1. The restraining force of the cardiac attachments, particularly at its base.

2. The density and resistance of the surrounding tissues. This latter may be positive, as in case of a solid tumor, or negative, as in pneumothorax.

3. The weight of the heart is, as a rule, of slight importance. In cardiac hypertrophy, however, it may be sufficient to produce temporarily, during decubitus on the left side, a decided displacement.

4. The direct exciting cause either of pressure or of traction.

These elements may produce one or both of two forms of motion—either distinct displacement or rotation of the heart about one of its axes. Rotation, owing to the peculiar attachments of the heart, is almost invariably about the long axis, or a fixed point at the base.

Passing now to a special consideration of the causes operative in the several varieties of displacement, we find that displacement toward the left, which is most common, is brought about by contraction of the left lung by accumulation of fluid or air in the right pleural cavity, or by solid enlargements of the right lung or right lobe of the liver. Displacement toward the right is most frequently due to the presence of fluid or air in the left pleural sac, but may also result from contraction of the right lung consequent upon chronic pleurisy or fibroid phthisis; and from the development of tumors in the left side of the chest or mediastinum. Displacement downward may be caused by aneurisms or solid tumors, emphysema of the lungs, etc., in the thorax, or by collapse of the stomach or intestines. Displacement upward may be due to solid, liquid, or gaseous accumulations in the abdominal cavity, ovarian tumors, enlargement of the liver, fibroid tumors of the uterus, etc. Enlargement of the left lobe of the liver usually displaces the heart upward and to the left, while great enlargement of the spleen may push it upward and to the right. Enlargement of the liver from abscesses will also raise the organ somewhat, a fact which may become very valuable as a means of distinguishing between hepatic abscess and abscess in the abdominal wall over the liver. Another cause of this displacement is contraction of the upper part of either lung, the most common pathological condition being phthisis. Forward displacements are usually caused by an aneurism, or a solid tumor in the posterior mediastinum. Backward displacement may be produced by tumors in the anterior mediastinum, collections of air, pus, or blood in the same situations, or a posterior exostosis of the sternum.

Symptoms and Physical Signs.—Simple displacement of the heart, unattended by compression of its walls or torsion of the great vessels, causes no distinctive subjective symptoms. This is especially true when the displacement has been gradual. When it has been rapid, however, or is due to pressure, symptoms of embarrassed cardiac action may be developed suddenly. They commonly consist of præcordial oppression, more or less severe pain, amounting even to true angina; palpitation with its accompanying peculiar sense of lack of breath, which may increase until it becomes absolute dyspnœa; a weak, irregular pulse, and a pale or cyanotic countenance. Such sudden compression results most frequently from a rapidly developed gaseous distention of the stomach or intestines, and may quickly become dangerous, and, if not relieved, produce collapse and death.

Lateral displacements are usually more readily detected when occurring toward the right than toward the left side. In the former case the apex beat not infrequently corresponds to the right nipple, between the fifth and seventh ribs, the apex itself being usually raised by the width of an intercostal space above the normal level. In displacement toward the left the apex beat may be observed in the axillary region, with little or no perceptible movement at the normal situation of the apex.

Dr. Douglas Powell has shown that in right lateral displacement the apex of the heart is depressed, but never so as to occupy a position external to the base; whereas in displacements to the left the apex is relatively elevated, and the long axis of the heart is nearly or quite horizontal. According to Hayden, the distinctive features of lateral displacement from liquid effusion into the opposite pleura are the slow and mensurable mode of its occurrence; percussion dulness on the side whence the heart has been displaced, and clearness on the opposite side, beyond the limit of cardiac dulness; and, in the event of the removal of the displacing medium, return of the heart to its natural situation, or beyond it when the lung previously compressed has become incapable of expansion. Since the apex of the heart is so much more movable than its base, it is evident that, in cases of displacement, the maximum points of its sounds will not be equally removed from their normal situation. Thus, in left pleuritic effusion, when the maximum point of the first sound is carried out of place to the extent of seven or eight inches, the maximum point of the second sound is scarcely ever changed more than an inch and a half. In displacement to the right a systolic murmur has been noticed, which is ascribed to a twisting of the great vessels. When displacement of the heart is caused by cancer of the lung or pleura—with or without effusion of serum—two diagnostic symptoms of great value are especially to be noted, viz., enlargement of the subcutaneous veins of the affected side of the chest, and distinct transmission, on the same side, of the sounds of the heart. These signs, when accompanied by dulness on percussion, absence of respiratory sounds, and vocal fremitus, except at the root of the lung, and by excentric displacement of the heart, become pathognomonic of cancer of the lung or pleura.

In downward displacement the heart not only lies lower than usual, but it generally sways a little to the right. Displacement in this direction is limited by the diaphragm, and by the attachments at the base of the heart. It is most frequently caused by emphysema of the lungs, which constitutes, in fact, one of the most valuable signs in this form of displacement. If the apex beat is perceptible at all, it is situated below and to the left of its normal position. There is pulmonary resonance instead of dulness in the præcordial area, and the cardiac sounds are transferred to the epigastric triangle and the lower left cartilages.

Upward displacement appears to reach its maximum when resulting from ovarian dropsy. In some of these cases distinct impulse is not to be felt below the second interspace. The sounds of the heart are carried upward and weakened. In pericardial effusion the cardiac impulse may correspond in position to the left nipple, or even be shifted farther in that direction. When the effusion is very copious the apex beat may be completely obscured.

Backward displacement occurs most frequently in connection with other forms of dislocation. It is unaccompanied by any physical signs due directly to the condition itself.

Forward displacement is ascertained with difficulty. The physical signs are: Increase of the area and strength of pulsation and of præcordial dulness; bulging of the same sometimes is noticed in young subjects. The cardiac sounds are intensified.

Diagnosis.—The diagnosis of acquired displacements of the heart is to be made by the position of the apex beat, and of the impulse of the organ generally, by the altered locality of cardiac percussion dulness, and by the comparative intensity of the heart's sounds, more especially of the first, at different parts of the chest. The chief conditions which simulate cardiac dislocation are the following: Physiological displacements, to which reference was made at the outset; præcordial bulging in cardiac hypertrophy; intrathoracic tumors and aneurisms lying behind the heart and pushing it forward—thus producing the closest possible resemblance to cardiac hypertrophy; adhesions of the pericardium; atrophy of the lungs.

Prognosis.—Displacements of the heart being, in most instances, attended by only a slight amount of functional disturbance, and oftentimes by none at all, their prognosis is determined by the disease in which they originate. As already mentioned, when they are accompanied by sudden and violent compression of the heart, these symptoms, if not relieved in time, may result fatally. Usually, however, the direct consequences of these displacements are annoying rather than serious.

Treatment.—There is no direct treatment of cardiac displacements, they are to be remedied only by the removal of their cause; but this, in the traction class of cases, is very rarely possible. In those, however, which result from pressure, treatment is often both urgently indicated and highly successful. The troublesome pulsation which is sometimes experienced may frequently be relieved by the simple application of a belladonna plaster, with assurances as to the unimportance of the symptom. *Alfred L. Loomis.*

HEART DISEASES: ENDOCARDITIS.—Endocarditis is an inflammation of the endocardium, *i.e.*, of the membrane lining the cavities and valves of the heart. In the great majority of cases the valvular endocardium is alone involved.

Classification.—Endocarditis is divided into two large classes, acute and chronic. We shall confine ourselves chiefly to a description of the acute form. Chronic endocarditis is practically synonymous with chronic valvular disease of the heart.

Acute endocarditis is divided into benign and malignant. These are differentiated both clinically and pathologically. The synonyms of benign are simple, rheumatic, verrucose and papillary; those of malignant are ulcerative and infective. It must be remembered that the distinction between benign and malignant is one not of kind, but of degree, as all stages between the two are found, and the same bacteria may be present in both.

History.—Bouillaud first recognized endocarditis as a distinct disease, publishing an account of it in his work on the heart and vessels in 1824. The discovery of the stethoscope by Laënnec made it possible to recognize endocarditis during life. Previous to this time it was impossible to diagnose this disease with any degree of certainty on account of its indefinite and unreliable symptomatology. In 1814 Kreysig of Dresden devoted special attention to inflammation of the lining membrane of the heart. Bouillaud by means of the signs furnished by Laënnec was able accurately to localize the endocarditis on certain valves. He declared there was a close relationship existing between rheumatism and endocarditis. He also called attention to the fact that other diseases than rheumatism, for instance, pneumonia, might produce endocarditis. He recognized likewise the fact that the acute form might be attended by symptoms of pyæmia. Virchow described the process of embolism, thus explaining the obscure complications on the part of the brain and kidneys, so intimately associated with endocarditis. Following this the important additions to our knowledge of the disease have been made by bacteriologists. Klebs in 1878 was the pioneer and insisted upon it that the various bacteria of any one of the infectious diseases might colonize on the endocardium and produce endocarditis. He showed that both the benign and the malignant forms were caused by bacteria. Litten found the same micro-organism in both forms and showed that there was no abrupt, but only a gradual difference between the two. Since this time multitudinous investigations have been made by bacteriologists all over the world, largely increasing our knowledge of the etiology of the disease.

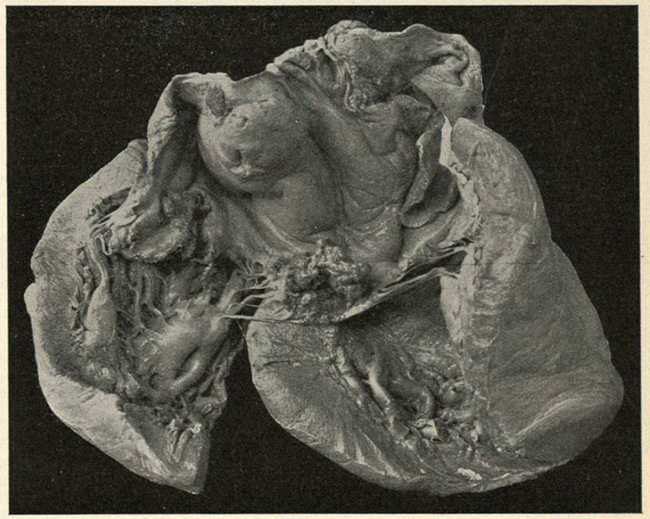

Fig. 2590.—Malignant Endocarditis Involving the Mitral Valves and the Left Auricle. (Original.)

Bacteriology.—The following bacteria have been found in the vegetations of endocarditis: Streptococci, staphylococci, pneumococci, and gonococci most frequently; less often the bacteria of typhoid fever, anthrax, diphtheria, tuberculosis, influenza, and the Bacterium coli communis.

Etiology.—Acute endocarditis is in most cases a product of bacterial activity. It is usually secondary to the various infectious diseases. When, as rarely happens, a causative factor cannot be found, it is termed cryptogenetic. Bacteria cannot always be demonstrated, but this may be due to the fact that they are likely to disappear as the vegetation becomes old. Sometimes it is possible to demonstrate them only by culture methods.

Since the time of Bouillaud acute articular rheumatism has been recognized as the most important associate of endocarditis. It is incorrect to speak of it as a cause, since it, like endocarditis, is the result of bacterial activity, with different localizations. When the germ or its toxin attacks the joint it is rheumatism; when the endocardium, endocarditis. In a certain percentage of cases, the pericardium is attacked either alone or together with the endocardium.

Chorea seems to have a special association with endocarditis. The vast majority of fatal cases show endo-

carditis. During childhood scarlet fever is more often associated with endocarditis than any of the other diseases of early life. Thayer and Lazear have analyzed thirty cases of fatal ulcerative endocarditis which occurred in gonorrhœa.

Osler has demonstrated the frequent association of pneumonia with malignant endocarditis. Infected

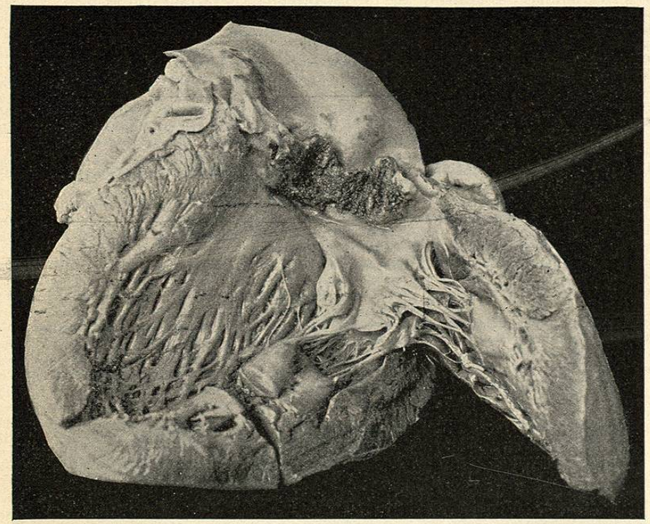

FIG. 2591.—Malignant Endocarditis; Ulceration of the Aortic Valves; Hole through One of the Valves; Old Atheroma. (Original.)

wounds, or septic processes, external or internal, such as puerperal fever, boils, gastric ulcer and bone necrosis may also be followed by the malignant variety. The infectious agent may find an entrance through lesions of the skin, mucous membranes of the throat, gastro-intestinal, respiratory, and genito-urinary tracts—and produce endocarditis.

It is frequently found in debilitating diseases, such as Bright's disease, diabetes, cancer, etc. These diseases reduce the vitality of the patient, rendering him more susceptible to the action of the infectious agent.

Not many years ago in discussing the etiology of this disease a prominent authority wrote that it arose from exposure to cold or without any cause being discoverable. Our present certainty regarding its etiology is in pleasing contrast to former guesswork. Benign endocarditis is common to childhood. This is supposed to be due to the greater vulnerability to bacterial action during early life. The malignant form is rare in childhood.

MORBID ANATOMY.—*Benign Form.*—As a rule endocarditis involves the valves, but it is possible for it to be located upon the chordæ tendineæ, or the walls of the cavities (mural endocarditis). Grayish-yellow, opaque bodies, called vegetations, are seen upon the diseased valves, usually near their edges. They may be minute and in great numbers, may resemble venereal warts, or may be so large as to resemble an extensive cock's comb. Microscopically, they consist of a superficial layer of coagulated fibrin, in the upper layer of which are entangled numbers of leucocytes deposited from the blood stream. Deeper down is a layer of simple fibrin free from corpuscular elements and below this again is a layer of granulation tissue. In these vegetations bacteria are frequently found. Sometimes they are demonstrated only by culture methods. Again they are absent if the vegetation has existed for some time. These vegetations are a constant source of danger, as they are frequently whipped off by the blood current and carried as emboli to different parts of the body, producing striking symptoms. The mitral valves are most frequently attacked by this process. Next come the aortic and very infrequently the tricuspid and the pulmonary valves. Fœtal endocarditis is confined chiefly to the right side of the heart. The sequel of this morbid process may be resolution with no impairment of the function of the valve, or, more commonly, thickening, retraction, curling, or even calcification, resulting in chronic valvular lesions.

Malignant Form.—All stages of endocarditis are found from the benign through the transitional to the malignant form. The same bacteria are present in all, but are more numerous and virulent in the malignant. There is a deposit upon the inflamed area, resembling in many respects that of the benign form, but containing many bacteria of great virulence. If this becomes detached there is loss of tissue with the formation of an ulcer. This may penetrate both layers of the valve, or only one layer of the endocardium, thus producing a valvular aneurism. The destruction of tissue may be very rapid and extensive (Fig. 2592). As in the benign form, the left side of the heart is usually attacked. The tendinous cords, the wall, or the papillary muscles are more likely to be involved in the process than in the benign form. The changes produced by the bacterial emboli constitute an important part of the morbid anatomy of ulcerative endocarditis. Small masses of bacteria are whipped off by the blood current and are carried to the most diverse parts of the body: brain, lungs, kidneys, spleen, skin, mucous membranes, eye, etc. Inflammation and suppuration soon occur and we have the production of the so-called metastatic abscesses. Few of the viscera may escape.

SYMPTOMATOLOGY.—Flint truly says, authenticated cases of endocarditis disconnected from other diseases, which serve to mask its symptoms to a greater or less extent, are wanting.

Benign Form.—The symptoms are usually very indefinite, the patient rarely complaining of more than a præcordial distress. Almost never does this amount to an actual pain. There may be oppression and shortness of breath. The heart action may be rapid and irregular, furnishing symptoms of palpitation. The temperature is usually elevated as a result of the associated disease. A slight extra elevation, a degree or two, usually occurs with the advent of endocarditis. Naturally then, because of the unobtrusiveness of the symptoms, the disease is frequently latent and not recognized. The physician who keeps in mind the etiology of endocarditis and makes routine heart examinations in all cases of rheu-

matism, chorea, pneumonia, etc., most frequently recognizes the disease. It is extremely important to remember that in children and adolescents, a rheumatism followed by grave heart complications may be very insidious, and show itself only by a history of pain in joints or limbs—no appreciable swelling or tenderness of joints and very little fever.

The appearance of signs due to emboli frequently directs our attention to the heart.

Malignant Form.—In this variety we find grave constitutional symptoms. There are evidences of a most severe infection, which requires considerable skill in differential diagnosis. It may be impossible to separate the symptoms from those of the associated disease, such as sepsis or pneumonia. According to the symptomatology we distinguish two large classes: (1) The *typhoid*, in which the case resembles typhoid fever, showing continued fever, delirium, dry coated tongue, distended abdomen, roseola, splenic enlargement, and possibly diarrhœa. (2) The *septic*, resembling septicæmia. The temperature shows the streptococcus curve, *i.e.*, rapid elevation and descent several times daily, accompanied by chills and drenching sweats. There is profound intoxication and depression of vitality. The intermittent temperature and chills may suggest to some malaria. In these cases infective embolic processes are common, showing themselves in the skin, the mucous membrane, retina, and the various internal organs.

Certain authors mention a *cardiac* group in which patients with chronic valvular endocarditis show marked fever and evidences of recent endocarditis. Many of these cases resemble either the typhoid or the septic form and may run a very acute course. Others run a chronic course lasting for a year or so.

Another group of cases may be termed the *cerebral* in which the symptoms resemble meningitis, either basilar or cerebro-spinal.

Some authors also made a *cutaneous* group. Here petechial rashes are found; also skin abscesses from infected emboli. Erythematous rashes are not uncommon. Because of petechial rashes, cases of malignant endocarditis have been diagnosed cerebro-spinal meningitis and black smallpox.

DIFFERENTIAL DIAGNOSIS.—Attention to etiology will assist one. Benign endocarditis is more likely to follow rheumatism; malignant, infective processes and pneumonia. The symptoms of the malignant form are much more intense, suggesting septicæmia or typhoid fever. The positive Widal reaction, the absence of leucocytosis, the presence of the diazo reaction, would all point to typhoid fever. Unfortunately the Widal and diazo reactions may both be absent, and with the advent of a complication there may be a leucocytosis. So in some cases it is impossible to differentiate endocarditis from typhoid fever. Even a past master in the art of diagnosis like Osler diagnosed a case typhoid fever, which post mortem was proven to be endocarditis.

Osler gives the following points for guidance: The more abrupt onset in endocarditis, and the absence of any regularity of the pyrexia in the early stage of the disease, the cardiac pain, oppression, and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. There is marked leucocytosis in infective endocarditis. Bacteriological examination of the blood as used by Cole of Johns Hopkins in the diagnosis of typhoid fever is of striking value, but unfortunately its field of application is limited. The specific variety of endocarditis has been diagnosed during life by the finding of the micro-organism in the blood.

PHYSICAL SIGNS.—These are very uncertain, because our main dependence is placed upon the presence of a soft blowing systolic murmur at the aortic or mitral areas. Such murmurs are extremely common and frequently do not mean endocarditis, but occur with the various acute febrile processes. Similar murmurs occur in anæmia, and in many conditions which cannot be explained. The size of the heart is not always of help to us, since an acute endocarditis may not enlarge it. If in the course of an acute disease a murmur develops under our observation, which cannot be accounted for by anæmia or weakness of the heart wall, and especially if accompanied by an increase in the fever, we are justified in diagnosing endocarditis. Sudden appearance and disappearance of murmurs may occur when vegetations are rapidly forming and changing their shape.

PROGNOSIS.—In the vast majority of cases simple endocarditis entails no immediate danger. The danger in such cases is more or less remote and dependent upon the chronic valvular defects which follow.

MALIGNANT ENDOCARDITIS.—Death may take place in

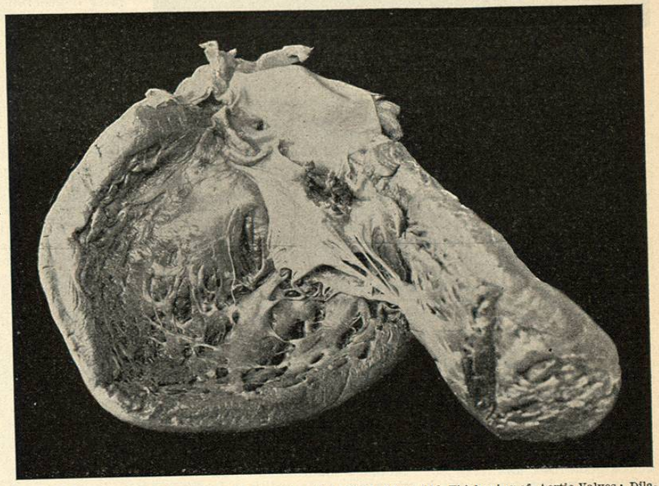

FIG. 2592.—Malignant Endocarditis; Destruction of Aortic Leaflet; Old Thickening of Aortic Valves; Dilatation of the Left Ventricle. (Original.)

three days. The disease may be protracted for months. Death results from progressive failure of strength or in consequence of secondary suppuration, or of embolism of an important cerebral vessel. The prognosis is almost always unfavorable.

TREATMENT.—This should combine treatment of the associated disease and the endocarditis. It will in most cases be necessary to treat the endocarditis after the patient has recovered from the associated pneumonia, rheu-

matism, etc. Whether the salicylates have any effect upon endocarditis is a much mooted question. I am of the opinion that they have a decided indirect effect upon the valvular disease. Any one who has administered

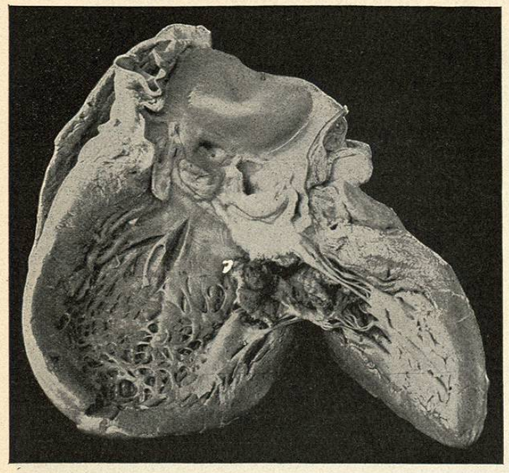

FIG. 2593.—Malignant Endocarditis on Top of Old Sclerosing Process. Marked Involvement of the mitral valves. (Original.)

salicylates in proper dosage in acute articular rheumatism knows of their remarkable, almost specific control over the disease. Endocarditis may develop at any time during the progress of a rheumatic fever; consequently if we shorten the time of the disease we lessen the possibility of the development of endocarditis. If the valvular inflammation has already developed, the chances of resolution are increased, if the fever, pain, and restlessness are controlled by the giving of the salicyl compounds. Bruce advises the combination of digitalis with the salicylates if the heart seems weak. In the direct treatment of endocarditis *rest* is by all means the most essential thing. The work of the heart and the movement of the diseased valves must be reduced to a minimum. This is the best accomplished by putting the patient to bed. An ice bag over the præcordium is often of service in quieting the action of the heart. The time required in bed must be decided by the progress of the case. It may require months in order to guard against serious and permanent injury to the heart. At best this frequently cannot be avoided. The development of symptoms of incompensation will demand the administration of digitalis and strychnine. Anæmia frequently develops as the disease progresses, and requires iron, tonics, good food, and fresh air. Mercuric chloride and quinine have been employed in ulcerative endocarditis for the purpose of destroying the bacteria upon the endocardium, but it is very unlikely that they are of any use.

ACUTE RECURRENT ENDOCARDITIS is a very interesting form occurring on old valve lesions. It may be benign or malignant (Fig. 2593).

CHRONIC ENDOCARDITIS is either a sequel of the acute form or is an evidence of senility, being a part of the process of arteriosclerosis, involving the general arterial system. This contracting endocarditis leads to thickening, curling, contraction, and frequently to calcification of the valves. The same causes which produce premature arteriosclerosis produce it, namely: excessive physical activity, excessive use of alcohol, syphilis, gout and diabetes, and chronic diseases of the kidneys. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valves.

The effects and treatment of chronic endocarditis are those of chronic valvular lesions and will be considered under that heading. *James Rae Arneill.*

HEART DISEASES: FATTY DEGENERATION.—Fatty degeneration of the heart muscle is a retrograde change characterized by the formation of minute fat droplets within the muscle protoplasm. It is distinguished from fatty infiltration, in that in the latter process the fat is deposited in connective-tissue cells, either beneath the pericardium or between the muscle fibres. The term *fat heart* is often used as a designation for both fatty degeneration and infiltration, but it should be applied to the latter condition only. It should be emphasized that the essential difference between these two conditions is, that in fatty degeneration the fat is in the muscle cell, formed there by the disorganization of the muscle protoplasm; while in fatty infiltration the fat is in connective-tissue cells, deposited there from the blood.

GROSS APPEARANCES.—Severe degrees of fatty degeneration may exist without recognizable changes in the appearance of the heart. The naked-eye diagnosis of this condition is in many cases difficult and uncertain. When the process is extreme the heart muscle becomes yellowish in color, and of softer consistence; it may have a fatty shine or give a fatty smear. The condition may be diffuse or localized to certain areas. The latter form is the more readily diagnosed, inasmuch as the heart muscle so affected presents a peculiar mottling of red and yellow, occasionally disposed in the form of stripes or broken bars, the so-called "tiger-heart." This appearance is best seen on the endocardial surface of the left ventricle. In the majority of cases the degeneration is most marked in the wall of the left ventricle near the apex; the septum, right ventricle and auricles being affected in degree according to the order given. In the left ventricle the papillary muscles and trabeculæ usually show the most severe change, and in some cases the process may be sharply localized to these, the yellowish color of the papillary muscles standing out in sharp contrast to the normal brownish-red of the remainder of the heart wall. The heart is usually increased in size without a corresponding increase of weight. Dilatation of greater or less degree occurs, and the organ as a whole is flabby, flattening when laid upon the table. On section into the muscle the color change may affect the wall uniformly throughout, but more often it is found in streaks and patches of varying form, size, and distribution. The discolored muscle is more opaque than normal, cloudy, easily torn, and gives a fatty smear to a warm knife. In very extreme cases the degeneration may be so severe in sharply outlined areas as to present an appearance closely resembling that of an abscess. It must be remembered that fatty degeneration of the heart muscle rarely exists alone, but is usually associated with cloudy swelling, atrophy, anæmia, etc., all of which processes complicate the diagnosis from the gross appearances.

MICROSCOPICAL APPEARANCES.—The diagnosis of fatty degeneration in many cases can be made only on microscopical examination. This should always be done with the fresh tissue when possible, inasmuch as the processes of fixation and hardening often completely destroy the appearances of the degeneration by the removal of the

fat droplets and the contraction of the tissue. Fixation of the muscle in osmic acid or Flemming's solution will obviate this danger. Slight degrees of fatty change are, however, always best brought out by the treatment of freshly teased muscle with osmic acid. The affected muscle fibres are found to contain minute fat-droplets arranged in rows between the masses of interfibrillary sarcoplasm, beginning about the nucleus and extending linearly toward the ends of the fibres. In the early stages the transverse striations are usually not affected, but in severe degrees of the change they gradually disappear, first at the fibre-ends and then toward the nuclei. The staining power of the nucleus is always affected; in extreme degrees the nuclei may be entirely lost. The changes are usually most marked in the fibres lying just beneath the endo- and pericardium, the endocardium itself being rarely affected. In the acute forms of fatty degeneration cloudy swelling, segmentation, and fragmentation, etc., are usually found in association with the fatty change; in the chronic cases atrophy of the fibres, both simple and pigment, with increase of the interstitial connective tissue, is present.

ETIOLOGY.—The causes of fatty degeneration of the heart are both local and general. Of the former disturbed nutrition resulting from sclerosis or obstruction of the coronary arteries is the most common and important cause. Fatty degeneration of the muscle may also be caused by disturbances of nutrition secondary to myocarditis, cardiac abscess, pericarditis, and endocarditis. In pericarditis the degeneration may be confined to the layer of muscle immediately beneath the epicardium.

The two great general causes of this degeneration are anæmia and intoxication. It is of common occurrence in pernicious anæmia, chlorosis, leukæmia, and all forms of secondary anæmia. It occurs after long-continued or frequently repeated hemorrhage (epistaxis, hæmoptysis, hæmatemesis, menorrhagia, metrorrhagia, hemorrhage from the intestines, hæmaturia, etc.). It has been observed after venesection. Fatty degeneration is also commonly associated with the cachectic anæmias of chronic suppuration, chronic diarrhœas, syphilis, tuberculosis, malaria, malignant growths, etc. In all of these conditions the systemic intoxication is to be regarded also as a possible factor in the production of the condition. Deficient oxygenation due to cardiac insufficiency, valvular lesions, emphysema, fibroid pneumonia, etc., also causes fatty change of the heart muscle. The relation of fatty degeneration to cardiac insufficiency is not entirely clear; the condition is almost always present in the failure of compensation of the hypertrophied heart in chronic valvular disease, and while it is usually regarded as being the cause it is also possible that it may be the result of the cardiac insufficiency.

Extreme fatty degeneration of the cardiac muscle occurs in poisoning with phosphorus, arsenic, antimony, etc. It may also occur to an extensive degree in the acute infectious diseases, especially in typhus and typhoid fevers, recurrent fever, scarlet fever, diphtheria, pneumonia, puerperal fever, pyæmia, etc. In acute yellow atrophy of the liver and in puerperal eclampsia the fatty degeneration of the heart may be so marked as to resemble that seen in phosphorus poisoning. In the infectious diseases the fatty change is to be attributed chiefly to the action of the poisons produced in these conditions, but the effect of the increased temperature of the body must also be regarded as an important factor. Long-continued mercurial inunctions, iodoform and chloroform poisoning, morphinism, the camphor habit, the long-continued use of dilute mineral acids in beverages, or as adulterants of vinegar, mushroom poisoning, etc., are among the many other supposed factors of fatty degeneration of the myocardium. In the later stages of chronic nephritis and gout there is almost always present extensive fatty change in the heart, resulting partly from the general cachexia and anæmia, and partly from sclerotic changes in the coronary vessels. The habitual use of alcohol is one of the most important causes of slowly progressive fatty degeneration of the myocardium. The condition is found particularly in the case of habitual beer drinkers. The condition is of very frequent occurrence among the lower classes in Germany and Austria with whom beer forms a very important part of the daily diet. In France the use of dilute mineral acids in some of the beverages consumed by the lower classes is said to be an important factor in the production of fatty degeneration.

High living, over-eating, lack of exercise as well as excessive muscular activity are regarded as either primary or predisposing factors to cardiac degeneration. Idiopathic hypertrophy is practically always followed by fatty degeneration. Cardiac over-strain in laborers, athletes, etc., is another very important etiological factor. A slight amount of fatty change in the senile heart is to be regarded as physiological. The pathological fatty degeneration of the heart due to anæmia or cachexia is found more frequently in women than in males; that due to coronary sclerosis, alcoholism, etc., much more frequently in males. The majority of the cases are in individuals past fifty years of age. Cardiac fatty degeneration occurring in young individuals is almost always due to intoxication or infection.

SYMPTOMS.—The symptoms of fatty degeneration do not differ from those of other forms of myocardial affections. It is usually impossible to differentiate between fatty and fibroid heart. The most extreme degrees of fatty degeneration, as found in severe cachexias, pernicious anæmia, etc., may give rise to no symptoms if cardiac dilatation has not taken place. It is the latter condition which produces the symptoms which are usually associated with fatty degeneration. Dyspnœa, cardiac irregularity, palpitation, præcordial distress, vertigo, the so-called pseudo-apoplectic attacks, rapid and weak pulse, etc., are symptoms of dilatation rather than of fatty degeneration. Very rarely anginal attacks may occur without coronary changes. In the late stages Cheyne-Stokes breathing may be present. The physical signs are those of dilatation. Death may occur from sudden over-strain or from slowly developing cardiac insufficiency. In rare cases aneurismal dilatation of the cardiac wall may result from local weakening of the myocardium due to fatty degeneration.

PROGNOSIS.—In the majority of cases of myocardial disease the prognosis is very unfavorable, especially in the slowly developing or chronic form. Acute degeneration of mild degree caused by acute infections or poisoning may recover completely; the more severe forms may be very quickly fatal. Within certain limits chronic fatty change of mild degree does not quickly lead to a fatal issue.

TREATMENT.—The occurrence of fatty degeneration may be many times prevented by careful regulation of the daily life. In suspected cases strict diet should be enforced, the amount and character of muscular activity carefully planned, and the patient protected from excitement of all kinds. Sudden death during sexual intercourse, straining at stool, lifting, running, etc., is extremely common in this affection. Alcohol should be entirely withdrawn, and the amount of fluid limited. When the signs of dilatation have not appeared digitalis is usually not advisable. Strychnine and nitroglycerin may be given. When symptoms of cardiac insufficiency are present the treatment should be carried out along the lines indicated for the treatment of failure of compensation in chronic valvular disease.

Aldred Scott Warthin.

HEART DISEASES: FATTY INFILTRATION.—Fatty infiltration of the heart is that condition in which the subepicardial fat alone is greatly increased, or in connection with this increase there is also an extension of adipose tissue into the myocardium. Normally there should be but a small amount of subepicardial fat and this should be confined to the sulci and grooves along the superficial coronary vessels, being greatest in amount at the base of the heart. Over the greater part of the ventricular surfaces in the active, muscular male of early adult life there should be no fat, but after the age of forty