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PRACTICE OF MEDICINE

A MANUAL FOR STUDENTS AND PRACTITIONERS

BY

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PREFACE TO THIRD EDITION

To render this Epitome of Practice suitable for hasty reference by physicians, as well as for the use of students, it was deemed advisable in its original preparation to avoid undue curtailment of the most important subjects, such as typhoid fever, tuberculosis, and pneumonia. For this reason the necessary restrictions as to space were met by the exclusion of diseases of the pharynx, larynx, and tonsils, subjects which are treated fully in the companion volume on *Diseases of the Nose and Throat.*

Under the headings of Diagnosis have been mentioned a few of the most important differential points of each disease. To Dr. D. T. Mitchell Prudden the author is indebted for assistance in the preparation of the introductory remarks on immunity.

In the present revision the same principles have been followed as in the former editions. While the entire volume has been thoroughly revised, the principal changes have been necessitated by advances in the knowledge of infectious diseases and diagnostic methods. Partial reclassification of diseases and exclusion of obsolete material has permitted the inclusion of Acute Poliomyelitis.

H. D.

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PRACTICE OF MEDICINE

SECTION I

INFECTIOUS DISEASES

INCLUDING under the term microörganisms the bacteria, yeasts, and moulds, and excluding animal parasites, an **infectious disease** may be defined as one incited by the entrance into and proliferation in the body of pathogenic microörganisms.

Infection is the process by which such diseases are incited.

The inciting organisms may enter the body through the respiratory or the alimentary tract, the skin or mucous membranes, and the sebaceous or sweat glands, or by inoculation into wounds or abrasions. Infection may also be *congenital*, through the placental circulation. When bodily resistance is weakened by the presence of one infectious disease, a so-called *mixed* or *concurrent infection* with microorganisms of another variety frequently occurs. In chronic diseases of the heart, lungs, liver, kidneys, etc., infection often occurs with a fatal result, and this is described as a *terminal infection*. Some diseases are usually communicable only by direct inoculation, for example, syphilis, rabies, and tetanus; the infectious agent of others, such as scarlatina and smallpox, appears to be transmitted directly through the air. Infectious agents of diseases which were formerly supposed to be transmitted through the air are being proved

one by one to have special modes of entrance, such as the malarial organism, which is conveyed by its intermediate host, the mosquito, and typhoid fever, the bacilli of which enter the alimentary canal in excreta of other patients, usually contained in food and drink.

The factors influencing susceptibility to infection include age, sex, impairment of the power of resistance by unhygienic surroundings, intemperance, malnutrition, or fatigue, or by existing infection of another variety, as in the case of tuberculosis following measles or bronchopneumonia; absolute or relative immunity acquired by a previous attack of the disease; and the virulence of the infecting agent and the mode of its introduction.

"**Immunity** is insusceptibility, or capacity for resistance on the part of the body to infection or its effects." It may be natural or acquired.

Natural immunity is a characteristic of certain species, races, and individuals. Thus many of the infectious diseases of man are not known to affect other animals, and *vice versa*. Racial immunity is relative rather than absolute—*e. g.*, the negro is rarely attacked by yellow fever. Age is an important factor in natural immunity, some diseases being confined almost exclusively to childhood or to other periods of life. Certain individuals may fail to contract an infectious disease though never having suffered from it and being frequently exposed.

Acquired immunity results either from (1) an attack of the disease in question, or from (2) some artificial process. As examples of the first may be cited the exanthemata; of the second, various forms of preventive inoculation.

Immunization, either natural or artificial, is a process of adaptation of the body to an unusual environment, and is not an entity any more than is disease under the modern conception. Recent studies have indicated that artificial acquisition of immunity is dependent upon the capacity of the body to adapt itself to the presence of certain foreign

materials introduced into it. This is accomplished by the production of new substances or of unusual amounts of substances natural to the body. Those concerning us most closely are the substances formed by the body under the influence of infectious agents. Such as neutralize the toxic products of microörganisms are known as antitoxins; those which are inimical to the life of microörganisms are called bactericidal or bacteriolytic substances. It is necessary to distinguish sharply between antitoxic immunity and bactericidal immunity. Another class of substances formed in response to the introduction into the body of foreign materials are those possessing the power of causing agglutination. (See Widal Reaction, p. 26.)

According to the mode of its appearance, an infectious disease may be: (1) *Sporadic*, occurring in isolated and scattered cases. (2) *Endemic*, appearing in a number of cases, but confined to certain localities. (3) *Epidemic*, affecting a large number of persons simultaneously and spreading rapidly to other localities.

The results of infection are shown by the occurrence of local or constitutional symptoms, or both. The constitutional symptoms usually include those of a sthenic or asthenic fever, which are given below for the sake of brevity, as they will be referred to under the individual diseases as one or the other of these types of fever.

The average normal temperature of the body taken under the tongue is 98.6°, though variations of 0.5° or more above or below this figure occur in health. The rectal temperature is about 1° higher. The pulse rate rises at an approximately constant rate of ten to the minute for each degree of temperature above the normal. The liability to error is less in the rectal than the mouth temperature, so the former is employed where accuracy is desired, as well as in infants, insane, delirious, violent, feeble, or unconscious patients, or when tenderness of the mouth requires. Taking the temperature by rectum is often prevented by the presence of painful local

lesions. When both mouth and rectal temperatures are difficult to obtain, the thermometer may be placed in an axilla or inguinal fold, taking care that it is well surrounded by tissues and not hastily removed.

Symptoms of a Sthenic Fever.—Skin hot, dry, flushed; mouth dry; tongue coated, moist or dry; thirst; at times vomiting; pulse increased in force and frequency, full; respiration increased in depth and frequency; headache, restlessness, sleeplessness; internal as well as surface temperature raised; urine diminished, often high-colored, containing albumin. These may pass into those of the asthenic type.

Symptoms of an Asthenic Fever.—Skin clammy; mouth dry; tongue heavily coated, dry, sometimes fissured; pulse frequent, small, feeble, of low tension; respiration increased in frequency, shallow; apathy, general weakness, headache, delirium or stupor; temperature elevated; urine diminished, high-colored, containing albumin.

The **general treatment** of certain of the infectious diseases includes isolation of the patient, ventilation of the sick room, from which unnecessary articles have been removed, and thorough disinfection of the room and all articles which have been in contact with the patient. The following are practical methods of securing these results:

1. *Disinfection of Sick Room, Furniture, etc.*—Walls, ceiling, and woodwork may be cleaned by washing with 1 to 1000 bichloride or 5 per cent. carbolic acid solution, or by rubbing with bread if solutions would injure them. All dust must be removed. Plastered walls and ceilings may be whitewashed. After washing with antiseptic solutions, woodwork should be scrubbed with soap and thoroughly wiped. Fumigation alone is uncertain, but is useful as an adjuvant. It is done by sealing all doors, windows, etc., with strips of paper and filling the room for several hours with formaldehyde gas, made by burning wood alcohol in suitable formaldehyde generators. Fumes of sulphurous acid are said to be less germicidal. At least three pounds

of sulphur should be burned in a room for each 1000 cubic feet of space, placing it in a pan supported in another containing water, to guard against fire. After scrubbing or fumigation, the room and its contents should be freely aired for several days, admitting sunlight, if possible. All worthless articles and badly soiled bedding should be burned. Such pieces of clothing, etc., as will not be injured may be boiled or soaked in 1 to 1000 formaldehyde solution (℥j of a 12.5 per cent. solution in 1 gallon of water) or 5 per cent. phenol. Clothing or bedding, etc., may be disinfected in the steam sterilizer.

2. *Toilet and Medical Articles.*—Bed pans, urinals, rectal tubes, and other articles which will permit should be boiled. Thermometers are kept in 1 to 1000 bichloride.

3. *Excreta.*—Feces, vomitus, or urine should be mixed with an equal volume of one of the following solutions: Formaldehyde, 1 to 500 (12.5 per cent. formaldehyde solution, ℥ij; water, 1 gallon), for one hour; chloride of lime, ℥iv; water, 1 gallon, for one hour; or 5 per cent. phenol for several hours. Sputum may be disinfected by burning, boiling, or 1 to 500 bichloride.

4. *Hands, Body, etc.*—Special outer garments may be worn while in the sick room and removed and clothing aired after leaving. Hands of attendants should be washed in bichloride, 1 to 1000. After deaths from the exanthemata, diphtheria, yellow fever, cholera, bubonic plague, etc., the bodies should be wrapped in sheets wet with bichloride, 1 to 500, or phenol, 5 per cent. solution, and the coffin should be sealed at once and buried as soon as possible. In convalescence from infectious diseases accompanied by desquamation, the body should be oiled and frequently bathed until desquamation ceases.

TYPHOID FEVER

Definition.—Typhoid fever is an infectious disease, characterized by hyperplasia and ulceration of intestinal lymph

follicles, enlargement of mesenteric lymph nodes and spleen, with parenchymatous changes in other organs, and clinically by continuous fever, prostration, diarrhea or constipation, and a characteristic eruption.

Etiology.—Bacillus typhosus, introduced into the alimentary canal most often in contaminated drinking-water, in milk infected directly by the milker or by water used in washing cans; or in food to which the germs are carried from the excreta by flies, unclean hands, or water used in washing the food, occasionally through oysters infected during freshening. Bacillus carriers, themselves in good health, may harbor typhoid bacilli in the gall-bladder or intestines for many years and repeatedly spread the disease. The disease occurs most frequently between August and November, and most often in persons from fifteen to twenty-five years of age. Individual and family susceptibility vary. One attack usually protects from another.

Pathology.—The *Peyer's patches* and *solitary lymph nodules* of the intestine undergo hyperplasia, are reddish or grayish, and more or less elevated. The lymphoid cells are increased in number, and the endothelium of the trabeculæ and sinuses proliferated. Resolution by fatty and granular degeneration of the cells may occur, or necrosis and sloughing may result from obstruction of the bloodvessels by proliferation of their endothelium and from direct action of the bacterial toxin. These newly formed endothelial cells are extremely phagocytic. The ulceration may be superficial or extend through the intestinal wall to any depth. Cicatrization may then occur. If the ulcer perforates the intestine there is local or general peritonitis. Necrosis of bloodvessel walls may occur, causing hemorrhage of varying degree. In some cases the intestinal lesions are apparently absent or found only by microscopic examination. The *mesenteric lymph nodes* show lesions similar to those in the intestinal nodules. *Spleen* is enlarged and soft from congestion and hyperplasia. *Liver* shows parenchymatous degeneration and focal cell collec-

tions which are sometimes necrotic. Suppurative cholecystitis may occur. *Kidneys*, cloudy swelling with granular degeneration of cells of the convoluted tubules. The bacilli are found in the urine and feces. Inflammations of the respiratory tract are common, and suppurative lesions, most often in the bones, may occur in many regions.

Symptoms are very variable. Those of a typical case are the following:

The **period of incubation** is eight to fourteen days, sometimes twenty-three days. The prodromata are lassitude, inability to work, chilly feeling, headache, anorexia, diarrhea or constipation, with or without abdominal pain, which is occasionally localized in the right iliac fossa; in some cases epistaxis. The *invasion* is usually dated from the time at which the patient is obliged to go to bed.

First Week.—The temperature often rises each evening until it reaches about 103° or 104°, with symptoms of a sthenic fever (p. 20). The pulse is characteristically slow in proportion to the temperature, being about 100 to 110, full, of low tension, often dicrotic. The tongue is coated; there is constipation or slight diarrhea, the abdomen is slightly distended and tender. There may be some mental confusion at night. Bronchitis often present. From the seventh to the tenth day the spleen becomes enlarged, and the typhoid eruption usually appears.

Second Week.—Symptoms intensified; fever is constantly high and of the asthenic type (p. 20); pulse more frequent; headache replaced by apathy; intestinal symptoms increase, stools becoming like "pea soup" if diarrhea is present. Apathetic facies; tongue coated in center, red along edges and at tip, becoming dry. There may be hemorrhage or perforation.

Third Week.—Morning remissions, with gradual fall of temperature; emaciation and weakness are marked. Perforation or hemorrhage may occur. Unfavorable symptoms

include delirium, muscular tremor, pulmonary complications, cardiac weakness, and meteorism.

Fourth Week.—Temperature gradually falls to normal, symptoms disappearing.

Termination.—Death may occur at any time after the second week, from the disease or complications. Sudden death may occur, with no cause apparent during life or at autopsy. Convalescence is very gradual, appetite intense, with great increase of weight and hemoglobin.

Special Symptoms and Variation.—1. *Invasion* may be sudden and marked by a chill. Sometimes it is obscured by severe nervous symptoms like those of meningitis, pulmonary or gastro-intestinal symptoms, or those of acute nephritis. In so-called "walking typhoid" the patient may keep up well into the course of the disease. These cases are often severe. In rare cases perforation or hemorrhage is the first diagnostic point.

2. *Fever.*—Evening temperature about 1° higher each day during the first week, up to 103° or 104°; constant high temperature, 103° to 105°, with slight morning remissions, in the second week; in the third week it is lower each day, with marked morning remissions; in the fourth week it falls to normal by lysis. The chief variations are: Sudden onset of high temperature after a chill; defervescence after the second week; fever sometimes absent or low; rises due to complications; rises due to indiscretions in diet during convalescence, but of no great significance; those due to one or more relapses and following a course like the regular but shorter; persistent post-typhoidal fever often relieved only by liberal diet and getting the patient up.

3. *Pulse.*—Not characteristic, but in the early stages it is more often dicrotic than at a corresponding period of any other acute disease, and generally slower than is usual with the existing temperature. Later it becomes frequent, feeble, and small.

4. *Eruption* of slightly elevated, flat, rose-colored papules, disappearing on pressure, usually appears from the seventh to the tenth day, and later in successive crops, each lasting two or three days. It is found most often first on the abdomen, and may be also on the rest of the trunk and on the extremities.

5. *Digestive System.—Tongue* white-coated and moist at first. Gradual disappearance of the coating leaves it red at the tip and edges in the second week. Later it may be dry, brown, and fissured, and teeth and lips covered with brownish material called sordes, consisting of mucus, epithelium, bacteria, and food. *Diarrhea* is an uncertain symptom. *Constipation* appears to be more frequent. One or the other seems to predominate in epidemics. The characteristic diarrheal stools are of the consistency and appearance of pea soup, and occur most often at the end of the first and during the second week. *Tympanites* is frequent, but is an unfavorable sign only when excessive. Gurgling in the right iliac fossa upon pressure is not indicative of typhoid, and attempts to elicit it may injure the ulcerated bowel. *Hemorrhage* occurs most frequently between the end of the second and the beginning of the fourth week, while the intestinal sloughs are separating. It is marked by a sudden feeling of collapse and rapid fall of temperature, often of many degrees. Hemorrhages may be repeated, and, though of serious import, are not always fatal. *Perforation* of the intestine is usually marked by sudden, sharp, paroxysmal pain, generally localized in the hypogastrium to the right of the median line, and sometimes with signs of shock, followed by symptoms of peritonitis. Special attention should be paid to local physical signs, as the general symptoms may be slight or masked by those of the typhoid state at the time when surgical aid is most valuable. Increase of leukocytes in the blood at the time of perforation is an important aid in diagnosis.

Complications and **sequelæ** are numerous. The more common are: *Skin:* partial loss of hair, bed-sores. *Digestive*

system: parotitis, cholecystitis. *Circulatory system:* secondary anemia, myocarditis, thrombosis of veins of the lower extremities, especially the femoral, popliteal, and internal saphenous. *Respiratory system:* bronchitis, bronchopneumonia or lobar pneumonia, pleurisy. *Nervous system:* neuritis, including so-called "tender toes." *Urinary system:* retention of urine, albuminuria, occasionally nephritis. *Osseous system:* periostitis, necrosis of bones, arthritis. *Other complications* are inflammatory processes in various parts of the body due to the Bacillus typhosus—*e. g.*, mastitis, orchitis.

Diagnosis.—There is no symptom pathognomonic of typhoid. The temperature curve, slow and dicrotic pulse, eruption, enlarged spleen, apathetic facies, and coated tongue with red edges and tip, with a history of a prodromal period, are valuable, as is the absence of leukocytosis. Isolation of typhoid bacilli from the blood, stools, or urine is of great diagnostic value. Ehrlich's diazo reaction is merely of value as slightly confirmatory of other tests.

The agglutination, or so-called *Widal reaction*, may not be obtained in typical cases, may appear only during convalescence, or remain for years afterward. It is not often obtained during the first few days, but usually within seven or ten days. The serum obtained from the blood or blisters, or by moistening the dried blood, is added to a young culture of typhoid bacilli until the serum is diluted to a certain degree. Clumping of the bacilli and loss of their motion should follow. A dilution of 1 to 50 should be employed, and a time limit of one hour.

Differential Diagnosis.—(1) Of cases beginning with cerebral symptoms from **cerebrospinal meningitis,** by appearance of abdominal symptoms or the eruption and absence of Diplococcus intracellularis from fluid obtained by lumbar puncture of spinal canal. (2) Of cases beginning with **pneumonia,** by later symptoms, physical signs, and Widal reaction. (3) From **malarial fever,** by presence of Widal reaction and absence of malarial parasites from the blood.

(4) From **pyemia**, by absence of leukocytosis, occurrence of Widal reaction, and identification by blood cultures. (5) From **acute miliary tuberculosis** by less irregular fever, presence of eruption; at times physical signs and bacilli in sputum of tuberculous cases. (6) **Tuberculous peritonitis.** (7) From **appendicitis** at the onset, by presence of other symptoms besides pain and fever (p. 171); the only other period when such confusion is likely is in the second and third weeks, when perforation may occur. (8) From **paratyphoid fever**, in which the Widal reaction is negative and paratyphoid-Widal positive. (9) From **typhus fever** (p. 31).

Prognosis.—The death-rate varies in different epidemics and in private and hospital practice. In hospitals it is 5 to 8 per cent. when hydrotherapy is employed. Unfavorable symptoms are high fever, delirium, tympanites, and hemorrhage. Ambulatory cases are dangerous. Alcoholic patients do badly.

Treatment.—*Prophylaxis.*—Care of drainage, boiling drinking-water and milk during epidemics. Individual utensils for patients. Screens to prevent access of flies to the patient, soiled bedding, excreta, etc. Disinfection of hands of attendants, vessels, bedclothing, feces, urine, and sputum (p. 21). Urotropin by mouth is an effective disinfectant of the urine. Preventive inoculation with sterilized cultures of typhoid bacilli is most efficient. Quarantine patients until stools and urine show no typhoid bacilli.

General.—Bed, careful nursing, ventilation.

Hydrotherapy.—Cold bath, usually for ten minutes, every three hours (omitting two during night, unless nervous symptoms or fever forbids), for temperature above 102.6° or 103°. Temperature of first bath, 80°; second, 75°; subsequent, 70°. Cloth around head, over which cold water is poured. Body and limbs rubbed vigorously while in the tub. Stimulant before or after bath, if necessary; hot milk after it. There should be no fixed rule for temperature of water or that of the patient for which baths are given, or for duration.

These must be adapted to individual cases, watching results. Those desired are tonic action on the nervous system and heart, diminution of stupor or delirium and of insomnia, a less important reduction of temperature, and lowering of mortality. Contra-indications are intestinal hemorrhage, peritonitis, phlebitis, severe nephritis, poor reaction, or no improvement from this treatment. Cold sponging or cold pack may be substituted when baths are not obtainable, or an ice-coil over the abdomen when they are contra-indicated.

Medicinal.—Antipyretics are rarely advisable. If circumstances forbid hydrotherapy, phenacetin (gr. v), antipyrin, or acetanilide may occasionally be used cautiously for the indications governing the use of tubs. Intestinal antiseptics are unsuccessful. Alcohol should be given only when there is severe toxemia, the so-called typhoid state. *Serum therapy* is as yet unsatisfactory.

Of Symptoms and Complications.—For nervous symptoms, hydrotherapy; trional and codeine if necessary. If active delirium is present, morphine may be needed. As it is frequently of a suicidal nature, all possible weapons should be removed, and the patient carefully watched, or at least fastened securely to the bed. For "tender toes," paint with tincture of iodine and alcohol, equal parts, once or twice a day, or apply methyl salicylate. For cardiac weakness, digitalis, caffeine, strychnine, etc. For dry mouth and sordes, frequent cleansing and mouth washes—tincture of myrrh, ℥j: sodium bicarb., ℥ss; water, ℥vij; or, glycerin with lemon juice; or benzoinated oil. For constipation, small doses of calomel and salines at first; enemata or suppositories after the second week, when intestinal complications are likely to occur. For diarrhea, enemata, bismuth, opium, etc. For abdominal distention, turpentine in enemata, ♏v to x by mouth, and in abdominal stupes, and passage of rectal tube. For hemorrhage, absolute quiet, application of cold to the abdomen, morphine if necessary, calcium lactate, gr. xv to xx every four hours; temporary

restriction of food, saline enemata or infusions if required. For perforation, early laparotomy; if this is impossible, morphine to secure absolute quiet, and treat the peritonitis. For bronchitis and pneumonia, the usual remedies; bathing not contra-indicated. For bed-sores, chiefly prophylaxis, protective dressings and lotions.

Diet.—Water, *ad lib.* Enough food should be given to prevent tissue waste, *i. e.*, about 2500 to 3000 calories for a man of 150 pounds, including 70 to 80 grams of protein. It must be regulated by individual digestive ability, and should consist chiefly of milk, cream, ice-cream, eggs, cereal gruels, scraped beef, beef juice, and lactose if needed to supply the caloric requirements. Gradually add solid food when the temperature reaches normal.

Convalescence.—Reduction of stimulants, increased diet, tonics, sleep, getting up gradually. If persistent temperature with no obvious cause and good appetite, get the patient up and give solid food. For bacilluria, urotropin, gr. v, t. i. d., or every four hours, for several weeks.

PARATYPHOID FEVER

A group of cases resembling typhoid clinically, pathologically, and in treatment, but not giving the Widal reaction with Bacillus typhosus. They are caused by the paratyphoid bacillus A or B. The patient's serum agglutinates in high dilution bacilli of the corresponding strain, A or B. The course is usually shorter than that of typhoid and the disease milder.

TYPHUS FEVER

Definition.—Typhus fever is an acute infectious disease, characterized by sudden onset, marked nervous symptoms, macular rash, and ending by crisis usually after two weeks.

It is also called **spotted, jail, camp, hospital,** or **ship fever,** or **exanthematic typhus,** in contradistinction to abdominal typhus, as typhoid fever is sometimes termed.

Etiology.—Bacillus typhi exanthematici, an obligatory anaërobe found in the blood during the febrile period. It is transmitted through the body louse, hence typhus occurs in epidemics of decreasing frequency and extent, usually beginning where there are filth and overcrowding.

Pathology.—No characteristic lesion except the eruption. Blood dark and fluid; moderate hyperplasia of spleen and lymph nodes, but no ulceration of Peyer's patches; albuminous degeneration with swelling of viscera; lesions due to complications.

Symptoms.—Duration of disease about two weeks. *Incubation* period twelve days or less, marked at times by slight malaise. *Invasion* usually sudden, by one chill or several, with high fever, headache, pain in back and legs, prostration, vomiting, and mild or active delirium. Face flushed; dull expression. Pulse not dicrotic. Often bronchitis. *Eruption* appears on the third to the fifth day, fever remaining high. During the second week all symptoms increase and are those of an asthenic fever, with marked delirium and often coma vigil. Death usually occurs from exhaustion at the end of the second week. Favorable cases terminate at this time by crisis; prostration is extreme, but convalescence rapid.

Special Symptoms.—*Fever.*—Sudden onset to even 104° or 105°; steady rise for four or five days, with slight morning remissions; maximum usually on the fifth day, 105° to 107°; then constantly high, with slight morning remissions; terminating by crisis on the twelfth to fourteenth day, falling, in some cases, below normal; in fatal cases a rapid rise to 108° or 109°.

Eruption appears first on the abdomen and upper part of the chest on the third to the fifth day; then on the extremities and face; is complete in two or three days. It consists

of fine, irregular, dusky red mottling, which appears as though some distance below the surface, and often resembles the rash of measles, and of rose spots which become petechial. These remain after death or fade during convalescence.

Complications and Sequelæ.—Bronchopneumonia; less often gangrene of lungs, extremities, nose or mouth, or pleurisy; rarely meningitis, parotitis, septic processes in subcutaneous tissues and joints, and nephritis.

Diagnosis.—By sudden onset, eruption, temperature curve, and severe nervous symptoms.

Differential Diagnosis.—(1) From **typhoid,** by its usual occurrence in epidemics, sudden invasion, temperature, eruption, severe nervous symptoms, slight polynuclear leukocytosis, absence of Widal reaction, duration, and termination by crisis. (2) From **measles,** in which the eruption is brighter, often of crescentic form, and appears first on the face, by absence of catarrhal and severity of nervous symptoms. (3) From **cerebrospinal fever,** by absence of severe spinal symptoms and of Kernig's sign, negative result of lumbar puncture, and general course. (4) From **malignant variola,** by less extensive hemorrhages in eruption and absence of bleeding from mucous membranes.

Prognosis.—Mortality, 12 to 20 per cent.; slight in childhood; increases with age; varies in different epidemics and under varying hygienic conditions.

Treatment. — Like that of typhoid, including hydrotherapy. *Prophylaxis:* destruction of lice; isolation of cases. Inoculations of polyvalent vaccine greatly reduce incidence of the disease but do not give absolute immunity.

RELAPSING FEVER

Definition.—Relapsing fever, known also as **famine, seven-day,** and **spirillum fever,** and as **typhus recurrens,** is an infectious disease characterized by a paroxysm of fever, which

usually lasts six days, and is followed by a remission of the same length, then by another paroxysm. This may be repeated three or four times.

Etiology.—*Predisposing:* overcrowding, deficient food. *Exciting:* spirochete or spirillum of Obermeier, found in the blood during paroxysms. Immunity is not conferred by one attack.

Pathology.—Albuminous degeneration of viscera; spleen large and soft; sometimes infarcts in the kidney and spleen. Nothing characteristic.

Symptoms.—After an *incubation* period of five to eight days, occasionally with slight prodromata, the *invasion* is sudden, with chill, intense pain in the back and extremities, rapid rise of temperature to 104° to 106°; pulse, 110 to 130. There may be gastric symptoms, jaundice, and delirium. After three to ten days (usually five or six) of high or increasing temperature, it falls by crisis within a few hours to normal or below, with profuse sweating, sometimes diarrhea. Rapid convalescence, with recurrence of all symptoms on the fourteenth day. The relapse is usually shorter than the first attack. There may be as many as five relapses.

Complications include pneumonia, nephritis, hematuria, rupture of the enlarged spleen, postfebrile paralyses, ophthalmia, and abortion, but are not common.

Diagnosis.—By typical temperature curve and presence of spirochete in the blood during the paroxysms. Differentiate from **typhoid, typhus, yellow,** and **malarial fevers, cerebrospinal meningitis,** and **acute miliary tuberculosis.**

Prognosis.—Mortality about 4 per cent. In pregnant women abortion usually occurs.

Treatment.—Hygienic and symptomatic, as in other continued fevers; stimulants and analgesics, when necessary.

SMALLPOX (VARIOLA)

Definition.—Smallpox is an acute infectious disease characterized by an eruption which is successively papular,

vesicular, pustular, and a crust, and by a peculiar febrile course.

Etiology.—An unknown poison in the contents of the pustules, crusts, exhalations of the lungs and skin, and apparently in the secretions and excretions. Protozoön-like bodies, cytoryctes variolæ, have been described as present in the cutaneous lesions. The negro and aboriginal races are especially susceptible. One attack does not always confer immunity for life. Severe forms may be contracted from mild cases. The disease is contagious from an early period; direct contact with the patient does not appear to be necessary. The poison is extremely tenacious, and may be carried by one who is not himself affected.

Pathology.—The skin contains areas of inflammation, in each of which develops a fluid-filled reticulum, its contents serous, then pustular. The surrounding tissue is infiltrated with cells, and there is a central area of necrosis. The pus and necrotic tissue dry and form crusts which fall off, leaving scars only if the true skin has been involved. In the hemorrhagic form there are red cells in the reticular spaces. Secondary lesions are diffuse suppurative inflammation of the skin, inflammation of mucous membranes, hemorrhages, and albuminous degeneration of the spleen, liver, and kidneys.

Varieties.—(1) Variola vera: (*a*) discrete, (*b*) confluent. (2) Variola hæmorrhagica: (*a*) purpura variolosa, (*b*) variola hæmorrhagica pustulosa. (3) Varioloid.

Symptoms.—1. **Variola Vera.**—(*a*) **Discrete Form.**—*Incubation*, lasting ten to fourteen days, is usually without symptoms. *Invasion* is sudden, with one or more chills in adults or convulsions in children, with headache, characteristically severe pain in the lumbar region and extremities, vomiting, and rapid rise of temperature to 103° or 104°, and symptoms of sthenic fever (p. 20). At this time there may be purpuric and erythematous initial rashes, diffuse-like scarlatina, or macular-like measles, usually on the lower part of the abdomen, inner surface of thighs, sides of thorax or axillæ.

Eruption.—Usually on the fourth day the characteristic eruption appears as small red papules on the forehead, along the line of the hair, and on the wrists, spreading within twenty-four hours over the face, extremities, trunk, and mucous membranes. With the appearance of the rash, the temperature falls and all symptoms diminish. The eruption is most marked on the face, and matures first there, then on other parts in the order of its appearance. On the fifth or sixth day the papules become umbilicated vesicles with clear fluid contents. About the eighth day these become pustular and their summits globular, while the surrounding skin is red, swollen, and painful. At the same time there is a return of the constitutional symptoms, with rise of temperature for about twenty-four hours. *Desiccation* begins on the tenth or eleventh day. The pustules dry, forming crusts, while the swelling of the skin disappears and the temperature falls by lysis. The crusts fall off, leaving scars only where the true skin has been destroyed.

(*b*) **Confluent Form.**—All symptoms are more severe. The eruption appears on the third or fourth day; the papules are so close together as to become confluent, especially on the face and extremities. The remission of constitutional symptoms at this time is not complete. When the formation of pustules occurs, the face and extremities are practically covered with a single abscess, the eruption on the trunk remaining discrete. The mouth, pharynx, and larynx may be greatly swollen from the eruption, and the temperature may reach 104°, with delirium. In fatal cases the pulse becomes more rapid and feeble, with severe cerebral symptoms, until death. Hemorrhagic symptoms occur in some cases from the eighth to the eleventh day. In favorable cases desiccation begins on the eleventh or twelfth day, and scarring may require three or four weeks.

2. **Hemorrhagic Smallpox.**—(*a*) **Purpura variolosa,** the most serious type of the disease, is marked by more severe general symptoms, and at the end of the second or the third day a

diffuse hyperemic rash, especially in the groins, with punctate hemorrhages which spread very rapidly and appear in the conjunctivæ, mouth, pharynx, and internal organs. Death occurs between the third and the sixth day.

(*b*) **Variola Pustulosa Hemorrhagica.**—Begins like the ordinary form, but hemorrhages occur into the vesicles or pustules and often from the mucous membranes. Usually fatal in seven to nine days.

3. **Varioloid.**—Smallpox modified by vaccination. The invasion may be sudden and severe. It is usually mild and gradual, but with severe pain in the back and head. A scanty eruption of papules, often only on the face and hands, appears on the third or fourth day, with disappearance of constitutional symptoms. The eruption matures rapidly without fever or other symptoms.

Complications include laryngitis with edema of the glottis, bronchitis, bronchopneumonia, pleurisy, rarely lobar pneumonia. Cardiac complications and nephritis are rare. Peripheral neuritis, diffuse myelitis, arthritis which may be suppurative, necrosis of bone, parotitis, conjunctivitis, keratitis, iritis, otitis, etc., may occur. Furuncles and acne are common; gangrene of the skin may be observed.

Prognosis.—Confluent form is often hemorrhagic, nearly always fatal, especially purpura variolosa. Even the discrete variola vera is very fatal to persons not protected. A mild invasion is favorable; high fever, delirium, and subsultus are serious signs. Age, poor constitution, alcoholism, and pregnancy are unfavorable to recovery. The average mortality is 25 to 35 per cent. The prognosis of varioloid is usually good.

Diagnosis.—By existence of an epidemic; chill, pain in back and head, and vomiting; later by the eruption in characteristic stages, with fall of temperature on its appearance and subsequent rise during pustulation.

Differential Diagnosis.—(1) From **measles**, by absence of coryza, severe conjunctivitis, and buccal spots. (2) From **typhus** (p. 31), **hemorrhagic scarlet fever, pustular glanders,**

and **impetigo contagiosa.** (3) From **varicella,** which may resemble mild variola, by initial rashes, the occurrence of the relatively scanty eruption more abundantly upon the trunk in varicella, while the papules are less shotty, and pocks in all stages of development can usually be seen at one time, and the constitutional symptoms of varicella are generally milder. (4) From **cerebrospinal fever** with purpuric symptoms, by the absence of Kernig's sign and by lumbar puncture. (5) From **pustular syphilide,** by the history and greater number of lesions on the face. (6) From **eruptions due to counter-irritants,** such as croton oil, by localization of the latter, with absence of constitutional symptoms, and by the history.

Treatment.—*Prophylaxis.*—Vaccination of children at the second or third month, of all persons from time to time, and always after exposure to the disease or during epidemics. Isolation of the patient in a bared room, with subsequent disinfection (p. 20). *General:* as in other infectious diseases; in bed during febrile stages; eyes and mouth cleansed frequently with saturated solution of boric acid; stimulation, as needed. *Diet:* milk, broth, and other digestible food. *Of symptoms:* for fever and nervous symptoms, cold sponging or baths. If delirious, they are given at about 70°, every three hours, for a temperature of 103°. For eruption, while papular and vesicular, cover with lint soaked in bichloride or carbolic acid solution, the latter also to diminish odor; for crusts, keep moist with oil, vaseline, or glycerin. For diarrhea, paregoric. For hemorrhage no known treatment is efficient. *During convalescence:* daily baths with carbolic soap to remove crusts, which should be burned. Isolation until desquamation has ceased.

VACCINIA (COW-POX)

A disease of the cow, with vesicles whose contents inoculated into man cause a local pock and constitutional symp-

toms, affording partial or complete immunity to smallpox for a variable time, sometimes permanently, usually not over ten to twelve years. Lymph from the calf is generally employed, though humanized lymph, from vaccinated persons, is sometimes used.

Etiology.—Bacteriology is uncertain. Probably vaccinia is variola modified by transmission.

Symptoms.—*Incubation.*—At first slight irritation at the place of vaccination. *Eruption* appears on the third or fourth day as a red papule surrounded by a reddened area. On the fifth or sixth day this becomes a vesicle with depressed center and clear contents. It reaches its maximum size on the eighth day. By the tenth the contents are purulent and the surrounding skin more inflamed and often painful. These symptoms diminish, and by the end of the second week the pustule has dried to a brownish scab, which falls off between the twenty-first and twenty-fifth days, leaving a depressed scar. Fever and mild constitutional symptoms usually accompany the eruption, and may last until about the eighth day. The regional lymph nodes are enlarged and tender.

Variations.—Irregular and atypical pocks may form; several vesicles may coalesce. **Generalized vaccinia,** a general pustular rash, usually developing about the eighth to tenth day, sometimes occurs.

Complications.—In debilitated persons, or as the result of infection, sloughing, cellulitis, erysipelas, general septic infection, or syphilis may follow.

Vaccination.—Reliable glycerinated calf lymph should be employed. Syphilis has been transmitted by the use of humanized lymph. The skin is cleansed near the insertion of the deltoid, where the muscular movement will be slight, and consequently the pock less liable to frequent irritation than elsewhere, or on the leg. With a sterile needle, knife, or ivory point a few short scratches are made, deep enough to allow slight exudation of serum, but no bleeding. The vac-

cine virus, moistened if dried on a point, is rubbed into the wound and allowed to dry. A piece of sterile gauze is used as a protective dressing. If unsuccessful, repeat the vaccination. If cellulitis occurs, wet dressings may be required.

VARICELLA (CHICKEN-POX)

Definition.—Varicella is an acute infectious disease occurring in children and characterized by a vesicular eruption.

Etiology.—Specific cause unknown. The disease is usually epidemic, sometimes sporadic, and is not related to smallpox. Several attacks may occur.

Symptoms.—The *incubation* period is ten to fifteen days. *Invasion* with slight fever, chilly sensation, occasionally vomiting, and pain in back and extremities. The *eruption* appears in twenty-four hours on trunk, face, or forehead, on which it is more abundant than on the extremities, usually as a few discrete red papules which soon become rounded vesicles. In thirty-six to forty-eight hours the contents are purulent, and on the third or fourth day crusts form and fall off later. There is often itching, which leads to scratching and infection of the pocks with subsequent formation of scars. The eruption appears in successive crops for two or three days, so that all stages of development are often seen at once. It may occur in the mouth and pharynx.

Complications are rare.

Differential Diagnosis.—From **smallpox** (*q. v.*).

Prognosis.—Good. Death has occurred from extensive involvement of the skin and from complications.

Treatment.—Symptomatic if necessary. Protect the eruption from scratching.

SCARLET FEVER (SCARLATINA)

Definition.—Scarlet fever or scarlatina is an acute infectious disease characterized by sore throat and a diffuse scarlet eruption.

Etiology.—An unknown germ, probably in the secretions of the respiratory tract or in the skin, possibly a modified streptococcus. It is extremely resistant and may be conveyed by milk, in bedding, etc. The disease is epidemic, sometimes sporadic, and usually occurs between the ages of two and ten years. One attack does not necessarily protect from others.

Pathology.—Catarrhal inflammation of tonsils, pharynx, and larynx. Sometimes cutaneous and internal hemorrhages, albuminous degeneration of the viscera, or acute nephritis. Croupous or gangrenous inflammation of tonsils, pharynx, and larynx, acute hyperplasia or suppuration of cervical lymph nodes, bronchopneumonia, endocarditis, or pericarditis may result from mixed infection with pyogenic cocci.

Symptoms.—*Incubation*, one to seven days, usually two to four. *Invasion* generally sudden with vomiting, convulsions in young children, rarely a chill, rapid rise of temperature, often to 104° or 105°, with all symptoms of sthenic fever (p. 20), especially frequency of pulse. *Eruption* develops in twenty-four to forty-eight hours as scattered red points on a reddened ground. It begins on the neck and chest and rapidly spreads, usually over the entire body, as a bright scarlet rash, rarely patchy, usually erythematous, occasionally with fine hemorrhagic dots or with sudamina (**scarlatina miliaris**). Other variations occur. On the face it appears first at the sides rather than on the central portion. When the finger is drawn over the rash the pressure causes a white line. The skin is swollen. The tongue is coated in the center, red at the tip and edges, the swollen red papillæ projecting through the central coating. Later the tongue is red and rough, with prominent papillæ—the "strawberry tongue." Parynx congested, with swelling of tonsils and pillars of the fauces, or with follicular tonsillitis or intense membranous inflammation of the entire pharynx and enlargement of cervical lymph nodes. There is leukocytosis. The fever remains high, often 105° or more, for two or three days

after the rash appears, when both gradually subside. Headache and nocturnal delirium cease with the appearance of the eruption. Albuminuria is common. *Desquamation* in large flakes, whose size is in proportion to the severity of the rash, begins when the fever subsides and lasts ten to fifteen days.

Malignant Scarlet Fever.—*Toxemic form:* Severe onset with temperature which may reach 108°, and all symptoms of a very acute infection, including delirium passing into coma. Death may occur in twenty-four to thirty-six hours. *Hemorrhagic form:* There are small hemorrhages into the skin gradually increasing in size, epistaxis, and hematuria. Death may take place on the second or third day. *Anginose form:* Throat symptoms appear early and are severe, with membranous exudate which may extend to the trachea, bronchi, Eustachian tubes, and middle ear. The appearance is that of a severe case of diphtheria. (See Pseudodiphtheria, p. 64.) Death may occur soon or extreme sloughing of the tissues of the neck take place.

Complications and Sequelæ.—(1) Nephritis in the second or third week, which may be severe, with death from acute uremia, moderately acute with some dropsy terminating in recovery or chronic nephritis, or very mild; (2) arthritis, which may become suppurative; (3) endocarditis, pericarditis, myocarditis; (4) pleurisy, often purulent; (5) otitis media and its complications; (6) simple or suppurative adenitis, with sloughing of adjoining tissues; (7) nervous system—chorea, progressive ascending spinal paralysis, etc.

Diagnosis.—By sudden onset, eruption, throat symptoms, tongue, desquamation, and albuminuria.

Differential Diagnosis.—(1) From **acute exfoliating dermatitis,** in which the throat symptoms and strawberry tongue are usually absent. (2) From **measles,** by rapid invasion, absence of catarrhal symptoms, early appearance and characteristics of the rash, absence of buccal spots, presence of sore throat, and character of desquamation. Leukocytosis is usually considered of diagnostic value, as it is generally

held to be absent in measles. (3) From **rubella** by severity of constitutional symptoms, temperature curve and tongue. (4) From **septicemia** of puerperal or surgical type. (5) From **diphtheria** by microscopic examination of throat cultures. (6) From **drug rashes**, which are usually transient and without constitutional symptoms. (7) From **digestive eruptions.**

Prognosis.—Always uncertain, varies with epidemics; worse in infants.

Treatment is that of any acute infectious disease, including fluid diet, bed until ten days after temperature is normal, and special care in isolation, disinfection, and ventilation. The mouth must be kept clean. During desquamation the skin should be greased with oil or vaseline and bathed. For fever and nervous symptoms, hydrotherapy—sponging, warm baths gradually cooled, or cold packs. Severe throat symptoms are treated as in diphtheria (p. 64); otitis media must be especially watched for and treated as usual; nephritis should be guarded against by avoiding exposure for several weeks after getting up, and is treated as in ordinary cases. Antistreptococcus serum and autogenous vaccines may favorably influence the streptococcic complications of scarlet fever. Prophylaxis demands early detection, especially of mild cases, and thorough isolation.

MEASLES

Definition.—An acute infectious disease characterized by initial coryza and a rapidly spreading, blotchy eruption.

Etiology.—An unknown and extremely infectious contagium attacking children most frequently. Measles is endemic and at times epidemic. One attack may fail to confer immunity.

Pathology.—No characteristic lesions; usually bronchitis. The kidneys may show albuminous degeneration or acute nephritis; the lungs, lesions of bronchopneumonia. The bronchial lymph nodes are swollen and often tuberculous.

Symptoms.—*Incubation* period, seven to eighteen days, usually fourteen. *Invasion* is marked by chilly feelings, coryza, redness of eyes and lids, photophobia, then cough. These are all symptoms of a sthenic fever (p. 20), the temperature rising to 103° or 104°. *Eruption* usually appears on the fourth day during the maximum temperature. It begins on the cheeks or forehead as small red papules, which increase in size and extend over the body, becoming rounded or crescentic reddish blotches. The rash is hyperemic; in malignant cases it may become hemorrhagic. The cervical and axillary nodes are swollen and tender. On the fifth or sixth day the temperature begins to fall and all symptoms diminish. The rash remains two or three days, then desquamation occurs in fine scales. Irregularities in the appearance of the eruption often occur. Buccal spots or Koplik's spots seem to occur almost constantly, sometimes before the cutaneous eruption. They are white or bluish-white spots surrounded by red areolæ, and are found "on a level with the bases of the lower milk molars on either side or at the line of junction of the molars when the jaws are closed." It is generally held that there is no increase of the leukocytes in the blood. *Hemorrhagic measles* is marked by a severe invasion, an eruption which becomes petechial, hemorrhages from mucous membranes, severe constitutional symptoms, and early death from toxemia.

Complications and Sequelæ.—The most common **complications** are bronchopneumonia, laryngitis, catarrhal or suppurative otitis media, and enteritis. Chief **sequelæ**, tuberculosis and pertussis.

Diagnosis.—By history of exposure, initial catarrhal symptoms, especially coryza and conjunctivitis, and the eruption.

Differential Diagnosis.—(1) From **scarlet fever** by longer and milder invasion with catarrhal symptoms, the blotchy rash, presence of buccal spots, and absence of leukocytosis. (2) From **German measles** is difficult. (See Rubella, Diagnosis, p. 43.) (3) From **smallpox** (p. 35). (4) From **drug eruptions** by catarrhal symptoms and fever.

Prognosis is favorable in uncomplicated cases. It is worse in armies, institutions, and with pulmonary complications.

Treatment.—Bed, ventilation, light diet, catharsis, protect eyes from light, keep eyes and mouth clean. For high fever, sponging or tepid baths gradually cooled. For bronchial symptoms, expectorants and sedatives if necessary, such as mist. glycyrrhizæ comp., or inhalation of compound tincture of benzoin. During desquamation oil and bathe the skin. Avoid exposure during convalescence, in order to avoid bronchopneumonia and tuberculosis. Isolate for at least five days after rash appears, and exposed, non-immune children from seventh to eighteenth day after contact.

RUBELLA (RÖTHELN, GERMAN MEASLES)

Definition.—Rubella is a disease resembling somewhat both measles and scarlet fever.

Etiology.—Unknown. The disease occurs in rapidly spreading epidemics. Previous attacks of measles and scarlet fever do not confer immunity.

Symptoms.—*Incubation*, ten to twelve days. *Invasion* is marked by chilliness, slight fever, pain in the head, back, and legs, coryza, and a macular rose-red eruption on the throat. The *eruption* appears on the first or second day on the face, then on the chest, and in twenty-four hours on the entire body. It is composed of slightly elevated rose-red spots, usually discrete and of a round or oval outline. It is brighter than the eruption of measles and less crescentic. In two or three days it fades and slight desquamation occurs. The cervical lymph nodes are often swollen.

Complications and Sequelæ are rare.

Diagnosis.—(1) From **measles** by slight prodromal symptoms, little or no fever, the color and diffuseness of the eruption, and early enlargement of the cervical nodes. (2) From **scarlet fever** (p. 41).

Treatment is like that of measles.

EPIDEMIC PAROTITIS (MUMPS)

Definition.—Epidemic parotitis, or mumps, is an acute infectious disease characterized by inflammation of one parotid gland or both, and sometimes by involvement of other salivary glands, or of the testes or ovaries and breasts.

Etiology.—Unknown. The disease is epidemic, is directly communicable, and may probably be conveyed through a third person. It rarely attacks infants and old persons, and is more common in males than in females.

Symptoms.—*Incubation* period, two or three weeks. The *invasion* is marked by slight fever, rarely reaching 103° or 104° in severe cases, and pain just below one ear, where a swelling appears if the parotid is the gland affected. This enlarges greatly within two days, reaching in front of the ear and backward to the sternomastoid and pushing the lobe of the ear forward. There is some difficulty in chewing and swallowing, but often no real pain. The other parotid is usually affected a few days later. Other salivary glands may be involved. The swelling gradually subsides after a week to ten days. Rarely the constitutional symptoms are severe and a typhoidal state may occur.

Complications and Sequelæ.—Orchitis is fairly common in adult males. Rarely it is the only sign of the disease, or develops before the swelling of the parotid gland. Usually it begins as the latter is subsiding, or even a week later. It is unilateral or bilateral, sometimes with effusion into the tunica vaginalis. After increasing for three or four days it generally subsides and only occasionally results in atrophy of the testicle. Ovaritis, vulvovaginitis, and mastitis may occur in women.

Diagnosis.—The swelling in front of and behind the ear, the lobe of which is raised and everted, differentiates the swelling from one in the superficial lymph nodes, while

absence of causes of secondary parotitis and a history of exposure point to its epidemic origin.

Prognosis.—Always good. Occasionally orchitis may interfere with the function of the testicles. One attack usually confers immunity.

Treatment.—Bed, light diet, and catharsis during the acute stage. A protective dressing of cotton or hot or cold applications is usually all that is necessary. If tension is great, leeches may be used. For orchitis, rest, suspension of the testicle, and application of an ice-bag or wet dressing if severe.

WHOOPING-COUGH (PERTUSSIS)

Definition.—Whooping-cough or pertussis is an infectious disease characterized by a paroxysmal cough followed by a long inspiration, the "whoop."

Etiology.—The exciting cause is Bacillus pertussis, found in the sputum. The disease is epidemic, often preceding or following epidemics of measles, and is usually transmitted by direct contact. Children are most often affected. One attack usually protects, and some persons possess natural immunity to the affection.

Pathology.—No characteristic lesions; sometimes complicating bronchitis, bronchopneumonia, or vesicular or interstitial emphysema.

Symptoms.—*Incubation* period, seven to ten days. The *catarrhal stage* begins with catarrhal symptoms in the conjunctivæ, nasal mucosa, and bronchi. After a variable period, usually seven to ten days, the cough gradually becomes worse and the *paroxysmal stage* dates from the first "whoop." These attacks consist of a series of short, spasmodic coughs, followed by a long inspiration through the spasmodically contracted glottis, which causes the shrill whoop, while the face often becomes cyanotic. Several attacks may succeed each other, ending with the expectora-

tion of thick mucus or vomiting. There is often an ulcer at the frenum of the tongue caused by its paroxysmal protrusion. Any irritation of the throat may precipitate an attack. Gradual recovery in from three weeks to four months. Marked leukocytosis appears early, chiefly lymphocytosis.

Complications and sequelæ include hemorrhages into skin, conjunctivæ, or brain, epistaxis, hemoptysis, bronchopneumonia, emphysema and tuberculosis.

Diagnosis.—By the "whoop;" a sublingual ulcer is suggestive; marked lymphocytosis is confirmatory.

Prognosis is favorable in simple cases. Complications, especially pulmonary, may make it very serious.

Treatment.—Isolation and fresh air, preferably outdoor country life. Expectorants and sedatives for the cough in the catarrhal stage. In the paroxysmal stage paregoric, bromides, antipyrin, and belladonna have often given favorable results. Pertussis vaccine seems to cure some cases. In convalescence care should be taken to avoid bronchopneumonia and tuberculosis. Tonics, cod-liver oil, and change of climate are indicated during this period.

INFLUENZA (LA GRIPPE)

Definition.—Influenza is an acute febrile pandemic disease, rapidly attacking large numbers of persons, and endemic or epidemic for several years afterward. It is characterized by inflammations in various regions of the body and great prostration.

Etiology.—Bacillus influenzæ, found at the site of local lesions and in the blood. One attack does not necessarily confer immunity. A distinction is sometimes made between epidemic influenza and pseudo-influenza, or la grippe, which is caused by various bacteria.

Pathology.—No distinctive lesions; rarely those of exudative inflammation of the respiratory, nervous, and digestive

systems combined or singly. The resultant pneumonia is usually lobular.

Symptoms.—*Incubation* period one to four days, usually three or four. The *invasion* is usually sudden, with a chill and all symptoms of a sthenic fever (p. 20) due to a general infection, and with those of the particular local lesion. The symptoms vary according to the site of these local affections, the following being the most common types:

1. **Respiratory Type.**—Begins like a severe coryza, with symptoms of sthenic fever, severe pain in the eyes, back, and extremities, often described as "in the bones," and prostration. After the fever subsides there is often intense general soreness. Symptoms of bronchitis, pleurisy, empyema, or pneumonia may develop. The last is usually a bronchopneumonia of irregular distribution, protracted course, defervescence by lysis, and slow resolution.

2. **Nervous Type.**—The leading or only symptoms may be severe headache, pain in the back and extremities, and prostration. Instead, there may be symptoms of meningitis, encephalitis, neuritis of any type resulting in acute pain or loss of sensation or motion, cardiac neuroses, and mental disturbances, such as melancholia and dementia.

3. **Gastro-intestinal Type.**—Begins with symptoms of acute gastritis—fever, nausea, vomiting, gastric pain—or of acute catarrhal colitis—fever, colicky abdominal pain, free diarrhea, sometimes jaundice. There may also be all the ordinary symptoms of the general infection. Duration, a few days to several weeks.

4. **Febrile Type.**—Fever may be absent or be the only symptom. It is remittent or continuous and lasts several days to two or three weeks. It is often accompanied by pain.

In all types convalescence is often gradual on account of the bodily and mental prostration, with general soreness for several days.

Complications and Sequelæ.—Many persons remain in permanently impaired health, with chronic bronchitis,

cardiac weakness, etc. Respiratory complications, such as pneumonia and pleurisy, herpes, conjunctivitis, and otitis media, are common: Acute nephritis, cardiac affections, and phlebitis are more rare.

Diagnosis.—By the existence of an epidemic, the extreme prostration, the suggestive pains, and by examination of the sputum in the respiratory type of cases.

Differential Diagnosis.—From the diseases which the particular type of influenza simulates, such as cerebrospinal meningitis from cases of the nervous type. The chief difficulty may be in recognizing the disease as the cause of the local lesions, such as neuritis.

Prognosis is usually good in regard to life in uncomplicated cases, but complications and sequelæ may cause death or lead to permanent invalidism.

Treatment.—*Prophylaxis.*—Theoretically, isolation is advisable, but in epidemics is difficult. The nasal discharge and sputum should be disinfected. *For the disease* itself there is no specific treatment. Bed, preliminary catharsis, and careful nursing and feeding are necessary. *Of symptoms:* For pain, aspirin, phenacetin, antipyrin, acetanilide, or morphine, watching for cardiac weakness while giving the antipyretics. A tablet of acetanilid, gr. iij; caffeine citrate, gr. j; and camphor monobromate, gr. ij, every two or three hours, for several doses, will often relieve the pain. For cardiac weakness, stimulation as required, such as strych. sulph., gr. $\frac{1}{30}$, every three or four hours. For catarrhal inflammations, their usual treatment. *In convalescence,* rest, nutrition, change of air, tonics, such as strychnine.

DENGUE

Definition.—**Dengue, break-bone fever,** or **dandy fever,** is an acute infectious disease characterized by pains in the joints and muscles, fever, an initial erythematous eruption, and a terminal eruption of variable type. It occurs

in tropical regions and the warmer portions of the temperate zone.

Etiology.—Specific germ unknown. It is transmitted by the bite of a mosquito, *Culex fatigans*. The disease appears in epidemics, rapidly attacking many persons. One attack is thought to confer immunity.

Pathology.—Unknown.

Symptoms.—*Incubation* period, three to five days, without symptoms. *Invasion* is marked by chilly feelings, symptoms of a sthenic fever (p. 20) with gradual rise of temperature, and severe pain in the muscles and in the large and small joints which become red and swollen. The pain in the head, eyeballs, back, and extremities is intense. The face is suffused, the eyes are injected, and the skin and mucous membranes flushed. This is the initial rash. After three or four days the high fever falls by crisis, sometimes with diaphoresis, diarrhea, or epistaxis, and the patient feels stiff and sore, but comparatively well. After two to four days there is a return of slight fever, sometimes absent, pains, and the appearance of a macular, scarlatiniform, papular, urticarial, or other eruption. Lymph nodes are often enlarged. There may be jaundice. An attack usually lasts seven or eight days and is often followed by a protracted convalescence, with pain and stiffness in the joints and muscles and great prostration. A relapse may occur at any time within two weeks.

Diagnosis.—By the occurrence of an epidemic and the severe pains.

Differential Diagnosis.—(1) From **acute articular rheumatism** by the existence of an epidemic. (2) From **yellow fever** is very difficult. In the latter the facies, slowing of the pulse, with constant or rising temperature, albuminuria, and earlier occurrence of jaundice are the most valuable differential points.

Prognosis.—Nearly always good.

Treatment.—Symptomatic, chiefly for pain; occasionally hydrotherapy for the fever; tonics during convalescence.

CEREBROSPINAL FEVER

Definition.—Cerebrospinal fever, known also as **epidemic cerebrospinal meningitis, petechial fever,** and like typhus as **spotted fever,** is an infectious disease, sporadic or epidemic, caused by the Diplococcus intracellularis, and characterized by inflammation of the meninges and an irregular course.

Etiology.—Children and young adults are most often affected. Overexertion and unhygienic surroundings seem to be predisposing factors. The Diplococcus intracellularis meningitidis is usually found in the meningeal exudate, generally within polymorphonuclear leukocytes. It is probably transmitted through the nose. Meningitis carriers may harbor the germ in the nasopharynx and convey it to others, yet be uninfluenced by it themselves.

Pathology.—There is congestion of the meninges of cord and brain which are covered with a fibrinopurulent exudate, confined, on the brain, chiefly to the base. Serous distention of the lateral ventricles occurs more often in children than in adults. The secondary lesions are those of any severe general infection.

Symptoms.—(1) **Ordinary form**: They are those (*a*) of a general infection; (*b*) of disturbances of the brain and spinal cord; (*c*) due to stimulation followed by paralysis of various motor, sensory, and cranial nerves. The *incubation* period, of unknown length, is occasionally marked by loss of appetite, headache, and pain in the back. The *invasion* is usually sudden, with a chill, projectile vomiting, severe headache, pain and rigidity of the back of the neck and of the back, pain in various parts of the body, cutaneous hyperesthesia, irritability, and a temperature of about 102°, with all symptoms of a sthenic fever (p. 20). Later the pains become severe, especially in the head, neck, and back; the head is retracted; often the back is rigid; the muscles of the neck and back are tender, and attempts to stretch them cause

intense pain. Kernig's sign is present (see Diagnosis). Vomiting becomes less marked. The temperature is extremely irregular, 99° to 105° or more, and its height is not indicative of the severity of the disease. The pulse is slow, often 50 to 60, full and strong at first. Severe delirium of variable type is common, alternating with partial or complete coma, the latter predominating toward the close of fatal attacks. There are symptoms due to stimulation of such nerve centres and cranial nerves as are involved, followed by those of their paralysis, such as strabismus, nystagmus, ptosis, unequal, contracted, dilated, or sluggish pupils, acute and painful hearing, succeeded by deafness, spasmodic contractions of groups of muscles of the face, trunk, or extremities, followed by paralysis, particularly of the facial muscles, etc. Herpes or a petechial, urticarial, or other eruption may occur. There is marked leukocytosis. The course of the disease is very variable, lasting from a few hours to several months, with frequent changes in the temperature, general condition, and symptoms. Many deaths occur within five days. In fatal cases the patient passes into coma, with symptoms of an asthenic fever (p. 20) and often retention of urine. The temperature may rise to 108° before death. In favorable cases convalescence is slow and often interfered with by complications. (2) **Mild cases** occur, with only slight fever, headache, stiffness of the neck, discomfort in back and extremities, nausea and little vomiting. (3) **Malignant type** occurs in epidemics or sporadically. Sudden *invasion* with severe chills, slight rise of temperature, pain in the back of the neck, headache, stupor, muscular spasms, a slow but feeble pulse, often a purpuric eruption, coma, and death within hours rather than days. (4) **Abortive type**: The *invasion* is severe, but the attack lasts only a day or two and is followed by rapid convalescence. (5) **Intermittent type**: In this the temperature curve is of a septic type. (6) **Chronic type**: This is described as lasting from two to six months, the successive febrile attacks probably being due to cerebral complications.

Complications and Sequelæ.—The complications include pleurisy, pneumonia, pericarditis, endocarditis, arthritis with serous or purulent effusion, inflammation of the eye, which may result in partial blindness, otitis media, and mastoiditis, often followed by deafness. The attack may be followed by persistent headache, mental feebleness, chronic hydrocephalus, or temporary paralysis of groups of muscles.

Diagnosis.—The occurrence of an epidemic is suggestive. Projectile vomiting with severe headache, rigidity, and tenderness of the neck and back, fever, delirium, coma, and symptoms due to irritation of nerves are important features.

Kernig's sign—inability to extend the leg passively while the thigh is flexed at right angles to the body—seems to be constant. *Lumbar puncture* yields a fluid usually turbid, purulent, or bloody, and containing the Diplococcus intracellularis meningitidis. *Technic:* The patient lies on the side with thighs and body flexed, and with strict aseptic precautions an aspirating needle is introduced at one side of the median line upward and inward into the third lumbar interspace until its point enters the spinal canal and the fluid flows out drop by drop. The fluid usually contains an excess of polymorphonuclear leukocytes.

Differential Diagnosis.—(1) From **tuberculous meningitis** by sudden onset, more severe symptoms, absence of signs of active tuberculosis elsewhere, usually a less protracted course, and by the presence of intracellular diplococci and excess of polymorphonuclear leukocytes in cerebrospinal fluid. (2) From **acute cerebrospinal leptomeningitis** by absence of the primary lesions from which this arises—acute infectious diseases, especially pneumonia, septicemia, and erysipelas, injury or disease of the bones of the ear, nose, etc.—and by examination of cerebrospinal fluid. (3) In pneumonia, typhoid, septicemia, and other **acute infections** it should be remembered, however, that the action upon the brain of toxins in the blood may cause cerebral symptoms closely simulating meningitis, though no lesion of the men-

inges except congestion can be found. (4) In children, **gastro-intestinal disturbance** may closely simulate meningitis.

Prognosis varies in different epidemics, being worse at the beginning and height of each. It is worse in children and in cases with deep coma, convulsions, high fever, or prolonged course. Mortality, formerly from 20 to 75 per cent., is reduced by serum therapy to from 16 to 42 per cent.

Treatment.—Aspiration of cerebrospinal fluid by lumbar puncture and injection into the canal of 30 to 60 c.c. of Flexner's antimeningitis serum, repeated daily until recovery is assured. If lumbar puncture is dry and symptoms continue or signs of intracranial pressure develop, puncture lateral ventricles and inject 15 to 30 c.c. of serum. Serum is far more efficient if used early. Bed, nutritious fluid diet, stimulation when necessary, and catheterization if retention occurs. Tub bathing is useful. For pain, ice-bags on the head and neck and along the spinal column, and morphine.

LOBAR PNEUMONIA

Definition.—Lobar pneumonia, called also **croupous** or **fibrinous pneumonia, pneumonitis,** and **lung fever,** is an acute infectious disease characterized by an exudative inflammation of one or more lobes of the lungs, with constitutional symptoms due to absorption of toxins, the fever usually terminating by crisis.

Etiology.—*Predisposing causes:* It occurs most frequently in children under six and again with increasing frequency after the fifteenth year, in males, in negroes, in city residents, in debilitated or alcoholic persons, in those subject to exposure, after injury of the chest or other region, and during the winter and spring. It is sometimes endemic or epidemic. Repeated recurrence is common. Diminished resistance from any cause seems to be the chief predisposing factor. *Exciting cause:* The Micrococcus lanceolatus, pneu-

mococcus, or Diplococcus pneumoniæ found in the bronchial secretion and the lung in pneumonia, and in the mouths of many healthy persons. Four types of the pneumococcus are recognized by agglutination tests. There may be an acute general infection without special involvement of the lung, or the pulmonary lesion may be part of a general infection with localized processes elsewhere, or these may occur alone. There may be epidemics of pneumococcus catarrh of the upper respiratory tract resembling influenza. The virulence of the pneumococci present, as well as the degree of resistance of the body, may determine the presence or absence of symptoms and the location and type of the lesion.

Pathology.—The whole of one or more lobes is usually involved. During the stage of **congestion** or **engorgement** the lung is deep red, edematous, and firmer than normal. The capillaries are congested; the air spaces are lined with swollen epithelium, and contain leukocytes, red blood cells, serum, fibrin, and proliferated epithelium. In the stage of **red hepatization** the lobe involved is distended by exudate, firm and airless. Its surface shows fibrinous or scrofibrinous pleurisy. The cut surface is of the reddish-brown color and consistency of liver, dry, coarsely granular from projecting plugs of exudate. The bronchi show catarrhal or fibrinous bronchitis. The air spaces are densely packed with leukocytes, red cells, fibrin, and alveolar epithelium, with pneumococci. From this stage the lung passes through an intermediate condition, in which it is mottled red and gray, to the stage of **gray hepatization** or beginning resolution. The lung is grayish white, softer; the cut surface moister and covered with a grumous fluid. The exudate degenerates and is absorbed or expectorated. These stages run into each other and may coexist in different parts of the lung, the consolidation and other changes spreading from the areas first involved. In delayed resolution or "organizing pneumonia" the exudate may be gradually replaced by connective tissue

which causes permanent fibrous consolidation. There is albuminous degeneration of heart, liver, and kidneys. Frequently associated lesions are pericarditis, endocarditis, meningitis, and empyema usually caused by the pneumococcus.

Symptoms.—*Incubation* period is uncertain. Bronchitis sometimes precedes the attack for several days. The *invasion* is usually abrupt, with a severe chill or chills lasting fifteen minutes to one hour, with sudden rise of temperature and symptoms of a sthenic fever (p. 20). In a few hours there is usually intensely sharp pain, generally referred to the lower part of the front of the chest or of the axilla of the affected side, occasionally to the abdomen, and increased by inspiration and coughing. The patient lies on the affected side, cheeks flushed, expression anxious, nares dilating with each breath. There are a short, dry, painful cough, and rapid, shallow, jerky, often grunting respiration increasing to severe dyspnea. The sputum is at first scanty, mucous, sometimes blood-streaked, then blood-tinged. It contains pneumococci. The characteristic "rusty sputum"—thick, tenacious, red, rusty, or orange-yellow, or "prune-juice sputum"—dark brown and fluid—usually appears after the first day; during resolution it becomes mucopurulent. In some cases the sputum is like that of bronchitis. The pulse is about 100 to 116, usually full and bounding, but may become small and feeble in serious cases. Sudden heart failure may occur after the third day. There is marked polymorphonuclear leukocytosis, which usually rises when a new area of lung is involved or with complications such as empyema. Herpes of the lips, nose, genitals, or anus is common even in abortive cases. Delirium may be mild or acute maniacal. There is constipation, sometimes meteorism in severe cases. Urine is diminished; its chlorides are nearly absent. After three or four days the pain disappears. After the initial rise the temperature continues high, usually about 104° to 105°, sometimes with a precritical rise, often with a pseudocrisis

in which it drops several degrees and rises again. The day of the disease upon which the crisis occurs is, in order of frequency, the seventh, fifth, eighth, sixth, ninth. In a few hours, usually five to twelve, the temperature falls to normal or below, usually with profuse sweating and with sudden relief of all symptoms, yet with no decided change in the physical condition of the lung. Convalescence is usually rapid. A prolonged rise of temperature after the crisis may be regarded as a relapse or as prolonged resolution, or indicate a complication such as empyema. Death may occur at any time after the third day from sudden cardiac failure, from the extent of lung involved, from severe general infection, or from complications.

Physical Signs.—During the stage of **congestion,** rapid respiration; sometimes slight dulness or a rather tympanitic note called Skoda's resonance, respiratory sounds diminished or harsh, crepitant or subcrepitant rales, sometimes sibilant and sonorous rales. These signs may be slight or absent. During the stage of **hepatization** patient lies on the affected side; face flushed or cyanotic; pupils at times unequal; respiration rapid, jerky, with dilatation of nares and diminished movement of the side where the lung is consolidated and compensatory increase of expansion of the other; vocal fremitus much increased unless bronchi are filled, when it is diminished or absent; sometimes pleuritic friction rub felt; dulness, varying from a tympanitic note to flatness, bronchial voice and breathing unless the bronchi are filled. Pulmonic second sound accentuated. During **resolution,** gradual diminution of these signs, with subcrepitant and moist rales.

Complications and Sequelæ.—The chief **complications** are pleurisy; empyema, usually due to the pneumococcus, occasionally to the streptococcus; pericarditis, especially with pneumonia of the left lung; endocarditis; acute arthritis; meningitis; jaundice. **Sequelæ** include delayed resolution, sometimes ending in chronic interstitial pneumonia, abscess,

or gangrene; mental disturbance; rarely temporary delusional insanity.

Varieties of Lobar Pneumonia. — 1. *Local variations:* (*a*) *Apex pneumonia,* marked by great prostration and delirium. (*b*) *Central pneumonia,* all symptoms may be present except those of pleurisy, with absence or incomplete development of physical signs of consolidation and pleurisy until late. (*c*) *Double pneumonia,* both lungs involved, greater respiratory disturbance. (*d*) *Migratory* or *wandering pneumonia,* involving different lobes successively. (*e*) *Massive pneumonia,* bronchi of consolidated lobes filled with fibrinous exudate; flatness, absence of fremitus, breathing and voice.

2. *In Infants.*—Invasion often marked by a convulsion. Apex often involved, with cerebral symptoms simulating meningitis. Pneumonic sputum seldom seen.

3. *In Old Persons.*—Atypical course, often with absence of many of the chief symptoms, poorly developed physical signs, great prostration, with slight local lesions; very fatal.

4. *In Chronic Alcoholic Subjects.*—High temperature, active delirium, pneumonic symptoms slight and often masked by those of delirium tremens.

5. *Terminal pneumonia* occurring in chronic diseases—tuberculosis, endocarditis, nephritis, diabetes, and arteriosclerosis—often causes only a slight fever and increase of respirations to foreshadow the fatal termination.

5. *Secondary pneumonia* in such infectious diseases as diphtheria, typhoid, typhus, influenza, and the plague, is usually an example of imperfect consolidation resembling multiple areas of bronchopneumonia. The symptoms and signs are incompletely developed. There is usually a mixed infection of the bacteria of the primary disease with the streptococcus, staphylococcus, or colon bacillus.

7. *Epidemic Pneumonia.*—Predominating symptoms vary in different epidemics. These cases are usually severe.

8. *Larval Pneumonia.*—Abortive cases with mild symptoms of the invasion, poor physical signs, moderate fever,

and a duration of two or three days. Herpes may occur even in these cases.

9. *Asthenic, Toxic, or Typhoid Pneumonia.*—Severe constitutional symptoms, particularly those referable to the nervous system, overshadow those of the local lesion in the lung. The appearance is that of great prostration like that of the third week of typhoid.

10. *Ether pneumonia* is more often a bronchopneumonia, and is probably due to diminution of the power of resistance of the bronchi to the germs already present.

11. *Delayed Resolution and Organizing Pneumonia.*—While resolution may go on so rapidly that the breath sounds are normal within a week to ten days after the crisis, it is sometimes delayed. There may be persistence of physical signs with no symptoms or with slight fever, rapid pulse, and sweating, suggesting tuberculosis; or persistent consolidation with or without return of fever, ending fatally; or replacement of the exudate by new connective tissue extending into the alveoli from their walls, with fibrous consolidation of the lungs and subsequent contraction. This is sometimes called "organizing pneumonia."

Diagnosis.—By the initial chill, fever, pain, sputum, change in pulse-respiration ratio, physical signs, crisis, polymorphonuclear leukocytosis, and positive blood cultures.

Differential Diagnosis.—(1) From **serofibrinous pleurisy** in which the physical signs, especially in children, may resemble it, can often be made only with the aspirating needle introduced through an intercostal space after sterilization of chest wall and needle. This is particularly the case in massive pneumonia. Leukocytosis, higher temperature, more rapid respiration, and in adults pneumonic sputum, are to be expected in pneumonia. (2) From **empyema** by the same methods as serofibrinous pleurisy. In empyema the temperature is septic. (3) From **acute pneumonic tuberculosis,** often very difficult, as examination of sputum in this disease may fail to show tubercle bacilli. (4) From

acute bronchitis by the more severe symptoms, the physical signs, and sputum. X-ray examination in bronchitis shows no consolidation. (5) From **meningitis**, especially in children, by physical signs, rapid respiration, absence of Kernig's sign, and negative results of lumbar puncture. (6) From **typhoid fever.** Absence of Widal's reaction may be the only feature differentiating pneumonia from typhoid in the third week with secondary pulmonary symptoms. (7) From **appendicitis.** The pain of pneumonia is sometimes referred to the abdomen, suggesting this affection. Diagnose by other symptoms and physical signs of each. (8) Pneumonia is often masked by the symptoms of chronic diseases when it is a **terminal infection.** Either fever or cough in such cases should lead to a physical examination of the chest. (9) **Delirium tremens** may be the chief symptom of pneumonia in chronic alcoholic subjects.

Prognosis.—Average hospital mortality 20 to 40 per cent. In persons over sixty it is 60 to 80 per cent. Prognosis is more unfavorable in the negro, in chronic alcoholic subjects, debilitated persons, and in those suffering from preëxisting serious valvular disease or chronic nephritis, tuberculosis, diabetes, cancer, etc. Meningitis, endocarditis, and pericarditis are complications which seriously affect the prognosis. Severe pneumococcus toxemia or simultaneous involvement of a large portion of both lungs is extremely unfavorable. Continued absence or very slight development of leukocytosis is often seen in fatal cases. Sudden cardiac failure may occur after the third day, even in cases apparently doing well.

Treatment.—*Prophylaxis.*—Keep mouth and throat clean; avoid lowering resisting power. Disinfect pneumonic sputum. *General:* Bed, careful nursing, and watching to prevent injury when delirious or sudden cardiac failure; as thorough ventilation as possible; open-air treatment, with sufficient covering in cold weather, but never excessive; tepid sponging, mouth kept clean, water given in abundance;

fluid diet, with eggs. Everything should be done to save the patient's strength by preventing unnecessary movement, severe respiratory effort, etc. Sleep is of great value. *Of the disease:* Antipneumococcic serum in Type I cases. *Of symptoms:* (*a*) For pain, strap chest, hot applications, morphine hypodermically. (*b*) For nervous symptoms and high temperature, cold sponging. In cases with severe delirium, particularly in alcoholic subjects, cold tub baths are of great value. Hydrotherapy acts as a nervous sedative, lowers temperature, and improves the heart action. In alcoholic subjects paraldehyde in large and repeated doses until nervous symptoms are controlled and sleep secured (℥j to ℥ss). Strychnine may replace the customary alcohol; rarely temporary use of the latter seems advisable. (*c*) For cardiac weakness, tincture of digitalis, ♏xxv t. i. d., is given for two or three days as routine, continuing in sufficient dosage if pulse is frequent or weak; caffeine sodiosalicylate, gr. v, q. 1–4 h., if more active stimulation is required; saline infusions if blood-pressure is very low; camphor and ether hypodermically for sudden cardiac failure; phlebotomy for dilated right heart. Oxygen inhalation gives great relief at times. *Of complications:* As under other circumstances. Pleurisy with effusion may require aspiration; empyema, incision, and drainage.

Summary of Treatment.—(*a*) Save the patient's strength in every way, by morphine and hot applications in the early stage to relieve pain and cause sleep, by free ventilation or actual open-air treatment, to diminish respiratory effort, and by avoiding disturbance. Digitalis in large doses from the onset, continuing and supplementing with caffeine if cardiac weakness occurs, remembering that the crisis may occur at any hour, and hence life should be prolonged in this way even if death seems inevitable. Serum for Type I cases.

DIPHTHERIA

Definition.—An acute infectious disease characterized by a fibrinous exudate, usually on a mucous membrane, with constitutional symptoms caused by the absorption of a toxin generated in it and by presence of the Klebs-Löffler bacillus.

Etiology.—Endemic in cities and epidemic in the cold autumn months. Its severity varies with different epidemics. It attacks most frequently children of from two to fifteen years. Catarrhal inflammations of the respiratory mucous membrane and individual susceptibility predispose to it. The exciting cause is the Klebs-Löffler bacillus, which may be communicated directly from the membrane or discharges, from nasal and buccal secretions of convalescents, from the throats of normal persons, "diphtheria carriers," by infected articles, and infected milk. It varies greatly in virulence, some strains causing no pathogenic effects. Associated with it are Streptococcus pyogenes, and less often Micrococcus lanceolatus, Bacillus coli, and Staphylococcus aureus and albus. The pseudodiphtheria bacillus and others resemble the diphtheria bacillus in form but not in virulence or cultural peculiarities.

Pathology.—The local lesion may be simple catarrhal inflammation or a greenish or gray fibrinous exudate containing pus, red blood, and epithelial cells, and involving chiefly the tonsils, pharynx, soft palate, nares, larynx, and trachea, less often the conjunctiva and alimentary tract from mouth to stomach. It is firmly adherent at first and leaves a bleeding surface when detached; later it is soft and can be removed. It is formed by necrosis of the epithelium and subsequent exudation of fibrin, etc., into this necrotic tissue. The diphtheria bacilli live in the membrane. They may not be found in cultures from it until it has begun to disappear, and are often present in the mouth and throat for weeks after its complete disappearance. The nose or larynx

may be filled by the membrane. Associated lesions are hyperplasia and focal necrosis of the kidney, liver, and spleen, and of the lymph nodes, especially those of the neck; fatty degeneration of the heart; bronchopneumonia; albuminous degeneration of the kidneys, liver, and spleen, or acute nephritis; degeneration of peripheral nerves.

Symptoms.—*Incubation* period, two to seven days, usually two. The *invasion* may be mild or severe. It is usually marked by chilliness, in young children sometimes convulsions, pain in the back and extremities, and a temperature of 102.5° to 104°.

Pharyngeal diphtheria, in typical cases, begins with slight dysphagia and redness of the fauces; then there is general pharyngeal congestion and the membrane appears on the tonsils. It is grayish white, then dirty gray or yellowish; it is adherent, and when removed leaves a bleeding surface upon which a fresh membrane forms. In three days the membrane covers the tonsils and pillars of the fauces, often the uvula. The cervical nodes are swollen and tender. Temperature, 102° or 103°; pulse, 100 to 120. The membrane separates and constitutional symptoms and glandular enlargement disappear in a week to ten days. The constitutional symptoms are usually in proportion to the severity of the local lesion, but not always. The local lesion may be simply a congestion resembling acute pharyngitis, or the membrane may be only in patches on the tonsils, or be represented merely by a soft exudate. In malignant cases all symptoms are severe and rapidly progressive, ending in delirium, stupor, and death in three to five days. In other malignant cases there are gradual invasion, low temperature, pharyngeal congestion with little or no membrane formation, discharge of a dirty brown fluid from nose and mouth, great prostration and death. Death may occur in diphtheria from sudden cardiac failure or complications. The infection may extend to the nasal duct and conjunctiva, antrum of Highmore, or Eustachian tube and ear.

Nasal diphtheria may be very mild or extremely severe. The membrane may be primarily in the nose or confined entirely to it.

Laryngeal diphtheria may occur alone or with a primary pharyngeal diphtheria. After several days of hoarseness and coughing, respiration suddenly becomes difficult, usually at night. The dyspnea at first is paroxysmal; later it is constant. There are depression of the suprasternal region and drawing in of the intercostal spaces during inspiration, with husky voice and increasing cyanosis. Temporary relief may be obtained by coughing up pieces of membrane. The fever is slight. The attack may recur the next night. If the laryngeal obstruction is severe the cyanosis and dyspnea increase and gradual asphyxia leads to coma and death.

Diphtheria of Other Regions.—Diphtheria may be primary in the conjunctiva or extend to the external auditory meatus, or to the skin around the mouth, anus, or genitals, or may occur in wounds.

Complications and Sequelæ.—**Complications** include bronchopneumonia, acute parenchymatous nephritis, hemorrhages from the nose or throat, and erythematous, urticarial, and purpuric eruptions. **Sequelæ.** The most important is paralysis, due to toxic neuritis, and affecting most frequently the soft palate, less often the pharyngeal, laryngeal, ocular, facial, or respiratory muscles or those of the extremities. It usually occurs during convalescence. Sudden cardiac failure is a sequela caused by neuritis of the cardiac nerve and showing itself during the second week or later.

Diagnosis can be made with certainty only by finding the diphtheria bacillus in cultures from the region affected.

Prognosis in mild cases is good. Mortality formerly about 40 per cent.; with antitoxin treatment about 12 per cent. Death may occur from sudden cardiac failure, laryngeal obstruction, severe infection, complications, or paralysis.

Treatment.—*Prophylaxis.*—Isolation of known or suspected cases until throat is germ-free; disinfection of every-

thing in contact with the patient; antiseptic mouth washes if exposed. Immunization by antitoxin if Schick test fails to show immunity; 500 units for child under two; 1000 for an adult. *General:* Bed, isolation, disinfection of mouth, sputum, surroundings, and attendants; careful feeding, liquid diet. Stimulants as required. No drugs are of certain value. *Local treatment* for *pharyngeal* cases: Swabbing the throat with solutions of bichloride, 1 to 1000; phenol, 3 per cent.; hydrogen peroxide, full or half-strength, etc. For *nasal* cases: irrigation of nose and throat with normal salt solution. For *laryngeal* cases, cold or hot applications to the neck, steam inhalations. For *laryngeal obstruction,* intubation or tracheotomy. The case is not considered cured as long as diphtheria bacilli are present. *Antitoxin:* This treatment should be employed as early in the disease as possible. The antitoxin is administered subcutaneously, intramuscularly, or intravenously, according to the severity of the attack. To cure the disease 2000 to 5000 units according to age are given in mild cases; 3000 to 10,000 in moderate; 5000 to 20,000 in severe; 10,000 to 40,000 in malignant cases. The smaller figures given are for infants, the larger for adults. The dose may be repeated if necessary. A few drops of antitoxin may be injected before the full dose to test for anaphylaxis. Antitoxin is injected beneath the skin of the abdomen, buttock, upper arm, or other region with loose subcutaneous tissue. Urticarial and other eruptions, or pain and swelling in the joints, may follow its use. A second dose given ten days after the first may cause anaphylaxis. *Of complications:* For paralysis, rest, strychnine, electricity, massage, etc. For cardiac weakness, rest, avoid exertion; tonics and stimulants.

PSEUDODIPHTHERIA

Definition.—So-called **pseudo-, false,** or **streptococcus diphtheria,** sometimes known as **diphtheroid** or **membranous angina,**

is an inflammation caused by the Streptococcus pyogenes and resembling true diphtheria in the character of the local lesion, which is membranous.

Etiology.—Usually Streptococcus pyogenes. The trouble often follows measles, scarlet fever, or typhoid, and frequently occurs in young children.

Pathology.—Croupous inflammation of pharynx, tonsils, larynx, or nose; sometimes secondary pneumonia.

Symptoms of a favorable case are the presence of a local membranous lesion of the pharynx, larynx, tonsils, or nose; sometimes nasal obstruction, dyspnea, or sore throat. The constitutional symptoms are like those of diphtheria, but the temperature is often relatively higher, the pulse less rapid, and prostration less marked. These symptoms may, however, be very severe. The duration is usually one or two weeks, unless complications arise.

Complications.—Bronchopneumonia, necrosis of tissues of the neck.

Diagnosis.—Cultures show streptococci or other bacteria but no Klebs-Löffler bacilli.

Prognosis.—Usually good, unless complications occur.

Treatment.—*General:* Bed, fluid diet, catharsis. *Local* irrigation with hot alkaline solution, hydrogen peroxide full to half-strength, bichloride 1 to 1000, or argyrol 15 per cent.

ERYSIPELAS

Definition.—Erysipelas is an acute infectious disease, characterized by rapidly spreading inflammation of the skin; usually caused by Streptococcus pyogenes, and generally accompanied by constitutional symptoms. Sometimes called **St. Anthony's fire.**

Etiology.—The disease is endemic, sometimes epidemic; occurs most often in the spring; is contagious, inoculable and transferable by a third person or in bedding, etc. *Predisposing:* Unsanitary surroundings, debility, diabetes,

chronic alcoholism or nephritis, wounds, the puerperal state, and family or personal predisposition. The last may lead to many recurrences. *Exciting:* Streptococcus pyogenes.

Pathology.—Edema of involved skin, with streptococci in lymph spaces, especially near the borders of these areas. There may be suppuration or gangrene of the skin, meningitis from extension, and visceral lesions of toxemia or pyemia.

Symptoms.—Facial erysipelas, the common medical type, is often described as idiopathic, but it seems probable that some solution of continuity of the skin exists in all cases as a point of entrance for the streptococcus.

The *incubation* period is probably three to seven days. The *invasion* is usually sudden, with a chill, followed by a sthenic fever (p. 20) and local symptoms. In other cases these local symptoms appear first. There is redness, usually of the bridge of the nose, and rapidly spreading to the cheeks and other parts. The area involved is elevated, red, shiny, tense, hot, and with an abrupt raised margin. Its surface may be covered with blebs. There may be suppuration or gangrene. The parts first affected become pale and more normal as others are involved. The eyes may be closed, the face and scalp greatly swollen, with edema of eyelids, lips, etc. The cervical lymph nodes are enlarged. Constitutional symptoms may be severe, and in debilitated persons the fever may early assume an asthenic type (p. 20). The temperature is high, often 104° to 106°, and may terminate by crisis. Death may occur from toxemia. In *erysipelas migrans* progressive invasion of various parts of the body is accompanied by corresponding constitutional symptoms and often defervescence by lysis or death from exhaustion.

Complications.—The most common are septicemia and malignant endocarditis. Pneumonia, nephritis, and especially meningitis are more rare.

Diagnosis.—By sudden onset, high temperature, and local elevated area of redness with sharply defined margin.

Prognosis.—Mortality 4 to 7 per cent. Prognosis is usually good, except in chronic alcoholic, diabetic, debilitated persons.

Treatment.—*General:* Bed, isolation, nutritious diet, symptomatic treatment if needed. For fever, cold sponging; for delirium and sleeplessness, trional, chloral, or morphine. *Local:* Any wet or oily dressing may relieve discomfort. There is no certain method of limiting the process, but ichthyol is much used, 50 per cent. in collodion, or as an ointment or a solution. Carbolic acid, 2 per cent. solution, injected just beyond the margin, may be efficacious.

SEPTICEMIA AND PYEMIA

Definition.—Septicemia and pyemia are terms formerly very loosely applied. Properly, *toxemia* refers to the group of symptoms and lesions caused by the presence in the blood of toxins, usually resulting from bacterial growths; *septicemia* or *bacteriemia,* to a condition caused by the presence in the blood of bacteria, as well as their toxins; *pyemia,* to the same condition, with the development of fresh foci of suppuration. *Sapremia* is a septic intoxication, the result of absorption of toxins. Toxins alone, when in the blood, usually cause a chill, sthenic fever (p. 20), prostration, general malaise, headache, and increased frequency of the pulse.

Septicemia

Etiology.—Presence of bacteria in the blood, introduced from a local lesion or with no obvious local infection (*cryptogenic septicemia*). The bacteria most commonly found are streptococcus and staphylococcus, and less frequently pneumococcus, Bacillus typhosus, gonococcus, Bacillus anthracis, and others.

Pathology.—No macroscopic lesions may be found, except that of the initial infection in the first group of cases. There may be visceral congestion, enlargement of the spleen, albuminous degeneration of the liver and kidneys, and lesions of the ganglion cells of the brain and spinal cord.

Symptoms.—*Prodromata:* If there is a local infection, symptoms of this precede the septicemia; in a cryptogenetic septicemia, there may be a chronic illness or a sudden onset during health. *Invasion,* sudden or gradual, with a chill or chilly feelings, followed by symptoms of a sthenic (p. 20) and later of an asthenic fever, with dry tongue, and apathy or delirium. Death may occur in from one to seven days.

Pyemia

Etiology.—The presence in the blood of bacteria, with resultant foci of suppuration, chiefly the staphylococcus, streptococcus, gonococcus, pneumococcus, Bacillus coli communis, Bacillus influenzæ, and others.

Pathology.—Same as in septicemia, with secondary suppurative foci, particularly in the heart, lungs, liver, spleen, and kidneys. There is often a primary suppurative lesion —external wound, otitis media, genito-urinary infection, empyema, appendicitis, osteomyelitis, etc. The bacteria are usually carried in emboli from a local lesion and cause metastatic or embolic abscesses elsewhere.

Symptoms are those of the local lesion at first. The *invasion* of the general infection is marked by a severe chill, followed by high fever, then sweating. The constitutional symptoms are those of a sthenic fever (p. 20). *Course of the disease:* Chills, fever, and sweating, repeated daily or at irregular intervals, with some irregular fever at other times. The fever is very variable, with sudden falls. There are often symptoms of infarctions and abscesses in the organs where emboli lodge. The spleen is enlarged. Hemorrhagic,

erythematous, and other eruptions occur. In severe cases the fever assumes an asthenic type, the patient's appearance resembling that in the third week of typhoid, and death soon occurs. In other cases the chills, fever, and sweating are repeated at irregular intervals. The patients are emaciated, and the skin has a sallow pallor. Death usually occurs eventually from exhaustion.

Diagnosis of Septicopyemia.—Septicemia and pyemia are often indistinguishable from each other. Their diagnosis is considered jointly. It is made by the history of a preceding septic process, such as otitis media or a genito-urinary infection, the general septic appearance, marked leukocytosis, and positive blood cultures.

Differential Diagnosis.—(1) From **malaria,** by leukocytosis and absence of malarial organisms in the blood. (2) From **typhoid,** by absence of rose spots and Widal reaction and the usual presence of marked leukocytosis, which is absent in uncomplicated typhoid. (3) From **meningitis,** by previous history, by absence of focal symptoms and of Kernig's sign, and by signs of infection in other regions. (4) From **acute miliary tuberculosis,** by septic appearance, presence of leukocytosis, which is lacking in uncomplicated tuberculosis, and absence of local tuberculous lesions elsewhere—*e. g.*, the choroid. (5) From **appendicitis** and **other abdominal conditions** confused with this. These diseases may be simulated by general septic infection.

Prognosis.—Death may occur in a few days or months.

Treatment of the primary local lesion is surgical. Autogenous vaccine or antistreptococcic serum may be tried. General treatment is supporting and stimulant, with symptomatic treatment of complications, including relief of pain.

RHEUMATIC FEVER (ARTICULAR RHEUMATISM)

Types and Definition.—Rheumatic fever, known also as **articular rheumatism,** may be acute or subacute. It is an

infectious disease characterized by inflammation of several joints.

Etiology.—*Predisposing:* It is most common in temperate climates with humidity, in winter and spring, in young adults, and in those whose occupations expose them to cold and wet.

Exciting: Probably bacteria, entering, perhaps, through the tonsils. Some ascribe the disease to a primary disturbance of the nervous system; others, to defective metabolism with accumulation of some chemical substance in the body.

Pathology.—Congestion of the soft parts of the joints and effusion into the joint cavities of serum sometimes containing fibrin, rarely purulent. There may be lesions of complicating endocarditis, pericarditis, myocarditis, pleurisy, and pneumonia.

Symptoms.—The *invasion* may be gradual, with malaise and often tonsillitis. It is usually sudden, with pain in one or more joints, and fever. The knees, ankles, elbows, and wrists are most often affected. They become red, hot, swollen, painful, and tender. *Course:* The temperature is 102° to 104°, with symptoms of a sthenic fever (p. 20), modified by profuse acid sweats. The urine is concentrated, very acid, and contains urates in excess. The symptoms frequently disappear partially from one joint as they begin in another, attacking several in rapid succession, the temperature varying with equal rapidity and corresponding with the degree of joint involvement. Anemia is very marked and progresses rapidly. The hemic murmurs associated with it are often confounded with those due to a complicating cardiac lesion. The course of the disease is often greatly modified by its complications. Defervescence is gradual, and the disease may become subacute or chronic. Pain and stiffness of the joints often last long after defervescence.

Subacute articular rheumatism is a milder form, often more prolonged.

Complications and Sequelæ.—*Hyperpyrexia:* The temperature reaches 105° to 108°, often with diminution of joint symptoms, and with marked nervous symptoms—delirium, stupor, sometimes convulsions—and great prostration. *Endocarditis*, most often affecting the mitral valve, is very common and often becomes chronic. The resulting valvular disease is frequently serious. *Pericarditis* occurs with this or independently. *Myocarditis* is less common. *Pneumonia* and pleurisy are not rare. *Chorea* is often associated with acute rheumatism, especially in childhood. *Sudamina*, urticaria, or a hemorrhagic eruption may be present. *Rheumatic nodules*, sometimes as large as a pea, may be found, especially on the fingers, hands, and wrists.

Diagnosis.—By exclusion of other joint inflammations, the multiplicity of joints involved, the rapid changing of the inflammation from one to another, profuse sweating, and the occurrence of cardiac complications and anemia.

Differential Diagnosis.—(1) From **gonorrheal arthritis**, which is usually monarticular, generally in the knee, with history of recent urethritis and presence of discharge. (2) From other forms of **secondary arthritis**, by absence of preceding scarlet fever, colitis, cerebrospinal meningitis, etc. (3) From **septic arthritis** or **osteomyelitis**, in which there may be a history of a preceding septic focus and more severe symptoms, and by the test of treatment with salicylates. (4) From **gout**, in which the first attack is often in the great toe; if polyarthritic, fever may be low or absent, and in chronic cases there may be tophi. (5) From **acute arthritis deformans** (see Diagnosis, p. 135).

Prognosis.—For life, usually good. Subsequent attacks frequently occur. The remote prognosis is often unfavorable on account of complicating cardiac disease, ankylosis, etc. Death often results in cases with hyperpyrexia; sudden death, sometimes from myocarditis.

Treatment.—*General:* Bed until convalescence is well established; flannel clothing; fluid diet, preferably milk;

water to drink *ad lib.;* frequent examination for cardiac and other complications.

Drugs.—During acute stage: (*a*) Salicylates internally and locally. By mouth, preferably sodium salicylate or aspirin, gr. xv to xxx; oil of wintergreen, ♏x to xx, every two or three hours until pain and temperature are relieved, then in decreasing doses less frequently. This may be combined with sodium bromide, gr. xxx to prevent or diminish cerebral symptoms caused by salicylates—buzzing in the ears, deafness, etc.—if these are annoying. Local application of methyl salicylate may be employed alone or in conjunction with internal medication. The drug so applied appears in the urine within thirty minutes, and the relief of pain is very rapid. It is useful also when gastric disturbance prevents internal medication. Method: A layer or two of gauze saturated with methyl salicylate is wrapped around the painful joints and covered with rubber tissue, paraffin paper, or other impervious dressing, held in place by a bandage. This is renewed once or twice daily until the pain in all the joints is relieved. (*b*) Alkalies may be combined with the above or used alone, especially in plethoric persons or when the urine is very acid, giving:

℞—Potassii acetatis,
Potassii citratis,
Sodii et potassii tartratis . . . . . āā gr. xxx
Aquæ . . . . . q. s. ad ʒij.—M.

Sig.—Two teaspoonfuls in water every three hours to which may be added the juice of a lemon.

The dosage and frequency should be sufficient to keep the urine neutral or faintly alkaline. In subacute cases, or after subsidence of acute symptoms, sodium salicylate and potassium iodide, āā gr. v to x, every four hours or after meals. *Of symptoms:* For pain, methyl salicylate locally; ice-bag or hot applications; phenacetin, acetanilid, or antipyrin, when necessary, watching the heart; immobilization

of the joint while symptoms are acute may give great relief, but should not be persisted in long enough to favor subsequent joint stiffness; morphine, if these fail. For persistent fluid in joint, counter-irritation by tincture of iodine, cautery, cantharidal blisters, or local hot-air baths. (See Arthritis Deformans, Treatment, p. 135.) For stiffness of joints, hot-air baths; massage after all inflammatory symptoms have subsided. For anemia, tonics, especially iron and cod-liver oil. *Of complications:* Hyperpyrexia: cold packs or baths. Cardiac and pulmonary complications: treat as usual, keeping the patients in bed until they subside. *In convalescence:* Food, tonics, warm climate.

ASIATIC CHOLERA

Definition.—Asiatic cholera is an acute infectious disease caused by a specific organism and characterized by profuse watery discharges from the bowels and great prostration.

Etiology.—*Predisposing:* Intemperance, general debility, unhygienic surroundings. *Exciting:* The Spirillum choleræ Asiaticæ, often called the "comma bacillus," which is found in the stools, watery discharges, and intestines of affected cases and of "cholera carriers" who may have had no symptoms, and is transmitted by infected water and food. The disease is endemic in India and epidemic in other countries. Whether one attack confers immunity is uncertain.

Pathology.—After death in the stage of collapse: often postmortem elevation of temperature, early and marked rigor mortis, sometimes preceded by muscular twitching. Skin dusky and shrunken. Blood dark and concentrated. Serous membranes may be coated with fibrin. Small intestine may show hemorrhages into the mucosa, which is usually soft and swollen, sometimes congested, generally or chiefly around the nodules of lymphoid tissue. Croupous inflammation and superficial necrosis may be present, and postmortem desquamation of epithelium is common. The

intestines contain the rice-water fluid of the disease or dark bloody fluid. Liver shows granular, hyaline, or fatty degeneration. The kidneys show cloudy swelling, with degeneration of the epithelium or necrosis. If death occurs in the stage of reaction, there may be signs of inflammation in various organs, while the body appears less desiccated.

Symptoms.—After an *incubation* period of one to five days the *invasion* is marked either by simple diarrhea with some malaise and prostration, or by abdominal pain, vomiting, and diarrhea. Mild cases may recover at this time. In the *stage of collapse* there are frequent watery movements resembling rice-water, with vomiting, great thirst, abdominal pain, and cramps in the legs. There is sudden collapse, with shrinking of the soft parts and shrivelling of the skin, feeble pulse, and subnormal temperature. Nearly all secretions are greatly diminished. In so-called **cholera sicca** death occurs before the diarrhea begins, although rice-water fluid is found in the intestines after death. After two to twenty-four hours those who have not died may recover or pass into the *stage of reaction,* in which the signs of collapse and purging disappear. After temporary improvement, with slight rise of temperature at times, there may be a relapse, or the patient may have inflammation of some of the viscera and suppression of urine, with delirium, coma, and death.

Complications and sequelæ include nephritis, diphtheritic inflammation of the larynx, pharynx, colon, bladder, and genitals, and parotitis, pneumonia, and pleurisy.

Diagnosis.—By the occurrence of an epidemic, the characteristic evacuations and collapse, and by bacteriological examination of the intestinal discharges.

Differential Diagnosis.—From **cholera nostras** or **morbus,** by bacteriological examination, including Pfeiffer's serum reaction in which known cholera spirilla are destroyed by addition of blood serum from a suspected case if it be actually one of cholera. From **poisoning** by arsenic, bichloride of mercury, and fungi.

Prognosis is worse in infancy, old age, and debilitated persons, and in cases with rapid collapse, low temperature, and great cyanosis. The mortality varies from 30 to 80 per cent.

Treatment.—*Prophylaxis.*—Isolation of cases, disinfection of excreta and clothing (p. 21), care to avoid pollution of water supply, use of boiled water for drinking during epidemics, and care of general health at such a time, particularly of attacks of diarrhea. Given before cholera is contracted, bacterial vaccine is said to immunize for about fourteen months. *Of the disease and symptoms:* For pain during the invasion, morphine hypodermically and hot applications to the abdomen. For vomiting: lavage or cocaine. Ice, coffee, brandy, or water freely by mouth. Intestinal irrigation with 2 per cent. solution of tannic acid. During the stage of collapse, hot applications to the surface of the body and hypodermic injections of camphor and ether, in addition to the use of morphine. Subcutaneous or intravenous injections of normal saline solution are of great value in replacing the fluids of the body. In the stage of reaction, nursing and careful diet.

YELLOW FEVER

Definition.—Yellow fever is an acute infectious disease characterized by jaundice, hemorrhages and albuminuria.

Etiology.—Endemic in some Spanish-American ports and epidemic in other ports, usually tropical, of America and Africa. It is most common in crowded, dirty, poorly drained portions of sea-coast cities. It is probably caused by a specific organism which is conveyed from one person to another by a mosquito. *Stegomyia fasciata,* and not in clothing, excreta, etc. One attack usually confers immunity.

Pathology.—Skin jaundiced and may contain ecchymoses. Stomach contains altered blood of dark color. Liver light yellow from marked fatty degeneration, sometimes with

focal necroses. Other lesions are those due to toxemia, including albuminous degeneration of the kidney.

Symptoms.—*Incubation* period, about three or four days. There may be slight prodromal malaise, but the *invasion* is usually sudden, with chilly feelings, headache, backache, rise of temperature, and general febrile symptoms, vomiting, and constipation. Early in the disease the face is flushed, with congestion and slight jaundice of the conjunctivæ. Temperature is usually 102° or 103°, falling gradually after from one to three days. Pulse is slow and falls while the temperature rises. Simple albuminuria or severe nephritis is present. After the fall of temperature is the stage of calm, followed by a rise of temperature, with increased jaundice and vomiting of dark altered blood, the "black vomit." Hemorrhages may also occur into the skin or mucous membranes. Cerebral symptoms are sometimes severe. Convalescence is usually gradual. The severity of the disease varies from great mildness to extreme malignancy.

Complications include abscesses and parotitis.

Diagnosis.—By the early flushing of the face and jaundice, the progressively slowing pulse with constant or increasing temperature, albuminuria, and black vomit.

Differential Diagnosis.—(1) From **dengue** is difficult. The facies, albuminuria, and pulse are the chief features. (2) From **malarial fever**, by early jaundice, absence of malarial organisms in the blood and of change in size of the spleen.

Prognosis varies with the epidemic. Mortality, 15 to 85 per cent.

Treatment.—*Prophylaxis.*—Prevention of spread of infected mosquitoes; use of screens and netting at night in infected districts. *Of the disease:* No specific known. Catharsis at first. Careful nursing, food by rectum while vomiting is frequent. *Of symptoms:* For fever, hydrotherapy; for hemorrhage, opium is most reliable; for vomiting, cracked ice, cocaine, etc.; for cardiac weakness, stimulants when needed; for severe toxemia, saline enemata.

PLAGUE

Definition.—Plague is an acute infectious disease characterized by inflammation and often suppuration of lymph nodes, pneumonia, and cutaneous hemorrhages. It has long been known as the plague or "black death," on account of the petechial eruption.

Etiology.—The Bacillus pestis, which is transmitted, in almost all cases, by the rat-flea and so indirectly by rats. For this reason it may be carried by persons not themselves infected.

Pathology.—(1) In the *bubonic type* the lymph nodes, particularly the axillary and inguinal, show inflammatory lesions with marked hyperplasia, sometimes suppuration, hemorrhage, or necrosis. The same changes may occur in the periglandular tissue. Other lesions are albuminous degeneration, focal necrosis, and secondary foci in the spleen and liver. The bacilli are found in great numbers in the affected nodes and secondary lesions. (2) In the *septicemic type* all lymph nodes and nodules show signs of toxemia, and the bacilli are found in the primary lesions and the blood. (3) In the *pneumonic type* there are areas of bronchopneumonia, with lesions of the bronchial lymph nodes. The bacilli occur in these situations and in the sputum.

Symptoms.—(1) In *bubonic plague*, the usual form, the *invasion* is marked by headache, depression, pain in the back, stiffness of the extremities, and fever. This rises for three or four days, then falls several degrees, and is followed by a more severe secondary fever of asthenic type (p. 20). About the third to the fifth day the lymph nodes usually become enlarged, most often in the inguinal region. This is followed by resolution, suppuration, or necrosis. A petechial eruption and hemorrhages from mucous membranes often occur. The mild cases, which often occur at the beginning of an epidemic and at its close, are marked only by

slight fever and glandular swelling, which may terminate in suppuration. The symptoms are mild and last only a few days. (2) *Septicemic plague* is characterized by symptoms of a severe general infection, with hemorrhages, rapid course, and death in three or four days, without the development of swelling of the lymph nodes. Cultures from the blood show the bacteria. (3) *Pneumonic plague.* The symptoms are those of a severe lobular pneumonia, with bloody sputum containing many bacilli. It is usually rapidly fatal.

Diagnosis.—By occurrence of an epidemic, the inflammatory lesions of the lymph nodes, petechial eruption, and bacteriological examination of urine, feces, sputum, or pus.

Prognosis.—Untreated and under unfavorable conditions, the mortality may reach 90 per cent. The septicemic form is very fatal; the pneumonic is more so. Haffkine's prophylactic inoculations appear to confer partial immunity for three to six months, to reduce the severity of infections, and to diminish the mortality.

Treatment.—*Prophylaxis.*—Prolonged isolation, disinfection of excreta (p. 21), cremation of persons dying from the plague, destruction of rats, and prophylactic inoculation of healthy persons with sterilized cultures of the Bacillus pestis. Immunity following this procedure is said to last from one to eighteen months. *Of the disease:* Several antitoxic serums have been tried, but have not yet been proved of great value. Haffkine's prophylactic inoculation of sterilized cultures of the bacillus seems to prevent infection in many persons, and also to diminish the mortality, even if not given until the disease has begun. Catharsis is recommended. *Of symptoms:* For pain, morphine; for weakness, stimulation; for fever, hydrotherapy; for buboes, applications of ice, injections of bichloride, and incision have been advised.

BACILLARY DYSENTERY

An acute intestinal infection by a specific bacillus, characterized by abdominal pain and frequent loose stools with blood and mucus.

Etiology.—Epidemic or sporadic, most severe in tropical climates. The specific cause is Bacillus dysenteriæ of which several strains exist: "Shiga type;" "Flexner-Harris type," the most common in the United States; and "Bacillus Y." It is found in the stools and is probably transmitted by fecal contamination of drinking-water.

Pathology.—Mucosa of the large intestine and sometimes the ileum is swollen, dark red, with hemorrhagic spots, and covered with a thin necrotic layer. The follicles are swollen, but not ulcerated. In subacute cases there is less thickening and necrosis, but the follicles are more prominent and erosions occur.

Symptoms.—*Incubation* period is less than two days. The *invasion* is sudden, with fever, pain in the abdomen, frequent passages with mucus. These soon contain blood and become very frequent, with great tenesmus, extreme thirst, and severe pain. The temperature may be 103° or 104°. In severe cases delirium may precede death on the third or fourth day. In milder cases convalescence is gradually established in two or three weeks, or the disease runs a subacute course for many weeks.

So-called **acute catarrhal dysentery** is a mild form. **Diphtheritic dysentery** is a type characterized by great congestion and necrosis of the mucosa; it is frequently secondary to other diseases such as pneumonia, and chronic cardiac and renal affections.

Diagnosis.—By bacteriological examination, and by agglutination of cultures of the bacillus with the serum of persons suffering from the disease after the third or fifth day.

Prognosis.—Mild sporadic cases often recover or may become chronic; severe epidemic cases, especially in the tropics, are often fatal.

Treatment.—In the acute stage, bed, fluid diet, free saline purgation, or castor oil, ℥ss, followed by castor oil, ♏v, with salol, gr. v, in capsules, every three hours, or bismuth subnitrate, ℥ss–j, every two or three hours; irrigation of the colon with normal salt solution, and after acute symptoms subside, with solution of silver nitrate, gr. xx to xxx to the pint, at about 100°, with a long rectal tube. For pain, morphine; hot applications to abdomen. Stimulants as needed. In chronic cases, change of climate and varied diet are more important than medication and irrigations.

MALTA FEVER (UNDULANT FEVER)

Definition.—An endemic, non-contagious disease, characterized by irregular fever or febrile relapses, joint pains, sweating, enlarged spleen, and presence of Micrococcus melitensis in the blood. It is transmitted by milk of infected goats and possibly by mosquito bites. The disease occurs in Mediterranean countries, India, China, the Philippines, and Porto Rico. There are no typical pathological lesions.

Symptoms.—*Incubation period*, six to ten days. The fever is irregular or marked by intervals of apyrexia of two or more days, with febrile relapses lasting one to three weeks. There are constipation, anemia, joint symptoms, debility, splenic enlargement, etc. Ordinary cases may last three months to two years. Malignant cases may prove fatal within a week. Mortality is 2 per cent.

Diagnosis.—The agglutination test with blood serum is the positive means of diagnosis. Typhoid and malarial fevers must be differentiated.

Treatment.—Like that of typhoid. Change of climate is important. Prophylaxis, avoid infected milk.

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS)

Definition.—An acute epidemic or sporadic infection involving chiefly the anterior horns of the spinal cord.

Etiology.—A microörganism transmitted through the mouth, tonsils, and nose from secretions of nose or mouth or stools of an infected person, probably often "carriers," themselves immune. Children from one to five years old are most often attacked.

Pathology.—Lesions are those of poliomyelo-encephalitis.

Symptoms.—*Incubation* two to fourteen days. Early symptoms: fever, vomiting, slight diarrhea, listlessness, fretfulness, drowsiness; then paralysis of any extremity, sensitiveness of skin and muscles, spinal pain especially on flexion, rigidity of neck with Kernig's sign; or without prodromata the child may awake with paralysis and slight fever. After two or three weeks the paralyzed muscles may begin to recover or atrophy and contractures ensue. Growth of the affected limbs may be arrested. Abortive cases are common, slight paralysis rapidly disappearing. Some cases resemble Landry's paralysis; others, cerebrospinal fever; those of the bulbar type show involvement of cranial nerves.

Diagnosis is aided by lumbar puncture. Spinal fluid is usually clear, with increase in quantity, albumin and globulin and good reduction of Fehling's. Cells increased, sometimes to 900 per c.c. At first over 50 per cent. may be polymorphonuclears, later, usually 90 per cent. or more are mononuclears.

Prognosis.—Mortality 4 to 25 per cent.

Treatment.—*Prophylaxis:* Isolation; cleanliness of home and person; handkerchief over mouth when sneezing or coughing; cleansing nasal spray; hexamethylenamine. *Of the disease:* Bed for five or six weeks; hexamethylenamine, gr. v to xv, t. i. d.; lumbar puncture; immune and normal human sera are on probation; support of paralyzed limbs to prevent deformity; aspirin for pain. After tenderness has gone treatment is orthopedic.

ANTHRAX

Definition and Etiology.—Anthrax, charbon, woolsorter's disease, or **splenic fever** is an acute infectious disease of animals, transmissible to man by inoculation into wounds or by inhalation of, or swallowing, the germs. Workers in animal products—butchers, tanners, shepherds, etc.—are most often attacked. The exciting cause is the Bacillus anthracis.

Pathology.—The local cutaneous lesion is a pustule containing the bacilli, which may also invade the general circulation. If the germs are inhaled, there is bronchopneumonia; if swallowed, areas of inflammation and sometimes necrosis in the intestine. The spleen and lymph nodes are hyperplastic.

Symptoms.—1. **External Anthrax.**—(*a*) *Malignant pustule:* This begins as a papule at the point of inoculation, becoming vesicular and pustular, surrounded by an inflammatory area, with marked edema. Neighboring lymph nodes enlarged and tender. The temperature rises rapidly at first, subsequently may be subnormal. Constitutional febrile symptoms may be severe. Death in three to five days or recovery. (*b*) *Malignant anthrax edema:* In this the lesion is a pustule, with very marked edema. The eyelid or other part of the face and the upper extremities are the most frequent sites. The edema may terminate in fatal gangrene.

2. **Internal Anthrax.**—(*a*) *Intestinal anthrax* or *mycosis intestinalis* is caused by the introduction of the bacteria into the alimentary canal in infected meat, milk, etc. The *invasion* is marked by a chill, followed by moderate fever, vomiting, diarrhea, pains in the back and legs, and restlessness; sometimes convulsions and hemorrhages into the skin and from mucous membranes. The spleen is swollen. Prostration may be extreme and often ends in death. (*b*) *Woolsorter's disease* occurs among those employed in picking over

wool or hair of infected animals, the germs being inhaled or swallowed. The onset is sudden, with a chill, then fever, pain in the back and legs, and severe prostration. There may be dyspnea and signs of bronchitis, or vomiting and diarrhea. Death is the common termination, sometimes within a day.

Diagnosis of both external and internal forms is usually suggested by the patient's occupation and verified by bacteriological examination of the contents of the pustule, sometimes of the blood.

Prognosis.—Mortality from 5 to 26 per cent.; least after wounds of the extremities, greatest after those of the head.

Treatment.—The local lesion should be cauterized and solutions of carbolic acid or bichloride injected around it and applied to its surface. Stimulation and feeding are important. In the internal forms early purgation may eliminate some bacteria.

HYDROPHOBIA

Definition.—Hydrophobia is an acute infectious disease chiefly of carnivorous animals, transmissible to man by inoculation. Called also **rabies.**

Etiology.—The unknown virus is communicated by the bite of an infected animal, usually the dog, cat, wolf, or cow, or by inoculation of the virus in its saliva through abrasions of the skin or mucous membranes.

Pathology.—There are collections of leukocytes around bloodvessels and ganglion cells of the central nervous system, with degeneration of nerve cells. Irregular bodies, probably protozoa, are described by Negri as constantly present in the central nervous system.

Symptoms.—*Incubation* period varies from two weeks to three months. In the *premonitory stage* there are uneasiness, dread of the disease, malaise, pain in the bite, and beginning dysphagia and excitability. In the *stage of excitement* the typical convulsive spasms of the pharynx and larynx occur after any slight external irritation, physical or mental.

There is great difficulty in swallowing on account of the resulting spasm, hence dread of water. There may be salivation with forcible expectoration. There is great anxiety, sometimes wild excitement. Temperature is slight or may reach 103°. After one and one-half to three days this stage passes gradually into the *paralytic stage* of increasing prostration, without convulsions, ending in death.

Diagnosis.—By the history of a bite, typical symptoms, presence of Negri bodies, and subdural inoculation of rabbits with the medulla of the animal which inflicted the wound, causing paralytic hydrophobia. **Lyssophobia,** or dread of the disease, must be differentiated; also **tetanus,** in which there are trismus and prolonged spasms of abdominal and dorsal muscles.

Prognosis.—Mortality has been said to reach 80 per cent.

Treatment.—*Prophylaxis.*—Muzzling of dogs, disinfection and cauterization of the bite, and Pasteur's preventive inoculations with attenuated virus gradually increasing in strength. *Symptomatic:* Chloroform and morphine to diminish spasm, cocainization of the pharynx, rectal feeding if necessary.

TETANUS (LOCKJAW)

Definition.—Tetanus is an infectious disease characterized by tonic convulsions.

Etiology.—Bacillus tetani is found chiefly in soil, manure, and the intestines of herbivora. Infection is more common in certain localities and often follows small punctured wounds of the exposed portion of the extremities. The bacilli grow only in the wound and the toxin travels along the nerves.

Pathology.—No characteristic lesion is known.

Symptoms.—*Incubation,* ten to fifteen days or less. The *invasion* is occasionally marked by a chill or chilly feelings, usually by rigidity of the neck, jaw, and face. This increases gradually to a tonic spasm and extends to the muscles of the trunk and extremities. The body becomes rigid in a straight

line or bent forward, backward, or laterally. There may be asphyxia from spasm of the glottis. The tonic spasm has frequent exacerbations after any slight irritation and is extremely painful. The temperature is usually low, but may rise very high, especially late in the disease and postmortem. Death occurs from asphyxia, heart failure, or exhaustion. In mild cases the paroxysms become less frequent and severe, and recovery may take place.

Diagnosis.—By history of wound, symptoms, and culture from pus in wound. (1) From **strychnine poisoning**, in which rigidity of the jaw muscles rarely occurs, and then only late, and the muscular contraction is intermittent. (2) From **tetany**, in which the spasm begins in and is chiefly confined to the extremities, particularly the hands, which are held with fingers flexed at the metacarpophalangeal articulation, their other joints extended and thumbs flexed against the palms. (3) From **hydrophobia** (p. 84).

Prognosis.—Mortality above 80 per cent.

Treatment.—Chiefly prophylactic: Excision and irrigation of wound with iodine or 2 per cent. silver nitrate solution. Tetanus antitoxin injections as prophylactic for wounds from fireworks, in gardeners and stablemen. Of the disease, isolation, absolute quiet, antitoxin intraspinally, intravenously, subcutaneously. Chloroform and morphine for spasm. Rectal or nasal feeding if necessary.

GLANDERS

Definition.—Glanders is an infectious disease of the horse and occasionally of man; called **glanders** when the local lesion is in the nares, **farcy** when in the skin.

Etiology.—Bacillus mallei introduced usually from infected horses through the nasal and buccal mucosa or cutaneous abrasions.

Pathology.—In the skin or the mucous membrane of the nose or mouth are nodules resembling miliary tubercles or larger. They consist of collections of leukocytes and young

connective-tissue cells and the specific bacilli. If superficial these result in ulcers; if deeper, in abscesses. Metastatic foci of inflammation and necrosis may occur in the internal organs or skin.

Symptoms.—(1) *Acute glanders.* Incubation, three or four days. Inflammatory signs at site of infection and constitutional febrile symptoms. In two or three days nodules appear on the nasal mucosa and ulcerate, with a mucopurulent discharge. They sometimes become necrotic and the discharge foul. The cervical lymph nodes are enlarged. A papulopustular eruption appears over the face and joints. Pneumonia may occur. Death takes place in eight to ten days. (2) *Chronic glanders* may last for months. It resembles chronic coryza with ulcer of the nasal mucosa. Some recover. (3) *Acute farcy.* The local and constitutional signs are those of an acute infection, with necrosis at the site of inoculation. Nodules known as farcy buds form along the lymphatics and suppurate. There may be purulent collections in the joints and muscles. Death often occurs in ten to fifteen days. (4) *Chronic farcy.* Tumors in the skin of the extremities, which suppurate. The process is local, the inflammatory symptoms slight, and the duration may be months or years. Death may occur from acute glanders or pyemia.

Diagnosis.—By history of possible infection, bacterial examination of the wound secretion, or intraperitoneal injection of this or its culture into a male guinea-pig. If positive, orchitis results in two days.

Treatment.—Excision of the wound, antisepsis, opening other local lesions. Symptomatic treatment.

ACTINOMYCOSIS

Definition.—A chronic infectious disease chiefly of cattle.

Etiology.—The actinomyces or ray fungus, which forms translucent to opaque, grayish to yellow granules composed

of radiating filaments with bulbous ends. Probably ingested with the food.

Pathology.—Around the parasite are round, epithelioid and giant cells, surrounded later by masses of connective tissue. These large masses may suppurate or become necrotic or calcareous. In the lungs the lesion is an acute bronchitis or bronchopneumonia with production of new tissue.

Symptoms.—(1) *Alimentary canal.* There may be swelling of the face or sarcoma-like tumor of the jaw, or the intestines or liver may be involved. Chronic. (2) *Pulmonary actinomycosis:* Cough with mucopurulent, sometimes fetid, sputum, irregular fever, loss of weight. Resembles pulmonary tuberculosis and fetid bronchitis with septic symptoms, and usually ends in death in about ten months. (3) *Cutaneous:* Tumors which suppurate, forming chronic ulcers. Resemble tuberculous lesions. (4) *Cerebral:* Rare. Symptoms of brain tumor or epilepsy.

Diagnosis.—Microscopic examination of pus for the fungus.

Treatment.—Incision, irrigation, etc. Potassium iodide has been successful. In the pulmonary form, supportive and symptomatic.

SYPHILIS

An acquired or hereditary infectious disease caused by the Spirochete pallida.

Medically we are concerned chiefly with:

1. **Syphilis of the Lung.**—Rare. (*a*) *White pneumonia of the fetus,* a white hepatization from diffuse thickening and infiltration of the alveolar walls and filling of the air cells with desquamated epithelium. (*b*) *Gummata* scattered through the lungs. (*c*) An *interstitial pneumonia,* not universally recognized.

2. **Syphilis of the Liver.**—(*a*) *Diffuse hepatitis,* especially congenital. Liver hard, with grayish points of new tissue. The new connective tissue is interlobular or intercellular.

(*b*) *Gummata*, pale gray nodules, size of a pea to that of a hen's egg, becoming cheesy except at the periphery. Contraction of the resulting cicatrices causes deformity of the liver.

Symptoms.—The symptoms may be: (*a*) Symptoms of cirrhosis of the liver with ascites (see Atrophic Cirrhosis, p. 193) and irregularity of the liver; (*b*) anemia with enlarged (amyloid) liver and spleen, increased urine with albumin and casts, dropsy; or (*c*) localized liver tumor. Confirm diagnosis by Wassermann reaction and therapeutic test.

3. **Syphilis of the Arteries.**—(*a*) *Obliterating endarteritis* from proliferation of the intima; (*b*) *gummata* in the adventitia. Presence of gummata or other syphilitic lesions and a positive Wassermann reaction verify the specific origin of an endarteritis.

Symptoms.—(For symptoms see Arteriosclerosis, p. 278.)

Treatment.—Potass. iodide, gr. x, t. i. d. p. c., increasing gradually to gr. xxx or more p. c. In syphilitic cirrhosis of the liver with ascites a pill containing calomel, digitalis, and squill, āā gr. j, aids the cure and also diuresis. Inunction of ung. hydrarg., ʒj q. n., or a mercurial preparation such as hydrarg. iod. rubrum, gr. $\frac{1}{32}$ to $\frac{1}{10}$, t. i. d. p. c. by mouth, or the bichloride or salicylate intramuscularly. Continue treatment for two years. A single dose of salvarsan intravenously may cause rapid disappearance of spirochetes and lesions; but several doses should be given.

GONORRHEA

The chief complications of medical interest are:

1. **Gonorrheal Septicemia and Pyemia.**—The symptoms are those of a severe general septic infection with a local gonorrheal suppurative focus in the genito-urinary tract.

2. **Gonorrheal endocarditis,** usually following joint involvement. (See Malignant Endocarditis, p. 253.)

3. **Gonorrheal Arthritis.**—More common in men than in women, occurring during, at the end of, or after a urethritis. It is usually polyarthritic and often involves such joints as the temporomaxillary and sternoclavicular, thus differing from acute articular rheumatism. The gonococcus may be found in the inflamed area or exudate. The inflammation is frequently periarticular and with tenosynovitis; the effusion is usually serous. The only **symptom** may be variable joint pains. The attack may resemble an acute articular rheumatism of one joint, or a subacute rheumatism of one or more. There is sometimes a chronic monarticular arthritis, usually of the knee. The tendon sheaths and bursæ may be involved alone or with joints. Gonorrheal septicemia may result from the arthritis. The course of a gonorrheal arthritis is protracted. Iritis is the most common **complication**; endocarditis and pericarditis are less frequent.

Treatment.—The urethral source of infection must be cured. Gonococcic vaccine (50,000,000 or 100,000,000 bacteria daily) may cure or improve many cases. Good results are claimed for antigonococcic serum. In the acute stage the pain may be relieved by joint immobilization or use of an ice-cap; in the chronic, hot-air baths, massage, and passive motion are useful. Tonics such as iron, arsenic, and strychnine are important. Methyl salicylate locally or the Paquelin cautery may diminish pain.

TUBERCULOSIS

Definition.—An infectious disease caused by the Bacillus tuberculosis and characterized by the formation of nodules or diffuse masses of new tissue.

Etiology.—*Predisposing:* Man, fowls, and cows are chiefly affected. Indians, negroes, and Irish are very sus-

ceptible. The disease is less common at great altitudes. Dark, poorly ventilated rooms, such as tenements and factories, and the crowding of cities favor infection, as do indoor life and occupations in which much dust is inhaled. Certain affections, such as measles, pertussis, chronic arterial, cardiac, renal, and hepatic diseases, and inflammations of the respiratory tract, are predisposing factors. *Exciting:* The Bacillus tuberculosis, a long, narrow, straight, or slightly curved bacillus, rarely branched, often staining in an irregular, beaded manner. The bacilli are found in tuberculous lesions and discharges, and in dust contaminated by sputum. Hereditary transmission is rare, inoculation infrequent; genital infection through coitus may occur. Milk of tuberculous animals may infect the lungs without causing intestinal lesions. Inhalation is the chief mode of transmission. Traumatism favors local tuberculous infection.

Pathology.—The most distinctive lesion is the *tubercle*, a structure which is, however, not characteristic of the disease but may originate under the influence of other germs. The bacilli multiply rapidly at the site of infection or the places to which they are carried by the lymphatics or bloodvessels. In adults this is most often the lungs; in children, lymph nodes, bones, or joints. The fixed connective tissue and endothelial cells in these regions proliferate, and there is an exudation of leukocytes around the foci. A stroma is formed from the connective-tissue cells. Giant cells arise through subdivision of the nuclei alone in growing cell bodies, or through fusion of adjacent cells. No new bloodvessels are formed. The tubercles are irregularly rounded, gray, and translucent. The action of the bacteria or their toxins and the lack of bloodvessels may cause coagulation necrosis beginning at the center of the tubercle and spreading outward, the necrotic region becoming a homogeneous mass filled with bacilli. This cheesy mass is now yellowish gray. Dense fibrous tissue may permeate and enclose the tubercle, resulting in its encapsulation or complete sclerosis. Calcifi-

cation may result from deposition of lime salts. To summarize: cell proliferation, inflammatory exudation, caseation, and sclerosis. The last two are respectively destructive and conservative processes. *Diffuse tuberculous inflammation* is characterized by the coalescence of many tuberculous foci forming large masses of proliferated cells, which undergo caseation, or by extensive production of inflammatory exudate around the foci with rapid and widespread coagulation necrosis. Suppuration in tuberculous lesions is usually the result of mixed infection with pyogenic microörganisms.

1. **Acute Miliary Tuberculosis.**—(*a*) **Acute General Miliary Tuberculosis.**—In this the presence in the blood of large numbers of tubercle bacilli leads to the formation of many tubercles throughout the body.

Symptoms are those of a severe general infection. There is usually a *prodromal period* with fever, loss of appetite and strength, as in typhoid, which it often closely resembles; more rarely the *onset* is sudden. The evening temperature is usually 103° or 104°, the morning 100° to 102° or less. Its course is very irregular. It may be low throughout. The pulse is rapid. There is usually some bronchitis, but pulmonary symptoms are not marked, and there may be no physical signs of pulmonary involvement. The spleen may be enlarged. The tongue becomes dry and brown, the pulse more frequent and feeble, with emaciation, delirium, or more often stupor, passing into coma. Death occurs in a few days or weeks.

Diagnosis.—By general typhoidal symptoms and occasionally by signs of pulmonary lesions. (1) From **typhoid fever**, by the irregular temperature, more frequent respiration, rapid pulse, later and less marked splenic enlargement, absence of rose spots, and occurrence of leukocytosis which is not found in uncomplicated cases of typhoid. Typhoid bacilli are not found in blood cultures, and tubercle bacilli may abound in fluid from a lumbar puncture; no Widal reaction is obtained. Tubercles may be seen in the

choroid. (2) From **malignant endocarditis**, in which there may be signs of cardiac disease. (See Diagnosis, p. 253.)

(*b*) **Acute Miliary Tuberculosis, Pulmonary Type.—Symptoms.**—In children this often follows measles or whooping-cough; in adults it is frequently preceded by protracted cough. The *invasion* is like that of acute bronchitis. Symptoms are cough, mucopurulent sputum, occasionally hemoptysis, marked dyspnea, and cyanosis; temperature 102° or 103°, and rapid and feeble pulse. **Physical signs** are those of a general acute bronchitis with sometimes those of a complicating pleurisy, or in children of bronchopneumonia; enlarged spleen in acute cases. Death occurs in from ten days to several months.

Diagnosis.—By previous history, symptoms of acute bronchitis with marked dyspnea and cyanosis, sometimes tubercle bacilli in the sputum and tubercles in the choroid. The *x*-rays may show areas of shadow where physical signs fail to reveal the presence of pulmonary lesions.

(*c*) **Tuberculous Meningitis, Basilar Meningitis, or Acute Hydrocephalus.—Etiology.**—Usually in children under five, often following old tuberculous lesions of the bronchial or mesenteric lymph nodes.

Pathology.—There may be miliary tubercles on the inner surface of the dura. The pia mater may be congested and dry or edematous, with clear or fibrinopurulent serum. Many small miliary tubercles or a few large tubercles are found in any or all regions of the pia, especially at the base of the brain. In children, especially, the convolutions may be flattened from distention of the ventricles with clear or turbid serum, their walls being covered with miliary tubercles. The lesions may involve the meninges of the cord. There is usually a generalized tuberculous infection.

Symptoms.—In children tuberculous meningitis is usually part of an acute general infection following bronchopneumonia, measles, or pertussis; in adults, generally secondary to a local pulmonary lesion. There is usually a *prodromal*

period in which there are poor appetite, loss of flesh, irritability, and restlessness. The *invasion* is marked by headache, projectile vomiting, and fever; less often by a convulsion in children, in adults by a chill. The headache is extreme, often causing a short cry, the hydrocephalic cry. The temperature is irregular and may reach 103°; pulse slow and irregular. There are symptoms of irritation of the different cranial nerves and cortical centers—contracted pupils, strabismus, nystagmus, muscular twitchings, rolling and burrowing of the head in the pillows, great restlessness. Delirium alternates with stupor. The child is apathetic, vomiting subsides, abdomen and head are retracted, and there is constipation. Pulse becomes rapid and feeble, temperature irregular. Convulsions, twitching, and other signs of nerve irritation gradually give way to those of paralysis of ocular and other muscles, including ptosis and dilatation of the pupils. A typhoid state with subnormal or high temperature may end in death between the second and fourth weeks. Severe cases may prove fatal in a few days. In children distention of the lateral ventricles is common, and so signs of compression—stupor, etc.—preponderate and other cerebral symptoms are less marked.

Diagnosis.—By history of a previous tuberculous focus, cerebral symptoms, presence of tubercle bacilli in the turbid fluid from lumbar puncture. Kernig's sign is usually obtainable. (See Cerebrospinal Fever, p. 52.) Cerebrospinal fluid increased, clear or slightly turbid; cells chiefly lymphocytes; tubercle bacilli may be found in the sediment or by inoculation of a guinea-pig. From **other types of meningitis** (see p. 52).

Prognosis.—Cases which recover are probably not tuberculous.

Treatment.—Ice-cap to the head; repeated lumbar puncture; sponging for fever; chloral or opium for pain.

2. **Tuberculosis of the lymph nodes** was formerly known as scrofula. The bacilli causing this form of tuber-

culosis are probably of attenuated virulence. An important predisposing factor is diminution of the resisting power of the natural barriers, as in tonsillitis and catarrhal inflammation of the mucous membranes, especially in nasopharyngitis enteritis, measles, and pertussis. Rarely the glandular affection is general and the case resembles Hodgkin's disease. It usually is localized in the cervical, mediastinal or mesenteric nodes, most commonly the cervical.

(*a*) *Cervical.*—Common in poorly nourished children living in badly ventilated or crowded rooms, and in the negro. Chronic nasopharyngeal catarrh and tonsillitis predispose. The submaxillary nodes are usually first involved. They are enlarged, smooth, firm, and generally become matted together. Later the skin may become adherent and suppuration occurs, the resulting abscess breaking through the skin and leaving an obstinate sinus. The posterior cervical glands may be involved also, or the axillary, supraclavicular, and bronchial. There is usually secondary anemia. A protracted course and spontaneous recovery are common. Pulmonary or general miliary tuberculosis may occur.

(*b*) *Mediastinal.*—The tracheobronchial nodes are often infected through the respiratory tract. The enlarged nodes may cause symptoms by compression of adjacent blood-vessels; by extension of the infection to the lungs, pleura, or pericardium; by rupture of a caseous node into trachea, bronchus, or esophagus; by general infection through the circulation.

(*c*) *Mesenteric.*—The symptoms are chiefly loss of flesh and strength, anemia, abdominal distention and tympanites with offensive diarrheal stools. The enlarged mesenteric and retroperitoneal nodes can sometimes be felt. Tuberculous peritonitis is often present.

Diagnosis from **Hodgkin's disease** (p. 290).

Treatment.—General hygienic measures; fresh air, nutrition, cod-liver oil. If localized, excise the infected nodes.

3. **Tuberculous pleurisy** may be primary or secondary to pulmonary tuberculosis. It may be acute at the outset, becoming chronic or recovering, or run a severe suppurative course. More often it is subacute or chronic, either dry or with serofibrinous or purulent effusion or chronic adhesions.

Symptoms and Physical Signs.—(See Fibrinous, Serofibrinous, Purulent, and Chronic Pleurisy, pp. 236 to 241.)

Treatment.—Constitutional treatment for tuberculosis (p. 102): local as for other forms of pleurisy (*q. v.*).

4. **Tuberculous pericarditis** may give no symptoms, may resemble an acute pericarditis with effusion, or result in an acute general infection or chronic adhesive pericarditis (p. 250).

5. **Tuberculous Peritonitis.—Etiology.**—Primary or secondary to tuberculosis of intestines, lungs, or Fallopian tube; is most frequent in males and between twenty and forty years, though occurring at all ages.

Pathology.—Miliary tubercles in the peritoneum or diffuse tuberculous tissue which may ulcerate or become fibrous. There may be a serous, serofibrinous, purulent, or hemorrhagic exudate which may be encapsulated by adhesions between the visceral and the parietal peritoneum. The adherent intestines or rolled up and matted omentum may form hard masses.

Symptoms are extremely variable. They may be entirely or almost absent. An attack may begin like acute peritonitis with sudden onset of high fever, abdominal pain, tenderness, tympanites, vomiting, and constipation, these symptoms passing into those of chronic peritonitis. In some cases symptoms are like those of subacute miliary tuberculosis, without focal signs. Often there are gradual loss of flesh and strength, low and irregular fever, frequently subnormal temperature, moderate ascites, tympanites, constipation, diarrhea, and abdominal masses. These may consist of the omentum rolled up and matted into a sausage-shaped tumor in the upper part of the abdomen, of thickened and adherent

coils of intestine, enlarged mesenteric lymph nodes, or sacculated collections of exudate. Spontaneous recovery may occur or the course of the disease may resemble that of a malignant neoplasm.

Diagnosis.—(1) Of cases with sacculated exudate, from **ovarian cyst,** by history and physical signs of other tuberculous lesions, irregular fever, gastrointestinal symptoms, and lack of the definite outline of a cyst. (2) From **malignant newgrowths,** by the youth of the patient, better nutrition considering the size of the tumor, and sometimes history and physical signs of tuberculosis in other regions.

Treatment.—In cases with effusion and few adhesions, laparotomy and removal of fluid may lead to cure. In other cases, surgery is ineffectual, and feeding, cod-liver oil, and symptomatic measures only are indicated.

6. **Tuberculosis of the Larynx.** (See volume *Nose and Throat.*)

7. **Acute Pneumonic Pulmonary Tuberculosis** (*Galloping Consumption*).

(1) **Pneumonic Type.—Pathology.**—Large areas or an entire lobe or lung solidified by an exudate of epithelium, pus, and fibrin, with miliary tubercles. Necrosis may occur, with formation of large cavities.

Symptoms.—Sudden *invasion* with chill, fever, pain in the chest, cough, mucous sputum. The case resembles acute lobar pneumonia. There are marked dyspnea, and rusty sputum, containing in some cases tubercle bacilli. Instead of the crisis, the general condition becomes worse, the pulse rapid, sputum greenish. The **physical signs** are dulness, increased fremitus, diminished and later bronchial breathing and voice and subcrepitant rales over a lobe or lung. Death may occur in two or three weeks or several months, or the disease may become chronic.

Diagnosis.—By microscopic examination of sputum for tubercle bacilli and elastic tissue, a history of previous pulmonary trouble, and irregularity of temperature.

(2) **Acute Tuberculous Bronchopneumonia.—Pathology.**—The bacilli enter the smaller bronchi by inhalation, by aspiration from an existing tuberculous lesion at the apex or bronchial lymph nodes, or by the bloodvessels or lymph vessels. Cut section of lung shows small gray or yellowish-white areas of consolidation around the small bronchi. The walls of these bronchi show catarrhal inflammation and coagulation necrosis, with exudate in the surrounding air spaces. The areas of consolidation coalesce, forming a grayish-white mass or areas of gray or yellowish-gray color surrounded by dark red. Necrosis may result in formation of cavities.

Symptoms.—In adults the *invasion* is usually sudden, with chills, fever, cough, mucous sputum, pain in the chest, rapid pulse and respiration, and some loss of flesh and strength. Physical signs of areas of consolidation gradually appear, especially at the apices—dulness, harsh breathing, and subcrepitant rales. The presence and extent of these areas may be shown by the *x*-rays. The sputum may contain tubercle bacilli. Symptoms of an asthenic fever (p. 20) may follow and end in death; or caseation and breaking down may occur; or the disease become chronic. In children, usually after pertussis or measles, there is sudden onset of signs of acute bronchitis, with gradual appearance of areas of consolidation. Death occurs within a few days or weeks, or the case becomes chronic.

8. **Chronic Ulcerative Pulmonary Tuberculosis.—Pathology.**—*Bronchial nodes* usually contain miliary tubercles and caseous foci. The *pleura* is usually thickened and adherent. If the pleurisy is tuberculous, miliary tubercles and caseous areas are present. There is often a serous, purulent, or hemorrhagic effusion. Pyopneumothorax is not uncommon. The lesions in the *lungs* are most often found near the apices of the upper lobes. They include (*a*) *miliary tubercles*, in the smaller bronchi or air spaces if the bacilli have been inhaled, in the bloodvessel walls if carried by the circulation, around the primary foci if disseminated by lymphatics; (*b*)

tuberculous bronchopneumonia beginning in the smaller bronchi, with tendency to coalescence of various foci, caseation, cavity formation by rapid caseation or ulceration of the bronchial wall, and formation of connective tissue around the tuberculous foci, more or less completely encapsulating them; (*c*) *exudative inflammation* around the tuberculous areas, the air spaces being filled by masses of epithelioid cells. This consolidated region is red, and if these cells are fatty it is yellowish white. (*d*) *Cavities:* The walls of fresh cavities are formed of caseous and necrotic masses. Many of the older cavities have thick walls of granulation tissue which constantly produce pus. They are crossed by bloodvessels which may still be permeable and from which hemorrhages may occur if they are eroded. These cavities contain purulent material, tubercle bacilli, and other bacteria. Chronic ulcerative pulmonary tuberculosis commences as a simple tuberculous process, but a secondary mixed infection with pyogenic organisms usually occurs. Small cavities may be walled in by connective tissue which converts the surrounding portion of the lung into a solid mass. Tuberculous lesions of other organs are often present, especially in the intestines and larynx. There is frequently amyloid degeneration of the liver, spleen, and kidneys.

Symptoms.—Chronic ulcerative pulmonary tuberculosis may be preceded by an acute pulmonary tuberculosis or tuberculosis of lymph nodes. It may become far advanced with practically no symptoms. The invasion may be marked by hemoptyses; by chills, fever, and sweating, suggesting malaria; by symptoms and signs of dry pleurisy; by those of laryngeal tuberculosis; or by dyspeptic and anemic symptoms with afternoon fever. Usually the onset is like that of acute bronchitis followed by protracted cough. The cough is at first dry and hacking, later accompanied by profuse expectoration. It is often severe during the night; also in the morning, after cavities have formed. There may be little or no cough in advanced cases. If the larynx is involved

the cough is husky. The sputum is usually scanty and mucous at first and may be absent for a long time; later it becomes profuse and mucopurulent, and after cavity formation is nummular, that is, in rounded, flattened, heavy, greenish-gray masses. It may contain tubercle bacilli at any time, especially in the later stages, when elastic tissue from erosion of bronchi, alveoli, or bloodvessels is also present. Hemoptysis may occur at any period and does so in 60 to 80 per cent. of the cases. It is usually slight at first and generally recurs. The later hemorrhages may be large. The blood is ordinarily bright red, frothy, and mixed with mucus. Dyspnea is usually not marked. Pain in the chest may be due to dry pleurisy or to coughing. There is remittent fever with a drop of 2° or 3° toward morning, or an intermittent or hectic fever in which there is a rapid fall to subnormal in the early morning accompanied by sweating and with a rapid afternoon rise. The intermittent fever and night-sweats are associated with mixed infection and necrosis. The temperature should be taken at least at 8 A.M. and 5 and 8 P.M. in doubtful cases. The pulse is frequent, full, and soft. There are progressive loss of weight and strength, anemia, anorexia, and sometimes nausea, vomiting, and gastric pain. Diarrhea often occurs as a terminal symptom, and is the result of tuberculous or catarrhal colitis. The urine is not characteristic. There may be complicating tuberculous laryngitis, nephritis, meningitis, or peritonitis, dry pleurisy or one with effusion, pneumothorax or pyopneumothorax, emphysema, pneumonia, or gangrene of the lung.

Physical Signs.—The chest is often long and narrow with winged scapulæ. There may be retraction above and below the clavicles and diminution of expansion and of diaphragmatic excursion (Litten's sign) on the affected side. Fremitus increased over consolidated areas; diminished or absent over thickened pleura or effusions. Relative or absolute dulness over infiltrated areas. Breath sounds feeble at first; later, harsh breathing with prolonged and high-pitched

expiration, becoming bronchial and with bronchial whisper, as consolidation increases. Subcrepitant rales, becoming moist as softening occurs. Signs usually appear first at one apex. Consolidation over a large bronchus simulates a cavity—tympanitic percussion note, cavernous or tubular breathing, and loud rales. Over cavities there are increased fremitus; dulness; tympanitic, amphoric, or cracked-pot resonance; modified bronchial, cavernous, or amphoric breathing; bronchophony, pectoriloquy, gurgling rales; occasionally succussion sound and coin sound over large cavities. Pulmonic second sound accentuated; pulse frequent, full, soft.

Course.—Patients may recover with cavities remaining as a permanent loss of lung tissue. The disease may progress slowly for years, and death occur from an acute exacerbation or extension to other organs. The immediate cause of death is usually asthenia, hemorrhage, general miliary tuberculosis, or lobar pneumonia. The hope of recovery is characteristic of even advanced cases of tuberculosis.

Diagnosis.—By presence in sputum of tubercle bacilli and elastic fibers, increased frequency of pulse, occurrence of afternoon fever, loss of flesh and strength, cough; physical signs usually first at apices. X-ray examination shows extent and multiplicity of areas of infiltration.

9. **Chronic Miliary Tuberculosis.**—A form of chronic pulmonary tuberculosis is sometimes described in which the chief **lesion** is the miliary tubercle, which occurs in large numbers, is very dense, contains few tubercle bacilli, and is accompanied by little exudation or necrosis. The tubercles may be confined to one or both apices or scattered throughout the lungs. There is little consolidation except that due to connective-tissue formation, so **physical signs** may be absent or those of small cavities formed by dilatation of bronchi with little necrosis. The **symptoms** are like those of chronic ulcerative tuberculosis without those due to a mixed infection. There is often a chronic bronchitis. Loss of flesh and strength are out of proportion to the apparent

insignificance of the pulmonary lesions. The sputum is often scanty and contains few tubercle bacilli.

10. **Tuberculosis of the Alimentary Canal.**—Tuberculosis may attack all parts of the digestive tract, especially the intestines. There it is secondary to pulmonary or peritoneal involvement or primary in the mucosa. The primary form is most common in children; the secondary often accompanies chronic pulmonary tuberculosis. The tubercles are in the Peyer's patches, solitary follicles, and mucosa. The ulcers are frequently ovoid, with their long axis extending transversely to that of the intestine. The subperitoneal lymphatics are often thickened. The **symptoms** are diarrhea or constipation, some abdominal pain, irregular fever, and sometimes intestinal hemorrhage. There may be physical signs of complicating tuberculous peritonitis or mesenteric lymphadenitis.

Death occurs from exhaustion, hemorrhage, or from peritoneal infection after perforation.

11. **Tuberculosis of the Brain.**—In addition to acute tuberculous meningitis, which in children is often accompanied by acute hydrocephalus, there may be a chronic type involving both brain and meninges. In other cases the tubercles are large and relatively few. Though known as solitary tubercles, these are usually multiple. They are most frequent in the cerebellum or cerebrum. The symptoms are those of brain tumor. (See *Diseases of the Nervous System*, this series.)

Diagnosis of Tuberculosis.—By symptoms, physical signs, gross appearance of lesions, microscopic examination, animal inoculations, tuberculin reactions, and *x*-rays. Inoculation of tissue or fluids suspected of containing tubercle bacilli into guinea-pigs causes tuberculosis in the latter if they are present. Tuberculin reactions cause local hyperemia if present—0.5 per cent. tuberculin in conjunctiva (Calmette), in abrasions of skin (von Pirquet), by inunction (Moro). A positive reaction in a young child

is of diagnostic value, a negative is inconclusive. In adults the reaction only shows a tuberculous infection of some part of the body at some time. Subcutaneous tuberculin injections are employed in the diagnosis of obscure cases. One milligram is given hypodermically, and if no reaction occurs two or three milligrams are given a few days later. In positive cases a rise of temperature of several degrees, perhaps to 102° or 104°, occurs within ten to twelve hours, with exacerbation of symptoms and local signs. The *x*-rays may show areas of partial consolidation which give no physical signs, or show involvement of the other apex when not suspected.

Prognosis of Tuberculosis.—There is a marked tendency to spontaneous cure, especially in disease of the bones and lymph nodes. An acute onset, repeated hemoptyses, rapid and extensive consolidation, great loss of weight and strength are unfavorable signs in pulmonary cases. Chronic cases usually last several years, acute often only a few weeks. Unhygienic surroundings and repeated pregnancies tend to prevent recovery. Cerebral cases are fatal; laryngeal, serious.

Treatment of Tuberculosis.—*Prophylaxis:* Destruction of all tuberculous sputum by heat or bichloride, exposure of patient's rooms to air and sunlight, inspection of dairies and slaughter houses and their products. Mothers with pulmonary tuberculosis should not nurse their infants.

General.—Fresh air at all seasons and hours, sunlight, rest until acute symptoms have disappeared, and liberal feeding. (*a*) *Fresh air* at home is secured by constantly open windows with an exposure admitting sunlight, and extra clothing if the weather necessitates. If possible, the patient should be out of doors for many hours each day. Far-advanced cases should not be sent away from home. Others who can do so should seek a suitable climate. This must be chosen with reference to the patient's idiosyncrasy, not by any fixed rules. Resorts at a moderate altitude, such as the Adirondacks, the neighborhood of Liberty, N. Y., and

Asheville are usually preferable, as patients can subsequently return to lower levels with safety. While lacking this advantage, high altitudes, such as Colorado, Arizona, and New Mexico, are very favorable in cases with limited consolidation, no large cavities, little or no hemoptysis, and no great emaciation. The climate of Arizona is especially suitable for cases of bronchitis with profuse sputum. Cases with involvement of both lungs and large cavities should be in a warm, dry climate, such as Southern California or the South Atlantic States. (*b*) *Rest:* All exertion should be avoided until acute symptoms disappear. Those with more than slight fever, hemoptysis, or great weakness should remain in bed in the open air. (*c*) *Overfeeding* until weight is slightly above normal, if this does not disorder digestion. One to three quarts of milk a day; raw eggs, at first one three times a day, increasing to three or more times that number, and red meats at each meal should be taken if digestion permits. If the appetite is poor and the stomach will not retain much food, small quantities of milk, koumyss, beef juice, etc., are administered frequently, adding eggs and meat later.

Medicinal.—All drugs should be avoided if found to interfere with digestion. Cod-liver oil, ʒj to ij, or its emulsion, ʒij to ʒss, t. i. d. p. c., is of great value in improving nutrition, if well digested. In some cases an emulsion of mixed fats is better borne. Cream or olive oil may be substituted.

Of Symptoms.—For cough and profuse expectoration, creosote, ♏j p. c., in capsule or in cod-liver oil, increasing gradually if tolerated by the stomach; or creosote, chloroform, alcohol, equal parts, by inhalation; or guaiacol. For temporary relief of cough, heroin, gr. $\frac{1}{20}$ to $\frac{1}{12}$, or codeine, gr. ¼, alone or combined with dilute hydrocyanic acid, ♏j to ij, and syrup of wild cherry, ʒj, given t. i. d. or less often. A certain amount of coughing is necessary to remove the sputum; but a dry or harassing cough which disturbs sleep and violates unnecessarily the principle of immobilization of tuberculous areas should be checked. For fever,

rest in bed, if at all high; cold sponging if necessary; antipyrin, acetanilid, or phenacetin only when this fails. For sweating, atropine, gr. $\frac{1}{120}$ to $\frac{1}{60}$; extract of belladonna, gr. $\frac{1}{8}$ to $\frac{1}{2}$; aromatic sulphuric acid, ♏x to xx, or picrotoxin, gr. $\frac{1}{100}$ to $\frac{1}{50}$. For large hemoptyses, absolute rest in bed. While bleeding is profuse, coughing must be permitted to prevent filling of the air passages with blood; if slight or arrested, morphine should be given in sufficient doses (Magendie's solution, ♏v to viii hypodermically) and sufficiently often to prevent recurrence due to coughing or hard breathing. Purgation to lessen blood-pressure in cases of persistent hemorrhage. Ice-bag over the chest is of uncertain value. For small hemorrhages from mucous membranes it is necessary only to keep fairly quiet. To increase coagulability of blood, calcium lactate, gr. xv to xx, every four hours. For pleuritic pain, strapping the chest, hot applications over the site of pain, or codeine. For diarrhea: bismuth, castor oil, or combination of opium, acetate of lead, and camphor. As tonics and for anemia, iron, arsenic, and strychnine.

Mechanical.—Immobilization of the affected lung by injection of the pleural cavity with nitrogen has been tried. In general, relative immobilization by avoiding exertion and coughing is advisable.

LEPROSY

Definition.—Leprosy is a chronic infectious disease caused by the Bacillus lepræ. It is of two types—tubercular, in which there are nodules in the skin and mucous membranes; anesthetic, with nerve lesions.

Etiology.—The lepra bacillus resembles the tubercle bacillus. It seems to be communicable to others at any age, but only by close contact, probably through the saliva, sputum, or nasal secretion.

Pathology.—The tubercles in the skin consist of a fibrous stroma containing masses of cells of variable form, in and between which are many of the bacilli. These nodules may become necrotic and form ulcers. In the anesthetic form the bacteria within the nerve sheaths cause a peripheral neuritis.

Symptoms.—1. *Tubercular leprosy* begins with areas of erythema of the skin, often hyperesthetic and distinctly limited, later becoming pigmented. The areas frequently become anesthetic and absolutely white. The eyelashes and other hairs on the face fall out; there may be keratitis. Cutaneous nodules appear. The mucous membranes of the upper part of the respiratory tract are attacked; the voice becomes harsh and is gradually lost. Death may occur from inhalation pneumonia or laryngeal involvement.

2. *Anesthetic leprosy* begins with pain and areas of hyperesthesia or numbness in the extremities. The anesthetic areas appear on the body and extremities, preceded or not by a macular eruption. Trophic changes in the affected areas are common. There are bullæ, often followed by ulceration; sometimes necrosis causing the loss of portions of the fingers and toes. The nerve trunks are enlarged and nodular. The disease may progress slowly for many years.

Diagnosis.—By areas of erythema with anesthesia or hyperesthesia, and by microscopic examination of nodules for the bacilli.

Treatment.—*Prophylaxis.*—Isolation. *Of the disease:* No known positive cure. Vaccines, Calmette's antivenene injections, gurjun and chaulmoogra oils are recommended.

ACUTE FEBRILE JAUNDICE (WEIL'S DISEASE)

This is an acute infectious disease, attacking most frequently males from twenty-five to forty, especially butchers.

The **invasion** is sudden, usually with a chill, pain in the head, back, and sometimes the extremities, and remittent fever. There is soon jaundice accompanied by swelling and perhaps tenderness of the liver, enlargement of the spleen, albuminuria, and sometimes delirium and coma. The duration is generally ten to fourteen days. Usually recovery.

Treatment.—The treatment is the same as that of acute catarrhal jaundice (p. 183).

SECTION II

DISEASES CAUSED BY ANIMAL PARASITES

ASCARIASIS

1. **Ascaris lumbricoides,** or **roundworm,** resembles the angleworm in form. It is yellowish brown. The male is 10 to 20 cm. long; the female, 17 to 30 cm. The ova are small, elliptical, and brownish red. Usually one or two worms in a patient, occasionally many. Found ordinarily in the upper part of the small intestine, they may enter any part of the gastro-intestinal or respiratory tract or the gall ducts and liver. They are most common in children, in whom they may cause picking of the nose, grinding of the teeth, restlessness, or even convulsions, or their presence in the stools may be the only indication of their existence.

Treatment.—Santonin in doses of gr. ij to iij for adults, and gr. ss to j for children, once or twice a day for two or three days, followed by salts or calomel.

2. **Oxyuris vermicularis, threadworm** or **pinworm,** is a small round worm, the male 4 mm., the female 10 mm. in length. They live in the rectum and colon, and may migrate at night, sometimes entering the vagina. The worms are most common in children, and are probably acquired through infected water or lettuce. They cause great itching, particularly at night, when they emerge from the anus. The itching may cause restlessness, loss of sleep, and consequently anemia.

Treatment.—Reinfection by ova in the feces must be avoided. Santonin or cathartics may be used, but large enemata containing salt, quassia, vinegar, or other drugs are usually most effective. For itching, belladonna ointment.

TRICHINIASIS (TRICHINOSIS)

Etiology and Pathology.—The disease is caused by the Trichina spiralis, a parasite introduced into the body by eating imperfectly cooked flesh of infected hogs. The encysted larvæ in the hog's muscles are liberated from their capsules by digestion, enter the intestine, and by the sixth or seventh day reach the adult size and sexual maturity. The female is 3 or 4 mm., the male 1.5 mm. long. Within seven to nine days after ingestion the female burrows through the abdominal wall and deposits several hundred embryos in the lymph spaces. They pass by the bloodvessels to the intermuscular connective tissue, and thence into the voluntary muscle fibers. Here they cause an interstitial myositis and become enclosed individually or in groups of two to four in translucent ovoid capsules, in which they lie coiled. The length of the larvæ is ½ to 1 mm. The capsules become calcified within a few months and the larvæ remain alive for years. These calcified capsules appear as white specks in the muscles. The mesenteric lymph nodes are enlarged.

Symptoms.—A few days after ingestion of the infected meat there may be *prodromal* general gastro-intestinal disorder, loss of appetite, vomiting, diarrhea, abdominal pains, and a sense of fatigue. The *invasion* from the seventh to the tenth day, or occasionally after several weeks, is marked by fever, with a chill in some cases, and pain, swelling and tenderness of the voluntary muscles involved. The extremities are held in positions which cause the least tension upon the muscles. If the diaphragm and intercostals are affected, there is great dyspnea, while involvement of the muscles of the pharynx and larynx causes difficulty in eating

swallowing, and speaking. There is early edema of the face, especially the eyelids, later of the extremities. Profuse sweating, tingling and itching of the skin, and various eruptions may occur. Temperature normal, or may reach 102° or 104°, rarely higher. It is usually intermittent or remittent. There is leukocytosis, often over 30,000, of which one-half may be eosinophiles. There are emaciation and anemia. In severe cases the appearance may be like that in the third week of typhoid. In mild cases the fever and muscular symptoms subside in ten to fourteen days, in others only after two or three months. The mortality, from 1 to 30 per cent., seems to depend upon the virulence and number of parasites.

Diagnosis.—By the history of eating raw pork, by the occurrence of muscular pains, swelling and tenderness, and edema of the face; by the occurrence of leukocytosis and eosinophilia; by examination of the infected meat or the stools, or of a section of the patient's muscle.

Differential Diagnosis.—(1) From **typhoid**, by muscular pain and swelling, edema, leukocytosis, and eosinophilia, and by absence of Widal reaction and enlarged spleen. (2) From **acute articular rheumatism**, by occurrence of the local symptoms in the muscles instead of joints, and by blood signs. (3) From **ptomain poisoning**, by more delayed and less severe gastro-intestinal symptoms. (4) From **cholera morbus**, by less marked diarrhea and absence of general symptoms of trichinosis.

Treatment.—*Prophylaxis.*—Care in feeding hogs, inspection and thorough cooking of pork. *General:* Purgation, especially in the early stages while the stools contain parasites. *Symptomatic:* Heat to the painful muscles, hypnotics and tonics.

ANKYLOSTOMIASIS

Synonyms: Tunnel, Mountain, Brickmaker's or Miner's Anemia, Egyptian Chlorosis, Hookworm Disease.—Etiology.—

It is caused by the Ankylostoma duodenale, called also Uncinaria, Dochmius, or Strongylus duodenalis. It is a common disease in the Southern States, where it is due to an allied parasite, Uncinaria americana. This is an almost cylindrical worm, 6 to 18 mm. long. The mouth has a set of hooks by which it attaches itself to the mucosa of the upper parts of the small intestine to suck blood. The eggs are oval. They may mature in the soil and become encysted. They are ingested in drinking water or by dirt eating, or by penetration of the skin of bare feet by larvæ in infected soil. The **symptoms** are gastro-intestinal disturbances followed by gradually or rapidly developing anemia; skin yellowish or pale, weakness, palpitation, rapid pulse, eosinophilia, ova and blood in stools. In rapid cases, dyspnea and edema. It is **differentiated** from other anemias, especially malarial cachexia, by rapid development of anemia, occurrence of gastro-intestinal disturbances, and finding of ova in the stools.

Treatment.—*Prophylaxis.*—Disposal of stools, boiling of drinking-water, compulsory wearing of shoes. *Of the disease:* Purgation with castor oil or salts at night, followed by thymol, ʒss, in the morning and again two hours later, with another dose of salts two hours after this. Bed and diet of milk and soup during treatment. Repeat weekly until ova are absent from the stools. Then treat the anemia as usual.

FILARIASIS

Caused by the presence in the blood of different species of the **Filaria sanguinis hominis**—Filaria bancrofti, Filaria diurna, Filaria perstans. (1) The Filaria bancrofti, the most common, is found in the peripheral circulation only at night, or only during the day if the sleeping hours are reversed. The adult female produces many embryos, which enter the blood with the lymph. They are supposed to

remain in the lungs during the hours of bodily activity and to enter the blood current during rest. Their life cycle is supposed to be completed in the mosquito, which ingests them in the blood at night and directly inoculates persons bitten later, or when dying leaves them in water. The embryos are of about the width of a red blood cell and $\frac{1}{90}$ inch in length. They move very actively. The adult worms are much larger. (2) The Filaria diurna is found in the blood only in the daytime or when the patient stays awake. (3) The Filaria perstans is supposed to be the cause of craw-craw, a parasitic skin disease.

Symptoms.—Filariæ may cause no symptoms. If the adult worms or ova block lymph channels they cause hematochyluria, lymph scrotum, elephantiasis, etc. (1) **Hematochyluria:** The only symptom is the passage at intervals of a milky, bloody, or chylous urine, which deposits a reddish sediment. It contains minute fat drops, usually red cells, and sometimes the embryos. The passage of blood clots from the bladder may cause pain. (2) **Lymph scrotum:** The scrotal tissue is much thickened, and enlarged lymphatics are seen.

Diagnosis.—By examination of the blood at about midnight for the embryos.

Treatment.—*Prophylaxis.*—Boil drinking-water. *Of the disease:* No known cure. Removal of the enlarged lymph nodes containing the adult worms may be of value. Dry diet and avoidance of fat are advised in hematochyluria.

TAPEWORMS

1. **Tenia solium,** or **pork tapeworm,** is six to twelve feet long. It has a round head smaller than that of a pin, with projected rostellum on which is a double circle of hooklets and below which are four sucking disks. By these hooklets and disks the parasite attaches itself to the mucosa of the

small intestine of man. Below the head is a constricted neck, which is followed by a large number of segments or proglottides, increasing in size from the neck onward. When full grown the segments are about 1 cm. long and 5 to 8 mm. broad. Each segment contains the generative organs of both sexes. The uterus has eight to fourteen lateral branches from a median stem. The parasite becomes fully grown in three to three and one-half months. Segments then continually break off and are discharged at stool. Each ovum contains a single embryo, armed with six hooklets and contained in a thick shell. When swallowed by pig or man these shells are digested and the embryos migrate to various parts of the body, where they change to cysticerci or "measles." Each cysticercus contains a scolex or tapeworm head. When meat containing "measles" and imperfectly cooked is eaten the cyst is dissolved in the human stomach, and the freed scolex attaches itself to the intestinal mucosa and grows into a tapeworm.

2. **Tenia saginata,** or **mediocanellata,** or **beef tapeworm,** is the common form in the United States. It is larger than the preceding, fifteen to twenty feet long, and possesses a large head, which is about 2 mm. in diameter, and has no rostellum or hooklets, but is square and has four sucking disks. The segments are larger and the uterus has more lateral branches, fifteen to thirty-five, than that in the Tenia solium, and they divide more dichotomously than in the latter. The ova have thicker shells and are larger than those of the pork tapeworm. Cattle are the intermediate hosts, and the development of the ova is similar.

3. **Bothriocephalus latus,** a still larger worm, and other species are rare in this country.

Symptoms.—These worms are found in persons of any age. The only symptoms may be the passage of segments. They may cause excessive appetite, nausea, vomiting, diarrhea, or abdominal pain, sometimes anemia. The knowledge of the presence of a parasite may cause great nervousness

or depression; some attribute epilepsy, convulsions, and other severe nervous disorders to them.

Diagnosis.—By the presence of segments or ova in stools.

Treatment.—*Prophylaxis.*—Destruction of all tapeworm segments; inspection of meat; sufficient cooking of meat to kill the parasites. *Of the disease:* Light diet and catharsis for one or two days. No supper except a glass of milk. Calomel, gr. ij to iij, with sodium bicarb., gr. x, at bedtime; Rochelle salt, ℥ ss to j, or magnesium citrate upon awakening. As soon as the bowels have moved give oleoresin of aspidium, ℥j, in capsules. Castor oil, ℥ss, or a saline should be given one-half to one hour later. It is essential that the worm be exposed to the drug employed by light diet and catharsis.

ECHINOCOCCUS DISEASE

The **Tenia echinococcus** is a parasite found in the intestinal canal of the dog, more rarely of other animals. It is therefore most common in Iceland, Australia, and other countries where the association with dogs is intimate. It is rare in the United States. The parasite is 4 or 5 mm. long, and consists of a head with four sucking disks and a double row of hooklets, and three other segments. The last is long and contains many ova. When these enter the human stomach the shell is digested and the embryos burrow into the muscles, peritoneal cavity, or vessels. They are often carried to the liver, less frequently to the lungs, brain, or other organs. The hooklets are then lost and each embryo changes into an **hydatid cyst**, with a lamellated wall enclosing an inner granular layer, and itself contained in a connective-tissue capsule of inflammatory origin. The cyst contains a clear fluid. From the granular layer buds form, which change into daughter cysts; and inside of these grand-daughter cysts, also of similar structure, may arise. From the granular inner layer of parent and daughter cysts brood capsules arise by budding, and from their lining membrane are formed projections which

develop into scolices or tapeworm heads with suckers and hooklets. These become free within the capsules and are capable of development when ingested by the dog. The scolices may live for many years, or die, liberating hooklets in the cyst cavity. The cyst contents then usually become changed into a dry, putty-like mass, which may become calcified. Rupture or suppuration of the cyst may occur. Rarer forms of hydatid cyst occur. The cyst fluid is clear, neutral, of specific gravity 1005 to 1009; it contains no albumin, but sometimes sugar, and may contain free hooklets.

Symptoms.—(*a*) **Echinococcus, or hydatids of the liver,** may give no symptoms if small. If large they may cause sensations of pressure, weight, or pain. The physical signs are those of enlargement of the liver upward, downward, or forward. If superficial, a tense or fluctuating, rounded or irregular prominence may be palpated. Holding the tumor gently with the fingers of one hand and percussing it with the other a sensation like the trembling of a bowl of jelly is sometimes obtained ("hydatid fremitus"). Large cysts give symptoms of pressure upon the abdominal or thoracic viscera. If suppuration occurs the symptoms are those of abscess of the liver (p. 197). Rupture is usually marked by sudden sharp pain and varying degree of shock. Subsequent symptoms depend upon the direction of rupture.

Diagnosis.—By the association of good health with a large, irregular liver tumor, sometimes fluctuating or elastic, or giving the hydatid fremitus; aspirated fluid may contain hooklets.

Differential Diagnosis.—(1) From **syphilis,** by absence of specific history and signs of former lesions. (2) From **cancer,** by less rapid growth, lack of hardness, and of constitutional symptoms of cancer. (3) From **cirrhosis,** by absence of symptoms of cirrhosis (*q. v.*) or history of chronic alcoholism. In atrophic cirrhosis without fatty degeneration the liver is reduced in size and ascites is greater; in hypertrophic there are jaundice, symmetrical enlargement of the liver, and a

large, hard spleen. (4) From **amyloid liver**, by absence of history of tuberculosis or syphilis and of a uniformly enlarged, smooth, hard liver. (5) From **abscess**, by absence of leukocytosis and septic symptoms. (6) From **distended gall-bladder**, by the shape and mobility of the latter and by the character of fluid obtained by puncture, if necessary. (7) From **hydronephrosis**, by the shape of the tumor and by aspiration. (8) Of hydatid cyst near upper part of the liver from **pleural effusion** of right chest, by difference in upper limit of dulness and of upper outline of *x*-ray shadow of liver.

(*b*) **Echinococcus of the pleura and lung** may give no symptoms. If in the pleura, the signs are those of hydrothorax, but with an irregular line of dulness which does not change when lying down. The heart may be displaced, with symptoms of compression of the lung. The cyst may perforate the chest wall, with spontaneous recovery. Cysts in the lung, if large, cause pulmonary inflammation or gangrene. They may rupture into the pleural cavity, giving signs of empyema, or into the bronchi, with expectoration of membrane and hooklets. Usually fatal.

(*c*) **Echinococcus of the kidney** resembles hydronephrosis or cystic kidney. Few symptoms unless rupture occurs. Diagnosis by aspiration only; if rupture takes place into the renal pelvis, cysts or membrane may be found in the urine, sometimes after attacks of renal colic.

(*d*) **Echinococcus of the brain** is rare. Symptoms of brain tumor.

Prognosis of Echinococcus Disease.—Fatal unless the echinococcus dies spontaneously or is removed.

Treatment.—*Prophylaxis* same as of tapeworm. *Of the disease:* Incision or removal.

AMEBIC DYSENTERY

Definition.—Acute or chronic colitis caused by the Ameba dysenteriæ. Sometimes called **tropical dysentery**,

because it is very common in tropical countries, but it also occurs frequently in temperate regions. It is endemic, epidemic, and sporadic.

Etiology.—Ameba dysenteriæ, found in mucus or pus in the stools, and in amebic abscesses of the liver and lungs, is a unicellular rounded organism, with variable protrusion, called pseudopodia. It is transmitted by contaminated water and green vegetables.

Pathology.—In the large intestine and sometimes part of the ileum there is proliferation of fixed connective-tissue cells, with subsequent ulceration of the overlying mucosa. The ulcers undermine the mucosa and may unite by necrosis of their margins. Amebæ are found in the walls and floors of the ulcers, in the lymphatic spaces, and at times in the bloodvessels. The ulcers may extend to any depth. There is no tendency to suppuration. If healing occurs there is contraction of the scar tissue in the walls and floors of the ulcers, constricting the intestine. In the liver there are focal necroses and may be abscesses, usually in the right lobe. They are single or multiple. (See Abscess of the Liver, p. 196.) If they point through the diaphragm, abscess of the lung or empyema results.

Symptoms.—(*a*) *Acute cases:* Sudden onset of frequent passages containing blood, or blood and mucus, with severe pain and tenesmus and slight fever. There is rapid loss of flesh and strength, and death may occur within a week from the disease or complications. Recovery is the rule, but the colitis may become chronic. (*b*) *Chronic cases:* In these the disease may begin as acute or subacute. There are alternating periods of frequent diarrheal passages with blood and mucus and constipation. The symptoms of the attacks are mild, but emaciation is marked in long-continued cases, and death occurs from asthenia. (*c*) *Mild cases* may cause almost no symptoms, only slight diarrhea and abdominal pain and lassitude.

Complications and Sequelæ.—Abscess of the liver (*q. v.*) and secondary to this of the lung; intestinal perforation, followed by local or general peritonitis; intestinal hemorrhage.

Diagnosis.—By tendency to recurrence and by examination of fresh stools on a warm stage for living amebæ showing protrusion of pseudopodia.

Prognosis.—There is a great tendency for the disease to become chronic and recurrent, ending eventually in death from asthenia or complications.

Treatment.—*Prophylactic:* Same as typhoid. *Of the disease:* Rest in bed; liquid diet in acute cases, more liberal if intestinal symptoms permit. Emetine hydrochloride, gr. ½ to ⅔ q. d. or b. i. d., is specific. Irrigation of the large intestine with large quantities of warm solutions of quinine, 1 to 5000, gradually increasing to 1 to 1000, or of silver nitrate, or thymol. Application of tincture of iodine to ulcers visible with proctoscope. In cases which have resisted medical treatment for several months, recovery may follow formation of an artificial anus, which relieves the diseased intestine entirely from fecal irritation and allows thorough irrigation of the colon from above. After recovery the artificial anus may be closed by operation. *Of symptoms:* For pain, hot applications to abdomen, morphine. For tenesmus, small starch-water and laudanum enemata.

MALARIAL FEVER

Definition.—Malarial fever is an infectious disease characterized by intermittent quotidian, tertian, or quartan fever, or remittent fever; there are also several pernicious types of the disease, and chronic malarial cachexia, with enlargement of the spleen and anemia.

Etiology.—It occurs chiefly in low lands, along seacoasts and swamps, particularly in the tropics and warmer portions of the temperate zone. In the temperate zone the attacks are most common in the autumn, less in the spring.

The *exciting cause* is the **Hematozoön** or **Plasmodium malariæ**, a parasite developing in the body of all species of anopheles, a common form of mosquito, and transmitted to man, its intermediate host, by the bite of infected mosquitoes. The *parasites* are of several classes, each causing a definite type of the disease. That of *tertian fever* is seen soon after a chill as a small, rounded, or irregular hyaline body within the red blood cells. This increases in size and shows ameboid movements, while fine granules of pigment appear within it, and the blood cell becomes paler and larger. About forty-eight hours after the chill the pigment has collected at the center of the organism and segmentation of the parasite occurs, the body dividing irregularly into fifteen to twenty nucleated subdivisions, like the original hyaline bodies, which are set free in the blood. This occurs at the time of the chill. The *parasite of quartan fever* requires seventy-two hours for its cycle in the human blood; ameboid movement is less marked than in the tertian form; the pigment is coarser; the segments number only six to twelve, and are arranged radially. In both types some full-grown bodies remain unsegmented as the sexual forms of the organism. The *parasite of estivo-autumnal fevers* is much smaller than the other forms, the pigment less abundant. Young signet-ring forms are common. The small hyaline bodies are found in the peripheral circulation, the older in the spleen and bone marrow. The cycle occupies forty-eight hours, and the number of segments formed is about fifteen. Ovoid or crescentic bodies contained in remnants of red cells, the sexually differentiated forms of the parasite, appear in the peripheral blood after a few days. The sexual forms of each variety of parasite enter the stomach of the anopheles when the infected person is bitten. Fertilization occurs there, and after completing a cycle of development in the mosquito, the resulting sporozoids may be inoculated into persons bitten by the insect, and the organisms pass through another cycle in their human hosts.

Pathology.—In *acute cases* the red cells and hemoglobin are diminished and the parasites are found in the blood; the spleen is enlarged, dark, soft, and shows congestion and the presence of many parasites. The kidneys may show albuminous degeneration. In pernicious cases with cerebral symptoms the brain is often congested and its capillaries may be filled with the parasites; in cases with intestinal symptoms they may be found in the gastric and intestinal capillaries. The bone marrow contains many organisms. Phagocytosis is marked in the marrow and spleen, and pigmentation in these structures and the liver. In *chronic cases* there is marked anemia. The spleen is very large, its capsule thickened, cut section slate-colored; microscopically, hyperplasia of all connective-tissue structures and pigmentation. Liver enlarged, slate-colored; pigmented, connective tissue sometimes increased. Kidneys may be pigmented and show chronic diffuse nephritis. The peritoneum and gastro-intestinal mucosa are sometimes grayish red from deposition of pigment.

Symptoms.—1. *Intermittent Malarial Fever:* (*a*) Tertian, (*b*) quartan, (*c*) quotidian. The symptoms of all of these are the same, except that in tertian fever, the parasite completing its cycle in forty-eight hours, the paroxysms occur every third day; in quartan they occur every fourth day, and an infection with two groups of tertian parasites causes a double tertian or quotidian fever.

The *incubation* period is unknown. The disease is characterized by paroxysms occurring at regular intervals, and usually consisting of three stages—cold, hot, and sweating. These usually occur in the morning. There may be premonitory disagreeable sensations. The *cold stage* is ushered in by yawning, lassitude, and headache, with rapid rise of temperature; sometimes nausea and vomiting, followed by shivering and later violent shaking with chattering of the teeth. The chill lasts from ten minutes to two hours. The internal temperature rises to 104° to 106°, while the surface

is blue and cold. There is severe headache; often nausea and vomiting. The pulse is small and quick. In the *hot stage*, which lasts from one-half hour to five hours, the temperature may increase somewhat; the face is flushed, the skin red and hot; there are great thirst, a throbbing headache, and full, bounding pulse. The *sweating stage* lasts two to four hours. In this the entire body is covered with a slight or profuse perspiration, the fever and all other symptoms abate, and sleep usually follows. The spleen can generally be palpated during the attack. In atypical paroxysms the cold stage may be absent or represented only by a chilly feeling, and sweating may be very slight. The patient usually feels well between the attacks. Spontaneous recovery, temporary improvement with recurrence, and malarial cachexia are the terminations if untreated.

2. *Remittent or Continuous Malarial Fever.—Estivo-autumnal fever:* This form, caused by the estivo-autumnal parasite, occurs, in temperate regions, especially in the summer and autumn. Its symptoms vary greatly. The fever may be regularly intermittent, but at longer intervals than in the tertian; there may be anticipation or retardation of the paroxysms, making the fever continuous with exacerbations. The individual attacks are of greater duration than the tertian. The cold stage is often absent, and in the hot the temperature often falls by lysis. The appearance is often like that of typhoid. Spontaneous recovery may occur after a week to ten days; there may be remissions and exacerbations, or the disease may assume the pernicious type.

3. *Pernicious malarial fever* is always caused by the estivo-autumnal parasite. The chief forms are the comatose, algid, and hemorrhagic: (*a*) The *comatose* form is characterized by delirium or sudden coma, with high temperature. (*b*) The *algid* or *asthenic form* begins with vomiting and great prostration. The temperature is normal or subnormal. There may be severe diarrhea and suppression of urine. (*c*) The *hemorrhagic forms* include *malarial hemoglobinuria* and

blackwater fever. The exact cause of the hemoglobinuria is unknown. By some it is attributed to the malarial infection; by others to the use of quinine.

4. *Malarial Cachexia.*—Chronic malaria results in symptoms of secondary anemia, with enlargement of the spleen.

Diagnosis.—By periodicity of symptoms, enlarged spleen, microscopic examination of fresh or stained blood specimens, and by the test of treatment with quinine.

Differential Diagnosis.—(1) From **typhoid**, by absence of Widal reaction. (2) From **septic processes**, by absence of **leukocytosis**. (3) From **yellow fever**, by absence of early jaundice, albuminuria, facies of yellow fever, and black vomit. Examination of blood is the final test in all of these cases.

Prognosis.—(1) Tertian and quartan, good as to life; sometimes cease spontaneously, may recur or become chronic. (2) Estivo-autumnal, more protracted; may become chronic or pernicious. (3) Pernicious, untreated, often causes death in a few days. (4) Malarial cachexia, usually recovery under treatment.

Treatment.—*Prophylaxis.*—Destruction of anopheles, protection from mosquitoes by screens, etc.; persistent treatment of infected persons; small prophylactic doses of quinine, gr. iij, t. i. d., for persons in malarial regions. *Of the disease:* Quinine, gr. v, t. i. d., will nearly always cure tertian and quartan cases, especially if kept in bed until the time for one or two paroxysms has passed. Attacks often stop spontaneously for a time when this is done, even without administration of quinine. A single dose of gr. x to xx may be given five or six hours before the expected paroxysm, and subsequently gr. v, t. i. d., for a week or ten days, and smaller doses for two or three weeks. If constipated, a cathartic before beginning quinine may aid absorption. In estivo-autumnal cases larger doses may be necessary. In pernicious forms, hydrochlorate of quinine and urea, gr. x to xx, hypodermically, every three or four hours until

improvement occurs, when the sulphate by mouth may be substituted. Intravenous administration of bimuriate of quinine has been recommended for very severe cases. In malarial cachexia, arsenic and iron, as well as quinine. *Of symptoms:* In the cold stage, blankets and hot drinks; in the hot stage, cold sponging if headache and fever are severe. In pernicious cases, caffeine or strychnine, when required. For anemia, iron and arsenic, *e. g.*, pil. ferri carb., ij, p. c., and liq. potass. arsenitis, gtt. iij to viij, p. c.

SECTION III

INTOXICATIONS AND SUNSTROKE

ALCOHOLISM

1. **Acute Alcoholism.—Symptoms.**—Face flushed, breath alcoholic, pulse full and bounding, respiration deep. Reason, memory, judgment, and will are first stimulated, then blunted. Individual peculiarities are exaggerated, the person becoming affectionate or pugnacious. There is loss of co-ordination, shown by staggering, then muscular relaxation, and finally coma with stertorous breathing. The person is unconscious, but can be partly aroused and will mutter when questioned. Pupils are contracted or dilated, and dilate when the face is slapped. Temperature, reflexes, and sensation are diminished; urine is increased, but often retained, causing distention of the bladder.

Differential Diagnosis.—(1) From **apoplexy,** in which coma is deeper, pupils are more often unequal, and there are increased arterial tension and paralysis of groups of muscles. (2) From **fracture of the base of the skull,** in which there may be a watery discharge and hemorrhage from the ears or mouth, with symptoms of intracranial hemorrhage. (3) From **uremic coma,** in which there are dry skin, characteristic odor of the breath, often twitchings or convulsions, irregular pupils, scanty, albuminous urine containing casts. (4) From **diabetic coma,** which is sudden and profound and in which

the breath is sweetish, with glycosuria and ketonuria. (5) From **epilepsy**, in which a convulsive attack precedes, and the tongue often shows old or fresh bites. (6) From **opium poisoning**, in which there are pinhead pupils and very slow, irregular respiration. (7) From poisoning by **chloral, chloroform, ether, gas**, etc., by odor of breath and gastric analysis. (8) From coma of **sunstroke**, by history. *Acute alcoholism is often associated with cerebral hemorrhage, fracture of the skull, or sunstroke*, hence diagnosis should always be guarded.

2. **Chronic alcoholism** results from protracted or periodical excesses.

Pathology.—Some or all of the lesions found in cases of chronic alcoholism are absent in many instances, and may also be the result of other causes. There may be chronic pachymeningitis, edema of the pia or brain, peripheral neuritis, pulmonary congestion, flabby and fatty heart wall, sclerosis and atheroma of the arteries, chronic gastritis, cirrhosis with or without fatty degeneration of the liver, albuminous or fatty degeneration of the kidneys, or chronic diffuse nephritis.

Symptoms.—Face red, capillaries dilated, eyes watery, conjunctivæ congested; chronic gastritis, the particular type of which is characterized by morning vomiting; often cirrhosis of the liver, tremor of hands and tongue, impairment of judgment, memory, and will, especially until a stimulant has been taken; often irritability, carelessness, loss of moral sense, in extreme cases dementia. Peripheral neuritis is more common in women. It begins with sharp pain and tingling in feet and hands; paralysis affects lower extremities, then upper, is most marked in distal muscles of limbs and is chiefly extensor. Pain may be very severe, with great tenderness. Arteriosclerosis; often cardiac dilatation.

3. **Delirium tremens** is a cerebral manifestation of chronic alcoholism, occurring in steady drinkers after excessive consumption or sudden withdrawal of alcohol, or after sudden excitement, an accident, pneumonia or other illness, or lack

of food. The symptoms are restlessness, insomnia, mental depression, then active delirium with great restlessness, talking, muttering, hallucinations of sight or hearing. The patient sees black or colored snakes, rats or other animals; occasionally he hears them or voices talking to him. To escape them he may jump from the window, etc. Tongue heavily coated and tremulous. Temperature usually below 103°; pulse frequent, soft. Tremor and insomnia are marked. After a few days all symptoms, mental and physical, diminish. In severe cases a typhoidal state, with high temperature and insomnia, supervenes, and death occurs from cardiac weakness.

Diagnosis.—By history and absence of other illness. It is necessary to differentiate cases with high temperature from meningitis, apex pneumonia, erysipelas, etc. Pneumonia should be particularly watched for.

Prognosis.—Usually recovery.

Treatment.—*Acute alcoholism:* Sleep, lavage if necessary, cold bath, aromatic spirit of ammonia, ʒss to j, for headache and reactionary symptoms. *Chronic alcoholism:* Withdrawal of alcohol, substitution of strychnine, gr. $\frac{1}{30}$, three or four times a day, nourishing food, confinement in sanitarium, bromides for restlessness and sleeplessness. Cure is often difficult, as the patient does not wish to coöperate. Drugging the liquor with apomorphine or tartar emetic may succeed. *Delirium tremens:* Bed, draw-sheet if necessary, avoidance of active restraint, constant watching, withdrawal of alcohol, substitution of strychnine, nourishing food given frequently. For restlessness and delirium, sodium or potassium bromide, gr. xxx, and chloral, gr. v, every three hours, paraldehyde, ʒss, then ʒj to ij, p. r. n., or hyoscine hydrobromide, gr. $\frac{1}{100}$, hypodermically if necessary; morphine, gr. $\frac{1}{4}$, may be given for a few doses if it quiets; cold bath or sponging or pack if febrile. For cardiac weakness, aromatic spirit of ammonia; whisky if necessary.

MORPHINE HABIT

Called also **morphinism**, or **morphinomania**, is usually acquired by repeated use of the hypodermic syringe for pain. Drug is used hypodermically, by mouth, or by opium smoking.

Symptoms.—The drug at first causes a sense of well-being and exhilaration, but must be gradually increased to produce this result. As the effect of a dose wears off there are weariness, mental and physical, nausea, slight epigastric distress, or pain like intestinal colic. These symptoms are relieved by repeating the dose. Eventually, the person becomes thin, with sallow face, pupils dilated or unequal except when under the influence of the drug, poor appetite, indigestion, sometimes itching of the skin, restlessness, irritability, disturbed sleep and a tendency to tell lies about everything. There may be hysteria or neurasthenia. Especially by Orientals, morphine may be used for years with little effect, or the dose may have to be increased tremendously and gradual asthenia may terminate in death.

Treatment.—Isolation from home and friends, constant watching, gradual withdrawal of the drug, and nourishing food given frequently. Codeine or antipyrin is sometimes substituted temporarily. For restlessness, trional (gr. xx to xxx), bromides, chloral, or hyoscine; for pains and restlessness, hot baths; cardiac stimulants if necessary—whisky, aromatic spirit of ammonia, or digitalis; for vomiting and diarrhea, intestinal astringents.

COCAINE HABIT

The drug is taken as snuff, hypodermically, or in sprays. **Large doses** cause great excitement, sometimes convulsions, followed by cardiac and respiratory weakness, general prostration, convulsions, and coma. The **cocaine habit** causes

emaciation, anemia, digestive disturbances, disordered heart action, bodily and mental weakness, nervousness, great depravity.

Treatment.—Same as for morphine habit.

CHLORAL HABIT

After slight primary exhilaration there are mental and physical depression, cutaneous eruptions, bad breath, spongy gums, poor appetite, indigestion, malnutrition, permanent dilatation of cutaneous bloodvessels, intermittent pulse, blunting of higher mental qualities, restlessness, sleeplessness, irritability, sensory and motor disturbances, sometimes dementia. Habit establishes little tolerance.

Treatment.—Same as for morphine habit.

LEAD POISONING (PLUMBISM, SATURNISM)

Etiology.—It is common in lead smelters and grinders, painters, glaziers, and plumbers, whose hands are not washed before eating. The lead is absorbed by mouth, skin, or lungs. It may be ingested in drinking-water, cider, etc., in new lead pipes, or from hair dyes and cosmetics.

Pathology.—Lead is deposited in the muscles, nerves, and other soft parts. It is eliminated slowly by kidneys, intestines, and skin. The muscles may be pale, flabby, or infiltrated with connective tissue. There may be peripheral neuritis, arteriosclerosis, and chronic nephritis.

Symptoms.—**Acute cases**: Symptoms develop rapidly from continued exposure. They are rapidly progressing anemia with acute neuritis, epilepsy, convulsions or delirium, or with severe gastro-intestinal symptoms. **Chronic cases**: Chief symptoms are anemia, lead line in gums, paralysis, colic, and cerebral symptoms. (1) *Anemia*, red cells and

hemoglobin above 50 per cent., granular basophilia of red cells. (2) *Blue-black line* of lead sulphide in the gums near the teeth. (3) *Colic,* preceded and accompanied by obstinate constipation. It resembles severe intestinal colic. There may be vomiting. (4) *Paralysis,* the result of peripheral neuritis, localized or generalized. The local forms are: (*a*) Wrist-drop, or antebrachial type, a musculospiral paralysis. (*b*) Brachial type, involving the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the pectorals; primary, or follows the first type. (*c*) Aran-Duchenne type, involving the small muscles of the hands, with marked atrophy. (*d*) Peroneal type, affecting peroneal muscles and extensor communis digitorum, causing steppage gait. (*e*) Laryngeal type. Adductors paralyzed; rare. (*f*) A general paralysis may develop slowly or rapidly like an ascending spinal paralysis. Diaphragmatic involvement may cause death. Saturnine arthralgias, pains in legs and joints, usually accompany beginning paralysis, but sensation may be normal. There is usually reaction of degeneration. (5) *Cerebral symptoms:* Hysteria, convulsions, delirium with hallucinations, epilepsy, optic neuritis. (6) *Arteriosclerosis* and *chronic nephritis* are common. Gout may be present.

Diagnosis.—From **other forms of colic,** by occupation, constipation, blue line on gums, character of pain, which is cramp-like, not cutting, sometimes by presence of paralyses or cerebral symptoms. From **other forms of neuritis.** (See volume on *Nervous and Mental Diseases,* this series.)

Prognosis.—Usually good. Cerebral symptoms may be permanent. Paralysis may persist in cases with reaction of degeneration.

Treatment.—*Prophylaxis.*—Cleanliness of hands and fingernails of lead workers; use of respirators if lead is present in the form of dust. *Of chronic poisoning:* Remove cause. Potassium iodide, gr. v to x, t. i. d., to liberate lead from the tissues; not given in acute cases or when symptoms are severe, until what is already in the intestines is removed.

For constipation, magnesium sulphate, ℥ss before breakfast, when needed, or in repeated small doses. This makes an insoluble salt of lead in the intestines. For pain, heat over abdomen, morphine if necessary; for anemia, iron; for paralysis, strychnine, massage, galvanism.

ARSENICAL POISONING

Acute Poisoning.—Caused by ingestion of Paris green or rat or insect poison. **Pathology** and **symptoms** are those of acute irritant poisoning.

Chronic Poisoning.—Etiology.—Inhalation of arsenic from dyes in wallpaper, carpets, etc.; ingestion by mouth in handling dyed paper, artificial flowers, etc., or of the drug given in repeated and excessive doses.

Pathology.—May be lesions of gastro-enteritis, fatty degeneration of viscera, peripheral neuritis. Arsenic in tissues.

Symptoms.—Dry throat, edema of eyelids, sometimes coryza, nausea, vomiting, diarrhea, cutaneous eruptions or pigmentation, falling of the hair, paralysis of arms and legs with ataxia, atrophy and numbness, but little pain. Legs most affected, causing steppage gait.

Diagnosis.—From **lead** and **alcohol neuritis.** (See volume on *Nervous and Mental Diseases*, this series.)

Treatment.—Remove cause. Treat symptoms. Massage, galvanism, and strychnine for paralysis.

FOOD POISONING (BROMATOTOXISMUS)

This includes poisoning by ptomains, by meat, milk, or fish which has become decomposed, and by mussels. The **symptoms** are those of acute gastro-enteritis, with great prostration ending in collapse. In mussel poisoning they are numbness, weakness, dilatation of pupils, rapid, feeble pulse, subnormal temperature, collapse.

Treatment.—The stomach should be emptied by emetics or stomach tube, then the bowels by catharsis. Stimulation if necessary. Opium if needed for diarrhea and pain.

SUNSTROKE

Called also **heat exhaustion, insolation, heatstroke, siriasis, thermic fever.**

1. **Heat exhaustion** is caused by continued exposure to high temperatures, especially while working hard. The **symptoms** are prostration with cool skin, temperature often subnormal, pulse small and frequent, sometimes restlessness and delirium.

Treatment.—Aromatic spirit of ammonia and strychnine, avoiding alcohol; rest, warm bath if temperature is subnormal.

2. **Sunstroke, Heatstroke, or Thermic Fever.**—Caused by severe exertion while exposed directly to the sun or to high temperature. The former cases are sometimes distinguished as sunstroke, the latter as heatstroke. Use of alcohol predisposes, as does a previous attack.

Pathology.—Early rigor mortis and putrefaction; blood remains fluid. Congestion of brain and meninges, sometimes exudate or hemorrhages beneath the pia. Left ventricle contracted, right dilated. Congestion of viscera; albuminous degeneration of liver and kidneys. Chromatolysis of ganglion cells as in auto-intoxication suggests that this is the cause of heatstroke. Some consider it an infectious disease.

Symptoms.—In severe cases, where exposed to intense heat, as during forced marches, the patient falls unconscious, and death occurs almost immediately or after a few hours of coma with dyspnea and cardiac weakness. In ordinary cases there may be sudden arrest of perspiration, premonitory headache, dizziness, sometimes nausea and vomiting; colored or indistinct vision, then unconsciousness, which may be

temporary or increase to deep coma. Face flushed, skin dry and hot, pupils temporarily dilated, then usually greatly contracted; muscular relaxation, sometimes muscular spasms or convulsions; temperature, 107° to 110° or higher; pulse frequent and full; respirations deep and labored or stertorous. In fatal cases coma deepens, pulse becomes frequent and feeble, respiration rapid, irregular, and shallow, or of Cheyne-Stokes type; and death occurs in twenty-four to thirty-six hours. In others, consciousness returns, temperature falls, pulse and respiration become normal, and recovery is complete or with **sequelæ**. The patient may be predisposed to subsequent attacks or suffer from physical weakness, impaired memory or power of mental concentration, or headache and mental disturbance whenever the weather is warm.

Diagnosis.—From **other forms of coma,** by history of exposure, general appearance, high temperature.

Prognosis.—High mortality in severe cases, especially if alcoholic and if treatment is delayed. Liability to sequelæ. (See **Sequelæ.**)

Treatment.—*Prophylaxis.*—Avoid exposure and alcohol. *Mild cases:* Rest in cool place, cold sponging, aromatic spirit of ammonia or strychnine if needed for prostration. *Severe cases:* Immediate bathing in ice-water containing ice, with cutaneous friction; enemata of ice-water. If ice cannot be obtained, strip and sprinkle with water until temperature is reduced. Aromatic spirit of ammonia by mouth, strychnine hypodermically. Gastric lavage, especially if alcoholic; catharsis. Subsequently cold sponging if needed. For severe asphyxial cases with dyspnea, coma, and cardiac failure, venesection. *Of sequelæ:* Avoid exposure to heat. Ice-cap to head to relieve pain.

BERI-BERI (KAKKE)

Definition.—Beri-beri is a disease due to lack of vitamines in the diet; a multiple neuritis with anasarca.

Etiology.—Exclusive diet of polished rice.

Pathology.—Degeneration of peripheral nerves, sometimes of the pneumogastric and phrenic, and of the cardiac and voluntary muscles.

Symptoms.—*Incubation* period unknown. (1) *Incomplete or rudimentary form: Invasion* marked by catarrhal symptoms, then pain and weakness of the extremities, diminution of sensibility of the legs and paresthesiæ in them and later elsewhere. There may be edema, dyspnea, palpitation, and muscular tenderness. These symptoms last from a few days to months, and often recur. (2) *Atrophic, dry, or paralytic form:* Similar symptoms, but with rapid paralysis, atrophy and pain in arms and legs, sometimes in the face. Little palpitation or edema. (3) *Wet or dropsical form:* Similar invasion, with rapidly developing anasarca, palpitation, and dyspnea, and little atrophy or sensory symptoms. (4) *Acute, pernicious, or cardiac form:* Rapid development of cardiac weakness early in the disease, with death in from twenty-four hours to several weeks.

Diagnosis.—Association of edema with multiple neuritis in persons from a tropical seaport.

Prognosis.—Mortality, 2 to 50 per cent.

Treatment.—*Prophylaxis.*—Diet should be richly nitrogenous, avoiding rice and raw fish and overcrowding of institutions. *Of the disease:* Catharsis, sodium salicylate, gr. xv to xx, four or five times a day, and phlebotomy for severe cases have been recommended. *Of symptoms:* For cardiac weakness, stimulants; for muscular weakness and atrophy, electricity.

SECTION IV

DISEASES OF THE JOINTS AND MUSCLES

ARTHRITIS DEFORMANS (RHEUMATOID ARTHRITIS)

A GROUP of chronic joint diseases, with degeneration of parts of the joint, bone formation, and deformity.

Etiology.—Occurs at any age, most often thirty to fifty-five; usually in women, generally at or after the menopause, and most frequently in sterile subjects; involvement of large joints is most common in adult males. The influence of heredity is doubtful, but there is often a family history of rheumatism, gout, or arthritis deformans. As exciting causes have been named exposure to cold and wet, improper food, unhygienic surroundings, worry, grief, traumatism, and acute infections. Probably the disease is of infectious origin, the trophic and metabolic changes being secondary. The arthritis is secondary to a focus of infection in teeth, gums, tonsils, nose, nasal sinuses, middle ear, bronchi, genito-urinary tract or possibly the intestine.

Pathology.—Several joints usually involved, often symmetrically. In the articular cartilages the basement substance becomes fibrillated, cartilage cells multiply, cartilage may become fatty and atrophy. Ends of bone are thus exposed, become eburnated and deformed; the spongy portion may undergo rarefying osteitis. At the edge of the

articulations there is formation of new bone covered with cartilage, causing enlargement of the bone and often partial ankylosis. The capsules become thickened; the synovial membranes vascular, thickened, and elongated; the synovial fluid is increased, then diminished. Enlargement of metacarpal joints causes ulnar deviation of the fingers; similarly with the toes. The adjacent muscles become atrophied.

Symptoms.—Several distinct types exist. (1) **General progressive type**: (*a*) *Acute:* Usually in women of twenty to thirty years or at the menopause. Invasion like acute articular rheumatism, many joints, permanent enlargement appearing early, rarely redness of joints, pain very severe, moderate rise of temperature, malaise, anemia, loss of flesh and strength. The first and later attacks are often associated with pregnancy, labor, or lactation. (*b*) *Chronic:* Gradual onset of pain or stiffness in one or more joints, usually of the fingers, then of corresponding joints of the other side, then of others. Involvement symmetrical. Swelling at first may be in its soft parts, with joint effusion and tenderness. Pain may be slight or severe. Periods of improvement and exacerbation alternate, the joints becoming enlarged and deformed, often incompletely ankylosed in partial flexion by thickening of bone and soft parts; joint crepitus. The muscles moving them atrophy and there may be trophic changes in the skin and nails of the extremities affected. Digestive disturbances and anemia are common. Heart not affected. A few joints only may be attacked or many, with great deformity, before the disease reaches a period of inactivity. (2) **Monarticular type**: Usually in males over fifty years, one joint or a few large joints may be affected, generally with atrophy of corresponding muscles. In the case of the hip this is called *morbus coxæ senilis.* (3) **Heberden's nodes**: Common in women between thirty and forty years. Often preceded by digestive disturbances. With occasional attacks of local pain and swelling, or insidiously, small hard tubercles form at the sides of the dorsal surface of the distal extremity of the

second phalanges. The presence of these nodes may be the only sign of the disease.

Diagnosis.—(1) **Acute articular rheumatism** may be confused with the early stage of the disease, but is less apt to involve the small joints, is less persistent, occurs more often in younger persons, responds better to salicylates, and shows with the *x*-rays no loss of cartilage or bony thickening. (2) From **chronic gout**, in which the first attack is often in the great toe, tophi may be present, arteriosclerosis is common, and lesions of bone and cartilage of arthritis deformans are not shown by *x*-rays. (3) From **gonorrheal arthritis**, by history, absence of urethral discharge, negative complement-fixation, and presence of bony enlargement. (4) From **Charcot joints**, by absence of history and signs of locomotor ataxia and by occurrence of local pain.

Prognosis.—Usually slowly progressive, with intervals of improvement, often resulting in great crippling and disability. In some cases it becomes almost stationary.

Treatment.—*General:* Warm, dry climate; avoid exposure; general hygienic life; as full nutritious diet as digestion permits. The chief indications are to improve the general condition and relieve pain. *Medicinal:* Cod-liver oil, arsenic, and iron; potassium iodide may be of value. Salicylates may relieve pain in acute attacks. *Hydrotherapy* and *local treatment:* Heat, in the form of baths or hot compresses. Frequent treatment with air heated to 250° or 350° F., for one-half to one hour, with the affected limb wrapped in gauze. Massage is a valuable adjuvant. Mercurial ointment locally may aid. Marked improvement sometimes follows use of a mixed vaccine of the common pathogenic bacteria, but an autogenous vaccine made from cultures from foci such as carious teeth, tonsils, etc., is preferable. Such foci should be treated.

MYALGIA

Definition.—A painful affection of voluntary muscles, called also, according to its special localization, **torticollis, pleurodynia, lumbago,** etc.

Etiology.—Predisposed to by previous attacks and the rheumatic or gouty diathesis. Follows sudden exposure or slight traumatism, hence most common in men. Actual cause unknown. It may be a myositis or a neuralgia of sensory nerves of the muscles.

Symptoms.—Local muscular pain, sharp or dull, aching, constant or caused only by certain movements, usually relieved by pressure. Duration, a few days to several weeks. Recurrence frequent. Fever is exceptional and slight. The common forms are **lumbago,** affecting the muscles of the lumbar region, sudden onset, chiefly in laborers, often completely disabling; **torticollis,** or **stiff neck,** in anterolateral or posterior neck muscles, usually of one side; **pleurodynia,** in intercostals, pectorals, or serratus magnus, with pain on all chest movements, resembling intercostal neuralgia or pleurisy.

Diagnosis.—By localization in muscles, increase of pain by motions contracting or stretching the muscles, absence of other symptoms. (1) From **intercostal neuralgia,** by local tenderness, constancy of pain, absence of tenderness along nerve trunks. (2) From **pleurisy,** by absence of its physical signs.

Treatment.—Avoid exposure; rest aided by strapping, heat locally, or counter-irritation by cautery. Galvanism, acupuncture, and the Turkish bath sometimes aid. Belladonna liniment or ointment may relieve. Aspirin or sodium salicylate, gr. x, every three or four hours, is often efficient. In chronic cases potassium iodide, nux vomica, or arsenic may help. If gouty, restrict diet and give alkaline waters.

SECTION V

DISEASES OF METABOLISM

GOUT

Definition.—A disorder of nutrition characterized by excess of uric acid in the blood, attacks of acute arthritis, with deposition of sodium urate in and around the joints, and various general symptoms.

Etiology.—Heredity; male sex; age, usually thirty to fifty years, rarely under twenty; continued use of alcohol, especially fermented liquors; food, excessive indulgence, especially in highly seasoned nitrogenous food, with little exercise. Unhygienic living with poor food and excessive consumption of ales and beers may be followed by "poor man's gout." Gout is common in lead workers. The actual cause of the disease is unknown. There is defective metabolism with an excess of uric acid in the system; but whether this is due to increased production, decreased elimination, or both, and why, is uncertain.

Pathology.—The blood contains an excess of uric acid. During acute attacks the affected joints are inflamed, with some effusion, and urates are deposited in crystals or masses, uniformly or in patches in the cartilages and synovial membranes, also in synovial fluid. In chronic cases the ligaments and subcutaneous tissues adjacent are infiltrated with urates, which form masses called tophi or chalk-stones. The skin over these may ulcerate. Similar deposits occur in bursæ,

tendon sheaths, cartilages of the ears, nose, and eyelids. Eventually the joint may become ankylosed. Other lesions in chronic cases are chronic interstitial nephritis, often deposits of sodium urate in tubules and stroma of renal papillæ, arteriosclerosis with hypertrophy of the left ventricle, emphysema, chronic bronchitis, eczema.

Symptoms.—(1) **Acute gout**: Often premonitory irritability, restlessness, indigestion, with twinges of pain in the hands and feet; urine scanty, dark, very acid, with diminished uric acid, depositing urates when cooled. *Invasion* usually early in the morning, with sudden intense pain in the metatarsophalangeal articulation of a great toe, generally the right, less often an ankle, knee, wrist, hand, or finger. This swells rapidly and is extremely tender, the overlying skin red, glazed, hot. Temperature may reach 103°. During the day the pain may subside, increasing again at night. Suppuration does not occur; symptoms usually decrease gradually, the entire attack lasting five to eight days. Desquamation may follow. After the attack the general health may be improved, and the joint normal or slightly stiff. Recurrence at intervals of a few months is common. (2) **Retrocedent gout** is the name given to serious symptoms which sometimes accompany rapid improvement of the local joint conditions—severe gastric pain, nausea, vomiting, diarrhea; cardiac pain, dyspnea, palpitation, irregular and feeble heart action; cerebral symptoms, probably uremic. These attacks may be fatal. (3) **Chronic gout**: Repeated acute attacks. Many joints, beginning with the feet, become stiff, deformed, perhaps ankylosed by deposits of urates. Tophi around joints, in ears, bursæ, and tendon sheaths. The overlying skin may ulcerate, especially over the knuckles. Dyspepsia, arteriosclerosis, increase of tension, hypertrophy of the left ventricle, abundant urine of low specific gravity are common. Moroseness and irritability are not unusual. Frequent complications are eczema, chronic bronchitis, emphysema. Death often occurs from uremia, meningitis,

pleurisy, pericarditis, or peritonitis. (4) **Irregular gout** or **gouty diathesis** or **lithemic state** is a term applied to certain symptoms often observed in members of gouty families or in others who have no typical acute attacks. It is also a convenient cloak for ignorance. The chief of these symptoms and conditions are eczema, "biliousness," hepatic torpor, constipation, chronic bronchitis, laryngitis, pharyngitis, emphysema; cardiac palpitation and irregularity from failure of hypertrophy; arteriosclerosis, cerebral endarteritis, and hemorrhage; chronic interstitial nephritis, iritis, headache, burning and itching of the feet at night.

Diagnosis.—Differentiated from **rheumatism** and **arthritis deformans** by family history, occupation (bartenders, brewery and lead workers), dietetic habits; recurrence of acute arthritis, with the first attack, at least, usually at night and in the great toe; affection of small joints chiefly; predominance of local over constitutional symptoms, especially fever, with absence of tendency to wander from one joint to another. In chronic cases, by presence of tophi.

Treatment.—*Prophylactic and general:* "Live temperately, abstain from alcohol, eat moderately." Fresh air, bathing, exercise, warm clothing. Avoid fermented liquors especially. Take water in large quantities on an empty stomach. Mineral waters are no better than others, but treatment of chronic and irregular gout at springs gives the advantage of regular hours, diet, etc. "The weight of opinion leans to the use of a modified nitrogenous diet without excess in starchy and saccharine articles of food." *Of acute gout:* For pain, phenacetin and hot applications or hot-air baths; elevate limb; wrap joint in cotton. Wine of colchicum, ℥xv to xxx, with potassium citrate, gr. xv, every four hours until pain is relieved, then wine of colchicum, ℥x every four hours, watching for symptoms of gastro-enteritis or renal irritation. Milk or fluid diet, returning gradually to gouty diet. *Of chronic and irregular gout:* Hygiene, potassium iodide, alkalies, tonics.

DIABETES MELLITUS

Definition.—A nutritional disorder characterized by an excess of sugar in the blood and its persistent excretion in the urine, which is much increased.

Etiology.—Heredity, male sex, adult life, Jewish race, obesity, cerebral or spinal disease or injury, infectious diseases, overwork and nervous strain predispose. Actual cause is unknown. Pancreatic disease is probably important.

Pathology.—The blood contains an excess of sugar and fat globules. Nerve lesions are inconstant. Common lesions are cardiac hypertrophy, arteriosclerosis, pulmonary tuberculosis, lobar or bronchopneumonia; liver generally enlarged, often fatty; chronic nephritis or fatty degeneration; pancreas shows inflammation, degeneration, atrophy, or a newgrowth of the islands of Langerhans.

Symptoms.—The invasion is usually gradual. Urine is passed frequently, six to forty pints in twenty-four hours; is pale, specific gravity 1025 to 1045, sometimes lower with chronic nephritis; contains 1 to 10 per cent. of glucose, often acetone, diacetic acid, also β-oxybutyric acid during coma. There are abnormal thirst and appetite (bulimia), often loss of weight and strength, sometimes constipation, headache, and depression. Mouth dry, tongue red and glazed, skin dry. Progress is more rapid the younger the patient. Death usually occurs in diabetic coma; in others it results from complications.

Complications.—Frequently boils and carbuncles, balanitis or pruritus vulvæ, less often eczema or gangrene; lobar or bronchopneumonia, pulmonary tuberculosis or gangrene; arteriosclerosis; peripheral neuritis sometimes simulating locomotor ataxia or paraplegia, occasionally herpes zoster; perforating ulcer of the foot; often loss of sexual power, conception rare, abortion usually the result; cataract, optic nerve atrophy, retinitis, sudden blindness. **Diabetic coma,**

most common in young patients, is probably caused by β-oxybutyric acid in the blood. (1) Sudden onset after exertion, of weakness, feeble pulse, stupor, coma, death in a few hours. (2) Sudden headache, coma, death in a few hours. (3) After nausea, vomiting, or a pulmonary complication, there are headache, delirium, abdominal pain, rapid, labored breathing, sweetish odor to breath, stupor, rapid, feeble pulse, coma, death within a few days.

Diagnosis.—By persistence of glucose in the urine, which is usually of high specific gravity (Fehling's, Trommer's, and fermentation tests for glucose). By hyperglycemia: sugar 0.3 to 0.8 per cent. (normal is 0.1 to 0.15 per cent.).

Differential Diagnosis.—(1) From **transient glycosuria.** (2) From **hysterical polyuria, chronic nephritis, and diabetes insipidus,** by the presence of sugar and high specific gravity. (3) From other forms of **coma**—alcoholic, opium, uremic, apoplectic, epileptic, etc.—by absence of characteristic signs of these and by urinalysis and blood-sugar determination.

Prognosis.—Recovery is rare. In patients under forty years the outlook is bad; in those over forty the progress is slow and symptoms are milder. If sugar is not diminished by carbohydrate-free diet and acidosis remains high, the prognosis is serious.

Treatment.—*General:* Avoid exertion, worry, and exposure. Moderate exercise, baths. *Dietetic:* Is the most important. Determine sugar excretion on ordinary diet and carbohydrate tolerance with carbohydrate-free diet, with addition of weighed amount of bread. Prohibit sugar. In severe cases with marked acidosis, oatmeal, butter, and white of egg sufficient to supply caloric needs may be given for several days. When acidosis is controlled by this, one or two "green" or "vegetable" days may greatly diminish sugar excretion, though increasing acidosis. This diet excludes proteins as far as possible; it includes green vegetables, egg yolks, sardines, and coffee. Carbohydrate-free diet with occasional "oatmeal" or "green" days should be continued until urine

has been sugar-free several days, then gradually increasing weighed amounts of white bread added unless sugar reappears. Caloric values are useful in feeding, but gain of weight is more important. Carbohydrates must not be so suddenly decreased as to cause acidosis. In severe cases even meats cause glycosuria, and must be restricted. Fats are usually well tolerated and of high caloric value. *Fasting treatment:* Determine glycosuria and ketonuria on ordinary diet for two days. Fast two to four days until urine is free from sugar and acidosis, giving only water *ad lib.*, tea, coffee, diluted whisky, or bouillon. Carbohydrate, protein and fat tolerances are then approached by graded diets, stopping at the lowest on which the patient can live comfortably while sugar- and acid-free. A weekly day of fasting or reduced diet is advisable. *Medicinal:* Absolutely unsatisfactory. Opium preparations, preferably codeine, gr. ss, t. i. d., increasing to gr. vj to viij in twenty-four hours. Withdraw gradually when sugar is absent or reduced so far as possible. Keep bowels open. Sodium bicarbonate in very large, repeated doses whenever diacetic acid is abundant in the urine. For coma, large intravenous injections of 4 per cent. sodium bicarbonate, up to ℥ iij a day, until urine is alkaline; or normal saline intravenous or subcutaneous injections; oxygen inhalations.

DIABETES INSIPIDUS

A chronic disease characterized by persistent passage of large quantities of normal urine of low specific gravity.

Etiology.—Most often in young males. Probably of nervous origin. Syphilis is often present.

Pathology.—There are no constant nerve lesions. The kidneys may be enlarged and congested, renal pelves and ureters dilated, and bladder hypertrophied.

Symptoms.—Onset usually gradual. Urine pale, ten to twenty quarts per day; specific gravity 1001 to 1005; total

solids often normal. Thirst, dryness of mouth and skin. Appetite and general condition are usually normal; sometimes there are feebleness and emaciation. Death may occur from intercurrent disease.

Differential Diagnosis.—(1) From **diabetes mellitus**, by low specific gravity and absence of sugar. (2) From **chronic interstitial nephritis**, by absence of albumin, casts, and physical signs of nephritis. (3) From **hysterical polyuria**, by constancy of polyuria and absence of hysterical symptoms.

Treatment.—No cure known. Opium, valerian, valerianate of zinc, gr. xv increased to gr. xxx, t. i. d., etc., are used. Antisyphilitic treatment should be tried.

OBESITY

Excessive development of fat may be hereditary. It occurs most frequently in middle-aged women and in children. Its chief causes are excess of food and drink, especially carbohydrates and malt liquors, and lack of exercise. The fat is increased in all its normal situations and the heart and liver are often large and fatty. The general condition may be good, or there may be mental and bodily inactivity, digestive disturbances, and symptoms of fatty heart (p. 272). The power of resistance to disease is diminished. Death may result from fatty infiltration of the heart, resulting in dilatation or rupture.

Treatment is chiefly dietetic. Excess of all kinds of food and drink should be avoided, especially starches and sugar. Systematic active exercise is important. Hot baths and massage are aids. Thyroid extract, gr. v, t. i. d., is sometimes useful.

SCURVY (SCORBUTUS)

Definition.—A metabolic disorder characterized by weakness, anemia, sponginess of the gums, and tendency to hemorrhages.

Etiology.—Formerly common among sailors and in armies and prisons. *Predisposing causes:* Overcrowding; dark, unhealthy rooms; prolonged fatigue; mental depression. The *exciting cause* is supposed to be (1) lack of fresh vegetables, (2) toxemia from slightly tainted food, or (3) an infection.

Pathology.—Variable, not characteristic. Gums swollen, sometimes ulcerated. Skin ecchymotic. Hemorrhages into mucous and serous membranes, muscles, viscera, sometimes joints, rarely subperiosteal. Bone necrosis is rare. Spleen enlarged and soft. Albuminous degeneration of heart, liver and kidneys.

Symptoms.—Gradual onset with weakness, loss of weight and anemia. Usually the gums become swollen, spongy, and bleed easily; the teeth may be loose. Tongue swollen, breath foul, salivary glands enlarged, saliva increased, hemorrhages into or through the buccal mucosa. Skin dry and pale, with general ecchymoses, beginning on the leg and especially in and around hair follicles. These are spontaneous or follow a slight injury. In severe cases subperiosteal hemorrhages may cause irregular swelling, especially in the legs, and these may break down, forming ulcers. Subcutaneous extravasations, especially in the lower extremities, may be followed by permanent induration and stiffness due to connective-tissue infiltration ("scurvy sclerosis"). There may be joint pains, often edema of ankles. Hemorrhages from internal mucous membranes are less common than cutaneous. Appetite is poor; constipation or diarrhea. Heart action feeble, often with a hemic murmur; blood shows secondary anemia. Urine scanty, concentrated, often albuminous. Lassitude, depression, headache, and finally delirium or coma, or symptoms due to intracranial hemorrhages, or day- or night-blindness may be present. Fever is rare except from secondary inflammations.

Diagnosis.—By history, condition of gums, hemorrhages, debility, affection of several persons simultaneously, and prompt recovery with proper food.

Prognosis.—Good if cause is removed, unless far advanced. Death may result from complications.

Treatment.—*Prophylactic:* Fresh or canned vegetables or fruit. *Of attack:* Fresh vegetables, meat, juice of two or three lemons or oranges daily. If digestion is feeble, milk, scraped beef, and lemon juice, adding meat, potatoes, lettuce, cabbage, water-cress, etc., later. *Of symptoms:* For stomatitis, mouth wash of potassium permanganate, dilute carbolic acid, or hydrogen peroxide, with local applications of silver nitrate solution. For constipation, enemata. For hemorrhages, as in purpura.

SECTION VI

DISEASES OF THE DIGESTIVE SYSTEM

DISEASES OF THE MOUTH

Acute stomatitis (**simple, catarrhal,** or **erythematous**) is caused by various irritants—gastro-intestinal disturbance, use of tobacco, or hot or highly seasoned food—acute infectious diseases, extension from the pharynx or tonsils. The entire mouth and tongue or portions are red, dry, painful; later swollen and moist, with the tongue furred and tooth-indented. Duration usually less than a week. In children there may be some fever.

Treatment.—Remove the cause. Mouth wash of borax, boric acid, chlorate of potash, alum, tannic acid, argyrol, or tinct. myrrh, ℥j, sod. bicarb., gr. xxx, in water, ℥viij. In severe cases silver nitrate 0.5 to 1 per cent. locally. Fluid diet.

Aphthous Stomatitis.—Usually in infants with gastro-intestinal trouble or infectious diseases. Vesicles appear on the lips, cheeks, and edges of the tongue, rarely on the pharynx. These soon rupture, leaving small, tender, gray ulcers surrounded by a red area.

Treatment.—Treat the cause. Touch with silver nitrate; mouth wash as in acute stomatitis.

Ulcerative Stomatitis.—Most common in children. May be contagious. Is favored by unhygienic surroundings and

lack of cleanliness of teeth. The edges of the gums are red and swollen; they bleed, and become retracted from the teeth. Ulcers, covered with adherent, grayish membrane, form along the gums. There is acute stomatitis of the rest of the mouth. Breath is fetid, saliva profuse, submaxillary lymph nodes are enlarged, and mastication is painful. Constitutional symptoms may be severe.

Treatment.—Potassium chlorate, gr. xx, t. i. d.; mouth wash of potassium permanganate, 1 to 5000, for fetor; silver nitrate for ulcers; mouth wash of tinct. myrrh and sod. bicarb. (see Acute Stomatitis) or of potassium chlorate, 1 to 16 to 1 to 32.

Gangrenous stomatitis (**noma, cancrum oris**) occurs in children after acute infectious diseases, usually measles, or under unhygienic conditions. Beginning as an ulceration of the mucosa of the cheek, it may perforate or destroy a large part of the latter. Constitutional symptoms are severe. Mortality very high.

Treatment.—Cauterize under anesthesia; mouth washes of hydrogen peroxide and potassium permanganate; free stimulation and feeding.

Parasitic stomatitis (**thrush, soor, muguet, mycotic stomatitis**) is caused by growth of the fungus Saccharomyces albicans on the tongue and mouth of children or adults with catarrhal stomatitis. It forms white patches, and leaves, when scraped off, a slight erosion or natural surface. The mouth is dry. Chronic course; tendency to recurrence.

Treatment.—Remove growth repeatedly; cleanliness of mouth, mouth wash of sod. bicarb., ʒj in ℥viij; fresh air and feeding.

Mercurial stomatitis (**ptyalism**) occurs usually in persons especially susceptible to the drug even in small doses, as when given unsuccessfully or continuously as a diuretic. A metallic taste in the mouth is followed by tenderness when the teeth are brought together. The gums then become red, swollen, and tender; saliva profuse, with fetid odor.

Ulceration may occur. The tongue may be affected. The teeth may become loose or fall out. Mastication is painful.

Treatment.—Stop mercury as soon as the gums are tender; fluid diet; mouth wash of potassium chlorate, 1 to 16 or weaker. May give potassium chlorate internally. Saline catharsis. Hot baths and hot drinks to cause diuresis. Atropine, gr. $\frac{1}{100}$, t. i. d., for salivation. Opium for pain, if needed.

DISEASES OF THE STOMACH

ACUTE GASTRITIS

Etiology.—Called also **simple gastritis, acute dyspepsia,** or **acute gastric catarrh,** this is an acute inflammation of the gastric mucosa caused by ingestion of excessive, improper, or partially decomposed food, overindulgence in alcohol, or hot or cold drinks. There is often an inherited or individual predisposition, and it occurs often after fatigue or with acute infectious diseases.

Pathology.—The gastric mucous membrane is red, swollen, sometimes with small hemorrhages or erosions, and covered with mucus, especially at the pyloric end. The epithelium shows swelling and albuminous degeneration; the cylindrical cells, increased mucus; the submucosa, edema, and infiltration with leukocytes.

Symptoms.—*In mild cases:* Gastric distress, headache, malaise, thirst, nausea, eructations, and vomiting; tongue heavily coated, saliva increased. Vomiting usually relieves. The attack rarely lasts more than one day. In children there is usually diarrhea with colic; in adults there may be acute duodenitis, sometimes with slight jaundice. *In severe cases:* Onset at times with a chill, rise of temperature to 102° or 103°, headache, tongue furred, breath foul, anorexia, great thirst, slight abdominal distention, epigastric tenderness; repeated vomiting of food, then of bile-stained fluid with much mucus, usually no hydrochloric acid, but lactic and

fatty acids; constipation or diarrhea; urine of febrile type. The attack lasts one to five days. Repeated attacks may lead to chronic gastritis.

Diagnosis.—*Of severe cases:* From **meningitis, biliary colic, typhoid,** and gastric crises of **locomotor ataxia,** by absence of other symptoms of these disorders.

Treatment.—*Of mild cases:* Calomel or castor oil, rest for the stomach. *Of severe cases:* Empty stomach by giving warm water with emetic if necessary; calomel, gr. ij to iij, at night, saline in the morning; little or no food for a day or two; for thirst, cracked ice; for nausea and acid eructations, sodium bicarb. and bismuth subnitrate, āā gr. v, every three hours.

TOXIC GASTRITIS

Definition.—An intense gastric inflammation, often with hemorrhages and necrosis, caused by ingestion of strong acids or alkalies or non-corrosive poisons.

Symptoms are those of severe gastritis with intense pain, repeated vomiting, often of blood, abdominal distention and tenderness, scanty albuminous or bloody urine, collapse at once, feeble pulse, cold sweat, sometimes convulsions. Death may occur in a few hours or days, or recovery with cicatricial stenosis of the esophagus or gastric atrophy.

Diagnosis is made by the history, examination of the mouth for burns, and of the vomitus for poison.

Treatment.—Chemical antidotes; lavage if seen early; morphine for pain; absolute rest for stomach; subsequently demulcents.

CHRONIC GASTRITIS

A chronic digestive disorder characterized by increased secretion of mucus, changes in the gastric juice, weakening of the gastric muscle, and pathological changes in the mucosa.

Etiology.—(1) Excessive, irregular, or hasty eating; improper, greasy, or poorly cooked food, hot breads, pastry, ice-water, ice-cream, tea, coffee, alcohol, tobacco. (2) Cancer, ulcer, and dilatation of the stomach, portal congestion, cirrhosis of the liver, chronic valvular disease, chronic lung diseases with circulatory obstruction. (3) Anemia, chronic nephritis, gout, diabetes, chronic tuberculosis, etc.

Pathology.—Stomach usually enlarged, its walls thickened or very thin, mucosa gray or congested and with hemorrhages or erosions, and covered with tenacious mucus. The surface epithelium may be degenerated or detached. The glands are long, irregular, dilated, atrophic, or hyperplastic, with degeneration of their epithelium. Interstitial tissue is infiltrated with round or polyhedral cells and new fibrous tissue which contracts and may cause almost complete atrophy of the glands.

Symptoms.—Appetite variable, tongue coated, bad taste in the mouth; distress, feeling of fulness or pain in the epigastrium soon after eating; sometimes nausea, eructations of gas and at times of bitter fluid; vomiting, spontaneous or induced to relieve pain, from a few minutes to two hours after eating; vomitus partially digested food with mucus, sometimes bile; diffuse epigastric tenderness. Especially in chronic alcoholic gastritis the nausea, retching, and vomiting occur before breakfast. The abdomen is often distended, especially after meals, with constipation or diarrhea, abdominal distress, at times pain. Headache, inactivity, and mental depression are common. Analysis of a test meal may show diminished or normal hydrochloric

acid and presence of lactic and fatty acids. In advanced cases hydrochloric acid, pepsin, and rennin may be absent and secondary anemia and loss of weight are pronounced. Slow digestion and absorption favor retention and fermentation of stomach contents and muscular atony, but gastric motility may remain good. Symptoms vary greatly in intensity from time to time.

Diagnosis.—(1) From **gastric dilatation,** in which periodical vomiting of large quantities occurs, an outline of the naturally or artificially distended stomach can be determined by inspection, palpation, and percussion, and its capacity measured. (2) From **ulcer,** in which the pain immediately follows ingestion of food, tenderness is distinctly localized; hematemesis and increased hydrochloric acid are common. (3) From **cancer,** in which loss of flesh and strengtb is more rapid and eventually a tumor may be felt. Chronic gastritis usually accompanies each of these diseases.

Treatment.—*General:* Mental diversion, systematic exercise. *Dietetic:* Correction of hasty, irregular, and excessive eating and of use of food known to disagree, *e. g.*, greasy food, pastry, etc. It is sometimes better not to restrict too closely, as patients may have already nearly starved themselves. For severe cases, milk with Vichy or lime-water, ʒvj to viij, every two or three hours, skimmed milk or buttermilk, adding eggs, toast, scraped-beef sandwiches, etc., when symptoms decrease or if stools show curds, and later other articles. With eructations and flatulence avoid farinaceous food. *Medicinal:* Dilute hydrochloric acid, ♏x to xv, p. c.; silver nitrate, gr. ⅛ to ¼, t. i. d.; pepsin, gr. x to xv, one hour p. c., or better, pancreatin and sod. bicarb., āā gr. x to xx, fifteen to twenty minutes p. c., only in cases with atrophy of the mucosa; sometimes malt extract p. c. To increase secretory and motor activity, daily lavage with plain water or sodium bicarbonate, 5 per cent., if much mucus is present, or boric acid, 3 per cent., if much fermentation; sodium

bicarb., gr. x to xxx in warm water, ʒviij, a. c.; tincture of nux vomica and compound tincture of gentian, āā ℿv, a. c. For pyrosis, bismuth subnitrate and sodium bicarb. For nausea and vomiting, cerium oxalate, bismuth, dilute hydrocyanic acid, cocaine, carbolic acid. For constipation, cascara, podophyllin, salines, small doses of calomel, suppositories, or enemata.

DILATATION OF THE STOMACH (GASTRECTASIS)

Acute dilatation is rare. *Chronic dilatation* results from pyloric stenosis by cicatrization of an ulcer, by cancer, by pressure of tumors, or from atony due to repeated overdistention or chronic gastritis, etc. The walls are thin or hypertrophied, and the cavity is increased in size.

Symptoms.—In atonic cases there may be no symptoms, or those of dyspepsia, neurasthenia, and enteroptosis. In stenotic cases, besides the symptoms of the accompanying cancer, ulcer, etc., there is occasional vomiting of large and increasing quantities of sour stomach contents, which separate on standing into three layers—the uppermost, of brownish froth; the middle, muddy gray; the lowest, food. Hydrochloric acid may be absent, normal, increased, or diminished. Lactic and butyric acids and gases of fermentation, bacteria, moulds, yeast fungi, and sarcina ventriculi are present. Constipation, loss of flesh and strength and dryness of the skin result.

Physical Signs.—The outline of the distended stomach may be seen, also peristalsis from left to right. The thickened pylorus may be palpable. A splashing sound may be obtained several hours after eating by shaking the patient or by sudden pressure. Percussion aided by distention with air or gas defines the gastric outline.

Diagnosis.—By periodical vomiting of large quantities and physical signs of enlargement. Radiography after barium feeding shows enlarged gastric shadow and often ptosis.

Prognosis is good in simple atony, fair in simple stricture, bad in cancerous pyloric stenosis.

Treatment.—Food in concentrated form, avoiding starches and fats, limiting fluids. Daily lavage with warm water, sodium bicarbonate, or boric acid solution. Strychnine and iron. In cases with stenosis of non-malignant or doubtful origin, pyloroplasty, pylorectomy, or gastro-enterostomy. In malignant pyloric disease, gastro-enterostomy gives great temporary relief. It is also indicated in cases of simple atony if other measures fail.

PEPTIC ULCER

Etiology.—Simple, round, or perforating ulcer of the stomach or duodenum is due to disturbance, of nutrition of a limited area of the mucosa, as by thrombosis, embolism, or traumatism, followed by its digestion by gastric juice which is often superacid. Gastric ulcer is most common in women of twenty to thirty, is often associated with anemia; servant girls, shoemakers, and tailors are frequently attacked. Duodenal ulcer usually occurs in males and may follow large superficial burns.

Pathology.—Usually single and most often situated near the pylorus on the posterior wall near the lesser curvature; duodenal ulcers, in the first portion. The shape of an acute ulcer is a truncated cone whose apex extends to any depth and may even perforate the peritoneum; edges clean-cut, floor smooth, diameter one-quarter inch up. In chronic ulcers the border is irregular, edges are thickened and hard. Perforation may result in general peritonitis or localized peritonitis with adhesions to other organs, and sometimes local abscesses which may point into adjacent organs; erosion of bloodvessels causes hemorrhage; cicatrization may cause pyloric stenosis or hour-glass contraction. Lesions of chronic gastritis are often present.

Symptoms.—While pain, localized tenderness, vomiting, and hemorrhages are the most characteristic symptoms, there may be none during life or at least until perforation or hemorrhage occurs. Usually there are distress after eating, eructations, often nausea and *vomiting* of very acid fluid soon or several hours after eating, loss of weight, and anemia. *Pain* in the epigastrium and back is the most constant symptom, usually sharp, increased at once by food, relieved by vomiting; sometimes gnawing or burning when the stomach is empty. *Tenderness* is usually marked and distinctly localized, but pressure relieves the attacks of pain. *Hemorrhage* occurs in at least one-third of the cases, usually profuse, bright red, and fluid; if retained in the stomach, the blood becomes clotted and brown. Blood in the stools makes them tarry. Small, latent, "occult" hemorrhages may be detected only by chemical test of vomitus or stools, yet gradually cause anemia. *Perforation* is marked by sudden severe epigastric pain which may radiate, and collapse, followed by death or symptoms of general or localized peritonitis.

Course and Prognosis.—Often protracted; usually recovery; recurrence frequent. Death may result from shock or peritonitis after perforation, from hemorrhage or from starvation. Cicatrization may cause pyloric stenosis and dilatation of the stomach, or hour-glass stomach.

Diagnosis.—A test meal may show hyperacidity and chemical reaction for blood, and stools show occult blood. A silk thread, one end weighted and swallowed and retained in the stomach and duodenum overnight, may show, by distance of blood stain from the teeth, the presence and site of bleeding; stain at 40 cm. from teeth indicates ulcer of cardia; 44 to 54 cm., of lesser curvature; 56 to 58 cm., of pylorus; above 59 cm., of duodenum. Radiography after barium feeding may show persistent irregularity of gastric outline at site of ulcer. The gastroscope may reveal site of an ulcer near cardia. Exploratory laparotomy is often necessary. (1) From **gastralgia**, in which pain and tenderness

are not localized, the former is not so constantly influenced by food; dyspepsia is usually absent between attacks; anemia and hematemesis are absent; vomiting is rare. (2) From **hyperacidity**, in which the pain usually comes on one to three hours after meals and is diffuse. (3) From **acute gastritis**, which is usually febrile, of short duration, and vomitus contains no blood or little. (4) From **cancer**, which occurs usually after forty, with pain and vomiting often absent or irregular; blood in vomitus scanty and like coffee grounds; hydrochloric acid usually diminished or absent; tenderness often absent; cachexia and tumor in later stages. (5) From **chlorosis** with hematemesis, by absence of local symptoms and by blood examination. (6) From gastric crises of **locomotor ataxia** and (7) **cirrhosis of the liver**, by absence of other symptoms of those diseases. (8) From **gall-stone colic**, by rapid onset and ending and situation of pain and tenderness in the latter, with jaundice and enlarged gall-bladder in some cases. (9) Pain in the right hypochondrium two or three hours after meals, with sudden intestinal hemorrhage during apparent health, suggests **duodenal ulcer.**

Treatment.— *Dietetic:* Modified Lenhartz treatment—bed for three or four weeks; administration by nurse at hourly intervals, except during night, of teaspoonful doses of iced milk and beaten raw eggs, gradually increasing dose and total of food, adding sugar after third day, scraped beef on sixth, rice and soft-boiled egg on seventh, zwieback on ninth, chopped chicken and butter on tenth; after that, broiled chop, steak, or chicken, ice-cream. After two weeks, high protein diet, avoiding coarse food. Enemata for bowels. *Symptomatic:* For pain, sodium bicarbonate, Hoffmann's anodyne, chloroform, camphor water, or morphine with bismuth subcarbonate and sodium bicarbonate, or hypodermically if needed. For vomiting, rectal feeding, mustard paste over epigastrium, cracked ice, cerium oxalate, bismuth, chloroform, hydrocyanic acid. For hemorrhage, morphine hypodermically to keep quiet for several days, cracked ice

only by mouth, hypodermoclysis if needed; later, iron for anemia. For thirst, saline enemata. For perforation, persistent hemorrhage, or intractable cases, surgery.

CANCER OF THE STOMACH

Etiology.—Usually after the age of forty and primary.

Pathology.—Four types in order of frequency are: Cylindrical-celled, encephaloid or medullary, scirrhous, and colloid carcinoma. The cylindrical-celled and scirrhous are usually localized, most often near the pylorus; the other forms, diffuse. Usually primary with secondary growths in other viscera. With growths at the pylorus the stomach may be dilated; if at the cardia, contracted, with esophagus dilated. The cylindrical-celled is a firm ulcer, usually single, sometimes in large irregular masses with abundant stroma and masses of cells. The encephaloid forms large, soft, grayish, vascular masses, ulcerating early, with scanty stroma. The scirrhous is very hard, consisting chiefly of stroma. The colloid is very diffuse, soft, and its alveoli are distended with gelatinous material.

Symptoms.—The first are often indigestion for a few months, with anemia and loss of weight. In a well-marked case: Anorexia; nausea; irregular vomiting, especially in pyloric cases, in which it occurs usually one hour or more after eating; hematemesis, rarely profuse, usually of "coffee-ground" type; dragging, gnawing, or burning pain in the epigastrium, back, loins, or shoulders, usually increased by food; progressive loss of weight and strength; secondary anemia; cachexia; often some fever. There may be symptoms of metastases or gastric dilatation, or of peritonitis from perforation.

Physical examination of advanced case: Skin pale or yellowish; emaciation; abdominal skin often dry and withered. In the epigastrium may be seen fulness, peristaltic

movements, or metastatic nodules. A gastric tumor may be seen or felt, descending with inspiration, transmitting aortic pulsation, firm, smooth, or nodular. Epigastric tenderness. There may be signs of gastric dilatation. Gastroscopic examination may show presence of neoplasm near cardia. Radiograms after barium feeding may show persistent irregularity or "filling defect" of stomach wall. Ewald's test breakfast may show hypo- or anacidity and presence of lactic acid and Boas-Oppler bacilli, with occult blood in gastric contents and stools.

Course.—Usually death in twelve to eighteen months, occasionally in three or four. Metastases or perforation may hasten the end.

Diagnosis.—(1) From **chronic gastritis**, by cachexia, sometimes tumor, and lactic acid after test meal. (2) From **ulcer** which usually occurs in younger persons, may cause symptoms for years, is accompanied by severe gastralgia and profuse hemorrhages, with hyperchlorhydria, and may be differentiated only by laparotomy. (3) From **pernicious anemia** and that due to parasites, by examination of blood and feces.

Treatment.—Easily digestible food; lavage for vomiting or dilatation; morphine and bismuth subnitrate for pain. Exploratory operation may permit early diagnosis and so allow complete removal, the only hope of cure.

HEMORRHAGE FROM THE STOMACH

Gastrorrhagia may be due to (1) *local conditions*—ulcer, cancer, or traumatism of the stomach, acute or toxic gastritis, rupture of aneurysm or esophageal varices into stomach; passive congestion from portal obstruction, as in hepatic cirrhosis, valvular disease, or with enlargement of the spleen; portal thrombosis or obstruction by external pressure;

(2) *general diseases*—acute infections, such as smallpox, yellow fever, measles, or acute yellow atrophy; severe anemias, leukemia, purpura, hemophilia, scurvy. Hematemesis may occur after swallowing of blood from the nose or pharynx, in hysteria, etc.

Symptoms may be those of internal hemorrhage or vomiting of dark, usually clotted blood, sometimes of coffee-ground material, followed by melena.

Diagnosis is chiefly from **hemoptysis**, in which there is history or signs of pulmonary or cardiac disease; blood is coughed up, is bright red, usually fluid or frothy, alkalines; moist rales are usually heard, and sputum remains blood-streaked. In hematemesis the blood is usually dark, clotted, acid, with a history of hepatic, gastric, or splenic disease, traumatism, or acute infection; stools tarry. Blood is recognized by the microscope, spectroscope, or chemical tests.

NEUROSES OF THE STOMACH

A group of motor, secretory, and sensory gastric disturbances without organic lesion, and usually occurring in combinations, constitute *nervous dyspepsia.* The neurotic condition is inherited or acquired by overwork, worry, excitement, hurried or irregular meals, or eye strain.

1. **Motor Neuroses.—Supermotility**, or **hyperkinesis**, abnormal motor activity, the stomach emptying its contents into the intestine too soon, is usually secondary to superacidity or supersecretion. No special symptoms.

Peristaltic unrest, supermotility evidenced by loud gurgling or rumbling after food or emotion, is often felt subjectively. Peristalsis may extend to the intestine or be reversed.

Nervous eructations, belching in rapid succession and with loud noise of large quantities of air swallowed involuntarily; common in hysteria.

Pyrosis or **heartburn** is a burning sensation due to regurgitation of normally or hyperacid chyme into the esophagus.

Rumination or **merycismus** is regurgitation of the food in small quantities into the mouth, where it is again chewed, as in cattle. It occurs in hysterical, idiotic, or epileptic persons, or may be hereditary or imitative.

Nervous vomiting, usually in hysterical women, is regurgitation of food after meals or at irregular times, without nausea, gagging, or effort.

Spasm of the cardia, usually painful, occurs in hysterical subjects after ingestion of hot or cold drinks or passage of a stomach-tube. It may cause **pneumatosis**, gastric distention.

Relaxation of the cardia is shown by regurgitation, eructation, or rumination.

Spasm of the pylorus, often painful, usually follows superacidity, ulcer, or chronic gastritis.

Pyloric insufficiency allows too early escape of gastric contents into the intestine. No characteristic symptoms.

Atony of the stomach, usually due to habitual overeating, organic gastric disease, or chronic wasting affections, is sometimes a neurosis. *Symptoms:* Gastric fulness and weight, eructations.

2. **Secretory Neuroses.—Hyperacidity, superacidity,** or **hyperchlorhydria**, most common in young chlorotic girls, is the secretion of abnormally acid gastric juice during digestion. One to three hours after meals there is a feeling of weight or burning and tenderness in the epigastrium, usually acid eructations, sometimes vomiting. Temporary relief is afforded by vomiting, food, fluids, or alkalies.

Supersecretion.—Intermittent: Increased flow of gastric juice, usually superacid, which may be independent of food. Gnawing in the stomach, with headache, is followed by vomiting of strongly acid watery fluid. **Continuous**: Pyloric spasm, followed by gastric dilatation, results from constant supersecretion. There are gastric distress and acid eructations. These, with vomiting, may occur at night or early in the morning.

Subacidity or **hypochlorhydria** is subnormal acidity during gastric digestion, without organic lesions. The ferments are present. May be no symptoms unless there is atony; or gastric distress soon after eating, followed by eructations.

Achylia Gastrica.—Gastric secretion, including ferments and mucus, permanently absent; motor function usually good. Symptoms may be slight or absent unless caused by associated atony, dilatation, and fermentation, or by intestinal indigestion—pain, nausea, vomiting, eructations, flatulence, diarrhea. Diagnosticate by repeated gastric analysis and exclusion of cancer and chronic gastritis.

3. **Sensory Neuroses.—Hyperesthesia.**—Pressure, burning, pain, or distress in stomach after ingestion of any food or drink, yet some fluids may be taken as medicine without complaint, and digestive functions are otherwise normal. Simulates organic disease; may lead to great emaciation. Other hysterical symptoms are usually present.

Gastralgia, or **gastrodynia**, is most common in constipated, neurasthenic women. Sudden onset of intense epigastric pain radiating to the back and sides, usually independent of taking food; may recur periodically, often at night; may be relieved by food or moderate pressure. Tenderness on deep pressure. Must be differentiated from gastric ulcer and cancer, pyloric stenosis, gall-stone, intestinal and renal colic, gastric crises of locomotor ataxia, and angina pectoris. Periodicity, independence of diet, freedom between attacks, and absence of characteristics of these other painful conditions are suggestive.

Nervous anorexia is hysterical absence of hunger. All food is refused; may terminate in death. Patient is not alarmed as in anorexia from organic disease.

Bulimia, or **hyperorexia**, is excessive hunger occurring in attacks in cases of hysteria, gastric disorders with superacidity, diabetes, epilepsy, brain tumor, etc. Enormous quantities of food may then be taken. Atony, dilatation, or chronic gastritis may result.

Polyphagia is a condition requiring excessive amounts of food to satisfy hunger. It occurs in short attacks or is chronic.

Akoria is absence of sense of satiety after eating.

Parorexia, perverted appetite in the insane or hysterical or in pregnant women.

Diagnosis of motor and secretory neuroses often requires use of the stomach-tube and analysis of test meals.

Treatment.—*General:* All gastric neuroses require treatment of the underlying nervous condition—by improving the general physical and mental condition by rest, with change of scene and of the mode of life; hydrotherapy, regulation of exercise, manner of eating, fresh air, etc. The Weir-Mitchell rest cure in obstinate hysterical cases, especially nervous anorexia and vomiting. Iron and arsenic for anemia; bromides temporarily in nervous cases. **Motor neuroses**: For *increased motility,* avoid irritating food and rapid eating; for *diminished motility,* strychnine, highly seasoned food; for *vomiting,* as in ulcer of stomach (p. 156); for *atony,* as for dilatation (p. 154). **Secretory neuroses**: For *hyperacidity,* diet chiefly of meat and milk, avoiding irritating and seasoned foods, starches, and alcohol; calcined magnesia and sod. bicarb., āā gr. xv, two hours p. c. or when distressed. For *supersecretion,* lavage with alkaline solution or silver nitrate, 1 to 1000, or give albuminous food or alkalies; for continuous supersecretion with atony and dilatation, lavage as above, alkalies, especially two hours p. c.; diet as in hyperacidity, given frequently in small quantities. For *subacidity,* restrict meat; tinct. nucis vom., ♏v, with tinct. gentian. comp., q. s. ad ℥j, a. c.; or sod. bicarb., gr. x to xx in glass of hot water, a. c.; or dilute hydrochloric acid, ♏x to xx, p. c. For *achylia gastrica,* large meals; restrict meats; lavage; bitter tonics before meals; dilute hydrochloric acid, ♏x to xx, p. c.; preparations of Carica papaya, a. c. **Sensory neuroses**: For *hyperesthesia,*

silver nitrate, gr. $\frac{1}{3}$ to $\frac{1}{2}$, on empty stomach. For *gastralgia*, same; heat locally; Hoffmann's anodyne, ʒss to j, or chloroform, ♏x to xx; opium if needed.

DISEASES OF THE INTESTINES

ACUTE CATARRHAL ENTERITIS (DIARRHEA)

Definition.—An acute catarrhal inflammation of any portion or the whole of the intestine. It includes the terms **duodenitis, jejunitis, ileitis, colitis,** and **proctitis,** which cannot be differentiated with certainty.

Etiology.—*Primary cases:* (*a*) Improper or excessive food, including green or overripe fruit; (*b*) toxic substances—organic, such as decomposed milk or meat either fresh or canned, and inorganic, such as arsenic, mercury, colchicum; (*c*) absence of pancreatic secretion or increase or decrease of bile in the intestine; (*d*) exposure to cold, wet, or drafts. *Secondary cases:* (*a*) Stomach disorders preventing the completion of gastric digestion; (*b*) extension of adjacent inflammation as in peritonitis, strangulated hernia, and carcinoma; (*c*) extensive burns; (*d*) acute infections, such as typhoid, malaria, tuberculosis, cholera, dysentery, and septicemia; (*e*) circulatory disturbance from portal obstruction or chronic pulmonary or cardiac disease, though this usually causes chronic diarrhea.

Pathology.—During life the intestinal mucosa is red, swollen, covered with mucus and degenerated epithelium. Solitary and agminated lymph nodules may be swollen from hyperplasia and sometimes ulcerated.

Symptoms.—Usually sudden onset of colicky intestinal pain, moving from one part of the abdomen to another with rumbling noises. Pain is intermittent and followed at intervals by sudden extreme desire to empty the bowels. The

stools are from four to twenty a day, watery or like gruel, sometimes containing mucus or undigested food. If the duodenum alone is involved there may be no diarrhea; if the lower part of the large intestine, there is tenesmus. Pain is temporarily relieved by defecation. The tongue becomes dry, abdomen tympanitic. There is thirst, but little or no fever. If the stomach is involved there may be nausea and vomiting. The attack lasts two or three days, more rarely a week. It may become chronic. In very acute and severe cases, sometimes called **cholera morbus**, or **cholera nostras**, there is occasionally a prodromal period with nausea and malaise; but the invasion is usually sudden, with nausea, vomiting, and cramp-like abdominal pain. The vomitus consists of normal stomach contents, then of watery fluid, sometimes bile-stained. Stools are at first diarrheal, later approach or assume the appearance of rice-water. Repeated vomiting and purging, with severe cramps, cause the patient to resemble one with true cholera (p. 74). After one or two days recovery usually begins and requires about two weeks. In severe cases a state of collapse is followed by death within two to four days.

Diagnosis.—Duodenitis is suspected when acute gastritis is followed by jaundice. Involvement of the rest of the small intestine is characterized by intensity and persistence of colic, moderate diarrhea, little mucus, and presence of undigested food in stools. Colitis may cause little or no pain or only just before and during defecation; stools are of uniform soupy consistence, often with much mucus. If the rectum is affected there is tenesmus. Acute catarrhal enteritis must be differentiated from (1) **nervous diarrhea**, which follows emotion in nervous persons or occurs immediately after eating; (2) **acute poisoning** by arsenic, bichloride, or fungi, by the history and analysis of gastric contents. The severe cases called cholera morbus are differentiated from (3) **Asiatic cholera** by absence of the spirillum of cholera from the stools.

Prognosis.—Primary cases usually recover in a few days; secondary often become chronic.

Treatment.—Rest in bed; no food at first; then only small quantities of milk with Vichy or lime-water every two hours for one or two days. Cracked ice for thirst. Unless purgation has been thorough, give castor oil, ℥ss to j, or calomel, gr. ij to iij, with sod. bicarb., gr. x, followed in eight hours by a saline cathartic. After catharsis, bismuth subnitrate, gr. xxx, or bismuth subgallate, gr. v, every three or four hours. For severe pain, hot-water bag on abdomen, morphine if necessary; if in the large intestine, enemata of starch-water, ℥ij, with tinct. opii, ♏xx. In severe cases resembling cholera, morphine hypodermically; heat to abdomen and extremities; for thirst, cracked ice by mouth or normal salt solution subcutaneously; for prostration, strychnine and brandy.

CHRONIC CATARRHAL ENTERITIS

Definition.—Chronic catarrhal inflammation of part or the whole of the intestine.

Etiology.—May follow acute catarrhal enteritis. Often results from chronic congestion due to portal obstruction, as in cirrhosis of the liver, or to cardiac or pulmonary diseases. Occurs in chronic affections, such as malaria, tuberculosis, and chronic nephritis, and with tuberculous or carcinomatous ulcers of the intestine.

Pathology.—The intestinal mucosa may be covered with mucus and show ecchymoses, pigmentation, and erosions. The submucous and muscular layers may be thickened by hyperplasia of connective tissue, or the same process may cause atrophy of the glands in the mucosa and muscular layer and so thinning of the intestinal wall.

Symptoms may develop gradually in cases of portal obstruction or other chronic disease, or follow acute catarrhal enteritis. There is diarrhea with or without colic, often

alternating with constipation. Stools vary greatly in size, color, and presence of mucus and undigested food, the last being found in cases involving the small intestine, while watery stools with much mucus show involvement of the colon. There may be abdominal distention, rumbling, and flatulence. Loss of flesh and strength, anemia, and hypochondriasis occur.

Diagnosis.—As in acute catarrhal enteritis (p. 165).

Prognosis.—Very persistent. May recur. Death may result from exhaustion or intercurrent disease.

Treatment.—Rest, fresh air, warm clothing, moderate exercise. Peptonized or plain milk for several weeks, gradually adding eggs, rare beef, toast, rice, etc., but avoiding starches. Pancreatin, gr. v, with sod. bicarb., gr. x, after meals; bismuth subgallate, gr. v, or bismuth subnitrate, gr. xxx, every four hours. For flatulence, salol, gr. v, every three hours. To empty bowels occasionally, calomel followed by saline, or castor oil, ℥ss to j, followed by castor oil, ♏v, with salol, gr. v, every three hours. In persistent cases, silver nitrate, gr. ¼, p. c. For colitis, irrigations with normal saline or weak silver nitrate solution. For anemia and loss of weight, cod-liver oil, iron, and arsenic.

DIPHTHEROID OR CROUPOUS ENTERITIS

An inflammation of any portion of the intestine with formation of membrane resembling that of diphtheria and in areas of various sizes and thicknesses. It usually occurs in acute infectious diseases—typhoid, pneumonia, pyemia, scarlet fever, etc.; in poisoning by mercury, arsenic, or lead; or as a terminal process in cancer, cirrhosis of the liver, chronic nephritis, etc. The **symptoms** are those of acute enteritis, varying with the cause and the extent and situation of the lesions. The **diagnosis** can be made only by finding the membrane in stools. **Treatment** is that of the etiological factor and as in acute catarrhal enteritis (p. 165).

ULCERATIVE ENTERITIS

1. **Simple ulcerative colitis** occurs usually in men past middle age. The intestinal lumen is enlarged or contracted, the wall hypertrophied, the mucosa thickened, with ulceration of variable extent, and sometimes intervening polypoid growths. The **symptoms** are lienteric diarrhea, often alternating with constipation, gradual loss of flesh and strength, with a cachectic appearance. Perforation or death may occur or the disease may become chronic.

2. **Follicular ulcers** are most common in children and as a secondary lesion in adults. They are small, with sharply cut edges, and usually limited to the follicles. **Stercoral ulcers** occur in the colon from pressure of hardened feces in chronic constipation. **Perforative ulcers** are caused by pressure or extension of newgrowths, abscesses, or inflammatory processes from adjacent organs. **Traumatic ulcers** result from the presence of foreign bodies of corrosive poisons. **Cancerous ulcers** are due to ulceration of neoplasms in the wall. **Solitary ulcer**, resembling the peptic ulcer, may cause perforation of the colon or cecum. Other forms are **typhoid, tuberculous, syphilitic,** and **duodenal.**

Symptoms are variable. Diarrhea is common if the ulcers are in the colon or lower, often absent if in the small intestine. Stools may contain fragments of tissue, pus, and blood, the last bright if from the sigmoid or rectum, dark if from higher up. Localized pain and tenderness may occur but not invariably. Perforation is followed by symptoms of general peritonitis or localized abscess.

Treatment.—Same as for chronic catarrhal enteritis (see p. 166). If the large intestine is involved, irrigations with 0.5 to 1 per cent. silver nitrate are valuable, or other astringents may be used, sulphate of copper or of zinc, etc.

INTESTINAL HEMORRHAGE

Etiology.—(1) General blood diseases with hemorrhages from mucous membranes—leukemia, pernicious anemia, purpura, scurvy, hemophilia; and severe infections—smallpox, malaria, bubonic plague, yellow fever. (2) Vicarious menstruation. (3) All causes of nasopharyngeal or gastric hemorrhage, the blood later entering the intestine; (4) traumatism without or within the intestine, ulceration (see Ulcerative Enteritis, p. 167), newgrowths, corrosive poisons, intestinal parasites (Ankylostomum duodenale), passive congestion from cirrhosis of the liver or valvular disease, embolism, or thrombosis of mesenteric vessels, polypi, fissure or fistula in ano, internal hemorrhoids.

Symptoms.—Blood is bright and fluid if from the lower part of the intestine; dark, clotted, and often mixed with the feces, if from high up.

APPENDICITIS

Inflammation of the vermiform appendix. The chief varieties are **catarrhal** or **obliterative, ulcerative, necrotic,** the last two often accompanied by local or general peritonitis.

Etiology.—Usually in young adults, especially males; heavy lifting, local traumatism, indiscretions in diet, foreign bodies in intestine, constipation, previous attacks of appendicitis, anything narrowing the lumen of the organ, bacterial invasion.

Pathology.—1. **Catarrhal or Obliterative Type.**—The mucosa is thickened and desquamates to a variable degree. Apposition of surfaces thus denuded results in their adhesion, and partial or rarely complete obliteration at one or more points of the lumen, which may contain fecal concretions. The entire wall becomes thick and stiff. Ulceration, perforation, or suppuration may follow (**purulent appendicitis**).

2. **Ulcerative Type.**—As a sequel to catarrhal appendicitis, or as the result of the presence of concretions, foreign bodies, or pyogenic bacteria, one or more ulcers of the appendix may form. They may extend to any depth, causing adhesive peritonitis when the surface of the appendix is reached, thus binding the appendix to any adjacent structure. If perforation occurs without complete limitation by such adhesions, acute general peritonitis results; if adhesions are sufficiently extensive, a localized abscess forms, whose site depends upon the position of the individual appendix. It is usually midway between the umbilicus and anterior superior spine of the ilium; but may lie in the cavity or over the brim of the pelvis or in any part of the right lower quadrant of the abdomen. Perforation may take place into retroperitoneal tissues and burrowing occur in the direction of least resistance. Local abscesses may rupture into the hollow viscera or externally, and spontaneous recovery ensue.

3. **Necrotic type** may possibly be primary, but usually follows one of the preceding forms. Localized necrosis results in perforation; general necrosis, in complete sloughing off of the appendix; each is followed by acute localized or generalized peritonitis. The pyogenic streptococcus and staphylococci and the colon bacillus are often found in the pus.

Symptoms.—Preceded in some cases by colic-like pains in the right inguinal region, loss of appetite, nausea and vomiting, constipation or diarrhea, there is usually sudden onset of **pain**, often sharp and severe, in the whole or any part of the abdomen, later becoming localized in the right iliac fossa. Pain is soon followed by **fever**, usually 100° to 102°, higher in children and in some severe cases, though it may be absent even with a local abscess. There are usually nausea and vomiting, nearly always **constipation.** Pulse corresponds with the degree of fever.

Physical Examination.—Patient lies on the back or right side with the right knee drawn up. At first there are only **muscular rigidity** of the right side of the abdomen and ten-

derness on deep pressure over this region, especially **localized tenderness** over the appendix, usually at McBurney's point midway between the umbilicus and right anterior superior iliac spine. Later a more or less definite **mass** may be felt in the region of the appendix, though in acute perforative cases without local reaction there may be none. Vagina and rectal examinations rarely are of value, except rectal in children. There is usually leukocytosis, especially with suppuration.

Course.—(1) **Gradual recovery** is usual in mild cases, symptoms diminishing in three to seven days, and convalescence being complete in ten days to three weeks, leaving in some cases an induration or mass with tendency to recurrence. (2) **Recurrent appendicitis** is the name given to cases in which, the induration persisting or not, there are repeated attacks at long or short intervals; **relapsing,** to those in which symptoms merely diminish, with exacerbations. Eventually complete recovery may occur, but any attack may prove fatal. (3) **Local abscess formation.** With perforation from ulceration or necrosis, localized peritonitis may wall in the resulting collection of pus. There are usually gradual increase in size of the mass and in the fever and other general symptoms, though typical symptoms may be absent. Death may result from septicemia or general peritonitis, or recovery may follow pointing of the abscess into a hollow viscus. (4) **General peritonitis** may result from rupture of a localized abscess or from direct perforation of the appendix without the formation of adhesions. The first symptoms may thus be due to general peritonitis, the existence of this condition being suggested chiefly by the intensity of the initial symptoms. Pain and tenderness become general, abdomen distended and tympanitic, with persistent nausea and vomiting dry tongue, rapid pulse, and a temperature which may be as low as 100° or as high as 105°. The patient lies on the back with knees drawn up and the facies is anxious and pinched. These typical symptoms are very variable, and many, espe-

cially fever, may be absent in even severe cases. Unless treated, death usually results in two to ten days.

Diagnosis.—By sudden onset of localized pain and tenderness and sometimes induration or a tumor at McBurney's point, rigidity of the right rectus with fever, vomiting, constipation, or diarrhea. From (1) **intestinal colic**, in which the objective signs are not localized in the appendiceal region. (2) **Hepatic colic**, in which the local symptoms and signs are usually higher up, the pain radiates upward or backward, and there may be jaundice. (3) **Renal colic**, in which the pain may radiate toward the bladder, and there is usually hematuria. (4) **Dietl's crises**, which cease when the movable kidney is replaced. (5) **Pyonephrosis, perinephritic abscess**, or **tumor of the kidney**, by urinary examination or laparotomy. (6) **Perforated gastric, duodenal**, or **typhoid ulcer**, by previous history and absence of the local signs. (7) **Acute intestinal obstruction**, which is accompanied by fecal vomiting; **intussusception**, by bloody stools with mucus and tenesmus; **fecal impaction**, in which the tumor is hard or doughy and local signs are less severe. (8) **Mucous colitis**, in which the patients are neurotic and membranous casts of the intestine are passed. (9) **Acute hemorrhagic pancreatitis**, in which the pain, tenderness, and swelling are in the upper part of the abdomen. (10) **Typhoid fever**, by history, severity of local symptoms and physical signs, including rigidity and presence of a tumor, presence of leukocytosis, absence of Widal reaction, and lack of the high and regular fever of typhoid with its slow pulse. (11) **Psoas abscess**, in which the thigh is held in flexion, adduction, and inward rotation, a spinal lesion can be detected and a fluctuating tumor felt. (12) **Ruptured extra-uterine pregnancy**, in which the local symptoms are lower and signs of pregnancy are found in the breasts, cervix, etc. (13) **Pyosalpinx, pelvic peritonitis, dysmenorrhea**, and other pelvic disorders, by history and vaginal examination. (14) **Hysteria**, by careful history and examination.

Prognosis.—The majority recover; but any mild case may suddenly become serious. The gravity of a case cannot always be judged by the intensity of the symptoms. The results of operation are favorable, especially in those operated upon in the interval after subsidence of the acute attack or before the onset of general peritonitis. A large number of the cases with general peritonitis die.

Treatment is essentially surgical. In the beginning and in mild attacks the patient should be kept in bed, with fluid diet, and an ice-bag over the appendix. Opium must be avoided, as it masks symptoms of danger. Cathartics should not be given; enemata may be employed only if operation is deemed unnecessary. Mild cases *may* be tided over by the above methods, and operated upon in the interval. If symptoms are severe and distinct improvement does not occur within twelve hours at the latest, operation is advisable. The only other class of cases in which operation should be postponed is that in which the patient's condition is such that an operation would probably prove fatal. In these sufficient improvement sometimes occurs under palliative treatment to permit abdominal section later. Severe onset or signs of perforation or general peritonitis are indications for immediate operation.

INTESTINAL OBSTRUCTION

Etiology.—Internal causes: *Impacted feces; foreign bodies,* such as false teeth, roundworms; *gall-stones; enteroliths; stricture,* rarely congenital, usually acquired, and either cicatricial or cancerous; *intussusception,* one portion of intestine becoming invaginated into the adjoining part, occurs in young children often after habitual constipation or diarrhea. **External causes**: *Strangulation* of internal hernia in peritoneal pouches or openings by bands, by adhesions, by slits in omentum or mesentery, or by diverticula; *volvulus* is most common in males between thirty and forty,

twisting of the gut being favored by long mesentery, traction by adhesions, and fecal masses in its lumen; *tumors* in pelvis may cause obstruction by external pressure; *adhesions*, causing kink or sharp bend. The **most common causes of acute obstruction** are intussusception, strangulation, and volvulus; of **chronic obstruction**, stricture and impaction of feces.

Pathology.—In **acute obstruction** the intestine above the constriction is much dilated, thin, congested; at the constriction there may be perforation, adhesions, or other signs of peritonitis; below it is usually small, pale, and empty. In **chronic obstruction** the intestine above is moderately dilated and its wall thickened by muscular hypertrophy; below it is small and its wall atrophied. In obstruction by strangulation or volvulus the strangulated portion of gut and mesentery is congested; then peritonitis and necrosis occur. In intussusception the portion involved is congested and swollen; peritonitis follows, the exudate binding the invaginated portions together; rarely spontaneous cure then occurs by sloughing of the invaginated portion and union of the middle and outer layers.

Symptoms.—(1) **Acute obstruction**: Sudden onset of **abdominal pain**, colicky, then continuous and severe; repeated **vomiting**, at first of stomach contents, then of bile-stained fluid, finally of **fecal matter**; absolute **constipation** after the intestine below the obstruction has been emptied; **abdominal distention** and tympanites most marked when the obstruction is in the lower bowel. In intussusception a sausage-shaped tumor may be felt. The face is pinched and anxious, skin cold, tongue dry, thirst marked, urine scanty or suppressed, pulse rapid and feeble, temperature often subnormal. Unless relieved, death occurs in three to six days. (2) **Chronic obstruction**: Usually caused by fecal accumulation or stricture, cicatricial or malignant. There is chronic constipation, with stools at long intervals or frequent passages of mucus or of liquid fecal matter from above the obstruction. Hard fecal masses may be felt by abdominal or rectal examina-

tion. There may be vomiting, severe cramp-like abdominal pain referred to the site of obstruction, or general abdominal distention, and visible peristaltic movements of the hypertrophied gut above the obstruction. Ulceration may occur from pressure (see Stercoral Ulcer, p. 167) and lead to perforation or peritonitis. Anemia and emaciation increase, and if obstruction becomes complete there is feculent vomiting, and death usually occurs with symptoms of acute obstruction.

Diagnosis.—1. **Of the Situation of the Obstruction.**—If in the **duodenum** or **jejunum**, the upper part of the abdomen is distended, collapse occurs rapidly with marked diminution of urine, vomiting occurs early, but vomitus is not fecal. If in the **ileum** or **cecum**, the umbilical region is most distended, the coils of small intestines standing out above one another like steps of a ladder; vomiting occurs early and becomes fecal. If low down in the **colon**, this may stand out distinctly around the sides of the abdomen, tympanites becomes general and marked, the course is slower, fecal vomiting occurs only late, urine is little reduced, collapse is delayed, tenesmus is common. The obstruction may be felt by abdominal, rectal, or vaginal examination, or its distance from the anus measured by passage of a soft rectal tube or by measurement of the amount of water which the bowel will hold below the obstruction. The site of obstruction may be definitely located by radiograms taken immediately and also six, twelve and twenty-four hours after barium feeding, and after large enemata of barium.

2. **Of the Nature of the Obstruction.**—**Intussusception**: Common in children; tenesmus with blood and mucus in stools and sausage-shaped tumor, usually in the region of the transverse colon, are significant. The tumor may be felt by rectal examination. **Strangulation** occurs usually in adults who have had peritonitis or abdominal operations causing adhesions. **Volvulus**: Patient usually over thirty, sigmoid often involved, with pain, tenderness, and tympany

often localized there at first; vomiting late and not fecal. **Impacted feces**: Detected by palpation at sigmoid, cecum, or hepatic or splenic flexure, or by rectal examination. **Gall-stones**: History of previous biliary colic, sometimes jaundice; vomiting occurs early.

Differential Diagnosis.—(1) From **perforative peritonitis**, in which pain is more constant, tenderness present, abdomen rigid from the first, peristalsis is absent, tympanites uniform, fever is present until collapse, vomiting is less frequent and rarely fecal; constipation not absolute, as it is in all acute obstructions except intussusception. (2) From **strangulated external hernia.** (3) From **appendicitis**, in which there is localization of pain and tenderness with fever. (4) From **acute hemorrhagic pancreatitis**, of which symptoms are the same as of obstruction of the upper part of the small intestine. (5) From **lead colic**, by history of occupation and absence of lead line in gums, anemia, wrist-drop. (6) From **renal** or **hepatic colic** by history, absence of distention, and relief of constipation by enemata in these cases. (7) From **thrombosis of mesenteric vessels.** If in doubt as to nature or site of obstruction make exploratory incision soon.

Prognosis.—Spontaneous recovery is rare, and in cases operated upon much depends upon *early intervention.* Intussusception *may* recover without this; after the fourth day is usually fatal even if operated upon. Obstruction by gall-stones or feces is much less serious.

Treatment.—**Acute obstruction**: High enema repeated once or twice after a few hours, to distend bowel and reduce intussusception or free strangulated intestine; rectal feeding, only ice by mouth. Impacted feces may be removed digitally through the anus. If too high, give a high enema of cottonseed oil, castor oil, and mag. sulph., āā ℥j; ox gall, ℥ss; turpentine and glycerin, āā ℥ij, to be retained fifteen to thirty minutes, then followed by a high soapsuds enema. If partially effectual, repeat. *Never give cathartics.* Avoid opium as much as possible. For vomiting, repeated lavage;

for tympanites, turpentine stupes. If medical treatment is not successful within twenty-four hours, early surgical intervention is imperative; never delay beyond the third day; operate earlier if possible. In **chronic obstruction**: Laxative diet; enemata; cascara; aloes, belladonna, and strychnine, etc. If it becomes complete, operate.

CONSTIPATION (COSTIVENESS)

Definition.—Prolonged retention of feces or habitual difficulty in defecation.

Etiology.—(*a*) **General Causes.**—Female sex, advanced age, sedentary habits, irregularity of hours for defecation; persistent use of cathartics; improper diet, leaving either too much or too little residue or poorly cooked and chewed, lack of fluids; many general diseases, *e. g.*, anemia, neurasthenia, hysteria, diabetes, acute infectious diseases, chronic cardiac, hepatic, gastric, and intestinal disorders; diseases of the brain and spinal cord.

(*b*) **Local, Intestinal Causes.**—Intestinal atony; pressure of tumors, gravid or retroverted uterus; fecal masses, foreign bodies, or strictures; diminution of digestive secretions, especially bile; persistent intestinal spasm.

(*c*) **In Infants.**—Congenital defects, lack of fat in milk, poor digestion, insufficient consumption of water.

Symptoms.—In some persons fecal retention for even a day causes great languor, headache, backache, loss of appetite, coated tongue, bad breath, abdominal distress. In others the bowels may not move for a week, yet cause slight symptoms. Dysmenorrhea is common. Hemorrhoids and periodical diarrhea frequently occur in chronic cases; ulceration and perforation or intestinal obstruction less often. Sallow complexion with acne in chronic cases.

Treatment.—*Prophylactic* and in *mild cases:* Systematic hour for defecation regardless of lack of desire; moderate exercise; sufficient fluid in diet, rough foods, such as oatmeal

and Graham bread; fruit. Glass of cold water on rising, abdominal massage following the colon, bandage to raise the abdomen if pendulous. If defecation does not occur spontaneously after waiting a few minutes, at the regular hour, a plain or soapsuds enema or glycerin suppository may be used. An enema of cottonseed oil, ℥iv to vj, given every night and retained until morning, often relieves even obstinate constipation. *Medicinal:* Avoid drugs or use the mildest which are efficacious, such as liquid petrolatum, ℥ss, b. i. d. or q. n. If given they should be varied frequently to avoid a habit and alternated in some cases with glycerin suppositories or enemata. Decrease the dose whenever possible. In all cases try to discover and remove the cause, giving, for example, iron if anemia is the etiological factor. If constipation is severe or prolonged it may be well to give a single dose of calomel, gr. ij to v, with sod. bicarb., gr. x, followed in the morning by Rochelle salt, ℥ss to j, or other saline, before beginning regular medication. The best drugs for constipation, to be given at bedtime, are fluidextract of cascara, ʒss to j; compound licorice powder, ʒj, or pills containing combinations of aloin, belladonna strychnine, podophyllin, and colocynth, such as aloini, ext. belladonnæ, ext. nucis vomicæ, res. podophylli, āā gr. $\frac{1}{10}$; or ext. cascaræ, gr. ij; ext. belladonnæ, gr. $\frac{1}{16}$; res. podophylli, gr. $\frac{1}{8}$; give 1 to 3 at night. In more severe cases, pulv. aloes, gr. j; ext. nucis vomicæ, gr. $\frac{1}{4}$; ext. belladonnæ, gr. $\frac{1}{6}$; ext. colocynth. comp., gr. ij; 1 or 2 at night. In children *avoid drugs.* Increase cream in the milk, give water, or barley- or oatmeal-water, regularity of habits, Castile soap suppository, enema, abdominal massage. Castor oil, citrate of magnesia, if drugs are needed. In older children, fruit, oatmeal, etc.

ENTEROPTOSIS (GLENARD'S DISEASE)

A group of symptoms due to prolapse of the intestines and usually of the other viscera on account of relaxation of their

natural supports. Descent of the stomach is called gastroptosis; of the spleen, splenoptosis; of the kidney, nephroptosis; of the colon, coloptosis.

Etiology.—Congenital relaxation of peritoneal attachment; loss of support by abdominal wall due to ascites, repeated pregnancies, ovarian cyst, etc.

Symptoms.—(*a*) In cases due to abdominal relaxation after distention there may be no symptoms, but the abdomen is pendulous, covered with striæ. Inspection and percussion after gastric inflation show this organ low down, with but slight peristalsis. (*b*) In another group, usually young women, there occur spontaneously or after a wasting illness, neurasthenic and dyspeptic symptoms with physical signs of enteroptosis. The chief complaints are dragging pain in the back, dyspeptic symptoms, constipation, and general debility. Examination shows a neurotic patient with pulsating abdominal aorta; stomach when dilated is seen lower than normal; kidney often movable (see Movable Kidney, p. 303) and displaced, liver slightly so, and spleen sometimes markedly so. The colon may be felt below the umbilicus; barium radiograms show gastro-intestinal ptosis.

Treatment.—None if symptoms are absent. Never inform patient of abnormal mobility of viscera. If symptoms occur, abdominal bandage to support viscera; fatten patient if enteroptosis is due to wasting illness. Treat constipation (p. 176) and nervous dyspepsia (p. 162). For neurasthenia, general hygienic and hydrotherapeutic measures with tonics. Surgical treatment is rarely necessary.

ENTERALGIA (INTESTINAL COLIC)

Etiology.—*Predisposing:* Poor general condition, worry, mental overwork, chronic gastric disease, and neurotic disposition. *Exciting causes:* Exposure; intestinal irritants, *e. g.*, gas, masses of feces, undigested or irritating food, cold

drinks, green fruit; mineral poisons, such as lead and arsenic; bacterial, such as cholera; cerebral and spinal diseases, hysteria, locomotor ataxia.

Symptoms.—Intermittent pain, usually in the umbilical region, radiating or moving from place to place, dull or sharp, relieved by pressure of bending forward. Abdomen is distended or retracted. Face pale, sweating often profuse. Duration, few minutes to many hours, ending gradually or suddenly, often after passage of gas or feces.

Diagnosis.—(1) From **intestinal obstruction,** by absence of fecal vomiting and persistent constipation. (2) From **biliary** and **renal colic,** by different location and type of pain in some cases, normal stools and urine. (3) From **peritonitis,** by absence of fever and tenderness. (4) From **appendicitis,** by lack of fever, local tenderness at McBurney's point, abdominal rigidity on right side, and tumor. (5) From **neuralgia** of the abdominal wall, in which pain is unilateral and confined to region of a definite nerve. (6) From **muscular rheumatism,** in which there are local tenderness and pain on motion.

Treatment.—Remove the cause. For mild cases, heat to abdomen by hot-water bag, turpentine stupes, etc.; peppermint water, ginger, Hoffmann's anodyne. Morphine hypodermically only when necessary.

MUCOUS COLITIS

Called also **membranous colitis, mucous colic, tubular diarrhea.** This is a chronic intestinal disorder in which mucous shreds or intestinal casts are passed; a secretory neurosis.

Etiology.—Most often in neurotic or hysterical women. Exciting causes: emotion, dyspepsia, or improper food.

Pathology.—Mucosa normal, covered with layer of mucin, which becomes detached in shreds or casts of the intestine.

Symptoms.—Periodical attacks of intestinal colic with tenderness, especially at the splenic flexure; constipation,

usually followed by expulsion of mucous casts or shreds. Attacks last from one to fourteen days, and these may be repeated for years. Hysteria is often associated.

Diagnosis.—By examination, gross and microscopic, of casts to differentiate undigested food, etc. From other causes of pain in abdomen. (See Enteralgia, Diagnosis, p. 179.)

Prognosis.—Usually chronic.

Treatment.—Improve the general condition by hygienic measures and tonics. Rectal irrigation with warm sodium bicarbonate solution. Treat constipation (p. 176). Avoid irritating food. Give no morphine. For pain, hot-water bag, turpentine stupes, etc. Right inguinal colostomy may cure in time.

DISEASES OF THE LIVER, BILE PASSAGES, AND GALL-BLADDER

JAUNDICE (ICTERUS)

Definition.—A symptom consisting in discoloration by bile pigment of the skin, conjunctivæ, other mucous membranes, and secretions. Two principal groups of cases are described: (*a*) **Obstructive**; (*b*) **toxemic** and **hemolytic.**

1. **Obstructive Jaundice.—Etiology.**—Obstruction of the gall ducts **from within** by gall-stones or parasites, catarrhal or suppurative inflammation of the wall of the duct or of the intestinal mucosa at the orifice of the common duct, stricture or occlusion of the duct, tumors of the wall of the duct; or **from without** by pressure on the ducts by tumors of the liver, pancreas, stomach, kidney, lymph nodes, or uterus, or aneurysm, or impacted feces.

Symptoms.—Skin and conjunctiva vary from pale lemon-yellow to dark olive or greenish black, this discoloration being obscured by yellow artificial light. Pruritus may be slight or intense, especially in chronic cases. Sweating is common and often confined to the palms, axillæ, or abdo-

men. Urticaria, furunculosis, and other cutaneous affections may develop, and in chronic cases telangiectases in the skin or mucous membranes. In chronic or severe cases hemorrhages into the skin or mucous membranes are more or less serious. The sweat, and less often the saliva, bronchial mucus, tears, and milk may be yellowish. Gmelin's test will show bile pigment in the urine, even before the skin and conjunctivæ become discolored; if more than a trace is present it colors the urine light greenish yellow to dark blackish green. The stools are of a pale slate color from lack of bile, and are often pasty and of offensive odor, with flatulence and constipation or diarrhea. Pulse is slow, sometimes only 40 to 20; respiration normal or slowed in proportion to the pulse. Languor, depression, irritability, and headache are usual. Visual disturbances may occur. Coma, convulsions, or delirium may supervene in persistent cases. Acute cases may recover in a few days or weeks; chronic may last several years.

Diagnosis.—By yellow or greenish skin and conjunctivæ, clay-colored stools, and urine examinations. Avoid yellow light in inspecting for jaundice. *Of the cause:* (*a*) Mild jaundice with light-colored stools, following acute indigestion in general good health, points to **acute catarrhal jaundice.** (*b*) Attacks of severe local pain suggest **gall-stones.** (*c*) Persistent jaundice, cachexia, dilated gall-bladder, or hard tumor occur in **cancer of the gall-bladder.** (*d*) Persistent jaundice with enlarged, nodular liver and emaciation is found in **cancer of the liver.** (*e*) Persistent jaundice with enlargement of liver and spleen occurs in **hypertrophic cirrhosis.** (*f*) **Valvular lesions** of the heart may cause slight jaundice through passive congestion. In persistent jaundice exploratory laparotomy is often necessary for diagnosis.

Prognosis depends upon the cause. Catarrhal jaundice may recover in a few days or weeks.

Treatment is that of the cause. For pruritus, hot alkaline baths; dusting powder of starch, ℥j; camphor, ʒiss; zinc

oxide, ℥ss; or carbolic acid solution, 1 to 60 to 1 to 100, or carbolated vaseline locally.

2. **Toxemic and Hemolytic Jaundice.—Etiology.**—(1) Certain acute infections—yellow fever, acute yellow atrophy, Weil's disease, malaria, typhus fever, icterus neonatorum, relapsing fever, sometimes pneumonia, typhoid and scarlet fevers. (2) Arsenic, phosphorus, toluylendiamin, snake venom, and other poisoning.

Symptoms may be much less marked than in obstructive jaundice, but in some cases cerebral symptoms, jaundice, and hemorrhages are intense. Fragility of the red cells may be increased.

Treatment is that of the causal disease.

ACUTE CATARRHAL ANGIOCHOLITIS

Definition.—Jaundice caused by obstruction of the terminal portion of the common duct by swelling of the mucosa. Called also **acute catarrhal cholangitis** or **catarrhal jaundice.**

Etiology.—Most often in young persons. Follows gastroduodenitis from ingestion of irritating food, drink, or drugs, emotion, exposure, and passive congestion in cirrhosis, chronic cardiac disease, or nephritis. May be epidemic; is then possibly an acute infection.

Pathology.—Acute catarrhal inflammation of the mucosa of the lower end of the common duct, which may be occluded by a plug of thick mucus.

Symptoms.—The first may be slight jaundice, or this may be preceded by gastro-intestinal disturbance. Epidemic cases may begin with a chill, headache, and vomiting. Symptoms of obstructive jaundice (p. 180) are slight or marked. Abdominal pain is usually absent or slight, unless from accompanying gastritis. Light or bright yellow skin and conjunctivæ, headache, pain in the back and legs, languor, anorexia, nausea, clay-colored stools, little or no fever, are the chief symptoms. Pulse may be slow; liver and spleen

usually slightly enlarged. In mild cases jaundice lasts less than two weeks; in others, one to three months.

Diagnosis.—By history of gastroduodenitis in a young person, moderate jaundice, and absence of emaciation. (1) From **gall-stones**, by absence of biliary colic or dilatation of gall-bladder. (2) From **cancer**, by absence of pain, nodules in liver, and emaciation. (3) From **hypertrophic cirrhosis**, in which course is chronic, liver enlarged, jaundice persistent, and hemorrhages from mucosæ occur. (4) From **Weil's disease**, which attacks chiefly young butchers, begins with symptoms of an acute infection and runs a febrile course.

Prognosis.—Recovery in two to twelve weeks.

Treatment.—Restrict diet until gastroduodenitis subsides. Milk alone at first. Cerium oxalate, gr. v, and bismuth subnitrate, gr. xxx, every three hours, if gastritis exists. Calomel, followed by sodium phosphate in small doses to keep the bowels moderately open daily. Alkaline mineral waters or sodium bicarbonate with large quantities of water. Daily cold colonic irrigations are recommended. Bed is needed only in cases with fever or very slow pulse.

CHRONIC CATARRHAL ANGIOCHOLITIS (CHRONIC CHOLANGITIS)

Etiology.—Obstruction, partial or complete, of the common duct by gall-stones, stricture, newgrowths, or external pressure. May possibly follow repeated acute attacks.

Pathology.—The walls of the bile ducts are thickened, their lumina enlarged, especially in cases with complete obstruction. In these the dilated ducts and gall-bladder contain clear, colorless, usually sterile mucus; in cases of partial obstruction the mucus is bile-stained. Gall-stones are often present.

Symptoms.—Chronic obstructive jaundice (see Obstructive Jaundice, Symptoms, p. 180), with periodical attacks of intermittent fever lasting a few days ("hepatic fever"),

with chills, sweating, and sometimes pain. These febrile attacks occur without suppuration, but are probably due to infection or passage of gall-stones.

SUPPURATIVE AND ULCERATIVE ANGIOCHOLITIS OR CHOLANGITIS

Definition.—A diffuse purulent inflammation of large and small bile ducts usually with cholecystitis.

Etiology.—Gall-stones, ascarides, infection by extension from pylephlebitis, cancer, or abscess of the liver, rarely from acute cholecystitis. May also occur in typhus, typhoid, pneumonia, pyemia, and cholera.

Pathology.—The ducts are dilated, their walls thickened and distended with fibrinous or purulent exudate. They may be ulcerated, with small abscesses in adjacent liver tissue. The gall-bladder usually shows similar conditions, adhesions to adjacent organs, and sometimes fistulous openings connecting with adjoining viscera or perforating the abdominal wall or peritoneum, and causing peritonitis.

Symptoms.—A history of cholelithiasis, followed by severe sepsis, with enlargement and tenderness of liver and gall-bladder, leukocytosis, jaundice, which may be slight or marked, pain also variable, and loss of weight and strength. (For terminations by perforative fistulæ, etc., see Cholelithiasis, p. 186.)

Treatment.—*Surgical:* Cholecystotomy with prolonged drainage.

ACUTE INFECTIOUS CHOLECYSTITIS

Acute inflammation of the gall-bladder caused by bacterial invasion may be catarrhal, suppurative, or phlegmonous.

Etiology.—Gall-stones predispose to this affection, but are not necessarily present. The inflammation may extend from the gall ducts or adjacent structures. Acute infectious

diseases are often followed by infectious cholecystitis, especially typhoid and pneumonia.

Pathology.—The inflammation is catarrhal, suppurative, or phlegmonous. The gall-bladder is distended, wall tense, contents mucopurulent, purulent, or hemorrhagic, with foul odor in phlegmonous cases. The bacteria usually found are colon bacillus, typhoid bacillus, pneumococcus, staphylococcus, and streptococcus. The cystic duct is usually occluded, even in the absence of gall-stones. The gall-bladder may be adherent to adjacent structures and perforation, localized abscesses, fistulæ, or general peritonitis may follow.

Symptoms.—Severe pain usually located at or near the gall-bladder, but sometimes in the epigastrium or low in the abdomen. Fever, rapid pulse, nausea, vomiting, abdominal distention, tenderness, and rigidity occur. Tenderness becomes localized, usually over the gall-bladder, the distention of which may sometimes be recognized by palpation. Jaundice usually occurs with gall-stones, seldom in their absence. Adhesions may cause partial or complete intestinal obstruction.

Diagnosis.—By history of rather recent attack of typhoid or pneumonia followed by the above symptoms, especially when jaundice develops. Difficult if pain and tenderness are not localized over the gall-bladder. Leukocytosis differentiates the suppurative or phlegmonous cases from simple **impaction of gall-stone.**

Prognosis.—Fatal unless relieved by operation.

Treatment.—*Surgical:* Cholecystotomy and drainage; cholecystectomy, if wall of gall-bladder is gangrenous, friable, or contains abscesses.

CHOLELITHIASIS (GALL-STONES, BILIARY CALCULI)

Etiology.—*Predisposing:* Rare under twenty-five years of age, frequent after forty. Three-fourths of the cases occur in women, nearly all parous. Sedentary habits, excess of

food, constipation, and enteroptosis predispose. *Exciting:* Bacteria (colon, typhoid, etc.) are probably the essential cause, acting as nuclei and causing a catarrhal inflammation, which leads to secretion of cholesterin and lime salts.

Pathology.—Calculi occur in the gall-bladder, less often in the bile ducts. They may be single, ovoid, or rounded, and so large as to distend the gall-bladder, usually multiple, each with four or more flattened surfaces or facets. They may number several hundred or thousand. If very small they are called gall sand. They are composed of (1) cholesterin, white or tinged with bile pigment; (2) cholesterin, bile pigment, and salts of calcium and magnesium, dark brown, green, reddish black, with smooth or rough surface; (3) rarely of bile pigment; or (4) calcium carbonate. They are lamellated, homogeneous, or crystalline. A central nucleus consists of cholesterin, bile pigment, epithelium, bacteria, mucus, or a foreign body. The gall-bladder may be distended if the cystic duct is obstructed; it may also show inflammatory thickening in chronic cases. The lesions of cholecystitis, cholangitis, ulceration, and perforation followed by localized abscesses or general peritonitis, abscess of liver, biliary fistulæ, and septicemia may appear later.

Symptoms.—Usually none while the stones are in the gall-bladder. There may be dyspeptic symptoms, with a feeling of discomfort or slight pain in the right side of the abdomen. Symptoms are those of (1) passage of a stone, biliary colic; (2) chronic obstruction of (*a*) cystic or (*b*) common duct; (3) remote results.

1. **Biliary Colic.**—Though sometimes absent, passage of a stone through the cystic or common duct is usually accompanied by intense pain in the right hypochondrium, epigastrium, or lower thoracic region, often radiating to the right subscapular region. There are often a chill, fever, profuse sweating, and vomiting, which is paroxysmal or continuous. Pain may also become continuous or recur at intervals. Symptoms of collapse may accompany the paroxysms. The

liver region may be tender, the gall-bladder enlarged, especially in chronic cases, and very tender. If the stone obstructs the common duct, there is jaundice; the stools become light-colored; the urine contains bile and often albumin and red cells. The attack may cease suddenly after a few hours or a number of days, or recur at intervals until the stone is passed into the intestine. It may end in chronic obstruction of the gall duct by impaction, or terminate in ulceration and perforation of the duct with peritonitis. Gall-stones may be found in the stools after an attack. Death may occur from collapse during an attack.

Diagnosis.—By sudden intense pain in right hypochondrium, vomiting, prostration, tender gall-bladder, sometimes jaundice and calculi in stools after an attack; radiograms significant only if positive; exploratory laparotomy. (1) From **gastralgia** or **intestinal colic**, by location of pain and by absence in these of serious symptoms, with speedy relief by pressure or treatment. (2) From **gastric** or **duodenal ulcer**, by sudden onset of pain not increased by ingestion of food, and by absence of blood in vomitus and stools. (3) From **lead colic**, by absence of lead line, constipation and wrist-drop, and by location of pain. (4) From **pyloric stenosis** with adhesions, by absence of characteristic vomiting and physical signs of this condition. (5) From **cancer of liver** or **bile ducts**, in which pain, if present, is continuous, and emaciation and cachexia may be accompanied by nodular enlargement of the organ involved. (6) From **renal colic**, by location of pain in upper abdominal or lower thoracic region, instead of radiating to the groin, and absence of red cells or calculi in the urine. (7) From **appendicitis**, by different localization of pain and tenderness, unless the appendix is high up, and by jaundice and stone in stool. (8) From gastric crises of **locomotor ataxia**, by absence of other symptoms of the latter. (9) From **hysteria**, by irregular character of attacks in this condition, with absence of serious symptoms or physical signs and presence of stigmata of hysteria.

2. **Obstruction of the Cystic Duct.**—(*a*) Great dilatation of the gall-bladder, with bile and mucus or mucopus; in chronic cases with clear mucus (*hydrops vesicæ felleæ*). The gall-bladder is usually recognizable by inspection, palpation, or percussion, unless relaxed or dilated upward under the liver in acute cases. If not too tense, gall-stone crepitus may be elicited through a lax abdominal wall. Jaundice is often absent. (*b*) Acute cholecystitis is common and often becomes suppurative. (See Acute Infectious Cholecystitis, p. 184.) The symptoms of these conditions are often the most prominent of those present. They may be followed by those of perforation, localized abscesses, or general peritonitis. Atrophy of the wall of the gall-bladder is not an unusual termination; calcification is less common.

3. **Obstruction of the Common Duct.**—**Complete obstruction** by a stone in the common duct leads to distention of the gall ducts with clear fluid, and jaundice is intense and persistent. **Partial obstruction** by one or more stones in the common duct, or a movable stone in the diverticulum of Vater acting as a ball-valve, causes great dilatation of the ducts, rarely dilatation of the gall-bladder. There may be no symptoms. The jaundice is variable in intensity; bile is present constantly or at intervals in the stools; the spleen is enlarged. Infectious cholangitis may occur. (For symptoms, see Chronic Catarrhal Angiocholitis, p. 183.) Suppuration may take place in the inflamed ducts, extending to the gall-bladder or causing liver abscesses, characterized by remittent or intermittent fever, enlargement of the liver, and septic symptoms, ending in death. The so-called "hepatic intermittent fever" may be due to the presence of a ball-valve stone. The symptoms of this are paroxysmal attacks of pain in the hepatic region, vomiting, chills, fever, and sweating, resembling malarial attacks, with varying jaundice for months or years, deepening after each paroxysm.

Diagnosis.—(1) From obstruction of the common duct by **cancer**, by absence of dilatation of the gall-bladder; explora-

tory laparotomy. (2) Of incomplete obstruction with infectious cholangitis from **malarial fever**, by presence of jaundice and pain and absence of plasmodia.

4. **Remote Effects of Gall-stones.**—(*a*) **Biliary Fistulæ.**—These may occur through the skin, gastro-intestinal canal, hepatic duct, peritoneum, lungs, or other viscera. Symptoms may be absent; bile and gall-stones may be found emerging from the natural outlet of the viscus perforated; abscess may result. (*b*) **Intestinal obstruction** by stones passed through the common duct or by ulceration and perforation through the gall-bladder and intestinal wall.

Treatment of Biliary Colic.—Morphine, gr. $\frac{1}{4}$, hypodermically, repeated when necessary, and preceded by chloroform temporarily, if pain is intense. Hot applications over the liver or hot baths. *General medical:* Bowels must be kept open by laxatives, preferably sodium phosphate or sulphate, ℥j to ij, t. i. d., as these are thought to prevent concentration of the bile, and thus formation of new and enlargement of old calculi. Alkaline waters in large quantities. Moderate exercise. Diet regulated, so as to avoid dyspepsia. No drug is known to dissolve gall-stones when administered. (For pruritus, see Obstructive Jaundice, Treatment, p. 181.) *Surgical:* Indicated, when medical fails, for repeated attacks of colic; sometimes for persistent jaundice or dilatation of gall-bladder, for accidents due to infection or perforation, or for diagnostic purposes.

CANCER OF THE GALL-BLADDER AND BILE DUCTS

Etiology.—Usually at forty to seventy years of age. Primary cases show no sexual predisposition; secondary are three times as frequent in women as in men. Chronic irritation by gall-stones probably is an important cause.

Pathology.—Primary or secondary growths usually begin in the fundus of the gall-bladder or the common duct. The tumor is cylindrical or polyhedral celled or gelatinous.

Symptoms.—Pain, often with paroxysmal exacerbations, and tenderness in the gall-bladder region, persistent and deep jaundice, palpable tumor due to dilatation of the gall-bladder or to the growth in the gall-bladder or adjacent structures, emaciation, cachexia.

Diagnosis.—By jaundice, tumor in gall-bladder, or primay tumor elsewhere, cachexia, and exploratory laparotomy. (See Obstructive Jaundice, Diagnosis, p. 181.)

Prognosis.—Fatal. In accessible primary growths early complete removal may cure.

Treatment.—Cholecystectomy if the growth is primary in the gall-bladder and sufficiently localized. For pain, morphine. (For pruritus due to jaundice, see Obstructive Jaundice, Treatment, page 181.)

CIRCULATORY DISTURBANCES OF THE LIVER

1. **Active hyperemia,** or **congestion,** occurs normally after meals, in acute infectious disease, etc. Symptoms are absent or uncertain.

2. **Passive hyperemia, chronic congestion,** or **nutmeg liver** is due to circulatory obstruction in the liver by chronic valvular disease with failure of compensation; pulmonary obstruction in emphysema, chronic interstitial pneumonia, or large pleuritic effusion; obstruction of inferior vena cava or hepatic vein by aneurysm or tumor.

Pathology.—Liver uniformly large, dark red, firm. Cut section shows nutmeg appearance, due to deeply congested central vein and capillaries. In a later stage the liver is contracted, central liver cells are atrophied, connective tissue is increased.

Symptoms.—Sometimes a feeling of weight in the hepatic region; gastro-intestinal disturbances; may be hematemesis or jaundice, with clay-colored stools and dark urine. Liver is enlarged, tender, and may pulsate. Ascites is common.

Treatment.—(1) Of the cause. (2) Purgation, especially by salts.

3. **Thrombosis of the portal vein** may occur in cirrhosis, syphilis or cancer of the liver, sclerosis of the portal vein, local inflammation from injury, proliferative peritonitis, external pressure by enlarged glands, tumors, etc. The clot may become organized and complete obstruction of the portal vein result, collateral circulation being established.

Symptoms are slight; there may be sudden enlargement of the spleen, with ascites and blood in vomitus and stools.

ACUTE YELLOW ATROPHY

Called also **acute parenchymatous hepatitis, malignant jaundice,** and **icterus gravis.** This is a rare disease, with rapid degeneration and necrosis of liver cells, resulting in atrophy, with severe cerebral symptoms, jaundice, and hemorrhages.

Etiology.—Unknown. Most common between twenty and thirty years; most often in women, especially in pregnancy.

Pathology.—Liver very small, flabby, capsule wrinkled. Cut surface mottled gray, yellow, and red, the degree of cellular degeneration increasing in these colored areas in the order named. The liver cells contain droplets of fat and granules of yellow pigment; then only masses of fat droplets and pigment, with crystals of leucin and tyrosin; finally only connective tissue, bloodvessels, and groups of newly formed cells remain. Spleen often enlarged; parenchymatous degeneration of heart and kidneys. Often hemorrhages into skin, gastro-intestinal canal, lungs, kidneys, bladder. Skin and viscera bile-stained.

Symptoms are those of simple gastroduodenitis—malaise, headache, anorexia, nausea, eructations, vomiting, constipation, followed by jaundice. After three or four days, or

even two or three weeks, jaundice becomes deep, vomiting constant; hemorrhages into the skin and mucous membranes, as shown by hematemesis, melena, epistaxis, hematuria; severe headache, maniacal delirium, muscular twitching, sometimes convulsions. Fever absent, occasionally present before death. Urine scanty, bile-stained, may contain casts, albumin, and blood, usually leucin and tyrosin crystals. Stools become light colored or contain blood. The liver rapidly decreases in size, becoming painful and tender, while the spleen is enlarged. Delirium passes into coma, which deepens until death.

Diagnosis.—By jaundice, vomiting, delirium, diminution of size of liver, and presence of leucin and tyrosin in the urine. (1) From **hypertrophic cirrhosis,** in which there is usually fever, the liver is enlarged, and leucin and tyrosin are absent from the urine. (2) From **acute phosphorus poisoning,** in which the symptoms begin suddenly, gastric symptoms are more intense, vomitus and stools are phosphorescent, and leucin and tyrosin are absent.

Prognosis.—Nearly always fatal.

Treatment.—Symptomatic only. Feeding, stimulation, and relief of vomiting and delirium.

CIRRHOSIS OF THE LIVER

Atrophic cirrhosis is called also **chronic interstitial hepatitis, sclerosis of the liver, fibrous hepatitis, hobnail liver, alcoholic cirrhosis, gin-drinker's liver.**

Etiology.—Most often in men of forty to sixty years; may occur even in young children. *Exciting cause:* Usually alcohol in excess, especially whisky, brandy, rum, or gin.

Pathology.—(*a*) **Atrophic cirrhosis:** Liver small, firm; capsule usually thickened; surface smooth, irregularly contracted, or covered with depressions and protrusions (*hobnail liver*). Cut section is greenish yellow, yellow, brown, or red, sometimes with grayish bands or patches of connective tissue.

Connective tissue is seen chiefly between the lobules; the enclosed parenchyma cells are atrophied. Groups of cells, apparently newly formed bile ducts, are seen. Hepatic and portal veins may be obliterated; bile ducts obliterated or inflamed. The circulatory obstruction is compensated by anastomosis of the superficial and deep vessels. The peritoneal cavity often contains fluid. Chronic gastro-enteritis and enlargement of the spleen are common; kidneys cirrhotic; heart degenerated; arteries sclerotic. (*b*) **Fatty cirrhosis**: Marked localized or general fatty degeneration. Liver often enlarged, yellow over fatty areas, but firm. Most common in beer drinkers.

Symptoms.—Few or none while compensating circulation is maintained. Fatty cirrhosis is often found only at autopsy. The first symptoms are the same as those of the accompanying chronic gastritis—poor appetite, nausea, retching, and vomiting, especially in the morning, epigastric distress, constipation or diarrhea. These increase, and vomiting of blood from the stomach or varices of the esophagus may occur early or late. Gastric or intestinal hemorrhage causes tarry stools, and may relieve symptoms temporarily. Epistaxis and hemorrhoids are common. Profuse hemorrhages may cause severe anemia. The epigastric and mammary veins are enlarged. Ascites usually occurs later and may be very marked, as much as twenty quarts at times, and recurs after aspiration. There may be œdema of the lower extremities and genitals. Jaundice is slight and occurs late. Fever is absent or slight. At a late stage the patient is emaciated, face sallow or subicteroid, vessels of nose and cheeks dilated, conjunctivæ watery, abdomen greatly distended, with dilated veins. After tapping, liver dulness may be found diminished, and the rough, hard surface may be felt; spleen enlarged. Delirium, stupor, coma, or convulsions may occur at any time. **Course** is chronic, sometimes with temporary improvement. Ascites usually recurs; emaciation and weakness increase; death usually from exhaustion.

Diagnosis.—By history of chronic alcoholism and chronic gastritis, with hepatic facies, ascites, and gastric or intestinal hemorrhages. (1) From **carcinoma of liver,** in which emaciation may be great and there is cachexia in the later stages. (2) From **syphilis of liver,** in which a history of syphilis may be obtained or other specific lesions be found, Wassermann reaction may be positive, general health is fairly good, and improvement follows trial of mixed treatment. (3) From **thrombosis of portal vein.** (4) From **tuberculous peritonitis,** in which there are often fever and tuberculous lesions in other organs, but no splenic enlargement or jaundice.

Prognosis.—Usually fatal, sometimes after temporary improvement. Ascites and hemorrhages are unfavorable.

Treatment.—*General:* No alcohol allowed. Simple diet; milk alone while gastric disturbance is marked; out-of-door life without overexertion. *Medicinal:* Bitter tonics, such as tinct. gentian. comp., ʒj, a. c., for appetite; dilute nitrohydrochloric acid, ♏x to xx, p. c., if digestion is poor. Keep bowels regular. *Of symptoms:* For ascites, diuresis by salines, such as potass. acetat., potass. citrat., potass. et sod. tartrat., āā gr. xx; aquæ, q. s. ad ʒij, in water, to which may be added the juice of a lemon, every three hours; or by pill of calomel, digitalis, and squill, āā gr. j, every four hours, for three or four doses; or by high rectal irrigations with hot normal salt solution; or catharsis with Rochelle or Epsom salt or compound jalap powder. Salt-free diet with restricted fluids. If these fail after a few days, tapping should not be delayed if ascites is pronounced. Repeat when necessary. For hemorrhage, ice-bag on abdomen, opium. *Surgical:* Scraping the peritoneal surface of the liver and the adjacent peritoneum, so as to induce formation of adhesions in which an anastomotic circulation may be established, and omentopexy, stitching the omentum to the abdominal wall, are sometimes successful.

Hypertrophic Cirrhosis (Hanot's Disease).—Etiology.—Most common in young adult males. Exciting cause unknown.

Pathology.—Liver uniformly enlarged; surface smooth or granulated, of an olive-green color. Section is of a uniform greenish-yellow color. Microscopically, in one or more lobes the connective tissue, rich in round cells, is between the liver cells as well as between the lobules. The connective tissue lacks the tendency to contraction seen in atrophic cirrhosis, and the hepatic cells are not degenerated, but may hypertrophy. The bile ducts are inflamed, and in so-called "biliary cirrhosis" the connective-tissue changes are chiefly around them. Spleen much enlarged; no ascites.

Symptoms.—The first is often enlargement of the liver, which is smooth and firm with a sharp edge; spleen enlarged, hard; gall-bladder not enlarged. Attacks of pain and tenderness of the liver, with nausea and vomiting, may occur early. Jaundice usually appears early; is slight at first, but may become marked. Urine contains bile, but stools are not clay-colored. Hemorrhages from gums, nose, stomach, or intestines are common, or purpura may develop. Superficial abdominal veins are not dilated. Ascites is absent. Urticaria and other cutaneous affections may be present.

Course is very chronic, four to ten years. At any time a severe febrile jaundice, with delirium, may develop. Death occurs from this or from cachexia, hemorrhage, or some intercurrent infection.

Diagnosis.—By chronic enlargement of liver and spleen, persistent jaundice, hemorrhages from mucous membranes, absence of ascites. From (1) **Carcinoma**, by absence of hard nodules, great cachexia, and emaciation. (2) **Amyloid disease**, in which there is no jaundice, but a history of prolonged suppuration, syphilis, or tuberculosis. (3) **Abscess**, which is usually secondary to colitis or some suppurative disease and is accompanied by fever of septic type, while tenderness and sometimes fluctuation are detected. (4) **Echinococcus cyst**, which is elastic or fluctuating, with good general health unless suppuration occurs. (5) **Syphilis**, in which irregular masses may be felt and specific history

obtained, Wassermann reaction is positive, and by mixed treatment.

Prognosis.—(See Course, above.)

Treatment.—Symptomatic. (See Atrophic Cirrhosis, Treatment, above.)

Syphilitic Cirrhosis.—(See Syphilis of the Liver, p. 87.)

Capsular Cirrhosis.—Called also **Glissonian cirrhosis** and **chronic perihepatitis,** is a chronic inflammation of the serous covering of the liver, whose capsule becomes thick and contracted, with or without increase of the interstitial connective tissue of the organ.

Etiology.—Usually occurs in adults. It may be secondary to acute perihepatitis, or chronic and accompanied by chronic proliferative peritonitis, chronic interstitial nephritis chronic perisplenitis, and sometimes chronic mediastinitis. Syphilis may be a cause.

Pathology.—Capsule thickened uniformly or in patches and contracted generally or irregularly; surface smooth or roughened by projecting connective tissue, sometimes adherent to adjacent peritoneum. Liver generally or irregularly contracted, hepatic tissue soft, at times traversed by bands of connective tissue extending inward from the capsule. Microscopically, the connective tissue is usually chiefly dense and fibrous. Associated lesions are mentioned under Etiology.

Symptoms are those of atrophic cirrhosis, with persistent ascites; no jaundice; small, painful, and tender liver, and usually symptoms of chronic nephritis.

Treatment.—(See Atrophic Cirrhosis, Treatment, p. 194.)

ABSCESS OF THE LIVER

Called also **suppurative** or **purulent hepatitis.**

Etiology.—(1) **Tropical abscess,** usually single, occurs chiefly in tropical countries, sometimes in the United States. "Idiopathic" cases are recognized, but there is usually a

preceding dysentery. The Ameba dysenteriæ is regarded as the active cause, though streptococci or staphylococci may be found in the pus. (2) **Traumatic abscess** results from direct injury of the liver or head. (3) **Embolic or pyemic abscesses** follow infection through the portal vein in dysentery, appendicitis, typhoid, intestinal ulcers, and pelvic abscesses; through the hepatic artery, as in malignant endocarditis or other septic processes in the systemic circulation; through the inferior vena cava and hepatic vein; or in pyemia. (4) Suppurative cholangitis, due to gall-stones, tuberculosis, or duodenitis, may cause abscesses. (5) Foreign bodies from the stomach may perforate and cause liver abscesses, as may roundworms or other parasites. Echinococcus cysts may suppurate.

Pathology.—(1) **Solitary or tropical abscess,** usually single and large; sometimes several. Usually in upper part of the right lobe. Size from that of an orange to one containing six quarts. In old cases the lining wall is thick. In recent cases it is irregular, necrotic, passing gradually into the surrounding inflamed liver tissue. The pus is reddish brown, white, yellow, or greenish. Microscopically, liver tissue around the necrotic area is infiltrated with round cells. The pus may be sterile or contain amebæ, streptococci, staphylococci, or colon bacilli. The abscess may perforate into the right pleural cavity, peritoneal cavity, gall-bladder, bile ducts, colon, vena cava, or pericardium. Spontaneous cicatrization may follow pointing of the abscess. (2) **Multiple or pyemic abscesses,** usually multiple. Surface of liver smooth or dotted with small, yellowish-white, subcapsular abscesses. Section shows many small communicating abscesses. The pus is usually thick, tenacious; in some cases fetid and bile-tinged. With suppurative cholangitis gall-stones are present, and the obstructed ducts and gall-bladder enlarged and filled with pus.

Symptoms.—1. **Tropical Abscess.**—Death may occur from rupture of the abscess without previous symptoms. Usually

there are pain, tenderness, and enlargement of the liver, with symptoms of sepsis. Pain is usually a dull ache, referred to the shoulder and back or right hypochondrium, with a dragging feeling when lying on the left side. Tenderness is generally most marked at the costal margin in the mammary line. Liver enlargement is usually of the right lobe, upward and to the right, the line of liver dulness rising beyond the nipple line, sometimes to the fifth interspace at the mid-axillary line, and nearly to the angle of the scapula behind. The right side of the chest may bulge. Fluctuation may be detected. Fever is irregular or intermittent, sometimes with chills and sweating. In chronic cases fever may be absent. Skin is pale, sallow, slightly jaundiced. Diarrhea may occur, with amebæ in the stools, or constipation. Empyema may develop without perforation of the abscess, or the latter may point through the skin or into the peritoneal cavity, lung, stomach, intestine, or pericardium.

Course and Prognosis.—Recovery may follow pointing into the lung or through the skin. Course is uncertain. Duration, a few weeks to several years. Mortality is high; death from septicemia or perforation.

2. **Pyemic Abscess.**—Symptoms of pyemia, with slight icteroid tint and slightly enlarged, painful, tender liver.

Prognosis.—Fatal.

Diagnosis.—From (1) **malarial fever,** by absence of plasmodia and splenic enlargement, presence of leukocytosis and irregular temperature, and failure to respond to quinine. (2) **Empyema,** in which there are usually a history of preceding pleuritic pain or pulmonary tuberculosis and physical signs of pus in the pleural cavity (p. 238). This may occur after perforation. X-rays show straight level of shadow of fluid in empyema, shadow convex upward if enlargement is subphrenic. (3) **Gall-stones** with catarrhal angiocholitis, in which jaundice follows the attacks, between which there is no fever, and leukocytosis is absent. Aspiration of the liver with a fair-sized needle is important in

cases of probable abscess. (4) **Hypertrophic cirrhosis** (see Diagnosis, p. 195). (5) **Carcinoma,** by absence of cachexia and hard nodular masses in liver. (6) **Echinococcus cyst.** (See Diagnosis, pp. 114 and 115.)

Treatment.—Supporting and symptomatic. Incise and drain. For amebic abscess, aspirate and inject emetine hydrochloride, gr. j; treat amebic dysentery if present.

FATTY LIVER

This may be due to fatty infiltration or fatty degeneration of hepatic cells.

Etiology.—Obesity; cachectic states due to cancer, tuberculosis, or other chronic wasting disease; other conditions interfering with oxidation, such as severe anemia, prolonged use of alcohol; poisons, including phosphorus and that of acute yellow atrophy.

Pathology.—Liver uniformly and often greatly enlarged, edge rounded, color yellowish, surface smooth, consistency diminished, cut surface yellowish and greasy. If the organ is fatty only in parts the surface and cut section are mottled brownish red and yellow. Chronic congestion is often associated (*nutmeg liver*). Microscopically, fat droplets of varying size in the liver cells.

Symptoms may be absent and are never marked. Stools may be light colored. No jaundice; rarely signs of portal obstruction.

Diagnosis is made by history of associated condition causing the fatty liver (see Etiology), and by enlargement of liver, excluding other causes for this. From (1) **amyloid liver,** by absence of history of causes of this disease (p. 200) and by less firm consistency. (2) **Hypertrophic cirrhosis,** by absence of jaundice and portal obstruction. (3) **Carcinoma,** by absence of cachexia, emaciation, pain, and hard nodules. (4) **Passive congestion,** which is often accompanied by fatty

changes in the liver, by absence of circulatory obstruction. (See Passive Hyperemia, p. 190.) (5) **Leukemia,** by blood examination.

Treatment is that of the cause.

AMYLOID, WAXY, OR LARDACEOUS LIVER

Etiology.—Wasting diseases, especially those with suppuration and ulceration, particularly of bones. The most common causes are tuberculosis and syphilis; less frequent, cancer, severe malaria, dysentery, and leukemia.

Pathology.—Liver often very large, edges rounded or sharp; surface smooth, pale, rather translucent; firm, tough. Cut section pale, translucent; often shows fatty change or cirrhosis also, amyloid portions being stained mahogany color by dilute iodine solution. Microscopically, hyaline degeneration, first of the walls of the intralobular vessels, then of the interlobular and of the connective tissue. Cirrhosis of the liver often, and amyloid disease of spleen and kidneys usually occurs.

Symptoms.—Few or none. Stools may be light colored. Liver enlarged, smooth, firm, not tender, edge rounded or sharp. Spleen usually enlarged.

Diagnosis.—By history of suppuration, tuberculosis, or syphilis with large, firm liver. From (1) **fatty liver,** by this history and consistence. (2) **Leukemia,** by blood examination.

Prognosis depends upon condition of kidneys and intestines.

Treatment.—None.

CANCER OF THE LIVER

Etiology.—Rarely primary; usually secondary to carcinoma of the stomach, intestine, pancreas, gall-bladder or

gall ducts, or other organs. Usually between forty and sixty years of age; more often in men. Heredity, trauma, and gall-stones are alleged causes.

Pathology.—(1) **Primary carcinoma**: (*a*) *Massive:* A uniform grayish growth in a large portion of the liver, which is much enlarged. (*b*) *Nodular:* A large primary mass with numerous small nodules of varying size. (*c*) *Cancer with cirrhosis:* Liver little enlarged, grayish yellow, filled and studded with many small yellowish nodules, looking like a cirrhotic liver. (2) **Secondary carcinoma**: Liver usually large, with many large nodules throughout. These may be umbilicated or show hyaline or fatty degeneration.

Symptoms are those of the primary growth: then loss of flesh, strength, and appetite; nausea, vomiting; sometimes pain or distress in the liver, which is usually enlarged; jaundice usually moderate; rarely ascites; anemia, cachexia; fever which is irregular or intermittent and may be high. The upper part of the abdomen is enlarged in most cases; superficial veins are dilated. The cancerous nodules may be seen or felt. Spleen rarely enlarged. Disease lasts three to eighteen months.

Diagnosis.—By enlargement with nodular surface. (1) From **gummata**, by lack of specific history and of rapid improvement under potassium iodide. (2) Of cases with smooth surface from **fatty** or **amyloid liver**, by jaundice, pain, cachexia. (3) From **hypertrophic cirrhosis**, by pain, cachexia, irregular, and more rapid enlargement of liver. (4) From **echinococcus cysts**, which are usually softer, may be detected by aspiration, and do not affect the general health as much.

Treatment.—Symptomatic—supportive and for pain and gastric disturbance.

Sarcoma is rarely primary; more often secondary to melanosarcoma of the eye or skin. Nodules in these structures and melanuria may aid the diagnosis.

MOVABLE LIVER

Most common in women. Occurs as part of an enteroptosis, after repeated ascites, or as a result of abnormally lax ligaments. The **symptoms** are a dragging feeling or pain. **Differentiated** from diseases causing enlargement of the liver by the upper border of liver dulness, which percusses low, while the lower border is felt abnormally low and at times the entire upper surface may be palpated. The liver may be displaced downward also by pleuritic effusion, intrathoracic tumor, pulmonary emphysema, pericardial effusion, or subphrenic abscess; upward by ascites, intestinal distention, or abdominal tumors. **Treatment** consists in replacement of the liver and application of a supporting belt; sometimes hepatopexy.

DISEASES OF THE PANCREAS

Hemorrhage of the Pancreas.—Etiology.—Not definitely known. Pancreatic hemorrhage occurs after traumatism; with valvular disease or portal obstruction; in cases with diseased bloodvessels or blood states accompanied with hemorrhage, such as hemophilia, purpura, pernicious anemia, scurvy, and acute infections; in acute pancreatitis, and associated with cancer or cyst of the pancreas, or fat necrosis.

Pathology.—The hemorrhage may be confined to a small part of the gland or even invade the adjacent tissues. It is often associated with gangrene or inflammation.

Symptoms.—While in perfect health, intense pain suddenly begins in the pancreatic region. Collapse rapidly follows, with profuse sweating, subnormal temperature, feeble pulse, nausea and vomiting, restlessness. There is tenderness, usually in the epigastrium. The abdomen may be tympanitic, the bowels constipated. Death occurs in coma, not from loss of blood.

Diagnosis.—By sudden onset without preceding illness, by location of pain and tenderness, and by exclusion.

Prognosis.—Very poor.

Treatment.—*Surgical:* For pain, morphine, and heat or cold locally.

ACUTE PANCREATITIS

1. **Acute Hemorrhagic Pancreatitis.—Etiology.**—Usually in adult males, often alcoholic. Entrance of bile or gastric juice into the pancreatic duct may be a cause.

Pathology.—The gland is enlarged, with blood infiltrating the interlobular tissue. Areas of necrosis of gland cells surrounded by inflammatory exudate. Fat necrosis in the omentum, mesentery, etc.

Symptoms are those of hemorrhage of the pancreas (p. 202), followed by fever, sometimes a chill. Death usually in two to four days or less.

Diagnosis.—(1) From **irritant poisoning**, by history and examination of vomitus. (2) From **perforating gastric** or **duodenal ulcer**, by previous good health. (3) From this and **intestinal obstruction**, by site of pain and tenderness, severity of pain, more profound collapse, and by laparotomy.

2. **Acute Suppurative Pancreatitis (Pancreatic Abscess).—Etiology.**—Unknown. Chiefly in men. Is primary or secondary.

Pathology.—One abscess or more, or diffuse purulent infiltration. The abscess may perforate into the intestinal canal, peritoneal cavity, or peripancreatic space. There may be fat necrosis.

Symptoms.—Severe localized pain, vomiting followed by fever, delirium, tumor in the pancreatic region; later, jaundice, fatty diarrhea, glycosuria, and symptoms of sepsis.

3. **Gangrenous pancreatitis** may be localized or general, and follows traumatism, hemorrhage, acute hemorrhagic or

suppurative pancreatitis or perforation of a gastric ulcer. The gland is usually dark slate-colored, often free in an abscess cavity. Fat necrosis is common. Symptoms of acute hemorrhagic pancreatitis may precede or accompany the affection. Usually fatal in ten to twenty days, but recovery has followed spontaneous evacuation through the intestine.

Treatment of Acute Pancreatitis.—*Surgical:* For pain, morphine, hot or cold applications.

CHRONIC PANCREATITIS

Etiology.—Usually follows obstruction of the pancreatic duct by calculi, or extension of inflammation along it from the duodenum, or results from infection, adjacent inflammatory processes, or syphilis. May be preceded by one or more attacks of acute pancreatitis.

Pathology.—Pancreas larger or smaller than normal, usually hard. Interstitial tissue of a part or the whole of the gland is increased. This increase may be interlobular or interacinar. Atrophy of the gland tissue follows.

Symptoms.—Dyspepsia, paroxysmal epigastric pain, with tenderness, prostration, fever, emaciation; sometimes a palpable pancreatic tumor; jaundice from pressure, and ascites. Lipuria, fatty stools containing undigested muscle fibers, and sometimes glycosuria are present.

Diagnosis.—By localization of symptoms and tumor, pressure symptoms, and examination of feces and urine.

Treatment is that of the cause, if discoverable.

PANCREATIC CYST

Etiology.—Usually in young or middle-aged adults. *Causes:* Traumatism, usually blows or prolonged pressure;

inflammation extending from the duodenum; obstruction of the duct by calculi or by pressure of tumors; obstruction due to chronic pancreatitis; cystadenoma.

Pathology.—Cysts are single or multiple; may be very small or contain several quarts of fluid, sometimes mixed with blood. This is usually dark brown, alkaline; specific gravity, 1010 to 1020; contains cell debris, fat granules, sometimes cholesterin. It will digest fibrin and albumin. One or both of the other pancreatic ferments may also be present.

Symptoms, except gradual abdominal enlargement, may be absent until the cyst is large, or there may be a history of one of the above causes. The firm or elastic rounded tumor is usually found projecting between the stomach and transverse colon, occasionally above or below both, or on the left side when developing from the tail of the pancreas. There are usually attacks of severe pain, often colicky, radiating to the back from the region of the pancreas, nausea, vomiting, loss of weight; sometimes jaundice or dyspnea from pressure, or undigested meat in stools; rarely fatty stools, glycosuria, or salivation.

Diagnosis.—By symptoms and by presence of tumor, generally between stomach and transverse colon in the median line, not usually moving with respiration. Functional disturbances—fatty stools, undigested meat in feces, and glycosuria—are merely suggestive. Aspiration of cyst fluid for diagnosis by presence of trypsin is dangerous, as it may allow pancreatic secretion to enter the abdominal cavity and cause fat necrosis.

Treatment.—*Surgical:* Incision and drainage, with care to protect adjacent structures from the fluid.

TUMORS OF THE PANCREAS

Carcinoma is the most common; sarcoma, adenoma, and lymphoma are very rare. Syphilitic gummata may occur.

Symptoms are not characteristic. They are paroxysmal epigastric pain, nausea, vomiting, emaciation, cachexia, persistent marked jaundice, dilatation of the gall-bladder, sometimes an epigastric tumor, and at times functional disturbances—fatty stools, glycosuria, salivation—or ascites from pressure or gastric dilatation from compression of the pylorus.

Treatment is surgical and symptomatic.

PANCREATIC CALCULI

Pathology.—Usually multiple, white, round, or rough, consisting chiefly of carbonate and phosphate of lime. They cause chronic interstitial inflammation of the gland, dilatation of the duct, formation of cysts, and possibly predispose to carcinoma.

Symptoms are indefinite. Occasional epigastric pain, glycosuria, fatty stools, emaciation.

Treatment.—Surgical and symptomatic.

DISEASES OF THE PERITONEUM

ACUTE GENERAL PERITONITIS

Etiology.—(*a*) **Primary peritonitis,** rare. Occurs without known preceding disease; is sometimes possibly rheumatic. (*b*) **Secondary peritonitis** results from traumatism, extension from inflamed adjacent organs, or bacterial infection without apparent lesion of the intestine. Peritonitis due to perforation is the most common. Peritonitis may follow acute infections or accompany chronic nephritis, rheumatism, pleurisy, tuberculosis, and septicemia. Bacillus coli, streptococcus, staphylococcus, Ameba coli, gonococcus, pneumococcus, and other organisms are found.

Pathology.—The peritoneum is congested uniformly or locally. The intestine is distended, and its coils are often

adherent to each other. In cellular peritonitis the congestion is the only visible lesion: microscopically, the endothelial cells of the peritoneum are seen to be increased and enlarged. In exudative peritonitis the exudate is fibrinous, serofibrinous, purulent, or hemorrhagic; it may be scanty or sufficient to fill the abdomen.

Symptoms.—There is often the history of one of the causes mentioned above, followed, in cases with perforation or sepsis, by a chill or chilly feeling and pain varying at first with the site where the inflammation begins. Later it becomes general. The patient lies on the back, with knees flexed and body bent to relax the abdominal muscles, which are often rigidly contracted at first on the side where the pain starts. Pain may be absent. The abdomen becomes distended, tympanitic. Vomiting is an early symptom and often repeated. Constipation is the rule, occasionally diarrhea. The temperature may rise rapidly to 104° or 105°, then become lower; it is sometimes not above normal. The pulse is frequent, small, wiry, often 100 to 150; respirations frequent and shallow. The tongue becomes dry, red, and often fissured. Micturition may be frequent and painful; sometimes there is retention. The Hippocratic facies develops—face pinched, eyes sunken, expression anxious, skin of face lead-colored or livid. Hiccough, muttering delirium, or stupor may be present.

Physical Examination.—Hippocratic facies. Tongue dry, red, often fissured. The abdomen, at first rigid and tender, later is distended, sometimes with friction fremitus, and signs of ascites may develop, with displacement of the apex beat upward and to the left.

Course and Progress.—Heart action becomes weak and irregular, respiration shallow, rectal temperature high, skin cold, pale and livid, and death occurs sometimes suddenly, usually in three to five days, less often in thirty-six to forty-eight hours, or even after ten days. Prognosis depends chiefly upon the cause of the inflammation and the nature of

the infection, being usually very bad after puerperal sepsis, induced abortion, perforation of the intestine or stomach, or rupture of an abscess.

Diagnosis.—By history of a preceding cause, such as appendicitis, gastric ulcer, or suppurative pelvic lesions, and by sudden onset of pain, tenderness and distention, vomiting, usually with fever, ascites, and prostration. (1) From **acute enterocolitis**, in which pain is more colicky, diarrhea and collapse are more marked. (2) From **intestinal obstruction.** (See Differential Diagnosis, p. 175.) (3) From **embolism of the superior mesenteric artery**, by history of cause and by exploratory incision. (4) From **acute hemorrhagic pancreatitis**, in which a tumor sometimes develops in the upper part of the abdomen, by laparotomy. (5) From **ruptured tubal pregnancy**, in which other signs of pregnancy are usully present and the tumor is palpable by the vagina. (6) From **hysteria**, in which there are hysterical stigmata and variability of symptoms, which may even disappear when the attention is diverted.

Localized Peritonitis.—1. **Subphrenic Peritonitis** (*Acute Perihepatitis, Subphrenic Abscess*).—**Etiology.**—May be caused by traumatism, but is usually secondary to diaphragmatic pleurisy, empyema, tuberculosis or cancer of the pleura, or to cancer, abscess, or hydatid cyst of the liver, or abscess of the gall-bladder or right kidney, and most often to perforation of a gastric or intestinal ulcer.

Pathology.—The peritonitis may be fibrinous or suppurative. It involves the apposed surfaces of the diaphragmatic and the hepatic peritoneum, or may be confined to the lesser peritoneal sac. Formation of adhesions usually results in localization of the abscess, which sometimes contains air (*subphrenic pyopneumothorax*), especially in cases due to gastric perforation.

Symptoms.—The symptoms generally follow those of one of the causes mentioned. The onset is usually marked by sudden local pain, increased by deep inspiration, tenderness,

and vomiting; then chills, irregular fever, sweating, dyspnea due to pressure upon the diaphragm, emaciation. Perforation of the diaphragm may cause symptoms of empyema or abscess of the lung. The abscess may point through the abdominal wall.

Physical Signs.—(*a*) If in the greater sac, the abdominal wall is prominent above the liver, which is displaced downward. Displacement of the diaphragm upward causes flatness like that of empyema, or tympany, as in pyopneumothorax, if gas is present in the abscess cavity. (*b*) If in the lesser sac, being behind the stomach, the tumor and area of dulness caused by the abscess are increased if the stomach is full of liquid, obscured if filled with gas. Pressure upon the diaphragm by air in the cavity may give signs like those of pneumothorax on the left side.

Diagnosis.—(1) From **empyema**, by absence of pulmonary symptoms at first and of cardiac displacement, and by aspiration in the area of abnormal dulness. In subphrenic abscess the flow of pus is not arrested with each inspiration. X-rays show curved shadow of diaphragm, not horizontal level of shadow of empyema. (2) Of subphrenic pyopneumothorax from true **pyopneumothorax**, by the absence of history of preceding pulmonary disease and by *x*-rays.

2. **Appendicular Peritonitis.**—(See Appendicitis, Pathology, Ulcerative Type, p. 169.)

3. **Pelvic Peritonitis.—Etiology.**—Usually puerperal infection, gonorrhea, tuberculosis of the tubes or uterus.

Pathology.—Usually pyosalpinx, with matting together of fimbriæ, ovary, and intestines, sometimes with localized abscesses. Spontaneous recovery may occur, or an abscess rupture, or tuberculosis extend, causing general peritonitis.

Treatment of Acute Peritonitis.—Absolute rest in the most comfortable position. Morphine, gr. $\frac{1}{3}$ to $\frac{1}{2}$, hypodermically, repeated in dose of gr. $\frac{1}{4}$ when necessary to relieve pain and keep the patient quiet. Ice-cold cloths, hot fomentations, or turpentine stupes for relief of pain, avoiding weighty

applications. Saline purgatives are recommended by some, but should never be given if perforation of the stomach or intestine is suspected. One should then rely upon restriction of peristalsis by morphine. For vomiting, stop food and drink temporarily; cracked ice. Diet, hot or cold milk with lime-water or Vichy or peptonized if necessary; rectal feeding if vomiting persists. For tympanites, high rectal tube, enemata containing turpentine. For cardiac weakness, strychnine and whisky as needed. Surgical treatment should be considered from the outset. Incision and saline irrigation with or without drainage are most likely to succeed in localized peritonitis.

CHRONIC PERITONITIS

1. **Local Adhesive Peritonitis.**—Adhesions often exist between the spleen or liver and the diaphragm, or between the appendix and adjacent structures, or between adjoining coils of intestine. There may be no symptoms, or persistent abdominal pain, or strangulation of the intestine may occur.

2. **General Adhesive Peritonitis.**—Results from acute general or tuberculous inflammation. The peritoneal cavity may be completely obliterated, all surfaces being densely adherent and often thickened. If any portions are not adherent they are filled with serofibrinous or purulent exudate.

3. **Proliferative Peritonitis.**—Often occurs in chronic alcoholics, especially with cirrhosis of the liver, tumors, or chronic passive congestions. The peritoneum is opaque, white, and much thickened, but not uniformly so, and not extensively adherent. The rolled-up omentum often forms a mass above the transverse colon. There may be many nodular thickenings of the peritoneum grossly resembling tubercles. Liver and spleen are adherent or their capsules greatly thickened and the organs constricted. The mesen-

tery is very thick and often contracted, the gastrohepatic omentum constricted, thus compressing the portal vein. The intestinal wall is thick and its mucosa lies in folds. There is usually a moderate serous effusion, more rarely marked ascites. The fluid may be sacculated by adhesions. Chronic inflammation of the pericardium and pleuræ may coexist.

4. **Chronic Hemorrhagic Peritonitis.**—Occurs most often in the pelvis. Circumscribed areas of the peritoneal surface become covered with thick layers of connective tissue containing large vessels, and hemorrhages occur from these at intervals.

Symptoms of chronic peritonitis may be absent, or there may be slight abdominal pain and tenderness and little distention, sometimes fever. Bowels irregular. Anemia, emaciation, and weakness follow. A friction fremitus may be felt, or masses due to thickening.

Treatment.—Symptomatic for weakness and pain. For ascites, tapping.

5. **Tuberculous Peritonitis.**—(See p. 95.)

Cancer of the peritoneum is rarely primary, and then usually endothelioma. Carcinoma is secondary to new-growths of adjacent organs, and corresponds in type with that of the primary tumor. The nodules are multiple and grossly resemble tubercles. The signs are usually those of chronic peritonitis with ascites and emaciation. After tapping, the hard nodules may be felt. In colloid cancer the abdomen is firm and distended with a semigelatinous substance.

Diagnosis.—(1) From **tuberculous peritonitis**, by absence of fever, multiplicity of nodules, and exploratory laparotomy. (2) From **echinococcus cysts**, which are usually secondary to one in the liver which causes enlargement of that organ, and by the poorer general condition.

Prognosis.—The prognosis is bad.

Treatment.—The treatment is symptomatic.

ASCITES (HYDROPERITONEUM)

Ascites, or hydroperitoneum, is an accumulation of serous fluid in the peritoneal cavity. It is a symptom.

Etiology.—(1) *Local causes:* (*a*) Chronic peritonitis, simple, tuberculous, or cancerous; (*b*) portal obstruction in cirrhosis, cancer, or other hepatic disease; in chronic passive congestion from cardiac disease or emphysema; in compression of the vein in the gastrohepatic omentum in proliferative peritonitis, or by pressure of tumors, aneurysm, or adhesions; pressure upon the inferior cava, hepatic vein, or lymph vessels may cause ascites; (*c*) solid abdominal tumors, as of the ovaries, or enlarged spleen. (2) *General causes:* (*a*) Those causing general dropsy, such as cardiac disease or emphysema; (*b*) chronic nephritis, chronic malaria, cancer, syphilis, or amyloid disease. Chylous ascites is due to presence of filaria or compression of the thoracic duct, etc.

Pathology.—Ascitic fluid is clear, light or dark yellow, of specific gravity 1010 to 1015, highly albuminous; may coagulate spontaneously. It may be hemorrhagic in cancer, tuberculosis, or cirrhosis; fatty or chylous in cancer of the peritoneum or in persistent lipemia due to milk diet or diabetes.

Symptoms.—Gradually increasing abdominal distention, causing sometimes a sense of weight, then dyspnea from pressure.

Physical Signs.—Abdomen distended, flattened at the sides unless very full. Skin may be tense and show striæ, superficial veins distended and the superficial epigastric anastomosing with the external mammary. The umbilicus may be flat or even protrude, and around it the vessels may be varicose (*caput medusæ*). Fluctuation: Fluid wave obtained by tapping sharply at one side while palpating the other, the transmission of vibration through the abdominal fat being prevented by pressure upon the whole median

line of the abdomen by an assistant. By "dipping" with the finger-tips suddenly one may displace the fluid and come down upon the abdominal organs or tumors. Dulness or flatness over fluid, tympany over the floating intestines, these signs changing as the position is altered. In the dorsal position the tympany is in the central portion; when on the side the tympany changes to the side uppermost, the dulness to the lower. If fluid is scanty, the knee-chest position will allow dulness to be obtained in the upper portion of the abdomen.

Diagnosis.—(1) From **ovarian cyst**, which is not preceded by the causes of ascites; is usually unilateral at first; gives dulness in the median line with its convexity upward, and tympany in the flanks; displaces the uterus, as is felt by vaginal examination; refills more slowly after aspiration than ascitic fluid accumulates, and has contents of specific gravity 1020 or more, which do not coagulate spontaneously as ascitic fluid may. (2) From **echinococcus cyst**, whose fluid contains hooklets. (3) From **pancreatic cyst**, whose fluid will digest albumin and fibrin and which is confined to the upper part of the abdomen until very large. (4) From **distended bladder**, by catheterization. (5) From **pregnancy**, by absence of other signs of this condition in breasts and abdomen, and by vaginal examination.

Treatment.—(1) Of the disease causing the ascites. (2) To remove the fluid (*a*) in cirrhosis, by repeated tapping or by forming an anastomotic circulation by scraping liver, diaphragm, and omentum so as to cause formation of vascular adhesions; (*b*) in cardiac or renal disease, by hydragogue cathartics, such as compound jalap powder, ʒj, at night, followed by a concentrated dose of salts before breakfast. Potass. bitartrate in repeated doses of ʒj to ij with lemon juice in water, calomel, gr. $\frac{1}{10}$, every half-hour, for ten doses, or a few doses of calomel, digitalis, and squill, āā gr. j, may cause diuresis to remove the fluid.

SECTION VII

DISEASES OF THE RESPIRATORY SYSTEM

DISEASES OF THE NOSE AND LARYNX

(See volume, *Nose and Throat*, this series.)

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

Etiology.—Acute catarrhal inflammation of the larger and medium-sized bronchi is probably an infection. It is predisposed to by youth and advanced age, lack of fresh air and exercise, dusty occupations, poor general health, dampness and changeable weather in winter and early spring. It may be secondary to coryza or pharyngitis or to measles, typhoid, malaria, asthma, and cardiac disease.

Pathology.—Congestion, swelling, epithelial desquamation, and exudation of mucus or mucopus from the walls of the bronchi.

Symptoms.—Often preceded by coryza or pharyngitis, there is a sense of oppression with chilliness and pain in the back; a dry, tight feeling beneath the sternum, with a dry, harsh cough, which may cause headache and pain and a feeling of rawness in both chests, chiefly in front. There is little fever or a temperature from 101° to 103°. After a

few days the secretion becomes mucopurulent, then purulent and profuse, while other symptoms except cough subside. In a week or ten days this usually stops and recovery is complete. The bronchitis may become chronic. In infants and old persons there is danger of extension causing bronchopneumonia.

Physical Signs.—Respiration may be more rapid; bronchial fremitus may be felt; general sibilant or sonorous rales; later, moist rales, which may disappear temporarily after coughing. Localization of these signs is most common in tuberculous cases.

Diagnosis.—(1) From **bronchopneumonia**, by absence of areas of dulness and bronchial breathing and less severe symptoms. (2) From **acute pulmonary tuberculosis**, by lower temperature, less prostration, and absence of other symptoms of tuberculosis.

Prognosis.—Good except in infants and the aged, in whom death may occur from exhaustion, asphyxia, or bronchopneumonia.

Treatment.—Bed, hot bath, hot drinks, Dover's powder, gr. x, at night, and extra blankets to encourage sweating. A saline cathartic in the morning. In the dry stage, counter-irritation with mustard paste; mist. glycyrrhizæ comp., ℥j to ij, or Dover's powder, gr. v, every three or four hours. Inhalation of steam from boiling water to which is added compound tincture of benzoin or turpentine, ℥j. Later, heroin, gr. $\frac{1}{20}$ to $\frac{1}{12}$, or codeine, gr. ¼, with dilute hydrocyanic acid, ♏j, ammonium chloride, gr. v, syr. prun. virg., q. s. ad ℥j, every three or four hours. If cough with profuse sputum persists, cod-liver oil after meals is often effective. In infancy and old age careful nourishment and stimulation are necessary, and opium derivatives must be used with care, especially if secretion is profuse. In children an emetic should be given if profuse secretion is not well expectorated or dyspnea and cyanosis are present.

CHRONIC BRONCHITIS

Etiology.—It occurs most frequently in persons beyond middle age, chiefly in winter, in changeable, cold, and damp climates. It may follow repeated acute attacks or accompany chronic pulmonary troubles, valvular disease, aneurysm of the aorta, nephritis, and gout.

Pathology.—The submucosa and muscularis of the bronchi may be hyperplastic or atrophied, with irregularity and desquamation of the epithelium of the mucosa. Emphysema is present and bronchiectatic dilatations are common.

Symptoms.—In ordinary cases the symptoms are very variable, and are present chiefly in winter and damp weather. Dyspnea on exertion; cough, often worse at night or in the morning; sputum variable, usually profuse, mucopurulent, or purulent, sometimes thin mucoid. There may be slight fever at times. Exacerbations occur every winter, and finally the cough and expectoration may become constant. There are often added the symptoms of complicating emphysema or of the accompanying chronic cardiac, pulmonary, or other diseases.

Physical Signs.—Sibilant and sonorous, fine and coarse mucous rales, usually over the entire chest. Subcrepitant rales at the bases if the small bronchi are involved. In prolonged cases usually signs of emphysema also.

Clinical Varieties of Chronic Bronchitis.—In addition to this usual form there are:

1. **Bronchorrhea,** cases with very profuse sputum, usually thin, purulent, greenish, less often thick and purulent. This usually begins in young adults and may cause no special trouble or be followed by dilatation of the bronchi and putrid bronchitis.

2. **Putrid or Fetid Bronchitis.**—Sputum of fetid odor, profuse, thin, grayish white, separating on standing into three layers, the uppermost of frothy mucus, the middle of clear mucoserous fluid, the lowest of thick pus containing yellow

lumps as large as peas, the so-called Dittrich's plugs. There is quite frequently fever. Pulmonary gangrene or abscess and empyema are sequelæ.

3. **Dry Catarrh.**—Severe paroxysmal cough, with little or no sputum.

Treatment.—*General:* Warm, equable climate, such as southern California, Florida, or the south of France, especially in the colder months; warm clothing. Avoid exposure and fatigue. Treat the underlying disease—tuberculosis, gout, nephritis, cardiac disease, etc. *Medicinal:* Potassium iodide, gr. v to x, p. c. or every four hours. For profuse secretion, ammonium chloride, senega, turpentine, terebene, balsam of Tolu, eucalyptol. For fetor, inhalations of creosote or carbolic acid.

BRONCHIECTASIS

General or localized dilatation of the bronchi.

Etiology.—(1) Congenital, rare. (2) Weakening of the bronchial walls by inflammation with accumulation of secretion and pressure due to coughing, in chronic bronchitis, emphysema, bronchopneumonia and chronic tuberculosis. (3) Accumulation of secretion due to compression of bronchus by aneurysm or tumor or to obstruction by foreign body. (4) Contraction of lung tissue from interstitial pneumonia or pleuritic thickening.

Pathology.—In congenital cases the dilatation is usually unilateral and general. In others it is sacculated or cylindrical, and involves only some of the bronchi. They may be very small or several inches in diameter. The walls are atrophic or thickened; the mucosa is sometimes ulcerated. The contents are thick, grayish brown, fetid, purulent, or mucopurulent.

Symptoms.—In moderate cases the symptoms are masked by those of the causal disease, or are those of fetid chronic bronchitis. With large sacculated dilatations the cough occurs

in attacks, usually in the morning on arising or after certain changes of position. The sputum then is brought up in large quantites. It is sour or fetid, grayish brown, fluid, purulent. It separates on standing into three layers—the top frothy, brownish, or yellowish; the middle clear, mucoserous; the lowest greenish, yellowish, or brownish, thick, purulent. It contains pus, fatty-acid crystals, bacteria, pigment, sometimes elastic fibers if ulceration has occurred. Hemorrhage quite frequently occurs. Osteo-arthropathy with cyanosis on exertion may develop. The disease is chronic. Death usually eventually occurs from the disease causing the bronchiectasis.

Physical signs of the condition causing bronchiectasis may be present. Signs of a cavity may develop—cracked-pot resonance or tympany, cavernous breathing and voice, and gurgling rales. These disappear if the cavity is full.

Diagnosis.—(1) From **chronic bronchitis**, by signs of a cavity and periodical profuse expectoration influenced by position. (2) From **tuberculous cavity**, by absence of tubercle bacilli, of signs of consolidation around the cavity, and of symptoms of tuberculosis. Situation low down posteriorly suggests bronchiectasis. (3) From **sacculated empyema** perforating a bronchus, by history and by infrequent expectoration. (4) From **actinomycosis**, by absence of ray fungus from sputum. (5) From **gangrene** or **cancer**, by absence of lung tissue except, rarely, a little elastic tissue.

Treatment.—*Medical:* Inhalations of creosote, carbolic acid, thymol, etc. For profuse expectoration, terebene or turpentine, ♏v to x, every four hours. Intratracheal injection twice a day of a dram of a mixture of guaiacol, 2 parts; menthol, 10 parts; olive oil, 88 parts, is recommended. *Surgical:* Drainage of the cavity may be tried.

BRONCHIAL ASTHMA (SPASMODIC ASTHMA)

Definition.—Dyspnea due to spasmodic change in the smaller bronchi, probably a neurosis.

Etiology.—Four theories are that it is due to: (1) Neurotic spasm of the bronchial muscle. (2) Swelling of the mucosa from simple hyperemia, a vasomotor change. (3) Inflammation of the small bronchioles. (4) Protein sensitization; an anaphylactic attack. Hereditary influence is evident and individual susceptibility marked, especially in neurotic persons. It occurs at all ages, but usually begins in the young, particularly males. Often follows pertussis or is associated with hypertrophic rhinitis, adenoids, polypi, etc. The exciting causes vary with the individual—change of climate or residence, dust, smoke, odors, indiscretions of diet, emotion, or reflex influences from other organs.

Pathology.—Emphysema and chronic bronchitis are usually present in cases of long standing.

Symptoms.—There are sometimes premonitory oppression in the chest, mental depression, dyspeptic or other symptoms, but the onset is usually sudden, often during the night. Dyspnea becomes intense. The patient cannot lie down, but often sits at an open window resting the elbows on a table. The face is pale and expression anxious. There is a feeling of great oppression in the chest and often dread of suffocation. Respiration, though labored, is not unusually frequent, as expiration is much prolonged. In severe or prolonged attacks there are cyanosis, sweating, coldness of the extremities, with small and frequent pulse and great drowsiness. The attack lasts a few minutes to many hours and may pass off suddenly, perhaps to recur soon or on several successive nights, with slight cough and difficulty in breathing in the intervals. The cough is nearly dry at first and sputum very tenacious. It consists of thin mucus containing small, round, gelatinous masses, the perles of Laennec, which can be unrolled under water into spirally arranged mucin casts of the small bronchi (Curschmann's spirals). Later it becomes mucopurulent. It contains also pointed octahedral crystals (Charcot-Leyden crystals). Eosinophiles are greatly increased in the blood. In protracted

cases symptoms of chronic bronchitis and emphysema develop.

Physical Signs.—Thorax fixed, expansion limited, inspiration quick, expiration prolonged; percussion note normal or hyperresonant if emphysema has developed; loud sibilant and sonorous rales; later, moist rales over the whole chest.

Diagnosis.—(1) From **spasm due to pressure** of an enlarged node or aneurysm, by other signs of mediastinal pressure. (2) From **spasm of the larynx**, in which dyspnea is inspiratory, and signs of emphysema and sibilant and sonorous rales are absent. (3) From **paroxysmal dyspnea of chronic nephritis** or **cardiac disease**, by absence of other signs of these troubles.

Prognosis.—May last for years. Not fatal in attacks, but bronchopneumonia may follow.

Treatment.—*Of attack:* Inhalation of chloroform or amyl nitrite, or of smoke from potassium nitrate paper or cigarettes containing stramonium, etc.; adrenalin chloride, 1 to 1000, ♏viii to xv hypodermically; oxygen inhalations may relieve and the pneumatic cabinet with compressed air sometimes benefits. *To prevent recurrence:* Potassium iodide, gr. v to xx, t. i. d. The heaviest meal should not be taken at night. Restrict carbohydrates to diminish flatulence, which may bring on attacks. Many do well in dry, elevated regions, but with emphysema they are usually better near the sea. A climate like that of Florida or southern California is of benefit to the asthma and the complicating emphysema and bronchitis.

FIBRINOUS BRONCHITIS

Definition.—A rather rare acute or chronic affection characterized by the formation and expectoration of so-called fibrinous casts of groups of bronchi. Casts are also sometimes formed in diphtheria, pneumonia, or tuberculosis, and blood casts in hemoptysis.

Etiology.—Unknown. It occurs most often in middle-aged, debilitated persons, in the spring.

Pathology.—The mucous membrane of the bronchi is congested, the epithelium intact or desquamated. The branching casts are white or cream colored, rolled up and mixed with mucus and blood, and consist chiefly of mucin.

Symptoms.—1. **Acute Form.**—Invasion usually with symptoms of simple acute bronchitis accompanying typhoid, pneumonia, or the eruptive fevers, sometimes with chill, high fever, dry cough, and dyspnea. Cough and dyspnea increase and casts of the bronchi are expectorated with or without hemoptysis. The relief so obtained is temporary and symptoms recur in a few hours. The attacks gradually become less severe and recovery takes place, or death from asphyxia occurs in from a few days to two weeks.

2. **Chronic Idiopathic Form.**—Attacks of dyspnea, cough, and pulmonary oppression occur at intervals of weeks or even years, with but slight fever and temporary relief after casts are expelled.

Physical signs during attacks are those of acute bronchitis, and over the region involved are diminished breath sounds and rales on coughing while the casts are present. To recognize the casts they should be placed in water to unfold.

Diagnosis.—From casts of bronchi in tuberculosis, pneumonia, hemoptysis, and diphtheria, and from fungus.

Treatment of attacks, as in acute bronchitis (p. 216). Also inhalation of atomized lime water, ether, or steam to loosen casts; expectorants; emetics to aid removal of casts. In the intervals, as in chronic bronchitis (p. 218).

DISEASES OF THE LUNGS

CIRCULATORY DISTURBANCES

1. **Active Hyperemia or Congestion.**—The result of inhalation of irritants, beginning inflammation, occlusion of

the vessels in another portion of the lung or great atmospheric pressure, as in caissons or deep-sea diving. It is practically the initial stage of lobar pneumonia, and the **symptoms** are those of the larval type. (See Larval Pneumonia, p. 57.)

2. **Passive Hyperemia or Congestion.**—(*a*) **Mechanical congestion** results from obstruction to return of blood to the heart, as in weakness, dilatation or valvular disease of the heart, or pressure on the vessels by aneurysm or tumor.

Pathology.—Lung is brownish pink or salmon colored, indurated (*brown induration*). The capillaries are distended and elongated, and the walls of the air vesicles show increase of connective tissue. The alveoli contain desquamated epithelium and brown, decomposed hemoglobin free in these cells and in the sputum.

Symptoms occur only when cardiac compensation fails. Then there are cough, dyspnea, and sputum containing numerous pigmented alveolar cells.

(*b*) **Hypostatic congestion** occurs in protracted diseases or adynamic states requiring prolonged maintenance of the same position while the heart action is weak.

Pathology.—The posterior portions of the lungs are dark, firm, and so heavy that they may sink in water. Much blood flows from the cut surface. The capillaries are engorged; the alveoli are filled with serum and sometimes red cells and desquamated epithelium.

Symptoms.—Slight cough and dyspnea, sometimes blood-stained sputum.

Physical Signs.—Slight dulness at the bases, with feeble breathing and moist rales. These clear up if the position is changed.

Treatment of Active Congestion.—Hot bath, cupping. For intense congestion, venesection. If this fails, the right auricle may be aspirated. **Of passive congestion**: Treat the causal condition. Venesection, if necessary. For hypostatic congestion, change position, stimulate gently.

3. **Pulmonary Edema.**—A transudation of serum into the alveolar walls and air cells.

Etiology.—Pulmonary edema occurs (1) as *acute infectious edema* in scarlet fever, smallpox, typhus, rheumatism, and influenza; (2) with pneumonia, pulmonary tuberculosis, etc., here a *collateral edema* or perhaps an acute infectious edema; (3) with acute cardiac dilatation or "a disproportion between the working power of the left" and right ventricles, in angina pectoris, myocarditis or valvular disease, or as a terminal affection in chronic nephritis, arteriosclerosis, cirrhosis of the liver, cachexias, or profound anemia; (4) in recurrent attacks of *paroxysmal pulmonary edema* in cases with myocardial weakness and high blood-pressure; (5) after paracentesis of thorax or abdomen, in some cerebral diseases, as angioneurotic edema, etc.

Pathology.—The entire lungs, or the bases and posterior portions, appear gelatinous, are heavy, pit on pressure, and when cut allow a large amount of serum, often frothy and also blood-stained if congestion is present, to exude from the air vesicles, alveolar tissue, and bronchi.

Symptoms.—Sudden or gradual onset of increasing dyspnea and cough, with profuse, watery, frothy, often blood-tinged sputum, audible rattling, orthopnea, and cyanosis; with infectious edema, fever, etc. **Physical signs**: Slight dulness at the bases, with fine moist rales over the whole lungs.

Prognosis.—May prove fatal within an hour. Sometimes recovery in chronic cases.

Treatment.—Dry cupping; atropine, gr. $\frac{1}{100}$, hypodermically; venesection; purgation; oxygen inhalations; amyl nitrite or glonoin to reduce blood-pressure and so reduce work of left ventricle; morphine; later, strychnine, caffeine, strophanthus or digitalis if needed for cardiac weakness.

4. **Pulmonary Hemorrhage.**—(*a*) **Bronchopulmonary hemorrhage** or **hemoptysis** signifies spitting of blood exclusive of blood from the nose or pharynx. It occurs in apparently

healthy young persons without lesion of the lung so far as can be ascertained, without recurrence or subsequent illness; in pulmonary tuberculosis; in beginning pneumonia; in bronchitis, bronchiectasis, emphysema, abscess, cancer, or gangrene of the lung; with ulceration of larynx, trachea, or bronchi; in chronic valvular disease, especially mitral; in aneurysm of branches of the pulmonary artery; as vicarious menstruation; in hysteria; in persons over fifty with the arthritic diathesis; in purpura hemorrhagica; in malignant fevers; in parasitic lung diseases.

Symptoms.—Usually sudden, without apparent cause or after exertion, a warm, salty taste in the mouth, with expectoration of a variable quantity of bright, frothy, fluid blood, followed by coughing. This may recur for several days, with blood-streaked sputum for some time longer. Some of the blood may be swallowed and vomited in dark, clotted masses, and the stools may become dark. Moist rales may be heard over the area of lung affected.

Diagnosis.—By occurrence of coughing rather than vomiting at first, and by bright, frothy, fluid character of blood. Bleeding from the nose and pharynx is excluded by inspection; blood from the stomach is usually dark, clotted, and vomited. Localized moist rales not previously present suggest pulmonary hemorrhage.

Prognosis.—Death rarely occurs during the hemorrhage, which usually stops spontaneously. If from a large vessel or aneurysm it is usually fatal.

Treatment.—Absolute rest, lying on the affected side if it is known. Light diet and saline purgation if necessary, to diminish blood-pressure. Aconite may be given if the heart is overacting. Opium freely for cough except during profuse hemorrhage, when coughing must be permitted to prevent the bronchi from filling with blood.

(*b*) **Pulmonary apoplexy** or **hemorrhagic infarct** is localized hemorrhagic extravasation into the lung, due to embolism or thrombosis of a branch of the pulmonary artery. It

usually occurs after valvular disease, sometimes after thrombosis of a distant vein.

Pathology.—Infarctions are single or multiple, most often in the lower lobes, and usually near their surface. They are wedge-shaped, dark red, firm areas the size of a walnut to that of an orange, with their bases toward the surface of the lung. If they reach the surface this area of pleura is often inflamed. The air cells are distended with red cells. The area may subsequently be replaced by cicatricial tissue, or possibly the blood may be absorbed. If the embolus is infectious, abscess or gangrene ensues.

Symptoms may be absent or there is sudden onset of severe pain, dyspnea, and sometimes chill and fever. Hemoptysis is the leading symptom. Over the area involved there may be dulness and bronchial breathing.

Diagnosis from **pneumonia**, by history and signs of valvular disease, milder general symptoms, and localization of physical signs—dulness, harsh breathing, and rales.

Treatment.—The treatment is symptomatic.

BRONCHOPNEUMONIA

Definition.—An acute inflammation of the terminal bronchi of individual lobules and then of their air cells. Called also **lobular** or **catarrhal pneumonia** or **capillary bronchitis.**

Etiology.—**Primary bronchopneumonia**, most common under two years of age, is usually a pneumococcus infection.

Secondary bronchopneumonia occurs particularly among children with rickets or after measles, diphtheria, pertussis, influenza, scarlet fever, typhoid, and smallpox. It is usually a mixed infection with the streptococcus, staphylococci, pneumococcus, etc. In adults it often occurs as a terminal affection in chronic pulmonary or renal disease. It also follows the entrance of foreign bodies into the larynx and bronchi during anesthesia or coma or after operations on the nose and mouth. These cases are called **aspiration, inhalation,**

or **deglutition pneumonia.** (For tuberculous bronchopneumonia, see p. 97.)

Pathology.—The surface of the lungs shows projecting areas over which there is fibrinous pleurisy if the consolidation reaches the pleura, darker areas of atelectasis, and sometimes compensatory emphysema of other portions. The solidified areas are firm. They may be small and consist of zones of inflammation around single bronchi, or these may coalesce until a large part of the lung is involved. On section each involved bronchus appears as a light spot from which pus exudes. Around these bronchi are slightly projecting red areas which may be reddish gray at the center. Microscopically, the bronchial wall is swollen and infiltrated with white cells. The lumen of the bronchus contains an exudate of leukocytes and epithelium. The surrounding air vesicles contain the same and also fibrin and red cells, less abundantly and less often than in lobar pneumonia. In aspiration or deglutition pneumonia there is more marked infiltration resulting in abscess formation or gangrene. Bronchopneumonia terminates in (1) resolution, which occurs rapidly; (2) suppuration or gangrene, usually in aspiration pneumonia; (3) chronic interstitial bronchopneumonia, with connective-tissue proliferation; (4) secondary infection with Bacillus tuberculosis or activation of a latent infection.

Symptoms.—(*a*) **Primary cases** begin suddenly with a convulsion or chill, vomiting, and rapid rise of temperature. Respiration is frequent. In some cases the cough may be very slight and cerebral symptoms marked. (*b*) **Secondary cases**: After an ordinary bronchitis, measles, pertussis, etc., the temperature rises, with increased frequency of pulse and respiration, dyspnea, and severe, often painful cough. Temperature, 102° to 104°; respirations may reach 60 to 80; inspiration is labored, with dilatation of the alæ nasi, and expiration may be grunting. Face anxious and becoming cyanotic. **Physical signs** are those of bronchitis—sibilant and subcrepitant rales. Later there may be areas of

slight or distinct dulness and harsh breathing. **Course**: Cyanosis may increase, other symptoms decreasing as asphyxia deepens, rales become moist, and death occurs from cardiac weakness. Favorable cases gradually improve and clear up in about a week. A few develop chronic interstitial pneumonia (p. 229), or tuberculosis may become active.

Diagnosis.—(*a*) *Of primary cases* from **lobar pneumonia**, by absence of dulness, or, if involvement is so extensive as to give a large area of dulness, by presence of signs in other lung also. (*b*) *Of secondary bronchopneumonia* from (1) **lobar pneumonia**, by preceding disease (see Etiology), absence of sudden invasion with chill or convulsion, and usual bilaterality of involvement, with absence of marked signs of consolidation. From (2) **acute tuberculosis** is difficult at first. Localization of signs at the apices, especially if signs of softening are present, and occurrence of night-sweats suggest tuberculosis. Sputum, if obtainable, may show tubercle bacilli.

Prognosis.—Good in primary cases. In secondary, many infants and old persons die, but much depends on the general previous condition. Aspiration pneumonia is usually fatal.

Treatment.—*Prophylaxis.*—Avoid exposure to sudden changes of temperature and use antiseptic mouth washes in acute infections. *Of the attack:* Bed; constant room temperature of 68°, with air kept moist. Bowels opened by calomel or castor oil and kept open daily if necessary. Diet of milk, broths, and egg albumen. Water *ad lib.* For high fever and cerebral symptoms, wet pack or a gradually cooled bath, or tincture of aconite, ♏j to iss. For cough, ammonium chloride, gr. j to ij, or ammonium carb., gr. ¼ to j, with syrup of ipecac, ♏v to x, or senega or squill. If mucus is raised with difficulty an occasional emetic will relieve. For pain in chest, hot applications. If these fail, or for obstinate cough, Dover's powder in small doses. For cyanosis, frequent inhalations of oxygen. For cardiac weakness, strophanthus, strychnine, brandy, ♏x to xx, for children, at intervals. For cardiac failure, hot and cold douches, ether hypodermically.

CHRONIC INTERSTITIAL PNEUMONIA

Definition.—Called also **cirrhosis of the lung.** This is a chronic inflammation of the pulmonary interstitial tissue resulting in increase and subsequent contraction of the connective tissue and obliteration of air spaces.

Etiology.—Primary cases are due to inhalation of dust, and are known as **pneumokoniosis** (p. 230). Secondary cases follow lobar or bronchopneumonia, chronic bronchitis, chronic pleurisy, and syphilis. Similar fibroid changes occur in emphysema and tuberculosis. Local fibrosis occurs around foreign bodies or from pressure by aneurysm or tumor.

Pathology.—Connective-tissue hyperplasia begins around the bronchi, in the exudate within air cells, in interlobar septa, or in the pleura. (*a*) The massive or lobar form involves one lobe or lung. The affected side of the chest is greatly contracted. The heart is drawn toward that side. The other lung is emphysematous; the one involved is small, hard, and may contain more or less dilated bronchi in a mass of grayish fibroid tissue. These bronchiectatic cavities may contain aneurysms of the pulmonary artery. (*b*) In the bronchopneumonic, insular, or diffuse form, the firm areas are smaller, less dense, and chiefly in the lower lobes.

Symptoms.—Chronic paroxysmal cough, dyspnea on exertion. Sputum usually profuse, mucopurulent, sometimes fetid. Hemorrhage often occurs. General condition fair. Disease may last many years. Death occurs from failure of the right heart, hemorrhage, or amyloid disease.

Physical Signs.—In marked cases the affected side is greatly contracted and without expansion, while the other is apparently enlarged. Lateral curvature of the spine. Fremitus is increased. Percussion shows dulness or flatness, with tympanitic quality over cavities. The other lung is hyperresonant. Breath sounds are tubular, cavernous, or amphoric, with coarse mucous rales. There are signs of cardiac displacement.

Diagnosis.—From **tuberculosis** by history, general health, absence of signs in other lung, and of bacilli in the sputum.

Treatment.—Mild climate, with care of general nutrition, cod-liver oil, etc. (For fetid sputum, see Putrid Bronchitis, Treatment, page 218.)

PNEUMOKONIOSIS

A form of pulmonary fibrosis due to inhalation of dust. It includes **anthracosis** due to coal dust; **siderosis** caused by metallic dust, especially iron; **chalicosis** from mineral dust, occurring in stonecutters.

Pathology.—When the dust inhaled passes the carrying capacity of the phagocytes and bronchial ciliated epithelium, it is stored in the lymph spaces and in the connective tissue of the lungs, which undergoes proliferation and pigmentation. Softening of affected areas may cause cavity formation, and if these cavities communicate with a bronchus mixed infection may occur. Chronic bronchitis and emphysema are common. Secondary tuberculous infection may occur.

Symptoms are those of chronic bronchitis with profuse mucopurulent sputum, and emphysema with dyspnea and asthmatic attacks.

Physical signs are those of emphysema, chronic bronchitis, sometimes of cavities.

Diagnosis.—By occupation and dusty sputum.

Treatment is that of chlorine bronchitis and emphysema.

EMPHYSEMA

Definition.—A condition characterized by dilatation of the alveoli and infundibula and atrophy of the alveolar walls.

1. **Compensatory Emphysema.**—Distention of the air vesicles of normal lung tissue to fill the space within the pleural cavity due to contraction of other portions, as in tuberculosis, bronchopneumonia, chronic interstitial pneu-

monia, pleurisy with effusion, etc. If the causal condition is removed the emphysema disappears. If the cause persists, true emphysema develops.

2. **Hypertrophic Emphysema.**—Called also **substantive, idiopathic,** or **large-lunged emphysema.**

Etiology.—Heredity appears to have a causal influence by causing congenital weakness of lung tissue, often in children. Occurs in glassblowers, in musicians using wind instruments, after pertussis, asthma, chronic coughs, and other conditions requiring increased pressure within the air cells.

Pathology.—Chest large, barrel-shaped. Anterior margins of lungs cover pericardium. Lungs large and pit on pressure. Large air vesicles seen under the pleura. Microscopically, atrophy of alveolar walls with junction of adjoining vesicles and disappearance of their bloodvessels. Bronchi show chronic bronchitis with thickening of their mucosa. Fibrous tissue around the bronchi is increased; the smaller bronchi may be dilated. The right side of the heart is hypertrophied and dilated; in chronic cases both sides hypertrophy. Passive congestion of other viscera.

Symptoms.—Beginning with slight dyspnea on exertion, the disease runs a very chronic course with exacerbations due to bronchitis and failure of cardiac compensation. Dyspnea may be nearly constant or occur only after exertion or eating. It is chiefly expiratory, often with distinct wheezing. Subsequently asthmatic attacks often occur, with both inspiratory and expiratory dyspnea. Cyanosis is common and often more marked than in any other condition in which the patient can move about except congenital heart disease. Bronchitis may be mild in summer, but is usually brought about by even slight exposure in winter, and causes marked increase of dyspnea and cyanosis and often severe asthma. It becomes more frequent as age advances. Death may occur from lobar pneumonia or bronchopneumonia or failure of cardiac compensation.

Physical Signs.—*Inspection:* Chest is barrel-shaped, its anteroposterior diameter increased, ribs nearly horizontal, intercostal spaces wide, sternum and clavicles prominent. The neck appears short from elevation of clavicles and shoulders; the back is rounded. Inspiration is short and energetic, but the chest expands very little and appears to rise as though a single structure. Expiration is prolonged and the thorax sinks slowly. The accessory as well as the ordinary respiratory muscles are large and prominent. There are cyanosis, distention, and often pulsation of the cervical veins. The apex beat of the heart is not visible, but there is usually strong epigastric pulsation. *Palpation:* Vocal fremitus diminished. Apex beat not felt; marked impulse under the lower part of the sternum; epigastric pulsation. *Percussion:* Hyperresonance, often of "woody" quality. Upper border of hepatic and of splenic dulness lower than normal; area of cardiac dulness diminished or obliterated. *Auscultation:* Inspiratory murmur short and feeble; expiratory prolonged, low-pitched, often with sibilant and coarse rales. With bronchitis moist rales are present. Pulmonic second sound accentuated. In late stage, tricuspid systolic murmur may be present.

Diagnosis.—By dyspnea, cyanosis, bronchitis, characteristic appearance of chest, hyperresonant note, diminished cardiac, hepatic, and splenic dulness, and prolonged, low-pitched expiration.

Prognosis.—Slowly progressive course.

Treatment.—Warm, dry climate to avoid bronchitis; hygienic mode of life; avoid gastro-intestinal disturbance; relieve nasopharyngeal obstructions. The treatment is chiefly that of chronic bronchitis (p. 218). For extreme cyanosis, inhalation of oxygen, strychnine; in robust patients, venesection.

3. **Atrophic Emphysema.**—Known also as **senile** or **small-lunged emphysema.** It occurs in thin old persons who have small chests with very obliquely placed ribs and other signs

of senility. There is usually a history of winter cough and dyspnea for years. The condition is one of atrophy of the walls of the air cells, numbers of which may coalesce. No treatment.

4. **Acute Vesicular Emphysema.**—Acute distention of the air cells by strong inspiratory efforts in bronchitis of the smaller tubes, bronchopneumonia, cardiac dyspnea, angina pectoris, etc. The lungs are enlarged, as may be detected by the hyperresonant note, diminished cardiac and hepatic dulness, prolonged expiration, and general sibilant rales.

5. **Interstitial Emphysema.**—The presence of air in the interstitial tissue of the lungs due to rupture of air vesicles by severe expiratory effort in pertussis or other affections causing cough, in straining at stool, in labor, etc. The air collects in the interlobular tissue and beneath the pleura, and may work along the trachea into the neck. It may develop in the reverse direction after tracheotomy. Pneumothorax may result from interstitial emphysema.

ABSCESS OF THE LUNG

Etiology and Pathology.—(*a*) Lobar and lobular pneumonia, rarely the former; often aspiration pneumonia, particularly after operations upon the nose and throat. These abscesses are usually multiple, one or two inches in diameter, with ragged walls. (*b*) Embolic abscesses, in pyemia, usually multiple, begin as hemorrhagic infarcts beneath the pleura. These suppurate, forming abscesses. There is septic pleurisy, and perforation may occur, causing pyopneumothorax. (*c*) Perforation of the lung from the exterior or by cancer of the esophagus, by hepatic or subphrenic abscess or echinococcus cyst, and foreign bodies. (*d*) Secondary pyogenic infection in chronic tuberculosis.

Symptoms.—Fever, pain in lung, dyspnea, cough, sputum containing or consisting of pus of offensive odor with

fragments of elastic tissue, and physical signs of cavities. The symptoms of embolic abscesses are usually masked by those of the causal pyemia.

Prognosis.—Aspiration and embolic abscesses are usually fatal; recovery may follow those from foreign bodies or pneumonia.

Treatment.—Incision and drainage. Otherwise symptomatic.

GANGRENE OF THE LUNG

Definition.—Secondary putrefaction of necrotic areas of lung tissue.

Etiology.—Aspiration pneumonia, pulmonary tuberculosis; rarely lobar pneumonia, bronchopneumonia, bronchiectasis, fetid bronchitis, embolism of the pulmonary artery, rupture into the lung of an empyema or hydatid cyst or ulcer, foreign bodies, pressure of aneurysm or tumor, etc.

Pathology.—*Diffuse form:* After lobar pneumonia or embolism of a large branch of the pulmonary artery a large part of the lobe is a greenish-black, extremely offensive mass, ragged at the centre. *Circumscribed form:* One or more well-limited, greenish-brown areas breaking down into a greenish fluid, and surrounded by a zone of congestion or consolidation, and this by one of edema. Erosion of blood-vessels and perforation of the pleura may occur. There is intense bronchitis.

Symptoms.—To those of the preceding disease is added the characteristic sputum which is profuse, intensely fetid, and separates on standing into three layers, the lowest of thick, greenish-brown material, the middle thin, sometimes greenish or brownish, the top thick and frothy. It contains fragments of lung tissue. Hemorrhage may occur. Fever is moderate, pulse frequent. Symptoms of sepsis and of marked bronchitis develop.

Physical signs are those of a cavity in some cases. Death may occur from exhaustion or hemorrhage.

Diagnosis.—By sputum and fetid odor of breath.

Prognosis.—In young, robust patients recovery may occur.

Treatment.—Inhalation of carbolic acid, guaiacol, or formaldehyde. If a localized cavity is detected it may be incised and drained or injected with an antiseptic solution. Feeding and stimulation are important.

NEOPLASMS OF THE LUNG

Etiology and Pathology.—Primary growths, benign and malignant, are rare. Secondary growths are chiefly carcinoma and sarcoma, usually multiple if metastatic, single if a direct extension of the primary growth. The pleura is often the portion first involved. There is usually malignant serofibrinous or hemorrhagic pleurisy. Sarcoma occurs in the young and middle-aged, carcinoma after forty.

Symptoms are indefinite. There may be pain, dyspnea, dry, painful cough, sometimes "prune-juice" sputum; signs of compression of veins of the head and upper extremities; fever, cachexia. Death in one to eight months.

Physical Signs.—The cervical, subclavicular, and axillary lymph nodes may be enlarged. The signs are those of consolidation with usually those of pleuritic effusion.

Diagnosis.—By knowledge of primary lesson, unilateral and irregular signs, bloody sputum, glandular enlargement and cachexia. Microscopic examination of tissue fragment obtained by exploratory puncture of consolidated area may confirm.

Treatment.—Morphine for pain. Stimulation.

DISEASES OF THE PLEURA

ACUTE PLEURISY

1. **Fibrinous, Plastic, or Dry Pleurisy.—Etiology.**—*Primary:* Due to exposure or contusion of chest. Often bac-

teria are probably present. *Secondary:* Due to pneumonia, hemorrhagic infarction, tuberculosis, abscess or gangrene of the lung, cancer or wounds of the lung or chest wall, or pericarditis.

Pathology.—Part or even the whole of one pleura is lusterless, dry, coated with homogeneous or granular fibrin which later causes adhesion to the apposed pleural surface, to which the inflammatory process early extends. The mesothelium is swollen, degenerated, proliferated, and desquamated; the connective tissue beneath is proliferated and swollen.

Symptoms.—Sudden onset of sharp pain, usually in the lower part of the side of the chest, increased by deep inspiration, coughing, or motion. Rarely an initial chill and slight fever. Dry cough.

Physical Signs.—Sometimes local tenderness, usually only localized crepitant rales or a to-and-fro dry friction sound. After a few days symptoms and friction sound disappear.

2. **Serofibrinous Pleurisy or Pleurisy with Effusion.**—**Etiology.**—Same as fibrinous pleurisy. The majority of cases are tuberculous. The pneumococcus, streptococcus, etc., have also been found.

Pathology.—The pleural surfaces are covered with fibrin and the cavity contains a variable amount of serum, sometimes as much as four quarts. It is straw-colored, clear or slightly turbid from presence of fibrin, leukocytes, mesothelium, and some red cells, and is highly albuminous. In slight exudations the lower part of the lung is atelectatic; in large, the entire lung may be airless and the heart displaced toward the opposite side. In large right-sided effusions the liver is pushed downward. If recovery takes place the serum and fibrin are absorbed and granulation tissue forms permanent thickenings of the pleura or adhesions.

Symptoms.—The onset may be gradual with slowly increasing dyspnea and accumulation of a large amount of

fluid, or sudden with chill, fever, severe sharp pain in the side, increased by respiration, coughing, or motion; dry cough, dyspnea, temperature 102° or 103°. The pain is usually referred to the axilla or mammary region, sometimes to the abdomen or back. In about seven to ten days, fever and other symptoms disappear and the fluid is absorbed, quickly if scanty, often very slowly if abundant. It may become purulent or persist for months. Sudden death sometimes occurs.

Physical Signs.—*Inspection:* Depending upon the amount of fluid, there is some diminution of mobility of the affected chest, which may appear larger than the other, and the intercostal spaces may be less visible. The cardiac apex beat may be displaced toward the opposite side and upward. *Palpation* confirms diminished mobility of chest and displacement of apex beat. Tactile fremitus is absent over fluid or greatly diminished. *Mensuration:* Affected side may be one-half to one inch larger than the other. *Percussion:* Below the level of fluid, flatness or dulness and sense of resistance; over condensed lung, above the fluid, dulness to tympany, occasionally "cracked-pot" resonance; above this may be normal resonance. The upper border of fluid in moderate effusions runs in an S-shaped line, the "Ellis line of flatness," when the patient is upright. Beginning low near the spine it runs upward and forward, in a line concave upward, to the highest point in the axilla, then descends toward the sternum. Slight mobility of the fluid is sometimes shown by percussing the upper border in the mammary region while sitting, then while recumbent, and noting the change of the level of dulness. Lower border of liver dulness is displaced downward by large right-sided effusions. *Auscultation:* At first a pleuritic friction sound, which disappears as fluid accumulates. Below level of fluid, absence of breath and voice sounds, sometimes loud to distant tubular breathing and voice, occasionally with an amphoric quality; the whisper may be transmitted; rales are rarely heard. Above the fluid,

over condensed lung, breathing bronchovesicular or bronchial, voice increased or bronchial; above this, breathing and voice normal. Above fluid may be friction sounds. Over the sound lung hyperresonance and exaggerated breathing. Heart sounds displaced. Pleuropericardial friction sound in some cases. X-rays show a dark area, often with sharply defined upper level, over fluid, and displacement of the heart if this is present.

As absorption progresses friction sounds appear; dulness and diminished breath sounds at the base persist for months. Other signs gradually disappear.

3. **Purulent Pleurisy, or Empyema.—Etiology.**—(1) Usually follows serofibrinous pleurisy. In children it may be primarily purulent. (2) Often secondary to scarlet fever, pneumonia, pyemia, less often typhoid, etc. (3) Perforating wound, fracture of rib, perforation of pleura by extension of a tuberculous cavity, cancer of lung, of chest wall; or of esophagus. The most common organisms are streptococcus, staphylococcus, pneumococcus, and tubercle bacillus. Apparently sterile purulent exudates are usually tuberculous.

Pathology.—Fluid turbid to thick pus. In pneumococcus cases it is thick, creamy, with sweetish odor. In some cases it is fetid. Pleura thickened; fluid may be sacculated by adhesions.

Symptoms.—Invasion sometimes abrupt, with symptoms like serofibrinous pleurisy, but usually develops insidiously. There are usually cough and gradual onset of dyspnea. There are nearly always symptoms of sepsis—pallor, weakness, sweating, irregular fever, sometimes chills, marked leukocytosis. The empyema may point and discharge through a bronchus, adjacent viscus, or chest wall. Recovery may follow such drainage or absorption of the fluid.

Physical Signs.—Same as in serofibrinous pleurisy (p. 237), but there may be greater distention of the chest, bulging of intercostal spaces, and displacement of heart and liver. Chest wall may be edematous and subcutaneous veins dilated.

Pulsation of the chest wall, synchronous with that of the heart, may be present.

4. **Tuberculous Pleurisy.**—(See p. 95.)

5. **Minor Varieties of Pleurisy.**—(*a*) **Hemorrhagic pleurisy** occurs in tuberculous pleurisy, cancer of the pleura, asthenic conditions, such as cancer, cirrhosis of the liver, chronic nephritis, and malignant infectious cases. Injuries in aspirations may cause enough bleeding to make a clear fluid appear hemorrhagic. (*b*) **Diaphragmatic pleurisy**: Inflammation limited to the diaphragm, hence no rales are heard; fluid usually scanty. (*c*) **Interlobar pleurisy.** A collection of fluid between two lobes becomes sacculated by adhesions. The aspirating needle reaches the pus only after penetrating the lung. (*d*) **Encysted pleurisy**: Collections of fluid shut in by adhesions. These can often be diagnosticated only with the aspirating needle.

Diagnosis of Pleurisy.—(1) *Fibrinous pleurisy*, by sharp localized pain on inspiration with friction rub or crepitant rales. (2) *Pleurisy with large effusion*, by symptoms, physical signs, *x*-rays, and use of aspirating needle. (3) *Pleurisy with moderate effusion*, from (*a*) **lobar pneumonia**, in which the onset is more severe, with a chill and high temperature, dyspnea greater, sputum rusty, dulness less marked and usually higher posteriorly than in the axilla, tactile fremitus and voice sounds are increased rather than diminished, and bronchial breathing and whisper more common and less distant than in pleurisy with effusion, in which breath sounds are usually absent. The patient should always cough to clear the bronchi if doubt as to diagnosis exists. From (*b*) **hydrothorax**, which is usually bilateral, painless, and develops during chronic cardiac, nephritic, or hepatic disease, or cancer, with fluid of lower specific gravity and less albuminous. (*c*) From **pneumothorax** (p. 243). (*d*) From **large pericardial effusion**, in which dyspnea is extreme, the apex beat obscured or absent instead of displaced to the right, heart sounds and pulse are feeble. Only an encysted

pleurisy should cause confusion, as in this the area of dulness may be situated anteriorly while the axilla is resonant. (*e*) From **abscess of the liver, hydatid cysts** or **tumors,** and **subphrenic abscess,** by history, sometimes by upward convexity of the area of dulness in the back and of radiographic shadow in hepatic enlargements. The aspirating needle may differentiate all except subphrenic abscess, in which there is no cardiac displacement and the flow of pus through the needle does not intermit with inspiration. From (*f*) **cancer of lung** or **pleura** or **hydatids of pleura,** by aspiration. (4) **Of the character of the effusion,** by the aspirating needle. Symptoms and general appearance, such as pallor, sweating, irregular fever, and leukocytosis suggest pus but do not prove it, nor does their absence disprove its presence.

Treatment.—*General:* Bed, fluid diet, initial catharsis. For pain, strapping the affected side with zinc oxide plaster, hot applications, codeine, or morphine if needed. *For effusion,* limitation of fluids ingested unless there is thirst due to fever; saline cathartics in concentrated form, *e. g.*, Rochelle or Epsom salt, ʒss to ʒiss, daily before breakfast if patient is robust; sometimes diuretics; counter-irritation with tincture of iodine or mustard. If these fail or effusion is large, aspiration, repeated at intervals if fluid reaccumulates. Strict disinfection of hands, instrument, and chest wall. Patient bending forward with hand on opposite shoulder to widen intercostal spaces. Needle inserted close to upper border of rib in eighth space below angle of scapula or in seventh in midaxillary line. Ethyl chloride or cocaine anesthesia of skin if desired. Withdraw fluid slowly, stopping if there is severe pain or coughing, dyspnea or marked faintness, or feeble and irregular pulse. Whisky, ʒss, may be given in case of weakness or before aspiration in nervous patients. Codeine or morphine after tapping if pain or paroxysmal cough is severe. Pneumothorax, subcutaneous emphysema, albuminous sputum, and sudden death are occasional sequelæ. *For empyema,* resection of ribs and

free drainage. If patient is very feeble postpone resection of ribs but incise at once. Empyema in children and pneumococcus empyema, unless very small and encysted, should also be treated surgically, although these sometimes recover after aspiration.

CHRONIC PLEURISY

Is either (*a*) adhesive or dry, or (*b*) with effusion.

Etiology.—Same as acute pleurisy. It is secondary to acute pleurisy, either dry or with exudation, or develops slowly primarily.

Pathology.—(*a*) **Chronic adhesive pleurisy**: Usually unilateral and at the base; if tuberculous, at the apices. Pleura thickened, covered with thick layers of fibrin, and adherent to apposed pleura. When this contracts the chest wall is drawn in, the lung compressed, and bronchiectatic cavities form. Prolongations of connective tissue may extend from the pleura into the interlobular tissue. (*b*) **Chronic pleurisy with effusion**: Same as acute form, but pleura much thickened, covered with fibrin, and fluid often sacculated by adhesions.

Symptoms.—Occasional attacks of acute pleurisy, dyspnea on exertion, sometimes a dragging or sharp pain at the site of an adhesion.

Physical Signs.—Diminished expansion of the affected side; later, retraction of chest wall if adhesions contract; dulness, diminished fremitus, voice and breath sounds, absence of Litten's sign of diaphragmatic excursion. The heart may be drawn toward the affected side. X-rays show shadow over the affected area.

Diagnosis.—By physical signs and the resistance encountered in introducing an aspirating needle.

Prognosis.—Predisposes to bronchitis, bronchopneumonia, and bronchiectasis.

Treatment.—General hygienic measures and nutritious diet. For pain, same as acute pleurisy (p. 240). For contraction of chest, deep breathing and blowing into James' bottles.

HYDROTHORAX

Non-inflammatory transudation into the pleural cavity.

Etiology.—Secondary to diseases which may cause dropsy, *e. g.*, cardiac, renal, and pulmonary.

Pathology.—Usually bilateral, often unilateral in cardiac cases.

Symptoms and **physical signs** are those of pleurisy with effusion, usually of gradual onset with no pain.

Diagnosis.—By aspiration, specific gravity of fluid below 1015 and albumin low, and by lack of symptoms of inflammation.

Treatment is that of the cause and saline purges or diuretics to remove fluid, or aspiration, repeated if necessary.

PNEUMOTHORAX, HYDROPNEUMOTHORAX, PYOPNEUMOTHORAX

Definition.—Pneumothorax, or air in the pleural cavity, is rare; hydropneumothorax and pyopneumothorax are respectively, air with serous and purulent effusions.

Etiology.—Perforating wounds or pointing of empyema, pulmonary abscess or hydatid cyst through the chest wall; perforation of the lung by straining, tuberculous process, gangrene, or septic bronchopneumonia; perforation of the diaphragm in cancer of the stomach, esophagus, or intestine; growth of Bacillus aërogenes capsulatus within the chest.

Pathology.—Usually serous or purulent fluid and gas in the pleural cavity; compression of the lung, displacement of the heart toward the opposite side. The site of perforation may sometimes be found. The cause, usually tuberculosis, is generally obvious.

Symptoms.—Usually sudden sharp pain, severe dyspnea, cough, cyanosis, rapid and feeble pulse and respiration, some-

times collapse. If due to tuberculosis, symptoms may be slight. Septic temperature develops, especially if the effusion is purulent. Termination by death, recovery, or chronic pneumothorax.

Physical Signs.—*Inspection:* Enlargement and immobility of affected side. *Palpation:* Fremitus diminished over air, absent over fluid; apex beat displaced toward opposite side. *Percussion:* Flatness over fluid, changing level with position; over air, hyperresonant, tympanitic, or dull note. *Auscultation:* Above the fluid, absence or diminution of voice and breath sounds, or feeble amphoric breathing and voice; moist rales, metallic tinkle, succussion and coin sound. Below level of fluid, absence of breathing and voice sounds.

Diagnosis.—Chiefly by succussion, metallic tinkle, coin sound, and amphoric breathing. (1) From **pleurisy with effusion**, by presence of these signs, absence or amphoric character of breathing above the water level instead of tubular breathing. (2) From **large lung cavity**, in which the general nutrition is usually much worse. (3) From **diaphragmatic hernia**, in which the tympany is confined to the base of the thorax, abdominal pain and vomiting may be present, and intestinal gurgling is heard over the chest. (4) From **subphrenic pyopneumothorax**, in which the condition is right-sided, heart not displaced, pulmonary symptoms are absent and gastric or intestinal have preceded, pus flows from the needle without inspiratory intermission, and *x*-rays show shadow convex upward.

Prognosis.—Tuberculous cases usually die soon; persons previously healthy often recover.

Treatment.—Same as serofibrinous pleurisy or empyema (p. 240), according to the character of the fluid.

DISEASES OF THE MEDIASTINUM

Inflammation.—Etiology.—(*a*) *Simple lymphadenitis:* Traumatism; inflammation of lungs, bronchi, pleura, or peri-

cardium. (*b*) *Suppurative lymphadenitis* or *mediastinitis* (abscess of mediastinum): Traumatism; extension from local suppurative foci in neck, lungs, bronchial glands, thymus or chest wall; perforation of esophagus; and metastasis in acute infectious diseases. (*c*) *Tuberculous lymphadenitis:* Primary in lymph nodes or by extension from vertebræ.

Pathology.—Simple lymphadenitis shows the usual lesions of the nodes. In rare chronic cases (*indurative mediastinopericarditis*), associated with chronic adhesive pericarditis, the fibrous tissue of the mediastinum is increased, with subsequent contraction and adherent pericardium. In suppurative mediastinitis the pus infiltrates the anterior or posterior mediastinal tissue, or forms distinct abscesses which may point through adjacent viscera or the skin. In chronic abscesses (tuberculous) the pus may become inspissated.

Tumors.—Chiefly cancer and sarcoma; most often in males from thirty to forty years. They originate in the thymus, lymph nodes, or pleura and lung. The growths may be part of a general lymphadenomatosis or Hodgkin's disease (p. 290). They may compress any of the intrathoracic structures and cause pleural effusion.

Symptoms.—(*a*) *Of suppurative mediastinitis* (abscess) are retrosternal pain; dyspnea, if large; symptoms of sepsis, with subsequent pointing through the skin or adjacent organs. A pulsating tumor may be present at the edge of the sternum, pulsation not expansile, no diastolic shock. (*b*) *Of chronic indurative mediastinitis.* (See Chronic Adhesive Pericarditis, p. 251.) (*c*) *Of tumor* are due to pressure upon bronchi, vagus, and recurrent laryngeal nerves, esophagus, heart, and great veins. Dyspnea, paroxysmal cough, sometimes brassy, hoarseness, dysphagia, pain beneath the sternum. In malignant cases, anemia, emaciation, cachexia. Death from exhaustion or asphyxia.

Physical signs are chiefly those of pressure, as in aortic aneurysm. There may be cyanosis of face and neck; dilatation

of jugular veins; dilated and tortuous branches of internal mammary artery anastomosing with superficial epigastric; bulging of sternum, or perforation of chest wall with protrusion of tumor; displacement of apex beat; clubbing of fingers; inequality of pupils; paralysis of vocal cord. Over the tumor there may be transmitted pulsation, dulness, absence of fremitus, breath and voice sounds. Often signs of pleural effusion. X-rays show shadow in mediastinal region.

Diagnosis.—*Abscess* is suggested by septic symptoms; *malignant disease*, by cachexia. Diagnosis of *tumor* from **aneurysm** is uncertain. Both cause pressure symptoms, but tumor usually develops more rapidly, has no diastolic shock, thrill, or expansile pulsation, rarely tracheal tug, but often pleural effusion; sometimes secondary tumors in neck. Improvement under potassium iodide favors aneurysm. The shape and position of the radiographic shadow may be conclusive; if not, fluoroscopy may differentiate the expansile pulsation of an aneurysm from tumor which is stationary or shows only transmitted pulsation.

Treatment.—Of abscess, drainage may cure. Of tumor, treat with mercury and iodide to exclude syphilis. Morphine for pain, cough, and dyspnea.

SECTION VIII

DISEASES OF THE CIRCULATORY SYSTEM

DISEASES OF THE PERICARDIUM

PERICARDITIS

Etiology.—(*a*) *Primary pericarditis* is rare. It apparently occurs in previously healthy children, but these cases may be tuberculous or due to unrecognized infection. It may be traumatic. (*b*) *Secondary* to (1) acute rheumatism or with rheumatic tonsillitis; (2) acute infectious diseases, as pneumonia, scarlet fever, influenza, etc.; (3) acute sepsis, as puerperal infection, malignant endocarditis, septicemia, necrosis of bone, and infection at the umbilicus in infancy; (4) tuberculosis, primary in serous membranes or secondary; (5) gout, chronic nephritis. (*c*) *By extension* from pleuropneumonia, especially in children and alcoholics, purulent myocarditis, ulcerative endocarditis, and aneurysm of the aorta.

Acute Plastic or Dry Pericarditis. — Pathology.—Usually secondary; often tuberculous. Partial or general. Pericardium dull or covered with a thin layer of fibrin, beneath which are many hemorrhagic spots. Later the fibrin may be thick and irregular, and the two fibrinous surfaces may become permanently adherent. There is usually some increase of

pericardial fluid. In some cases pericarditis with effusion follows, or the disease becomes chronic (tuberculous cases).

Symptoms may be absent. There may be slight pain in the precordia. Fever moderate. These subside or symptoms of pericarditis with effusion develop.

Physical Signs.—There may be friction fremitus, especially over the right ventricle. A friction sound, usually to and fro, less often single or triple, is more frequently present. It is superficial, dry, most often heard in the fourth and fifth intercostal spaces near the sternum, is increased by pressure of the stethoscope or by bending forward, and is often alternately present and absent. It is usually harsher than endocardial murmurs and is not transmitted along bloodvessels, though sometimes along the sternum.

Diagnosis from double murmur of **aortic insufficiency**, by the characteristics just mentioned and by the absence of the pulse of that lesion. A **pleuropericardial friction sound** is loudest along the left side of the heart, varies greatly with respiration, and may disappear if the breath is held.

Pericarditis with Effusion. — Pathology. — Usually occurs with acute rheumatism, tuberculosis, and septicemia. Beginning as in plastic pericarditis (see Pathology, p. 247), a serofibrinous, hemorrhagic, or purulent exudate accumulates in the pericardial cavity, varying from two ounces to two quarts. In serofibrinous cases the pericardium is covered with a thick, irregular layer of fibrin. Hemorrhagic exudate is usually more scanty, with hemorrhage into the pericardium. It is found chiefly with tuberculous or cancerous pericarditis. Purulent exudate may be such from the beginning or follow serofibrinous. It contains pus and red cells as well as serum and fibrin. With this form myocarditis or fatty degeneration of the heart may occur.

Symptoms.—Invasion usually without marked symptoms. There may be discomfort or sharp pain in the cardiac region, increased by pressure at the lower part of the sternum. Temperature is irregular or intermittent, and not high; with

purulent exudate it is of distinctly septic type. With the effusion comes dyspnea, which may become intense. The patient is cyanotic and lies on the left side, or must even sit up. Pulse is frequent, small, and weak, or is the pulsus paradoxus, which is weak or lost during inspiration. There may be distention of the veins of the neck, cough, aphonia, and dysphagia, all from pressure. In severe cases insomnia and restlessness and later delirium and coma may be present. Melancholia with suicidal tendency and chorea may occur, rarely epilepsy during paracentesis. Effusion may reach its maximum in two or three days or several weeks, and absorption varies similarly. (See Prognosis.)

Physical Signs.—*Inspection:* Pallor, cyanosis, face anxious, veins of neck distended, respiration frequent and irregular. In children there may be bulging of the precordia and intercostal spaces, with apparent slight enlargement of the left chest, whose expansion is diminished. Skin of precordia may be edematous, apex beat not seen, epigastrium prominent. *Palpation:* Apex beat displaced upward and to left, or not felt. Friction fremitus lost or felt only at the base. *Percussion:* Dulness in second left intercostal space and fifth right near the sternum. Increase of area of cardiac dulness or flatness, which becomes irregularly triangular or pyriform, with apex at about the second costal cartilage, and base downward. It extends below and to the left of the apex beat and to the right so far as to make the cardiohepatic angle of dulness obtuse. There may be dulness in the left infrascapular region and tympany over compressed portion of the left lung. *Auscultation:* Friction sound at the base only and lost when effusion increases, returning as it is absorbed. Heart sounds become distant and feeble; action frequent and irregular. Bronchovesicular or feeble breathing over compressed portion of left lung, varying, as does change in resonance, with alteration of position. X-rays show enlarged cardiac shadow, with absence of pulsation along its left border if effusion is large.

Diagnosis.—Daily cardiac examination in acute rheumatism and other infectious diseases. (1) From **dilatation of the heart,** in which there are, usually, a history of valvular disease; visible but wavy apex beat; an area of dulness rarely triangular, not often extending to the left beyond the apex beat or, except in mitral stenosis, so high along the left border of the sternum, nor altering the cardiohepatic angle without visible or palpable impulse; while heart sounds are clearer, endocardial murmurs may be heard, and signs due to compression of left lung are absent. (2) From **encysted pleural effusion** (See Diagnosis of Pleurisy (*d*), p. 239). (3) From **pneumonia,** by disproportion between dyspnea, cyanosis, etc., and amount of lung apparently involved—the compressed area in infrascapular region.

Prognosis.—Good in serofibrinous cases, but with formation of adhesions. In septic cases death may occur in a few days. Tuberculous cases are slow, but recovery is rare.

Treatment.—Absolute rest; fluid diet; ice-bag or ice-coil over the precordia; local cupping or leeches are sometimes recommended for robust cases. If effusion is present, dry diet; in strong patients, purgation with salts in concentrated solution; diuresis by potassium acetate, citrate, or bitartrate, or infusion of digitalis, or calomel in small, repeated doses; sometimes blisters to the precordia. Potassium iodide, gr. v to x, t. i. d., is given. If effusion is so large as to cause dyspnea or other pressure symptoms, aspiration in serofibrinous cases in the fourth left intercostal space near the sternum or an inch from it, or in the fifth an inch and one-half from the sternum, or near the axillary border of the area of dulness. In purulent cases, incision and drainage. In convalescence, iron, arsenic, and strychnine.

Chronic Adhesive Pericarditis.—Etiology and Pathology.—Commonly follows acute pericarditis. It is partial or general; pericardium is thick, covered with fibrin with adhesions. There is simple adhesion of the two layers of pericardium; or the same with chronic mediastinitis, the outer

layer of pericardium being adherent to the pleura and chest wall and the heart often greatly hypertrophied and dilated.

Symptoms.—(1) With simple adhesion of the two layers they may be slight. (2) With chronic mediastinitis there are symptoms of cardiac hypertrophy and dilatation with failure of compensation, sometimes of associated chronic peritonitis with ascites, perihepatitis, and perisplenitis. Dyspnea and cyanosis may be marked.

Physical Signs.—In cases with chronic mediastinitis: *Inspection:* Precordia prominent; area of cardiac impulse large; systolic retraction of the chest wall at the apex; systolic tug visible at the attachment of the diaphragm if the heart is extensively adherent to the latter; diastolic collapse of cervical veins. If chronic peritonitis is associated there are signs of ascites. *Palpation:* Apex beat does not move with respiration or change of position. Diastolic shock felt. *Percussion:* Increased area of cardiac dulness. Adherent lung borders may be fixed so as not to move during respiration, area of cardiac dulness remaining unchanged. *Auscultation* may show murmurs of associated endocarditis or of relative insufficiency of one or more valves.

OTHER PERICARDIAL AFFECTIONS

Hydropericardium, or **dropsy of the pericardium,** is a non-inflammatory collection of serum in the pericardium as part of a general dropsy due to chronic nephritis, valvular disease, emphysema, or cirrhosis. The **symptoms** and **physical signs** are those of pericarditis with effusion, but without pain, fever, or friction sound. It may cause increase of dyspnea.

Chylopericardium is rare, the serum milky white.

Hemopericardium.—The pericardium is filled with blood from a penetrating wound or a ruptured aneurysm, heart,

or coronary artery. Death usually occurs rapidly from heart failure; in rupture of the heart it may be postponed several days, with dyspnea, cardiac failure, and signs of effusion.

Pneumopericardium is rare. The pericardium is distended with air or gas as the result of a penetrating wound or of perforation of the esophagus, lung, etc., or the presence of the Bacillus aërogenes capsulatus.

Symptoms are those of pericarditis with effusion and rapid heart failure.

Physical Signs.—Movable areas of dulness and tympany; splashing, churning, and sometimes friction sounds; heart sounds distant and feeble.

Treatment.—Surgical.

DISEASES OF THE HEART

ENDOCARDITIS

Inflammation of the endocardium, chiefly confined to the valves, may be acute or chronic. The former is divided clinically into simple or benign and malignant or ulcerative, although both are accepted as being of bacterial origin and the one runs imperceptibly into the other.

1. **Simple Acute Endocarditis.**—**Etiology.**—Occurs at all ages, but most often in children and young adults. Rarely primary. Probably always of bacterial origin. It is most frequently associated with acute rheumatism, chorea, tonsillitis, scarlet fever, and pneumonia, or is an exacerbation or chronic endocarditis. It is less common with other acute infectious diseases and with cancer, gout, diabetes, tuberculosis, and chronic nephritis.

Pathology.—The valves, chiefly in the left heart, and most often the mitral, are simply swollen or bear vegetations

1 to 4 mm. in diameter. These are sometimes pedunculated, sometimes undergo ulceration. They may become small and sclerotic, or become detached and form emboli, or be followed by lesions of malignant or of chronic endocarditis. The basement substance is first swollen and a growth of new connective-tissue cells may break through it. On such surfaces fibrin, leukocytes, and bacteria collect. These vegetations may become organized by further growth of connective tissue and later undergo contraction or ulceration. Streptococci, staphylococci, pneumococci, and other bacteria are found.

Symptoms and **physical signs** are very often absent or unrecognized. In cases of acute rheumatism there may be rise of temperature without increase of joint symptoms, but with increased infrequency and irregularity of heart action. Impurity of the first sound or murmurs may develop, but the latter are often hemic. Sudden variability of a murmur suggests acute endocarditis.

Prognosis.—Recovery or death may occur. More often chronic valvular disease with acute exacerbations follows.

Treatment.—Rest during attacks of rheumatism, chorea, or diseases with which endocarditis is commonly associated. Treatment of tonsillitis, etc. Absolute rest after the disease develops. For overaction of the heart, ice-bag over the precordia, sometimes aconite. There is no known curative treatment, but salicylates and alkalies should be given in rheumatic cases, as they may influence the causal disease. Diet chiefly fluid, but not restricted. Stimulation as needed. Avoid early exertion after convalescence.

2. **Malignant endocarditis** is called also **ulcerative, infectious,** and **diphtheritic.**

Etiology.—Rarely primary; usually secondary to pneumonia, acute rheumatism, other acute infectious diseases, or septic processes, such as septicemia from any cause, erysipelas, and gonorrhea.

Pathology.—Vegetations form upon the valves as in simple acute endocarditis (p. 252), but are often larger and covered with large thrombi containing many bacteria, which may break off and form embolic abscesses in various parts of the body. Necrosis of the vegetations, underlying endocardium, and sometimes of the deeper cardiac tissue, is characteristic of the malignant type and may cause perforation of a valve of the heart. Blood cultures often show bacteria.

Symptoms.—Two general types occur, resembling sepsis and typhoid. Arriving during another disease, the onset is marked by a sudden rise of temperature, often to 104° or 105°, or in some cases by a chill also, and the pulse becomes frequent, irregular, and feeble. In the *typhoid type*, the more common, the temperature remains quite uniformly high; the tongue is heavily coated; sordes collect on the teeth; vomiting, abdominal distention, diarrhea, or constipation may be present. Cerebral symptoms are usually marked, and may even simulate meningitis—headache, restlessness, somnolence, delirium, and coma. Sweating and petechial or other eruptions may occur. In the *septic type* there are chills, sweating, and a septic temperature. The chills sometimes occur periodically, simulating quotidian or tertian malaria. *Cerebral* and *cardiac types* are also recognized, according as symptoms of either of these classes predominate. In any type there may be symptoms due to embolism and then abscess of the brain, retina, spleen, liver, kidneys, lung, or other organ. There is usually a sudden localized pain, followed by symptoms of local inflammation and then abscess. Cardiac symptoms are usually subordinate and often overlooked. In cases of chronic valvular disease symptoms of failure of compensation are more likely to be present. The disease usually terminates fatally in from a few days to six weeks.

Physical Signs.—As in simple acute endocarditis (p. 253).

Diagnosis.—By history of preceding illness, sudden onset, septic or typhoidal symptoms, sometimes by physical signs,

by blood cultures, and by exclusion of the following diseases; (1) **Simple endocarditis**, by greater severity of symptoms and by blood cultures. (2) **Typhoid fever**, by preceding infection, sudden onset, and precocity of severe symptoms, irregularity of temperature, symptoms of embolism, physical signs of endocarditis, occurrence of leukocytosis, and absence of Widal reaction. (3) **Malarial fever**, by blood examination. (4) **Acute miliary tuberculosis**, in which the local symptoms may be pulmonary rather than cardiac, and sputum may contain tubercle bacilli. (5) **Typhus fever, cerebrospinal meningitis**, and **hemorrhagic smallpox.**

Treatment.—Absolute rest; ice-bag over heart; autogenous vaccine; otherwise as in pyemia (p. 69).

3. **Chronic Endocarditis.—Etiology.**—Usually in persons under middle age. Generally follows acute endocarditis, and therefore, acute articular rheumatism or other acute infectious disease, but may be caused by syphilis, alcoholism, gout, and prolonged overexertion.

Pathology.—The lesions are chiefly in the valves, whose edges become thickened and opaque. In the aortic valve the process begins about the corpora arantii; in the auriculoventricular, just within the valve margin. The thickening is due to proliferation of connective tissue in the endocardium, and as this increases and contracts, the valve segments become rounded at the edges, distorted, sometimes adherent to each other or to the mural endocardium or intima of the aorta. The chordæ tendineæ and sometimes part of the papillary muscles usually become sclerotic and their contraction causes stenosis of the valve by drawing the segments together. Contraction of valve segments which have become adherent to each other also causes stenosis. The thickened, distorted segments cannot meet exactly and the valve becomes incompetent. Insufficiency and stenosis may exist separately or together. Localized necrosis and calcification of the sclerotic valves may occur. Grayish patches of sclerotic tissue may be present in the mural endo-

cardium. All the lesions in the valves may extend to the cardiac muscle. Secondary changes in the heart wall are compensatory hypertrophy, especially of the ventricles, and sometimes thinning due to dilatation. In the fetus the right heart is usually involved; after birth the left is almost always the one attacked.

CHRONIC VALVULAR DISEASE

Results of Valve Lesions.—Narrowing of a valve causes increased difficulty in emptying the chamber of the heart behind it; insufficiency of a valve allows the return of blood through the valve during diastole, thus increasing the amount of blood entering the chamber beyond the normal. Either causes dilatation of the chamber, and compensatory hypertrophy of its wall must take place in order to perform the extra work thus demanded constantly, for the normal reserve force of the heart muscle can accomplish the extra task only temporarily. This hypertrophy increases the working power of the heart to above normal, but the organ is relatively less efficient than the normal heart, as its reserve force is less and sudden or unusual exertion may cause disturbance or failure of the compensation acquired by the hypertrophy. If this loss of reserve force is temporary, compensation is restored by further hypertrophy and by diminution, by rest, of the work demanded of the heart.

Aortic Insufficiency or Incompetency.—Etiology.—(*a*) Arteriosclerosis, involving the valves also, due to prolonged severe exertion, alcoholism, or syphilis; most cases in young or middle-aged men are caused by syphilis; (*b*) acute, ulcerative, or chronic endocarditis; (*c*) rupture of a segment by sudden exertion, as in piano-movers; (*d*) congenital valve deformity; (*e*) dilatation of the valve ring in atheroma and aneurysm of the aorta, a relative insufficiency.

Pathology.—The valve cusps may be sclerosed and deformed, or ulcerated or calcareous, or adherent to the intima

of the aorta. The left ventricle is greatly dilated with compensatory hypertrophy, forming the so-called *cor bovinum*. Relative mitral insufficiency due to dilatation of the left ventricle is common. Dilatation and hypertrophy of the left auricle follow, and subsequently the same changes in the right heart. The ascending aorta is often dilated; myocarditis and general arteriosclerosis are frequently associated.

Symptoms are often long absent. Headache, dizziness, faintness, flashes of light, dyspnea and palpitation on exertion, and pain in the cardiac region may occur early. Pain is dull and localized, or sharp and radiating to the neck or left arm, rarely to the right. With failure of compensation, dyspnea, worse at night, sometimes orthopnea, edema of legs and feet, cough, anemia. There may be intercurrent acute endocarditis or embolism. Great restlessness and delirium are common in the late stages. Sudden death is more common than with any other valvular lesion.

Physical Signs.—*Inspection:* Apex beat heaving; may be in sixth or seventh intercostal space as far out as anterior axillary line. Precordia may bulge. Visible jerky pulsation of superficial arteries, which are tortuous, and in suprasternal notch, also in retinal arteries. Alternate blushing and pallor beneath finger-nail, when sufficient pressure is exerted to cause blanching, or of a hyperemic line caused by pressure with a hard substance upon the forehead ("capillary pulsation"). This is sometimes present in anemia, neurasthenia, or with very low tension. *Palpation:* Forcible apex beat unless dilatation predominates, sometimes heaving of entire precordia, and occasionally systolic retraction of intercostal spaces between left border of sternum and mammary line. Diastolic thrill at the base is very rare. The pulse is known as the "Corrigan pulse" or "water-hammer pulse." It rises very suddenly to an unusual height, then collapses abruptly as blood regurgitates from the aorta into the left ventricle. This is best recognized when the patient's arm is held high,

Pulse is often retarded. It is regular in force and rhythm, unless compensation fails. When pressing down the finger-nail to obtain capillary pulsation, expansile pulsation of the finger-tip with sudden fall may be felt. *Percussion:* Increased area of cardiac dulness, especially downward and to the left. *Auscultation:* Usually a soft, blowing, long diastolic murmur, with maximum intensity at the left of the sternum at the level of the third or fourth costal cartilage, or at the second right intercostal space near the sternum, or near the apex or at the ensiform. This is sometimes heard even in the axilla and back. The aortic second sound may be present; sometimes heard only over the carotid artery. At the apex the first sound is usually loud, but may be replaced by a systolic murmur of relative mitral insufficiency. A localized presystolic thrill and rumble at the apex ("Flint murmur"), resembling that of mitral stenosis, is often present with simple aortic insufficiency. At the second right intercostal space is often a systolic murmur transmitted to the vessels of the neck, due to complicating roughening of the aortic valve or aorta. A sharp systolic murmur ("pistol-shot sound"), sometimes also a diastolic, may be heard over the large peripheral arteries. X-rays show cardiac enlargement, chiefly downward and to the left, and increased systolic excursion of its left border; often enlarged shadow of aorta.

Aortic Stenosis.—Etiology.—Chronic endocarditis; atheroma of the aorta; arteriosclerosis.

Pathology.—The aortic valve segments are usually adherent to each other by their margins, and are thickened and distorted or little altered. The valves are often insufficient also. The left ventricle is slightly dilated, but shows great concentric hypertrophy. If compensation fails there are dilatation of the left ventricle, relative mitral insufficiency; then dilatation of the left auricle, pulmonary congestion, hypertrophy and dilatation of the right ventricle.

Symptoms.—While compensation is maintained there are no subjective symptoms. Later, diminished cerebral bood

supply causes dizziness and faintness. If dilatation occurs, signs of mitral insufficiency and pulmonary congestion develop.

Physical Signs.—*Inspection:* Apex beat usually indistinct; may be invisible; sometimes looks strong and displaced downward and to the left. *Palpation:* Apex beat is a deliberate thrust of no great violence. Usually a systolic thrill at the base, maximum intensity in the aortic area. Pulse small, regular, infrequent. *Percussion:* Area of dulness not much enlarged. *Auscultation:* Rough, loud, systolic murmur; maximum intensity second right intercostal space or higher; soft if compensation fails. It is transmitted to the vessels of the neck and may be heard over the whole precordia. The aortic second sound is absent or feeble. X-rays show cardiac enlargement.

Diagnosis.—(1) From **arteriosclerotic roughening** of the valve or of the ascending aorta, or **aneurysm** of the aorta, by absence of accentuation of aortic second sound and in the last instance of other signs of aneurysm. (2) From **functional** or **hemic murmur,** which is less harsh and loud and without thrill, cardiac hypertrophy, or slow pulse. (3) From **pulmonary stenosis,** in which the murmur is not transmitted to the vessels of the neck and the pulse is not altered as in aortic stenosis. (4) From **patent ductus arteriosus,** in which the thrill and murmur are not accompanied by changes in pulse and second sound.

Mitral Insufficiency or Incompetency.—Etiology.—This, the most common valvular lesion, is caused by (1) changes in the valves and usually in the chordæ tendineæ, due to rheumatic endocarditis; (2) weakness of the papillary muscles (muscular incompetency); (3) dilatation of the ventricle, making the valve ring too large to be closed by the normal valve segments (relative insufficiency). Such dilatation occurs with aortic insufficiency; with failure of compensation in hearts with hypertrophy due to chronic nephritis, alcoholism, or hard work; with myocarditis.

Pathology.—Segments of the mitral valve may be shortened and deformed, sometimes calcified, with thickening and contraction of the chordæ tendineæ. There is often some stenosis caused by this deformity. In relative insufficiency there may be lesions of hypertrophy of the heart wall or myocarditis with dilatation. The effects of mitral insufficiency are: Regurgitation from left ventricle to left auricle, which is also receiving blood from the lungs, causing dilatation of the auricle and its hypertrophy to expel the extra blood; dilatation and then hypertrophy of left ventricle on account of the large quantity of blood forced in by the auricle; obstruction to flow of blood from pulmonary veins due to extra blood in left auricle, hence dilatation and hypertrophy of right ventricle, which forces blood through the lungs; dilatation and hypertrophy of right auricle. The pulmonary vessels become dilated and eventually brown induration results. Ventricular hypertrophy maintains compensation until increase of the valvular lesion or increased muscular exertion interrupts it.

Symptoms.—During perfect compensation, symptoms are absent. If it is slightly disturbed there are some cyanosis, dyspnea on exertion, bronchitis, hemoptysis, and eventually clubbing of the fingers. If compensation is seriously disturbed, the cyanosis may become more marked, heart action feeble and irregular, dyspnea constant, with cough and watery or blood-stained sputum, dropsy beginning in the feet and extending upward and involving the serous cavities. The liver is enlarged and gastro-intestinal disturbance results from portal congestion. Urine scanty, with albumin, casts, and sometimes red cells. After repeated attacks, death usually occurs from asystole due to extreme dilatation or from pulmonary edema; rarely sudden.

Physical Signs.—(*a*) *Before compensation is established:* Systolic murmur loudest at the apex, often transmitted to axilla and back. (*b*) *Compensation established—Inspection:* Apex beat displaced to the left and downward. In children

the precordia may bulge. *Palpation:* Impulse forcible; systolic thrill at the apex is rare. Pulse normal or slightly irregular and small. *Percussion:* Increased area of cardiac dulness, especially to the right of the sternum and to the left. *Auscultation:* Blowing or musical systolic murmur, which may replace the first sound; maximum intensity at the apex, usually transmitted to the axilla, often to the angle of the scapula, often heard over the entire precordia or chest. Pulmonic second sound accentuated. X-rays show transverse increase of heart shadow. (*c*) *Failing compensation:* Impulse wavy and feeble, pulse weak and irregular, increased cyanosis, crackling rales at the base of the lungs, dyspnea, sometimes signs of relative tricuspid insufficiency. The **chief signs of mitral insufficiency** are lateral enlargement of the heart; systolic murmur at the apex, often transmitted to the lower part of the precordia, back, and axilla; accentuation of pulmonic second sound.

Diagnosis.—(1) From **functional murmurs**, which are not usually transmitted to the left and are not accompanied by hypertrophy and by accentuation of the pulmonic second sound. (2) From **aortic systolic murmur**, which may be loudest at the apex, by accentuation of pulmonic second sound, transmission to the left, and signs of venous congestion. (3) From **tricuspid insufficiency**, in which the maximum intensity is nearer the sternum, the pitch may be different, and pulsation of cervical veins occurs. (4) The existence of actual rather than relative insufficiency may be shown by the coexistence of mitral stenosis.

Mitral Stenosis.—Etiology.—Most common in young persons, chiefly females. Usually due to endocarditis with rheumatism or chorea; possibly other infections.

Pathology.—Narrowing of the valve lumen may be due to thickening or calcification of the valve segments, adhesion of their edges, thickening and contraction of the chordæ tendineæ or of the valve ring. Such a valve is usually insufficient also. Owing to the increased resistance to the passage

of blood through the stenosed mitral valve the left auricle becomes greatly dilated and hypertrophied. The obstruction then affects the pulmonary vessels and the right ventricle becomes dilated and hypertrophied, and eventually the tricuspid valve relatively insufficient. The left ventricle is not enlarged unless the mitral is also insufficient. Antemortem thrombi are sometimes found in the left auricle, frequently white thrombi in its auricular appendix. The lungs are greatly congested.

Symptoms.—Similar to mitral insufficiency, but develop more slowly, and those of venous congestion of the lungs, liver, etc., are more marked, hemoptysis is more common. Pulmonary inflammations seriously interfere with the right heart, and even slight bronchitis causes dyspnea and some cyanosis.

Physical signs vary greatly from time to time. (1) *In the early stage:* Presystolic thrill and rough, crescendo murmur localized at the apex, with accentuated first sound, long diastolic pause, and irregularity in force and rhythm of heart beat. These signs are more marked after exertion. (2) *With compensation established—Inspection:* In children there may be bulging of the lower sternal region and lower left costal cartilages. In this region is usually seen and felt the most marked impulse, though the apex beat is displaced slightly to the left. Pulsation may be seen and felt in third and fourth left intercostal spaces near the sternum. *Palpation:* Usually a short, grating presystolic thrill, terminating in a sharp shock localized in the fourth or fifth intercostal space just internal to the apex beat. Pulse irregular in force and frequency, small as compared with forcible heart action. *Percussion:* Cardiac dulness increased to the right of the sternum, but less than in mitral insufficiency, and somewhat to the left. *Auscultation:* Long, rough, purring or rumbling murmur, ending with a loud, snappy first sound, and localized just internal to the apex beat. Second sound is accentuated at the pulmonic area, diminished at the aortic,

and often absent at the apex. Often a systolic murmur at the apex, transmitted to the left, due to associated mitral insufficiency. X-rays show area of cardiac dulness increased transversely. (3) *With failure of compensation:* Heart action becomes more irregular; pulse beats often fail to reach the wrist; presystolic murmur, less often the thrill, is lost, but the sharp, snappy first sound at the apex and accentuated pulmonic second sound persist, with the lateral enlargement of area of dulness. Signs of tricuspid insufficiency may develop (see below). The **chief signs of mitral stenosis** are transverse increase of dulness, localized presystolic thrill and murmur and snappy first sound at the apex, accentuated pulmonic second sound, and irregularity of pulse.

Diagnosis.—By the above chief signs even in the absence of thrill and murmur, or these may be brought out by exertion. (1) From **Flint murmur** in aortic insufficiency, by absence of signs of that lesion and predominance of signs of pulmonary congestion (in cases where mitral insufficiency can be excluded). Great irregularity of pulse suggests mitral stenosis. (2) Of cases with paralysis of left vocal cord due to pressure upon recurrent laryngeal nerve by large left auricle, from **aneurysm of the arch of the aorta**, by absence of other signs of aneurysm. (3) From **tricuspid stenosis**, rare, usually congenital; presystolic murmur and thrill generally nearer the sternum than that of mitral stenosis, which is often associated.

Tricuspid Insufficiency.—Etiology.—Rarely endocarditis of the valve; usually relative insufficiency due to dilatation of right ventricle in mitral disease or with pulmonary emphysema or to other obstruction to the pulmonary circulation.

Symptoms are masked by those of the preceding valvular lesion, but those of visceral congestion and anasarca are marked.

Physical Signs.—Transmitted systolic pulsation from the right ventricle through the tricuspid and right auricle, visible in the jugular veins and causing expansile pulsation of the

enlarged liver; increased area of dulness and *x*-ray heart shadow, especially to right of sternum; soft murmur in lower sternal region, maximum intensity usually just to left of sternum, may be heard as far to the right as the anterior axillary line. True jugular pulsation is distinguished by immediate refilling from below and pulsation, when the vessel is emptied by stroking from below upward. The distention is greatly increased by coughing. Sphygmographic tracing shows the "positive" or "ventricular" jugular pulse. The murmur may be distinguished from a coexisting mitral systolic by one area of maximum intensity at the apex, and another in the lower sternal region with difference of pitch.

Tricuspid Stenosis.—Rare, except in congenital cases, and usually associated with lesions of valves of the left heart. A presystolic murmur and sometimes thrill may be present at the base of the ensiform or at the right of it. This is to be distinguished from the mitral presystolic usually present by difference in pitch, quality, or position. Cardiac dulness and *x*-ray shadow are enlarged, especially to the right. Cyanosis is often very marked and anasarca finally develops. Prognosis is very bad.

Pulmonary Insufficiency.—Very rare, usually congenital, sometimes caused by endocarditis. There is a diastolic murmur, loudest at the second left intercostal space, not transmitted to the apex or great vessels. Pulmonic second sound accentuated. Temporary pulmonary regurgitation may result from high pressure in the pulmonary artery, *e. g.*, in mitral stenosis (Graham-Steell murmur).

Pulmonary Stenosis.—Usually congenital and associated with compensating patency of the foramen ovale or incomplete ventricular septum, sometimes tricuspid stenosis. Cyanosis and dyspnea are present almost from birth. The heart is hypertrophied, especially the right ventricle, increasing the cardiac dulness and *x*-ray shadow to the right. There may be a systolic murmur with thrill at the second left intercostal space. It is not transmitted, as in aortic stenosis,

to the great vessels. Pulmonic second sound is feeble or absent.

Functional Pulmonary Murmurs.—A purely functional, soft, blowing systolic murmur is frequently heard, especially when recumbent, in anemia, febrile conditions, after exertion, and in children and less often healthy adults with thin-walled chests. Its only significance is that it suggests the advisability of blood examination for anemia.

Prognosis of valvular disease depends upon the degree of compensation maintained, and is best when this is acquired spontaneously. This is to be judged by the heart action. In children prognosis is poor, in women usually better than in men.

Treatment.—(*a*) *While compensated:* Quiet life, avoiding excitement, worry, and overexertion, especially with aortic lesions. Moderate exercise, such as does not disturb the cardiac rhythm or cause dyspnea. Avoid high altitudes. Moderate diet. No cardiac stimulants or tobacco. (*b*) *With disturbed compensation:* Rest, as complete as possible. This may be the only treatment necessary. If venous congestion is very marked, phlebotomy, or saline purgation. *Medicinal:* Tinct. digitalis, ♏v to xv, or infus. digitalis, ʒj to ʒss, every three hours to t. i. d., beginning with the larger dose at short intervals, reducing dose and decreasing frequency as heart action improves. It is often necessary to continue small doses indefinitely. Contra-indications are perfect compensation, and nausea, vomiting, onset of irregular pulse (partial heart block), or reduction of urine while taking. Tinct. strophanthi, ♏ij to v, is often a good substitute. In aortic cases with positive Wassermann of blood or spinal fluid give mercury and potassium iodide. If anemic, iron and arsenic are indicated. *Symptomatic:* For edema, digitalis as above; hydragogue cathartics; diuretics, such as calomel, gr. $\frac{1}{10}$, every half-hour, for ten doses; calomel, digitalis, and squill, āā gr. j, every four hours, for four doses; or potass. bitartrate or sodiosalicylate

of theobromine. If edema of the lower extremities becomes extreme, light bandaging with flannel; scarification with aseptic precautions, if necessary. For dyspnea, support in sitting position in bed; aspiration, if hydrothorax exists. If dyspnea is due to cardiac failure, cardiac stimulants should be pushed. If arterial tension is high, nitroglycerin may be given frequently and in increasing doses. Morphine, gr. $\frac{1}{8}$ to $\frac{1}{4}$, or codeine, gr. $\frac{1}{4}$, is most valuable, especially if there is great restlessness. Associated emphysema and bronchitis may cause dyspnea and require special treatment. For pulmonary edema, dry cupping. For palpitation and cardiac pain, ice-bag over heart. Tinct. aconite, ♏iij to x, every two or three hours, if heart continues overacting. For pain, potass. iodide, gr. x, t. i. d., or nitroglycerin. Relieve gastric or intestinal distention if present. For gastric disturbance, stop medication and food by mouth if necessary, giving cracked ice, milk with lime-water, etc., and cerium oxalate, hydrocyanic acid, etc., as in chronic gastritis. Venesection or purgation, if necessary to relieve portal congestion. For cough, treat the heart condition; heroin, gr. $\frac{1}{20}$ to $\frac{1}{12}$, or codeine, gr. $\frac{1}{4}$, p. r. n. For insomnia, bromides with camphor, or compound spirit of ether; paraldehyde or amylene hydrate by mouth or rectum. If these fail, morphine. For diminished urine, drugs mentioned for edema (see above).

HYPERTROPHY OF THE HEART

This may be *simple hypertrophy*, the form here described, in which the heart walls are thickened without change in size of the cavities, or *hypertrophy with dilatation.* (See Dilatation of the Heart, p. 268.) The auricles do not show simple hypertrophy.

Etiology.—Increased resistance, local or general, to the blood flow through the heart; overaction. (*a*) *Hypertrophy of left ventricle or of the organ as a whole:* Prolonged muscular exertion; cardiac disease, such as aortic stenosis or insuffi-

ciency, mitral insufficiency, pericardial adhesions, overaction due to cardiac neurosis, exophthalmic goiter, or use of alcohol, tobacco, tea, coffee; arterial changes, such as arteriosclerosis, with or without nephritis, conditions associated with high tension, congenital stenosis of the aorta.

(*b*) *Hypertrophy of right ventricle:* Mitral stenosis or insufficiency, later stage of aortic lesions, lesions of valves of right heart, pericardial adhesions; obstruction to circulation in the lungs, as in emphysema and interstitial pneumonia.

Pathology.—Heart may be greatly increased in size, the apex broadened, and in cases of hypertrophy of the right ventricle this forms the greater part of the apex. The thickness of the walls involved is increased, the muscle darker and denser than normal. The increase is probably due to increase in size and number of muscle fibers. Arteriosclerosis may result from the constant increased strain upon the vessels, and cerebral hemorrhage follow.

Symptoms.—None while the general condition is good. If impaired, there may be cardiac discomfort, consciousness of heart action, headache, tinnitus, flushing of the face, and visual disturbance.

Physical Signs.—*Inspection:* Precordia may bulge, especially in children. Apex beat forcible and diffuse. *Palpation:* Heaving apex beat, displaced downward and outward, occasionally as far as the eighth intercostal space three inches outside of the nipple line. Pulse regular, full, high tension. *Percussion:* Increased area of dulness, especially downward and to the left. *Auscultation:* First sound often prolonged, dull, and reduplicated; second sound accentuated and perhaps reduplicated in aortic region. X-rays show increase of cardiac shadow, chiefly downward and to the left.

If the hypertrophy is of the *right ventricle,* the cardiac dulness and enlarged *x*-ray shadow extend more to the left than downward and the pulmonic second sound is accentuated.

Diagnosis.—(1) From **nervous palpitation,** in which the apex beat is not heaving; action is rapid; area of dulness not much increased; first sound is sharp and second not accentuated. (2) From **hypertrophy with dilatation,** by stronger and more regular action and pulse, more distinct second sound, and absence of pulmonary congestion and murmurs. (3) From **pericardial effusion,** in which dulness is triangular with base downward, heart sounds are feeble, though action is frequent, pulse is rapid and feeble, and general condition is poor. (4) From other causes of increased area of dulness—retraction of left lung by adhesions, tuberculosis, pneumonia, aneurysm, mediastinal tumor, etc.—in which other cardiac physical signs are normal except as influenced by these lesions.

Prognosis.—Good, unless compensation is broken by undue exertion or mental strain, poor nutrition, or intercurrent cardiac or general disease.

Treatment.—Avoid all excesses.

DILATATION OF THE HEART

Etiology.—(1) *Increased endocardial pressure* due to overfilling of the chambers or to undue obstruction to the flow, either at the valves or in the bloodvessels. These causes are the muscular exertion and cardiac and vascular changes named as leading to hypertrophy of the heart (see Etiology, p. 266), which condition is usually also present, except in extremely acute dilatation. (2) *Weakness of heart walls* caused by myocarditis, fatty heart, or parenchymatous degeneration in acute infectious diseases, such as typhoid fever and pneumonia, or associated with endocarditis and pericarditis, or anemia, or chronic alcoholism. Both factors, muscular weakness and increased pressure, are usually present.

Pathology.—Dilatation usually involves two or more chambers, most often of the right heart, and is generally

associated with hypertrophy. If great dilatation exists, the valves are relatively insufficient, and the venous orifices are often greatly dilated. The lesions are those of the causal condition.

Symptoms.—*Of acute dilatation:* Sudden onset of dyspnea, with signs of venous circulatory obstruction and weak, diffuse impulse. Death if compensation is not established. *Of dilatation with hypertrophy:* None while compensatory hypertrophy is complete. When this is disturbed, so that a cavity fails to empty itself entirely during systole, signs of venous congestion develop—dyspnea, cough, gastro-intestinal disturbance, cerebral symptoms, from dizziness and headache to delirium and coma, dropsy of extremities and serous cavities extending gradually upward, and diminished urine. These disappear if the work of the heart is sufficiently decreased or compensatory hypertrophy occurs.

Physical Signs.—Cardiac impulse diffuse; area of dulness and *x*-ray shadow of heart enlarged, depending upon the site of dilatation. First sound resembles the second. Murmurs due to causal endocarditis or relative insufficiency may be present. There may be gallop rhythm. All variations in frequency and diminished force of the pulse may occur.

Diagnosis.—From **simple hypertrophy** and **pericardial effusion**, etc. (See Hypertrophy, Diagnosis (2) and (3), p. 268.)

Treatment.—*Of acute dilatation:* Venesection. Subsequently, and for dilatation with hypertrophy, see Valvular Disease, Treatment, p. 265.

DISEASES OF THE MYOCARDIUM

DISEASES OF THE CORONARY ARTERIES

These are embolism, thrombosis, and arteriosclerosis.

(*a*) **White Infarction or Anemic Necrosis.**—Sudden death usually follows embolism of a coronary artery, but blocking

of one branch by a thrombus or embolus leads to **white infarction** or **anemic necrosis**, as these arteries are terminal. The area involved is grayish or grayish red, often projects slightly above the surface, is of irregular outline, and usually in the left ventricle and septum. Microscopically, it shows necrosis, which may be so extensive and complete as to cause rupture or aneurysm of the heart or terminate by formation of new connective tissue or chronic myocarditis. If the embolus is septic, a local abscess forms, which may perforate the heart wall.

Symptoms.—Sudden death or the indefinite symptoms of myocardial disease. (See Chronic Interstitial Myocarditis, Symptoms, p. 271.) Death sometimes occurs later from rupture of the heart.

(*b*) **Endarteritis** of the coronary arteries leads to chronic interstitial myocarditis (see below), most often in the left ventricle and septum.

ACUTE MYOCARDITIS

This occurs with acute infectious diseases, such as typhoid, diphtheria, and scarlatina, or with endocarditis, or pericarditis (parenchymatous and interstitial forms), and as the result of septic embolism of the coronary arteries (suppurative form). Granular degeneration of the muscle cells, infiltration of the interstitial tissue with small round cells, and sometimes small abscesses of the heart wall are seen. Abscesses may point into the pericardial sac or the cavity of the heart, or become encysted, or cause aneurysm of the heart.

Symptoms are indefinite. They and the **physical signs** are in general those of slight cardiac dilatation and of sepsis, if due to septic embolism. The heart action and pulse become progressively weaker. Sudden death after slight exertion may occur.

Treatment is that of the accompanying disease, and as in valvular disease with disturbed compensation (p. 265), but avoiding overstimulation.

CHRONIC INTERSTITIAL MYOCARDITIS

Etiology.—Usually disease of the coronary arteries, sometimes endocarditis or pericarditis. It occurs generally after middle age in persons subject to the causes of arteriosclerosis, especially chronic intoxications.

Pathology.—The process is a fibrous replacement hyperplasia rather than an inflammation. The whole, or usually a limited area, of the heart wall is grayish and firm and shows microscopically connective-tissue hyperplasia, which may become very fibrous. Compensatory hypertrophy of the heart may occur, followed by aneurysm. Coronary endarteritis and various degenerations of the heart muscle may be present.

Symptoms are indefinite. They may be absent until sudden death occurs. Arrhythmia is common. Pulse may be very infrequent, feeble, and irregular; sometimes frequent. With this may be dyspnea, cardiac oppression, or attacks of angina. There may be symptoms and physical signs of hypertrophy and dilatation of the heart. Death may occur suddenly or after symptoms of gradual cardiac failure.

Diagnosis of individual myocardial affections is uncertain. Fatty heart is suggested by general obesity.

Treatment.—Moderation in everything. If there is marked cardiac insufficiency with rapid, feeble, irregular heart action, dyspnea, and edema, treat as in valvular disease with disturbed compensation. (See Treatment (*b*), p. 265.) If pulse is very slow, feeble, and irregular, with syncope, rest, cardiac stimulants, such as aromatic spirit of ammonia, p. r. n.; strychnine, gr. $\frac{1}{60}$, t. i. d. If the pulse is of high tension, small doses of nitroglycerin. If there is great restlessness with tumultuous but inefficient heart action, morphine is

often the best drug, gr. $\frac{1}{12}$ to $\frac{1}{8}$, repeated as necessary. (For attacks of angina, see Angina Pectoris, Treatment, p. 277.)

The Schott or Nauheim treatment—warm carbonated saline baths with graduated resisted exercise—is valuable in some cases.

FATTY HEART

1. **Fatty degeneration** occurs often in old age, prolonged infections, wasting diseases, anemias, alcoholism, poisoning by phosphorus and arsenic, and with diseases of the coronary arteries, pericarditis, or cardiac hypertrophy.

Pathology.—General or local yellowish fatty areas. If extensive, the heart is soft, flabby, secondarily dilated. Microscopically, many minute fat droplets in the muscle fibers. These may be seen when gross appearances are nearly normal.

2. Fatty overgrowth, or infiltration, is most common in men over forty years, often as part of a general obesity. The subpericardial fat may be greatly increased and may extend into the heart between muscle bundles. The heart is large and yellow. Secondary dilatation and sometimes fatty degeneration may occur.

Symptoms are usually absent until dilatation occurs, and then are those of that condition. The pulse may be very slow. Dyspnea, angina, syncope, mental symptoms, and Cheyne-Stokes respiration may be prominent symptoms. Sudden death may occur from syncope or cardiac rupture.

Physical signs are those of dilatation.

Treatment.—Same as chronic myocarditis. (See Treatment, p. 271.) The Oertel and Schott treatment—gradually increased hill climbing with reduction of fluids and fats in the diet—may give good results, especially in cases with general obesity. The Schott or Nauheim method—saline baths charged with carbon dioxide and graduated resisted exercise—may be employed at Nauheim or in artificial baths at home.

FUNCTIONAL AFFECTIONS OF THE HEART

1. **Palpitation.** — **Definition.** — Irregular or forcible heart action perceived by the individual.

Etiology.—Hysteria, neurasthenia, violent emotions, or sexual excesses; overuse of coffee, tea, alcohol, or tobacco; gastric distention or dilatation. The violent action in pure organic heart disease is not properly palpitation in the sense here understood, as it is not a neurosis.

Symptoms may be only a sensation of fluttering with that of distention or emptiness of the heart. There may be flushing of the skin, violent pulsation of superficial arteries, forcible apex beat, with rapid pulse, dyspnea, and nervousness. Functional murmurs at the second left intercostal space and apex are common. The attack lasts a few minutes to several hours, and may recur after exertion or excitement.

Prognosis good, though often an obstinate affection. Cardiac hypertrophy often results.

2. **Tachycardia, or Rapid Heart.—Etiology.**—While rapid heart action occurs normally in some individuals, and also with gross lesions of the medulla or pneumogastric nerves, it may be a neurosis of either constant or paroxysmal type, and has been ascribed to auricular fibrillation. Its causes are those of palpitation (see Etiology, above), especially excitement. It often occurs at the menopause.

Symptoms.—Tachycardia is itself really only a symptom. The heart action may be over 200 a minute, yet the patient be unconscious of it, or experience only a feeling of uneasiness in the cardiac region. Paroxysmal tachycardia occurs in repeated attacks, lasting a few minutes to many days. Rarely fatal.

3. **Bradycardia, Brachycardia, or Slow Heart.**—Physiological bradycardia occurs in individuals or families, in the puerperal state and after exhaustion. Pathological bradycardia is found in convalescence from acute febrile disease;

in gastro-intestinal diseases, such as chronic gastritis, cancer, ulcer, or dilatation of the stomach; in pulmonary emphysema; in myocardial or coronary artery disease; under the influence of toxic agents, such as coffee, tea, alcohol, tobacco, lead, digitalis, aconite; in diabetes, chronic nephritis, and anemia; in diseases of the brain, medulla, and cord; in neurasthenia, hysteria, and insanity; in sunstroke and diseases of the sexual organs and skin. It may be a disorder of conductivity.

4. **Arrhythmia.**—According to the myogenic theory, the cardiac rhythm is governed by impulses arising in the heart muscle itself, which possesses the functions of rhythmicity, excitability, contractility, conductivity, and tonicity. Disturbance of these functions causes various forms of arrhythmia.

(*a*) **Sinus** or **fundamental arrhythmia,** common especially in young persons, also found in neurasthenics, is a neurogenic disturbance through the vagus. There is a variation in the length of diastole, but no change in the relation of auricular and ventricular contraction. Clinically unimportant.

(*b*) **Extrasystolic Arrhythmia.**—The heart responds with its full force to a stimulus which is strong enough to cause a contraction. Immediately after a contraction all the functions of heart muscle except tonicity are exhausted—the refractory phase. Stimuli arise normally at the junction of the superior vena cava with the right auricle and pass through the bundle of His to the ventricle. In response to a stimulus arising elsewhere, a premature contraction, an "extrasystole," occurs. After this the heart is still in the refractory phase when the next physiological stimulus reaches it, so can make no response until the next one after that. The polygraphic tracing shows at each extrasystole two systoles in abnormal proximity followed by a long diastole, then resumption of normal rhythm. Several extrasystoles may occur successively, producing the bigeminal or trigeminal pulse. A weak extrasystole may not be palpable at the

wrist, causing an intermittent pulse. The extrasystole is usually a functional condition occurring in persons over middle age, in neurasthenic or debilitated persons, or as a result of toxemia of intestinal or metabolic origin, or from tea, coffee, alcohol, or tobacco. It is not of clinical importance.

(*c*) **Nodal arrhythmia, ventricular form of venous pulse, or permanently irregular pulse,** has been described as a disturbance in which the stimulus probably arises in or near the auriculoventricular node and travels toward both ends of the heart, so that ventricular systole is contemporaneous with auricular, or precedes it by a short period. It is more generally attributed to *auricular fibrillation.* It occurs in paroxysmal tachycardia, and in mitral stenosis its onset is marked by loss of the presystolic murmur. It is a persistent, continuous, and irregular rhythm in which the auricular wave disappears from all tracings. Digitalis is most efficient in this condition.

(*d*) **Pulsus alternans,** a disturbance of contractility. A regular alternation of large and small beats without other irregularity of rhythm is important as a sign of exhaustion of the heart muscle.

(*e*) **Heart block** is a serious form of irregularity. **Partial heart block** is a condition due to defective conductivity of the auriculoventricular bundle, shown by lengthening of the *a-c* interval and occasional dropping out of ventricular systoles. When conductivity is more depressed, only every second or third auricular systole initiates a ventricular systole, giving a 2 to 1 or 3 to 1 rhythm. Digitalis is strictly contra-indicated, as it can itself cause partial heart block. In **complete heart block** there is complete loss of conductivity of the auriculoventricular bundle, apparently on account of local lesions of this structure. The ventricle and auricle follow completely independent rhythms. The **Stokes-Adams syndrome** is complete heart block, a bradycardia with syncopal attacks.

Treatment of Palpitation and Arrhythmia.—Remove the underlying cause and reassure the patient. Regularity of life and moderation. For palpitation, avoid tea, coffee, alcohol, and tobacco; also food causing flatulence, and excitement. Iron, arsenic, and strychnine, the last in large doses. For arrhythmia, cure the cause. For tachycardia, rest on back, ice-bag over heart, aconite, and bromides. For bradycardia, treat the cause only; strychnine in some cases.

5. **Angina Pectoris.**—Known also as **stenocardia** or **breast pang**, is a symptom; paroxysms of intense cardiac pain, with sense of impending death.

Etiology.—Usually in adult males; it may be hereditary. A history of syphilis, gout, diabetes, or influenza is common. Arteriosclerosis, either general or of the coronary arteries near their origin, or myocardial disease, is usually present; often aortic insufficiency or adherent pericardium. *Exciting causes:* Exertion, emotion, distention of stomach, or chilling. The theories are that it is (*a*) neuralgia of cardiac nerves due to coronary endarteritis or changes in cardiac ganglia or pneumogastric or phrenic nerve; (*b*) cramp of the heart muscle; (*c*) due to sudden increase of ventricular tension from acute dilatation or to spasm of the coronary arteries; (*d*) ischemia of the heart due to coronary spasm or disease.

Symptoms.—Usually during exertion or excitement, sudden onset of agonizing pain in the heart, radiating to the neck and left arm, with sense of impending death. The patient remains motionless and silent, face usually pale or ashy, with profuse perspiration. Pulse usually of increased tension, though often regular and full. Sudden death may occur in any attack, or one may last a few seconds or minutes and end in syncope or be followed by exhaustion and sometimes passage of a large quantity of clear urine. Flatulent gastric distention is common in the attacks; asthmatic breathing may be present. There may be attacks at intervals

for years before death occurs. Sudden death may occur without warning, and not in an attack.

Diagnosis.—From **functional angina,** by occurrence in males over forty, after exertion, emotion, or exposure to cold, without nervous or other symptoms, of agonizing pain and sense of impending death for a very short time, during which immobility and silence are maintained, and pulse is of high tension.

Prognosis.—Often fatal in attack or at some other time.

Treatment.—*Of the attack:* Inhalation of amyl nitrite, gtt. iij to v, or of chloroform if this fails; morphine hypodermically if necessary. *In the interval:* Have perles of amyl nitrite always on hand. Potassium iodide or nitroglycerin in small and increasing doses if tension is increased. Treat the underlying disease if ascertainable.

6. **Functional Angina Pectoris.—Etiology.**—(*a*) *Neurotic angina:* Usually in neurotic women of any age; spontaneous, often periodical or nocturnal attacks of cardiac pain of moderate severity lasting several hours, accompanied by excitement and restlessness, often even hysterical symptoms. (*b*) *Toxic angina* follows excessive use of tobacco, tea, or coffee. The heart pains are sharp, shooting, often with palpitation, arrhythmia, pallor, sweating, nausea, and tendency to syncope.

Prognosis.—Neurotic angina is never fatal; toxic is rarely so.

Treatment.—Removal of the nervous or toxic cause, and general tonics.

CONGENITAL HEART AFFECTIONS

These are due to abnormal development or fetal endocarditis. The most common form is pulmonary stenosis (p. 264), with patency of the foramen ovale or ventricular septum. The chief **symptoms** are extreme cyanosis, appearing very early, increased by any exertion; clubbing of

fingers and toes; dyspnea and cough on exertion. The heart is enlarged, and murmurs differing from those of ordinary valvular lesions may be heard. Red blood cells may be of nearly double the normal number. These "blue children" usually die of bronchopneumonia or other disease before puberty. Some congenital defects give no symptoms.

Treatment.—General hygienic life, avoiding exposure. Venesection, if congestion becomes too severe. If compensation fails, treat as in valvular disease. (See p. 265 (*b*).)

DISEASES OF THE ARTERIES

ARTERIOSCLEROSIS

Definition.—A localized or diffuse thickening of the intima and then of the other coats of the arterial wall.

Etiology.—This probably begins as a compensatory hyperplasia of the intima to restore the normal lumen of a vessel dilated through weakening of its wall by a degenerative process, or to resist the strain accompanying high tension. The tendency to arteriosclerosis may be inherited or an individual predisposition may exist. The affection is usual after forty years of age. Muscular overexertion, worry, mental overwork, habitual overeating, chronic intoxications (especially by alcohol, lead, gout, and syphilis), and chronic interstitial nephritis are the chief causes.

Pathology.—Arteriosclerosis may be confined chiefly to the aorta, single vessels, cerebral or other groups, or be general. The veins may be similarly involved.

(*a*) *Nodular or Circumscribed Form.*—Chiefly in the aorta and larger branches are yellowish areas, usually hemispherical, flat, and projecting slightly from the intima. After atheromatous degeneration occurs these may be replaced by ulcerated patches, on which thrombi may form and which

may be the starting-point of aneurysm. Microscopically, the adventitia and media show infiltration and degeneration, then compensatory proliferation of fibrous and elastic tissue in the intima, sometimes of its endothelium. Fatty degeneration of this new tissue is common, and necrosis of the thickened area leads to atheromatous cysts, which may extend through the intima, forming ulcers. Calcification may occur in the new tissue of the intima and in the necrotic foci.

(*b*) *Senile Arteriosclerosis.*—The larger arteries are tortuous, dilated, with stiff, thin, often calcified walls. Atheromatous cysts and ulcers are present. All coats, especially the media, show degeneration. The intima often contains calcareous plates, and has thrombi on its surface. Other viscera show senile atrophy. The heart may show brown atrophy or hypertrophy.

(*c*) *Diffuse arteriosclerosis* involves the aorta and its branches, even the small vessels. They are dilated, their walls thickened; the intima may be smooth or show slightly elevated white areas, some yellowish from atheroma. The media is degenerated and necrotic, sometimes calcified. The subendothelial connective tissue is greatly proliferated, especially where the adjoining media is weakened, and the endothelium itself may proliferate. The degeneration of the media, with thickening of the intima, is most marked in small arteries. The left ventricle is hypertrophied. There are often interstitial myocarditis, chronic interstitial nephritis, and other sclerotic visceral changes.

Symptoms and Physical Signs.—If the lesion is extensive and advanced, enlarged tortuous superficial vessels may be seen and felt as thicker and harder than normal. Pulse is often of increased tension; this may be obscured by the rigidity or beading of the walls. Calcification may be detected with the *x*-rays. Physical signs of hypertrophy of the left ventricle, with accentuated aortic second sound, are present. There may be no symptoms except increased quantity of urine. Depending upon the vessels involved and the severity

of the lesion, there may be: *Renal symptoms*, those of chronic interstitial nephritis. *Cardiac symptoms* due to involvement of the coronary arteries—thrombosis with sudden death, angina pectoris, chronic myocarditis, aneurysm, or rupture of the heart. *Cerebrospinal symptoms*, headache, dizziness, insomnia, psychical disorders, aphasia, hemiplegia, monoplegia, symptoms of cerebral hemorrhage; symptoms resembling myelitis, locomotor ataxia, and various sclerotic lesions of the cord. *In the extremities*, coldness, numbness, tingling, pain, sometimes paraplegia or intermittent claudication, or thrombosis or gangrene. *Aneurysms.*

Diagnosis.—By palpable thickening of arteries, increased tension, hypertrophy of left ventricle, and accentuation of aortic second sound.

Treatment.—General regulation of mode of life, avoiding alcohol, excesses of eating, drinking, or exertion, excitement and worry, with care to maintain regular action of the bowels and kidneys. Potassium iodide, especially in syphilitic cases. For high tension, glonoin or sodium nitrite, p. r. n. For very high tension or in cases with dyspnea and other signs of cardiac insufficiency, venesection.

ANEURYSM

Aneurysm or local dilatation of an artery may be: (*a*) *True aneurysm*, whose sac is formed by one or more of the arterial coats; (*b*) *false aneurysm*, a rupture of all the coats, with the sac formed by surrounding tissue; (*c*) *dissecting aneurysm*, perforation of the intima and dissection between the coats of the wall by the blood; (*d*) *arteriovenous aneurysm*, due to communication of an artery with a vein; if a sac intervenes it is called *varicose aneurysm;* if communication is direct the vein is dilated and is called *aneurysmal varix.*

Etiology.—Syphilis and alcohol are the chief predisposing causes. The exciting causes are general weakening of the

media or of the entire wall by arteriosclerosis, with increased arterial pressure due to hard work or sudden exertion. An embolus or pyogenic bacteria may cause aneurysm.

Pathology.—True aneurysm may be fusiform or sacculated. In the fusiform all coats are distended, often irregularly; the walls are thin or thickened, sometimes degenerated or calcified. In sacculated, the entire circumference of a small part of the artery is dilated or part of one side. The entire wall is dilated, then atrophy of the media occurs, sometimes the intima is destroyed by endarteritis, or the aneurysm may follow laceration of one or more coats. The cavity contains fluid or clotted blood or lamellated fibrin along its wall. The aneurysm may erode adjacent structures by pressure, may rupture, or become stationary through deposition of fibrin on its wall.

1. **Aneurysm of the Thoracic Aorta.** — **Symptoms** vary with the size, location, and direction of growth. They are: *Pain*, severe, lancinating, often paroxysmal, most marked when erosion of bone occurs. *Cough*, due to pressure on bronchi or trachea or to bronchitis; brassy, if paralysis of the left vocal cord is caused by pressure upon the recurrent laryngeal nerve. *Dyspnea*, from compression of trachea or bronchi or of the recurrent laryngeal, which also causes hoarseness. *Hemorrhage*, due to pulmonary congestion bronchial compression, or rupture of the sac. The last may cause sudden death, though the sudden bleeding may be concealed by taking place into the esophagus or thoracic cavities. *Dysphagia*, from compression of the esophagus. *Dilatation of superficial veins*, from pressure on the great veins. *Dilatation of one pupil*, sometimes unilateral pallor or flushing and sweating of the face. Symptoms predominate in aneurysm of the transverse portion, physical signs in that of the ascending.

Physical Signs.—*Inspection:* There may be pulsation in the first and second right or left intercostal spaces near the sternum or in the suprasternal notch. Sometimes a perfo-

rating aneurysm is seen as a thin-walled tumor near the upper sternal region. *Palpation:* Apex beat displaced downward to the left; impulse heaving; diastolic shock may be felt over the sac; sometimes a systolic thrill. Tracheal tug may be obtained. Pulse may be small, delayed, or unequal in size in the radials. With large thoracic aneurysm it may not be palpable in the femorals. *Percussion:* If pressing against the chest wall, area of flatness and increased resistance over the upper sternum or at one side of it. *Auscultation:* Systolic murmur transmitted along the vessels may be heard; sometimes a diastolic murmur of aortic insufficiency, if the first portion of the arch is involved. Accentuated aortic second sound. X-rays show shadow of the dilated aorta, sometimes pulsating.

Diagnosis of Site of Aneurysm.—(*a*) *Ascending portion:* Pain, pulsation, dulness, and *x*-ray shadow in neighborhood of second right intercostal space near sternum. (*b*) *Transverse portion:* Pressure symptoms, as inequality of pupils, tracheal tug, paralysis of vocal cord, cough; *x*-ray shadow, and sometimes dulness in manubrial region. (*c*) *Descending aorta:* Pain in the back, radiating along intercostal nerves or downward; sometimes dulness and pulsation in or below left scapular region.

Differential Diagnosis.—(1) From aortic pulsation due to **aortic insufficiency** may be difficult, as the latter usually causes dilatation of the aorta, and dilatation of the aorta often leads to relative insufficiency of the valve. (2) From **pulsating empyema,** in which there are no diastolic shock and a different history, and use of a small aspirating needle, if necessary, shows pus. (3) From **mediastinal tumors,** in which pulsation is not expansile, diastolic shock is absent, aortic second sound not accentuated, tracheal tug less common, and cachexia may be present. The *x*-ray shadow may assist. (4) Dislocation of the heart in **spinal curvature** and retraction of the right lung and sometimes displacement of the heart to the right in **interstitial pneumonia** may cause pulsa-

tion, dulness, and loud aortic second sound in the upper right spaces near the sternum. Differentiate by history and other signs.

Prognosis.—Always serious. Though spontaneous cure may occur or life be prolonged for years, sudden death may occur at any time.

Treatment.—If seen early, absolute rest, restriction of fluids. In all cases avoidance of excitement and worry, stimulants, exertion and straining at stool. Potassium iodide, gr. x to xx, t. i. d. If the Wassermann reaction is positive, mercury and salvarsan should be given. Electrolysis and insertion of wire occasionally give good results with sacculated aneurysm. *Symptomatic:* For pain, potassium iodide; morphine if needed. For dyspnea with cyanosis, due to pressure on veins, venesection; tracheotomy only for bilateral abductor paralysis. For protruding aneurysm, ice-bag or elastic bandage.

2. **Aneurysm of the Abdominal Aorta.**—Usually near the celiac axis, most often fusiform. It may erode the vertebræ and cause numbness and tingling in the legs and paraplegia, also vomiting from pressure upon the stomach. The chief **symptom** is dull pain in the back, sometimes radiating to the sides. The **physical signs** may be epigastric pulsation, sometimes with visible protrusion. A tumor with expansile pulsation is always present, usually with systolic murmur, sometimes a diastolic also. It must be **differentiated** from (*a*) *simple pulsation of the aorta,* especially in neurotic subjects, by presence of tumor with expansile pulsation; from (*b*) *solid abdominal tumors,* with the pulsation transmitted and not expansile.

Prognosis is bad. Death occurs from rupture, obliteration of the lumen by clots, embolism of the superior mesenteric artery, or compression paraplegia.

Treatment.—As in thoracic aneurysm.

SECTION IX

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

ANEMIA

Definition.—Diminution of the total blood, of its red cells, or of their hemoglobin.

Primary or **essential anemia** includes chlorosis and pernicious anemia; **secondary anemia** results from hemorrhage, inanition or intoxications.

1. **Chlorosis.**—A primary anemia, chiefly of young girls, characterized by marked relative decrease of hemoglobin.

Etiology.—Usually in blondes of from twelve to twenty years, most often from fourteen to seventeen, with a family history of chlorosis or tuberculosis. Poor food, confinement in close rooms, constipation, and homesickness in girls coming from Europe are possible causes. Actual cause unknown.

Symptoms and Physical Signs.—Dyspnea, palpitation on slight exertion, tendency to syncope, headache, languor, irritability, poor or variable appetite, disturbances of digestion, constipation, coldness of hands and feet, dysmenorrhea, amenorrhea, or irregular menstruation, leucorrhea, sometimes slight fever. Pallor, often of yellowish-green tinge, though cheeks may be flushed; sometimes pigmentation of skin, especially around joints; scleræ bluish white. General nutrition good. Systolic murmur, loudest at second left intercostal space or at apex; rarely a diastolic murmur.

Venous hum (*bruit de diable*) over right jugular. Thrombosis of femoral, cerebral or other veins, with pulmonary embolism, may occur; otherwise improvement is rapid under treatment. The blood is pale. Red cells are diminished, but usually to not below 80 per cent. of the normal; hemoglobin is greatly reduced, sometimes to 35 or 40 per cent. Poikilocytosis and diminution in size of red cells are common. Slight leukocytosis, relative lymphocytosis.

Diagnosis.—By age, greenish tint of pallor, bluish scleræ, good nutrition, clinical history, and great relative diminution of hemoglobin. (1) From **secondary anemia** (tuberculosis, syphilis, malaria, chronic nephritis), by absence of other signs of these disorders and by examination of sputum, blood, or urine. (2) From **pernicious anemia**, by low color index and absence of megaloblasts.

Treatment.—Fresh air, good food, care of bowels, and rest in bed at first if symptoms are marked. Blaud's pill, j to iij, t. i. d., p. c., or other iron preparation, continued for several months.

2. **Pernicious Anemia.**—**Idiopathic** or **progressive pernicious anemia** is characterized by great diminution of red cells, with relatively high color index and presence of megaloblasts.

Etiology.—Unknown; probably some toxin, of gastro-intestinal or other origin. Similar blood changes may be associated with gastric atrophy, intestinal parasites, pregnancy and the puerperium.

Pathology.—Emaciation rare; skin shows lemon-yellow pallor. Muscles dark red; all other organs anemic and fatty. Heart is large, flabby, intensely fatty. Liver and spleen of normal size or slightly enlarged, with excess of iron pigment. Both lymph and hemolymph nodes may be dark red, and their sinuses are filled with red cells free and in large phagocytic endothelium. Bone marrow usually red; shows many nucleated red cells, especially megaloblasts. In so-called *aplastic anemia* it shows no cellular activity, but is pale and

fatty. The destruction of red cells appears to occur chiefly in the spleen, lymph nodes, and hemolymph nodes. *Blood:* Red cells average 1,500,000 and may even fall to 200,000. The color index may be normal or increased, hemoglobin averaging 30 to 35 per cent. Poikilocytosis is often marked. Megalocytes, microcytes, megaloblasts, sometimes normoblasts, and instances of polychromatophilia and granular degeneration of red cells are present. Leukocytes are actually diminished, but there is relative increase of lymphocytes, with usually some myelocytes.

Symptoms.—Gastro-intestinal symptoms—dyspepsia, nausea, vomiting, diarrhea, or constipation—may precede other manifestations or last throughout. The onset is insidious, with gradually increasing languor, pallor, dyspnea, and palpitation on exertion. Pallor of skin and mucous membranes becomes extreme, the former often having a lemon-yellow tint. Without emaciation the muscles become flabby, the ankles edematous. There are hemic murmurs; visible pulsation of arteries; full pulse, sometimes of water-hammer type; often capillary pulse. Hemorrhages may occur into the skin, mucosæ, or retina. The blood shows the changes described above. There may be slight fever. Nervous symptoms are not uncommon. Pallor and weakness become extreme, sometimes with intervals of improvement, and death usually occurs.

Diagnosis.—(1) From **chlorosis**, by low red-cell count, relatively high color index, preponderance of megaloblasts over normoblasts. (2) From **secondary anemia** due to **cancer of the stomach**, in which the blood changes may be similar, by low blood count, absence of rapid emaciation, and by gastric analysis.

Prognosis.—Usually fatal. Low red-cell count with great number of megaloblasts is unfavorable. Partial recovery of long duration may occur.

Treatment.—Bed and light nutritious diet at first; later, fresh air and light exercise. Fowler's solution of arsenic, ℳiij,

p. c., increased ♏j, every day, up to ♏xx to xxv, or sodium cacodylate, gr. ss to ij, every day or every two days, hypodermically. If toxic symptoms arise, discontinue and begin again with reduced dose. Injections of defibrinated blood or blood serum may cause temporary improvement.

3. **Secondary Anemia.—Etiology.**—(1) *Hemorrhage:* (*a*) Rapid, from rupture of aneurysm, traumatism, or erosion by an ulcer of bloodvessels; (*b*) slow, from repeated epistaxis, uterine flow, hemorrhoids, or in "bleeders." (2) Inanition, from interference with ingestion or assimilation of food, as in cancer of the esophagus or gastric diseases. (3) *Toxic causes:* Acute and chronic diseases, as typhoid, tuberculosis, rheumatism, syphilis, malaria, nephritis; chronic poisoning by lead, mercury, arsenic, and copper.

Symptoms.—Pallor, dizziness, headache, palpitation, and dyspnea on exertion; weakness, tendency to syncope, poor appetite, gastric dyspepsia, and constipation. Red cells are diminished, but less than in primary anemias, and hemoglobin is relatively more decreased. After a single large hemorrhage death may occur. In other cases the red cells are small; if severe, poikilocytosis may be marked, and some normoblasts are present. Polynuclear leukocytosis is usual. In severe cases the blood closely resembles that of pernicious anemia.

Diagnosis.—By history of cause; blood examination.

Prognosis.—Good, if cause is removed.

Treatment.—Removal of cause, rest, good food, fresh air, regulation of bowels. If necessary, iron and arsenic.

LEUKEMIA

Definition.—A disease characterized by persistent increase of leukocytes with lesions of spleen, lymph nodes, or bone marrow alone or combined. The chief types are splenomyelogenous and lymphatic.

Etiology.—Unknown. It is most common before middle age.

Pathology.—Emaciation, dropsy. Heart and veins often contain large blood clots of greenish color. In *splenomyelogenous leukemia* the spleen is greatly enlarged, capsule may be thickened; spleen at first soft, then firm on section; reddish brown, or mottled red and gray, and shows hyperplasia of connective tissue and many myelocytes. The bone marrow loses its resemblance to fat. In acute cases it may look like pus; in chronic cases it is firmer and dark brown. It contains many myelocytes, normoblasts, megaloblasts, eosinophiles, and spheroidal cells containing red cells. In *lymphatic leukemia* a single lymph node or group of nodes, most often the cervical, is enlarged, hard or soft, separate. Later other groups are involved. There is hyperplasia of lymphoid tissue and reticulum. Other lymphoid structures may be enlarged, and liver, kidneys, etc., increased in size by lymphatic infiltration.

Symptoms of either type may be acute or chronic. Invasion insidious, sometimes with gastro-intestinal disturbance or epistaxis. (*a*) **Splenomyelogenous leukemia** is the common type. The spleen generally becomes greatly enlarged, sometimes painful and tender, may occupy over one-half the abdomen, and varies in size after hemorrhage, diarrhea, or a meal. There may be pallor early or late. Nausea, vomiting, diarrhea, and dysentery are common, as is ascites. Pulse is rapid, full, soft. Fever is usual. Hemorrhages occur into the skin, retina, pleura, peritoneum, etc. Headache, dizziness, dyspnea, and syncope may result from anemia. The liver may be large. Many other symptoms occasionally occur. The *blood* shows great increase of white cells, usually 1 to 10 red, sometimes more numerous than the red, and very variable in the same case. Myelocytes form about 30 per cent. of the leukocytes; polymorphonuclear leukocytes are relatively diminished, though actually increased; lymphocytes are relatively greatly decreased in number; eosinophiles are

present in normal or increased proportion to other leukocytes; mastzellen are increased. Red cells are normal in number at first; later may be reduced to 2,000,000. Normoblasts are usually numerous, megaloblasts less so. Hemoglobin varies with the red cells, and may fall to 30 per cent. It often tends to crystallize on a slide. Blood plates often very numerous.

(*b*) **Lymphatic leukemia** is rare. Various groups of lymph nodes are enlarged, usually separate, sometimes matted together. Other lymphoid structures, such as the tonsils, may be large. The *blood* shows increase of leukocytes, but less than in the previous form. About 90 per cent. of these are small mononuclear leukocytes; in some cases large lymphocytes predominate. Eosinophiles and nucleated red cells are rare; myelocytes usually absent. Red cells are about 3,000,000; hemoglobin, about 40 per cent. Spleen is usually somewhat enlarged. Pressure symptoms may accompany enlargement of bronchial or other nodes. Acute leukemia is usually of lymphatic type in young persons and large lymphocytes predominate.

Diagnosis.—From **Hodgkin's disease** or other disease with **enlarged lymph nodes** or **spleen,** by blood examination.

Prognosis.—Recovery is rare; intervals of improvement are common. Lymphatic cases may last only six to eight weeks. The course usually is progressive for two or three years.

Treatment.—Same as pernicious anemia (see Treatment, p. 287). Splenectomy has rarely been successful. Recovery has followed use of the *x*-rays.

HODGKIN'S DISEASE

Hodgkin's disease is a progressive anemia and enlargement of lymph nodes and spleen, with secondary lymphoid growths in the liver, spleen, and other organs.

Etiology.—Usually in young persons, chiefly males. Probably an acute infection; is regarded by some as tuberculous, but histological changes in lymph nodes are different.

Pathology.—General or localized enlargement of lymph nodes. The groups most often involved are the cervical, axillary, and inguinal; also bronchial, mediastinal, and retroperitoneal. The nodes are usually soft at first; later, may be firm and form large masses enclosed in a dense capsule, which the growths may perforate. The nodes may compress or perforate adjacent structures. They are grayish white on section. There are proliferation of endothelial and reticular cells, formation of lymphoid and giant cells, great proliferation of connective-tissue stroma, and presence of many eosinophiles. Spleen is usually somewhat enlarged and often contains grayish-white lymphoid nodules. Bone marrow may be changed to lymphoid tissue. Lymphoid growths may be present in tonsils, liver, or other organs.

Symptoms.—Painless enlargement of cervical, axillary, or inguinal *lymph nodes*, followed by anemia, usually slight fever of irregular or intermittent type, and loss of strength. the nodes enlarge slowly or rapidly, forming large masses, while the growth extends to other regions. Pressure by enlarged nodes may cause swelling and pain in the extremities. In cases with involvement of deep-seated nodes the first symptoms may be those of pressure on bloodvessels, nerves, trachea, bronchi, or other structures. The *spleen* may be palpable. The *blood* is not characteristic. It is normal in the early stages; later it usually shows secondary anemia, sometimes severe; only slight leukocytosis. The course is acute or chronic, often with intervals of improvement.

Diagnosis.—(1) From **leukemia,** by blood examination. (2) From **tuberculous adenitis,** which is more common in children; often begins in the submaxillary region; and in which the nodes are often matted together even when small, often suppurate, usually do not increase rapidly in size, and are

more often limited to a single region. Excise for microscopic examination if in doubt. (3) From **syphilitic adenitis,** by the history, absence of other lesions of syphilis, and negative Wassermann reaction.

Prognosis.—Course is rapid or slow and irregular. The disease usually results fatally from asthenia or pressure.

Treatment.—Excision of nodes if small and localized. Arsenic, phosphorus, quinine, cod-liver oil, iron, strychnine, and the *x*-rays are employed. Their value is uncertain, except that of arsenic and those given as tonics. For pain, morphine.

PURPURA

This includes a group of affections characterized by hemorrhages into the skin—petechiæ or ecchymoses. Blood platelets are said to be decreased, bleeding time increased, coagulation time normal or slightly increased, with failure of the clot to retract.

1. **Symptomatic Purpura.**—(*a*) *Infectious:* Always in typhus fever, sometimes in smallpox, measles, scarlet fever, pyemia, malignant endocarditis, cerebrospinal meningitis, typhoid, etc. (*b*) *Toxic:* Snake bites, potassium iodide, quinine, copaiba, belladonna, ergot, etc., and with jaundice. (*c*) *Cachectic:* With cancer, tuberculosis, leukemia, pseudoleukemia, scurvy, senility, etc. (*d*) *Neurotic:* With hysteria, neuralgia, and some organic diseases. (*e*) *Mechanical:* Due to venous stasis or violent effort.

2. **Arthritic Purpura.**—(*a*) **Purpura Simplex.**—A mild form, usually in children, sometimes with joint pains, rarely febrile. Anemia, digestive disturbances, and purpuric spots on the legs, less often on the arms and trunk.

(*b*) **Purpura Rheumatica** (*Peliosis Rheumatica, Schönlein's Disease*).—Usually in men of twenty to forty years. There are usually pain and swelling of several joints, temperature 101° to 103°, purpuric eruption chiefly on the legs and about

affected joints, often urticaria, and digestive disturbances. Sore throat and sometimes endocarditis and pericarditis suggest a rheumatic origin.

(*c*) **Henoch's Purpura.**—Usually in children; is characterized by recurrent attacks of slight joint pains or swelling, gastro-intestinal disturbances, cutaneous lesions resembling purpura and erythema multiforme, hemorrhages from mucous membranes, splenic enlargement, sometimes fatal nephritis.

3. **Purpura Hemorrhagica** (*Morbus Maculosus of Werlhof*).—A severe form, usually in delicate girls. Cause unknown. Symptoms are: Weakness, extensive purpuric eruption, hemorrhages from mucous membranes, which may cause secondary anemia, slight fever, slow coagulability of the blood. Duration, ten to fourteen days. Death may occur within a day in cases marked by profuse cutaneous hemorrhages and prostration only. Differentiated from **scurvy,** by preceding health and absence of swelling of gums; from **malignant cases of exanthemata,** by low temperature and absence of prodromata; from **leukemia,** by blood examination and absence of glandular and splenic enlargement.

Treatment.—(*a*) *Of symptomatic purpura:* That of the cause, and fresh air, food, and tonics. (*b*) *Of purpura simplex:* Fowler's solution of arsenic in full doses. (*c*) *Of purpura rheumatica:* Sodium salicylate. (*d*) *Of symptoms:* For hemorrhages, application of suprarenal extract, calcium chloride or lactate, gr. xx, every four hours, to increase coagulability of the blood. Transfusion and subcutaneous injections of human blood serum have apparently given good results. Ergot, lead acetate, aromatic sulphuric acid, gallic acid, or oil of turpentine, ♏x to xv, t. i. d. or every four hours, may be tried. After the attack, iron, arsenic, food, and fresh air.

HEMOPHILIA

An hereditary disorder characterized by tendency to persistent bleeding spontaneously or after even slight injury.

Etiology.—Usually hereditary through many generations; transmitted through daughters, themselves usually not "bleeders," to their male children. Most often in Anglo-German races.

Pathology.—Vessel walls thin; skin delicate. Though increase of red cells and decrease of leukocytes and blood plates have been observed, no constant blood changes occur. Coagulation is usually retarded.

Symptoms.—Bleeding spontaneously or after slight traumatism; extremely obstinate. The hemorrhages occur from the skin or mucous membranes or from wounds, rarely during menstruation or parturition. They vary from petechiæ or ecchymoses to bleeding which may terminate fatally or in recovery, often with marked anemia. There may be pain and swelling of the joints with extravasation of blood or synovitis. This may leave deformities resembling those of arthritis deformans.

Diagnosis.—By hereditary history and tendency to repeated obstinate hemorrhages from different locations, sometimes with articular symptoms.

Prognosis is worse the earlier the disease shows itself. The patients may live to old age.

Treatment.—Avoid all traumatism and operations in bleeding families. Marriage of the women should be discouraged. For bleeding, rest, compression, thyroid substance, gr. v, t. i. d.; ice, tannic or gallic acid, or adrenalin locally if bleeding point is accessible. For epistaxis resisting these, plug the nostrils anteriorly and posteriorly. Normal human blood serum subcutaneously may be tried.

DISEASES OF THE SUPRARENAL BODIES

ADDISON'S DISEASE

Definition.—A constitutional disease characterized by asthenia, gastro-intestinal symptoms, cardiac weakness, and pigmentation of the skin.

Etiology.—Usually in men of twenty to forty years. Whether due to disease of the adrenals or of the abdominal sympathetic nerves, or both, is uncertain.

Pathology.—Skin, mucous and sometimes serous membranes pigmented. Adrenals are tuberculous, atrophied, atrophic with sclerosis, or the seat of malignant disease or hemorrhagic extravasation. These lesions may, however, be present without symptoms of Addison's disease. Alone or with adrenal lesions the semilunar ganglia or solar plexus may show sclerosis, degeneration, and pigmentation. There may be fatty degeneration of the heart, and hyperplasia of intestinal lymph nodules and spleen.

Symptoms.—Gradual onset of weakness, variable gastro-intestinal symptoms, and pigmentation. Great fatigue and feeble, irregular heart action; nausea and vomiting, often with anorexia and diarrhea. Late in the disease the abdomen may be painful and retracted. The pigmentation varies from light yellow to dark brown or olive. It usually begins on the face and hands, and is most marked on the exposed portions of the skin and on regions naturally pigmented or where pressure is exerted by clothing. Pigmentation of mucous membranes occurs. Blood-pressure is low, 70 to 80. Death may occur with syncope, asthenia, convullions, or delirium, or through tuberculous lesions elsewhere.

Diagnosis.—By presence of asthenia, pigmentation, and gastric irritability, and by exclusion of normal pigmentation and of its increase due to pregnancy, valvular disease, arteriosclerosis, passive congestion of the liver, hypertrophic cir-

rhosis, cancer of the pancreas, diabetes bronzé, tuberculous peritonitis, exophthalmic goiter, melanosarcoma, scleroderma, pediculosis, argyria, and prolonged use of arsenic. As tuberculosis is the most common lesion of the adrenals, the tuberculin test, in the absence of ascertainable tuberculous lesions elsewhere, is of suggestive value.

Prognosis.—Usually death within one year, though this may occur in a few weeks to ten years, after intervals of improvement.

Treatment.—Suprarenal gland, gr. xv, or its extract, gr. j, t. i. d., apparently cures some cases, and has no effect in others. Bed if asthenic; iron, arsenic, and strychnine as tonics and for anemia. For vomiting, ice, cerium oxalate, dilute hydrocyanic acid, champagne, or creosote. For diarrhea, salol and bismuth.

DISEASES OF THE SPLEEN

MOVABLE OR WANDERING SPLEEN

Usually in women with enteroptosis. Its lower border may reach the pelvic brim. It may cause no **symptoms**, or pain and dragging sensation referred to the splenic region and back. Torsion of the pedicle may cause pain, fever, and splenic enlargement or necrosis. The splenic tumor is recognizable by its shape and sharp, notched edge with change in or absence of normal splenic dulness. Unless adherent, it should be replaced and held up by a suitable belt. Surgical measures may be necessary.

RUPTURE OF THE SPLEEN

Rare. May occur spontaneously or from traumatism in cases of typhoid or malaria.

Symptoms are severe pain and signs of internal hemorrhage and collapse.

Treatment.—Surgical.

ACUTE SPLENITIS

Occurs in acute infections, after traumatism, and by extension.

Symptoms are pain, tenderness and enlargement of the spleen.

Treatment is that of the cause and for relief of pain.

CHRONIC SPLENITIS

Hypertrophy with induration and pigmentation; usually due to malaria, syphilis, or leukemia, or surrounds infarcts, abscesses, or foreign bodies.

Symptoms are a feeling of weight and symptoms of pressure on lungs or bowel.

Treatment is that of the causal disease; sometimes splenectomy.

INFARCTION AND ABSCESS OF THE SPLEEN

Infarction results from simple or septic embolism in endocarditis, septicemia, or typhoid; abscess from septic embolism or extension from perforated gastric ulcer.

Symptoms are pain, tenderness and swelling of spleen, with irregular fever; sometimes general peritonitis from rupture of an abscess.

Treatment.—Surgical.

SPLENIC ANEMIA (BANTI'S DISEASE)

A disease of unknown cause, characterized by great splenic enlargement with secondary anemia. The spleen is enlarged, smooth, firm; its capsule is adherent. Some find, chiefly in adults, sclerosis and atrophy of the Malpighian corpuscles; others describe, in children, endothelial hyperplasia in spleen, liver, and lymph nodes. The red cells average 3,000,000; hemoglobin is relatively lower; leukocytes normal or diminished, otherwise normal. There may be ascites or gastric hemorrhages, rarely slight jaundice. **Course** is that of progressive asthenia; duration six months to five or more years. It must be **differentiated** from splenic leukemia, Hodgkin's disease, chronic malaria, rickets, and alcoholic, syphilitic, or hypertrophic cirrhosis with splenic enlargement.

Treatment.—Splenectomy.

DISEASES OF THE THYROID GLAND

THYROIDITIS

Acute inflammation of the gland, simple or suppurative. It may develop in a patient with goiter, or after acute infectious diseases, or from metastasis or traumatism. The gland is enlarged and soft and may contain abscesses.

Symptoms are pain, tenderness, and enlargement of part or all of the gland. Fever may be present even in cases without signs of suppuration. If the enlargement is great there may be symptoms of compression of vessels, nerves, or trachea.

Treatment.—Symptomatic; surgical if suppurative.

GOITER (BRONCHOCELE)

Etiology.—Chronic enlargement of the thyroid is sporadic, and endemic in certain mountainous regions. Young women are most often affected. Excess of lime in drinking-water may be the cause.

Pathology.—The lesions may be (*a*) *parenchymatous*, a general hyperplasia with distention of follicles with colloid material; (*b*) *vascular*, a dilatation of bloodvessels without glandular hyperplasia; (*c*) *cystic*, the gland containing large cysts filled with colloid or other material; (*d*) *interstitial*, with connective-tissue proliferation.

Symptoms.—Gradual painless enlargement of the gland or of one lobe or the isthmus. The tumor rises with the larynx during deglutition. Usually no other symptoms unless large enough to compress the trachea, vessels, or nerves.

Diagnosis.—From **exophthalmic goiter,** by absence of exophthalmos, tachycardia, tremor, and nervousness.

Prognosis.—Usually good. Sudden death may occur.

Treatment.—In the vascular form, belladonna and ergot; in the interstitial, potassium iodide; arsenic and electrolysis may aid in early cases. Thymus and thyroid extracts and ointments of iodine or mercuric biniodide are used.

EXOPHTHALMIC GOITER

Called also **Parry's, Graves', or Basedow's disease.** It is characterized by exophthalmos, goiter, tachycardia, tremor, and nervousness.

Etiology.—Most common in women of from twenty to thirty years. Several cases may occur in a family. Though no exciting cause is observed in many cases, in many others worry, grief, or excitement precedes. The theories are that

it is (*a*) a pure neurosis; (*b*) due to lesions in the medulla or sympathetic nervous system; (*c*) disease of the thyroid with superactivity, as the symptoms resemble those caused by an overdose of thyroid substance.

Pathology.—Hyperplasia, proliferation of new tubular spaces with absorption of colloid material and its replacement by a mucinous fluid. Lesions in the medulla and sympathetic nerves have been observed. The thymus usually persists.

Symptoms.—(*a*) *Acute Cases.*—Sudden onset, vomiting, diarrhea, rapid heart action with throbbing arteries, exophthalmos, enlargement of the thyroid, sometimes delirium. Death may occur in three or four days.

(*b*) *Chronic Cases.*—Usually gradual onset of *tachycardia,* pulse 100 to 180 or more if excited, regular. Later, physical signs of cardiac hypertrophy may develop. Throbbing of arteries and of the thyroid; capillary pulsation. Fine involuntary *tremor,* about eight to the second, appears early. Bilateral or unilateral *exophthalmos* of variable degree, sometimes extreme. When the eyeball is turned downward the eyelid is said to lag behind (Graefe's sign). The palpebral opening is wider than normal (Stellwag's sign). Winking is infrequent. Moderate general or unilateral *enlargement of thyroid,* variable, with pulsation, thrill, and systolic or double murmur or bruit de diable. There are usually slight fever, anemia, emaciation, weakness, nervousness, hot and cold feelings, perspiration, dyspnea, sometimes melancholia, acute mania, or pigmentation of the skin or leukoderma.

Prognosis.—Usually lasts several years. Spontaneous recovery in six months to a year is not uncommon. Death may occur in a few days in acute cases. Recovery is rare in advanced cases.

Treatment.—Prolonged rest in bed, ice-bag constantly over heart or over lower part of neck and manubrium. Avoid all excitement or worry. Galvanism with the anode over

the cervical spine and kathode over peripheral nerves may aid. Drugs are uncertain. Belladonna, up to the physiological limit, ergot, small doses of opium, digitalis or strophanthus to slow the heart, bromides, and aconite may be of value. Serum therapy is of very doubtful value. *Surgical:* Partial thyroidectomy, ligation of thyroid arteries, or division of cervical sympathetic nerves may give good results, but death during anesthesia is common.

MYXEDEMA

Definition.—A constitutional disease due to atrophy of the thyroid gland and characterized by myxedematous condition of the subcutaneous tissue, atrophy of the thyroid, and mental failure. Three forms exist—cretinism, myxedema, and operative myxedema.

Etiology.—*Cretinism,* congenital or developing before puberty, is due to absence or loss of function of the gland. Sporadic cretinism may follow an acute infectious disease or be congenital. The cause of endemic cretinism is unknown. *Myxedema* may be hereditary, and is most common in women.

Symptoms.—(*a*) *Cretinism.*—Slow mental and physical development. Facial expression heavy, tongue looks large and protrudes, hair thin, skin dry, face pale and puffy, nose flat, dentition delayed, fontanelles open. Supraclavicular pads of fat. Abdomen large, legs short and thick, hands and feet poorly developed. Muscles too weak for sitting or standing. Imbecility or idiocy.

(*b*) *Myxedema.*—Infiltration of the skin, causing loss of lines of facial expression, skin dry and harsh, much thickened, especially in supraclavicular region. Face broad, features coarse, nose broad and thick, mouth large, lips thick, hair scanty and coarse. Slowness of motion and thought, weak memory, irritability, headache, suspicious-

ness, followed sometimes by hallucinations, delusions, and dementia. Temperature normal or subnormal. There may be albuminuria. The disease may progress slowly for ten to fifteen years or death occur early. It usually results from intercurrent disease, especially tuberculosis. Myxedema may follow or be associated with exophthalmic goiter.

(*c*) *Operative Myxedema* (*Cachexia Strumipriva*).—Rarely develops except after total extirpation of the gland, and then only if no supernumerary glands are present. Symptoms same as above.

Diagnosis.—From **chronic nephritis**, by infiltration of skin not pitting on pressure, dryness of skin, coarseness of hair, and mental dulness.

Treatment.—Equable, warm climate. Thyroid extract or powdered gland, gr. j to v, t. i. d. After recovery occasional small doses may be necessary for some time, or in cretinism, for life.

SECTION X

DISEASES OF THE KIDNEY

MOVABLE KIDNEY

Called also **floating kidney, palpable kidney,** and **nephroptosis.**

Etiology.—Usually acquired. More common in women, possibly due to lacing and abdominal relaxation from pregnancies. Congenital relaxation of peritoneal or vascular attachments, emaciation, trauma, and lifting weights may be causes. Often associated with enteroptosis.

Symptoms are often absent. There may be pain or dragging sensation in the lumbar region, or intercostal neuralgia. Neurasthenia and hysteria, with nervous dyspepsia and constipation, are common reflex manifestations. The kidney can be felt by bimanual palpation in the lumbar region and over the hypochondrium, to descend with deep inspiration, so that its lower end is palpable or even so that the entire organ passes below the abdominal hand and may be so held. Firm pressure causes only slight dull pain. *Dietl's crises* are attacks of severe abdominal pain with chill, fever, nausea, vomiting, and collapse, probably due to compression or kinking of the renal vessels or ureter or to excessive eating and drinking. The kidney becomes enlarged and tender; the urine contains excess of uric acid or oxalates, sometimes blood, pus, or albumin. Permanent or inter-

mittent hydronephrosis may result from ureteral compression.

Diagnosis.—From tumors of gall-bladder, intestine, omentum or ovary, chiefly by shape.

Treatment.—Unless symptoms are present do not mention it to the patient, as neurasthenia is a common sequel of knowledge of its presence. If symptoms exist, replace kidney while recumbent and retain by suitable belt. Treat neurasthenia. With severe symptoms, nephrorrhaphy may be necessary. For crises morphine may be needed. If due to excess of uric acid or oxalates, regulate diet.

HYPEREMIA OF THE KIDNEY

1. **Acute hyperemia,** or **congestion,** occurs in beginning acute nephritis, in acute infectious diseases, after ingestion of turpentine, copaiba, cantharides, carbolic acid, potassium chlorate, alcohol, etc., and after removal of one kidney. The kidney is enlarged, dark red, with tense capsule, and bleeds freely when cut. Urine is scanty, of increased specific gravity, and contains albumin and few casts.

Treatment is removal of the cause if possible, rest in bed, milk diet; if very marked, hot pack, dry cupping over the kidneys, or large hot rectal irrigations with normal salt solution.

2. **Chronic hyperemia,** or **congestion,** is caused by (*a*) obstruction to flow of blood through the inferior vena cava, as in valvular disease, myocarditis, adherent pericardium, pulmonary emphysema, etc.; (*b*) cirrhosis of the liver; (*c*) pressure on renal veins by tumors, gravid uterus, or ascitic fluid. Kidney is enlarged, dark red, firm, smooth, capsule not adherent. Epithelium of convoluted tubules is swollen, flattened, or degenerated; interstitial tissue may be increased. Urine is diminished, of increased specific gravity, with albumin and casts, sometimes blood.

Treatment is that of the cause, fluid diet, sometimes hot packs or dry cupping over kidneys. If due to pregnancy, abortion may be necessary.

ANOMALIES OF URINARY SECRETION

1. **Anuria.**—Results from congenital absence of kidneys, acute nephritis, obstruction of both ureters by calculi, acute infectious diseases, acute poisoning by drugs mentioned as causes of acute hyperemia (p. 304), injuries, operations, catheterization, removal of single kidney, and hysteria.

Symptoms may be absent; convulsions and other uremic symptoms are rare. Even with complete suppression, death may not occur for from ten to fourteen days.

Treatment is surgical if due to mechanical obstruction. If from congestion, hot packs or hot-air baths, large hot rectal irrigations with normal salt solution, dry cupping over kidneys, purgation.

2. **Hematuria.**—Blood is found in the urine in (*a*) diseases causing chronic hyperemia of the kidney (p. 304), diseases with serious changes in the blood, as pernicious anemia, leukemia, scurvy, malaria, etc.; (*b*) renal affections, as acute congestion and nephritis, infarction, tuberculosis, calculus, pyelitis, tumor, or traumatism; (*c*) traumatism, calculus, malignant, tuberculous, or other disease or parasites (*Filaria sanguinis hominis* and *Bilharzia*) of the urinary passages. Urine is smoky to black or bright red and the microscope shows red cells. Albumin and hemoglobin are found by chemical tests. The site of bleeding is determined by history, symptoms, and physical examination, sometimes including cystoscopy and catheterization of the ureters, and by examination or urine for blood casts, clots, and moulds of ureter or urethra.

Treatment depends upon the source of the blood. If from the kidney, rest, opium, lead acetate, gallic acid, or ergot, or cold applications to the loins.

3. **Hemoglobinuria.**—Blood pigment may be present in the urine, usually as methemoglobin. (*a*) *Toxic hemoglobinuria* is caused by poisons which rapidly dissolve blood cells, as potassium chlorate, carbolic acid, pyrogallic acid, etc.; or toxins of malarial, yellow, scarlet, and typhoid fevers and syphilis; or severe blood diseases, such as pernicious anemia, scurvy, etc.; or occurs as epidemic hemoglobinuria in the newborn. (*b*) *Paroxysmal hemoglobinuria* occurs usually in adult males after exposure or exertion, sometimes with Raynaud's disease. After chills and fever, or with subnormal temperature, there is sudden onset of hemoglobinuria with vomiting, diarrhea, and lumbar pain, sometimes jaundice. Several paroxysms may occur in a day, with clear urine in the intervals. Rarely fatal.

Treatment.—Rest, dry cups over kidneys, hot drinks, amyl nitrite. If there is a malarial history, give quinine; if syphilitic, potassium iodide.

4. **Albuminuria.**—(*a*) *Functional*, occurs without evident renal lesions, as after ingestion of much albumin in food. Cyclic albuminuria is a form in which albumin appears periodically. (*b*) *Febrile*, is probably associated with such changes as cloudy swelling during fevers. (*c*) *Hemic*, occurs with such profound blood changes as anemia, scurvy, purpura, poisoning by lead, mercury, turpentine, or carbolic acid, irritant diuretics, and anesthesia with ether or chloroform. (*d*) *Neurotic*, with epilepsy, convulsions, apoplexy, tetanus, sometimes exophthalmic goiter or cerebral injuries. (*e*) *With kidney lesions*, congestion or organic disease. (*f*) *With suppuration* in any part of the urinary tract. The nitric acid ring test and that with heat and acetic acid are most employed in detecting albumin.

Prognosis depends upon the cause, and it is often determined by association of increased tension, thick arteries, altered specific gravity, and casts. Presence of a large amount, especially with casts, is unfavorable, but small

quantities, even with casts, may be present, especially in persons over fifty years, without special significance.

5. **Other Anomalies of Secretion.**—**Pyuria,** or pus in the urine, occurs with suppurative processes in any part of the genito-urinary tract, and is detected microscopically. **Chyluria,** milky urine, due to presence of granules or fat droplets, may occur independently of the Filaria sanguinis. **Lithuria,** or excess of uric acid, occurs periodically in gout or lithemia. A deposit of uric acid crystals or brick-dust sediment of urates after cooling proves only the absence of conditions favoring solution in the urine. **Oxaluria,** shown by excessive number of calcium oxalate crystals, usually follows ingestion of rhubarb, tomatoes, apples, etc., or is associated with indigestion. Nervous symptoms, such as irritability and lassitude and neurasthenia, may be present. **Phosphaturia** occurs in wasting diseases, diseases of the nervous system, those causing much bone destruction, in dyspepsia, after excessive ingestion of phosphates, in cystitis, and in so-called phosphatic diabetes. **Indicanuria** occurs in wasting diseases, with excessive intestinal decomposition of proteids, and in intestinal obstruction.

UREMIA

A toxemia occurring in acute or chronic nephritis or with anuria. The cause is unknown, but it is usually supposed to be retention of one or more nitrogenous excrementitious bodies, either an excess of normal products or abnormal. The importance of urea, uric acid, and urates in this connection has probably been greatly overestimated. Uremia is acute or chronic.

Symptoms.—1. **Acute Uremia.**—Onset sudden or gradual. Headache severe, usually occipital and extending to the neck; persistent vomiting with nausea and diarrhea, which is sometimes due to severe colitis; dyspnea, which is constant

or paroxysmal (renal asthma), worse at night, when it may resemble asthma; often Cheyne-Stokes respiration; fever, if persistent, is usually slight until just before death. The arteries are often contracted; the urine is that of the causal condition, often acute nephritis. Either with or without previous twitching of facial or other muscles, epileptiform general convulsions may occur. These may occur frequently, the patient usually becoming comatose before the attack and remaining so. Hemiplegia or monoplegia may develop before or during convulsions, with no evident cerebral lesion save edema; sometimes sudden temporary blindness. Noisy delirium or suicidal mania may be present. Coma may develop either with or without convulsions or delirium. This is usually soon followed by death, sometimes by chronic uremia or recovery.

2. **Chronic Uremia.**—Develops most often in cases of arteriosclerosis or chronic interstitial nephritis. Symptoms are similar to those of acute uremia, less severe, and of gradual onset, sometimes with acute exacerbations. Headache and dyspnea are often constant; tongue is dry and brown, with occasional nausea, vomiting, and diarrhea, sometimes marked stomatitis. Insomnia, cramps in the calves, or pruritus may be present. Mania, delusional insanity, or delirium may develop, or muscular twitchings or convulsions. Death may occur in coma or from a terminal infection, such as acute endocarditis, pericarditis, peritonitis, pleurisy, or meningitis. The duration of the condition may be years. The urine is that of the causal renal lesion.

Diagnosis.—(1) From **opium poisoning,** in which pupils are fixedly contracted, respiration is slow, and albuminuric retinitis and urinary changes of nephritis are absent except as coincidences. (2) From **alcoholic coma,** by odor of breath and examination of retina and urine. (3) From **diabetic coma,** in which onset is sudden and urinalysis shows glycosuria and acidosis. (4) From **cerebral hemorrhage,** in which onset of coma is often sudden, with unequally contracted pupils,

complete hemiplegia, arteries usually thickened, while urinary changes may be absent. (5) From **meningitis**, by absence of rigidity of neck, of focal symptoms, and of persistent vomiting. (6) From **stupor of typhoid, miliary tuberculosis**, etc., by absence of characteristic features of these infections and presence of urinary changes.

Prognosis.—Usually unfavorable.

Treatment.—(See Chronic Interstitial Nephritis, Treatment, p. 314.)

ACUTE NEPHRITIS

Definition.—Called also **acute diffuse, exudative, parenchymatous, desquamative, catarrhal,** and **croupous nephritis,** and **acute Bright's disease.** This is an acute inflammation of the renal parenchyma and interstitial tissue.

Etiology.—Chiefly in young persons, and among adults in males. Exciting causes are (*a*) exposure to cold and wet, burns, extensive skin lesions; (*b*) toxins of scarlet fever, diphtheria, typhoid, measles, other exanthemata, syphilis, and acute tuberculosis; (*c*) cantharides, turpentine, carbolic acid, potassium chlorate, etc.; (*d*) pregnancy.

Pathology.—Kidneys may appear normal or be enlarged, dark red or pale or mottled red and gray, surface smooth, capsule not more adherent than normally, cortex thickened, glomeruli red or pale. Microscopically, there is swelling of cells covering the glomerular tufts and lining Bowman's capsule, with exudate containing red and white cells in its cavity. The capillaries may be filled with cells and thrombi. The epithelium of the tubules is swollen, fatty, or otherwise degenerated. Their lumina may contain desquamated epithelium, red and white cells, serum and casts. The interstitial tissue may be edematous or infiltrated with leukocytes and red cells, or show small-celled infiltration around glomeruli or vessels or uniform connective-tissue

thickening. In individual cases either glomerular, degenerative, exudative, or interstitial changes may predominate.

Symptoms.—*After exposure* or *scarlet fever* the onset may be sudden, sometimes with chills or chilliness; variable fever, which is often absent in adults, 101° to 103° in children; pain in the loins; edema of face and extremities, then of dependent portions of the body, then general; sometimes nausea, vomiting, headache, delirium, or coma. Urine is scanty, dark, of increased specific gravity, contains albumin, red cells, and hyaline, epithelial, and blood casts. Pulse is of increased tension, aortic second sound accentuated. Anemia is marked. *After some febrile diseases* the onset is gradual, with anemia, edema of the eyelids, face, and extremities; scanty, concentrated, albuminous urine containing casts; then headache, nausea, vomiting, little or no fever, dry skin. In these cases there may be gradual recovery, a uremic attack, or termination in chronic nephritis.

Diagnosis.—By routine examination of urine in scarlet fever, pregnancy, etc., and especially when any edema is noticed. In simple **febrile albuminuria** the marked symptoms and urinary changes are absent. **Exacerbations of chronic nephritis** are distinguished by high tension, cardiac hypertrophy, and absence of blood casts.

Prognosis.—Poor in scarlatinal acute nephritis, especially in young children. If they survive, recovery or chronic nephritis may follow. In adults with nephritis due to exposure recovery is the rule.

Treatment.—Bed until recovery is complete, flannel garments, milk diet, water or mineral water in large quantities. Cream of tartar, ʒj, juice of one lemon, sugar, q. s., water, Oj, may be given whenever desired. Hot-air or hot-water baths or hot packs daily or more frequently. Pilocarpine, adult dose, gr. $\frac{1}{4}$ to $\frac{1}{8}$ or less hypodermically, is sometimes given if these fail to cause diaphoresis. If urine is very scanty, dry cups over the kidneys may aid. Bowels moved by salines every morning if necessary, especially if dropsy

persists. Magnesium sulphate, ℥ij to ℥j, compound jalap powder, or effervescent citrate of magnesia may be used. If distention of the skin is extreme, it may be necessary to scarify with strict aseptic precautions. For vomiting, restrict fluids by mouth, rectal feeding if necessary, cracked ice, dilute hydrocyanic acid, carbolic acid, etc. For uremic convulsions, chloroform, pilocarpine hypodermically, and sometimes venesection. For high-tension pulse, glonoin or chloral, repeated as needed. For dyspnea due to hydrothorax or for great ascites, aspiration. Diuretics, except water and salines, should be given only after the acute stage; digitalis and sodiosalicylate of theobromine are useful, the former in cases with low tension. For anemia, iron after acute symptoms subside. In convalescence, avoid exposure; increase diet gradually, avoiding meat for some time.

CHRONIC PARENCHYMATOUS NEPHRITIS

(**Chronic Diffuse, Desquamative, or Tubal Nephritis, Chronic Diffuse Nephritis with Exudation, or Chronic Bright's Disease.**)

Etiology.—Most common in young adult males. It may be secondary to acute nephritis due to exposure, pregnancy, or scarlet fever; or follow excessive use of alcohol, malaria, septicemia, syphilis, chronic suppuration, or tuberculosis; or occur without apparent cause. In children it usually follows acute nephritis of scarlet fever.

Pathology.—The kidneys may be enlarged, with thin capsule, white surface, cortex thickened and yellowish or whitish (*large white kidney*). The epithelium of the tubules is granular or fatty or tubules are distended and contain casts. Cells of the glomeruli and their capsules are swollen and proliferated. There is moderate increase of interstitial tissue. In other cases (*small white kidney*) the organ is small and pale either primarily or as a later stage of the large white kidney. Surface pale, rough, and granular; capsule

thickened and partially adherent; cortex thin, with white or yellowish areas of fatty degeneration. Interstitial tissue markedly increased, epithelial degeneration in tubules extensive. *Large red kidney* is another type, differing from the large white only in that it is dark red or mottled red and yellow from hemorrhages into tubules and interstitial tissue. With any of these types the left heart may be hypertrophied and arteries thickened.

Symptoms.—If it follows acute nephritis, the symptoms of that affection subside, but anemia and urinary changes persist. Usually the onset is gradual, with pallor and puffiness of eyelids, ankles, or hands in the morning. Later, dyspnea, increased edema of face, extremities, and dependent portions of the body, most marked in the morning; pasty, yellowish pallor; subsequently dropsy of serous cavities. Vomiting and diarrhea may be severe. There is increased tension with accentuated aortic second sound, and later arteriosclerosis and cardiac hypertrophy are common. Urine is diminished, high colored, specific gravity usually 1020 to 1025, with much albumin, many hyaline, granular, epithelial, and fatty casts. Later the specific gravity may become low and quantity increased. Headache is common; uremic attacks may occur.

Prognosis.—Death from pulmonary edema, uremia, or terminal inflammations of the serous membranes is the rule. Recovery is rare after the disease has lasted a year, but may occur in children.

Treatment.—Avoid exposure; milk diet, water, and other diuretics, and symptomatic treatment as in acute nephritis (p. 310). For dropsy, salt-free diet, later gradually adding salt to food up to near the limit of tolerance.

CHRONIC INTERSTITIAL NEPHRITIS

(Sclerosis or Cirrhosis of the Kidneys; Granular, Contracted, or Gouty Kidney.)

Etiology.—(*a*) Primary cases—heredity, syphilis, alcohol, habitual overeating, gout, chronic lead poisoning; (*b*) continued sclerosis of the small white kidney; (*c*) secondary to arteriosclerosis,. chiefly in men over forty years who have overeaten, overworked, and used alcohol to excess.

Pathology.—Kidneys small, surface rough and nodular, capsule thick and adherent, color usually dark red, frequently with cysts beneath the capsule; section resistant; cortex thin. Microscopically, connective tissue is increased generally or in patches, especially between the medullary rays. Epithelium of the tubules in fibrous areas is degenerated or atrophied, tubules are sometimes enlarged and contain casts. In the glomeruli, Bowman's capsule and capillary tufts may be thickened, with proliferation and exfoliation of cells, or partially or completely replaced by small nodules of connective tissue. The arteries and veins are markedly sclerotic. The connective-tissue increase is regarded as in part a replacement hyperplasia following degeneration of glomeruli and tubules. General arteriosclerosis and cardiac hypertrophy are associated lesions.

Symptoms.—Both invasion and course may be insidious and symptoms entirely absent or present only in the late stages. The disease often lasts many years. Loss of appetite, dyspeptic symptoms, vomiting, headache, migraine, dyspnea, and increased urine are common. Urine is light colored, specific gravity low, often 1005 to 1012, frequently with a trace of albumin and a few hyaline or granular casts. In the late stages the quantity may be decreased, specific gravity and albumin increased. In the arteriosclerotic forms urine is of normal or diminished quantity, specific gravity normal or increased, casts are more numerous, and albumin, while variable, is usually more abundant. The heart is hypertrophied, with accentuated aortic second sound; pulse of increased tension, arterial wall usually thickened. Skin usually dry, eczema common, edema rare except when due to cardiac failure. Visual disturbances,

sometimes even sudden blindness, may be present. Albuminuric retinitis with hemorrhages may lead to diagnosis before other symptoms. Bronchitis, pneumonia, pulmonary edema, attacks of uremic or cardiac dyspnea frequently occur, sometimes cerebral or meningeal hemorrhage. These pulmonary and apoplectic complications are often fatal, or death may occur after cardiac failure or with uremic symptoms. Hematuria may be the first or only symptom or occur with other symptoms of nephritis. It comes on insidiously, is spontaneous, unilateral, usually continuous but may be intermittent.

Diagnosis.—By abundant urine of persistent low specific gravity, often with a trace of albumin and a few hyaline or granular casts, increased tension, and signs of cardiac hypertrophy with ringing aortic second sound.

Prognosis.—Incurable, but life may be prolonged for years. Test functional capacity with phenolsulphonephthalein.

Treatment.—*General:* Avoid worry, mental and physical overexertion, excess of food and drink, alcohol, and tobacco. An equable warm climate is desirable. Bathing, regularity of the bowels, flannels to prevent chilling, and ingestion of a sufficient amount of water are important. *Symptomatic:* For too high tension, restricted diet, occasional saline purgation, diaphoresis by hot-air or hot-water baths; if necessary, chloral, gr. v, every four hours, or glonoin, gr. $\frac{1}{100}$, t. i. d., increasing the dose to gr. $\frac{1}{10}$ or more every two or three hours if necessary, until the desired effect is produced, limiting it if headache or flushing occurs, and discontinuing it occasionally for a few days after giving it for several weeks. Care must be taken not to decrease the tension too much. For anemia, iron in large doses. For cardiac failure, rest, strychnine, or digitalis. For uremia, saline purgation, hot-air or hot-water baths, hot packs, high rectal irrigations with hot normal saline solution or enemata of the same (℥viij at 115° to 120° every three hours, if retained and effectual); chloral or glonoin for high tension; morphine, chloroform inhalation during convulsions, venesection if necessary.

AMYLOID KIDNEY

Amyloid, waxy, or lardaceous degeneration of the kidney usually develops, with similar changes in the liver, spleen, and other organs, in syphilis, prolonged suppuration, especially of bone, tuberculosis, sometimes in malaria, leukemia, lead poisoning, and gout.

Pathology.—Kidneys usually enlarged, firm, pale; cortex thickened and showing the peculiar translucent amyloid appearance, beginning in the glomeruli. The pyramids are dark red. Tincture of iodine on the cut surface stains the amyloid area deep mahogany color. Lesions of chronic nephritis are also present.

Symptoms.—Urine increased, pale, of low specific gravity; albumin usually present in large quantity; usually hyaline, granular, and fatty casts. Anemia, dropsy, and diarrhea may be present; cardiac hypertrophy and increased tension only when there is complicating chronic nephritis.

Diagnosis.—By the above urinary changes in person suffering from syphilis, tuberculosis, prolonged suppuration, etc.; often associated with enlargement of liver and spleen.

Prognosis depends upon the cause.

Treatment is that of chronic interstitial nephritis (p. 314).

PYELITIS

Etiology.—Pyelitis, inflammation of the renal pelvis, is caused by invasion of bacteria from the blood or by ascending pyogenic or tuberculous infection from the lower parts of the genito-urinary tract. *Pyelitis* is inflammation of the pelvic mucosa; *pyelonephritis* includes extension of infection to the kidney; *pyonephrosis* or *surgical kidney* signifies conversion of the kidney into a pus sac enclosed or not be renal tissue.

Pathology.—Kidney and sometimes the ureter enlarged; mucosa swollen, sometimes ecchymotic, later rough, gray, and thick; extension to the kidney may cause abscess of that structure. Tuberculous pyelitis usually begins at the apices of the pyramids, and its pus often becomes caseous or calcified. Calculus is more often secondary to than a cause of pyelitis.

Symptoms.—Pain in the back, tenderness on deep pressure over the affected kidney, and turbid urine, either acid or alkaline, containing pus, mucus, and sometimes red blood cells. Chills, high fever, and sweating occur. When the affection becomes chronic, pyuria is often intermittent or at least variable; fever may be intermittent, with chills so periodical as to suggest malaria. Anemia, emaciation, and finally cachexia result; sometimes pyemia, which may simulate typhoid. The kidney is tender and eventually enlarged, the urine often increased.

Diagnosis.—(1) From **perinephric abscess,** by history, presence of definite renal tumor, with absence of edematous swelling in the lumbar region. (2) From **cystitis,** by polyuria, greater albuminuria, localized pain and tenderness in one lumbar region, with absence of pain in the bladder and of frequent micturition, or by catheterization of the ureters showing pus from higher up. (3) Catheterization of the ureters, with microscopic examination and determination of the freezing point of the urine from each side, shows the side affected, while tuberculous pyelitis is differentiated from other cases by examination of urine for tubercle bacilli or its inoculation into guinea-pigs, which develop tuberculosis.

Prognosis.—Mild cases usually recover; suppurative cases may terminate in amyloid disease, perinephric abscess, or death from exhaustion.

Treatment.—In mild cases, large amounts of alkaline waters, urotropin, gr. v to x, t. i. d., tonics, and simple diet. In severe cases or with a tumor, surgical treatment.

HYDRONEPHROSIS

Dilatation of the pelvis and calices may be congenital or due to obstruction of the ureter or urethra by external pressure, kinking, calculus, or stricture. Atrophy of the parenchyma results. If unilateral, the **symptoms** are merely those of a fluctuating tumor. This may be intermittent if the causal factors act intermittently, as in kinking of the ureter in movable kidney, and disappearance of the tumor is accompanied by passage of a large amount of urine. Uremia in bilateral cases and pyonephrosis are possible sequelæ.

Prognosis for life is good.

Treatment.—Surgical if necessary. In intermittent cases without symptoms, no treatment, or a belt to retain the kidney in place. Inversion or the knee-chest posture may replace the kidney and so relieve obstruction due to kinking of the ureter.

NEPHROLITHIASIS (RENAL CALCULUS)

Etiology.—Formation of a stone or gravel in the kidney or its pelvis may occur in intra-uterine life or at any age. A family tendency, sedentary life, excesses in eating and drinking, and very acid urine predispose. The nucleus may be bacteria, a blood clot, etc. The exciting cause is uncertain.

Pathology.—The concretions vary in size from that of fine sand to that of a bean, or may be a complete cast of the pelvis and calices (coral calculus). They may consist of uric acid, smooth, hard, reddish brown; of calcium oxalate, hard, white, or rough and dark from spicules of uric acid; of either of these covered with urates; of calcium phosphate; more rarely of other substances.

Symptoms.—Gravel may be formed and passed or a large coral calculus be present without symptoms. A stone remaining in the kidney may cause dull, aching pain in the affected organ or referred to the opposite side, sometimes hematuria or pyuria, with chills and fever due to pyelitis (p. 316). Passage of a calculus through the ureter causes "renal colic," intense pain beginning in the kidney, radiating along the ureter to the testicle, which is retracted, and to the inner side of the thigh. It lasts from an hour to several days, until the stone reaches the bladder, and is often accompanied by a chill, fever, profuse sweating, collapse, strangury, and hematuria. Suppression during the attack is rare, but a large quantity of urine is usually passed after it, and a feeling of soreness may be present for several days. The stone may again cause pain in passing through the urethra or remain in the bladder as a nucleus for a vesical calculus.

Diagnosis.—(1) From **intestinal colic**, in which the pain is not localized in the region of renal colic, frequently moves to other regions and may be relieved by pressure, and urinary changes are absent. (2) From **biliary colic**, by direction of radiation of pain, absence of jaundice, of clay-colored stools, and of bile in urine. (3) From **vesical calculus**, in which usually the pain is in the bladder or referred to the glans penis and urine alkaline. The *x*-rays may aid in diagnosis by showing the presence and position of the calculus.

Prognosis depends chiefly upon secondary renal changes—pyelitis and pyonephrosis.

Treatment is surgical. Increase of size of uric acid calculi may be prevented by keeping urine alkaline with citrate of potassium; of phosphatic calculi, by maintaining acidity of the urine with benzoic acid. In either case large amounts of water should be ingested and diet restricted to maintain the desired urinary reaction. For renal colic, chloroform inhalations, morphine hypodermically, hot applications to the lumbar region, hot baths, hot drinks. Inversion or knee-chest posture may relieve.

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