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

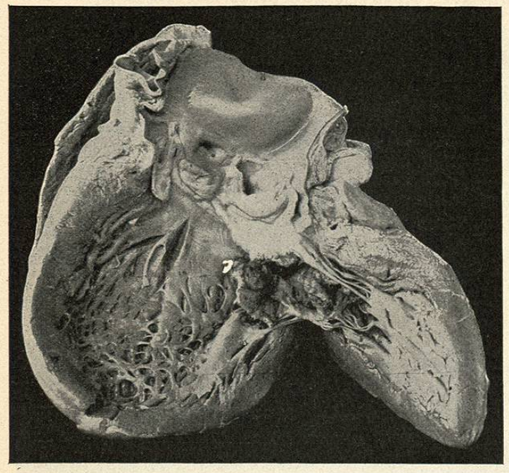

FIG. 2593.—Malignant Endocarditis on Top of Old Sclerosing Process. Marked Involvement of the mitral valves. (Original.)

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

there is a physiological increase in the amount of the cardiac panniculus. There are, however, wide individual variations in the amount of subepicardial fat within apparently normal limits, depending upon intrinsic peculiarities of metabolism, habits of life, etc. The presence of fat cells between the muscle fibres in the interstitial tissue of the myocardium is under all circumstances to be regarded as pathological. Such an encroachment upon the muscle is usually due to a direct extension of adipose tissue from the subepicardial fat, chiefly along the lines of the arterial branches. Secondary to the infiltration of adipose tissue there is an atrophy of the heart muscle. In extreme cases the greater part of the myocardium may be replaced by adipose tissue which may extend even to the endocardium. In some cases the deposit of fat in the cells of the interstitial tissue may be secondary to a primary atrophy or degeneration of the heart muscle, the formation of fatty tissue being of the nature of a compensatory process. Two forms of fatty infiltration may therefore be distinguished clinically: primary and secondary fatty heart. Of these the first variety is by far the most common and important.

In extensive hyperplasia of the subepicardial panniculus the heart is usually increased in size, partly from increased thickness of its wall, and partly from dilatation. The weight of the organ may or may not be above that of the normal. The entire heart may be encased in a thick layer of fat (lipoma capsulare cordis, obesitas cordis, lipomatosis cordis, fatty overgrowth, Mastfettherz, etc.). The most extensive deposit is found at the base of the ventricles along the auriculo-ventricular groove, in some cases having a distinct lobular arrangement. The heart muscle may not be visible through the epicardium at any point. On section through the heart the layer of adipose tissue may be found to form almost the entire thickness of the wall, the muscle consisting of a thin layer beneath the endocardium. In the most extreme cases the fat may reach to the endocardium in the form of trabeculæ extending along the intermuscular septa, gradually diminishing in size as they approach the endocardium. On microscopical examination groups of fat cells are seen between the muscle fibres. The latter may show varying stages of simple or pigment atrophy. Fatty degeneration of the atrophic muscle is not infrequently present.

Fatty infiltration of the heart may exist as a part of a general obesity, either congenital or acquired. Lack of exercise, over-eating, excessive use of carbohydrates, alcohol, etc., as well as excessive water drinking are the most important factors in the production of this condition. It is most common in males at middle age; in women it may exist as a part of general obesity associated with or following the menopause. Deficient oxygenation from anæmia or from chronic pulmonary disease also leads frequently to fatty infiltration of the heart. The condition is often found in the cachexias of chronic tuberculosis, carcinoma, etc. It occurs in the early stage of diabetes, and in certain forms of chronic poisoning, drug habits, etc. (iodoform, morphine, camphor, chloroform, dilute mineral acids, etc.).

A moderate degree of fatty infiltration may exist without signs or symptoms, but beyond a certain limit the over-accumulation of fat beneath the epicardium or its infiltration into the myocardium causes impairment of the cardiac function. In such cases the apex beat is lost, the cardiac impulse is very faint or not present at all, the area of dulness is increased, and the heart sounds are muffled. The pulse is soft, of low tension, often rapid and irregular. The affected individual is stout, with excessive abdominal panniculus, and usually shows signs of general venous congestion. Palpitation, præcordial distress, and dyspnœa are produced by slight exertion, and there is a tendency to excessive sweating. The urine is usually increased in amount, pale and of a low specific gravity. In an individual with general obesity these signs and symptoms may be taken as certain indications of fatty heart. In the cases of primary atrophy of the heart muscle with secondary fatty infiltration the differential diagnosis is not so easily made. In these cases the subcutaneous panniculus may be greatly decreased, and the cardiac symptoms obscured by those of the condition causing the myocardial atrophy. In the later stages of fatty infiltration the symptoms are those of cardiac dilatation, and the condition cannot be diagnosed from other forms of myocardial affections.

The prognosis on the whole is much more favorable than in the case of fatty degeneration. In cases of acquired obesity due to manner of living it is quite good. In congenital obesity it is much less favorable, and when secondary to other disease processes the prognosis rests wholly upon that of the more serious condition. It must be emphasized that the individual with fatty heart cannot meet without danger many conditions involving slight degrees of heart-strain. Intercurrent affections such as influenza, bronchitis, pneumonia, typhoid fever, etc., are very likely to cause death through cardiac insufficiency. Anæsthesia, surgical operations, excessive fatigue, muscular strain, intense emotion, etc., are to be regarded as dangerous factors in the case of individuals affected with fatty infiltration. While sudden death is not infrequent in this condition it is not nearly so common as in the case of fatty degeneration.

In the cases associated with obesity the treatment is very simple and if carefully followed very successful. The administration of drugs is of secondary importance and is usually limited to general tonics and to the improvement of elimination. The essential factors of the treatment are entirely hygienic. The diet should be chiefly nitrogenous, fats and carbohydrates being reduced to the smallest possible proportions. The use of alcohol should be entirely stopped, and but little fluid should be taken with the meals. The quantity of food and drink taken should be most carefully regulated. The amount and character of physical exercise taken are of prime importance and should be most carefully prescribed. The use of certain mineral waters is of great value in many cases as a means of promoting elimination. Turkish baths may be employed toward the same end. In the forms of fatty infiltration due to anæmia, etc., the treatment is carried out along lines depending upon the primary disease.

*Aldred Scott Warthin.*

**HEART DISEASES: FIBROID HEART.**—The terms cardio-sclerosis and chronic fibrous myocarditis are synonyms. There is either a hyperplasia of interstitial connective tissue, or a replacement of heart muscle, more or less extensive, by fibrous tissue.

Etiology.—It is intimately associated in most instances with the process of arterio-sclerosis, especially in its involvement of the coronary arteries. The blocking of one of these arteries by a thrombus or an embolus leads to a condition termed anæmic necrosis, or white infarct. Fibrous myocarditis may result from the gradual transformation of such processes into fibrous tissue. It frequently follows the narrowing of a coronary branch in the process of obliterative endarteritis. According to Broadbent, syphilitic disease of the myocardium and chronic interstitial myocarditis in association with subacute pericarditis and adherent pericardium, are the commonest causes of diffuse interstitial fibrosis. Chronic nephritis, with associated high tension in the arterial system, produces thickening in the walls of the arterioles, and increase of connective tissue in their proximity. Chronic alcoholism may be associated with a diffuse fibroid infiltration. Myocarditic cicatrices may sometimes result from a previous pericarditis or endocarditis, the inflammation extending through to the heart muscle.

Morbid Anatomy.—This sclerosis occurs most frequently at the apex of the left ventricle and in the septum, but may occur in any portion. In the septum there are often streaks of fibroid degeneration which do not reach the endocardium; it may be necessary to divide the muscle in order to see them. This degeneration is the invariable precursor of aneurism of the heart.

Symptoms.—It occurs in men past middle life. The symptoms are extremely unreliable. The so-called clinical symptoms are just as likely not to be present. In rare cases the pulse may be infrequent, even as low as 26; there may be shortness of breath and œdema. In other cases we may find a rapid, irregular, and unequal pulse, with signs of incompensation. Those cases in which coronary arterio-sclerosis is present frequently show symptoms of angina pectoris.

Physical signs are very indefinite. There may be some hypertrophy. Murmurs, systolic in time, may be present during incompensation. The sounds may be weak, irregular, and valvular in character; accentuation of the aortic second, and reduplication of some of the sounds, a diffuse slapping cardiac impulse, and even a gallop rhythm may be present. Post-mortem examination has proven the unreliability of symptoms and physical signs in the diagnosis of this condition, even in the hands of experts. Cabot states that a prominent Boston pathologist told him he had never seen a case of myocarditis correctly diagnosed. So far as symptoms and signs go it is impossible to distinguish the fibroid from the fatty heart. My observation is that physicians usually diagnose fatty heart if the patient is obese, fibroid heart if he is lean.

Course of Disease.—These patients may die suddenly without having previously shown any symptoms; there may be rupture of the heart or a blocking of a coronary artery. Again, they may present symptoms exactly similar to those of an incompensated valvular lesion. Dilatation of the cavities with the development of murmurs of relative insufficiency make it impossible to distinguish it from valvular lesions.

Prognosis.—It is distinctly unfavorable except in syphilitic cases, which may respond promptly to the use of iodides.

Treatment.—Appreciating the uncertainty of a diagnosis, our treatment of suspected cases must be based upon the broad lines of common sense and must depend mostly upon indications drawn from the symptoms and signs of weak heart. If symptoms of incompensation are present, the patient must be put to bed and placed upon digitalis and strychnine, with a light, nutritious diet. As routine measures these patients should observe moderation in food and drink, and avoid variations in blood pressure by a very even life; the bowels should be kept open with mild mercurials and saline purgatives. Strychnine, gr. $\frac{1}{30}$, every six hours, or in the form of nux vomica, gtt. xx. three or four times daily, is useful. If arterial tension is high, nitroglycerin is indicated.

*James Rae Arneill.*

**HEART DISEASES: HYPERTROPHY AND DILATATION.**—A discussion of these pathological conditions under a separate heading necessitates more or less repetition of facts stated in the articles on valvular and myocardial diseases.

These conditions usually go hand-in-hand; at times the hypertrophy exceeds the dilatation, at other times the dilatation exceeds the hypertrophy. It is very unusual to find a pure hypertrophy or a pure dilatation.

*Hypertrophy* is almost invariably a conservative process; *dilatation* on the other hand, though at times conservative, as in aortic insufficiency, is generally destructive. Both conditions may affect the whole heart, but are more likely to be limited to one part. We may even have hypertrophy limited to one papillary muscle and dilatation to a conus arteriosus.

*Hypertrophy* means an increase in the mass of muscular tissue, usually associated with an increase in volume. *Dilatation* means an enlargement of the cavities of the heart. The same laws which affect the development of muscular tissue in other parts of the body apply to the heart. Under conditions of proper nutrition, increased work causes an enlargement of the cardiac muscle. Ziegler says this increase in size takes place by enlargement of the muscle cells. Whether or not a proliferation of the cells also is present is not yet positively known. Some authorities believe there is a definite increase in the number of muscle cells.

Schroetter divides hypertrophy into three classes:

I. Simple hypertrophy,—increase of the heart in volume with normal cavities.

II. Concentric hypertrophy,—thickening of the walls with diminution in the size of cavities.

III. Excentric hypertrophy,—increase in muscular tissue, with enlargement of cavities.

Of these three classes only the third, excentric hypertrophy, is of special interest to us. The first two divisions are comparatively rare.

If work above its normal capacity is thrown upon any part of the heart, hypertrophy results. This is brought about by the presence of unusual obstruction to the flow of blood, and by excessive stimulation of the nervous mechanism of the heart.

Let us now consider in some detail the different causes which produce hypertrophy of special portions of the heart:

*Left Ventricle.*—The following are the conditions under which hypertrophy of this part takes place:

(*a*) Valvular lesions: Aortic insufficiency, aortic stenosis, mitral insufficiency; (*b*) the various conditions which cause increased resistance in the greater circulation, namely: aneurism, congenital narrowing of the aorta (rare), small arterial system (rare), arterio-sclerosis (very common) in which there may be both diminution in the calibre of the artery and loss of elasticity. The elasticity of the arterial wall is a powerful aid in propelling the blood current forward. Various intoxications cause a contraction of the peripheral circulation and thus raise blood pressure. Chief among these are the various forms of Bright's disease; also gout and lead poisoning. With these diseases, especially chronic interstitial nephritis, we frequently find a high grade of arterio-sclerosis. The entire heart may become involved in this hypertrophy. At present I have under observation a middle-aged patient who shows considerable cardiac hypertrophy, the apex being in the anterior axillary line. Valvular lesions have never been demonstrated in this case; there is, however, considerable arterio-sclerosis.

The excessive use of tea, coffee, alcohol, and tobacco, through their exciting action on the nervous mechanism of the heart, may produce slight hypertrophy. Exophthalmic goitre and various nervous conditions, such as neurasthenia, if of sufficient duration, may cause hypertrophy. But valvular lesions are a frequent complication of Graves' disease.

(*c*) Pathological conditions in the heart wall, such as chronic myocarditis and adherent pericardium, give rise to hypertrophy, because of the obstruction offered to the orderly and regular contractions of the heart.

*Right Ventricle.*—It is hypertrophied under the following conditions:

(*a*) Valvular lesions; most frequently, mitral insufficiency, and stenosis; the very rare lesions of the right heart, pulmonary stenosis and insufficiency, tricuspid stenosis and insufficiency. Relative tricuspid insufficiency secondary to left heart lesions is rather frequent.

(*b*) Increased resistance in the pulmonary circulation, due to narrowing of the calibre of the pulmonary artery by pressure from without, as by aneurism, tumors, and so forth; congenital narrowing and arterio-sclerosis of the pulmonary artery (rare); also the cutting off of capillary regions in the lungs by pleurisy, pneumothorax if of sufficient duration, chronic fibroid conditions, tumors, chronic infiltrations with or without cavities, *emphysema*, spinal curvature with dislocation of the heart.

*Auricles.*—Hypertrophy of these chambers is much rarer than dilatation, and is usually associated with a much greater degree of dilatation. The left auricle hypertrophies more than the right.

Hypertrophy of the whole heart takes place when there is a combination of several causes, for instance, aortic and mitral insufficiencies, combined with a relative tricuspid insufficiency; of valvular disease complicated by lung or arterial disease.

*Athletic Heart.*—Allbutt and Stengel have made special studies of the athletic heart among the students of Cam-

bridge and the University of Pennsylvania. They have demonstrated by means of percussion and location of the apex, that several years of the severer forms of athletics, such as distance running, rowing and football, will increase the size of the heart considerably. In these cases the valves are healthy. It is true that in some instances a so-called athlete's heart is one enlarged by aortic or mitral insufficiency. Seitz in Germany, and Da Costa in this country, have called our attention to a very similar condition in soldiers who have undergone severe strain from long marches.

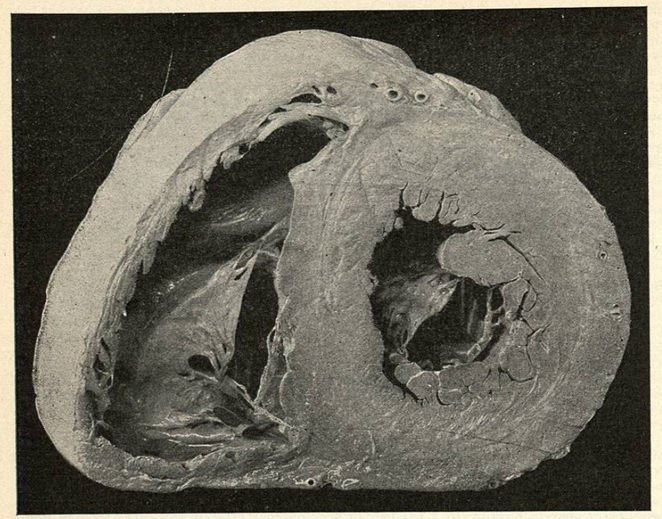

FIG. 2594.—Hypertrophy of the Left Ventricle and Dilatation of the Right. (Original.)

*Morbid Anatomy.*—The size of the average normal heart is about equal to the patient's closed right fist. The heart of the adult male weighs about nine ounces; of the female eight ounces. The majority of hypertrophied hearts weigh from sixteen to twenty ounces. Hearts of enormous size have been reported; Beverley Robinson has reported a heart weighing fifty-three ounces, Alonzo Clark one weighing fifty-seven ounces, and Stokes one weighing sixty-four ounces. Another good method of determining the extent of hypertrophy is by measuring the thickness of the wall after the heart has been thoroughly relaxed by soaking in water. The left ventricle normally has a thickness of 9 to 12 mm.; so a thickness of 20 to 25 mm. indicates hypertrophy. The right ventricle is thinner than the left, measuring on an average 4 to 7 mm. When hypertrophied it measures from 13 to 20 mm. The left auricle has a normal thickness of about 3 mm.; the right scarcely 2 mm. The auricles may be doubled in thickness when hypertrophied.

The conical shape of the heart is lost in hypertrophy and dilatation, by reason of the apex being greatly broadened and rounded. When the right ventricle is chiefly affected it occupies most of the apex. The hypertrophied muscle is deep red in color and firmer than the normal heart.

*Dilatation.*—Schroetter divides it into three classes:

I. Simple dilatation,—enlargement of cavities with normal thickness of the walls.

II. Active dilatation,—enlargement of the cavities of the heart with increased thickness of the walls, *i.e.*, excentric hypertrophy.

III. Passive dilatation,—enlargement of the cavities with thinning of their walls.

Active and passive dilatation are of special interest. The former has been discussed under its synonym, excentric hypertrophy.

*Dilatation* is always a secondary process and is due either to (*a*) increase of pressure within a cavity whose walls are not hypertrophied, or (*b*) normal pressure

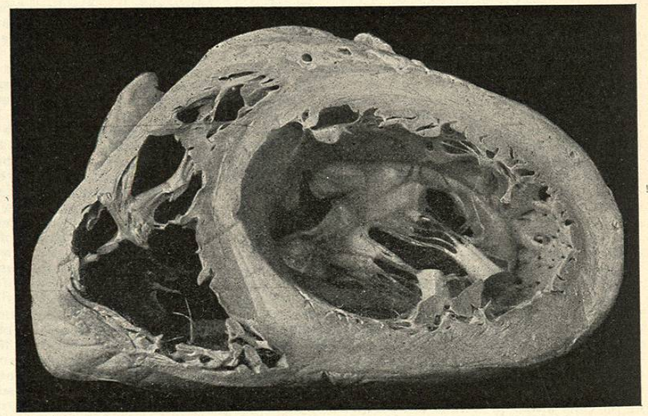

FIG. 2595.—Dilatation Involving Both Sides of the Heart. (Original.)





within cavities whose walls have been weakened by myocardial disease. Aortic insufficiency offers the best illustration of the first class. In it the dilatation of the left ventricle is due to the chamber being filled from two sides at once, the blood from the left auricle and that which regurgitates from the aorta. As a result of the increased pressure the ventricle yields. Soon hypertrophy develops, producing a typical example of excentric hypertrophy. Whether dilatation or hypertrophy will prevail depends on the nature and quality of the heart tissue.

When the capillary circulation of the lungs is overdistended, as it may be in mitral insufficiency and stenosis, the right heart meets an unusual resistance in discharging its blood into the pulmonary artery. This back pressure, together with the pressure of the blood which normally enters the ventricle from the right auricle, causes dilatation, and just so much sooner on this side than on the other, as the right ventricle is weaker than the left.

Pulmonary insufficiency and tricuspid insufficiency also cause dilatation of the right ventricle. Diseases of the lungs and pleura, especially if associated with myocardiac disease, as in lobar pneumonia, may cause a fatal dilatation of the right heart. Emphysema is usually associated with dilatation of the right ventricle.

The auricles are prone to extensive dilatation, not having muscular tissue enough for the development of much hypertrophy. The left auricle dilates in mitral insufficiency and stenosis, the right in tricuspid insufficiency and stenosis. The development of relative insufficiency of the auriculo-ventricular valves from dilatation of the ventricles, is a frequent cause of dilatation of the auricles. When incompensation develops in the course of the various heart lesions, marked dilatation of all the cavities is likely to follow.

*Morbid Anatomy.*—The cavities may be vastly increased in size, even trebled in capacity. The auricles may contain from eighteen to twenty ounces of blood. The auriculo-ventricular rings may be greatly dilated. The mitral orifice, which normally has a circumference of about three and one-half inches, may be increased to five and one-half inches and more; the tricuspid orifice normally has a circumference of about four and one-half inches and may be increased to six inches. The bundles of muscle fibres may be greatly thinned and separated. In Figs. 2594 and 2595 there was marked separation of the bundles of muscle with only a thin semitransparent membrane between; the endocardium and epicardium were in places in apposition.

SYMPTOMS AND PHYSICAL SIGNS.—Very little space need be devoted to them as they have been considered in detail under valvular lesions of the heart.

The so-called idiopathic hypertrophy produces a strong first sound at the apex and an accentuated aortic second, unaccompanied by murmurs. Hypertrophy of the right ventricle, due to disease in the lungs and pleura, produces a strong first sound in the tricuspid area with an accentuated pulmonary second. The apex beat is heaving and dislocated to the left, and usually downward. The pulse is likely to be much stronger than in the hypertrophy due to valvular lesions. The forcible heart action may or may not be perceptible to the individual.

In dilatation the apex beat loses its heaving character, is diffuse, undulatory and irregular, and taps against the chest rather than thrusts; dulness increases, especially to right and left. The heart sounds become weak, the first approaching the second in its valvular character; if murmurs are present they become weak, or even may disappear; fatal gallop rhythm may develop. The pulse becomes small, irregular, and unequal. All of the classical symptoms of incompensation may develop, such as cyanosis, dyspnœa, œdema, bronchitis, gastro-intestinal disturbances, and marked nervousness.

The treatment of these conditions has been discussed in detail in the article devoted to valvular lesions of the heart.

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**HEART DISEASES: MYOCARDITIS.**—Under this heading I shall discuss (1) Acute Interstitial Myocarditis, (2) Parenchymatous Degeneration of the Myocardium, (3) Segmentation and Fragmentation, (4) Abscess of the Heart.

I. *Acute Interstitial Myocarditis.*—This is a disease in which the intermuscular connective tissue is swollen and infiltrated with small round cells and leucocytes; the blood-vessels are dilated and the muscle fibres the seat of granular, fatty, and hyaline degeneration. Acute endocarditis and pericarditis, diphtheria and typhoid fever are most commonly associated with it. Councilman has described a very similar condition as a sequel of gonorrhœa and was able to demonstrate gonococci in the diseased areas.

II. *Parenchymatous Degeneration.*—This is extremely common because of its intimate association with the numerous acute infectious diseases and toxæmias. It varies much in degree, in most cases being mild. In practically all of the continued fevers the myocardium is affected, but not so much from the fever as from the associated toxæmia. It may develop in the course of a pericarditis or an endocarditis.

The heart is pale, turbid, and extremely soft. Microscopically there is granular degeneration of the muscle fibres. They may even lose their striæ. The sudden deaths which occasionally occur during the convalescence of diphtheria, pneumonia, typhoid fever and influenza, are undoubtedly dependent on the presence of a severe grade of parenchymatous degeneration. Acute dilatation of the heart develops very readily under these circumstances and explains the fatal termination.

The physical signs are those of cardiac weakness. The pulse is feeble and may be both irregular and unequal; the first sound at the apex becomes weaker and more valvular in character. Soft blowing systolic murmurs may develop especially in the pulmonary regions. The patient is weak and becomes short of breath on slight exertion.

III. *Fragmentation and Segmentation.*—These conditions of the heart muscle interest the pathologist rather than the clinician. Renaut and Landouzy first called direct attention to the dissociation of the muscle cells in 1877. Renaut has gone so far as to consider this condition a distinct disease from the clinician's standpoint. However, it seems to us impossible to attempt to distinguish with any degree of certainty this from numerous other diseases of the myocardium, by means of signs and symptoms. In segmentation there is a separation of fibres at the cement line. In fragmentation the fracture is across the fibre and perhaps at the level of the nucleus; longitudinal division is very rare. Such hearts are soft and easily torn, and are often pale and cloudy. Renaut attributes the dissociation of cells to solution of the cement substance, on account of senile involutionary changes, alcoholism, infection, and the associated high temperature. Hektoen says "of the morbid changes of the myocardium proper, none occurs more frequently or under a greater variety of circumstances, than the lesions known as segmentation and fragmentation of the muscle fibres. They are due to a disproportion between the vigor and order of muscular contraction and muscular cohesion."

IV. *Abscess of the Heart.*—This develops in the course of a pyæmia or infective endocarditis. It is a metastatic pyæmic process localized in the heart. Infected emboli are carried into the coronary circulation with a resulting inflammation and suppuration. These abscesses vary much in size, from very small to fairly large.

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**HEART DISEASES: NEOPLASMS.**—Primary tumors of the heart are very rare. They may arise in the myocardium, or in the peri- and endocardium, from which points they may invade the heart muscle. They may also take their origin from the valves. They may be single or multiple. They may form nodular circumscribed masses in the heart wall, or project as polypoid growths