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.

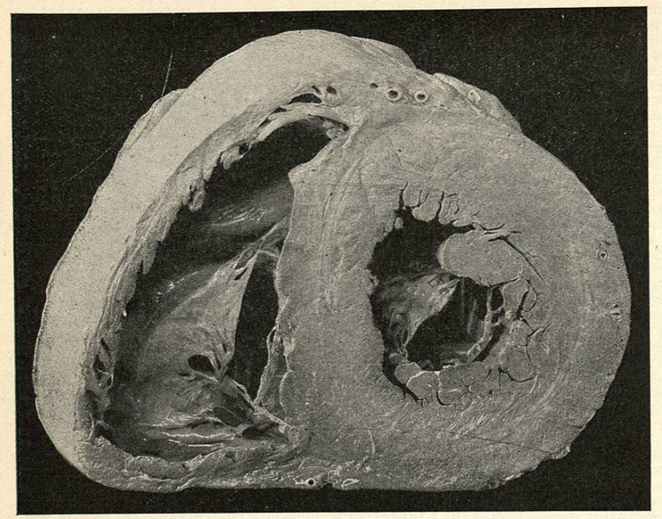

FIG. 2594.—Hypertrophy of the Left Ventricle and Dilatation of the Right. (Original.)

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

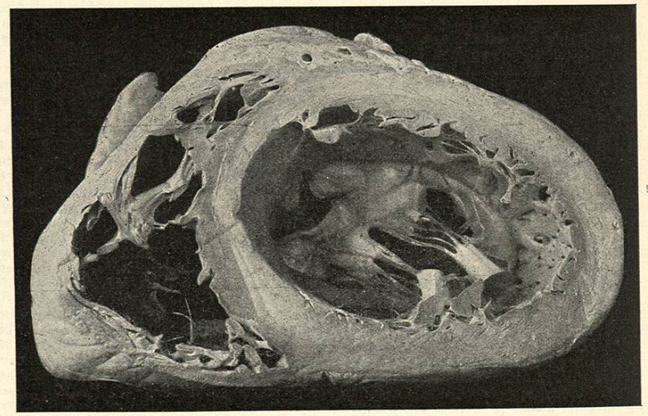

FIG. 2595.—Dilatation Involving Both Sides of the Heart. (Original.)

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.

James Rae Arneill.

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.

James Rae Arneill.

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

into the heart cavity. Only rarely do they form diffusely infiltrating growths. Many are of congenital origin. The majority of cases occur in young or middle-aged individuals; a few have been found in old age.

The *myxoma* is the most frequent variety of primary neoplasm of the heart, eighteen cases having been observed. Of these the majority were pure myxomata, the others fibromyxomata. In one case the growth was described as an angiectatic myxoma. As a rule they form polypoid growths projecting into the heart cavities. It is very doubtful, however, if some of the polypoid formations found on the endocardium were really of the nature of neoplasms; rather is it probable that they were masses of newly formed connective tissue of myxomatous type resulting from the organization of thrombi. In a case of fibromyxoma of the left auricle occurring in a girl of four years, Jacobsthal found large numbers of newly developed fibres of yellow elastic tissue. In the majority of cases the myxomatous tumor has been found in the left auricle, very often being attached to the endocardium by a slender pedicle. Papillary myxomata have also been found upon the tricuspid flaps.

Next in order of frequency of the primary cardiac neoplasms is the *rhabdomyoma*. Its peculiar histological structure gives to it a great interest. Cases have been described by von Recklinghausen, Virchow, Hlava, Kolisko, Cesaris-Demel, and Seiffert. The tumor is of congenital origin, and is usually multiple. The nodules are not encapsulated, but are well outlined from the cardiac muscle, and quite sharply demarcated from the pericardium and endocardium. The growths may be as large as walnuts, and are scattered through the ventricular walls and the interventricular septum. On section the nodules are reddish-gray in color. The microscopical appearances are very striking. Trabeculæ of varying thickness showing transverse striations are arranged so as to form small spaces. These spaces do not communicate with each other; they contain large polygonal cells of irregular outline having one or more oval nuclei which are centrally situated. Their photoplasm shows a faint striation. In some cases the cell appears to form part of the wall of the space and sends prolongations across the latter. Both the protoplasm and prolongations of these cells stain like muscle. The spaces and their contents have received very different interpretations from the different observers who have studied this tumor. Hlava regarded them as intracellular, Kolisko as intercellular. According to Seiffert each space and its wall represents a greatly enlarged embryonal heart-muscle cell. The smallest tumor nodules may consist of bundles of delicately striated fibres only, without the peculiar spaces. Of the significance and mode of formation of the cardiac rhabdomyoma we as yet have no satisfactory explanation.

Primary *sarcoma* of the heart is much more rare; and it is very probable that some of the cases diagnosed as such were secondary to some undiscovered focus elsewhere in the body. The reported cases have been in both young and old individuals. The varieties observed were round and spindle-cell forms, myxosarcoma, fibrosarcoma, and angiosarcoma.

Cases of primary *fibroma* of the heart have also been reported, but the true nature of these cases is uncertain. It is quite probable that they represent organized thrombi. Observations have also been made of the primary occurrence of *lymphadenoma*, *cavernous angioma*, and *lipoma* in the heart. The polypoid *cysts* which have been described by a number of writers as primary cardiac neoplasms are without doubt to be regarded as degenerating or partly organized polypoid thrombi. They are found most frequently in the left auricle, are reddish-gray in color, and have a smooth shining surface. On section they show a cystic space filled with the products of the simple softening of the central portion of the thrombus. When organization of the periphery of the thrombus has begun the resemblance to a true cyst is very close.

Primary neoplasms of the valves are more rare than those of the heart wall. A *papillary myxoma* of the posterior tricuspid flap, *multiple myxomata* of the size of peas of the same valve, and *spindle-cell sarcoma* of the pulmonary valves have been reported.

Secondary neoplasms of the heart are also rare but much more frequent than the primary. Secondary *carcinoma* is the most common tumor of this organ. Primary cardiac cancer is of course an impossibility, though in the older literature descriptions of growths regarded as such are found. These cases were either cancer-secondaries, or, as was not infrequently the case, sarcomata were diagnosed as carcinomata. Secondary carcinoma of the heart may arise through metastasis, or by extension through continuity or contiguity through the pericardium from carcinomatous growths in the lungs, œsophagus, or secondary deposits in the mediastinal lymph glands. Metastatic carcinoma of the heart may arise from emboli of cancer cells in the coronary vessels, on the mural endocardium, between the papiliary muscles, and on the valves. From a secondary focus thus formed further extension may take place through the cardiac lymph channels. In this manner diffuse carcinomatous infiltration of the entire cardiac wall may take place. In other cases multiple metastases in the heart may be found. These may be found in the walls of both auricles and ventricles. There does not appear to be any favorite seat of deposit. In the majority of cases the carcinoma is of the medullary type. In the event of carcinomata breaking into the vena cava large emboli of carcinomatous tissue may be carried to the right heart where they may become attached to the endocardium or develop as free carcinomatous masses.

Secondary *sarcoma* of the heart, while less common than secondary carcinoma, has been observed by a number of writers. The growths are usually metastatic, but the heart may be involved also by extension through the pericardium from primary mediastinal sarcomata. The tumors may be single or multiple. They may be nodular, diffusely infiltrating, or in the form of flattened plate-like growths more or less sharply outlined. Free sarcomatous masses may be found in the right heart in cases in which the primary tumor has invaded a large vein. In a case described by Osler of sudden death in a child the tricuspid orifice was found blocked by a sarcomatous mass coming from the renal vein. In a case of primary sarcoma of the nose reported by the writer the entire heart wall was found to be infiltrated with sarcoma, while in the right ventricle there was a free sarcomatous mass of the size of a walnut.

Small tumors of the heart may produce no symptoms; larger growths may lead to cardiac hypertrophy, dilatation, valvular stenosis or insufficiency, rupture of the heart wall, secondary embolism, etc. An absolute diagnosis in the majority of cases is impossible. In suspected cases the Roentgen ray might reveal changes of size or form in the heart. If the pericardium is involved and a pericardial exudate present, some of the latter should be withdrawn for examination. The presence of a hemorrhagic exudate would be regarded as favoring the presence of a malignant tumor. Of greater diagnostic significance would be the character of the cells found in the exudate. The presence of numerous mitoses in these, particularly atypical mitotic figures, could be taken as almost positive evidence of the presence of malignancy. The treatment is wholly symptomatic.

Aldred Scott Warthin.

HEART DISEASES: NEUROSES.—The Nervous Mechanism of the Heart.—It has become known to physiologists that the power of pulsation of the heart is not engendered in, or derived from, the nervous system, but rests in the musculature of the organ itself. In the frog and other cold-blooded animals the heart continues for a short time to pulsate with a true rhythm after removal from the body and complete separation from nervous or other connection. The same is true in a minor degree in the higher animals, and while not actually verified by observation, will undoubtedly hold good in the case of man himself. Although striated the cardiac muscular structure is incompletely differentiated, and thus retains the power of spontaneous movement common to all primordial protoplasm. The nervous system, however, exercises a supreme influence over the cardiac movements. Two important sets of nerves derived from the cerebro-spinal and the sympathetic systems are concerned in the regulation and control of the circulatory centre. The scope of this article does not contemplate a complete and detailed description of the nervous supply of the heart and the phenomena of its mechanism. It is sufficient for our purpose to call attention in this place to the major facts relating to the functions of the two kinds of cardiac nerves which have been well established by physiological research.

1. Those derived from the vagus which are chiefly inhibitory or slowing to the action of the heart when subjected to a stimulation of either a direct or a reflex character. The vagus is thus known as the anabolic nerve of the heart. The pneumogastric trunk also conveys the depressor cardiac nerve which enables it to govern and regulate the arterioles in accordance with the demands of the heart.

2. The accelerator, or more properly the augmentor, nerves of the heart, derived chiefly from the first, second, and third cervical ganglia of the sympathetic system. The function of this cord, known also as the *katabolic* nerve of the heart, is to accelerate the frequency of the cardiac contractions and at the same time to augment their force. But when the question is asked, by virtue of what events produced in the heart itself do the impulses of one kind bring about inhibition and those of another augmentation of the impulses, we are met with difficulty. We may speak if we choose of an inhibitory mechanism placed within the cardiac structure, but we have no exact knowledge of the nature of such a mechanism, still less of an augmentor mechanism (Foster). It has been suggested that some of the intracardiac ganglia may serve as a medium for the distribution of these inhibitory or augmentor influences, but the evidence goes to show that the inhibitory impulses produce their effect by acting directly on the muscular fibres themselves. Nor are we in a position at the present time to assign the proper function of these ganglia, although it is believed that their influence is chiefly of a trophic or nutritional character. But, while the nervous system neither initiates nor controls the rhythmic movements of the heart, this function at once goes astray if removed from nervous governance. It is by the nervous system that the heart-beats and the calibre of the small arteries are brought into relation with each other and with almost every part of the body. It is by the nervous system acting either on the heart or on the small arteries that a change of circumstances affecting either the whole or a part of the body is met by compensatory or regulating changes in the flow of blood; that an organ has a more full supply of blood when at work than when at rest; that the tide of blood through the skin rises and ebbs with the rise and fall of the temperature of the air; that the work of the heart is tempered to meet the strain of overfed arteries, and that the arterial gates open and shut as the force of the central pump waxes and wanes. We have thus learned the seat of the automatic and rhythmic action of the heart, and are able to descry the part played by the nervous system in controlling the force, frequency, and rhythm of the muscular movements, and the tonicity or laxity of the vascular walls. But this knowledge does not aid us greatly in a definite appreciation of the pathogeny of the various disordered states of the organ which we designate as neuroses, nor does it quicken to any great extent our perception of the clinical phenomena, or our knowledge as to the therapeutic management of these conditions. The physiological relations existing between the vagus and sympathetic supplying cords on the one hand and the intracardiac ganglia and muscular structure on the other have not been sufficiently defined to enable us to agree upon any fixed or definite anatomical, pathological, or even clinical basis in our attempts to explain the cardiac neuroses.

Nomenclature of the Cardiac Neuroses.—Until a very recent period the term palpitation was made to cover practically all of the nervous disturbances of the heart. With increased experience and additions to our clinical knowledge this restrictive appellation no longer serves its former purpose. It would appear indeed that we are in danger of going too far in the other direction. During the last decade or two there has developed a tendency to an embarrassing multiplication of terms in labelling the functional derangements of the heart. Furthermore, no two writers appear to agree exactly in classifying these disorders. It thus transpires that the nomenclature of the subject is at the present time in a most bewildering, it might almost be said chaotic, state. This is perhaps to some extent inevitable. Some of the conditions which we find it convenient to describe under distinctive titles so shade into each other that it is difficult to decide where one begins and another ends. For example, the condition described as palpitation may present at times the symptoms of neurasthenia cordis, or of arhythmia; while in tachycardia, the features of palpitation, or even of pseudo-angina, may obtrude themselves. Some of the terms employed do not constitute true symptom groups—they are mere symptoms themselves and not distinct diseases.

After much anxious thought and study of this subject, it has seemed to the author proper to present an account of these nervous manifestations, each under the head of the most prominent clinical features exhibited, noting the points of contact with other neurotic manifestations and such additional facts as our present state of knowledge would appear to warrant. It must be said that no perfectly satisfactory clinical classification of these disorders is possible until further light has been shed upon the physiological inter-relations existing between the supplying nerves, the cardiac ganglia, and the muscular structure of the organ itself. It has seemed to the writer, however, that all we know at the present time concerning the clinical and pathological features of the cardiac neuroses may be properly included under the following heads: (1) Palpitation; (2) tachycardia; (3) bradycardia, (4) the dyspeptic heart; (5) neurasthenia cordis; (6) the irritable heart; (7) arhythmia, including delirium cordis and tremor cordis; (8) the heart in Graves' disease; (9) angina pectoris and pseudo-angina. Of these terms it may be said that palpitation, tachycardia, neurasthenia, arhythmia, and angina are necessary and essential; the remainder might properly be omitted, but they are found in contemporaneous literature and may for the present be used provisionally as marking points of which we have no exact scientific conception.

Palpitation.—According to Balfour all who suffer, or think they suffer, from disease of the heart are prone to palpitation. Under Laënnec's definition, any person whose heart's action obtruded itself upon the consciousness might be said to have cardiac palpitation. In accordance with the modern terminology, however, the name is used to cover a much more limited class of cases. One by one symptoms formerly referred to as falling under this term have been relegated to other forms of disorder, until at the present time palpitation has become quite unfashionable. Several writers mention it only as a symptom of other neuroses. In the author's opinion, however, it may still be regarded as a substantive affection covering a distinct symptom group.

Symptoms.—Probably some palpitation of the heart falls within the experience of every one. Being very near to our consciousness, eccentricities of the heart are usually attended by great perturbation of the nervous system. The attacks usually come on suddenly, independently of any mental or physical effort, and vary in severity from a mere sense of discomfort or uneasiness to a high degree of pain and distress. Some persons experience darting pains through the heart during the attacks, especially on attempting to take a deep inspiration. It is often brought on by a full meal and the belching of gas gives relief. The heart appears, as variously described by the patient, to jump, to roll, to throb,