few days. Sometimes the elbows and other joints are affected. In severe cases paraplegia develops,

with diminished sensation or hyperesthesia, increased patellar reflex, and swelling and soreness of the muscles. The joints do not swell. Headache, nausea, vomiting, vertigo, tinnitus, deafness, chilliness, and retention of urine are common accompaniments. The urine may be albuminous. Monoplegias and hemiplegia have been exceptionally observed, and in the worst cases there is general paralysis, followed in a few hours by death in coma.

Prognosis.—Most cases recover. The paralysis subsides in from one to three weeks.

Treatment.—Prophylaxis requires care in the passage from a high to low pressure. A series of chambers having graded pressures is provided in properly constructed caissons. The immediate return of the patient to the higher pressure relieves all symptoms, and he may then, in a short time, be gradually removed to the surface. When this cannot be done, the patient must be confined to bed. Morphin may be required for the pain; hot fomentations and massage ameliorate the suffering. Strychnin should be administered in the treatment of the paralyses.

MYELITIS.

ACUTE, GENERAL OR TRANSVERSE MYELITIS, WHITE SOFTENING OF THE CORD, INFLAMMATION OF THE SPINAL CORD.

Definition.—A localized transverse or diffuse inflammation of the spinal cord, followed by softening or sclerosis.

Etiology.—The disease usually occurs in males between the ages of 10 and 30 years. It most frequently follows exposure to cold and wet, but may result from severe trauma, as fracture of the spine, strong muscular strain, or from emotional disturbance. It occasionally develops upon one of the acute infectious diseases, as the exanthemata, rheumatism, septicemia, or smallpox. It is sometimes associated with syphilis or nephritis, and it may be induced by one of the metallic poisons or chronic alcoholism. Peripheral neuritis and meningitis are thought to be possible causes. A syphilitic history is frequently obtained.

Morbid Anatomy.—In *transverse myelitis* the disease is limited to a small vertical area extending entirely across the cord; when a larger portion is affected, it is termed *diffuse myelitis*. When several areas are involved in different parts of the cord, it is a *disseminated myelitis;* and when only the gray matter is affected, it is a *central myelitis*. The disease is most frequently situated in the upper dorsal region, next in the cervical, and then in the lower dorsal; it rarely affects the lumbar cord, except in the disseminated form. In some cases the cord shows little or no change upon ocular inspection, while in other, advanced cases it is extremely soft, almost diffluent, or greatly hardened, sclerotic, owing to the proliferation of interstitial connective tissue. The cord may appear swollen, the membranes congested, the fibers in a state of yellow atrophy (the color being due to pigmentation), or there may be fatty degeneration in cases of long standing. In central myelitis there is often red softening, occasionally also small cavities. Such new elements as the so-called Deiter's "spider" cells and granular fatty masses may be found. Amylaceous bodies are not uncommon. The nerve fibers and cells may

be swollen and disintegrated in the lesions and for some distance above and below them. Blood-corpuscles are sometimes present. Obliterative arteritis is observed in some cases.

Symptoms.—The onset may be acute with fever, subacute, or chronic. Convulsions may occur in children. The manifestations vary with the portion of the cord affected. Such premonitory symptoms as numbness, tingling, formication, and weight or girdle pains may be present, but, as a rule, the motor disturbances precede the sensory. The first symptoms are irritative; they may be motor, sensory, vesical, or rectal. A girdle sensation corresponding to the location of the lesion soon develops, and a partial or complete paraplegia follows. Hyperesthesia may be present, especially in the zone above the girdle, but in a short time all sensation is lost in the parts affected. The application of heat to the hyperesthetic area occasions a sensation of pain. When the myelitis extends to the cervical spine, the power of motion is lost in the upper extremities. The reflexes, both of the tendons and of the skin, are diminished or obliterated in the beginning, but later become exaggerated, except in central myelitis, when they are lost unless the disease be confined to the cervical and upper dorsal region. The electrical reactions generally remain normal, but the reaction of degeneration is sometimes obtained. When the centers of the sphincters are involved, there is involuntary fecal evacuation and incontinence of urine from overdistention of the bladder. Atrophic changes are unusual. The muscles become soft and relaxed, but there is little atrophy except when the gray matter is involved. Rigidity often occurs. Bedsores frequently develop early; they are superficial and not regarded as trophic. In chronic cases, however, deep sloughs of this character may occur. Optic neuritis is sometimes observed in these cases.

The duration of the disease varies from a few days to several years. Apparent improvement may occur, but, as a rule, the disease becomes chronic and the patient is left a hopeless paralytic, greatly tormented at times by muscular twitching, spasm, or flexures, involuntary evacuations of urine and feces, and bedsores. Recurrent cases are occasionally encountered. In another group spontaneous recovery occurs after a year or more of complete rest.

Acute central myelitis is distinguished by a more violent onset, with hyperpyrexia, sometimes with convulsions and complete paralysis. The course is rapid and usually fatal.

In *transverse myelitis* of the cervical cord as high as the sixth or seventh vertebra, the upper extremities are more or less completely paralyzed, and sensation is gradually lost. In some instances, however, only the arms are involved, and the shoulder muscles sometimes escape. Vomiting, hiccough, slow pulse, contracted pupils (miosis), dysphagia, dyspnea, and syncope are sometimes met with.

Diagnosis.—Acute ascending paralysis is excluded in the diagnosis of central myelitis, which it most resembles, by the less marked sensory and trophic disturbances and the absence of fever. In *multiple neuritis* there is not usually so marked anesthesia, and the control of the sphincters is not usually lost. *Tumors* and *hemorrhages* of the cord are sometimes difficult or impossible of diagnosis, from the fact that they produce a form of pressure myelitis.

through the affected roots often follows, but in a large group of cases attended with extensive vertebral disease the nerve-roots escape.

3. *Cord Symptoms.*—These vary with the region affected. There may be paraplegia or paralysis of all the extremities (quadruplegia), dyspnea from involvement of the phrenic nerve, dilatation of the pupils, trophic and vasomotor disturbances, as muscular atrophy, cutaneous eruptions and desquamations, bedsores, sweating, and alterations of local temperature. The sphincters may be paralyzed, a girdle sensation may be present, and other symptoms like those of subacute or chronic myelitis are commonly observed.

The ***diagnosis*** rests upon a careful study of the symptoms and the discovery of the underlying condition. The pain is most severe in cases due to aneurism, and the nerve-roots are more constantly affected in malignant cases. The symptoms are often obscure in tuberculous cases, but in these, as in those due to syphilis, there are usually other lesions and a definite history of infection. Persistent lumbago, Janeway states, is a significant sign of vertebral caries in some cases.

Prognosis.—Cases due to tubercular caries sometimes become quiescent and those due to syphilis may subside under treatment, but in all other conditions the prognosis is exceedingly unfavorable.

Treatment.—Tubercular cases should be treated according to the general methods for tuberculosis, with the addition of orthopedic appliances for the removal of pressure and the prevention or correction of deformity. Confinement to bed, with extension, is often necessary for a time. Cases due to gummata are generally relieved by potassium iodid. Excision of the vertebral laminæ (laminectomy) has proved beneficial. In hopeless cases, as those due to tumors, morphin should not be withheld, and every effort should be made for the prevention of bedsores and excoriations.

ACUTE ANTERIOR POLIOMYELITIS.

1. ACUTE ANTERIOR POLIOMYELITIS OF CHILDREN.

INFANTILE SPINAL PARALYSIS, ESSENTIAL OR ATROPHIC PARALYSIS OF CHILDREN.

Definition.—An acute, febrile disease affecting the gray matter of the anterior horns of the spinal cord of young children, and producing paralysis of certain muscles, followed by rapid atrophy of them.

Etiology.—The disease usually appears before the fourth year of life and in previously healthy children of either sex. It rarely occurs in adults, mostly males. It is often erroneously attributed to injury, as by a fall. It is sometimes attributed to cold, dentition, muscular exertion, or mental strain. Most cases occur in summer, and for this reason, in part, but more particularly because the disease has appeared in epidemic form, it has been regarded by some writers as an infection. It may follow acute disease, menstrual suppression, sexual excess, dissipation, or syphilitic infection.

Morbid Anatomy.—The essential lesion is an acute hemorrhagic myelitis in the cervical or lumbar enlargement. Degeneration follows, with rapid destruction of the ganglion cells, the growth of sclerotic tissue, vascular dilatation, and endarteritis. The disease is believed to originate probably as an embolism or thrombosis, in the ventral spinal artery,

usually of one side, rarely of both; since the lesions correspond to the distribution of its terminal or cornual branches. The cord is more or less deformed as a result of atrophy and sclerosis. The affected muscles also undergo degenerative changes, and the interstitial tissue becomes sclerotic.

Symptoms.—The invasion is abrupt, or preceded by slight fever and malaise for a day or two. The course of the disease may be acute, subacute, or chronic. The first recognizable symptom, as a rule, is paralysis of one or more limbs. Convulsions very rarely occur. The paralysis becomes complete within 24 hours, and the affected member is generally hyperesthetic and painful. In some instances, however, the disease progresses more slowly. When more than one part is affected, the lesions are not symmetrical, except occasionally in adults. Monoplegia is the rule, with many exceptions, in children; paraplegia in adults. All the muscles of a limb are not usually affected to the same degree, and only a certain group may be involved, especially in the upper extremity. Crossed paralyses are peculiarly common to this disease. The paralyzed muscles undergo atrophy within a few days. Sensation is not affected, but the reflexes are obliterated in the affected limb, and the electrical reaction of degeneration is early established. The essential feature of this reaction is a sluggish contraction of the muscle in a state of degeneration or supplied by a degenerated nerve to the galvanic current. The contraction is not instantaneous, as in health, but it may be induced by a weaker current, and the anode-closure contraction may be greater than the cathode-closure contraction. The less important features are an absence of response on the part of the muscles to the faradic current and a failure of the nerve to react to either the galvanic or faradic current.

After a week or two, less frequently after three or four days, recession of the paralysis occurs in some or all of the muscles, but complete restoration of voluntary motion is seldom, if ever, obtained. Later the affected limb falls behind the sound one in its growth, and muscular contractures increase the apparent shortening and deformity. All the forms of talipes are produced in different cases. The head of the humerus sometimes slips from its place, owing to weakness of the deltoid.

Prognosis.—Error is rarely possible. *Multiple neuritis* seldom occurs in young children. It affects the peripheral muscles of the limbs symmetrically and is accompanied with sensory disturbances. In the pseudoparalysis of rickets the legs are usually affected, but the motion is restricted on account of pain, the power is not lost, and the muscles do not atrophy. The rachitic prominences upon the head and joints, and along the sternum, the hyperesthesia and sweating, assist in the diagnosis.

Prognosis.—The general health is not impaired, except by the loss of exercise. Complete recovery cannot be promised, but a great deal can be accomplished by persistent efforts for the improvement of the paralyzed members.

Treatment.—The patient should be made comfortable in the beginning by bandaging the affected limb loosely in a thick layer of cotton. A purgative dose of magnesium citrate should be administered. Excessive fever should be reduced by cool sponging. Morphin is rarely required for the pain. Sodium bromid may be given for the restlessness. The fluid extract of ergot should be administered in doses of ♏x to xx (0.60—1.20) t.i.d for the first week. Counter-irritation over the spine only adds

through the affected roots often follows, but in a large group of cases attended with extensive vertebral disease the nerve-roots escape.

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Treatment.—The patient should be made comfortable in the beginning by bandaging the affected limb loosely in a thick layer of cotton. A purgative dose of magnesium citrate should be administered. Excessive fever should be reduced by cool sponging. Morphin is rarely required for the pain. Sodium bromid may be given for the restlessness. The fluid extract of ergot should be administered in doses of ♏x to xx (0.60—1.20) t.i.d for the first week. Counter-irritation over the spine only adds

to the suffering and accomplishes nothing. As soon as the acute symptoms have subsided, a regular course of treatment by massage and electricity should be begun. A mild galvanic current should be applied at least twice a week to the spine and the affected muscles. The case should not be abandoned as hopeless for several years. Orthopedic treatment is often necessary for relief of the deformity. The nutrition of the child must be maintained with proper food, codliver oil, and malt preparations.

2. ACUTE POLIOMYELITIS IN ADULTS.

When the disease occurs in adults, it does not differ materially from that of children, except in the more frequent occurrence of symmetrical paralyses, as paraplegia or quadruplegia. Multiple neuritis is probably mistaken for this disease in some instances, although its onset is usually less sudden, the atrophy is less rapid and less profound, and the reaction of degeneration is very exceptionally present. When complete recovery follows a doubtful case, the diagnosis of multiple neuritis is established.

ACUTE ASCENDING PARALYSIS.

LANDRY'S PARALYSIS.

Definition.—An acute paralysis beginning in the legs and extending rapidly upward to the trunk, arms, neck, and face, ultimately involving the muscles of respiration, and generally terminating fatally.

Etiology.—Men between 20 and 30 years of age are generally affected. Little is known of the cause, and pathological lesions have seldom been found. The disease sometimes follows an acute infection, and it has been regarded as a peripheral neuritis. A plausible theory is that it is produced by a toxic agent affecting the lower motor neurons. Some authors look upon it as purely functional in character.

Symptoms.—The first manifestation of the disease is usually a weakness of the legs, which develops into complete paralysis, often within a few hours. The muscles of the trunk, arms, neck, and, finally, those of respiration, deglutition, and articulation are affected in rapid succession. The entire course of the disease in fatal cases may occupy only two or three days, and it rarely extends over more than two weeks. The muscles do not atrophy, and the electrical reactions may remain normal; the reflexes are lost. The sensation may be normal or slightly impaired. The sphincters are not involved. Profound dyspnea is induced when the respiratory muscles become implicated, respiration being carried on solely by the diaphragm. The mind and organs of special sense escape. The spleen and lymph-glands are sometimes moderately enlarged, and albuminuria has been noted.

Diagnosis.—The diseases to be excluded are myelitis, especially the acute central form, anterior poliomyelitis, neuritis, and possibly the paralytic form of hydrophobia. From all these the distinction is based upon the rapid ascent of the paralysis, the great predominance of motor symptoms, if not entire absence of sensory manifestations, the presence of fever, and absence of electrical changes or sphincter involvement.

Prognosis.—The disease usually terminates fatally within a few days, sometimes not for several weeks, from involvement of the bulbar centers

controlling the action of the heart. Recovery is possible only when the disease stops before it reaches the medulla.

Treatment.—The patient should be given complete rest and freedom from noise or other disturbance. Ergot should be given in conjunction with potassium iodid. Quinin proves beneficial in some cases. It may be given in doses of gr. ij to iij (0.10—0.20) three or four times a day along with the other remedies. If the case does not terminate fatally, the after-treatment is the same as that of poliomyelitis or multiple neuritis.

PROGRESSIVE MUSCULAR ATROPHY.

CHRONIC POLIOMYELITIS, WASTING PALSY, ARAN-DUCHENNE TYPE OF MUSCULAR ATROPHY.

Definition.—A progressive atrophy of the muscles, generally combined with paralysis and spastic rigidity, due to degeneration of the motor tract of the cord.

Etiology.—The disease usually affects men between 20 and 60, developing after exposure or during convalescence from an acute infection, as typhus or typhoid fever or diphtheria. A hereditary tendency can sometimes be traced. Syphilis, lead-poisoning, or occupational strain of particular muscles may lead to its development.

Morbid Anatomy.—The essential lesion is a degeneration of the motor neurons of the lower segments of the cord, subsequently extending also to those of the upper segments. The muscles at the same time undergo degeneration and atrophy, with hyperplasia of their connective tissue (sclerosis). A distinct atrophic degeneration of the anterior columns of the cord is often visible, and the microscope reveals atrophy or destruction of the multipolar ganglion cells. A similar change extends to the gray matter of the medulla. The neuroglia undergoes hyperplasia, which extends to the lateral columns in the so-called amyotrophic lateral sclerosis. In rare instances the degeneration can be traced to various levels in the motor areas of the brain, and the cortical centers may show degeneration.

Symptoms.—The disease advances slowly and is for a time confined to groups of muscles in the upper extremities, much more frequently on the right side than the left. It occasionally begins in the legs, and rarely affects all the muscles except those of the eyeball and jaw. The onset may be preceded by pains like those of chronic rheumatism. The hands are generally first affected; the ball of the thumb becomes soft and wasted, then the interossei and lumbricales, and the characteristic claw hand is finally produced. In the forearm the flexors precede the extensors; in the shoulder the deltoid is first affected, and sometimes it is the first to be involved in the upper extremity. The disease generally skips some of the muscles, and these falsely appear hypertrophied. Even the bones seem abnormally large in an advanced stage of the disease. Sensory disturbances are not usually observed. The atrophied muscles show a fibrillary twitching, which is intensified by percussion or a draught of air. The reflexes are greatly increased, and it is in this disease that a jaw-clonus can oftenest be obtained. The excitability of the nerves may remain after the muscles have become completely paralytic, and a partial reaction of degeneration is generally obtained. There is often a feeling

of numbness and coolness in the affected limbs. Sweating is often increased, the skin becomes harsh and pigmented, the nails curved and brittle, from trophic change.

In the tonic form of the disease, the amyotrophic lateral sclerosis of Charcot, an interesting form of spastic paraplegia is sometimes observed. When the patient starts to walk, he is unable to step. After a moment's hesitation he takes several rapid short steps with the body inclined forward, then walks at a rapid gait until he attempts to turn, when the process must generally be repeated. The wasting of the muscles in this form is less than in the atrophic.

As the disease extends upward in the spine, the symptoms of bulbar paralysis are produced, and the patient may still later become demented.

Three other types of the disease, described also as muscular dystrophies, are occasionally encountered. These are: (*a*) Erb's *juvenile type*, usually hereditary, occurring in young patients and similar to the atrophic, except that muscular tremors and the reaction of degeneration are absent; (*b*) the *facial type* of infants, beginning as an atrophy of the muscles of expression. The eyes show animation and seem to protrude, but the muscles respond but slightly in an attempt to smile. The disease usually extends to the shoulders; (*c*) the *peroneal type* (Charcot, Marie, Tooth). This begins in the legs, and several years later attacks the hands and forearms. Clubfoot is often produced. The duration of the disease is from 5 to 25 years or longer.

Diagnosis.—In *chronic myelitis* the paralysis precedes the atrophy, and contiguous muscles are affected. The same is true of simple neuritis. There are also in some cases pain and hyperesthesia. The pain and sensory disturbances of *multiple neuritis* suffice for its exclusion, and the atrophy is never primary. *Muscular pseudohypertrophy* may cause confusion in the early stages, but the hypertrophy always affects the lower extremities, and the defective movements are not seen in the apparently atrophic muscles. *Syringomyelia* cannot always be differentiated, but, as a rule, the sensory disturbances enable one to recognize it.

The ***prognosis*** is always unfavorable. A few cases of recovery have been reported.

Treatment.—Strychnin should be given in full doses, and the vitality of the muscles should be further stimulated as long as possible by faradization, with an occasional application of the galvanic current, and massage. The patient should live in the open air and take light and regular exercise. The needle bath and other methods of hydrotherapy should be tried, and nutrition must be maintained. Gowers favors the use of arsenic. Potassium iodid and mercury must be employed in cases having a syphilitic taint.

GLOSSOLABIOLARYNGEAL PARALYSIS.

PROGRESSIVE BULBAR PARALYSIS.

Definition.—A progressive paralysis and atrophy of the muscles of the tongue, lips, and larynx.

Etiology.—The disease rarely occurs in those under 40. It is most common in men of neurotic type. Syphilis, alcoholism, and mental strain are predisposing causes.

Morbid Anatomy.—Degenerative changes are found in the nuclei of the hypoglossal, facial, spinal accessory, and vagus, and in the anterior pyramids. The lesions are identical with those of progressive muscular atrophy, and the symptoms of the latter disease are often present.

Symptoms.—Prodromal numbness in the back of the neck or slight pain has been noted, but it is unusual. The first symptom is generally a difficulty in the pronunciation of the labials, dentals, and linguals: *p*, *b*; *t*, *d*; *l*, *m*, etc. As the lower lip becomes affected, it droops and the saliva escapes. The food collects between the lip or the cheek and the gums. Mastication and deglutition soon become difficult, for the tongue is unable to propel the bolus into the fauces, and the food is often regurgitated into the nares. Then the voice becomes feeble and nasal. The patient becomes emotionate and neurasthenic. The affection of the facial muscles causes partial loss of expression, and the nasolabial folds are deepened. The involvement of the pneumogastric causes alteration of the heart's action and dyspnea.

Diagnosis.—The conditions most likely to be confounded with the disease are cerebral hemorrhage, especially bulbar hemorrhage, multiple sclerosis, and possibly facial trophoneurosis. From all these, however, it is readily distinguished by its slow development and progress, the symmetry of the lesions, and the reaction of degeneration.

Prognosis.—Recovery probably never occurs, but the course of the disease is often slow, and interrupted by intervals of more or less complete remission. Death is seldom delayed beyond five years, however, and may occur at any time as a result of aspiration pneumonia from the entrance of food into the trachea, or from asphyxiation due to its lodgment in the larynx.

Treatment.—If medication be employed, it should be the same as for progressive muscular atrophy. It is more important, however, to maintain the nutrition and strength by a concentrated liquid diet. After the patient becomes unable to swallow, gavage must be resorted to.

THE SPINAL SCLEROSES.

POSTERIOR SPINAL SCLEROSIS.

LOCOMOTOR ATAXIA, TABES DORSALIS.

Definition.—A chronic disease characterized by degeneration and sclerosis of the posterior columns of the spinal cord, producing inco-ordination, sensory and trophic disturbances, and sometimes associated with degeneration of the spinal ganglia and peripheral nerves.

Etiology.—The disease occurs, as a rule, in men between 30 and 40, and particularly in those who have at some time been the subjects of syphilis. Exposure to cold and wet, fatigue, dissipation, sexual excess, and injury are often contributing influences, any one of which may induce the disease in a syphilitic subject, even though that disease may have been dormant for many years.

Morbid Anatomy.—The disease begins as a degeneration of the posterior root-zones. From the several sets of fibers the degeneration advances into the tract of Lissauer and the columns of Burdach, Clark,

and Goll, and the fibers are ultimately replaced to a great extent by connective tissue the contraction of which causes compression of the cord. Several investigators trace the beginning of the disease a step further to a chronic inflammation of the pia mater. The nerve-fibers are very unequally affected in some cases, one set showing an early stage of degeneration, while another is far advanced in the sclerotic process. Finally, the entire posterior columns become converted into a mass of connective tissue containing few remnants of nerve-fibers. In some advanced cases the anterolateral ascending tract is involved and a peripheral neuritis is developed, particularly in the sciatic nerve, sometimes in the optic, fifth, sixth, eighth pneumogastric, and glossopharyngeal. The articular affections are attributed to asymmetrical lesions sometimes found in the anterior columns. Lesions are occasionally found within the cranium.

Symptoms.—The course of the disease is exceedingly chronic and by no means uniform; it may last for many years, or it may terminate at any stage through various accidental complications. The symptoms are conveniently considered under three heads, corresponding to different stages of the disease. These stages are not separated by distinct lines, and there is great diversity in their sequence.

(1) **Initial or Preataxic Stage.**—Sensory symptoms are often first to be recognized, and one of the most common is pain. This is manifested in the form of sudden, spontaneous, often atrocious, darting, or "lightning" pains, shooting down the arms or thighs, or visceral and affecting the stomach and bowels. The latter form is usually accompanied with violent retching and vomiting independent of the ingestion of food, and constitutes the gastric crises. In the same manner crises may affect the heart, larynx, kidneys, bladder, urethra, clitoris, or rectum. These crises may precede other symptoms for several years. Paresthesia is often an early symptom, alone or associated with the pain and other manifestations. It usually begins as a numbness, tingling, itching, creeping (formication), or burning in the feet and legs. A highly characteristic complaint is an absence of normal sensation in the soles. The patient feels in walking as if he were treading upon a soft carpet or upon the air. The hands may be involved early or late, and he experiences difficulty in executing delicate movements, as in tying a cravat or buttoning his clothing, and his handwriting is altered. The tactile sense is affected so that the sense of pain, as that of a pin-prick, travels slowly and may be referred to the wrong extremity or to both. The temperature sense may be impaired or completely lost. A feeling of constriction, or girdle pain, is sometimes experienced at the wrist, knee, or ankle.

Loss of the patellar reflex, or knee-jerk, is a valuable and early symptom in many cases and may precede all others (Westphal's symptom). This feature is rarely absent. The patient should sit for the test upon the edge of a table, or the leg may be supported by the hand of the examiner placed under the knee, and his attention should be diverted by having him grasp the sides of the table or chair, while a sharp blow is struck upon the lower border of the patella. The other reflexes are usually lost during the progress of the disease. Ocular symptoms appear early in some cases and late in others. The most common of them are a gradual loss of vision, sometimes terminating in blindness, due to atro-

phy of the optic nerve; single or double ptosis (drooping lids), paralysis of one or more of the external muscles of the eye, and the Argyll Robertson pupil, in which the iris contracts during accommodation, but not to light. The pupils are often closely contracted (spinal miosis).

Trophic and vasomotor symptoms are occasionally observed early, but not, as a rule, until the ataxic stage. Difficulty in the evacuation of the bladder and decrease of sexual power and desire are sometimes early manifestations; incontinence of urine and cystitis may appear later.

(*2*) **Ataxic Stage.**—The typical symptom of this stage is the loss of the muscle sense. This is usually gradual, and, as stated, may appear early or it may be well advanced before it is recognized. In it the patient loses the normal sensation in the feet, as already described, and he cannot recognize the position of his limbs in bed. He is unable to walk steadily in the dark, to stand upon one foot, or with his feet together, when his eyes are closed (Romberg's symptom). A toppling forward while in the act of washing the face is highly typical. Inco-ordination of movements soon develops, and the characteristic ataxic gait is produced. The patient can guide the movements of his feet only through the sense of sight. He walks with his body inclined forward, his feet wide apart, and usually with the aid of a cane. The feet are swung outward and forward and brought down flat or on the heel. Inco-ordination of the arms develops later in most cases, occasionally before that of the legs. The patient experiences difficulty in all delicate movements. If asked to extend the arm and to immediately touch the tip of his nose with the forefinger, he misses the mark and may fail to touch his face. The muscles become relaxed and the joints can be abnormally extended; the knees sometimes bow backward, yet the muscular strength is retained.

Sensory Symptoms.—In addition to the sensory symptoms referred to under the Initial Stage, areas of hyperesthesia or of anesthesia may be present, particularly in the lower extremities, but sometimes in the form of bands about the thorax.

The affections of the eye sometimes develop in this stage, but it is a peculiar fact that atrophy of the optic nerve is rarely associated with ataxia. Deafness may develop, but the sense of smell is seldom affected. There may be incontinence of urine and cystitis in this stage, as stated, and the inflammation may extend to the kidneys. Obstinate constipation is the rule; the anal sphincter becomes relaxed.

Trophic disturbances are common. The fulgurant pains may be accompanied with an eruption of herpes, edema or local sweating. The nails become atrophic and the hair falls. A perforating ulcer sometimes forms in the foot, back of the big toe or in the heel, occasionally in the cheek, and a round ulcer may be found in the rectum. The joint lesions most frequently affect the knees. In the so-called Charcot joint, the conditions are very similar to those of arthritis deformans, but suppuration sometimes develops, and dislocation or spontaneous fracture may occur. Extensive effusion sometimes collects about the joint (hydrarthrosis) and may rupture spontaneously. Late in the disease the muscles undergo atrophy, probably as a result of degeneration in the ventral horns or of peripheral neuritis. Fränkel has called attention to the ability of the patient to extend his legs completely when they are at a right angle to the body, lying upon the side (hypotonia). Cerebral symptoms are

frequent: hemiplegia at any time; melancholia, paralytic dementia, or paranoia, at a late period.

3. **Paralytic Stage.**—This stage begins when the patient finally becomes unable to walk. Absolute helplessness often characterizes the condition, and the sphincters are often paralyzed. The patient may linger for months in this state, often blind and deaf, until death supervenes from exhaustion or some intercurrent disease.

Diagnosis.—There is seldom difficulty in the diagnostication of a well-marked case. The loss of patellar reflex, the inco-ordination, the loss of muscular sense, the crises, and the Argyll Robertson pupil are all pathognomonic. Several conditions may, however, enter into consideration.

Disease of the cerebellum is accompanied with inco-ordination, nausea, and vomiting, but there are also headache and vertigo, and the pain and pupillary reaction are absent. *Ataxic paraplegia* is characterized by an increase of the patellar reflex. *Multiple neuritis* produces hyperesthesia along the course of the affected nerves, without inco-ordination, crises, or ocular symptoms. *Polyneuritis* is attended with a steppage gait, without the more characteristic symptoms of tabes. Its onset and course are much more acute. There is often ataxia in *cerebral disease*, but only one limb is affected, as a rule. *General paresis* is sometimes accompanied by the symptoms of locomotor ataxia or it may develop late in the history of the latter disease; under such circumstances a diagnosis may be difficult. Recurrent attacks of visceral neuralgia are often an initial manifestation of this disease, and its absence can be determined only when, after careful study of the case, all other symptoms are found to be absent. In a syphilitic subject a conclusion should not be too hastily arrived at.

Prognosis.—Recovery is impossible after the disease has become fully established and the cord sclerotic. Long periods of quiescence may occur, however, and a slow progress of fifteen or twenty years is possible.

Treatment.—The first essential is a removal of all causal influences, as alcoholism, sexual or other excesses, and the patient must avoid fatigue. Since the mind remains unaffected until a very late stage, he may continue his occupation if a professional or business man. It is well in all cases to administer mercury and potassium iodid for a period of several months, as the disease is often arrested in this manner, for a time at least. Gold and sodium chlorid and silver nitrate are occasionally employed, but they are of doubtful utility, and the danger of argyria exceeds any possible gain to be obtained from the silver salt. Ergot, calabar bean, arsenic, strychnin, and other drugs are recommended by some writers.

Local applications, ice-bags, cold douches, blisters, and the cautery, are recommended, but they are to be thought of only in cases presenting an unusually acute onset, and they, as a rule, unnecessarily confine the patient to bed. Galvanism of the spine is extolled by Gray and others, but it is not always well borne.

The *suspension treatment* is now seldom resorted to. It consists in suspending the patient by the arms and head by means of a suspension apparatus for from 30 seconds to three minutes twice a week. The object is to make traction upon the cord. Many patients are much relieved for a time, but the method can exert no influence upon the sclerotic tissue.

The crises can be overcome in some cases only by the administration of an opiate, which is to be avoided as long as possible. Sodium salicylate, phenacetin, acetanilid, or canabis indica should be employed; and when they fail, codein may be given in doses of gr. ¼ (0.01). Regulation of the diet and relief of constipation often diminish the frequency of the gastric crises. The application of the faradic brush is sometimes of benefit. When the crises are accompanied with high arterial tension, they are sometimes relieved by continued administration of glonoin. Fränkel's method of re-educating the patient in co-ordinated movements yields good results in some cases in the hands of a skillful instructor.

In the paralytic stage the utmost care is necessary for the avoidance of bedsores and excoriations. Catheterization, vesical irrigation, and high rectal injections are often beneficial.

PRIMARY LATERAL SCLEROSIS.

SPASTIC PARALYSIS OF ADULTS, SPASTIC SPINAL PARALYSIS.

Definition.—A gradually increasing paresis with spasm of the muscles, without atrophy or sensory disturbance, usually beginning in the lower extremities and probably due to degeneration of the pyramidal tracts.

Etiology.—The disease is most frequent in middle-aged men with syphilitic taint.

Symptoms.—The patient complains of fatigue and stiffness of the legs, sometimes of pain, and later his legs become rigid when he stands. He walks stiffly upon the balls of the feet without touching the heels and without bending the knees, taking short, quick steps and, in an advanced stage, crossing one leg in front of the other. The legs can be passively flexed slowly to any angle, and remain there, but an attempt at sudden flexion is resisted. In the worst cases the legs are drawn closely together by the adductors. All the reflexes of the lower extremity are usually much increased. The strength of the muscles is retained until late, as a rule. In an advanced stage, the arms often become similarly affected, rarely simultaneously with the legs. Ocular symptoms are rare. The sphincters are involved late in some cases. The plantar reflex is so altered, in some cases, that slight irritation of the sole causes extension of only the great toe (Babinski sign of organic disease of the pyramidal tract). The course of the disease is exceedingly chronic, but it may not interfere materially with the general health for many years.

Diagnosis.—In the absence of a definite pathology, the diagnosis is necessarily difficult. Tumors, hemorrhage, vertebral caries, transverse myelitis, and hysteria can sometimes be excluded with great difficulty. General paresis sometimes begins with the symptoms of this affection.

Prognosis.—Recovery is not to be hoped for.

Treatment.—Potassium iodid and mercury should be administered. Ergot may also be employed. Hot baths and massage may be of benefit.

Hereditary Spastic Paraplegia (Hereditary Spastic Spinal Paralysis).—(See also Cerebral Paralyses of Childhood.) Two groups of cases are recognized. One develops in infants or young children and is accompanied with cerebral disturbances, as epileptic seizures or mental dullness.

Erb referred the symptoms to degeneration of the lower part of the pyramidal tract. In the other group the disease develops between the 20th and 30th years as a spastic condition of the legs, short of paralysis, progresses slowly for years, and finally affects the arms. At the end the paralysis becomes complete and may slightly involve the bladder.

Amaurotic Family Idiocy.—This is another form of infantile paralysis occurring in families and characterized by mental disturbances which deepen into idiocy; paresis ending in paralysis, partial and later total blindness, with normal, increased, or decreased tendon reflexes.

Secondary Spastic Paralysis.—This term is applied to spastic paralysis developing in the course of any disease affecting the pyramidal tract, as in transverse, compression, or chronic myelitis, or in multiple sclerosis. The condition is recognized by the rigidity and the exaggeration of the reflexes.

Erb's Syphilitic Spastic Spinal Paralysis (Toxic Spastic Spinal Paralysis—Osler).—This is regarded by Erb as a form of transverse myelitis. It is characterized by muscular rigidity, exaggeration of the deep reflexes, sometimes with paresthesia and the girdle sensation, disturbance of the sphincters, and impotence.

Hysterical Spastic Paraplegia.—This is characterized by partial loss of power, moderate rigidity, atrophy, increased reflexes, and in some cases a spurious ankle clonus (Gowers).

ATAXIC PARAPLEGIA (GOWERS).

Definition.—Sclerosis of the posterior and lateral columns, sometimes annular or diffuse and not confined to the pyramidal tracts.

Etiology.—The disease usually occurs in men of middle age without syphilitic history, sometimes after injury or exposure to cold.

Morbid Anatomy.—The sclerosis in many cases is confined to the terminal branches of the dorsal spinal artery (Marie).

Symptoms.—The legs feel tired and the gait is unsteady and stamping. There is no pain or sensory disturbance. The inco-ordination and rigidity increase, and the arms finally become involved. The reflexes become exaggerated. The sphincters are affected late. Eye symptoms are rare. The diagnosis is based upon the inco-ordination without loss of reflexes, ocular or sensory changes.

HEREDITARY ATAXIA.

FRIEDREICH'S ATAXIA, HEREDITARY TOXIC PARAPLEGIA, POSTEROLATERAL SCLEROSIS.

Definition.—A combined degeneration and sclerosis of the posterior and lateral columns of the cord and posterior nerve-roots, producing ataxia and paraplegia.

Etiology.—The disease occurs in families, affecting, as a rule, several brothers and sisters, but it is not always hereditary. It generally develops in childhood or early life and more commonly in males. Syphilis is not a constant factor in its production.

Morbid Anatomy.—The lesions are a combination of those belonging to locomotor ataxia and those of the ataxic paraplegia of Gowers. The sclerosis is thought by some writers to be neuroglear and different from other spinal scleroses. The lesions are generally found in the cervical and lumbar regions.

Symptoms.—The inco-ordination begins in the legs, and the gait is more irregular and swaying than that of locomotor ataxia. The arms are more pronouncedly affected than in the latter disease. There is often a swaying movement of the arms and head (static ataxia) when the body is at rest; sometimes the movements are more like those of chorea. The patient is generally unable to walk. The paraplegia is rather a paresis than a paralysis. Late in the disease, contractures, scoliosis, and talipes equinus, with dorsal flexion of the great toe, develop. Hyperesthesia and retardation of sensation are occasionally present, but, as a rule, there is no sensory disturbance. The deep reflexes are early lost, but the cutaneous and pupillary remain normal. Nystagmus is a constant and characteristic symptom. The speech is slow and scanning. The mind remains normal until late.

Diagnosis.—*Ataxic paraplegia* is excluded by the absence of the knee-jerk, ankle clonus, and muscle spasm. *Locomotor ataxia* is distinguished by its ocular symptoms, and it is a disease of later life. *Disseminated sclerosis* is characterized by greater inco-ordination of the arms and less of the legs. *Heriditary chorea* is not accompanied with nystagmus, loss of the deep reflexes, scoliosis, talipes, or flexion of the toe.

Prognosis.—There is no possibility of recovery, but the disease is not incompatible with many years of life.

Treatment.—This is the same as that of locomotor ataxia, but fewer cases are benefited by the potassium iodid. Massage and other methods for the prevention of contractures should be employed.

Cerebellar Type.—Marie and others have described a type of the disease occurring in adults and due to atrophy of the cerebellum. The legs become rigid, but the tendon reflexes are retained. Scoliosis and talipes are absent.

Toxic Combined Sclerosis.—A combined sclerosis affecting several columns of the cord is sometimes observed after poisoning with ergot and in pernicious anemia, pellagra, and some of the chronic wasting diseases, probably as a result of poisons produced in the body.

Progressive Interstitial Hypertrophic Neuritis of Infants.—This is a rare family disease occurring in early life, described by Dejarine and Sottas. It is characterized by the symptoms of locomotor ataxia combined with those of progressive muscular atrophy, including the face, and hypertrophy and sclerosis of the peripheral nerves.

SYRINGOMYELIA.

Definition.—A disease of the spinal cord due to the growth of gliomatous tissue about the central canal, and resulting in the formation of small cavities.

Etiology.—The disease is an infrequent one, usually affecting males

from the fifteenth to the thirtieth year. Nothing is known of the exciting cause.

Morbid Anatomy.—Small cavities of various shapes are formed around the central canal resembling, but distinct from, hydromyelus. The cavity is usually situated in the dorsal region, sometimes in the cervical, and may extend the entire length of the cord. It may involve only one dorsal cornu. The morbid process is a gliosis, a growth of embryonic neuroglear tissue, with subsequent degeneration or hemorrhage and cavity formation.

Symptoms.—The disease begins with pains in the arms and paresthesia of the hands, followed by anesthesia. Its progress is slow, extending to the trunk and then to the lower extremities. A spastic condition develops, with exaggeration of the reflexes and sometimes the peculiar symptoms of amyotrophic lateral sclerosis. The tactile and muscular senses and the special senses are retained, but the perception of pain and differences of temperature is lost. Injury often results from the latter condition. Trophic and vasomotor changes are common, particularly in the hands. The sphincters are not involved until late, when the medulla becomes involved. Scoliosis may be produced. Very irregular symptoms are sometimes observed which have been described by Schlesinger as belonging to different types of the disease, as the (*a*) motor, (*b*) sensory, (*c*) trophic, or (*d*) tabetic manifestations predominate.

Diagnosis.—The differentiation is to be made from progressive muscular atrophy, Morvan's disease, and the anesthesia of leprosy. In a typical case the muscular atrophy, with the gait of amyotrophic lateral sclerosis, loss of pain and temperature perception, and retention of the tactile sense, is pathognomonic. Morvan's disease is further distinguished by its unilateral invasion and the loss of tactile sense; and in leprosy the anesthesia is complete, perineuritis and tubercles are present, and the bacillus is found.

Prognosis.—The course of the disease is slow and interrupted by remissions. It may thus persist for fifteen or twenty years, but is ultimately fatal.

The treatment is purely symptomatic.

Morvan's Disease.—This is the trophic type of syringomyelia in the classification of Schlesinger. It occurs in neurotic subjects, usually young adult males, sometimes after injury or exposure, with pains and atrophy of the hands and arms, followed by anesthesia, analgesia, and whitlows, sometimes with necrosis of the phalanges. The course of the disease is protracted.

TUMORS OF THE SPINAL CORD.

Tumors of almost every variety may be found in the cord or its membranes. With the exception of congenital lipomata, they usually occur between the ages of 30 and 50, more frequently in men. Tubercular, syphilitic, and gliomatous growths are the most common within the cord, while fibromata, sarcomata, syphilitic, and tubercular growths generally attack the dura. Parasitic cysts are occasionally found in the

extradural space. The probable exciting causes are trauma and exposure. The tumor generally begins in the meninges; tumors of the vertebra sometimes extend to the cord. The tumor is small, and is usually situated in the lower cervical or in the dorsal region. The symptoms are a result of hemorrhage or softening and degeneration due to pressure; myelitis is occasionally set up.

Symptoms.—There are often no local symptoms, in the absence of vertebral disease. Pain, referred to the distribution of the nerves that are involved in the pressure, is the most constant symptom, which is generally accompanied with disturbed sensation, at first unilateral, but becoming bilateral with the increasing growth of the tumor. Muscle rigidity and contractures develop, the reflexes are increased, and paralysis follows, with such trophic affections as bedsores. Sometimes a different picture is presented, with loss of reflexes, girdle sensation, paresthesia, hyperesthesia, or anesthesia in different areas, spasm of the muscles, and finally paralysis. The anesthetic areas are sometimes painful. The diagnosis is generally based upon the character of the disturbances in the nerve-roots from the affected region and the gradual paralysis, but in many cases the manifestations are so slight or so vague as not to be diagnosticated during life. In caries of the vertebræ, the pain is not usually so severe, and there are local tenderness and swelling or angular curvature (kyphosis). Cervical meningitis yields symptoms identical with tumor, but its progress is more rapid, as a rule. Transverse myelitis has also a more rapid course, and is further distinguishable by the different order and location of the pain and other symptoms.

The ***prognosis*** is unfavorable, except in early recognized syphilitic cases. The duration seldom exceeds three years.

Treatment.—Potassium iodid should be given in large doses when there is a probability of syphilis. In other cases the treatment is symptomatic, unless surgical measures can be resorted to.

MALFORMATIONS OF THE SPINAL CORD.

Spina Bifida (Meningocele, Myelocele, Hydrorrhachis).—This is a congenital affection, chiefly of surgical interest, due to imperfect closure of the spinal canal. A fluctuating tumor is formed beneath the skin, which contains a portion of the dura and arachnoid and cerebrospinal fluid. The tumor, usually situated over the lumbar or sacral region, varies in diameter from one to five inches. The cord may be normal or atrophic, and the dilated central canal sometimes communicates with the cyst cavity. Various pressure symptoms are occasionally produced, as talipes and perforating ulcer of the foot. Suppuration may develop in the sac, or rupture may occur, with immediately fatal result. Pressure upon the sac sometimes distends the fontanels and may produce dyspnea and coma. The condition usually goes from bad to worse, but the patient occasionally recovers spontaneously or through operative measures.

Lesions of the Conus Medullaris and Cauda Equina.—Injury, disease, or tumor situated below the second lumbar vertebra may produce paralysis in groups of muscles or areas of anesthesia, loss of sphincter control,

and disturbance of sexual function through pressure upon the lumbar nerve-roots. When the cauda equina alone is affected, the loss of sphincter power may be the only symptom.

DISEASES OF THE BRAIN AND ITS MENINGES.

DISEASES OF THE MENINGES.

EXTERNAL PACHYMENINGITIS.

Definition.—An inflammation of the external layer of the dura mater of the brain.

Etiology.—The most common cause is injury, especially fracture of the skull, caries, or inflammation extending from without, chiefly from the middle ear, frontal, or ethmoid sinus. Erysipelas is occasionally responsible for an acute attack; syphilis is a common factor in the more chronic cases.

Morbid Anatomy.—The dura is thickened, hyperemic, edematous, and opaque; it may be detached and separated by an accumulation of blood or exceptionally by pus, but it is more commonly bound down by firm connective tissue. Laminæ of bone are sometimes formed in it in chronic cases.

Symptoms.—There are no typical manifestations. Headache and localized tenderness are common to this and many other affections. Convulsions or local paralyses, usually of a mild type, are sometimes observed as a result of pressure. In a large group of cases, especially in the insane, the disease is discovered only upon autopsy.

Treatment.—Medicinal treatment is limited to syphilitic cases, in which the usual remedies should be employed. An early resort to the trephine is indicated in most nonsyphilitic cases, especially when there is evidence of suppuration. Counter-irritation with the cautery has been recommended.

INTERNAL PACHYMENINGITIS.

This is an infrequent affection occurring as a pseudomembranous, purulent, or hemorrhagic inflammation, of which the hemorrhagic is the most common.

Etiology.—The affection is met with chiefly in advanced life, occasionally in children. It may occur, however, in chronic tuberculosis, syphilis, pernicious anemia, leukemia, valvular disease of the heart, or other conditions leading to degeneration of the blood-vessels.

Morbid Anatomy.—One or more punctate, rarely profuse hemorrhages occur, as a result of which an accumulation of blood of variable quantity is formed between the dura and arachnoid. The clot becomes organized to some extent. It is usually situated beneath the parietal bone, and the condition is sometimes bilateral. A hematoma is formed in the more extensive cases.

Symptoms.—In many cases these are absent. Pressure symptoms are sometimes produced, however, and, from the situation of the affection over the cortical centers, monoplegia or hemiplegia may result. Aphasia may be produced. When the hemorrhage is extensive, convulsions

or fatal coma may be induced. In another group of cases, recurrent symptoms, not unlike those of brain tumor, may exist for many years. Spontaneous recovery has been noted in a few instances. The treatment is the same as that for external pachymeningitis.

LEPTOMENINGITIS.

Definition.—An inflammation of the pia and arachnoid membranes of the brain.

Etiology.—Inflammation of the meninges in acute cerebrospinal meningitis, and that due to tuberculosis or syphilis, are not included under this heading, but are considered elsewhere.

The disease under consideration occurs, as a rule, in the third and fourth decade of life, occasionally in childhood, and somewhat more frequently in males. (*a*) It may arise from secondary infection through the bacilli or toxins of such diseases as typhoid fever, influenza, the acute exanthemata, rheumatism, septic pneumonia, or chronic nephritis. The pneumococcus or micrococcus lanceolatus is found in most of these cases, independently of pneumonia. The typhoid bacillus and the bacillus coli communis have been found. (*b*) Another group of cases owes its origin to extension of the inflammation from the middle ear, wounds, fractures, or caries of the skull, in which staphylococci or streptococci are usually present. Or the disease may arise from abscess of the brain, thrombosis of the sinuses, suppurative inflammation of the nose, frontal or ethmoid sinuses, or to erysipelas.

Morbid Anatomy.—The lesions may be limited to a small, circumscribed area, or they may extend over the entire brain and cord. They may be limited to the ventricles, particularly in children. When due to otitis, the disease is usually unilateral; due to pneumonia or ulcerative endocarditis, it is bilateral and generally confined to the cortex. In nephritis and cachectic conditions it is ordinarily confined to the base. The inflammation becomes suppurative almost from the beginning. (Consult also the paragraphs on the Morbid Anatomy of Cerebrospinal Meningitis, p. 111, and Tubercular Meningitis, p. 179.)

Symptoms.—Although a more or less typical train of symptoms is common to nearly all cases, there are often no peculiar manifestations by which the exact character or location of the lesions can be diagnosticated. In a majority of cases the features are the same as those described under Cerebrospinal or Tubercular Meningitis. It should be borne in mind also that the supervention of headache, photophobia, retraction of the head, possibly with vomiting, constipation, and increase of temperature in the course of typhoid fever or other acute infection, may be due to hyperemia of the meninges, without actual inflammation. When, however, these symptoms persist, and especially if convulsions occur, the pulse becomes slow, the vision obscure, the hearing impaired, and, when hyperesthesia develops, the face assumes a pained expression, and opisthotonos becomes pronounced, there is no longer doubt of the presence of meningitis. These symptoms often develop gradually, and the diagnosis may be in doubt for several days. In the early suppurative cases there are often chills, irregular fever, sweating, with projectile vomiting, and the pulse may be accelerated

instead of slow. The slow pulse, with fever, is more significant of this disease. Rigidity and twitching or spasm of the muscles, sometimes unilateral, are frequently observed. Incontinence of urine and feces often develops toward the close.

Basilar meningitis is characterized more especially by pressure symptoms due to involvement of the nerve-trunks within the cranium. Strabismus, ptosis, slight facial paralysis, anesthesia, and throphic disturbances due to involvement of the fifth nerve are common symptoms. The pupils, at first small, become dilated and often unequal. Optic neuritis, with deepening blindness, is not uncommon, and the respiration often becomes irregular. The reflexes may be exaggerated in the beginning, and lost at a later stage.

Diagnosis.—The differentiation from acute cerebrospinal meningitis may be difficult, but this can generally be excluded by the absence of an epidemic, and the primary development of symptoms on the part of the spinal cord, as pain and tenderness, with retraction of the neck and rigidity or contractures of the extremities. The differential diagnosis between meningitis and other affections is considered under Cerebrospinal Meningitis and Tubercular Meningitis.

Prognosis.—Acute suppurative cases usually terminate fatally, but surprising exceptions are occasionally observed. In cases due to secondary infection, especially when they develop near the natural termination of the disease, the outlook is more hopeful, but the disease is always a grave one, and death is often preferable to the blind, paralytic, and frequently imbecile condition in which the patient is left after recovery.

Treatment.—All the measures employed in the treatment of acute cerebrospinal meningitis are applicable in acute cases. The patient must be given complete rest in a quiet room. Ice-bags should be applied to the head, and to the spine when the cord is involved. Thorough examination should be made in cases of obscure origin in order to determine the cause, and, this done, the propriety of attempting relief through surgical measures should be considered. The assistance of a specialist is generally advisable. Counter-irritation by means of the cautery lightly applied to the back of the neck is often of benefit, and the barbarous seton of fifty years ago was often followed by prompt remission of symptoms. Lumbar puncture has yielded good results in some cases. The bromids should be administered freely; morphin is sometimes necessary for the relief of pain. Potassium or ferric iodid is of benefit during convalescence. The diet should be nutritious and for the most part liquid. The action of the bowels must generally be regulated with laxatives.

AFFECTIONS OF THE BLOOD-VESSELS AND CIRCULATION OF THE BRAIN.

Endarteritis and Arteriosclerosis.—Degenerative changes are exceedingly common in the blood-vessels of the brain. The process is the same as that described under the heading Arteriosclerosis in the chapter on Diseases of the Circulatory System. It may be localized or general, and may result in moderate thickening of the intima or a complete obliteration of the lumen of the vessel (endarteritis obliterans). Atheromatous patches are frequently produced, and a further result in many

cases is the formation of miliary aneurisms. Syphilis is an important etiological factor, and a great majority of the cases not so produced occur in advanced life. Nodular periarteritis is peculiar to syphilitic cases.

Aneurism of the Cerebral Arteries.—Aneurisms, other than miliary, which are considered under the head of Cerebral Hemorrhage, are occasionally met with, chiefly upon the outer surface of the brain, and, as a rule, in middle-aged men. They result from endarteritis or embolism and they are sometimes associated with endocarditis, since it is the most frequent cause of embolism. The left middle cerebral artery is most frequently affected, then the basilar or internal carotid and the communicating arteries. The aneurism is generally saccular, occasionally sessile, seldom fusiform, and rarely exceeds a half-inch in diameter. Its structure is the same as that of aneurisms in other locations. They erode the overlying bone, produce moderate compression of the brain substance, and not infrequently rupture.

Symptoms.—A constant headache is the most important feature. This is aggravated by anything which increases the blood-pressure, as exertion, straining, or stooping. There is usually a throbbing sensation, and the patient may hear a bruit with each pulsation. Vertigo, nausea, and vomiting are frequent symptoms. Manifestations distinctive of the location of the aneurism are seldom present. Rupture, with the production of apoplexy, is the usual termination.

Diagnosis.—Aneurism is differentiated from other tumors of the brain chiefly by the intensification of the symptoms upon slight increase of the circulation. Optic neuritis favors tumor, endocarditis aneurism. Pressure symptoms involving the nerve-roots at the base generally point to aneurism, since it is more commonly situated in that region.

Prognosis.—The disease terminates fatally by rupture in most cases within a few weeks after the development of pronounced symptoms.

THROMBOSIS OF THE SINUSES AND VEINS.

Thrombosis may arise primarily or secondarily through extension of inflammation from contiguous parts.

Primary thrombosis is occasionally encountered in infants under six months of age, and generally in connection with diarrhea. Gowers regards thrombosis of the veins as a frequent cause of infantile hemiplegia.

The so-called **autochthonous sinus-thrombosis** is met with in cases of anemia and chlorosis, usually in connection with thrombosis of the veins in other parts of the body. It occurs also in the late stages of cancer, tuberculosis, and other chronic wasting disease (marantic thrombosis).

Secondary thrombosis is a more frequent affection. It usually arises from disease of the internal ear, rhinitis, meningitis, tubercular caries, or fracture of the skull, compression by tumors, erysipelas, or suppurative disease in the tissues outside of the skull.

Morbid Anatomy.—(See Thrombosis, p. 15.)

Symptoms.—These are by no means uniform. Many cases begin with fever, and chills follow, often preceded by a constant headache, dizziness, and vomiting. The patient becomes listless, stupid, and finally delir-

ious, or he may have convulsions. Hemiplegia is not unusual, and other manifestations arise which more definitely point to the situation of the thrombus.

Longitudinal Sinus.—Thrombosis of the longitudinal sinus is occasionally discovered at autopsy in cases presenting no symptoms. Headache, epistaxis, convulsions, vomiting, and other disturbances may, however, occur, and the veins of the face and head may be distended and the side of the head edematous. The fontanels are distended in an infant, and meningitis may develop or convulsions and coma may be produced.

Lateral Sinus.—When the cause is suppuration of the internal ear, this affection becomes aggravated and the tissues about the ear become edematous. The external jugular vein of the affected side, receiving less blood than its fellow, is more rapidly emptied during a full inspiration (Gerhard). The clot may extend into or through the internal jugular vein, causing it to become indurated and sensitive to pressure. Optic neuritis and nystagmus may develop, and in rare cases there is hoarseness or aphonia, dysphagia, and spasm of the muscles of the neck.

Cavernous Sinus.—The important feature of this location is obstruction of the flow of blood from the ophthalmic vein, causing edema of the conjunctiva and eyelids of the affected side, with protrusion of the globe. The retina becomes edematous, its veins distended and pulsating. The orbital muscles may become paretic, with the production of strabismus. The ophthalmic branch of the fifth nerve becomes painful. Suppurative panophthalmitis may develop. In secondary thrombosis the onset is often sudden and the symptoms of septicemia may be present from the beginning, along with others to some extent of a localizing order.

The ***diagnosis*** is based upon the peculiar symptoms of localization, for the general features are common to tumors, abscess, and other affections. The development of the symptoms described, in connection with chlorosis or anemia, is highly diagnostic of thrombosis.

The ***prognosis*** is grave and usually extremely unfavorable.

The ***treatment*** is surgical, especially in cases due to ear disease; or it is only palliative.

ANEMIA OF THE BRAIN.

The brain becomes anemic in those conditions in which there is general anemia, as after profuse hemorrhages, pernicious anemia, leukemia, or inanition. Anemia of the brain may result also from the accumulation of a large quantity of blood in certain regions, as in the peritoneal cavity after tapping for ascites. A more permanent condition results from aortic stenosis. Sometimes it is due to such local conditions as obliterative endarteritis, the pressure of tumors, or an obliteration of a portion of the circle of Willis, accumulation of fluid in the ventricles, the anemia involving the entire brain or only a part of it.

The appearance of the brain after death is typical. The smaller vessels are empty, and the entire brain substance is moist and extremely pale. Anemia of the pia mater is usually associated with that of the brain.

Symptoms.—When the anemia develops acutely, vertigo or syncope is produced. When a little less acute there is roaring in the ears, flashes of light before the eyes, the sight becomes dim, respiration is rapid, sometimes nausea and vomiting occur, and the patient may become delirious.

HYPEREMIA OF THE BRAIN.

Hyperemia of the brain may be active or passive. Active hyperemia probably accompanies any marked increase of the general circulation; but hyperemia of the brain is a condition of high intravascular tension rather than an increased quantity of blood. A more or less general active hyperemia occurs in all inflammatory conditions, and perhaps in some of the acute infectious diseases, especially those accompanied with restlessness, insomnia, delirium or other cerebral manifestations.

Passive hyperemia is induced by any influence which retards the return of blood from the cerebrum, as in general venous engorgement of valvular disease, emphysema, asthma, and sometimes from the pressure of tumors. The symptoms of hyperemia are not uniform. Insomnia, restlessness, convulsions, are generally attributed to active hyperemia, while mental dullness and coma are regarded as belonging to passive hyperemia.

Treatment.—That of anemia is general, consisting in the administration of remedies for the improvement of the condition of the blood, relief of inanition, and tonics to strengthen the circulation. In hyperemia an effort should be made to reach the cause. Some relief is afforded by the application of ice-bags to the head, and the administration of full doses of the bromids, or 15-drop doses of hydrobromic acid.

EDEMA OF THE BRAIN.

This occurs, for the most part, in atrophy of the cerebral convolutions, thrombosis of the sinuses, passive hyperemia, chronic nephritis, occasionally in acute alcoholism, and locally in the vicinity of tumors and abscesses of the brain. The appearance of the brain is similar to that of anemia. The quantity of fluid in the ventricles and tissues is increased and there is general pallor. The symptoms are not clearly defined. If the view of Leube is correct, they embrace many of the cerebral manifestations of uremia.

Treatment.—An effort should be made to hasten the absorption of fluid by catharsis, diuresis, and diaphoresis. The treatment must be modified to a great extent, however, to conform to the causal indications. Digitalis and strychnin should be employed when the circulation is feeble.

CEREBRAL HEMORRHAGE.

APOPLEXY, INTRACRANIAL HEMORRHAGE, "PARALYTIC STROKE."

Definition.—Hemorrhage due to the rupture of a cerebral blood-vessel.

Etiology.—Cerebral hemorrhage occurs most frequently in individuals

over 50 years of age. It is occasionally met with, however, in infants or in young adult or middle life. In the latter group of cases it is generally a result of syphilitic disease of the arteries. It is more common in men than in women, and the transmission of a hereditary tendency is often apparent, particularly in gouty families. Individuals with habitually high arterial tension, whether natural or the result of alcoholic or other poisoning of the blood, are doubtless more liable to apoplexy than others, but there is no type of stature or physique by which such tendency can be invariably prognosticated.

Predisposing Causes.—Conditions which favor a degeneration of the blood, with the production of endarteritis or arteriosclerosis, and particularly the production of miliary aneurisms, strongly tend to the development of cerebral hemorrhage. Among these are syphilis, chronic alcoholism, chronic nephritis, gout, lead and other metallic poisons, anemia, leukemia, and purpura hemorrhagica.

Exciting Causes.—Violent muscular effort, nervous excitement, anger, fright, and intoxication are frequent exciting causes, but many cases occur independently of any such influences, at night and during sleep. Engorgement of the stomach by overeating and drinking, perhaps associated with constipation, often precedes an attack.

Morbid Anatomy.—The essential lesion in a majority of cases is a rupture of a miliary aneurism. Next most frequent is the rupture of a vessel at a point weakened by atheromatous degeneration. Either of these conditions results from a primary hyalin or other degeneration of the intima, with softening, degeneration, and finally distention or destruction of the media; or beginning in the media and involving the intima. The aneurism which has ruptured is often found with great difficulty. It is most frequently situated upon a branch of the middle cerebral artery, especially in the anterior perforated space, but any of the cerebral vessels may be involved. Secondary changes are found in the clots, and the nerve-fibers that have been subjected to pressure become sclerotic or otherwise degenerated.

Symptoms.—The onset is usually sudden, often in the midst of apparently good health and without premonitory symptoms. Such prodromes as headache, vertigo, thickness of speech, or numbness and tingling of the hand are observed in some cases for a few hours, possibly for several days before the seizure. Following the hemorrhage the symptoms may be divided into two groups, those of the attack (chiefly reflex), and later or localizing symptoms.

The Seizure.—The first symptoms are in great measure due to shock. The patient becomes unconscious; rarely he is seized with a convulsion. Occasionally the seizure is less violent, intense headache is complained of, there are vertigo and nausea, vomiting, and psychical disturbance; the loss of consciousness is less sudden, and the paralysis may be recognized before coma has supervened. The coma is generally profound. The face is intensely cyanotic, or it may have an ashen hue. The respiration is rapid, full, snoring, and often stertorous. Expiration is accompanied with dilatation of the cheeks and puffing of the lips. The pulse is at first slow and full, the arterial tension may be relatively normal or greatly increased. When the tension is high, the pulse often becomes rapid. The pupils are usually dilated, often unequal, and do

not respond to light. Conjugate deviation of the eyes and rotation of the head toward the side on which the hemorrhage has occurred are often observed; rarely the opposite or an alternating deviation. The temperature is subnormal during the first twenty-four hours, but generally rises to 100° or 101° F. (37.8°—39.3° C.) when the attack is not immediately fatal. The skin is cool and moist. This reactionary fever, as it is called, probably due to changes in the blood-clot and its absorption, may last a week or two. All the muscles, even the sphincters, are at first relaxed—those which are paralyzed to a more profound degree than those of the opposite side. The face, particularly the mouth, is drawn toward the sound side. All the reflexes are abolished for a time. The paralyzed limbs may be warmer than those of the unaffected side.

The localizing symptoms vary with the situation of the lesion. Since this is, in a majority of cases, in the anterior portion of the internal capsule, compression of the motor fibers from the cortex is produced, and hemiplegia results. The hemiplegia is said to be complete when the face, arm, and leg are affected, and incomplete when either of these parts escapes. The paralysis affects the side opposite the lesion within the brain, owing to the decussation of the fibers. In those instances in which the fibers do not decussate, and when the pressure is exerted below the point of decussation, the paralysis involves the muscles of the same side. In such cases the lower part of the face only is affected, the frontalis and orbicularis palpebrarum escape. The hypoglossal nerve is involved in hemiplegia, the tongue deviates to the affected side. Aphasia is sometimes present.

The completeness of the paralysis varies in different cases and in different parts. That of the arm is usually deeper than that of the leg. There is often absolute paralysis of the arm and leg, with only partial loss of power in the face muscles, and occasionally the leg is most deeply affected. A paralysis that is absolute in the beginning may rapidly subside into a partial one. Certain muscles frequently escape. Such trophic changes as the formation of vesicles or sloughing are sometimes developed in the paralyzed member. Monoplegias are sometimes observed, particularly when the hemorrhage is in the motor area of the convolutions. This is, however, an infrequent form of paralysis, except in children, when it is generally accompanied with convulsions. When confined to the frontal or right parietal lobe, focal symptoms may be absent. In other cases localizing symptoms are observed in the following relations: (*a*) Hemorrhage into the crus causes hemiplegia of the opposite side with oculomotor paralysis of the same side. (*b*) Hemorrhage into the posterior region of the first and second temporal convolutions produces word-deafness. (*c*) Hemorrhage into the occipital lobe, the lingual or fusiform lobule, or angular gyrus produces blindness in one-half of the field of vision (hemianopia). (*d*) Hemorrhage into the pons, if slight, causes paralysis of one side, with conjugate deviation of the eyes from the side of the lesion. When in the lower portion of the pons, the fifth or other cerebral nerve may be involved, producing paralysis or anesthesia of the parts supplied. (*e*) Hemorrhage into the lateral ventricle produces paralysis and rigidity of the opposite side, with stertorous breathing and often with high temperature and convulsions.

(*f*) Hemorrhage into the cerebellum causes vertigo, vomiting, incoordination, severe occipital pain and sometimes unconsciousness. A staggering gait, once thought pathognomonic, occurs when the peduncles are involved, and it may occur in hemorrhage of the pons. (*g*) Hemorrhage into the medulla causes involvement of the cranial nerves. When at all extensive, it is usually rapidly fatal from embarrassment of the respiration and heart's action. Other peculiarities are occasionally observed.

The course of the disease varies with the extent and location of the hemorrhage. After a slight hemorrhage the symptoms often disappear rapidly; that of the face and tongue often improves within a few days. Sometimes the leg shows the most rapid recovery, and it usually precedes that of the arm. The muscles of the shoulder recover before those of the arm. In most cases the leg recovers to such an extent that the patient becomes able to walk in a few weeks, although he may never gain full use of the limb, and the arm may remain limp and useless. The early improvement may prove transitory, and be followed by a deepening of the paralysis, with or without recurrence of the hemorrhage. Post-paralytic rigidity of the muscles develops in most cases in which the paralysis persists, and it is most pronounced in the arm and hand. The contractures are often painful for many months, strongly flexing the wrist and fingers. Contractures and rigidity sometimes fail to develop, however, particularly in cases in which the power of the leg has been fully restored. Tremor often develops late in the paralyzed muscles, or there may be a choreic movement or almost rhythmical swaying of the limbs (athetosis). The reflexes become increased in the later stages of the disease. Lesions sometimes develop also in the joints of the affected members. The muscles usually atrophy to a variable extent.

Diagnosis.—The recognition of a hemiplegia may be difficult either in cases which develop suddenly with profound coma, or in those in which several days elapse before the paralysis becomes complete. In the former the paralysis can generally be determined by raising the limbs and permitting them to drop. The paralyzed member always falls in a sudden, lifeless manner, while slight muscular resistance can be observed in the sound side. In the gradual cases the diagnosis must be established chiefly by the history of the case, the recognition of a tendency or an exciting cause, and the presence of premonitory symptoms. A slight rigidity and numbness of the affected side may be observed. As a rule, however, a positive diagnosis must be deferred.

Embolism and *thrombosis* often produce symptoms almost identical with those of hemorrhage, and a diagnosis may not be possible. The former occurs, as a rule, in younger subjects, the latter in older ones, than hemorrhage. Fever and stertorous breathing are absent in both. Embolism can be traced to endocarditis. The pupils are usually unaffected, convulsions rarely occur, and the exciting causes are different from those of hemorrhage.

Hemorrhage of the pia mater may cause symptoms of cerebral hemorrhage, but it is rarely an independent affection.

The *coma* of cerebral hemorrhage is distinguished from that of uremia and other conditions chiefly by the history of the case, the absence of

albumin or sugar from the urine, and the recognition of rigidity or paralysis. The pupils are usually contracted in uremia, and the eyes do not, as a rule, deviate. The odor of alcohol on the breath may be of value, but it is often dangerously misleading. The possibility of fracture of the skull, tumors of the brain, or cerebral syphilis should be borne in mind.

Prognosis.—The disease is always a grave one. Complete recovery from a first attack is possible; incomplete recovery is more common. The patient may live many years, succumb to a second or third attack or to die of another disease. The third attack is generally, though not invariably, fatal, as is popularly believed. Much depends upon the extent and location of the lesion. Hemorrhage into the ventricles, or rupturing into them, is usually rapidly fatal. Hemorrhage upon the cortex is less dangerous than at the base or within the cerebrum. High fever, delirium, and coma, especially increasing coma for 48 hours, are exceedingly unfavorable symptoms. The improvement of the first week or ten days may be deceptive. The late contractures and tremor are permanent, and subject to little or no improvement under treatment.

Treatment.—The patient should be gotten to bed with as little delay as possible. He should be placed on his back in a low reclining posture, with the head high. The treatment then to be pursued depends upon the condition of the circulation. When the arterial tension is high, it should be promptly reduced. Venesection is the surest and best means, withdrawing from 10 to 20 ounces of blood. It must not be practiced, however, in a case of low arterial tension or when the diagnosis does not clearly exclude embolism and thrombosis. Nitroglycerin should be administered when bloodletting is objectionable. It should be given in drop doses every twenty minutes until its effect becomes apparent. When the heart's action is rapid, the tincture of aconite should also be administered, but its action must be watched, and the administration stopped as soon as the pulse becomes slower. Next in importance is an early evacuation of the bowels. Owing to the condition of unconsciousness, this is best accomplished by placing on the back of the tongue gr. iij (0.20) of calomel, or gtt. ij of croton oil in emulsion or mixed with butter (Thompson). If there is muscular twitching or other premonition of convulsions, potassium bromid should be given in doses of gr. xx (1.30), repeated twice or three times a day. An ice-cap should be kept constantly upon the head. The patient must not be disturbed for any purpose, but his position should be occasionally changed to prevent the formation of bedsores. He should also be provided with an air-cushion. Catheterization should be performed during the coma. Alcoholic stimulation should be avoided. Small doses of strychnin (gr. 1-120 to 1-60) may be employed if the patient becomes weak as the acute stage subsides. The patient must not be permitted to sit up too soon, and the ice-cap should be kept upon the head for several days. As the paralysis subsides, the affected muscles should be gently rubbed for ten minutes each day or morning and evening, and after the second week has passed they should be treated with massage, the faradic current, and passive motion, as these measures prevent, to some extent, the rigidity and contractures. Potassium iodid is regarded by some phy-

sicians as beneficial in the removal of the clot. After recovery, the patient's life should be a quiet one, free from anxiety, worry, dissipation, and other forms of excess.

EMBOLISM AND THROMBOSIS OF THE BRAIN.

Etiology.—The embolus is generally derived from the mitral or aortic valve, and consists of fibrin or a vegetation formed as a result of simple or malignant endocarditis. It may originate from a thrombus or from the interior of an aneurism of the aorta, carotid, or other vessel. Embolism may occur at any age, but generally between 10 and 40, and somewhat more frequently in women. The point of arrest is most frequently the middle cerebral artery, sometimes the anterior or posterior cerebral or the vertebral.

Symptoms.—The involvement of a small area of the brain in either of these processes may give rise to no prominent disturbance. In a more extensive embolism the patient suddenly becomes unconscious or rarely falls in a convulsion, which may prove fatal within a few hours, or it may be followed by paralysis closely resembling that of cerebral hemorrhage. In thrombosis the onset is generally more gradual, beginning with headache, vertigo, nausea, vomiting, paresthesia, confusion of ideas, and terminating in unconsciousness and paralysis. The conditions are differentiated from cerebral hemorrhage under that disease.

THE CEREBRAL PARALYSES OF CHILDHOOD.

INFANTILE PARALYSIS.

The principal forms of paralysis from cerebral lesions met with in infancy and childhood are hemiplegia, paraplegia, and diplegia, occasionally also quadruplegia.

Etiology.—Most cases occur during the first two years. Some, no doubt, originate in intrauterine life; others are caused by injury during delivery, as by the forceps, especially in unnatural positions. Falls, blows, and penetrating wounds are responsible for some cases, and others follow more or less immediately upon one or other infectious disease, especially whooping-cough and those attended with convulsions. Paraplegia and diplegia are generally congenital and nearly always observed in premature infants.

Morbid Anatomy.—The early lesions are usually those of embolism, thrombosis, hemorrhage, or one of the forms of encephalitis. Later, there is sclerosis, either atrophic or hypertrophic, limited to a few convolutions, to a lobe, or involving an entire hemisphere. The tissue is extremely hard. The membranes are adherent over the affected area, and there is often pachymeningitis or leptomeningitis. Porencephalus, or cyst-formation, is usually observed. One or many cysts are formed in the brain substance, and they occasionally communicate with the ventricles.

Symptoms.—In congenital cases there may be no symptoms except the paralysis. In either form of the disease developing after birth there may be a convulsion or sudden unconsciousness, with fever and fol-

lowed by paralysis. The paralysis is usually most profound in the upper extremities; only the lower portion of the face is affected in most cases. The electrical reaction of the muscles is retained. Recovery may occur during the course of a month or several months, but it is often incomplete. Rigidity, tremor, athetosis, epilepsy, aphasia, or an arrest or retardation of mental development is usually a sequel of the condition.

Treatment.—The treatment of the initial convulsions is the same as that of convulsions from other causes, by the administration of bromids, or chloroform inhalation when protracted, and a warm or cold bath according to the degree of fever. For the paralysis much can be accomplished by persistent, systematic massage, little, if anything, by electricity, and the child should be encouraged and taught to use the paralyzed members. The mental condition can be improved also by proper instruction in an institution for the feeble-minded or by a trained tutor. Little is to be expected, however, when a condition of idiocy exists; imbecility often increases, but when the mental development is merely retarded, remarkable results are often obtained.

ACUTE ENCEPHALITIS.

INFLAMMATION OF THE BRAIN, ACUTE CEREBRITIS.

Inflammation of the brain may be localized (focal) or diffuse; it may develop independently or in connection with meningitis. It usually results from injury, embolism, or thrombosis; poisoning by ptomains, alcohol, illuminating gas; malignant endocarditis or acute infectious diseases, particularly influenza. It is seen especially in the insane. The histological appearances are the same as those of acute poliomyelitis. In the focal form the affected areas are often intensely hyperemic, but they sometimes show little change at autopsy. The hyperemia is less marked in any case in which the brain is not examined until after the chest has been opened.

The ***symptoms*** are often obscure, resembling an acute infection, with headache, vomiting, constipation, restlessness, sometimes passing into delirium or coma. Paralyses of different types, muscular spasm and paresthesia, are often developed. The disease usually terminates fatally within a few weeks, but sometimes after several months. The treatment is that of meningitis.

SUPPURATIVE ENCEPHALITIS.

ABSCESS OF THE BRAIN.

Etiology.—The disease may occur at any age, but it is somewhat more frequent in middle-aged men. It is rarely, if ever, primary, although the cause cannot always be determined. In most cases it is a result of the extension of suppurative inflammation from contiguous parts, trauma, or embolism; occasionally of tumor. (*a*) The most frequent source of suppurative inflammation in the vicinity is a chronic otitis, especially after involvement of the mastoid cells. The abscess has been traced also to a suppurative rhinitis, abscess of the frontal sinus, and to suppuration or the injury caused by a foreign body in the orbit.

(*b*) Blows, and more particularly penetrating wounds of the skull, are a frequent cause, especially when the brain is lacerated, or when suppuration develops in the wound. (*c*) The third source of the disease is the transmission of septic emboli from a suppurative focus, notably in pyemia, malignant endocarditis, occasionally from bronchiectasis, septic pneumonia, suppuration of bone, gangrene of the lung, or abscess of the liver. It may follow an acute infection, particularly influenza, and it has resulted from ligation of the external carotid too near to its origin.

Morbid Anatomy.—There may be but one abscess or many. The solitary abscess usually varies from an inch to two inches (2.5—5.0 cm.) in diameter; rarely a lobe or hemisphere is almost completely excavated by it. Multiple abscesses are usually small. All the pus-forming bacteria may be found in the pus, and other micro-organisms are occasionally met with. In the rapidly fatal cases the suppuration is diffused, but in cases of long standing a distinct wall is formed. The pus has an acid reaction, may be tinged with blood and the débris of the brain substance, but it has usually a greenish color and a strong odor of hydrogen sulphid. Any part of the brain may be the seat of suppuration, especially the temporal lobe and the cerebellum. The overlying membranes are involved in the inflammation when the abscess is near the surface. The surrounding brain substance is inflamed or degenerated and always compressed.

Symptoms.—The symptoms may develop suddenly with chills and fever, or a convulsion in a child, headache, nausea, vomiting, delirium or coma, especially in traumatic cases or those due to otitis. In other cases the manifestations are exceedingly vague or they may resemble meningitis or meningoencephalitis for days and weeks, until localizing symptoms develop. These frequently do not appear, however, until a large area of brain substance has been destroyed, especially in the anterior lobe of the cerebrum. The pupils may be dilated or unequal and optic neuritis may exist. Hemianopia has been observed. The temperature is not high when the membranes are not involved, and it may be subnormal, even as low as 97° or 96° F. (36.0°—35.6° C.) until near the termination of the disease. The pulse may be accelerated, but is often slow as 60 or less. Leucocytosis is present.

The localizing symptoms depend upon the situation of the abscess, and may be exceedingly confusing when several foci of suppuration are present. They consist of different paralyses, aphasia, irregular respiration, rigidity, spasm, convulsions, and other manifestations of cerebral disease. The cranium may be tender, especially upon percussion, and slight resonance over the compressed portion of the brain has been described. Symptoms of sepsis may develop toward the termination of the disease.

Diagnosis.—The diagnosis is usually difficult, except in traumatic cases. The persistent, severe headache, stupor, gastric irritability, low temperature, leucocytosis, inequality of the pupils, retinitis, delirium, and the localizing symptoms arouse suspicion of the disease, but do not always afford data for a differentiation from brain tumor unless a probable cause of abscess has been discovered. When these symptoms follow injury or suppuration, and when chills or convulsions with fever supervene, the diagnosis is practically established.

Prognosis.—The disease is fatal except in those cases in which the abscess can be evacuated. Death may occur within a week or two, or not for several months.

Treatment.—The medical treatment is purely palliative. Remarkable success has followed evacuation and drainage of the abscess in some cases.

CHRONIC MENINGOENCEPHALITIS.

DEMENTIA PARALYTICA, GENERAL PARESIS, PROGRESSIVE PARALYSIS OF THE INSANE.

Definition.—A slowly progressive inflammation and degeneration of the brain and meninges characterized by psychical and motor disturbances and leading to dementia and paralysis.

Etiology.—The disease is more frequent in men than women, and between the ages of 20 and 50. It is a product of the mental strain and worry, the ambition and constant struggle, the "strenuous life" of our age, but in more than three-fourths of all cases it attacks individuals who have been the victims of syphilis. Alcoholism is a most potent cause in the syphilitic subject. Sexual excess, loss of rest, deferred hopes, disappointment, and business reverses are important factors in many cases. The frequency of the disease has doubled in the last half-century.

Morbid Anatomy.—The disease usually begins with inflammatory changes in the adventitia of the blood-vessels of the brain, and degenerative changes in the brain substance follow. The membranes are found in a state of chronic diffuse meningitis, thickened, opaque, and hyperemic. The pia is also opaque, thick, and adherent to the cortex, but may later become detached. As the disease progresses, the hemispheres undergo atrophy, particularly in the frontal and parietal convolutions, so that they appear small and weigh less than normal. The gray matter is especially reduced. Hemorrhagic points or areas of pigmentation are generally found. The microscope reveals fatty and other degeneration of the nerve elements and hyperplasia of the connective tissue.

Symptoms.—The clinical history is divided into three stages, which are usually fairly distinct—the prodromal, the maniacal, and the stage of dementia.

1. **Prodromal Stage.**—The important feature of this stage is a change, a deviation, in some particular from the normal traits, habits, or disposition of the individual. There may be merely an exaggeration of the natural characteristics, or a complete reversal of them. The business man becomes unnaturally ambitious and optimistic in his affairs, or careless and indifferent, forgetful of engagements and promises, or reckless in investments. The man of gentle manners becomes more quiet and reserved, or rude, profane, vulgar, and utterly lawless. His conduct at home is often the reverse of that among other associates. He becomes irritable and morose, abuses his family, and possibly deprives them of all comforts, to spend his wealth lavishly upon the courtesan and other new associates. In many cases there is extreme exaltation. A man of moderate means boasts of fabulous wealth. He subscribes freely to charity, buys blocks of the most worthless stocks, and fre-

quently squanders a fortune before his condition has been recognized. A characteristic feature of the disease is the fact that the patient cannot be forced into an explanation of his suddenly acquired wealth. Although previously showing little affection, he may suddenly become extremely fond of his wife and children and boastful of them in the presence of others.

In many cases even during this stage a tremor of the tongue and lips is apparent, and the patient may speak with unusual deliberation and some difficulty. He generally becomes fatigued after slight exertion. The Argyll Robertson pupil or an unequal dilatation may be present.

2. The **acute or maniacal stage** is marked by an exaggeration of all the features of the prodromal. His egoism becomes expanded until his wealth is no longer within the bounds of figures; his strength is that of a Hercules, and he is in a constant state of excitement, restless, sleepless, often furious and violent. In some cases, however, the condition is directly the reverse, and the patient becomes melancholic and hypochondriacal, or he may have alternating attacks of depression and buoyancy, mania, or delirium. The face assumes a fixed expression, the tongue and lips become more tremulous, so that the tongue may be protruded with difficulty, and the speech becomes drawling and indistinct. The handwriting is also tremulous and irregular, and words or parts of words are omitted. The eye symptoms are more constantly observed in this stage, and the tremor affects also the fingers and toes. All these symptoms are subject to periodical exacerbation and remission. The knee-jerk is usually increased, but it may be normal or absent. Transient incontinence of urine sometimes occurs.

The evidences of paresis are often first seen in the face as a partial obliteration of the nasolabial fold, or in the tongue as a slight deviation to one side. Spinal symptoms like those of locomotor ataxia sometimes precede the mental, and the patient has difficulty in walking, especially in going up and down stairs.

3. In the **stage of dementia and paralysis** the delusions of grandeur and wealth give place to emotional disturbances, sometimes characterized by great religious fervor, sometimes by deep melancholia and depression. Epileptic seizures or brief attacks of petit mal sometimes occur as the disease progresses. The face becomes flushed, the breathing stertorous, and there is brief unconsciousness, or the patient may fall and the paroxysm may prove fatal.

Paralysis soon becomes a prominent feature, and the patient becomes bedridden, the helplessness increases, emaciation becomes extreme, and extensive bedsores develop. Death finally ensues from exhaustion or an intercurrent disease, particularly bronchopneumonia.

Diagnosis.—The diagnosis is based upon the gradual change of disposition and habits, the delusions of grandeur or depression, the tremor, ocular symptoms, and the affection of speech. In the later stages there is rarely difficulty in its recognition. *Cerebral syphilis* is generally to be distinguished by the early development of paralysis, not usually affecting the speech or the tongue. Epileptic seizures are more common and occur earlier, and delusions are not usually present.

Prognosis.—The disease terminates fatally in from one to five years, as a rule, seldom before two years, but occasionally exceeding ten.

Treatment.—Potassium iodid should be administered early in full doses, whether or not a history of syphilis can be elicited. The nervousness and sleeplessness call for the administration of large doses of the bromids. The bowels should be kept active. The patient should be early sent to an asylum, where the regular life and constant attention often add much to his comfort and prevent unfortunate accidents. In the last stages great care must be exercised to prevent bedsores. When so desired, this stage may be passed in the care of a good nurse at home.

SCLEROSIS OF THE BRAIN.

Etiology.—Sclerosis is generally a disease of early adult life, but it has been repeatedly observed in children, and a few congenital cases have been described. It may be caused by any influence which is capable of producing irritation of the connective tissue of the brain, the most prominent of which are: (*a*) The toxins of the infectious diseases, particularly syphilis; (*b*) metallic poisons, especially lead; (*c*) acute inflammation (encephalitis); and (*d*) degeneration of the nerve-fibers. (*e*) A capsular area of sclerosis forms also around foreign bodies, abscesses and tumors, as in other situations.

Morbid Anatomy.—The process may involve the neuroglia, fibrous tissue of the meninges, and walls of the blood-vessels. The lesions are grouped under four heads: (*a*) The diffuse, affecting all parts of the brain and peculiar to idiocy and imbecility; (*b*) the miliary, in which exceedingly small sclerotic areas are scattered over the surface or throughout the substance of the brain; (*c*) the tuberous, in which hypertrophic sclerosis produces large white nodules on the surface of the convolutions; and (*d*) insular sclerosis. It is only the last of these that produces definite manifestations.

INSULAR SCLEROSIS.

MULTIPLE CEREBROSPINAL SCLEROSIS, DISSEMINATED SCLEROSIS, SCLEROSE EN PLAQUES.

Definition.—A chronic disease of the brain and cord due to the development of disseminated sclerotic areas at the expense of the nerve-tissues.

Etiology.—The disease attacks either sex at the ages and as a result of the influences already stated. Syphilis and the acute infectious diseases, particularly scarlatina, are the most important factors. Some cases follow exposure to cold and wet.

Morbid Anatomy.—The sclerotic areas are firm and of a reddish gray color, regular contour, discrete or confluent, and consist of reticulated connective tissue. They may be found in either the gray or white matter and are generally found in the walls of the ventricles, in the centrum ovale, corpus callosum, septum lucidum, optic thalamus, corpus striatum, sometimes in the cerebellum, pons and medulla, and throughout the spinal cord. In some cases the brain or cord is affected alone; in others both are involved. Arteriosclerosis is also associated with these lesions. The myalin sheaths are early destroyed, but the axis cylinders (axons) remain normal.

Symptoms.—The disease usually develops slowly, often with (*a*) slight weakness and incoordination, and possibly some pain in the lower extremities. The reflexes are increased and the condition may resemble spastic paraplegia. With this, or before it, (*b*) a tremor develops which is known as the intention tremor or volition tremor, from the fact that it occurs only when voluntary movements are made, as when the patient attempts to write or to lift a glass of water to his lips. It usually disappears when he is at rest. It is ordinarily a fine tremor, but it may become extremely coarse when strong efforts are made. (*c*) Slight paresis generally accompanies the tremor; the grasp of the hand is feeble. (*d*) Nystagmus usually develops during voluntary movement. (*e*) Scanning or staccato speech is one of the most important symptoms. The patient hesitates, tremulous movements of the tongue and lips occur, and the words are uttered slowly and separately, or each syllable is accented. (*f*) Other more or less constant symptoms are vertigo, atrophy of the optic nerve, and a great variety of symptoms of a localizing character depending upon the situation of the lesion and embracing the manifestations of nearly all the other diseases of the central nervous system. The course of the disease is exceedingly chronic; it may terminate suddenly in an apoplectic seizure similar to those of paresis, or the latter disease may develop.

Diagnosis.—The disease is distinguished from paralysis agitans by the absence of tremor during rest and the occurrence of nystagmus. From general paresis it is distinguished by the absence of early delusions and the ocular symptoms. Hysteria sometimes imitates the other symptoms, but seldom the intention tremor and never the nystagmus.

Prognosis.—The disease is incurable, but it may last for many years with intervals, and the patient finally succumbs to exhaustion or an intercurrent disease.

TUMORS AND CYSTS OF THE BRAIN.

Etiology.—Tumors may occur at any age, but rarely before the tenth year, and most frequently between the twentieth and fortieth. Males are more commonly affected. Tubercular granulomata are more frequent in childhood, syphilitic in adult life. Sarcoma and carcinoma are generally met with at a later period than these. Heredity can seldom be traced except in tuberculosis and cancer. Some tumors follow prolonged nerve-strain, and others can be traced to injury.

Morbid Anatomy.—The growth may be single or multiple, benign or malignant, and histologically may belong to the epithelial, connective-tissue, or nerve-tissue type. Tubercular, syphilitic, and aneurismal growths are more frequent forms of multiple neoplasms. Enchondromata and osteomata develop from the bones of the skull or from the falx cerebri. Gliomata, fibromata, myxomata, lipomata, cholesteomata, adenomata, angiomata, and neuromata are occasionally found. The ray fungus is sometimes found as an invasion from the face. Of cysts, the cysticercus, echinococcus, and cavities formed by congenital atrophy, hemorrhages, or the softening due to disease are the usual types.

Symptoms.—The clinical manifestations are of two kinds, general and localizing or focal.

1. *General Symptoms.*—Persistent headache is the most frequent of these. It is not usually violent, but it is often accompanied with tenderness. Vertigo and projectile vomiting, often without distinct nausea, are usually persistent symptoms. Local or general convulsions of an epileptic type are present in many cases. Optic neuritis (choked disk) is found in a majority of cases, estimated by some authors as high as 60 to 80 per cent, at some time in the course of the disease, yet mania, delirium, and the eye symptoms of general paresis are sometimes observed. As a rule, however, they are more in the nature of hebetude. Somnolency or coma may develop. Variations in the character of the pulse and respiration often occur, and the pupils may be affected, but in no characteristic manner. Fever is not present. Trophic changes, as bedsores and paralysis of the sphincters, often develop toward the close.

2. *Focal Symptoms.*—These depend wholly upon the situation of the growth. They may be indirectly produced by pressure, however, in the larger tumors. Small neoplasms confined to a single area produce the most typical symptoms, among which the following may be studied:

a. Motor Area.—Localized muscle spasms, followed by monoplegia, or more extensive convulsive movements with other types of paralysis. Spasm with a tingling sensation confined to a single muscle group has been denominated a *signal* symptom by Seguin.

b. Psychical Centers.—The frontal lobes were formerly regarded as the seat of the metaphysical mind, but at the present time the integrity of the mind is believed to depend upon the total integrity of the cortex; hence any destructive lesion of this area may be attended with mental disturbance.

c. Prefrontal Region.—Mental disturbances and sometimes disorders of the sense of smell.

d. Third Left Frontal Convolution.—Motor aphasia. The patient understands the meaning of words, but cannot utter them properly.

e. Island of Reil.—Aphasia of conduction. The patient utters words entirely different, sometimes having a contrary meaning to that he desires to express.

f. Parietal Region.—Word-blindness or mental blindness. In the former the patient can repeat or write a word, but cannot comprehend its meaning; in the latter he sees objects, but cannot recognize them.

g. Temporal Region.—Word-deafness or psychical deafness. Words are heard, but not comprehended. Symptoms may, however, be absent.

h. Occipital Lobe.—Hemianopia, or total blindness when both sides are affected. When on the left side, word-blindness or psychic blindness may be produced.

i. Basal Ganglia.—Hemiplegia of the opposite side, sometimes hemianopia.

j. Corpora Quadrigemina (Usually Involving also the Crura).—Nystagmus with loss of pupil reflexes, hemiplegia of the opposite side, and oculomotor paralysis on the same side.

k. Cerebellum.—Symptoms may not be produced, or there may be vertigo, projectile vomiting, optic neuritis, pain in the cervical region, and incoordination with a peculiar reeling gait, always turning toward the same side.

l. Pons and Medulla.—Involvement of the cranial nerves, dyspnea,

disturbance of the heart's action, sometimes hemiplegia, sensory and other disturbances.

Diagnosis.—The gradually increasing intensity of the general symptoms described is ordinarily sufficient for diagnosis. It should be remembered, however, that chronic nephritis, lead-poisoning, tobacco, myelitis, multiple sclerosis, and other affections are often attended with symptoms suggestive of brain tumor.

Prognosis.—All forms of brain tumor are fatal except the tubercular and syphilitic. Death may occur suddenly or it may be delayed for many months. Some cases are amenable to surgical treatment.

Treatment.—The therapeutic test of large doses of potassium iodid should be made in every case. Temporary improvement is often obtained even in nonsyphilitic cases. When this fails, surgical measures should be considered. Operation is indicated only in definitely localized tumors of the dura or cortex. An exploratory operation is generally justifiable. In other cases the treatment is palliative, limited to measures for the comfort of the patient. Spontaneous recovery sometimes occurs in a tuberculous case, and it may be assisted by the administration of tonics and the general measures for the treatment of tuberculosis. An ice-cap should be worn to relieve the headache. Large doses of the bromids and the coal-tar preparations may be tried, but they generally fail to relieve it. Morphin should be administered in a hopeless case.

APHASIA.

Definition.—Aphasia, or loss of the power of speech as a result of cortical lesions, is a not infrequent accompaniment of diseases and injury of the brain. It may be motor or sensory in character.

Etiology.—The causes embrace all the causes of cortical affections, particularly trauma, hemorrhage, arteriosclerosis, embolism, thrombosis, and tumors, especially when the third frontal convolution is involved. Different varieties of aphasia are recognized as a result of disconnection of the centers of hearing, sight, and motion, involved in the comprehension of language and production of articulate sound.

Symptoms.—1. **Motor Aphasia** (Ataxic Aphasia.)—The lesion in this form of aphasia is generally situated in the posterior part of the third left frontal convolution. The individual comprehends words and can remember them; in rare cases he is able to write them and to understand what is written, though agraphia is more frequently associated with the condition, yet he is unable to utter a word. In many cases the ability is retained to utter a few words, often only a single word, and every effort to speak calls forth the stock word or phrase. In another group of cases there is what is termed aphasia of conduction. In this also the patient understands the meaning of words and can write from dictation, but cannot express them.

Word-dumbness is a rare form in which the individual can read to himself, but can neither speak spontaneously nor repeat words that are spoken to him.

Transcortical motor aphasia is another rare form in which the patient understands what is said, can read aloud and write from copy or dictation, but cannot speak or write from his own volition.

2. **Sensory Aphasia.**—In this form of the affection, often termed mind-blindness, visual amnesia or apraxia, there is an interruption of the auditory and visual communication with the motor centers of speech. In many cases, indeed, the perception conveyed by any of the senses fails to excite the centers of speech, and may fail entirely of recognition. The patient sees, hears, smells, tastes, and touches objects, but cannot recognize or name them. In rare instances recognition remains through one of the senses, particularly that of touch. The patient may be able to read aloud without understanding what he reads. The situation of the lesion is believed to be in the angular and supramarginal convolution. Several other terms are employed to designate peculiar conditions, as word-blindness when the patient does not comprehend written or printed words. He may be able to write correctly, but cannot read what he has written.

Word-deafness is a condition in which the patient cannot understand spoken language.

In *mind-deafness* the patient does not comprehend sounds. The ringing of a bell or the barking of a dog fails to arouse a concept of the object producing the sound.

In *apraxia* proper there is a dissociation, not only of the centers involved in the use of language, but of all the sensory centers, and the individual can neither recognize objects nor make proper use of them.

Agraphia is the term applied to the condition in which the individual is unable to express his thoughts in writing. Alexia signifies an inability to understand words which the patient may be able to read aloud. In most cases two or more of these conditions are associated.

Prognosis.—The completeness of recovery depends in part upon the character of the lesion and in part upon the age of the patient. Young subjects usually recover, probably through the education of centers on the opposite side of the brain. In adults with hemiplegia the prospect is less favorable. Sensory aphasia is generally less permanent than motor aphasia.

Treatment.—The treatment of the lesion is that of hemiplegia. The aphasia is to be overcome, if at all, through the re-education of the patient as in childhood.

HYDROCEPHALUS.

1. **External or Subdural Hydrocephalus.**—A chronic condition in which the arachnoid space is distended with fluid. It may be a congenital condition, a result of arrested brain development, or excessive growth of the skull. In other cases, without atrophy, the subdural fluid becomes excessive and the cranium enlarges without recognizable cause. In some instances the fluid becomes sacculated through the formation of adhesions, doubtless inflammatory, between the dura and pia. This may lead to unilateral dilatation of the skull. Hydrocephalus may result from rickets in childhood and may follow hemorrhage, softening, or sclerosis in adults. It is most frequent, however, in advanced life as a result of atrophy (hydrocephalus ex vacuo). The cranium becomes exceedingly thin in children, and the fontanels widely open. The dura may remain unchanged, but the cerebral cortex is thinned through pressure.

The ***symptoms*** are variable; in some cases there are no distinctive manifestations. In children, however, idiocy is generally produced; in the aged, a condition of dementia.

2. **Internal or Ventricular Hydrocephalus.**—A chronic distention of the ventricles of the brain with serous fluid.

Etiology.—The condition may be congenital or acquired. In the former class of cases it sometimes appears to be hereditary and may affect several members of a family. The acquired form usually results from meningitis, tumor, abscess, obstruction of the venæ Galeni, or from an unknown cause. Tumors sometimes operate by preventing the escape of fluid from the ventricles.

Morbid Anatomy.—In congenital cases the cranium becomes greatly distended and thin, the fontanels enlarged, and the sutures separated. Wormian bones may develop in the spaces. Changes in the brain result from the compression. The fluid may be confined to the fourth ventricle alone, to the third and lateral ventricles, or to the lateral alone. The quantity of fluid varies from a few ounces to several quarts. It is albuminous and has a specific gravity of 1.010 or 1.012. In the acquired form, occurring in adult life, the quantity of fluid is necessarily less, and the compression of the brain substance may be more extensive owing to the rigidity of the skull.

Symptoms.—In the congenital form the enlargement of the skull gives the child a typical appearance. It sometimes reaches a circumference of twenty-four to thirty inches (60 to 75 cm.). The fontanels are widely open, the sutures separated, and the tables of the skull may be so thin as to be indented by slight pressure. The orbital plates are depressed, and exophthalmos is produced. The subcutaneous veins become prominent. The face appears diminutive in comparison to the skull, and the expression is aged.

Various nervous manifestations may occur, as choreic movements, nystagmus, optic neuritis, conjugate deviation of the eyes, sometimes convulsions. The child is unable to support the weight of the head, and rolls it from side to side. The body becomes emaciated. Arrest of intellectual development may occur, but the mind may remain active. Some cases are bedridden, others can walk.

The symptoms in adults are those of brain tumor without localizing manifestations. Headache, stupor or coma, optic neuritis, and incoordination are usually present.

Diagnosis.—In cases occurring in children the differentiation must be made from rickets. This is not usually difficult, since the enlargement of the epiphyses and other bone changes are not present unless the two diseases are associated, as is not infrequently the case. In the adult the differentiation from brain tumor depends chiefly upon the absence of focal symptoms, and the gradual development of optic neuritis.

The ***prognosis*** is exceedingly unfavorable. Death may occur suddenly after repeated attacks of coma, sometimes lasting for several weeks.

Treatment.—Little can be done for the relief of the condition. When the pressure symptoms become urgent, aspiration of the ventricles may be antiseptically performed. Lumbar puncture is a safer procedure and may prove effective. Strapping and compression of the skull with rubber or adhesive bands is sometimes practiced, but it can operate

only by increasing the pressure upon the brain. Potassium iodid and mercury are generally administered, but without marked or permanent benefit, except, perhaps, in some cases in infants with syphilitic taint. The bromids should be employed to relieve the restlessness.

FUNCTIONAL NERVOUS DISEASES.

ACUTE DELIRIUM.

ACUTE MANIA, TYPHOMANIA, BRAIN FEVER, BELL'S MANIA.

Definition.—A rare and rapidly fatal form of delirium, characterized by intermittent outbreaks of violent maniacal excitement.

Etiology.—This affection is generally encountered in distinctly neurotic individuals, often in those who have been subject to hysteria, neurasthenia, or insanity. The exciting causes are injury, chronic alcoholism, autointoxication, acute infection, especially typhoid fever or pneumonia, and sunstroke. Many cases strongly suggest the probability of infection.

Morbid Anatomy.—Hyperemia of the cerebral and spinal meninges is found after death, sometimes the gray matter is congested. Granular degeneration of the cortical ganglion cells and engorgement of the perigangliónic spaces with leucocytes have been observed by Spitzka. Hypostatic congestion of the lungs or deglutition pneumonia is frequently found.

Symptoms.—The disease may be divided into three stages—the prodromal, the stage of excitement, and the stage of collapse.

1. *Prodromal Stage.*—This is usually of short duration, varying from a few hours to several days. The patient presents symptoms like those of profound autointoxication; headache, restlessness, stupor, furred tongue, fetid breath, and constipation, and he becomes rapidly emaciated.

2. *Stage of Excitement.*—There is a sudden violent outbreak, in which the patient has hallucinations of sight and hearing, screams, strikes, kicks, and threatens the lives of those about him, but rarely does violence to any but himself. In several instances he has gnawed off a part of his finger. He may escape from his attendants, naked or clothed. There is persistent insomnia. The temperature rises to 103° F. or higher. The pulse reaches 120 or more and is feeble. The patient soon sinks into a typhoid state bordering on collapse. Hyperesthesia, carphology, and subsultus are generally prominent, and the reflexes are increased. Several repetitions of the attack may occur before the final stage is developed, or death may occur after the first seizure. In a few instances deep melancholy or paresis, with cyanosis and sweating, takes the place of mania.

3. *Stage of Collapse.*—This stage is all that the name implies. The patient is completely exhausted; his temperature remains high, the pulse rapid and extremely feeble, the respiration irregular and weak, the pupils dilated, and the face expressionless. In a few cases the consciousness returns at intervals, but a profound coma soon ensues.

Diagnosis.—The sudden development of violent delirium, with fever and emaciation, renders the disease easy of recognition. In *meningitis* the invasion is slower, the delirium less violent and generally of a muttering

type. There are photophobia and retraction of the head. *Typhoid fever* can be distinguished by its slow onset, the rose spots, enlargement of the spleen, and Widal reaction. *Acute mania* is not accompanied with fever or so rapid emaciation. *Alcoholic delirium* is seldom so violent, there is little or no fever, and it occurs only in those addicted to excess. It must be remembered, however, that acute delirium often attacks these individuals.

Prognosis.—The disease is almost invariably fatal, usually within a few days, or the patient is left with a mental defect which often passes into dementia or general paresis.

Treatment.—The bowels should be thoroughly evacuated by calomel, followed with a saline cathartic. The bromids of ammonium, sodium and potassium, gr. x (0.65) of each, should be given every three hours. A few doses of hyoscin, gr. 1-100 (0.0006), should be given during the acute stage. Dram doses of the fluid extract of ergot every two hours have been highly recommended. An equivalent dose of ergotin may be administered hypodermically. Osler advises bloodletting, even in the presence of bodily prostration. Cold baths or the cold pack has a quieting effect.

PARALYSIS AGITANS.

SHAKING PALSY, PARKINSON'S DISEASE.

Definition.—A chronic, incurable affection of advanced life in which there is a tremor of the muscles, with gradual loss of power and increasing rigidity, a characteristic gait, and sensory disturbances.

Etiology.—The disease rarely develops before the fortieth year, but may begin as early as the twentieth or late as the seventieth. Men are somewhat more frequently affected. A tendency to nervous disease can sometimes be traced. Among the exciting causes are emotional disturbance, shock, care, worry, fatigue, exposure to cold and wet, trauma, as wounds and lacerations, and specific fever, as malaria.

Morbid Anatomy.—No lesions in any way distinctive of the disease have been found in the nervous system.

Symptoms.—The characteristic symptoms are tremor, weakness, rigidity, attitude, and gait. *Tremor* begins slowly, usually as a fine, constant or intermittent tremulousness of the fingers of one hand, often the left. The foot of the same side next becomes affected. In some cases it is confined to the hands alone or to the feet alone. As a rule, the hand becomes affected. The face-muscles escape the tremor, but not the rigidity. The tremor ceases during sleep, and until the disease becomes advanced it can be more or less completely arrested for a short time by the will and by voluntary motion. All the exciting causes, fatigue, fright, etc., are capable of increasing it. The movements are typical. The thumb and finger have the movement of rolling a pill; the wrist has all the motions of pronation, supination, flexion, and extension. The head nods. The rate of the tremor is five or six in the second.

Weakness.—This may be noticeable at the beginning, but is more marked in the late stages. It is always a relative, not a complete, loss of power.

Rigidity.—This affects all the muscles to an increasing degree and renders all movements slow and deliberate.

Attitude and Gait.—The patient stands with the body inclined forward and the hands drooping in front of him. As a result of this attitude there is a tendency to move forward (propulsion). Rarely there is backward movement (retropulsion), and more rarely lateral movement (lateropulsion). The gait is quick and shuffling. The face becomes expressionless, the eyebrows elevated, the voice shrill and piping. The patient hesitates in the beginning of speech, then speaks rapidly.

The reflexes are normal or slightly increased. The sensory disturbances are usually limited to a sense of heat or cold, often confined to one side. Localized sweating often occurs.

Diagnosis.—The symptoms are so distinctive as to leave little possibility of error. *Disseminated sclerosis* develops in younger subjects, as a rule. The tremor occurs during motion and ceases during rest. The nystagmus, scanning speech, and paralysis are typical. *Postparalytic tremor* is generally unilateral and accompanied with paralysis and

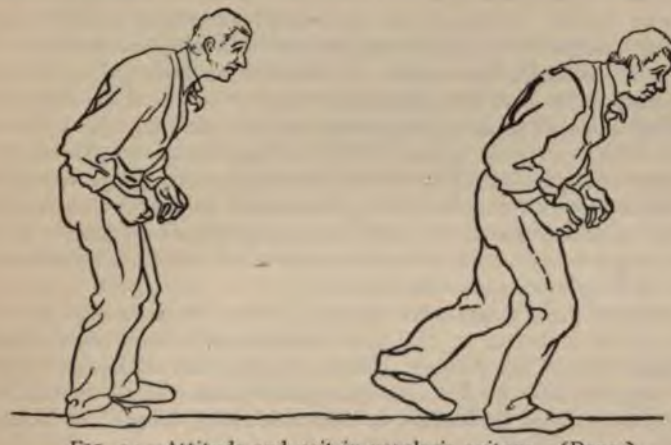

FIG. 24.—Attitude and gait in paralysis agitans. (Dana.)

greater rigidity of the affected muscles. *Senile tremor* is limited to the head, hands, and fingers in extreme old age.

Treatment.—The patient should be freed from care and worry. The tremor is sometimes diminished by the administration of hyoscin hydrobromate. Morphin or codein is more effective, but should not be used until it becomes imperative. The administration of arsenic has been followed by prolonged improvement in some cases. Frequent warm baths, with friction, massage, and galvanization of the limbs and spine, are sometimes of benefit.

OTHER TREMORS.

Simple tremor develops in those debilitated by illness, overwork, inanition, or without discoverable cause,and may be of short or long duration.

Toxic tremor follows the excessive use of tobacco or alcohol and poisoning by lead or other metals, and affects chiefly the hands during voluntary motion. The tongue is tremulous in alcoholic cases.

Hereditary Tremor.—Cases of this character have been reported by C. L. Dana, affecting the children of one family from infancy.

Senile tremor is a fine tremulous movement of the fingers, hands, and head in extremely old persons during voluntary movement.

Hysterical tremor usually accompanies other manifestations of hysteria. It affects the face and fingers chiefly, and ceases when the attention is diverted.

ACUTE CHOREA.

CHOREA MINOR, SYDENHAM'S CHOREA.

Definition.—A functional disorder of the nervous system in childhood and youth marked by a wavy contraction or sudden twitching of muscles, a variable degree of mental disturbance, and sometimes accompanied by endocarditis.

Etiology.—The disease is doubly more frequent in females and the ratio increases after puberty. An inherited neurotic tendency is nearly always to be traced. It rarely if ever affects the negro in our country. A rheumatic tendency is often found in the individual or family. Other infections, as malaria, pertussis, or scarlatina, anemia or eyestrain, sometimes appear to bear an etiological relation to it. The disease may occur during the first half of pregnancy or after delivery, particularly in connection with puerperal sepsis. The exciting cause in about 20 per cent of cases is fright, nervous strain, or injury, and many cases begin through imitation of those affected. Occasional epidemics are attributed to the last of these influences. Sudden pronounced barometric disturbances with high humidity of the atmosphere are believed to induce the attack or a relapse in some instances.

Many writers have supported the theory that the disease is due to the lodgment of an embolus in the smaller cerebral vessels as a result of endocarditis. While the theory offers a plausible explanation of the disease, it cannot be applied to all cases, for endocarditis is often absent; and when it is present, embolism is not uniformly found. A theory of infection is supported by some authors.

Morbid Anatomy.—The changes which have been most frequently found in the nervous system are congestion, extravasation, embolism, and softening. Hyalin degeneration of cells, perivascular exudations of leucocytes, minute hemorrhages, and thrombosis have also been discovered, but none of the lesions is constant. Simple or malignant endocarditis is often present.

Symptoms.—The disease may be mild throughout, or severe, even maniacal, and cases of the former type sometimes develop into the latter. In the mild form only a few groups of muscles may be affected, and the movements so slight as to be discovered with difficulty. In many cases there is little more than an inability to sit in repose for more than a moment at a time. The affection begins with twitching of the hands, arms, or face, and soon involves the lower extremities. Sometimes the movements are unilateral, often confined to the right side; or, beginning in this manner, they may later involve the other side and become general. They can be, to a considerable extent, controlled by the will, and do not prevent voluntary action; but they are increased by excitement, fatigue, and imitation, and they are always worse when the patient is conscious

of being observed. Anemia, digestive disturbances, and muscular weakness are generally present, and the patient often becomes fretful, irritable, and restless during sleep.

The severe form develops suddenly or follows a mild onset, and frequently manifests itself as a constant violent action of all the muscles, so violent in rare cases that the patient must be placed in a padded cell (chorea insaniens), where he pitches and tosses about like one "possessed of many demons." Fever is often present. The distress of the patient is greatly aggravated by his inability to sleep, eat, drink, or perform any voluntary act of more than a moment's duration. The voice is affected and speech may become impossible. Between these violent cases and the mildest there is every grade of severity. Some of the most violent and fatal cases are those occurring during pregnancy.

Cases have been observed in which more or less pronounced paresis existed with slight movements (paralytic chorea), and monoplegia or paresis may persist after recovery. The affected muscles are often slightly painful and tender to pressure.

Heart-murmurs can be heard in about half the cases. In many they are significant of endocarditis, but in some cases they are hemic, due to the anemia. The heart's action is rapid, but not, as a rule, irregular. Such cutaneous eruptions as urticaria, herpes, purpura, and rarely subcutaneous fibrous nodules, are observed.

Diagnosis.—Few diseases enter into the differential diagnosis of chorea. *Disseminated sclerosis* is distinguished by the constant tremor rather than twitching of the muscles, the nystagmus, and scanning speech. The tremor associated with *brain-tumors* presents finer movements, and there is headache with focal symptoms not seen in chorea. The *hysterical tremor* is more uniform, and it is accompanied with other sensory and motor symptoms of the disease.

Prognosis.—Recovery occurs in all but the most violent cases. A favorable prognosis should not be too hastily pronounced, however, owing to the possibility of a mild case assuming a violent form.

Treatment.—The child should be confined to bed in a cheerful room, and quietly entertained in such manner as will afford it the greatest rest of body and mind. Too great restraint is not profitable; punishment is injurious. All associates should be excluded. In a severe case complete rest must be afforded and potassium bromid, with chloral, if necessary, should be given in order to reduce irritability and promote sleep. Arsenic should be given in all cases, beginning with from one to three drops of Fowler's solution and increasing a drop each day, or less rapidly in the case of a young child, until slight symptoms of excess are produced. It should then be discontinued for a few days, and resumed in smaller doses followed by gradual increase. The danger of too prolonged use of arsenic must be guarded against. Hyoscyamin, physostigmin, cimicifuga, quinin, belladonna, the salts of zinc, and other remedies are highly recommended by different writers. Strychnin in small doses is useful as a tonic to the muscles. The diet should consist of the most digestible food and the bowels should be regulated. The patient need not be confined to bed all day after improvement has been obtained, but he should not be set at liberty until all choreic movement has ceased, and only moderate exercise should be permitted for several weeks.

CHOREOID AFFECTIONS.

Chronic chorea (Huntington's chorea) is a disease of adult life, usually appearing after the thirtieth year, sometimes inherited, in which there is slow movement of inco-ordination in the hands, face, and legs, accompanied with progressive dementia, and often a suicidal tendency.

Hysterical chorea is usually limited to rhythmical movements of certain groups of muscles, often consisting of a constant nodding of the head or, by affecting the abdominal muscles, producing salaam swaying of the body.

Chorea major is a form of hysterical chorea which was prevalent in the Middle Ages (St. Vitus's or St. Anthony's dance).

Saltatory chorea (latah, jumpers) is probably also a form of hysterical chorea in which the individual suddenly springs forward when he rests his weight upon his lower extremities, and sometimes utters a sharp cry. It is extremely rare in this country.

Habit chorea (habit spasm) consists in a frequent, sudden movement of one or more groups of muscles, especially those of the face, shoulder, or head, usually appearing in childhood and sometimes persisting through life. In some cases respiration is affected, producing sniffling or the sound of hiccough. It is increased by excitement or fatigue.

Posthemiplegic chorea is a jerking of paralyzed muscles usually accompanied with anesthesia and contractures.

Athetosis (Hammond's disease) is a form of postparalytic chorea consisting of rhythmical movements of the fingers and toes, sometimes of the mouth. Contractures and subluxations of the phalangeal joints are sometimes produced.

Convulsive tic (Gilles de la Tourette's disease) is a psychosis of neurotic children resembling, if not belonging to, hysteria. Irregular, sometimes violent movements affect the face and arms or the entire body. The seizures may be accompanied with an inarticulate outcry or the continued repetition of a sound or word that is heard (echolalia), or of profane or vulgar language (coprolalia), and actions may be mimicked (echokinesis). In some cases also there is a fixed idea that certain actions must be performed at definite times, that some object must be touched or a certain number counted before some other act can be performed (arithmomania). The disease is often persistent, but recovery sometimes occurs.

Complex tic is a name applied to many peculiar, more or less rhythmical movements occurring in idiots and imbeciles.

Treatment.—The treatment of all these conditions should begin early. More can be accomplished by moral and hygienic means than with medication. As in chorea, punishment aggravates the condition. Arsenic and strychnin should, as a rule, be administered.

CONVULSIONS OF CHILDREN.

INFANTILE CONVULSIONS, ECLAMPSIA.

Definition.—Convulsive seizures like those of epilepsy, generally due to reflex irritation or toxemia, and sometimes developing into epilepsy.

Etiology.—There is in many cases an inherited or acquired predisposi-

tion to convulsive seizures, especially in the children of neurotic, epileptic, or drunken parents.

The exciting causes are many: (1) Irritation of the nerve-centers by the toxins of the acute infectious diseases, the convulsion replacing the chill which occurs in adults. (2) Aside from the toxemia developing in the prodromal stage of the acute infections, the most frequent cause of convulsions in young children is probably gastrointestinal irritation. It is impossible to say, however, to what extent this irritation is obstructive and produced simply by the irritation of the stomach and intestines, and to what extent it is due to ptomain-poisoning. Other forms of intestinal irritation capable of exciting convulsions are the presence of improper food and such foreign bodies as the intestinal parasites. In the absence of a more definite cause the attack is often attributed to teething, phimosis, heat-eruption, and other peripheral irritations. (3) Intense paroxysms of fright, anger, crying, or coughing, as in pertussis, sometimes produce convulsions as a result of cerebral congestion. (4) The malnutrition and debility accompanying rickets may lead to convulsions, but more frequently to localized spasms. (5) Trauma and exposure to heat are occasional causes. (6) Organic disease of the brain, and meningeal hemorrhage during delivery, occasionally incite convulsions, but such seizures do not properly belong to this class.

Symptoms.—The paroxysm is generally preceded for a few moments by twitching of the muscles of the face, often confined to the lips, and often accompanied with grinding of the teeth and spasmodic swallowing. The convulsion begins with twitching of the fingers. The eyes are fixed in a stare, the body becomes rigid, respiration momentarily ceases, and the face becomes cyanotic. This is followed by a clonic spasm, most pronounced in the upper part of the body. The hands and arms jerk, the face is contorted, the head drawn back and usually to one side. The eyes are rotated upward or spasmodically drawn to one side. All the movements are rhythmical and synchronous, as if due to the discharge of an electric current. After a few seconds or several minutes, relaxation ensues, and the child usually falls asleep. Vomiting often occurs during or after the paroxysm, especially in cases of engorged stomach. Sometimes the convulsions are more pronounced on one side or they may be unilateral throughout. Fever is usually present. Death rarely occurs except in foudroyant cases of infection, or when the convulsion is the termination of a chronic diarrhea, cholera infantum, hydrocephalus, meningitis, or other previous disease. The attack is often repeated in a few hours, possibly in a few minutes. Frequent repetitions may beget a strong tendency to convulsions or a true epilepsy.

Diagnosis.—The important element in diagnosis is to determine whether the seizure denotes the onset of an infection. When this is the case, the fever is usually higher and there is sore throat, enlarged lymph-glands, an eruption, or other symptoms of a specific disease. Epilepsy can generally be excluded by the age of the patient, the absence of an aura or previous attacks, less typical course, and the recognition of an exciting cause.

Prognosis.—Death may follow repeated convulsions or the initial convulsion of a violent infection. Cerebral hemorrhage occasionally occurs during the seizure, and the child is left hemiplegic. In a great majority of cases, however, complete recovery occurs.

Treatment.—The paroxysm should be cut short by a few inhalations of chloroform, especially in a case of repeated convulsions, for in this manner serious results may be prevented. In the absence of the drug, the child should be put into a tepid bath, the head douched with cold water, and, if the temperature be high, the water should be cooled with ice or by the gradual addition of cold water. To conform with popular custom a tablespoonful of mustard may be tied in a rag and dropped into the water. Friction should be applied to the body during the bath. Any possible source of irritation should be removed. If the seizure be due to gastrointestinal irritation, an emetic should be given and followed with an enema. An ice-cap should be applied to the head. If the convulsion recur, morphin should be administered hypodermically in the dose of gr. 1-100 to 1-20 (0.0006—0.003), according to age, or chloral may be given by enema in dose of gr. v (0.30) or less. Following the attack, the child should be kept under the influence of the bromids for several days, and other precautions, particularly regulation of the diet, should be taken to prevent recurrence.

EPILEPSY.

FALLING SICKNESS, EPILEPTIC FITS.

Definition.—A paroxysmal disease manifesting periodical attacks of unconsciousness, with or without convulsions, which are usually preceded by an aura or warning. The principal types of the disease are: (*a*) *Grand mal* or *haut mal*, in which the unconsciousness is profound and the convulsions violent; (*b*) *petit mal*, exhibiting transitory unconsciousness without convulsions; (*c*) cortical, parietal, or Jacksonian epilepsy, consisting of localized spasm without loss of consciousness; (*d*) psychical epilepsy, or double consciousness, a state of somnambulism in which acts of violence may be committed, takes the place of the convulsion. Several other types are included by some writers, most of which belong rather to what are termed epileptiform convulsions than to the disease proper.

Etiology.—The disease usually begins before the fifteenth year, seldom before the tenth, and rarely after the twentieth. Epileptic seizures occurring in later life are generally due to cerebral syphilis, occasionally to other forms of intracranial disease. Sex is practically without influence.

Heredity of the disease cannot usually be traced, but a very large percentage of the cases occur in families of neurotic type exhibiting a strong tendency to such affections as neuralgia, hysteria, insanity, chorea, drunkenness, wantonness, drug habits, or syphilis. Repeated intermarriage intensifies the family predisposition.

Exciting Causes.—The most important of these are the acute infectious diseases, local diseases of the brain compressing the cortical layer; emotional disturbance, particularly fright, trauma, habitual convulsions; profound malnutrition, and reflex irritation. In the last group are usually enumerated many influences which much more frequently do not excite the disease, as dentition, intestinal worms, constipation, adherent prepuce, masturbation, foreign bodies in the nose or ear, and irritation of the eye. Osler records a case cured by removal of a retained testis. Epilepsy is sometimes associated with arteriosclerosis.

Symptoms.—1. **Grand Mal.**—This is the typical and most frequent form of the disease. In a majority of cases the seizure proper is preceded by a prodrome or premonition known as the aura. The warning may be given in many ways. Rarely it is like a puff of air, as the name signifies. More frequently there is a peripheral sensation of some kind, pain, numbness, tingling, or burning, starting in a finger, in the hand, over the region of the heart, or most commonly of all from the stomach or intestines (pneumogastric aura), and often apparently traveling toward the head. It is always the same sensation and in the same place in each case. Psychic auræ occur in some cases. There is a sensation of impending danger, a dreaminess, or a flash of light, an odor; a peculiar color, or a definite object is seen (visual aura). Odd sounds, a musical note, or voices may be heard (auditory aura). The aura may be followed by a sudden cry. In many cases, however, the outcry is absent, or it may occur without the aura. The duration of the aura is variable. In some cases it is but momentary, while in others the patient is given time to prepare for the attack, even time to walk to a place of safety and lie down. Cases have been described in which the fit could be prevented by quickly wrapping a cord around the finger in which it starts, but such cases are exceptional. In some instances peculiar movements take the place of the aura. The patient may turn around rapidly or run a short distance at great speed (procursive epilepsy).

The Paroxysm.—The patient suddenly becomes unconscious and falls forward. The head and face are thus often injured, and the old epileptic can often be recognized by his scars, bruises, and nodosities. The fit consists of three stages, the tonic spasm, the clonic spasm, and coma.

(*a*) *Tonic Spasm.*—This is usually momentary in duration. The body becomes rigid, the head is drawn back and to one side, there is conjugate deviation of the eyes, the face is pale and becomes cyanotic. The forearms, wrists, and fingers are strongly flexed and the legs extended. Respiration is arrested by the contraction of the thoracic muscles.

(*b*) *Clonic Spasm.*—The rigidity is immediately followed by a fine muscular tremor, which rapidly passes into a coarse jerking of all the muscles, increasing in intensity and rapidity until the limbs are violently thrown about. The face is contorted, the eyelids open and close, and the eyeballs rotate. The jaws participate in the spasm, and the tongue is often bitten. Froth appears at the mouth and it may be stained with blood. The urine and feces are sometimes discharged. The duration of this stage is usually from one to two minutes. As it passes, the convulsive movements become less pronounced, and the patient falls into a state of coma.

(*c*) *The Coma* is profound. All rigidity has passed away. The breathing is heavy and rapid, often stertorous, and the cyanosis of the face gives place to congestion. After a few moments, as a rule, the sleep becomes more natural, and the patient can be aroused, but if undisturbed he may sleep for several hours. When he awakes, the mind is clear and the recovery is complete, except for the muscular soreness and whatever of injuries may have been received. After the attack the reflexes are generally increased and the ankle-clonus may be obtained, but they may be diminished or absent. Slight albuminuria may be induced, and a large quantity of clear urine is generally voided. An elevation of temperature of a degree or less may be observed.

The periodicity of the epileptic seizures is variable. In the beginning the seizures may occur at intervals of several weeks or months; but as the disease becomes more firmly established, they recur with greater frequency until finally a day may not pass without its paroxysms. A condition known as the status epilepticus is ultimately developed in some cases, in which the fits follow each other so rapidly that consciousness is not regained in the intervals, a febrile state is produced, and the patient succumbs to exhaustion. The paroxysms occur at any time of day or night, in some cases at a definite hour. They are sometimes entirely nocturnal in the beginning, and the disease may be unrecognized for many months until the injury of the tongue, bruises about the head or neck, the dislocation of a shoulder or other joint, or the fracture of a limb leads to an investigation.

Postepileptic State.—In a majority of cases, as stated, the patient awakes fully recovered from the attack. Occasionally, however, there is a condition of semiconsciousness or trance in which acts may be performed which the patient does not recollect after the condition has passed. Rarely it assumes the form of mania with homicidal propensity. The mind becomes impaired in the confirmed epileptic; thought becomes sluggish and the speech is slow and drawling, often indistinct. Hemiplegia is rarely induced by the attack.

2. **Petit Mal.**—In petit mal or mild epilepsy there is momentary unconsciousness without a convulsion. There is at most a slight muscular tremor of the face or fingers. The patient may be seized at any moment. In some cases he suddenly becomes pale, his eyes are momentarily fixed, and he drops whatever may be in his hands. If writing, the seizure may be recorded by a scrawl of the pen. In a moment consciousness returns, and he resumes the conversation or other employment. He may not be conscious of the lapse and his associates may not observe it. There is no aura, as a rule, and rarely a cry. There may be a slight sense of vertigo or faintness. The patient seldom falls; the stage of coma and the sleep are absent, but after repeated recurrences the paroxysms often develop into grand mal. In some cases a peculiar act is performed, as rapid rubbing of the face, nose, or ear, or the patient may begin rapidly to disrobe. Acts of violence are sometimes committed in the unconscious state.

3. **Jacksonian epilepsy** (cortical, symptomatic, or partial epilepsy) manifests itself in twitchings of a single group of muscles, as those of the face, arm, or leg, without loss of consciousness. A prodromal numbness or tingling of part of the area may be felt before the attack, and the sensation may persist after it has passed. The spasm is both tonic and clonic, and may extend from the original area to the other muscles of the face or limb. Like those of petit mal the paroxysms are liable, in the course of months or years, to lapse into typical epileptic seizures. The affection generally arises from irritation confined to a single motor region of the cortex, by a tumor, localized meningitis, depressed bone the result of fracture, hemorrhage, abscess, or sclerosis. The attacks are sometimes observed in uremia and general paresis or after hemiplegia in children (posthemiplegic epilepsy). The attack is followed by local paresis, sometimes accompanied with loss of the sense of touch and the perception of heat.

Diagnosis.—The true epileptic fit can generally be differentiated from epileptiform convulsions due to other affections by the typical course of the manifestations in a fully developed case. When the seizure is preceded by a distinct aura, and consists of tonic spasm followed by clonic spasm lapsing into a deep coma, and accompanied by relaxation of the sphincters, the condition is clearly one of epilepsy. When many previous attacks have occurred, the diagnosis is usually further supported by the presence of scars, bruises, and nodes on the scalp that have been referred to. Nocturnal fits are almost always epileptic.

Uremic convulsions can be recognized through the condition of the urine. A persistent small trace of albumin, with low specific gravity and particularly the presence of casts, is the usual condition. Headache and vertigo are generally present in these cases.

Hysteria is rarely difficult of exclusion. The symptoms are usually overacted. There is no aura, the cry is prolonged and repeated. The patient never injures herself, but may scratch and bite those about her. Opisthotonos is often present, but the sphincters are not relaxed. The clonic spasms are less regular and more prolonged. The unconsciousness is well feigned, but coma is absent.

Fainting from any cause and the vertigo of Ménière's disease are distinguished by the absence of both aura and unconsciousness.

Prognosis.—Complete recovery is occasionally observed in young subjects and in women, seldom in men, but in a great majority of cases the disease is incurable. When death occurs during a fit it is generally due to some accidental injury, as falling into fire or water, or the obstruction of the larynx by food. Remissions of many months or years are sometimes followed by a renewal of the attack. Cases due to peripheral irritation are generally most benefited by treatment.

Treatment.—The first element in the treatment should be a careful investigation of the case, and the removal of any possible source or irritation. A diet should be prescribed which is proper for the age of the patient and the condition of his digestion. As a rule, meat should be eaten sparingly, and an abundance of water should be drunk to promote the secretions. The action of the bowels must be regulated.

The best remedy for the control of the paroxysms is potassium or sodium bromid; they may be combined. From two to four drams should be administered daily in the beginning, and if the attacks are not arrested, chloral should also be given. Bromism is generally induced within a week or two, and if it becomes excessive, the dose must be reduced; but bromism is more tolerable than epilepsy, and the treatment should be continued for a month or longer in most cases. After the seizures have been prevented or greatly modified, the dose may be gradually diminished, but a half-dram (2.0) of the bromid should be given daily for several years afterward. The success of the bromid treatment lies in the persistent use of large doses. The acne eruption can be greatly diminished by means of full doses of arsenic administered for a few days at intervals.

Many other remedies are employed independently or in conjunction with the bromids, as valerian, asafetida, cannabis indica, and zinc; but they are inferior to them. Nitroglycerin is beneficial more particularly in petit mal. It should be given in increasing doses and persistently.

Inhalation of amyl nitrite immediately upon recognition of the aura arrests the attack in some cases.

Excellent results have been obtained in the treatment of this disease in the epileptic colonies. Here a case that is of such severity that home employment is impossible is given the benefit of outdoor occupation, which is infinitely better than idleness. Surgical treatment is successful in some cases, particularly those of the Jacksonian type. Trephining has proved beneficial for a considerable time at least, even in cases in which the operation was technically a failure.

TETANY.

TETANILLA.

Definition.—An affection attended with paroxysmal or continued bilateral tonic spasm of the muscles of the extremities.

Etiology.—The frequency of the affection diminishes from infancy to the twenty-fifth year, after which it is rarely encountered. It is often associated with rickets, sometimes with fevers, especially typhoid, and occasionally with dilatation of the stomach, pregnancy, or lactation, and it may follow chronic diarrhea and other debilitating diseases. It has developed after removal of the thyroid gland. Epidemics of an acute type (rheumatic tetany) have been encountered in Europe. The disease is more frequent in the winter season. The exciting cause is probably an irritation of the cortical centers by toxins.

Symptoms.—An intermittent spasm usually develops, first in the hands, then in the feet. The thumbs and fingers are firmly flexed into the palms, the distal phalanges being extended in some cases; the wrists are bent upon the forearms, and the elbows are often flexed. The feet are extended (the flexor muscles contracted), and the toes are adducted. Trismus is often developed later, and the angles of the mouth are drawn down, but the face may entirely escape. The muscles of the eyelids are often involved, sometimes those of the globe, with the production of strabismus. The skin of the hands and feet is usually tense and may become edematous. Retraction of the head sometimes occurs late in the disease, and the thoracic muscles may become implicated, producing dyspnea and cyanosis. The attack is usually intermittent, but it may become constant for a period of two or three weeks. The entire body rarely becomes rigid. A spasm can be induced as long as the affection lasts by pressure upon the affected extremities, over the nerve-trunks or blood-vessels (Trousseau's symptom). The excitability of the motor nerves is greatly increased so that a light tap over the nerve-trunk throws the supplied muscles into contraction (Chvostek's symptom), and the electrical excitability is also increased (Erb). Fever develops in the more acute cases.

Diagnosis.—Few conditions are to be differentiated. The *carpopedal spasm* from severe gastrointestinal irritation or occurring in rachitic infants is more transient. *Tetanus* is characterized by an earlier development of trismus, the cause is different, and the bacillus can be demonstrated.

Prognosis is usually good, although the cases sometimes last for sev-

eral weeks, and recurrences are not infrequent. After thyroidectomy the disease is often fatal.

Treatment.—Any recognized irritation must be removed. The bowels should be freely moved and kept regular. The diet must be proper for the age of the infant and easily digestible for the adult. Hot baths often relieve the spasm for a time. Some writers prefer cold douches and an ice-bag to the spine. The reflex irritability should be reduced by the free administration of bromids and chloral. Urethane is also recommended. The thyroid extract has been curative in some cases, doubtless associated with atrophy or absence of the thyroid gland, as in the cases reported by Stewart. Massage and electricity have been employed with benefit in some cases, but they aggravate the spasm for the time.

MIGRAINE.

MEGRIM, HEMICRANIA, SICK HEADACHE.

Definition.—A severe paroxysmal headache usually confined to one side, and associated with disturbance of digestion and disordered vision.

Etiology.—The affection ordinarily develops before puberty and may subside in later life, in women after the menopause. It is often distinctly hereditary and several members of a family are often affected, particularly on the female side. It is often associated with other neurotic affections in the family or ancestry. A gouty or rheumatic history is also common. It is often associated with menstrual disorders or ovarian and uterine disease. Fatigue, excitement, anxiety, worry, and other debilitating influences are operative in some cases.

The exciting cause is not known. The attack often follows a disturbance of digestion, constipation, or the eating of some article of food that does not "agree" with the individual. Some persons invariably suffer from it when traveling. Eye-strain, due to astigmatism or uncorrected errors of refraction, is a frequent cause. Irritation of the nose, throat, or ear has been given as exciting it in some instances. Autointoxication is probably one of the most potent causes, but the nature of it is not known. An accumulation of uric acid or of one or more of the xanthin group in the blood have been urged as the cause by different writers. The output of these substances has been shown to be diminished by Haig, Rachford, and others, before and during the attack; but their accumulation in the blood has not been demonstrated. The exciting cause is probably not always the same.

Symptoms.—Prodromal languor, drowsiness, or visual disturbances occur in some cases, while in others the patient awakes with the headache or is attacked soon after rising and without warning. The pain is at first confined to one temple, to the forehead or the occiput, and continues more severe in that region; but it soon becomes general. Light and sound aggravate it. There are sometimes hemianopia or flashes of light, tinnitus, and vertigo, accompanied with nausea, and the vomiting of bile-stained mucus. When the stomach is at fault, this vomiting affords relief. The pupil of the affected side, sometimes both, may alternately dilate and contract. Numbness and tingling of the tongue and fingers may be complained of. The headache becomes violent and

throbbing, and complete prostration ensues. Various psychical disturbances of excitement or confusion are sometimes exhibited. The arterial tension is increased, particularly in such vessels as the temporal of the affected side; but the pulse is usually slow. The face is at first pale, but becomes flushed on the affected side. Constipation is usually present. The tongue is dry or pasty, but not always furred. Arteriosclerosis sometimes develops in the temporal artery of the affected side after a long series of attacks. The attack usually subsides in the course of a few hours or a day, the patient then falls asleep and awakes greatly relieved. The recovery may not be complete, however, in the worst cases for two or three days.

Treatment.—The attack is shortened and rendered less violent in most cases by the prompt administration of a saline cathartic. An emetic is sometimes equally beneficial. In other cases greater relief is afforded by such remedies as sodium salicylate (gr. xv; 1.0), citrated caffein (gr. v; 0.30), with phenacetin or acetanilid (gr. x; 0.65), or hydrobromic acid (gtt. xv), repeated every two to four hours. A cup of strong tea or black coffee is effective in some cases. Morphin should never be given, for the habit is almost invariably developed in this class of patients; even phenacetin and the myriad of proprietary mixtures of acetanilid often acquire a fascination that is injurious to the general health. An ice-cap should be applied to the head, and a mustard-leaf placed on the nape of the neck.

An effort should be made in all cases to determine the exciting cause, in order to treat the condition intelligently. As general measures, errors in diet or habits should be rectified. In many cases, abstinence from meat and a diet consisting largely of fruits are beneficial. Excitement and fatigue should be avoided, errors of refraction should be corrected. Any cause of throat irritation should be removed. Any abnormal condition of the uterus or ovaries should receive treatment, and if an anemic state of the blood is revealed, iron and arsenic should be prescribed.

NEURALGIA.

Definition.—A disorder of the sensory fibers of the peripheral or visceral nerves, the chief manifestation of which is pain. It is not always possible in practice to adhere to a close distinction between this true neuralgia and peripheral pain due to central irritation or neuritis affecting the nerve trunks.

Etiology.—The affection is rare in childhood, except as a result of caries of the teeth. Women are more commonly affected than men. A hereditary tendency to neurotic affections is generally to be traced. In many cases there is: (*a*) An underlying malnutrition of the nerves due to anemia, malnutrition, pressure, or endarteritis; (*b*) such affections as chronic nephritis, rheumatism, gout, or diabetes, or such poison as alcohol, lead, or arsenic; (*c*) pressure upon the nerve by a tumor or inflammation in its vicinity. (*d*) The condition may be induced by an acute infection, especially by influenza, sometimes by malaria. (*e*) In some cases no cause can be discovered beyond probable exposure to cold (idiopathic neuralgia). (*f*) Reflex irritation from a carious tooth, disease of the middle ear, the nose, sinuses or antrum, eye-strain, or

that of pathological conditions in the ovaries, uterus, or intestinal canal may excite it.

Symptoms.—Abruptly, or after premonitory tingling or sense of discomfort, an aching or more severe pain develops which is burning, boring, darting, or stabbing in character, sometimes constant, but usually paroxysmal. The skin of the affected region may be acutely sensitive, and tender points can be found along the course of the affected nerve at the places where it passes from a deeper to a more superficial level. The skin is usually abnormally cool or hot, as a result of trophic disturbances; it may be edematous, and atrophy and induration occur in protracted cases. Herpes often appears. Rarely the hair becomes white or falls out, when the nerves of the scalp are affected. An erythema due to vasomotor irritation sometimes appears. The pain often shifts from one nerve to another.

Any nerve of the body possessing sensory fibers may be involved. The more important of the resultant affections may be conveniently arranged under the following groups:

1. **Trifacial Neuralgia** (Ticdouloureux, prosopalgia).—This is a severe affection of either or all three branches of the fifth pair of nerves. (*a*) In the affection of the ophthalmic division the painful points are: (1) The supraorbital, just above the supraorbital foramen; (2) the palpebral, in the upper lid; (3) the nasal, on the bridge of the nose at the junction of the bone and cartilage; (4) the ocular, in the globe of the eye; and (5) the trochlear, at the inner side of the orbit.

(*b*) In the infraorbital branch, the points are in the infraorbital or malar region and in the upper lip.

(*c*) When the third division is affected, the painful points are, the temporal, inferior dental, sometimes the inferior labial, rarely the lingual in the side of the tongue.

Motion aggravates the pain in all of these forms. A spasmodic contraction of the muscles (spasmodic tic) often accompanies the pain and greatly increases the suffering.

2. **Neuralgia of the Neck and Trunk.**—(*a*) *Cervico-ocipital* and (*b*) *Cervicobrachial.*—These have been considered under the heading of Neuritis of the Cervical and Brachial Plexuses.

(*c*) *Intercostal Neuralgia.*—This is a severe form of the affection involving the intercostal nerves and intensified by the movements of respiration. Three tenderpoints are usually found—one in front, one in the axillary region, and one near the spinal column.

(*d*) *Phrenic neuralgia* is a rare form, manifested by pain along the insertion of the diaphragm, in the neck, chest, and shoulder.

(*e*) *Lumbar neuralgia* exhibits pain in the lumbar region, extending to the femoral region, or along the crest of the ilium to the groin and vulva or scrotum.

(*f*) *Coccygodynia.*—(See Neuritis of the Sacral Plexus.)

3. **Neuralgia of the Upper Extremities.**—This affects the branches of the four lower cervical nerves and brachial plexus, nerves which are much more frequently the seat of neuritis. The pain is in the arm and forearm, sometimes in the hand and fingers. The painful points are; *a*, the axillary, over the brachial plexus; *b*, the scapular; *c*, the shoulder, where the cutaneous branch of the circumflex emerges from the deltoid;

d, the median cephalic, at the bend of the elbow; *e*, the external humeral about three inches above the elbow; *f*, the superior ulnar, over the ulnar nerve, between the olecranon and the epitrochlea; *g*, the inferior ulnar, just anterior to the annular ligament at the wrist, and; *h*, the radial, on the lower external part of the forearm.

4. **Neuralgia of the Lower Extremities.**—The sciatic nerve is sometimes affected, giving a painful point midway between the trochanter and the tuberosity of the ischium. Neuritis is more common in this nerve, however. (See Sciatica.)

Neuralgia of the feet occurs in the forms of pododynia or tender heel, metatarsal and plantar neuralgia. In addition to the pain there may be burning, itching, and local sweating.

The visceral neuralgias are considered under the headings of Neuroses of the Heart, Stomach, Intestines, and other organs.

5. **Herpes zoster** (shingles, zona) is a neuralgia now generally regarded as a specific disease of the posterior-root ganglia, probably an acute hemorrhagic inflammation, and, therefore, not strictly a functional condition. Of all so-called neuralgias it appears to bear the closest relationship with malarial infection. After the neuralgic pain has persisted for three or four days, accompanied with general malaise, an eruption of small vesicles appears over the peripheral filaments of the affected nerve. Any of the peripheral nerves may be affected, the intercostals more frequently than others.

Diagnosis.—The differentiation from *neuritis* is not always an easy one. The latter affection, however, is generally distinguished by greater severity, longer duration, less tendency to migrate, and a greater liability to trophic and vasomotor disturbances. The nerve pain produced by cranial and spinal tumors or syphilis, caries of the vertebræ, and the crises of locomotor ataxia are generally more permanent, more restricted in location, and accompanied by other symptoms of the causal disease.

Prognosis.—With the exception of tic douloureux, sciatica, and cases due to organic disease which cannot be removed, the prognosis is good, but recurrences are by no means exceptional.

Treatment.—The treatment consists in relief of the attack, removal of the local or exciting cause, and the improvement of the general condition which acts as a predisposing factor. The pain is relieved by hot fomentations, poultices, stupes, sinapisms, or embrocations containing menthol. The ethyl-chlorid spray affords temporary relief, but cannot be used continuously. Liniments containing chloroform, camphor, aconite, or chloral may diminish the pain, but cannot be applied to the face. Galvanism and cauterization are often beneficial.

Many internal remedies are recommended. The most active are phenacetin or acetanilid in doses of gr. x (0.13); sodium salicylate, gr. xv (1.0); an active tincture of gelsemium in frequently repeated doses until slight drooping of the eyelids is observed; and aconite. This remedy, to be effective, must be carefully pushed until tingling of the lips is produced—often a dangerous limit. Nitroglycerin in full doses is sometimes of benefit, especially in trifacial and sciatic neuralgia. For visceral neuralgia such remedies as the compound spirit of sulphuric ether, aromatic spirit of ammonia, and chloroform are the most useful.

Morphin, chloral, alcohol, and other habit-begetting drugs should be avoided.

The removal of the exciting cause embraces the treatment of all the influences referred to under Etiology, including the removal of cicatrices, tumors, and improvement of conditions which cannot be removed.

Of even greater importance is the improvement of the general health by means of rest and tonics. Iron and, more particularly, arsenic should be given when anemia is present. Quinin in tonic doses, combined with iron or arsenic and strychnin, rapidly improves the condition in many cases. Codliver oil is beneficial in malnutrition. Outdoor exercise is essential, and many cases are greatly benefited by a trip to the mountains or removal to a dry, temperate climate.

HYSTERIA.

Definition.—A psychoneurosis, or functional disorder of the nervous system, in which the perverted ideas inhibit volition and master the functions of the body.

Etiology.—The disease is most frequent in women between puberty and the menopause, but it is occasionally met with in children after the fifth year; it occasionally persists into old age, and it is not infrequent in men. It is more prevalent in the Latin races, but occurs in all others. The severe forms of it are rare in our country.

Heredity is a strong predisposing factor. The disease occurs particularly in neuropathic families, and often alternates with epilepsy, insanity, alcoholism, and drug habits. It is sometimes closely related to degeneracy. *Consanguinity* intensifies the predisposition. Habits of life and education have much to do with bringing out the individual tendency. Pampering and petting, the yielding to whims, the gratifying of all desires, sympathizing in every childish sorrow, the cultivation of a selfish nature, and the too early training in the artificial life of society, all prepare the daughters of wealth for a leading rôle in hysteria. Fortunately, this tendency is being to a great extent counteracted by a greater devotion to outdoor games and instruction in calisthenics. But hysteria is by no means limited to the higher classes. It is often encountered among the poor and overworked saleswomen and working-girls, among whom it is often superinduced by ovarian or uterine diseases, sometimes by alcoholism or drug habits.

Exciting Causes.—Physical exhaustion from overwork or suffering, worry, grief, fright, fear, financial loss, religious excitement, disappointment in love, sexual excess, especially masturbation, shock or injury as in railway accidents or witnessing a disaster, and many other influences are capable of inducing the seizure in a susceptible person. Imitation is a strong factor in some cases, and in this sense the disease is often spoken of as contagious.

Symptoms.—The course of the disease may be divided into a prodromal, a convulsive, and a nonconvulsive stage, although many cases pursue a most atypical course.

1. *Prodromal Stage.*—This may be of but a few hours' duration, or it may last for several days. In it the patient is despondent or restless and emotionate. She laughs and cries without occasion, and often

complains of a choking sensation (globus hystericus) and an inability to swallow, or of pain and hyperesthesia, especially in the breast or ovarian region, sometimes of numbness or anesthesia, dizziness, dyspnea, or other abnormal sensation.

2. *Convulsive Stage.*—The second stage follows abruptly. The patient may fall into a more or less violent convulsion, usually with a prolonged cry, or she may walk rapidly about, gesticulating and screaming. The severity of the seizure places the case in either of two classes, known as hysteria minor and hysteria major.

(*a*) **Hysteria Minor.**—In this, the more common form, the fall is guarded. The patient sinks with dramatic grace to the floor, into a chair, or across the bed; she does not injure herself. She becomes apparently unconscious, and is usually seized with convulsive movements, the chief characteristic of which is irregularity. The arms are thrown about, they may be rigid or flaccid; the trunk and pelvis may be brought into motion. The screaming may be continued, especially if the voice has been cultivated, or the seizure may consist largely of violent respiration. Its duration is from a few minutes to several hours. When it subsides, consciousness returns, and the patient usually has a pretty definite recollection of all that has transpired, although she may not admit it. In other cases she sinks into a semiconscious stupor, from which she can be aroused with difficulty. After the attack, a large quantity of clear urine of low specific gravity is generally voided, and much flatus may be expelled.

(*b*) **Hysteria major,** or hystero-epilepsy, is not often seen in this country. It is not infrequent in France. The initial stage is much like that of the milder form, except that the action is more violent. The patient often suffers from gastric disturbance for a few days; the abdomen becomes distended with flatus, eructations are common, and micturition is frequent. Fantastic acts of all kinds may be performed, and the patient finally becomes intensely excited. Various symmetrical, acutely sensitive spots are complained of or found upon examination, over the dorsal vertebræ, at points on the abdomen, and over the ovaries. The convulsive seizure is described under four stages: (1) The epileptoid, in which the paroxysm resembles one of epilepsy, but is more prolonged; (2) a condition called by Charcot clownism; (3) a state in which the patient assumes peculiar attitudes suggestive of certain passions and may be cataleptic; and (4) a state in which, although the patient appears to have regained consciousness, she has hallucinations or is delirious. She sees visions, communicates with absent persons, and makes assertions that are false, with the utmost confidence in their truth. She may even become dangerous to her attendants through charges against their conduct, a belief which may cling to her after she has fully recovered.

3. The *nonconvulsive stage* is closely allied to malingering, although the patient does not voluntarily deceive. Any disease may be simulated, and the completeness of the simulation corresponds, as a rule, to the patient's knowledge of the affection, or the possibility of imitation. Paralyses are especially common, paraplegia more so than hemiplegia. In the latter affection, however, the face is not usually involved. The reflexes are generally increased or normal. Aphonia, retention of urine, and monoplegias are sometimes observed. Paralysis of the sphincters does not occur. Atrophy does not follow the paralysis, but con-

tractures of the various muscles are often present, which disappear under anesthesia. Tremors, spasms, and inco-ordination are frequently produced. Although the muscles retain their full power, the patient is often unable to walk or stand. The so-called hysterical joint, with swelling, pain, stiffness, and contracture of the associated muscles, is often observed.

Such sensory symptoms as formication, numbness, heat or cold, and other paresthesias, with loss of vision, hearing, or smell, occur in some cases. Vasomotor symptoms are also of occasional occurrence, as localized edema, congestion, or cyanosis.

The heart's action may be rapid, and palpitation is often complained of; syncope is a frequent manifestation. Attacks of pseudoangina are met with, and they are sometimes of almost daily occurrence.

Hysterical fever is one of the most interesting phenomena. In perhaps a majority of the cases the elevation of temperature is due to deception, and the thermometer often runs to the limit of its capacity, 110° F. or higher; 150° F. has been recorded. But cases have been repeatedly observed by clinicians whose acumen cannot be questioned, in which a moderate degree of fever has been more or less persistent for a long period. In some instances few or no other manifestations of hysteria were present, but the patient was of a neurotic type.

Diagnosis.—A correct diagnosis rests upon a close observation of the symptoms. The gradual onset, the explosive outburst, but above all the emotional condition of the patient and the overacting of the symptoms in almost every instance, are generally sufficient. In some instances, however, the exclusion of the affections simulated is extremely difficult. The administration of an anesthetic often removes doubt, particularly in the hysterical paralyses, contractures and joint affections.

Prognosis.—The disease is never fatal, but permanent cure is not always obtainable. The more violent seizures often cease under treatment, but the emotional nature remains, and the nonconvulsive manifestations are apt to crop out independently or in connection with any undue excitement or illness.

Treatment.—There is no other disease in which tact on the part of the physician is more truly the key to successful treatment. Although the patient is in a sense malingering, she is not guilty of intent to deceive. To her disordered mind the condition is one of real and serious illness. Many victims of hysteria are intellectually and morally far above suspicion of intentional deception, and they are often greatly chagrined by the realization of acts they have committed during the convulsive seizure or in a state of trance. The condition must be treated with a due appreciation of these facts. The first essential for the young physician is to gain the confidence of the patient and her family. An error of diagnosis, a failure to recognize the true condition, is fatal to him. He should impress the patient and attendants with the fact that the attack is not a serious one. With a statement of this fact, all the members of the family except one should be requested to retire from the room. The examination should be brief and only sufficiently thorough to satisfy the physician that there is no organic disease of consequence in addition to the hysteria. If the patient refuses to answer questions or feigns unconsciousness, let her alone. By the time the prescription is written, her curiosity will probably predominate, and she will open her

eyes in a wild stare. She can then be addressed, assured of speedy improvement, and will probably reply. When this end is reached, the physician should retire. A peremptory order must be given, however, after leaving the room and out of earshot of the patient, that she must be isolated and attended by but one member of the family, a friend who is capable of withholding sympathy, or, better, a trained nurse. The word hysteria should not be employed, but the family should distinctly understand that the nervous condition of the patient requires absolute rest and a total lack of sympathy. Her actions must be overlooked as though they were not seen. In many cases she should be left alone a greater part of the time, and in every way impressed with the idea that no anxiety is being aroused by her condition.

The more severe cases are not always so easily overcome, especially in patients who have gone through many previous attacks and have therefore become more adept. In these, strategy must sometimes be resorted to. The violent stage may often be promptly terminated by dashing or spilling, as if by accident, a glassful of icewater over the face and chest of the patient; the necessary change of garments proves beneficial. The application of a mustard-draught or of a cloth saturated with stronger ammonia to the abdomen, or the ice-pack, will often restore consciousness. The mere preparation of them is often sufficient in future attacks. The application of ammonia to the nostrils may break up a violent seizure, but the patient must not be tortured.

Sodium or potassium bromid should be prescribed in doses of gr. xv to xxx (1.0 to 2.0). Valerian or asafetida is often a useful addition. A cathartic should generally be ordered, and the bowels should be kept regular in action.

The general treatment consists of measures to restore the patient's confidence in herself and to increase her self-control. This can be accomplished by kind but firm advice toward the regulation of the habits of life and measures for the improvement of her health. Unfortunately, in many cases there are problems of domestic infelicity which are difficult of solution. In ordinary cases an outdoor life and exercise are beneficial. Strychnin and other tonics are often useful, but, as a rule, few drugs should be employed. In the chronic cases, patients who have been bed-fast for perhaps years, the Weir Mitchell treatment has proved most successful. This consists of a graduated milk diet, complete isolation, rest, massage, and electricity. It can seldom be properly carried out at home. The patient is given four ounces of skimmed milk every two hours for the first week; the milk may be peptonized, or Vichy or barley-water may be added to it. The quantity is then gradually increased and solid food slowly introduced. Massage is practiced daily, at first for twenty minutes, and gradually increased. The course requires about six weeks. Much can be accomplished in many cases by suggestion, but hypnotism has generally proved harmful.

NEURASTHENIA.

NERVOUS PROSTRATION.

Definition.—An exhaustion of nerve force resulting in an impairment and perversion of mental and physical functions.

Etiology.—The disease is most common during the age of greatest strain, particularly from the twenty-fifth to the fortieth year. Women are oftener attacked than men. Predisposition may be inherited or acquired. The patient usually belongs to a neurotic family; the parents have at least been of a nervous type or they may have been debilitated by alcoholism, tuberculosis, or other disease. All the influences mentioned under Habits of Life and Education as related to the Etiology of Hysteria prepare the way as well for neurasthenia. Too constant devotion to business, with disregard of exercise and other relaxation, is an important factor in many cases, especially among women. Physical strain is much less potent than mental strain, the so-called mind-fag. Excessive idleness, on the other hand, is the only explanation of the condition in some cases. The disease sometimes develops in those who have lived for a few years in a less exhilarating climate than they have been accustomed to, as in those who have removed from the interior to the seacoast or from the continent to Hawaii. It is, as a rule, a disease of city life, but cases occur in the country. Acute disease, as typhoid fever or influenza; organic disease, especially of the ovaries or uterus; reflex irritation from the eyes, especially exophoria; from the nose, throat, stomach, heart, kidneys, and other organs; injury, particularly of the spine, sexual excesses, late hours, financial loss, and bereavement are often influential.

Symptoms.—The patient experiences a constant feeling of fatigue. His energy is exhausted and his reserve force has been expended. Every application of mental or physical energy produces a sense of both mental and physical weariness. The appearance of the patient usually corresponds to his condition. He generally becomes emaciated and anemic, but in some cases the general appearance remains remarkably good. The disposition becomes irritable and despondent. In his selfishness the patient loses all regard for his family and others. Introspection is a prominent feature of most cases. The patient concentrates his mind upon his condition. He soon arrives at the conclusion that he has a serious organic disease or that he is on the verge of insanity, paresis, tuberculosis, or other serious illness, or he may brood over imaginary business reverses or losses, and in women the condition often develops into hysteria.

Headache, more commonly a sense of pressure within the head, insomnia, muscular tremors, gastric and intestinal indigestion, are frequent accompaniments of the affection. In the most severe cases the fears ("phobias") predominate. The patient fears not only disease and calamities, but he dreads to be alone (monophobia), or fears a crowd and shuns all assemblages (anthropophobia); he is afraid of lightning and thunder, afraid to pass a high building, to cross an open space, and a great many other groundless fears may be entertained. When the disease becomes extreme, the patient is confined to bed and believes himself unable to perform any exertion without severe palpitation and dyspnea. The mental condition becomes so distressing that he often contemplates and may attempt suicide. Various disorders of the special senses are sometimes observed. The eyes become irritable (asthenopia). Reading for a few moments is followed by burning or an aching and flashes of light.

Spinal symptoms are present in many cases. The patient has a weak back, and a slight exertion produces aching and a tired feeling in the spine and legs. Tender points are complained of, and may be found upon pressure along the spinal column. Intercostal and visceral neuralgias, especially in the ovarian region, are complained of, as in hysteria.

Vasomotor symptoms may be present. The face is flushed, localized sweating, throbbing of the arteries, abdominal pulsation, are seen, and the patient is often conscious of the heart-beats. Displacement of organs, as enteroptosis, nephroptosis, etc., are associated with some cases. The disease is often separated by various writers into many classes or types corresponding to the predominance of particular symptoms, as cerebral, cardiac, gastric, sexual, spinal, etc.

Diagnosis.—The neurasthenic condition often resembles in its clinical manifestations many other diseases which must be excluded by careful examination, but, as a rule, the mental state of the patient is so evident that the diagnosis rests between this disease, hysteria, hypochondriasis, and possibly general paresis.

In *hypochondriasis* there is the fixed idea or morbid sensation, particularly a pain under the ribs. The patient may be neurasthenic, but the delusion predominates.

In *hysteria* the symptoms are more violent, and there is the imitation of diseases rather than the fear of them, or the idea that they exist when they do not. A neurasthenic patient may, however, become hysterical at times, or the condition may merge into one of hysteria.

General paresis may begin with lassitude, melancholia, despondency, and tremors closely resembling neurasthenia, but the delusions soon develop and there is a feeling of exhilaration rather than of nervous exhaustion. It should be remembered also that the initial symptoms of locomotor ataxia and those of exophthalmic goiter occasionally resemble neurasthenia for a time, but the differentiation is not usually difficult after thorough examination.

Prognosis.—The results of early treatment are generally good, but in the cases of long standing recovery is often slow and recurrences are common.

Treatment.—The management of this disease is in many respects like that of hysteria, but the illness cannot be made light of. The patient cannot be made to believe that he is not seriously ill or that his disease is to any extent imaginary. His complaints must be listened to and kindly considered, but every effort should be made to divert his attention from his condition and to overcome the habit of introspection. In a severe case in which the patient is confined to bed or so weak as to be practically helpless, the Weir Mitchell treatment, referred to under the Treatment of Hysteria, is the most appropriate. In less severe cases it may be sufficient to recommend an outdoor life, with recreation and diversion. Fishing, hunting, boating, games, driving, and horseback riding in moderation are beneficial. The patient should retire early and rise late, obtaining from eight to ten hours of sleep. This should be secured without drugs if possible. Frequent baths should be administered. Hydrotherapy, including the cold pack and douches, especially the Scotch douch of alternating hot and cold water, beginning moderately, is applicable to nearly all cases, but it cannot usually be effectually

applied at home. Sea-bathing benefits some cases in which the weakness is not too great, especially nervous cases, but patients who are much depressed do better in the mountains than at the seashore. Electricity is often of benefit, directly and through the psychical impression, and for this reason the static machine is especially valuable.

A change of environment hastens the cure, but the patient should not be permitted to travel alone. A congenial companion is essential, for solitude affords too great an opportunity for introspection. Nothing is more important than the removal of the influences which have led to the breakdown, and in this regard every case is peculiar to itself. Idleness is more harmful than restful employment in some cases. Some are injured by even reading or writing, while to others it is a relief.

Tonics, particularly strychnin and iron, benefit many cases in the beginning, but medication should be suspended, as a rule, as soon as the improvement has become well established. The use of tea, coffee, tobacco, and stimulants should be abandoned. When sleep cannot be secured otherwise, the bromids, trional, or codein may be employed, but the more powerful opiates should be avoided and the patient must not be informed of the nature of the remedies he is taking.

OCCUPATION NEUROSES.

PROFESSIONAL SPASMS.

Definition.—An irregular involuntary spasm of certain groups of muscles as a result of their constant action in some habitual movement usually peculiar to the individual's occupation.

Etiology.—The principal types of the affection are the writer's cramp or scrivener's palsy, the telegrapher's cramp, pianist's cramp, and the milker's cramp. Among writers and telegraph operators the cramp is much more frequent in men; in some of the other forms, women are about equally subject to it. Improper methods of holding the pen or key are regarded as influential in many cases, but, aside from this, little is known of the cause. Many patients are in robust health, without neurotic or other taint or recognizable predisposition. Nothing is known of the pathology of the affection further than that there is probably a fatigue of the cortical motor centers governing the movements of the hands, which results in loss of co-ordination.

Symptoms.—In some cases the cramp comes on instantly when the pen is taken up; in others the individual can write for a few moments before it occurs. There is a violent spasm of the muscles of the fingers, sometimes accompanied with pain and tremor, and the pen is often thrown forcibly from the hand. Neuritis is sometimes developed, and there may be a constant feeling of fatigue in the muscles. The fingers often become hot and red or purple, and the skin may become glazed as a result of vasomotor influence. The strength of the muscles is not impaired, and the spasm is not excited by other use of them. The electrical excitability is often disturbed in cases of long standing.

Diagnosis.—The symptoms are generally so characteristic as to render the diagnosis perfectly apparent. Paralysis agitans, paresis, and other

central diseases, accompanied with tremor, can usually be excluded without difficulty.

Prognosis.—The prospect of recovery is poor, except through an early change of occupation.

Treatment.—An early change of occupation sometimes arrests the disease to such an extent that the individual can resume his former employment, providing he correct his method of writing or hold his pen in a different manner. The disease seldom occurs in those who use the free-hand method of writing, with the elbow as the fixed point. Some patients can prevent the cramp by using a very large cork penholder. Other devices are employed in the form of hand-rests, but they are often of no benefit. Some improvement may follow massage and cold douching of the arm, galvanism, and the administration of strychnin.

TRAUMATIC NEUROSES.

RAILWAY SPINE.

TRAUMATIC HYSTERIA.

Definition.—A condition of hysteria or neurasthenia resulting from the shock sustained in railroad accidents, fires, or explosions, or from witnessing the injury of others.

Etiology.—As stated in the definition, the injury may be physical or mental, but the effect is the same. The most pronounced cases are often those in which no lesion can be discovered or in which it is positively known that no physical injury has been received. These cases are well described by the term "traumatic hysteria," since the condition is purely a psychosis or psychoneurosis. A strong element in the etiology of many cases is found in the prospect of winning damages from a corporation. The pathology of the condition is generally expressed in such terms as spinal irritation or spinal anemia, but pachymeningitis and degeneration of the pyramidal tracts have been discovered after death in a few cases.

Symptoms.—The clinical features are those of hysteria or of neurasthenia. They may develop immediately, after several days, or as long as two or three weeks. Pain and tenderness over the back of the head and spine are complained of, and one or more tender spots can usually be found upon pressure. Numbness, formication, or sensations of heat and cold are often felt in the limbs. Paralyses sometimes develop in the form of either hemiplegia, paraplegia, or monoplegia. The sight, hearing, or other sense may be impaired.

Diagnosis.—An exact diagnosis is important from a medico-legal standpoint, but it is often extremely difficult. The differentiation is to be made especially from organic disease of the brain or cord and from malingering. It is often impossible in practice to make an early differential diagnosis between the purely functional cases and those in which injury has actually been received, and the malingerer is not always readily detected. A great deal is to be inferred from the persistence of the symptoms. When due to organic disease they are more permanent and less likely to be overlooked when the attention is diverted from them than when purely functional, and improvement is less likely to occur

after the fright and shock have had time to wear away. The malingerer usually exaggerates the symptoms to such an extent that he can be detected in it, as when a sharp tendon reflex is produced by a blow over the tibia, and the tender spots are forgotten during conversation.

Prognosis.—Complete recovery is the rule, but it may not occur for several months. It is often very rapidly completed after the termination of litigation. In cases of real injury, however, it may be permanent.

Treatment.—The case is to be treated as one of neurasthenia or hysteria.

FUNCTIONAL PARALYSES.

PERIODICAL PARALYSIS.

This is a form of general paralysis occurring in families, and transmitted through the maternal side. In some cases only the arms and legs are affected; in others, all the muscles from the shoulders down; and in a few cases the muscles of the tongue, pharynx, and neck have been involved. The attack comes on suddenly or after prodromal malaise, often during sleep and when the individual is in perfect health. When it begins gradually, it is generally complete within twenty-four hours. There are seldom sensory disturbances; the cranial nerves and special senses usually remain unaffected. The reflexes are diminished or abolished, and the electrical excitability of both the nerves and muscles is greatly reduced or lost. The temperature is normal or subnormal, and the pulse is usually slow. After lasting from two or three hours to several days, the paralysis disappears as suddenly as it developed. After a variable period of from a few days to several months, the attack recurs The recurrences cease, however, after the fiftieth year.

ASTASIA.—ABASIA.

These names have been given to two peculiar symptoms which are sometimes observed as independent affections in neurotic subjects; they are not infrequently combined.

In astasia the patient is unable to stand, while in abasia he is unable to walk. There may be rigidity of the legs, with tremor, or ataxia; or there may be complete limpness, so that the legs cannot support the weight of the body, although the strength of the muscles remains intact when the patient is in a recumbent position. The condition is usually associated with hysteria, epilepsy, chorea, or intention psychosis. Recovery generally occurs after a variable length of time, but recurrences are apt to follow. The treatment consists of rest, electricity, and other methods employed in hysteria.

VASOMOTOR AND TROPHIC DISORDERS.

RAYNAUD'S DISEASE.

Definition.—A symmetrical disorder of the circulation due to vasomotor influence beginning as a local anemia or syncope of the extremities and passing into asphyxia, followed by gangrene.

Etiology.—The disease is more frequent in women and children of neurotic type, but not infrequently affects men. Hysteria, neurasthenia, epilepsy, and other nervous affections are often present in the individual or family. The attack often follows exposure to intense cold.

Morbid Anatomy.—Raynaud's theory attributed the cause to a spasm of the vasomotor constrictors arising in the centers of the spinal gray matter, but these lesions have not been observed. Peripheral neuritis and endarteritis obliterans have been found in the affected extremities. The gangrene is usually superficial, but it may involve the entire member to such an extent as to produce spontaneous amputation.

Symptoms.—*Stage of Local Syncope.*—Following a nervous paroxysm, emotional disturbance or exposure to cold, less frequently after a gastric derangement, one or more fingers or toes, occasionally all the fingers and toes, or the hands with the fingers and the feet with the toes, become white, cold, and numb (dead fingers, dead toes). Rarely the tip of the nose and the lobes of the ears are involved. This condition lasts only a few hours, as a rule, but may continue indefinitely. It is then followed by the

Stage of Asphyxia or Engorgement.—The color is restored as it is after a part has been frozen. The vessels become engorged and the skin is livid, intensely red or purple, often mottled. There is slight swelling, with itching and burning pain. This condition occasionally develops primarily. The pain may be excruciating, but in some cases a state of anesthesia is developed. After a time the affection may subside, but similar attacks then occur, as a rule, at intervals, for several years, especially after exposure to cold. The general health may not be affected. In some cases, however, a chill occurs, and a condition of hemoglobinemia, with hemoglobinuria, is produced. The central artery of the retina is often contracted and the vision is impaired. Cerebral symptoms sometimes develop, varying from mental torpor with transient loss of consciousness or mania to aphasia and a temporary hemiplegia.

Stage of Gangrene.—A dry gangrene frequently follows the stage of engorgement, limited, as a rule, to the tips or pads of the fingers and toes, often to a single finger or toe of each side. The affected part becomes cold and dead, turns black and mummifies. A line of demarcation soon forms, and the flesh sloughs away or the entire phalanx may be amputated by a dry gangrene. This is followed by slow cicatrization, often resulting in deformity and ankylosis.

Diagnosis.—The disease is distinguished from the symmetrical gangrene of leprosy, diabetes, and other affections by the history of the case and the absence of the symptoms typifying these affections.

Prognosis.—The disease is rarely fatal except in feeble children. Many recurrences often take place, but the patient usually succumbs to another disease.

Treatment.—Prophylaxis is important after the first attack. The patient should avoid exposure to cold and other influences liable to excite an attack. Nitroglycerin has been found beneficial in some cases, but of no benefit in others. After the condition has developed the treatment is that of chilblains, elevation of the limb, and the application of dry dressings. When the pain is severe, morphin should be administered. Massage and electricity have been found of great benefit in some

cases. The treatment of the gangrene is surgical. Some writers advise early amputation, while others oppose it. It is a question which should be determined from the extent of the gangrene, the probability of sepsis, and the condition of the patient.

ERYTHROMELALGIA.

RED NEURALGIA.

Definition.—A rare chronic vasomotor disease manifested by painful localized redness and swelling of the skin, usually affecting the heels or balls of the feet, sometimes the entire foot and rarely the hands.

Etiology.—The disease has generally been observed in young adult males of nervous type or following rheumatism or other febrile disease. It is generally aggravated by warm weather, but may prove to be worse in winter. The nature of the affection is not known, but it is looked upon as a vasomotor disturbance or possibly due to neuritis. Arteriosclerosis has been found, and it has been suggested that the disease may depend upon irritation of the cells of the ventral horns of the spinal cord.

Symptoms.—The feet first become extremely painful, then, especially after walking, they become hyperemic and swollen. The blood-vessels are engorged and stand out prominently. Constitutional symptoms are sometimes present, as headache, vertigo, syncope; and the disease has been associated with Raynaud's disease.

Treatment.—The application of ice-water affords temporary relief. Massage and electricity have proved of benefit in some cases. The constitutional treatment with tonics is important. Excision of the nerves supplying the part has been followed by relief.

ANGIONEUROTIC EDEMA.

Definition.—A neurosis characterized by recurrent acute edema of localized areas of the skin or mucous membranes.

Etiology.—Young adult males are most commonly affected. The disease is often hereditary, passing through several generations, and it is generally encountered in persons of neurotic temperament. Its nature is not known, but it is regarded as due to nervous action upon the blood-vessels or lymph-channels through which local accumulation of lymph is produced. The disease has been associated with Raynaud's disease and erythromelalgia.

Symptoms.—The affection is generally strictly local, although gastric disorders and cardialgia are sometimes present. Hemoglobinuria has been observed. The swelling begins, as a rule, in the face, affecting the eyelids, the forehead, or cheeks, sometimes the backs of the hands or feet, the tongue, throat, or genitalia. The affected part is generally red and warmer than the surrounding skin. The swelling usually lasts for one or two days and subsides, but recurrences are the rule; sometimes they are daily, sometimes at longer intervals, and different regions are often affected in succession.

Treatment.—This is confined to hygienic measures, and the administration of tonics to improve the general physical and mental condition.

FACIAL HEMIATROPHY.

Definition.—A slow, progressive atrophy of the bones and soft tissues of one side of the face.

Etiology.—The disease is rare. It has usually been seen in girls about the age of puberty; in a few instances it has developed in adults. The cause is generally a disturbance of the trifacial nerve, as neuritis, trauma, or compression by a tumor. Some writers attribute it to a lesion of the sympathetic. It has been associated with epilepsy, migraine, or neuralgia.

Symptoms.—The disease generally begins on the left side as a wasting of the subcutaneous tissues in a small area, less frequently as a general atrophy. From this spot it spreads to adjacent structures, especially the upper maxilla, but affects the muscles least. White spots and areas of brownish pigmentation appear along the course of the larger nerves. The hair is lost and the teeth fall out on the affected side, which is sharply defined at the median line, except in a few cases in which the disease has been bilateral. The tongue and soft palate are usually involved and the eyeball, although not atrophied, sinks into the orbit on account of the loss of fat. In a few instances atrophic spots have appeared on the arm and back of the same side. Paresthesia and twitching of the muscles have been observed.

Diagnosis.—Other forms of unilateral atrophy are readily distinguished by the normal color of the skin, the greater involvement of the muscles, and exemption of the bones.

The disease is incurable, but it may remain stationary for many years and the health is not impaired. There is no treatment.

SCLERODERMA.

Definition.—A diffuse or localized induration of the skin, probably a trophoneurosis.

Etiology.—The disease is more frequent in young or middle-aged women, but may affect men. Sclerema neonatorum is not the same affection. The cause is not known. The disease is a sclerosis of the connective tissue, probably following nutritive changes produced by alteration of the blood-supply to the skin. Atrophy of the thyroid has been associated in some cases.

Symptoms.—The diffuse form is less frequent than the localized. The induration begins on the extremities or face, sometimes on the chest or back, and gradually extends. There is at first a thickening of the skin, which interferes with the natural movement, especially of the face. The expression is lost and mastication is impeded. When the fingers are affected, they cannot be moved, and forced flexion may tear the skin. The skin becomes adherent to the underlying tissues. The surface becomes white, with areas of pigmentation, or there may be diffused discoloration. It may be normal or drier. The integument of the entire body finally becomes involved, after which the process remains

cases. The treatment of the gangrene is surgical. Some writers advise early amputation, while others oppose it. It is a question which should be determined from the extent of the gangrene, the probability of sepsis, and the condition of the patient.

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clinical manifestations is peculiar to the disease. A well-marked cachexia develops sooner or later. The reflexes and special senses, except vision, remain normal, and sensation is not affected.

Diagnosis.—The appearance of a well-marked case is typical, especially the great width of the fingers and toes, and enlargement of the jaws.

Osteitis deformans is distinguished by the lengthening of the bones without great increase of width, and by the less marked involvement of the head. *Arthritis deformans* is characterized by enlargement of the ends of the bones, dryness of the joints, and pain upon motion. In pulmonary osteoarthropathy the enlargement is confined to the fingers and toes, and there is generally a history of pulmonary disorder.

Prognosis.—The disease is progressive, but subject to long intermissions. The patient may live many years, and death is generally due to another disease.

Treatment.—Extracts of the thyroid, pituitary body, and of the lung have been employed, but with doubtful success. The most that can be hoped is to obtain an arrest of progress.

RARE VASOMOTOR AFFECTIONS.

Micromegaly is attributed to disease of that part of the nervous system which presides over nutrition. In it some parts of the body become prematurely enlarged, and others remain abnormally small. Nothing is definitely known of its cause.

Ainhum is a condition in which the toes, usually the little toe of one or both feet, become enlarged and spontaneously amputated. It is met with chiefly in tropical or subtropical countries, but has been seen among the negroes of the Southern States. A groove forms under the base of the toe, this becomes swollen, red or purple. The groove becomes a line of demarcation, and the toe is separated by a dry gangrene. Sometimes the entire foot becomes involved or other toes may be affected. An intense burning pain extends up the leg in some cases; in others there is entire freedom from suffering.

Hypertrophic Pulmonary Osteoarthropathy.—In this affection there is an enlargement of the hands and feet, including the nails, and generally of the distal half or three-fourths of the bones of the forearms and legs. It usually affects adult males and is almost always associated with a chronic pulmonary affection, tuberculosis, empyema, or chronic bronchitis, but may occur without recognizable cause.

Osteitis Deformans (Paget's Disease).—The bones become enlarged much as in acromegaly, but they are at the same time softened. As a result of this, the long bones and the spine become curved and are often painful. The bones of the head are little, if at all, affected, and those of the face escape. The cause is unknown. The pathological condition is a combination of rarefying and hyperplastic osteitis. Some of the Haversian canals are enlarged, others obliterated. New lamellæ are formed. There is often an apparent relation between the disease and the development of sarcoma and carcinoma in the patient. The health generally remains good and the mind is not affected. No treatment has been found of any benefit.

Leontiasis ossea is a rare disease, in which the bones of the head and face, and often the soft tissues of the head and neck, take on an abnormal growth. The clavicles and hands may be affected. It begins in early life and may last indefinitely. The cause is not known. It has been attributed to injury in at least one case. Osteophytic growths may form upon either table of the skull. When upon the inner table, they may give rise to symptoms of tumor. No results have been obtained from treatment.

Hydrops articulorum intermittens is a rare affection of the large joints, characterized by sudden, painless swelling, which persists for several days, then subsides. It usually recurs at intervals of a few weeks for several years. It is thought to be due to a nervous influence which interferes with the circulation of the blood and lymph in the vessels of the joint. It occurs for the most part in neurotic subjects, and has been associated in a few instances with angina pectoris or exophthalmic goiter.

PART III.

CLINICAL METHODS OF EXAMINATION.

Clinical Methods of Examination.

The following pages are devoted to the methods of chemical and microscopical examination applicable to clinical study. As far as possible only such methods are given as can be employed in general practice, without the use of apparatus found only in a fully equipped laboratory. The student is assumed to possess the elementary knowledge of the use of the microscope and chemical apparatus which is now a part of the laboratory instruction in all medical colleges.

EXAMINATION OF THE BLOOD.

Obtaining the Specimen.—Just as in a surgical operation, everything to be made use of should be at hand before this little operation is undertaken. The hemoglobinometer, pipettes, hemacytometer, enough slides and cover-glasses, and the diluting fluid should be in readiness. The puncture is generally made on the posterior margin of the lobe of the ear with a small knife or a Hagadorn needle. For bacteriological examination, however, a larger quantity of blood must be secured from a vein of the arm, under strict antisepsis, with a hypodermic syringe. The skin to be punctured is first cleansed with a mixture of alcohol and ether, and the instruments must be sterilized. The puncture of the ear should be large enough to produce a spontaneous flow of several drops. The first two drops should be discarded.

The color of the blood should be noted, although it is perceptibly deficient only in extreme anemia. The time required for spontaneous coagulation is also important in some cases. This is normally from three to four minutes, while in hemophilia it may be as long as ten or fifteen minutes.

One specimen of blood should always be examined fresh without dilution or staining. A small fraction of a drop of the blood is placed on the center of a cover-glass and spread into a thin film upon the slide. If the specimen is not to be examined immediately, a circle of the proper size should be painted with vaselin upon the slide before the cover is inverted upon it. In this manner the specimen can be kept for several hours. From the examination of the undiluted specimen one obtains an idea of the size, form, and nucleation of the corpuscles (Fig. 25), the depth of color in the red, their rouleaux-formation, any apparent excess of leucocytes, the presence of fat, pigment, or other granules, bacteria or parasites, and roughly of the richness in fibrin. The specimen should be examined both with the ⅛-inch objective and with the 1-12-inch oil-immersion lens.

THE BLOOD-COUNT.

Enumeration of Red Corpuscles.—The blood must be diluted to precisely 100 or 200 volumes with Gowers' solution, the formula of which is:

Sodium sulphate	7.5
Acetic acid	20.0
Distilled water	125.0

The diluting fluid must be kept free from sediment and solid particles by frequent filtration. The dilution is made by means of the Thoma-Zeiss hemacytometer, Fig. 26.

In counting the red corpuscles, the pipette shown in Fig. 27, and readily recognized by the figures 101 at the base, is employed. The tube S is immersed in the drop of blood flowing from the ear, and by gentle suction through the mouthpiece *m* just enough blood is drawn in to fill it exactly to one of the marks, preferably to .05 or 1. The tube must

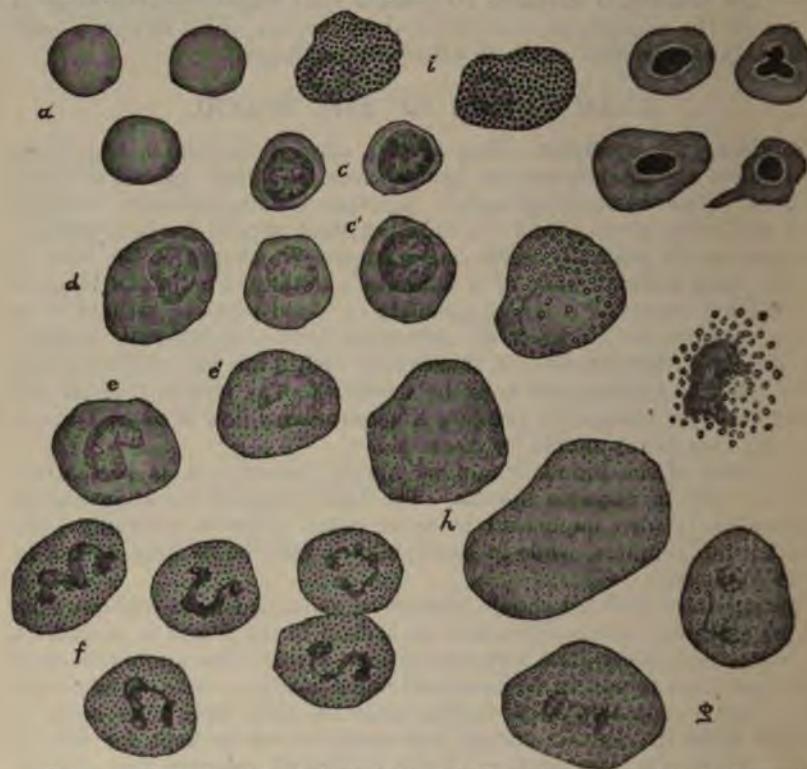

FIG. 25.—Normal and pathological red and white blood-corpuscles. *a*, Normal red corpuscles; *b*, nucleated red corpuscles; *c*, lymphocytes; *d*, large mononuclear; d^1, transitional forms between *c* and *d*; *e*, transitional forms; e^1, transitional neutrophiles; *f*, polymorphous forms; *g*, eosinophiles; *h*, myelocytes.

be perfectly clean and dry, and the manipulation rapid in order to prevent coagulation of the blood within the tube. If too much blood is drawn in, the tube must be cleansed and dried before the procedure is repeated. After the desired quantity has been secured, the tip is quickly cleansed and immersed in the diluting fluid. Suction is again applied and the fluid drawn exactly to the mark 101. The blood is thus diluted in a definite ratio. If the tube has been filled to the mark 1, the ratio

is 1 to 100, and if to only the mark .05, it is 1 to 200. The latter ratio is generally the better, hence it is better to draw the blood only to the .05. The rubber tube is now removed, and the tube is closed by placing the thumb and finger over its ends, and shaken in order to mix the blood and diluting fluid in the chamber E. After this the tube may be

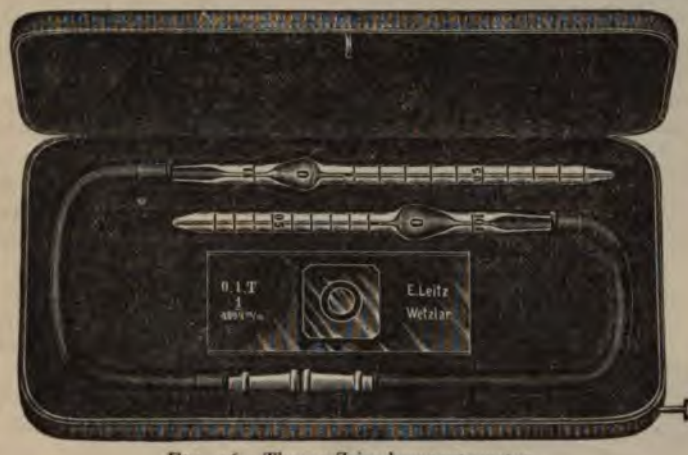

FIG. 26.—Thoma-Zeiss hemacytometer.

laid aside or transported, providing the ends be closed by placing a rubber band around it longitudinally.

Another valuable diluting fluid is that of Hayem:

Mercuric chlorid	0.5
Sodium sulphate	5.0
Sodium chlorid	2.0
Distilled water	200.0

The dilution may be made also with either a 3 per cent solution of sodium chlorid, or a 15 to 20 per cent solution of magnesium sulphate.

In making the count, the slide and ruled cover-glass belonging to the hemacytometer must be used, and the utmost care is necessary to have all parts perfectly clean, and to secure exact coaptation of the surfaces.

FIG. 27.—Thoma-Zeiss pipette for diluting red blood-corpuscles.

A low objective with a high ocular generally gives the best results, the adjustment being so arranged that the ruled squares will occupy a little less than the entire field. The illumination should be moderate. The corpuscles should then be counted in a definite number of squares, or until about 1,200 have been counted. A regular order must be followed

to avoid error, and it is well to check off the squares as they are counted on an extemporaneous diagram. Cells lying on the lines should be counted with the square below or to the right, to avoid counting them twice. After the count has been completed, the number of corpuscles in the cubic millimeter is computed by multiplying the average number in each square by 4,000, and this by the dilution. The percentage may then be calculated on the basis of 5,000,000 to the cubic millimeter in normal blood. In women, however, the normal is usually a little lower, from 4,000,000 to 4,500,000.

Enumeration of Leucocytes.—In counting the leucocytes, a lower dilution is necessary, as a rule, 1 to 10 or less. When the leucocytes are greatly in excess, however, as in leukemia, the count is more readily made in a dilution of 1 to 20 or even 1 to 50. The diluting fluid is a 0.33 to 0.50 per cent solution of acetic acid, which dissolves the red corpuscles. The addition of a few drops of a gentian-violet solution facilitates the count by staining the leucocytes. The tube must be held in a horizontal position or closed with the rubber band, for its large caliber permits the escape of the fluid. The manipulation is the same in all respects as in the dilution of the red corpuscles. The blood is drawn into the tube exactly to one of the divisions, quickly freed from superficial blood, and the diluting fluid is drawn in to the mark 11. The computation is made by multiplying the average number of leucocytes in each square by 4,000, and the product by the dilution. In extreme leucocytosis, the erythrocyte tube should be employed.

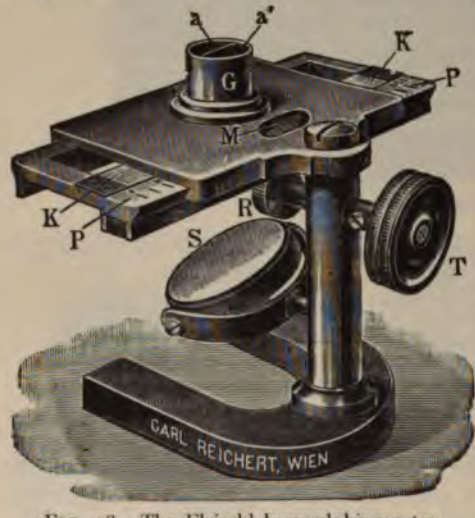

FIG. 28.—The Fleischl hemoglobinometer.

Estimation of Hemoglobin.—The most accurate method is that by means of the Fleischl hemoglobinometer (Fig. 28). The end of the capillary pipette accompanying the instrument is touched to the drop of blood. As soon as the tube is filled, its contents are washed into the compartment *a* with water from a medicine-dropper or pipette. This compartment is then completely filled with water and mixed by stirring. The upper surface of the mixture of blood and water should be slightly convex, care being taken that there is no overflow into the other compartment. Compartment a^1 is filled with pure water. An artificial light of moderate strength, as that of a candle, is reflected through the compartments from the mirror S. The color of the blood-mixture is compared with that of the red glass slide KK, which is thinner at one end than the other, and therefore lighter in color. It is moved by the thumb-screw T. The percentage is read from the scale PP at the line M. The estimate is usually a trifle low. The accur observation is not

entirely complete, and the result obtained by two observers is seldom exactly the same, since the eye is not capable of discriminating the delicate shades of color. The ratio of hemoglobin to the individual corpuscles may be determined by dividing the percentage of hemoglobin by the percentage of the red corpuscles to the normal as determined by the blood-count.

Staining the Blood-Specimen.—Staining is resorted to chiefly for the purpose of facilitating the differential count of the corpuscles, bringing out nuclei, granules, and other peculiarities. Permanent specimens may be mounted in this manner.

A small drop of blood is spread between two covers in the usual manner; the covers separated and the smear allowed to dry. It is then fixed by passing it rapidly through the Bunsen flame ten to twenty times, or by immersion in (*a*) absolute alcohol for 15 to 30 minutes, (*b*) in equal parts of absolute alcohol and ether for two hours, (*c*) in a 5 per cent alcoholic solution of mercuric chlorid for three or more hours, followed by thorough washing and drying, or (*d*) a 1 per cent alcoholic solution of formalin for one or two minutes. The alcoholic fixing fluids answer well for the red corpuscles and malarial parasites, but heat is better when the Ehrlich triple stain is to be used.

Methods.—The *eosin and methylene-blue* solution is one of the most useful stains. Its action varies, however, chiefly with the quality of the methylene blue, and, like all staining fluids, it should be tried and modified until good results are obtained with normal blood. The formula is:

Saturated alcoholic solution methylene blue	40.0
5 per cent solution eosin in 70 per cent alcohol	20.0
Distilled water	40.0

The solution should be filtered just before it is used.

The cover-glass smear should be immersed in the solution at a temperature of 37° C. for 24 hours. The erythrocytes are stained red, the nuclei of either red corpuscles or leucocytes blue, eosinophile granules a bright red, neutrophile granules pink, basophile granules blue. The malarial parasites take a pale blue stain. Better results are sometimes secured by adding to 50 c.c. of this solution 10 to 15 drops of 1 per cent acetic acid.

Ehrlich (or Biondi) Triple stain—The formula for this is:

Orange G, clear saturated solution	6.0
Acid fuchsin, clear saturated solution	4.0
Methyl green, clear saturated solution, added drop by drop, constantly shaking the mixture	6.6
Glycerin	5.0
Absolute alcohol	10.0
Distilled water	15.0

The orange G, acid fuchsin, and methyl-green solutions must be made with reliable ingredients, preferably with Grubler's, and the solutions should stand for several days before mixing. It is better to prepare at first only a small quantity of the triple solution in order to test its action, for better results are often obtained by slightly modifying the formula. After a satisfactory result has been obtained the solution

can be kept indefinitely. The blood-smear must be thoroughly fixed, preferably by heat, as otherwise the stain often acts too deeply.

The solution may be dropped upon the cover-glass held in a forceps and permitted to act for 5 or 6 minutes, washed, dried, and mounted in balsam.

Results.—Erythrocytes, orange, varying in intensity with their richness in hemoglobin; polychromatophile-red corpuscles, brownish or black; nuclei of red or white corpuscles, a variable shade of blue or green; neutrophile granules, violet or reddish blue; eosinophile granules, brilliant red; basophilic granules, not brought out. The malarial parasites become distinct, although unstained.

Basophile Stains.—A saturated aqueous solution of methylene blue may be applied to the specimen for 5 to 10 minutes. The basophile granules and nuclei are stained blue. Another useful stain consists of:

Toluidin	1.0
Phenol	5.0
Distilled water	95.0

After fixing with a mercuric-chlorid solution, the specimen is submitted to the stain for five minutes, washed in 1 per cent hydrochloric acid in 70 per cent alcohol. The nuclei and basophiles are stained blue.

Staining the Malaria Plasmodium.—Spread the film in the usual manner and fix by gentle heat or in absolute alcohol, then stain in a 1 per cent aqueous solution of eosin for five minutes, counter-stain for five minutes in a saturated aqueous solution of methylene blue or in the Ehrlich triple stain for a half-hour; wash, dry, and mount. The specimens stained in the Ehrlich triple stain may be examined dry. The malaria parasite and the nuclei of the leucocytes are stained blue; the erythrocytes, red, except after the Ehrlich stain, when they become distinct by contrast.

Whitney's Method.—Spread the film as before and thoroughly dry with gentle heat. Then immerse in the following solution:

Potassium bichromate	2.0
Sodium sulphate	1.0
Distilled water	100.0

Add, while warm, enough mercuric chlorid to saturate, and add, just before using, 5 per cent of nitric acid.

After allowing the specimen to remain in this solution for 20 minutes, wash in water, dry with cigarette-paper, and stain for three minutes with Ehrlich's triple stain. Wash, dry, and mount.

Plehn's Method.—Fix the smear in absolute alcohol for three to five minutes, then stain five or six minutes in the following solution:

Concentrated aqueous solution methylene blue	60.0
One-half per cent solution of eosin in 75 per cent alcohol	20.0
Distilled water	40.0
Twenty per cent NaOH	gtt. 12.0

After staining, wash in water and mount in balsam.

This method is one of the most rapid and satisfactory in use.

Widal's Serum Test.—This test is based on Pfeiffer's agglutination reaction. The test is applied thus: A drop of fresh or dried blood from the ear of the patient is diluted with 10, 20, and 30 or more times the quantity of distilled water. A drop of fresh, virulent bouillon culture of typhoid bacilli is then added to each, and the specimens are immediately examined under the microscope in the hanging drop. The agglutination may occur immediately or after ten or fifteen minutes. The bacilli appear grouped together in irregular tufts of variable size (Fig. 29) and become motionless. The time at which the reaction becomes distinct in the different dilutions should be recorded. In the dilution of 1:10 an immediate agglutination generally occurs. It may occur in a dilution of 1:50, 1:80, or even higher. The absence of this reaction throughout a disease may be regarded as positive evidence that typhoid fever is not present, since it has been found in 97.9 per cent of 4,879 cases collected by Brill. An agglutination of the typhoid bacillus has been obtained from the blood of patients suffering with malaria, typhus, miliary tuberculosis, cerebrospinal meningitis, and other acute infections, but rarely in a higher dilution than 1:5. A reaction obtained from a

FIG. 29.—Widal test for typhoid fever. *a*, Negative; *b*, partial reaction; *c*, positive reaction. (Nichols.)

dilution of 1:30 is, therefore, a positive demonstration of typhoid fever in nearly all cases, unless the patient has previously passed through the disease, for the blood often continues to agglutinate the bacilli for many years after recovery. About half the cases do not give a positive reaction before the beginning of the second week, and about a third of the cases do not give a reaction before the early part of the third week. It may appear, on the other hand, as early as the fourth or fifth day. Rarely it is first obtained in a relapse.

SPECIFIC GRAVITY OF THE BLOOD.

Hammerschlag's Method.—A mixture of chloroform (sp. gr. 1.526) and benzin (sp. gr. 0.889) is prepared in such proportions that the specific gravity of the mixture is nearly that of the blood (1.050 to 1.060). A drop of the blood, which must be free from air, is dropped upon the surface of this mixture. If the drop sink, it is heavier than the mixture, and chloroform must be added; if it rest on the surface, it is lighter, and benzin must be added. The fluids must be thoroughly mixed after each addition. When the blood-drop remains stationary, neither sinking nor rising, the specific gravity of the blood is the same as that

of the mixture, which can be determined with the urinometer. This mixture can be kept indefinitely after being used, the blood-drop having been removed with a pipette.

BACTERIOLOGICAL EXAMINATION OF THE BLOOD.

Bacteria are found in the blood in such small numbers, as a rule, that they can be detected only after cultivation on suitable media. This can seldom be accomplished without laboratory facilities. The method consists in flowing the blood removed from a vein with a hypodermic syringe over the surface of a blood-serum and agar culture-medium. The culture must be kept at a temperature of 37° C. Plate cultures are the most satisfactory. Any growth that occurs may be examined by the usual methods of bacteriological examination.

Other Tests.—***Hemoglobinemia.***—The hematocrit tube is filled with blood and revolved rapidly for three minutes upon the centrifuge. Normally the blood separates into three portions, the erythrocytes occupying about half the space, leucocytes a narrow band, and clear plasma the other portion of the tube. If free hemoglobin be present, the plasma is tinged with red.

Diabetes.—There are two fairly reliable tests of diabetic blood. *Bremer's test* consists in the application of the acid stains. While in normal blood these are promptly taken up by the erythrocytes, they have no effect upon these cells in diabetic blood.

Williamson's test consists in adding the blood to a methylene-blue solution. Diabetic blood changes the color to yellow, while normal blood produces no change.

TESTS FOR BLOOD.

Hemin Test.—Crush a small crystal of sodium chlorid, or, better, evaporate a drop of a 0.5 per cent salt solution on a slide, and add to it a small particle of the substance to be tested in a dry state. Fluids should be previously evaporated to dryness without scorching. Over these particles place a cover-glass, and allow to flow under this a drop of glacial acetic acid. Heat the specimen gently for about a minute, adding more acetic acid as it evaporates. As soon as a brownish stain is produced, allow the specimen to evaporate to dryness and mount in glycerin. Small rhomboidal crystals of hemin (hematin chlorid) are seen (Fig. 30) if blood be present in the specimen.

FIG. 30.—Hemin crystals.

Guaiacum Test.—To a few cubic centimeters of a freshly prepared tincture of guaiacum add half as much hydrogen peroxid in a test-tube. Under the mixture flow, through a tube or down the side of the test-tube, the fluid to be tested. Immediately or perhaps after ten or fifteen minutes, a blue ring appears at the junction of the two fluids. Stains may be tested by this method by first impregnating a piece of pure filter-paper with the stain or a solution obtained from it with distilled

water, then moistening an adjoining portion of the paper with the guaiacum-hydrogen-peroxid mixture. If blood be present the blue line is formed at the junction of the moistened areas. It must be remembered, however, that iodin, iodids, and many other substances produce this reaction.

Blood-Plates.—These are irregularly shaped bodies seldom recognized in the ordinary blood examination. They can sometimes be recognized, however, in a strictly fresh specimen. To obtain this, a cover-glass should be placed upon the slide, and a drop of blood deposited immediately from the ear at one edge of the cover. The plates are highly cohesive bodies about half the diameter of a red corpuscle and are usually found clinging together in irregular masses. They are colorless and have no ameboid movement. They may be stained, however, with eosin.

Muller's Blood-Dust.—These are small, highly refractile, colorless granules from ¼ to 1μ in diameter, or equal to the finest fat-droplets, and exhibiting rapid molecular motion, but no independent motility. They are insoluble in alcohol or ether, and stain with eosin or the triacid stain, but not with osmic acid. They are best seen with the Welsbach light. Similar granules are sometimes seen in hydrocele fluid and pus.

EXAMINATION OF STOMACH-CONTENTS.

The stomach-contents should be obtained for examination one hour after a test-meal, which should be ingested in the morning without other food. The contents are obtained by means of the stomach-tube. In conditions in which the digestion is slow, and when the food is retained in the stomach longer than is normal, the stomach should be washed out the evening before. Several test-meals have been proposed, but those of Ewald and Boas are most employed.

Test-Meals.—Ewald's test-breakfast consists of a wheat-roll to be eaten without butter, and 300 to 400 c.c. of water or weak tea without sugar.

Boas's test-breakfast consists of a tablespoonful of oatmeal added to a quart (liter) of water with a little salt, and boiled down to a pint (500 c.c.). The only advantage claimed for this meal over that of Ewald is that it is free from lactic acid.

Passing the Stomach-Tube.—The tube should be moistened with clear water before it is introduced. The patient should sit erect with the head thrown a little backward and the mouth wide open. He should be instructed to breathe regularly during the passage of the tube. Attempts to swallow as advised by many writers do not always facilitate its passage and often confuse the patient. The tip of the tube is placed against the posterior wall of the pharynx, and then steadily pushed onward until the white ring is about on a level with the incisor teeth. The first passage of the tube is disagreeable to most patients, and it may be rendered difficult, if not impossible, by the gagging that is excited. Usually, however, with a little persuasion and by quick manipulation it can be made to reach the stomach before the patient becomes greatly alarmed. The gagging and nervous excitement can be diminished in many cases by holding the end of the tube in chopped ice until it becomes thoroughly cold before it is introduced. The contents of

the stomach can generally be made to flow by having the patient contract the abdominal muscles with the glottis closed, as in straining in the act of defecation. In some cases, however, it must be started by means of suction with the Politzer bag, or by "stripping" the tube. The tube is held firmly between the thumb and finger of the left hand, while the right thumb and finger are pressed firmly, and drawn down the tube in such a manner as to produce suction within it. After the flow has been started, the tube is converted into a siphon by holding the external end below the level of the stomach. From 50 to 75 c.c. of contents are usually obtained.

The stomach-contents should be filtered through dry filter-paper, a process requiring considerable time unless a filter-pump is used. After this the contents may be examined for free and combined acids, the digestive ferments, the products of digestion, and other ingredients, if desired.

QUALITATIVE TESTS.

Test for Free Acids.—A few drops of a 1 per cent aqueous solution of Congo-red are added to a few drops of the filtered stomach-contents. If free or combined hydrochloric acid or the organic acids be present, a dark-blue or blackish-brown color is produced, while in a neutral or alkaline solution the red color is imparted.

Test for Hydrochloric Acid.—*Töpfer's Test.*—One or two drops of a 0.5 per cent alcoholic solution of dimethylamidoazobenzol are added to about double the quantity of stomach-contents. If hydrochloric acid be present, a bright red color is produced; if it be absent, a bright yellow is produced. The test is sensitive to about 0.01 per cent of the acid, more minute traces yielding a brownish color.

Boas's Test.—The test-solution consists of

Resublimed resorcin	5.0
White sugar	3.0
95 per cent alcohol	100.0

To a few drops of the gastric contents in a porcelain evaporating-dish a nearly equal quantity of the test-solution is added. The mixture is then gently evaporated over a Bunsen burner. In the presence of hydrochloric acid a rose-red color appears around the edge of the mixture as it evaporates; if it be absent, a yellow or brownish color is produced.

Günzburg's Test.—This test is now less frequently employed than formerly, owing to the instability of the test-solution and the cost of the ingredients. The formula is: Phloroglucin 2.0, vanillin 1.0, absolute alcohol 30.0. Its application and the results are the same as those of the Boas test.

Test for Organic Acids.—A small portion of the gastric contents is shaken with eight or ten times its quantity of ether having a neutral reaction. The reaction of the ethereal extract is then tested with litmus-paper. An acid reaction indicates the presence of organic acids. If, however, the Congo-red test fails, organic acids are not present.

Test for Lactic Acid.—The acid reaction obtained in the foregoing test is usually due to the presence of lactic acid. Its presence may be

more positively determined, however, by Uffelmann's test. The test-solution is made fresh as it is required by adding a few drops of a dilute aqueous solution of ferric chlorid to a 2 per cent phenol solution, and diluted with water until an amethyst color is obtained. Two or three cubic centimeters of the filtered stomach-contents is added to a like quantity of this solution. In the presence of lactic acid a bright lemon or canary color is produced. In its absence the amethyst color is retained or changed to a gray. Reacting to lactic acid alone, the test is sensitive to about 0.01 per cent, but its accuracy is impaired by the presence of hydrochloric and other acids. This difficulty may be avoided by applying the test to the ethereal extract of the contents.

Test for Fatty Acids.—The simplest test for the volatile fatty acids, acetic, butyric, etc., is made by holding a piece of moistened litmus-paper in the vapor arising from boiling gastric contents in a test-tube.

Test for Pepsin.—The test for pepsin is made by submitting small fragments of egg-albumen, coagulated by boiling, to the action of the stomach-contents. If the stomach-contents contain hydrochloric acid, the fragments of albumen are dropped into 5 or 10 c.c. of it in a test-tube and kept at a temperature of 37° C. If the pepsin be normal in quantity, the albumen is completely digested and dissolved in six or seven hours; if it be deficient, the digestion is delayed, and if it be absent, no digestion occurs. In case the hydrochloric acid is absent from the gastric contents, it must be added in the ratio of 0.1 to 0.2 per cent.

Test for Rennet.—A few drops of the stomach-contents are added to 10 or 15 c.c. of milk, and kept at a temperature of 37° C. If the rennet-ferment be normal, the milk will coagulate in 10 or 15 minutes. Delayed coagulation indicates deficiency of rennet. When hydrochloric acid is absent, however, a few drops of calcium chlorid must be added in order to convert the rennet-zymogen into active rennet.

Test for Proteids.—Acid albumin, or syntonin, is precipitated by exactly neutralizing the filtered stomach-contents. An excess of either acid or alkali causes it to be again dissolved.

Albumin.—The acid albumin is first removed by filtration after precipitation in the foregoing test. The filtrate is then boiled or otherwise tested for albumin. A cloudiness or precipitate indicates its presence.

Albumose (Propepton).—The syntonin and albumin are first removed by boiling and filtering a small quantity of the gastric contents (both are thus thrown down without neutralization); the filtrate is then allowed to cool, and it is mixed with an equal volume of a saturated solution of sodium chlorid, and a drop or two of acetic acid is added. If albumose be present, the fluid becomes turbid; the turbidity disappears upon heating and reappears upon cooling.

Pepton.—Albumin, syntonin, and albumose are first removed by the above methods. The filtrate is then tested with the biuret test. A purple or a violet red color indicates the presence of pepton. For accuracy, the absence of albumin should first be determined by means of the ferrocyanid test.

Blood.—When the presence of blood cannot be determined by inspection or by means of the microscope, the hemin or guaiacum test may be employed (p. 718).

Bile.—The presence of bile can generally be determined by the green color of the gastric contents, but a more reliable method is the Gmelin nitric-acid-contact test, in which a play of colors is produced, of which green is characteristic.

Carbohydrates.—A few cubic centimeters of Lugol's solution of iodin and potassium iodid are diluted until only a faint color remains. A few drops of the filtered contents are then added. A blue color indicates the presence of unchanged starch; a deep mahogany brown indicates erythrodextrin. The presence of sugar can be determined by the usual copper or fermentation tests.

QUANTITATIVE TESTS.

Volumetric analysis is employed in testing the gastric contents chiefly with a view to determining the total acidity, the acidity due to free or combined hydrochloric acid, and that due to the organic acids. These tests are usually made by titration with a decinormal solution of sodium hydroxid and a suitable indicator—a solution by means of which the exact neutralization of the acidity can be recognized. The sodium solution must be made with the greatest accuracy, and owing to the hygroscopic nature of the salt it cannot be made by weight. About 4 grams of sodium hydroxid must be dissolved in 8 or 9 c.c. of distilled water, and the solution tested with a decinormal solution of hydrochloric acid which can be more readily prepared by the specific-gravity method, or purchased ready for use. After the exact alkalinity of the solution has been thus determined, sufficient distilled water is added to reduce it to the decinormal standard, *i.e.*, representing 0.1 gram of the hydroxid to the liter, or 0.3996 per cent.

Determination of Total Acidity.—A definite quantity of stomach-contents, as 5 or 10 c.c., is placed in a beaker (a porcelain dish or capsule is even better on account of its white color). To this are added a few drops of 1 per cent solution of phenolphthalein in 50 per cent alcohol as an indicator. This is then titrated with the decinormal sodium-hydroxid solution, drop by drop, from the burette, and followed by agitation or stirring of the mixture until a permanent pale-pink color is produced. The degree of acidity is then determined by multiplying the quantity of sodium-hydroxid solution employed, expressed in cubic centimeters, by its acidity, which is 100, and dividing the product by the number of cubic centimeters of the gastric contents tested. The normal acidity is generally from 40 to 65.

Determination of Free Hydrochloric Acid.—To a definite quantity of the gastric fluid add a few drops of a 0.5 per cent alcoholic solution of dimethylamidoazobenzol. When HCl is present, a cherry or brownish color is produced; if it is absent, the color is a pure yellow, and titration is unnecessary. To determine the acidity, the mixture is titrated with the decinormal sodium-solution until a permanent, pure yellow color is obtained, and the calculation is made as in the preceding test. Normally the acidity due to free HCl is between 40 and 60. To determine the percentage of the acidity, the degree should be multiplied by .003637.

Determination of Combined Hydrochloric Acid.—*The Alizarin Method.*

—The gastric fluid is titrated as before, using a 1 per cent aqueous solution of alizarin-sodium-solfonat as an indicator. When the acidity has been neutralized, a permanent, pale violet color is produced. The percentage of acidity may then be calculated as in the preceding test. The method lacks accuracy, however, owing to the wide range of almost imperceptible color that can be produced. The more accurate tests are not applicable to clinical work.

Determination of Organic Acids.—After the total acidity of the specimen has been determined, the organic acids should be removed from another portion of the gastric filtrate by extraction with ether. To accomplish this, a portion of the filtrate is shaken with a quantity of neutral ether, and the fluids allowed to separate. The gastric portion is then shaken with another quantity of ether, and the process is repeated until the gastric fluid has been extracted with eight or ten times its volume of ether. Its total acidity is then determined as before, and the difference in the result represents the loss occasioned by the removal of the organic acids.

Determination of the Fatty Acids.—Since the fatty acids are volatile, they can be removed by heat. After the total acidity has been determined, another definite quantity is thoroughly boiled, and the fluid lost by evaporation is replaced by the addition of water. The total acidity is again determined by titration, and the loss represents the degree of acidity due to the fatty acids.

Determination of Lactic Acid.—The degree of acidity due to lactic acid is represented by the difference between the total acidity due to organic acids and that due to fatty acids. If fatty acids be absent, the acidity due to lactic acid represents the total organic acidity. To determine the percentage of lactic acidity, multiply the degree by .008979.

MICROSCOPIC EXAMINATION.

This examination is generally of minor importance, owing to the almost constant presence of a great variety of unimportant substances. For the examination, a small mass of the solid matter left in the filter should be picked up on the platinum loop, spread on a slide, and examined with low and medium power. After the Ewald breakfast the field is largely made up of starch-granules. These can be more distinctly brought out by passing a drop of dilute iodin solution under the cover-glass. Other substances commonly seen are fragments of other undigested food, fat-globules, crystals of fatty acids, erythrocytes, leucocytes, various micro-organisms, and sometimes leucin, tyrosin, or cholesterin crystals from the intestine. The bacteria may be stained in the usual way, or cultures may be made in order to differentiate the varieties. Fragments of solid tissues may be hardened and cut for histological examination.

EXAMINATION OF THE STOMACH-CONTENTS AFTER FASTING.

This examination is sometimes desirable in order to determine the presence of superacidity and its character. The stomach must be washed out and emptied the evening before. No food or drink is then taken until the stomach-contents have been obtained with the stomach-tube

in the morning. If the quantity obtained exceed 60 c.c., there is supersecretion. The percentage of acidity, pepsin, and rennet may then be determined by the methods that have been given.

EXAMINATION OF STOMACH-WASHINGS.

The chief importance of this is in cases of suspected poisoning. It is best in such cases, if possible, to secure some of the stomach-contents before lavage; otherwise, the water must be examined for the various poisons. If the examination is to be made by a chemist with a view to criminal prosecution, the specimen should be placed in a clean bottle, securely sealed, and marked in some manner, as by pasting a strip of paper over the stopper and neck of the bottle with the legend of the case and the signature of the physician. It is well, also, to give the date and hour of the examination.

EXAMINATION OF VOMITUS.

This examination is seldom of importance except for the purpose of determining whether, in a case of suspected stricture of the esophagus, the food ingested reaches the stomach. The presence of HCl, pepsin, and rennet is generally sufficient evidence that the vomitus has come from the stomach, and that it has not simply been regurgitated from the esophagus. In achylia gastrica, however, these substances may be absent. The coloring matter of bile should be tested for, and, if this be present, it is conclusive evidence that the food has come from the stomach.

Test of the Motor Power of the Stomach.—If food is brought up with the water in lavage of the stomach seven hours or longer after its ingestion, the motor power of the stomach is deficient; if food be absent when the washing is done within three or five hours after a meal of mixed food, it is evidence of increased motility.

Salol Test.—A gram (gr. xv) of salol is administered in capsules immediately after a meal. As soon as this substance reaches the alkaline juice of the intestine it is converted into phenol and salicylic acid, and the latter substance appears almost immediately in the urine. Its presence is detected by the addition to the urine of a few drops of a dilute aqueous solution of ferric chlorid. A violet or brown color is significant of salicylic acid. The test should be repeated every half-hour until a reaction is obtained. This occurs normally in an hour to an hour and a half. Should the stomach-contents be alkaline, however, the reaction appears much earlier and the test is of no value.

Test of Absorptive Power.—From 0.20 to 0.40 gram (gr. iij—vj) of potassium iodid should be administered, during fasting, in a capsule freed from the drug upon its exterior. The saliva is then tested for iodin with starch-paper at intervals of one or two minutes. Normally the reaction is obtained in five to fifteen minutes. A much longer interval indicates delayed absorption.

EXAMINATION OF INTESTINAL DISCHARGES.

The feces and other intestinal discharges may be submitted to macroscopic, microscopic, chemical, and bacteriological examination.

Macroscopic Examination.—Simple inspection of the feces reveals their color, consistence, and form, and affords an adequate idea of the completeness of the processes of digestion as well as of the presence of parasites, foreign bodies, blood, pus, fat, shreds of tissue, and other abnormal substances. The source of blood may be inferred from its color and other conditions. When the fecal mass is merely streaked with it, the source of the hemorrhage is usually at or near the anal orifice; when bright fluid blood accompanies the dejection, it is generally from the rectum and may be due to hemorrhoids or ulcer. When the blood is coagulated or black and tarry, its source is higher up in the intestine or stomach. The odor also reveals to some extent the completeness of the digestion. Segments of tapeworm should be compressed between two slips of glass in order to render them more translucent.

Microscopic Examination.—A small portion should be picked up on the platinum loop, transferred to a slide, and mixed with a drop of water, after which the cover-glass is applied. The examination should be made first with a low power. The substances commonly revealed are fragments of incompletely digested animal or vegetable food, as muscle

FIG. 31.—Ova of intestinal worms (X275). *a*, Tenia sagginata with and without albuminous covering; *b*, ascaris lumbricoides; *c*, trichocephalus dispar; *d*, anchylostoma duodenale. (Nichols.)

fibers, connective-tissue fibers, starch-granules, chlorophyll, fat, fatty-acid crystals, cholesterin, amorphous and granular matter, ammoniomagnesium-phosphate crystals, calcium oxalate and carbonate, spermin and hematoidin crystals, erythrocytes, leucocytes, epithelium, saccharomyces and other fungi, bacteria, small animal parasites, and crystals or other remains of drugs that have been ingested.

In examining for small parasites and ova, a moderate quantity of fecal matter should be placed in a cylindrical glass, thoroughly mixed with water and allowed to stand for ten minutes. The parasites and ova settle to the bottom, and by pouring off the supernatant fluid and repeating the process several times they may be obtained free from extraneous matter. They can then be subjected to microscopic examination. The more frequent varieties of ova are illustrated in Fig. 31.

Fat-globules can be more distinctly brought out by staining them red with Sudan III. To prepare this solution, first allow a saturated alcoholic solution of the Sudan III to stand a few days, then add one part

of it to one part each of alcohol and water. The solution is ready for use as soon as it has become clear.

Starch-cells are rendered more visible by treatment with dilute Lugol's solution. A drop of acetic acid renders leucocytes and epithelial cells more distinct, and dissolves phosphates and carbonates, the latter bodies evolving minute bubbles of carbonic acid.

Fibrin may be stained red with Ehrlich's triple stain or blue with Weigert's gentian-violet stain. Mucin is always present, and when abnormally abundant it can generally be recognized without the microscope. If its identity is doubtful, it may be dried, fixed on a slide with alcohol or mercuric-chlorid solution, and stained blue with methylene blue, green with the triple stain, and reddish with toluidin blue. Solid tissue-particles should be broken up, teased, or sectioned, as their character permits.

Chemical examination is seldom of sufficient importance to justify the labor. The reaction is often of importance, however, and this is determined with litmus-paper. If the fecal mass be firm and dry, it must be broken open, and the moistened litmus-paper pressed between its surfaces. A quantitative test of the degree of acidity or alkalinity can be made by testing a watery extract of a definite quantity of the fecal matter, using the titration method employed for stomach-contents.

The *proteids* may be recognized by digesting feces with water acidulated with acetic acid, and, after filtering, applying the usual tests for albumin, albumose, and pepton.

For the *carbohydrates* boil a small quantity of the matter in water, filter, concentrate the filtrate by evaporation, and test for starch and erythrodextrin with iodin, and for sugar with the urine tests.

Fat and the *fatty acids* may be recognized with the microscope or by extracting with ether and applying the tests given in the examination of the gastric fluid.

Bilirubin may be detected with the nitric-acid test applied to the aqueous or chloroform extract, or the acid may be applied directly to the fecal mass. The green color is distinctive.

Blood is distinguished by the hemin test applied either to a dried fragment of the clot or to an evaporated aqueous extract of the fecal matter. The guaiacum test may also be applied to the aqueous extract.

Calculi.—For the discovery of these, the feces should be mixed with water and forced through a sieve of fine mesh. The sandlike particles, if too small for macroscopic recognition, may be examined chemically and microscopically.

Bacteriological Examination.—The micro-organisms ordinarily found in the intestinal discharges are so numerous that little can generally be gained from an attempt to isolate them. Many of them can generally be recognized, however, with any of the usual stains applied to a dried and fixed specimen on the slide. A few are worthy of note.

The Ameba Coli.—The feces must be fresh and examined before cooling, on a warm slide, in order to retain the ameboid movement. *Tubercle bacilli* may be stained in the usual manner after drying and fixing on a slide. *Typhoid bacilli* can sometimes be obtained. The *cholera vibrios* are usually so numerous as to be recognized without difficulty.

Plate-cultures may be made from the dejections, but the growth is generally so luxuriant as to render the isolation of species next to impossible in clinical work.

DISINFECTION OF DEJECTA.

The best disinfecting agents for this purpose are a 1:500 acidulated solution of mercuric chlorid, a 1:20 solution of carbolic acid, a 1:20 solution of formalin, and chlorinated lime. The latter substance should be used in the dry state. It is especially suited to the disinfection of trenches and privies. The carbolic-acid and formalin solutions are less corrosive to metallic drain-pipes than the corrosive sublimate.

The disinfection of stools, especially in typhoid fever, should be begun as soon as the disease is recognized, and continued for at least ten days after the fever has subsided. The following rules should be observed:

1. The bedpan should contain a pint of the bichlorid solution at all times, ready to receive the dejection. The pan must be cleansed with boiling water and one of the disinfecting solutions.
2. Enough of the solution should be poured over the stool to cover it and be thoroughly mixed with it; the vessel should then stand two hours before it is emptied.
3. Lumps of fecal matter should be immediately broken up with a stick, and the stick subsequently burned.
4. The urine should be disinfected by the addition of enough carbolic acid or mercuric chlorid to convert it into a 1:20 or 1:500 solution, respectively.
5. All linen and bedclothing should be soaked in a 1:20 carbolic-acid solution, and afterward boiled for two hours.
6. As a further precaution, the nurses, physicians, and other attendants should wash their hands, and immerse them in a 1:1000 mercuric-chlorid solution after handling the patient, the bedpan, syringe, thermometer, or other articles coming in contact with him.

EXAMINATION OF THE URINE.

The Specimen.—To secure accurate results the specimen should be taken from a mixture of all the urine voided in twenty-four hours. When this cannot be done, a specimen passed three hours after a meal is most likely to reveal any abnormalities present. To preserve the specimen, a few drops of formalin, chloroform, or alcohol, or a few grains of chloral or salicylic acid may be added. The average normal quantity in twenty-four hours is from 1,200 to 1,500 c.c. (40 to 50 ounces). The reaction of a mixed specimen is usually acid, but after a meal consisting largely of carbohydrates it may be alkaline, in health.

Reaction.—This is tested with litmus-paper. Acid urine turns blue litmus red; alkaline urine turns red litmus blue. An amphoteric reaction may occur in which both papers are changed by the same specimen. Huppert attributes this to the presence of acid and neutral phosphates. When the blue color of red litmus fades upon becoming dry, the reaction is due to a volatile alkali.

Specific Gravity.—This can be determined with sufficient accuracy with the ordinary urinometer. An instrument of certified accuracy should be employed, however, as many of those offered in the market are unreliable. If the specimen be fresh, its temperature should be measured, and one degree added to the reading of the urinometer for every seven degrees of temperature above the standard of the instrument, which is usually 60° F.

Solid Ingredients.—The simplest method of approximately estimating the solids of the urine is to multiply the last two figures of the specific gravity by 2.33, which gives the number of grams in each 1,000 c.c.

TESTS FOR NORMAL INGREDIENTS.

Urea.—*Test.*—To a drop of the fluid (urine) on a slide add a drop of pure nitric acid and gently warm. Rhombic or hexagonal prisms or plates of urea nitrate are formed, and are visible with a low power.

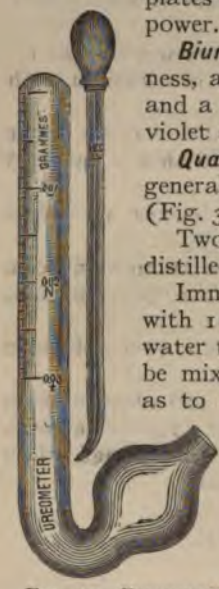

FIG. 32.—Doremus apparatus for the estimation of urea.

Biuret Reaction.—To the urine evaporated almost to dryness, and while warm, add a trace of potassium hydroxid and a drop of dilute cupric-sulphate solution. A rose-red or violet color denotes urea.

Quantitative Determination.—The hypobromite method is generally employed by means of the Doremus ureometer (Fig. 32).

Two solutions are required: (*a*) Sodium hydroxid 100, distilled water 250; and (*b*) bromin.

Immediately before the test is made, mix 1 c.c. bromin with 10 c.c. of the sodium-hydroxid solution, and add enough water to fill the long arm of the ureometer. The fluids may be mixed in the ureometer. The instrument is reclined so as to allow the fluids to fill the long arm, then restored to the upright position. One cubic centimeter of the urine is now slowly injected from a pipette into the bulb so as to come into contact with the hypobromite mixture only at the base of the long arm, allowing the liberated nitrogen to collect at the top. The result in fractions of a gram of urea to the cubic centimeter of urine may be read from the scale after about fifteen minutes. The mark 0.02 represents the normal 2 per cent.

Uric Acid.—*Murexid Test.*—To a small quantity of the sediment or to the residue after evaporation, in a porcelain capsule, add a few drops of nitric acid, evaporate over a flame to almost dryness, and add a drop of ammonia. In the presence of uric acid, a beautiful purple-red color is produced.

Silver Test.—Moisten a piece of white filter-paper with a little silver-nitrate solution. Touch the spot with a drop of urine made alkaline with sodium-carbonate solution. A brownish yellow color indicates a trace, and black 0.001 per cent or more of uric acid.

Xanthin.—With a few drops of urine mix an equal quantity of nitric acid, and evaporate to dryness. The yellow residue is changed to red by potassium hydroxid, and reddish purple by heat.

Creatinin.—*Weyl's Test.*—Add to the urine, solution of sodium nitrocyanid, followed by sodium hydroxid. A red color is produced, which turns to yellow upon standing.

By the addition of zinc-chlorid solution, and evaporation, groups of characteristic crystals of creatinin-zinc chlorid are formed (Fig. 33).

Ferments.—Pepsin has been found in normal urine, particularly in that voided in the morning. It is detected by soaking small pieces of fibrin in the urine, then removing them to a 0.1 per cent solution of hydrochloric acid at 37° C., where they are quickly digested. Traces of a milk-curdling ferment like rennet and a diastatic ferment, probably trypsin, have been isolated from the urine.

Chlorids.—Add to the urine in a large test-tube a little nitric acid to hold the phosphates in solution, then a drop of silver-nitrate solution (1 : 8). A precipitate forms, which is soluble in ammonia, but insoluble in nitric acid. If the precipitation be merely a flaky white cloud, the chlorids are diminished in quantity; if it be a heavy white mass, falling quickly, they are at least normal in quantity; if there be no precipitate, they are absent.

Quantitative Test.—*Mohr's Method.*—The following solutions are used: (1) A standard silver-nitrate solution made by dissolving 29.075 gm. of pure silver nitrate in 1,000 c.c. of distilled water, each cubic centimeter of which will precipitate 10 mgm. (0.010) of sodium chlorid; and (2) a saturated aqueous solution of neutral potassium chromate.

FIG. 33.—Creatinin-zinc chlorid crystals.

Dilute 10 c.c. of urine with 100 c.c. of distilled water, and add a few drops of the potassium-chromate solution. Then titrate with the silver solution. A white precipitate of silver chlorid is produced until all the chlorids have been removed; then a red precipitate of silver chromate begins to form. As soon as the pink color becomes permanent, the calculation can be made from the quantity of silver solution that has been used. One cubic centimeter should be deducted from the quantity of silver solution, however, to offset the other substances which unite with the silver before the potassium chromate.

Phosphates.—*Earthy Phosphates.*—Render the urine strongly alkaline with sodium, potassium, or ammonia, and gently warm. The earthy phosphates are precipitated and soon settle. If in a test-tube of 2 cm. diameter they form a deposit to the depth of 1 cm.; their quantity is normal.

Alkaline Phosphates.—Remove the precipitate formed in the preceding test. To the filtrate add one-third its volume of a solution consisting of magnesium sulphate and ammonium chlorid each 1 part, distilled water 8 parts, and pure liquor ammonia 1 part. The alkaline phosphates are precipitated in a white cloud. If a distinctly creamy appearance is produced, they are increased; if a very slight opacity, they are diminished.

Sulphates.—Acidulate 10 c.c. of urine with hydrochloric acid, and add one-third the quantity of barium-chlorid solution. A milky white precipitate indicates a normal quantity of the sulphates.

Carbonates.—The addition of an acid to urine containing carbonates liberates carbonic acid. By passing this gas through lime-water or baryta-water, a cloudy precipitate is formed.

ABNORMAL CONSTITUENTS.

Albumin.—Heat the upper portion of a column of urine in a test-tube. A precipitate which is not redissolved by the addition of nitric acid is due to albumin. Excess of acid must be avoided. Alkaline urine must be rendered acid before boiling. This test reacts to globulin, mucin, pine acids from cubebs, copaiba, etc., and albumose is precipitated after the specimen becomes cold. The pine-acid precipitate is redissolved by alcohol.

Nitric Acid Test (Heller).—Underflow the urine with nitric acid without mixing. A white ring is formed at the line of junction corresponding in depth of whiteness with the quantity of albumin. The precipitate may not appear for an hour or two. Slightly warming the urine intensifies the reaction and renders nucleoalbumin less apt to appear. A cloudiness due to mucin may appear at a little distance above the line of contact.

Purdy's Heat and Acid Test.—Add to the urine enough of a filtered saturated solution of sodium chlorid to raise its specific gravity ten or fifteen degrees, to prevent reaction with mucin. To two-thirds of a test-tubeful of this mixture add one or two drops of strong acetic acid, and boil the upper inch of the column for about half a minute. Albumin will appear in the boiled portion as a milky turbidity. This is one of the most delicate tests.

Potassium-Ferrocyanid Test.—Pour into a clean test-tube 15 to 30 drops of acetic acid, add to it three or four times as much of a 5-per-cent solution of potassium ferrocyanid, and shake. Then fill the tube two-thirds full of the urine. Albumin will appear as a more or less milky cloudiness. To recognize a slight cloudiness, the urine tested should be compared with another sample of the same urine in a tube of the same size. This is one of the most reliable tests, as it reveals all forms of albumin, and nothing else. It must be performed just as directed.

Potassium Mercuric Iodid Test.—The following solution is used: Potassium iodid, 3.32 gm.; mercuric chlorid, 1.35 gm.; acetic acid, 20 c.c.; distilled water, q.s. to make 100 c.c. The salts are dissolved separately, the solutions mixed, the acetic acid added, with enough water to make up the volume. A little of the reagent is poured into a test-tube, and the urine flowed over it without mixing. The test is sensitive to albumin, but reacts also to pepton, proteoses, mucin, and the pine acids.

Picric-Acid Test.—Over the surface of the urine flow a saturated solution of picric acid (6 or 7 grains dissolved in an ounce of hot water). Albumin is revealed by a cloudiness in the area in which the fluids mix. Albumin, mucin, pepton, proteoses, and vegetable alkaloids respond to the test, but all except albumin and mucin are redissolved by heat.

Biuret Test.—Add to the urine a solution of potassium hydroxid, then

a weak solution of cupric sulphate, drop by drop from a pipette. If albumin be present, the greenish precipitate is redissolved and the mixture assumes a reddish violet color. Albumose and globulin yield the same reaction, urea a rose-red or violet, and pepton a red color.

Millon's Test.—Dissolve one part of mercury in two parts of nitric acid of 1.42 sp. gr., and dilute with two volumes of water. To one dram of urine add ten minims. A trace of albumin is recognized by a red color on heating; larger quantities produce a precipitate which becomes red on heating. The test reveals the aromatics and some of the benzol group.

Alcohol Test (Truax).—From a pipette passed down nearly to the surface of 98 per cent alcohol in a test-tube, drop a little urine. If albumin be present, it is precipitated in a whitish streak extending to the bottom of the tube; if mucin be present, a general cloudiness is produced.

Quantitative Determination.—*Centrifugal Method.*—To 10 c.c. of urine in the centrifuge tube add 3.5 c.c. 10 per cent potassium-ferrocyanid solution, and 1.5 c.c. acetic acid, and mix thoroughly. After revolving until the fluid is left perfectly clear, each tenth c.c. of precipitate at the bottom of the tube denotes 1 per cent of albumin by bulk. It should be remembered that a precipitate amounting to 50 per cent by bulk in any of the tests rarely exceeds 2 per cent by weight.

Globulin.—Exactly neutralize the urine, and filter; then add magnesium sulphate until it no longer dissolves. If globulin be present, a white precipitate is formed.

Roberts's Test.—Globulin falls out of solution when the specific gravity is reduced below 1.002. To a test-tubeful of distilled water add the urine by drops. If globulin be present, each drop is followed by a milky streak until the entire volume becomes opaque. The cloudiness is removed by acetic acid.

Hemoglobin.—*Heller's Test.*—Render the urine strongly alkaline with sodium-hydroxid solution, and heat to boiling. The precipitate of earthy phosphates is colored red by hematin. If the urine be alkaline, a few drops of magnesium solution produces an artificial precipitate, which, when heated, brings out the hematin. The guaiacum and hemin tests may also be employed.

Fibrin.—The urine may become flaky, coagulæ may form, or it may coagulate into a firm mass after being voided, especially in chyluria. The fibrin is not soluble in water, swells on the addition of hydrochloric acid, and is dissolved by pepsin added to the acid fluid.

Alkapton.—*Test.*—Add to the urine one drop of a very dilute ferric-chlorid solution; a dull green color is produced, which immediately vanishes. Repeated additions of the same solution produce a repetition of the reaction. The surface of alkapton urine becomes dark upon exposure to the atmosphere, and the discoloration gradually extends to the entire specimen.

TESTS FOR SUGAR.

Trommer's Test.—To a quantity of urine in a test-tube add half as much sodium or potassium hydroxid solution (1:3); to this add a drop or two of cupric-sulphate solution (1:10), and shake. An azure-blue

color is produced. Heat the mixture to boiling. If sugar be present, the color changes first to yellow, then to an orange-red. Although the azure-blue color is not sufficiently distinctive, it does not usually appear in the absence of sugar.

Fehling's Test.—The following reagents are required: (*a*) Copper solution: Dissolve of pure crystallized cupric sulphate, 34.64 gm., in enough of distilled water to make 500 c.c. (*b*) Rochelle salt solution: Dissolve sodium hydroxid, 125 gm., and chemically pure potassium-sodium tartrate, 173 gm., in sufficient distilled water to make 500 c.c. These solutions may be preserved in well-stoppered bottles. Fehling's solution is made by mixing them in equal parts at the time of using.

The Method.—About 1 c.c. of the Fehling solution is diluted in a test-tube with three or four times as much distilled water, and boiled. If no change of color is produced, the urine is added drop by drop until a slight reduction of the copper occurs, as indicated by a yellow color, or until the quantity of added urine equals that of the reagent. The mixture is again heated, and if no reduction occurs the test is set aside for several hours. If there is still no reaction, sugar is absent. The test is one of the most delicate. If the reagent change color in the preliminary boiling, new solutions must be obtained.

Haines's Test.—Formula: Copper sulphate 30 grains, distilled water

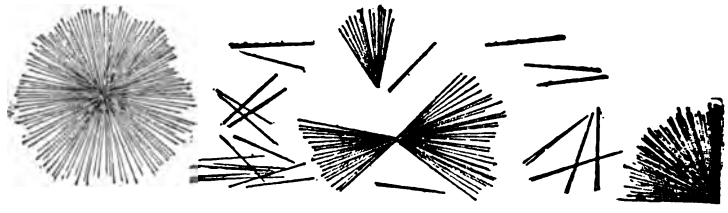

FIG. 34.—Rosette and rays of phenylglucosazone crystals.

a half ounce; make a perfect solution and add glycerin a half ounce; mix thoroughly and add liquor potassæ 5 ounces. Boil about a dram of the solution in a test-tube, add not more than 6 or 8 drams of the urine, and boil gently. If sugar be present, a copious yellow or orange precipitate is thrown down.

Böttger's Bismuth Test.—Add to the urine in a test-tube an equal volume of sodium-hydroxid solution and a very small quantity of pure bismuth subnitrate. Boil gently for one or two minutes. If sugar be present, the mixture turns gray, brown, or black, according to the quantity of sugar. If the quantity be very small, only the bismuth is discolored. Albumin and other sulphur compounds must first be removed. The test must not be made in a test-tube that has been previously used for either of the copper tests.

Phenylhydrazin Test.—To 25 c.c. of urine in a capsule add 1 gm. phenylhydrazin hydrochlorid, 0.75 gm. sodium acetate, and 10 c.c. distilled water. Warm the mixture for an hour on a water-bath, remove, and let cool. If sugar be present, a yellowish deposit is formed, which the microscope shows to be composed of fine, brilliant yellow crystals arranged singly or in stars (Fig. 34). This phenylglucosazon melts

at 204° C. Yellow scales or spheres do not denote sugar. Care must be taken not to allow the phenylhydrazin to come in contact with the hands.

Fermentation Test.—The simplest accurate method of performing this test is by filling a test-tube about half full of mercury, and completely filling the remainder of the tube with the urine, introducing a small piece of compressed yeast and inverting the tube over a vessel of mercury. Then set the tube in a warm place for several hours. If fermentation occur, the carbonic-acid gas collects in the upper extremity of the tube. A special apparatus may be purchased, or it may be easily made by passing a doubly bent tube through a cork nearly to the bottom of a bottle containing the urine and yeast, hermetically sealing the cork. If sugar be present, the urine is expelled through the tube, its place being taken by the carbonic-acid gas. A control test should be made with distilled water and yeast from the same piece, for yeast sometimes undergoes spontaneous fermentation, probably due to the presence of sugar in it. A second control may be made with a weak solution of glucose, in order to prove the vitality of the yeast.

Heller-Moore Test.—To a little urine in a test-tube add half its volume of sodium-hydroxid solution and heat to boiling; if the precipitation of earthy phosphates is abundant, filter. As the mixture becomes hot, a yellow, yellowish brown, or brownish black color appears if sugar be present. Now add a few drops of nitric acid. The color vanishes and an odor of molasses (caramel) is given off. Albumin must first be removed. Highly colored urine should be decolorized by filtration through animal charcoal.

Picric-Acid Test.—To a small quantity of urine add two-thirds as much saturated solution of picric acid, and the same quantity of liquor potassæ. An orange color results from the incipient reducing action of creatinin on the picric acid. Turbidity denotes albumin, but does not interfere with the test. Boil the mixture for one minute; if sugar be present, a deep mahogany-color is produced, much deeper than that which occurs in normal urine.

Quantitative Determination.—*Purdy's Method.*—The formula of the standard solution is: Cupric sulphate, C.P., 4.742 gm.; potassium hydroxid, C.P., 23.50; glycerin, C.P., 38 c.c.; strong ammonia, U. S. P. (sp. gr. 0.9), 450 c.c.; distilled water, to make 1,000 c.c. Dissolve the copper and glycerin in 200 c.c. of distilled water with the aid of gentle heat. In another 200 c.c. of the distilled water dissolve the potassium hydroxid. Mix the two solutions, and when cold add the ammonia with enough distilled water to bring the whole volume up to 1,000 c.c.

Place exactly 35 c.c. of this test solution in the flask, dilute it with two volumes of distilled water, and bring the whole thoroughly to the boiling-point. Fill the burette to the zero mark with the urine to be tested, and slowly discharge it into the test-solution, drop by drop, until the blue color begins to fade, then more slowly until the color permanently disappears and leaves the fluid perfectly clear and translucent. The blue color may return after some time, but it is due only to the absorption of oxygen. The percentage of sugar is thus calculated: If the 35 c.c. of solution were reduced by 2 c.c. of urine, the

latter contained 1 per cent of sugar; if by 1 c.c., 2 per cent of sugar; if by 0.75 c.c., 3 per cent; if by 0.5 c.c., 4 per cent, etc.

Bile Acids.—*Pettenkofer's Test.*—Concentrated sulphuric acid free from nitric or sulphurous acid is very slowly added in nearly equal volume to the urine, the test-glass being held in ice-water to prevent rise of temperature above 60° C. A 10 per cent solution of cane-sugar is then added drop by drop, with constant stirring. A beautiful red color indicates the presence of bile acids. The color becomes a bluish violet in the course of a few days.

Bile Pigment.—*Gmelin's Test.*—Place in a test-tube a little strong nitric acid containing some commercial yellow nitrous acid, and flow over it the urine to be tested. A layer of green will form at the line of contact, surmounted from below upward by layers of blue, violet, red, and yellow, the green being distinctive of the bile pigment.

Rosenbach modifies the foregoing test by passing the urine through a fine, thick filter, then applying a drop of the nitrous-nitric acid to the filter. A pale yellow spot is formed, surrounded by rings of yellowish red, violet, blue, and green.

Heller's Test.—About a dram of pure hydrochloric acid is placed in a test-tube, and just enough urine mixed with it to distinctly color it. The mixture is then flowed upon a column of nitric acid, and a beautiful play of colors is produced. If the nitric acid be now stirred with a glass rod, the colors are distributed in layers throughout the mixture.

Ultzmann's Test.—Ten c.c. of urine are treated with 3 or 4 c.c. of a strong potassium-hydroxid solution and acidified with hydrochloric acid. If bile pigment be present, a beautiful green color is produced.

Indican (Indoxyl-Sulphuric Acid).—*Heller's Test.*—To a dram of HCl in a small wine-glass add slowly, with constant stirring, about 20 drops of urine. If the color produced be a pale yellow, the indican is normal in quantity; if blue or violet, it is increased. The addition of a drop or two of nitric acid renders the test more delicate.

McMunn's Test.—Equal parts of urine and HCl with a few drops of nitric acid are boiled together, cooled, and agitated with chloroform. If much indican be present, the chloroform takes a violet color.

Diazo Reaction.—Two solutions are required: (1) Sulphanilic acid 2 gm., and hydrochloric acid 50 c.c., in 1,000 c.c. of distilled water; (2) a 0.5 per cent solution of sodium nitrate.

Mix one part of No. 2 with 50 parts of No. 1, add to the mixture an equal volume of the urine, render strongly alkaline with ammonia, and shake well. The characteristic reaction consists in the production of a carmine color both in the mixture and in the foam. Normal urine yields a yellow color. The test may be performed by the contact method, by carefully flowing the ammonia upon the surface without mixing. A brownish-red ring is formed at the junction of the fluids.

DRUGS IN THE URINE.

Arsenic.—*Reinsch's Test.*—Add to the urine in a test-tube a few drops of hydrochloric acid, then introduce a piece of pure, bright copper foil ⅛ inch square, and boil for several minutes. If arsenic be present, a

dark gray coating is deposited on the copper. The test is more delicate if the urine be concentrated by slow evaporation.

Lead.—*Wood's Test.*—For four or five days before securing the specimen, the patient takes 5 to 10 grains of potassium iodid three times a day. A liter of urine is then obtained, evaporated to dryness, and fused in a crucible with a little pure potassium nitrate until it becomes white. When cool, the residue is extracted with hot dilute hydrochloric acid, filtered, the filtrate rendered alkaline with ammonia to precipitate the phosphates and iron. Ammonium sulphid is now added to precipitate the lead, the precipitate is washed three times with hot distilled water and decanted, water acidified with hydrochloric acid is added, and the whole is allowed to stand until the next day. It is then filtered through a small filter, the precipitate is washed, and a little nitric acid is added drop by drop to dissolve the lead and carry it through as a nitrate. The filtrate is collected in a watch-glass and evaporated to dryness, and the final test is made by adding a drop of water and a crystal of potassium iodid. The formation of a yellow precipitate denotes the presence of lead.

Mercury.—To a liter of urine add 10 c.c. of hydrochloric acid, introduce a little piece of copper foil, and apply heat. After letting the urine stand for twenty-four hours, remove the copper foil, wash it with water, alcohol, and ether, and let it dry. Then introduce it into a long test-tube and heat it to redness. If mercury be present, it condenses on the cool part of the tube. If fumes of iodin be now introduced, the mercury is changed into mercuric iodid, having a red color.

Bromin and Iodin.—Add to the urine a little fuming nitric acid or some freshly prepared chlorin-water, and shake with chloroform. If bromin be present, the chloroform assumes a brownish yellow color. If iodin be present, a carmine or purple color is produced. If the urine be ammoniacal, potassium-hydroxid solution should be added to the urine before making the test (Gillett).

Quinin.—To about 10 c.c. of urine in a test-tube add a drop or two of HCl, then 2 drops of chlorin-water and an excess of ammonia. An emerald-green color is produced, which corresponds to the quantity of quinin present. Carefully neutralize the mixture, and the color turns to blue; add an excess of an acid and it becomes purple or red; again add an excess of ammonia, and the green color is restored.

Acetanilid.—Evaporate the urine to about half its volume, add HCl and boil for a few minutes; extract with ether, evaporate the ether, treat the residue with distilled water, add a few c.c. of an aqueous solution of phenol and half as much of a 1 per cent solution of calcium hypochlorite. A pale green (onion-peel) color is produced, which changes to blue on the addition of ammonia. If the urine is pale, the extraction with ether may be omitted.

Antipyrin.—The addition of ferric chlorid to urine containing antipyrin produces a red color.

If antipyrin be also present in the test for quinin, the urine acquires a red color on the first addition of ammonia.

Morphin.—Add to the urine a little chloroform containing one or two drops of iodic acid. As the chloroform sinks to the bottom, it takes up iodin and acquires a pink color corresponding in depth to the quantity

of morphin present. Now render the mixture alkaline with ammonia; the pink color is discharged from the chloroform, and the supernatant fluid becomes deep brown.

Salicylic Acid.—To 10 c.c. of urine add 1 c.c. of strong ferric-chlorid solution. Salicylic acid (salicyluric acid) produces a violet color. Diabetic urine may give the same reaction without the presence of this substance.

Santonin.—The bright yellow urine becomes red on the addition of an alkali, and the color gradually fades.

Rhubarb and senna give the same color-change on the addition of an alkali to the urine, but the color is permanent. Add baryta-water to the fluid, and filter; if the color pass through with the filtrate, it is due to santonin; if it remain with the precipitate, it is due to rhubarb or senna.

Pine Acids.—The acids and salts of pine appear in the urine after the ingestion of balsams, cubebs, and sometimes after turpentine has been taken. The addition of strong nitric or hydrochloric acid produces a precipitate like that of albumin, but it is dissolved by strong alcohol. Urine containing turpentine often has the odor of violets.

URINARY SEDIMENTS.

To obtain the sediment from a specimen, the fluid should stand about seven hours in a conical glass. The supernatant portion may then be decanted slowly; the last cubic centimeter will usually contain a representative quantity of the sediment. A much quicker and better method is by means of the centrifuge, since time is not thus allowed for the destruction of the anatomical elements by bacteria.

FIG. 35.—Uric-acid crystals. *A*, Crystallization on a cotton fiber.

Chemical Sediments.—*Uric-acid* crystals are found almost exclusively in acid urine, and they constitute the only ingredients of acid urine which have a yellow color. They frequently crystallize upon the side of the vessel or upon foreign bodies. They are for the most part rhomboidal, but may be rectangular, or having rounded ends may appear ovoidal or circular. They are usually flattened, but may be cubical and often form stars or clusters (Fig. 35).

Urates.—The urates of sodium and potassium may be found in acid urine, rarely also that of calcium. Ammonium urate is nearly always

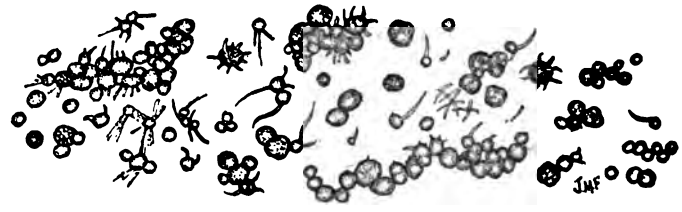

FIG. 36.—Crystals of ammonium urate.

found in alkaline urine. Sodium urate appears as an amorphous, "brick-dust" deposit, and is very insoluble, sometimes in the form of fan-shaped

or stellate clusters or needles. Potassium acid-urate appears only in amorphous form, more soluble than the sodium salt. Calcium urate occurs in acid urine as an amorphous white or gray deposit. Ammonium urate occurs in the form of dark brown crystalline spheres studded with fine spiculæ, which are known as mulberry crystals (Fig. 36).

FIG. 37.—Calcium-oxalate crystals.

Calcium Oxalate.—These crystals occur in either acid or alkaline urine, as small, highly refracting octahedra, "envelope" crystals, or as circular or ovoid disks with central depressions, "dumbbell" crystals (Fig. 37).

Phosphates.—Only the alkaline phosphates are found in urinary sediment. They occur as triple ammonium-magnesium phosphates or as calcium phosphates. The most frequent appearance is that of triangular

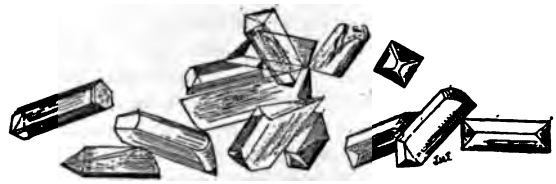

FIG. 38.—Crystals of ammonium-magnesium phosphate (from a camera-lucida sketch). The crystal at the extreme left is probably calcium phosphate.

prisms having beveled ends, the "coffin-lid" crystals (Fig. 38), rarely that of rosettes or star-shaped bunches of feathery crystals resembling fern leaves. They are found only in alkaline urine.

Leucin and Tyrosin.—Leucin occurs in yellowish, highly refracting spheres, resembling fat-globules, but in a pure state it crystallizes in irregular scales or rosettes having a greasy feel. The spherules are insoluble in ether.

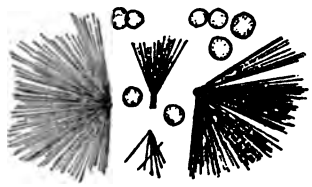

FIG. 39.—Leucin spherules and tyrosin crystals.

Tyrosin crystallizes in the form of fine needles arranged in sheafy bundles; sometimes, in alkaline urine, in the form of rosettes (Fig. 39). It is readily soluble in hot water, acids and alkalis, insoluble in alcohol or ether.

Melanin.—The urine may be dark colored when voided, or becomes so after exposure to the air, or upon the addition of sulphuric or hydrochloric acid or ferric chlorid. The addition of bromin-water produces a yellow precipitate which turns to black. (See also Alkapton test, p. 731.)

The microscope reveals small granules insoluble in cold alcohol, ether, acetic acid, or dilute mineral acids, but soluble in strong solutions of ammonium, sodium, or potassium hydroxid, and in boiling acetic, lactic, and mineral acids.

Fat appears in the urine as small, highly refracting granules with dark margins, which are soluble in ether, chloroform, benzol, carbon disulphid, and hot alcohol.

ANATOMICAL SEDIMENTS.

Epithelium is almost invariably found in the sediment, whether the urine be normal or pathological. Each division of the urinary tract has its typical surface epithelium, but of no cell can it be said that it

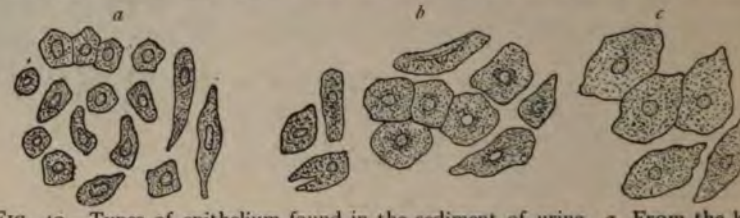

FIG. 40.—Types of epithelium found in the sediment of urine. *a*, From the kidney and ureter; *b*, from the bladder; *c*, from the vagina.

originates in one division alone, especially in pathological urine, for immature and transitional forms from the deeper layers often appear. The three principal types of cells are shown in Fig. 40.

Pus.—Purulent urine is often cloudy or milky. Under the microscope pus-cells appear as pale, finely granular spherical cells about the size of leucocytes, containing from one to three nuclei. Water and acetic acid cause them to swell and become more delicate in outline, the acid at the same time causing the granular matter to disappear and rendering the nuclei more distinct. The pus-cells in urine are dead and show no ameboid movement. In alkaline urine they usually fuse into a glairy mass at the bottom of the specimen, and cannot be recognized with the microscope.

FIG. 41.—Blood-cells and blood-casts.

Blood.—When abundant, blood gives to the urine a color varying with its quantity from a dark red or smoky hue in acid urine to a bright red in alkaline urine, or there may be a reddish brown granular sediment. Coagula may be found. Albumin is always to be detected. The corpuscles are usually scattered singly over the field of the microscope, rarely forming rouleaux. When the blood originates in the kidney, casts are usually present (Fig. 41). Gum-

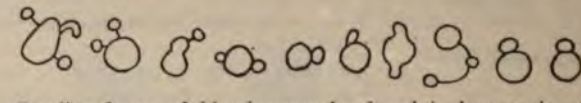

FIG. 42.—Peculiar forms of blood-corpuscles found in hematuria of renal origin. (After Gumprecht.)

precht has shown that in renal hematuria the red cells are fewer in number than in that of vesical origin; the corpuscles usually undergo fragmentation and often assume peculiar forms (Fig. 42).

URINARY CASTS.

The urine should be examined as fresh as possible, and for this reason centrifugal precipitation is to be preferred. It is better to place a

piece of hair under the cover-glass in order to avoid forcing the casts out from under it. A moderately low power should first be used in order to determine the presence of casts, then a high power (⅛) to determine their character. The light should not be too strong. Oblique illumination is often better, especially for the hyalin-cast.

FIG. 43.—Renal casts. *a*, Hyalin; *b*, granular; *c*, epithelial.

The principal forms of casts are: hyalin, granular, epithelial, waxy, fatty, and blood casts. These are shown in Figs. 43 and 44. Amorphous urates and foreign matter sometimes assume the form of false casts (Fig. 44, *c*).

Spermatozoa are little threadlike bodies about 1-600 inch in length and have flattened, oval heads. Under favorable conditions of heat and

FIG. 44.—Renal casts. *a*, Waxy; *b*, fatty; *c*, amorphous urate and other false casts.

moisture they exhibit a vermicular motion, but are usually motionless when found in the urine.

Fragments of tumors are sometimes found in the urine, but they are seldom of sufficient size to permit of proper hardening and cutting. A diagnosis should not, as a rule, be based upon their appearance under the microscope.

ANIMAL PARASITES.

Distoma Hematobium.—The ova of this parasite are oval, about 1-200 inch long, with a sharp, projecting anterior extremity and containing a distinctly visible embryo. They are usually accompanied with blood, and sometimes with fat.

Filaria Sanguinis Hominis.—This parasite is usually found in chylous urine. It is about as wide as a red blood-corpuscle and about fifty times as long. It has a short, rounded head and a long, pointed tail. The body is granular and has transverse striations.

Echinococcus.—The hooklets and scolices of the echinococcus rarely find their way into the urine. They may appear, however, entire or in fragments, and may be accompanied by pieces of the chitinous membrane, usually with blood, pus, and cellular débris.

Other parasites rarely found in the urine are the trichomonas, oxyuris vermicularis, and the strongylus gigas. A peculiar ameba and an infusorium have also been described.

VEGETABLE-PARASITES.

Nearly forty varieties of bacteria have been recognized in the urine These belong to the two classes of pathogenic and nonpathogenic or-

FIG. 45.—Yeast plant.

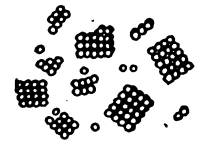

FIG. 46.—Sarcinæ of urine.

FIG. 47.—The micrococcus ureæ.

ganisms. Molds seldom form, except when sugar is present. The yeast plant (Fig. 45) is occasionally found, in single cells or in chains. Sarcinæ (Fig. 46) have been found in acid urine. They are sometimes larger than those found in the stomach. The micrococcus ureæ is a rather large micrococcus, growing in chains in alkaline urine. All these nonpathogenic bacteria are found, for the most part, in connection with retention due to stricture, cystitis, enlarged prostate, paralysis, etc.

Pathogenic Bacteria.—The pathogenic bacteria may be described under the heads of micrococci and bacilli.

The micrococci belong chiefly to the pus-formers and include the streptococci ureæ, pyogenes, and rugosus; the staphylococci pyogenes albus, aureus, and citreus; several diplococci and many others. The gonococcus belongs to this class.

The most important bacilli are the coli communis, tuberculosis, and typhosus.

The micrococci, as a rule, require no special methods of staining, all that is necessary being to place a drop of the sediment on a slide, add

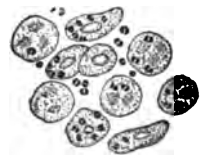

FIG. 48.—Gonococci.

FIG. 49.—Tubercle bacilli.

a drop of any anilin stain, remove the excess of coloring matter after a few minutes, and examine with a high power.

The *gonococcus* is hemispherical, and so arranged in pairs that the inner, flat or slightly concave side of each is separated from that of its

fellow by a narrow interval (Fig. 48). They are sometimes grouped as tetrads, and are generally found in pus-cells or attached to epithelium. Anilin blue or violet is the best stain.

The *Bacillus tuberculosis* (Fig. 49) is detected in the urine with much difficulty, as a rule, owing to the uncertainty of securing the specimen from so great a quantity of fluid, and still greater difficulty of fixing it. The method of staining is the same as that employed in examination of sputum.

Bacillus Typhosus.—This organism may be stained by Ziehl's method, acetic acid being used to decolorize instead of sulphuric or nitric acid. Better results are obtained by staining for twenty-four hours in Löffler's alkaline methylene-blue solution.

The *Bacillus coli communis* is differentiated from the B. typhosus with much difficulty, and chiefly by its behavior upon different culture-media, which the student will find fully described in his textbook on bacteriology.

CRYOSCOPY.

The apparatus necessary for the determination of the freezing-point of urine consists of a double test-tube, a stirrer, a freezing-bath, and a thermometer capable of registering the hundredths, or, better, the thousandths, of a degree centigrade. A lens is necessary to recognize these minute variations. The test-tube may be made by placing a flat-bottomed test-tube one inch in diameter and seven inches long within a slightly larger tube, preventing contact by placing rubber bands around the inner tube. The stirrer is made by bending a loop five-eighths of an inch in diameter at a right angle on the end of a stiff wire of suitable length, to serve as a handle. To the outer edge of the loop is then fastened with fine wire a strip cut from the side of a goose-feather. This must fit into the inner tube with sufficient accuracy to prevent the clinging of ice crystals to its sides. The thermometer is placed within the inner tube.

An approximate test of the freezing-point is made by immersing the tube in a freezing-mixture. A mixture of shaved ice and water is then prepared in a felt-covered vessel, and enough salt is added to reduce the temperature of the slush to that of the approximated freezing-point of the urine. The test-tube is filled one-third full of the urine and placed in the freezing-mixture until the temperature falls to 0.3° or 0.4° C. below that of the freezing-point just determined. The tube is then transferred to the mixture of ice and water, and a minute crystal of ice is dropped into the urine in order to start the crystallization. The temperature rises slightly as the urine congeals, and in about one minute it may be read from the thermometer. In order to secure the slightest variation it is necessary to tap the top of the thermometer with rapid but delicate blows. The stirrer must be kept in motion during the entire process.

BACTERIOLOGICAL METHODS.

Success in bacteriological work can be attained only at the expense of considerable time and with precise methods. The more elaborate investigations can be made only in a fully equipped laboratory, but

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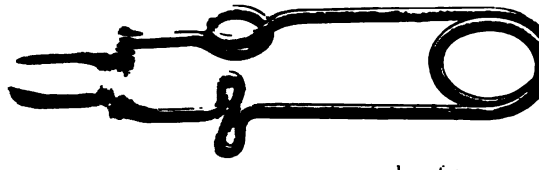

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erally employed. For convenience, a saturated alcoholic solution should be kept in stock. This is made by placing in the bottle a little more of the dye than will dissolve in enough alcohol to fill the bottle. The quantity of the dye is usually about one-fourth of the capacity of the bottle. The alcohol is then added, and the bottle is well shaken and set aside for 24 hours. A few crystals should remain undissolved. For use, enough of the stock solution is added to a small bottle about two-thirds full of distilled water to make a solution which is barely transparent. The proportion is usually about 5 c.c. of the saturated solution to 95 c.c. of water.

Some varieties of bacteria are slow to take up the stains, and for these special solutions must be employed. In some instances the staining is so strongly retained that the resistance to the action of bleaching-fluids is a distinguishing feature of the organism, as is notably the case with the Bacillus tuberculosis.

Löffler's Alkaline Methylene-Blue Solution.—The formula is:

Saturated alcoholic solution of methylene blue	30.0
Solution of potassium hydroxid in water (1 :10000)	100.0

This solution is especially applicable to the staining of the diphtheria bacillus.

Koch-Ehrlich Anilin Water-Fuchsin Solution:

Anilin-water	50.0
Saturated alcoholic solution of fuchsin	5.0

The anilin-water is made by adding about 5 c.c. of anilin oil to 100 c.c. of distilled water, a few drops at a time, and shaking well after each addition; then, after allowing the mixture to stand for several hours, filtering it through a moistened filter-paper. The specimen must remain several hours in this solution. Anilin solutions of gentian-violet or anilin blue may be made in the same manner.

Kuhne's Methylene Blue:

Methylene blue	1.5
Absolute alcohol	10.0
Carbolic-acid solution (1 :20)	100.0

This stain requires about five minutes for films.

Carbol-Thionin Blue.—Thionin blue, 1 gm.; carbolic-acid solution (1 :40), 100 c.c. For use, dilute with three times as much water, and stain from three to five minutes.

Ziehl's Carbol-Fuchsin Solution:

Fuchsin	0.5 gm.
Absolute alcohol	5.0 c.c.
Carbolic-acid solution (1 :20)	50.0 c.c.

This solution may be made also from the stock solution of fuchsin by slowly adding it to the 5 per cent carbolic-acid solution until an opalescent hue is produced or until the surface acquires a metallic luster.

Gram's Iodin Solution:

Iodin	0.10
Potassium iodid	0.20
Distilled water	30.00

This solution is employed as a bleach after staining with a gentian

violet, especially for the pneumococcus, and may be followed with a counter-stain of carmin or Bismarck brown. After staining with the gentian-violet solution, the specimen should be immersed for a few moments in the Gram solution, then washed in alcohol. If the cover-glass still retain a trace of violet color, it must be immersed in the iodin solution until thoroughly bleached. The method is valuable also for staining the capsules of certain bacteria.

Welch's Capsule Stain.—Another method of staining the capsules of bacteria thus enveloped is to cover the film with glacial acetic acid for a few seconds, then drain it off and replace it with gentian violet-anilin solution, repeating the application several times. Then wash in a 2 per cent sodium-chlorid solution, and mount in the same fluid.

Gabbet's Blue.—This is a solution of 2 grams of methylene blue in a 25 per cent solution of sulphuric acid. It is employed chiefly as a combined bleach and counter-stain. It should be allowed to stand for several hours to become perfectly cooled before it is used. Specimens intended for permanent mounts must be thoroughly washed after being immersed in it. In case the counter-stain is not sufficiently deep, the specimen may be immersed in the ordinary aqueous solution of methylene blue.

Staining the Spores.—*Abbot's Method.*—Apply one of the anilin stains to the film in the usual manner, warm until steam rises over a Bunsen flame, and keep the specimen at about this temperature for a minute or two, then wash and immerse in a 2 per cent alcoholic solution of nitric acid until the stain becomes invisible. Now immerse for ten seconds in an eosin solution composed of saturated solution of eosin 10 parts, distilled water 90 parts. The spores are stained blue, and the bodies of the bacilli rose-red.

Moeller's method is a little more penetrating than the preceding, and it is therefore more successful with some bacteria. The film should be immersed for two minutes in chloroform before the stain is applied, then washed with water and placed for from a half-minute to three minutes in a 5 per cent solution of chromic acid. It is then washed in water and restained with Ziehl's carbol-fuchsin solution, warmed over the Bunsen flame for several minutes. The specimen is again washed and decolorized with a 3 per cent solution of hydrochloric acid or 5 per cent sulphuric acid, and finally stained for one minute in methylene blue. The spores are stained red, and the bacilli blue.

Staining the Flagella.—A thin film of a fresh 18-hour culture of the motile organism to be examined is placed upon a perfectly clean cover-glass and allowed to dry in the air. It is then passed three times through the flame and immersed in a mordant solution of ferric alum and fuchsin. This solution is made by adding to 3 parts of a saturated solution of alum 1 part of diluted liquor ferri sesquichlorid (1:20 of distilled water). For use, add to 10 c.c. of this solution 1 c.c. of a strong aqueous solution of fuchsin. The mordant must be let stand, however, for several days, and filtered before it is used. The film is immersed in it for five minutes, then slightly warmed and washed. It is then dried, stained faintly with carbol-fuchsin, washed, dried, and mounted. It is sometimes necessary to repeat the process several times before a good result is obtained.

Culture-Media.—For clinical work, the culture-media should, as a rule, be purchased ready for use. The time consumed and the difficulties of preparing them are rarely justifiable unless some special line of investigation is to be undertaken. The most generally useful media are (1) nutrient bouillon, (2) bouillon to which has been added one-third of its volume of ascitic fluid, (3) slanting nutrient agar, and (4) solidified blood-serum. Of these, the last meets the greatest number of requirements.

Bouillon.—This may be prepared directly from meat, or with less difficulty from the beef extract. For the meat bouillon, take 500 grams of lean beef or mutton, freed from fat, tendon, and fiber, chop it fine and soak it in one liter of water for 24 hours. During this time it must be kept in a refrigerator. Then squeeze out through muslin all the juice. In this extract dissolve 10 grams of pure pepton and 5 grams of pure sodium chlorid, then boil in a porcelain-lined pan for 30 minutes, or until all the albumin has been coagulated. During the boiling, a few drops of a strong solution of sodium hydroxid should be added to render the fluid very faintly alkaline. The water lost through evaporation must be replaced so that the quantity at the end of the process shall be one liter. After the boiling, the fluid is filtered through paper into a flask; this is closed with a cotton plug and placed in the sterilizer. If the Arnold sterilizer be used, and it answers the purpose well, the sterilization must be done by the discontinuous method; the material must be heated to boiling (100° C.) for from 15 to 30 minutes on three successive days, or for a greater number of days if the sterilization is found to have been incomplete. If an autoclave be available, one sterilization under an additional pressure of one atmosphere (15 pounds to the square inch) is sufficient.

Beef-Extract Bouillon.—The formula for this is: Beef extract (Liebig's or Armour's) 3 grams, pepton 10 grams, sodium chlorid 5 grams, water enough to make 1 liter, and sufficient sodium-hydroxid solution to render the bouillon neutral or faintly alkaline. The processes of boiling, filtration, and sterilization are the same as those given under the preceding caption.

Glucose, lactose, sucrose, and other saccharine bouillons are made by adding to either of the foregoing bouillon preparations 1 or 2 per cent of the sugar designated. Litmus bouillon is made by adding a small quantity of litmus to the bouillon.

Nutrient Gelatin.—This is made by adding to bouillon 10 per cent of the best sheet gelatin, then boiling, neutralizing, and filtering. Or the gelatin may be added to the ingredients of the bouillon in its original preparation, 100 grams being required for the liter. It is better, however, to add the gelatin after the beef extract has boiled for about 15 minutes. As soon as the gelatin has become dissolved, the mixture is boiled vigorously for 10 or 15 minutes. Prolonged heating of the gelatin reduces the temperature at which it congeals. An attempt may now be made to filter the medium through two or three thicknesses of moistened filter-paper, pouring it down a glass rod. If the filtrate is not perfectly clear, the liquid must be cooled to below 60° C. and mixed with the albumen of one or two eggs well beaten in 50 or 100 c.c. of distilled water, then boiled for 10 minutes and filtered. The large coagula

may be removed with a strainer. The filter-paper should be folded into small plaits or placed on a wire frame, and the filtration is much facilitated by placing the filter and flask in the sterilizer or oven to prevent cooling and solidifying of the gelatin. After the medium has been thoroughly sterilized, it is poured into tubes, 5 to 10 c.c. in each; these are closed with cotton plugs and placed in the sterilizer for 15 minutes on three succeeding days.

Agar is made by adding, to the liter of bouillon prepared according to either of the given formulas, 15 grams of agar-agar cut into short pieces, boiling and filtering in the same manner as in the preparation of nutrient gelatin. The bouillon should, as a rule, be diluted with an equal quantity of water before the agar-agar is added, and the boiling continued until the quantity has been reduced to a liter, or until the agar has been fully dissolved. The scum should be removed as it forms on the surface. The fluid is then cooled, the white of egg added and again boiled, as in the last formula. It is finally filtered, sterilized, poured into tubes, and again sterilized. After the last sterilization the tubes should be laid in a reclining position, in order that the agar may solidify with the proper slant.

Blood-Serum.—These tubes may be made with either the blood of cattle, or with human blood when obtainable. The blood should be received in a tall glass jar and kept in a cool place for from 24 to 48 hours, in order to permit complete separation of the clot and serum to take place. The serum is then removed from the jar, with a pipette or by decanting, and poured directly into the culture-tubes. The tubes are then placed at a proper slant in the sterilizer and carefully raised to a temperature of 90° C. until the serum has become thoroughly coagulated. If raised to the boiling-point, the surface is rendered rough by the formation of bubbles. After the coagulation has been completed, the tubes can be raised to an upright position and the sterilization continued on succeeding days as with the other media.

Loffler's Blood-Serum.—This is prepared in the manner just described, except that one part of bouillon containing 1 per cent of glucose is added to 3 parts of beef blood-serum before the coagulation.

Potato.—After washing and paring, the potatoes are cut into pieces of proper size to be placed in the tubes, and with a beveled side to correspond to the slant given to the other media. The pieces are then passed into the tubes, a little space being left at the bottom for the accumulation of water. The tube is closed with cotton and sterilized in the ordinary way.

Milk is sometimes employed as a culture-medium, especially for the study of bacteria that coagulate casein or develop acids. For the latter purpose just enough litmus solution should be added to the milk to give it a pale blue tinge. The milk is then poured into tubes and sterilized by the three-day method.

Making Cultures.—Cultures from the living patient should be made at the bedside; for if the specimen be transported to any distance, many of the bacteria will die, and there is greater danger of contamination of the specimen. Cultures from the dead body should be made at the earliest possible moment after death, and directly from the body to the culture-medium. For inoculating the bouillon-culture the platinum

loop is most suitable, but solid media may be inoculated with either the loop or a sterilized cotton swab. Only heat is to be used to sterilize the loop or swab; chemicals must not be employed. The bacteria are transferred by lightly touching the wire, after it has been passed through the flame, to the culture or other source from which they are to be obtained, and rubbing this gently over the culture-medium. For the isolation of different species, the plate-culture affords the most satisfactory results, since the growth of different colonies can be more readily recognized and more easily transferred to tubes for the purpose of further study. A pure culture can seldom be obtained directly from tube-cultures. After a pure culture has been obtained, however, further propagation may be continued indefinitely by means of the tubes.

Plate-Cultures.—These are made by inoculating nutrient media spread upon glass plates or in shallow dishes (Petri dishes). Two methods are employed. In the first the matter containing bacteria is streaked over the surface after the medium has hardened; in the other, the material is mingled with the nutrient substance while it is still warm and in a fluid state, but cool enough to exert no harmful influence upon the organisms introduced into it. Both agar and gelatin plates are made use of. Nutrient 1 per cent agar begins to solidify at about 36° C., while the 10 per cent gelatin solidifies at a temperature of 23° C. The former is therefore used for cultures to be kept at the temperature of the body, and the latter for those requiring room temperature. In inoculating agar plates by the method of mingling the substance containing the bacteria with the medium, care must be taken in the first place not to add the contaminated matter to the agar while it is too hot, and on the other hand not to permit the culture-medium to become partially solidified before the mixing has been accomplished. The bacteria should be added, as a rule, when the medium is at about 40° C. Every precaution against contamination must be exercised. The thermometer must be sterile, and the Petri dishes must be opened only long enough to permit the pouring in of the culture. As the fluid solidifies, the organisms are sealed within it, and after a proper time each will develop a colony if the conditions for its growth have been right. The number of colonies, as well as the different varieties of bacteria, should be carefully determined.

In making primary cultures for the purpose of isolating a microorganism whose culture peculiarities are not known, several plates or tubes and several different media should be inoculated, using different quantities of the material in the different plates. On the solid media it is well to make both the stroke and the stab culture.

The *stroke culture* is made thus: Sterilize a straight platinum needle in the flame and touch it to the substance, pus, organ or tissue, to be investigated; then, after removing the cotton plug from a fresh culture-tube held on a slant, draw the needle in a straight line over the surface of the medium, and immediately replace the cotton.

The *stab culture* is made in the same manner, except that the needle is thrust down through the center of the medium nearly to the bottom of the tube. Media for this purpose are solidified in an upright position.

The principal features to be observed in the stroke culture are the extent and form of the growth, the shape of the margins, its color,

and whether it is flat or raised, clear or opaque. In the stab culture the principal features to be noted are the extent of the growth along the entire line of inoculation or only at the surface, its abundance, its color, and whether the medium has been altered in color, odor, or consistence. If liquefaction occurs in gelatin cultures, one should observe whether this takes place at the surface or along the line, and the form of the area that has undergone liquefaction.

EXAMINATION OF SPUTUM.

Microscopic examination of the sputum is of value not merely for the discovery of bacteria, but for the recognition of various other elements that may be present in it. Fragments of tissue are found, either as separate filaments or in a more or less complete alveolar arrangement, when there are disintegrating lesions of the lungs or bronchi. Very rarely the cellular structure of neoplasms can be recognized. Curschmann's spirals and the Charcot-Leyden crystals are often found in connection with asthma. Hematoidin plates and crystals are occasionally found after hemorrhagic affections. Cholesterin, leucin, tyrosin, calcium carbonate and oxalate, the triple phosphate, and other crystals have been occasionally discovered.

The Tubercle Bacillus.—The most frequent and probably the most important object of sputum examination is to determine the presence or absence of the Bacillus tuberculosis. For this purpose, a small particle of sputum is spread upon a cover-glass or slide in the usual manner, passed three times through the Bunsen flame, and stained. In selecting the specimen, a small, white, cheesy mass should be picked up with the loop, or if such particles are not found, an otherwise representative portion of the sputum should be selected. There are three principal methods of staining. The carbol-fuchsin method is probably more generally used than any other. The formula for this solution has been given on page 743. After the film has been made and fixed by heat, it is covered with the solution and held over the Bunsen flame for two or three minutes at such a distance that vapor rises without ebullition. The coloring fluid is then washed off with water; next the specimen is decolorized in a 1 per cent solution of hydrochloric acid in 70 per cent alcohol, or in 2 per cent nitric acid-alcoholic solution. The decolorization is stopped by quickly washing the specimen in water immediately upon the disappearance of all perceptible color. If it be thought necessary to exclude the smegma bacillus, the cover-glass is now immersed for a minute in pure alcohol. The film may then be given a counter-stain with the simple solution of methylene blue, applied cold, after which it is ready for the final washing with water, drying, and mounting. By this method the bacilli are stained a brilliant red, other bacteria and cellular elements blue.

Gabbet's Method.—This method yields excellent results and is preferred for routine work on account of the greater simplicity of the manipulations. The sputum is spread, fixed, stained with the carbol-fuchsin solution, and washed in water, just as in the preceding method. It is then decolorized and counter-stained by means of Gabbet's combined solution (see p. 744), allowing the solution to act from 15 seconds to a minute,

or until all trace of the red stain has vanished and the film has acquired a pale blue tinge. The specimen is then washed and mounted in the usual manner. The result is the same as that of the preceding method. This method has the advantage that, after a few trials, a uniform regulation of time can be adopted with a greater certainty of good results. When the counter-stain does not act so deeply as is desirable, the specimen can be treated for a few moments with the akaline methylene-blue solution.

Koch-Ehrlich Method.—The only essentially different feature in this method is the use of the anilin water-fuchsin solution. The cover-glass is floated, film downward, on the surface of a small quantity of the solution in a watch-glass held over the flame for about two minutes, the solution being raised almost to the point of boiling. The specimen is then decolorized in 3 per cent hydrochloric acid in 70 per cent alcohol, washed, dried, and mounted. Better results are sometimes obtained by allowing the stain to act for several hours, after having been warmed.

Staining the Bacillus in Sections.—The sections should be as thin as the tissues will permit. They may be stained in the same solutions as are employed for the films, but at a lower temperature.

Ehrlich Method.—The sections are placed in anilin-fuchsin solution and allowed to stand for from two to twelve hours, then decolorized for about 30 seconds in a 10 per cent solution of nitric acid and washed in 60 per cent alcohol until the free coloring matter has been removed. They are then counter-stained for two or three minutes in a saturated aqueous solution of methylene blue, washed in water, dehydrated with absolute alcohol, cleared in xylol or cedar oil, and mounted in xylol balsam.

Carbol-Fuchsin Method.—The sections are stained in the carbol-fuchsin solution warmed to not more than 50° C. and allowed to stand in the fluid for an hour. They are then decolorized in 5 per cent sulphuric acid, washed in 70 per cent alcohol, counter-stained as in the Ehrlich method, washed, dehydrated, clarified, dried, and mounted.

Examination of Water and Other Fluids.—The number of bacilli in water, milk, and other liquids can be approximately determined by means of plate-cultures. A definite quantity of a representative portion of the fluid to be tested, either in its purity or diluted with sterilized distilled water in case it is highly contaminated, is added to a liquefied culture-medium, poured into the Petri dish, allowed to harden and kept at the proper temperature until growth can be recognized. The number of colonies is then counted with the aid of a lens. Each colony represents an individual micro-organism, and the number of colonies, therefore, corresponds to the number of bacteria in the original quantity of fluid. Tufts of bacteria may, of course, be counted as single germs by this method, but the result is sufficiently accurate for practical purposes.

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