f. mik. Anat., Bd. 33, and Röse, *Morph. Jahrbuch*, Bd. 15 and 16).

The opening of the vein is guarded by two folds of the walls of the heart, which really form a complete bicuspid valve (Fig. 2571). An additional fold extends to this from in front, the sectum spurium, *S.sp.* Later on, the orifice enlarges to incorporate a portion of the veins into the auricle, but the right lip of the opening remains permanently as the Eustachian valve. The pulmonary veins are formed later. They grow from the lungs to the left auricle.

Returning to a stage of heart as represented in Fig. 2571, we notice that the common ventricle shows a constriction on the outside which corresponds to a septum forming on the inside. As this septum from the inside, the septum inferius, grows toward the S. intermedium, it must also grow toward the aorta to join a septum aorticum which is forming there. Figs. 2575–2577, show how this is brought about. Fig. 2575 represents a stage in which the auricles empty wholly on the left side and the aorta arises wholly on the right. As the inferior septum arises the auriculo-ventricular opening moves, as it were, toward the aorta; and when the septum inferius unites with the S. intermedium it cuts the auriculo-ventricular opening in such a manner that the blood coming from the right auricle passes to the right ventricle, and the blood from the left auricle to the left ventricle. A small opening exists for quite a while between the two ventricles, but, later on, this is closed by a membrane of connective tissue. Before the ventricles are completely separated from each other, a view of the septum appears much as is shown in Fig. 2578. The aorta is already completely separated into two vessels: the one which communicates with the right ventricle, communicating above with the fifth aortic arch, and the one from the left with the fourth. The fourth is destined to become the aorta, and the fifth the ductus arteriosus from which the pulmonary artery arises.

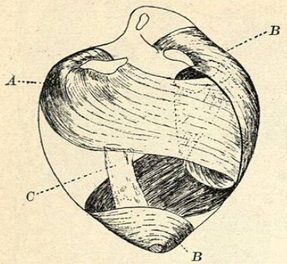

FIG. 2582.—Diagram of the Course of the Superficial Muscle Layers Originating in the Right and Left Auriculo-ventricular Rings and in the Posterior Half of the Tendon of the Conus. (After MacCallum.) *C*, Anterior papillary muscle.

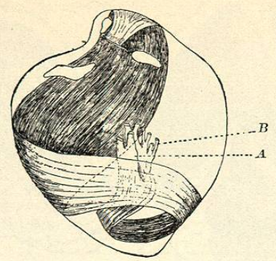

FIG. 2583.—Diagram of the Course of the Superficial Muscle Layers Originating in the Anterior Half of the Tendon of the Conus. (After MacCallum.) *A*, Posterior papillary muscle; *B*, papillary muscle of the septum.

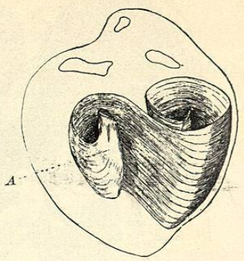

FIG. 2584.—Diagram of the Course of the Deepest Layer of Muscle of the Left Ventricle. (After MacCallum.) *A*, Posterior papillary muscle.

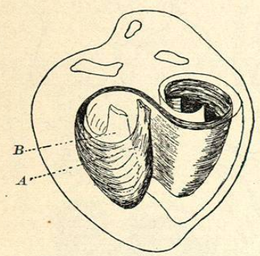

FIG. 2585.—Diagram of the Course of the Layer Superficial to that Shown in Fig. 2584. The outline of the deep layer is also shown. (After MacCallum.) *A*, Posterior papillary muscle; *B*, papillary muscle of the septum.

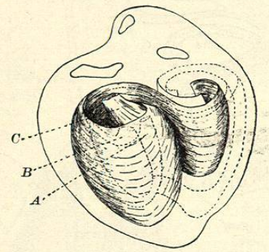

FIG. 2586.—Diagram of a Layer Still more Superficial to that Shown in Fig. 2585, and Ending in the Anterior Papillary Muscle. The other layers are represented in dotted lines. (After MacCallum.) *A*, Posterior papillary muscle; *B*, papillary muscle of septum; *C*, anterior papillary muscle.

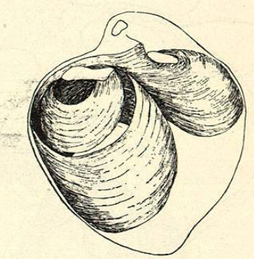

FIG. 2587.—Diagram of the Muscle Originating in the Tendon of the Conus and Upper Part of the Right Ventricle. (After MacCallum.) This layer surrounds the layers represented in the left ventricle in Figs. 2584, 2585, and 2586.

The muscle walls of the ventricles are at first of the same thickness, but, later on, that of the left becomes heavier than that of the right. The spongy layer in the ventricle is covered with endocardium, and from this layer the columnæ carneæ arise.

At an early stage the tissue of the ventricle walls becomes spongy on the inside. These meshes and columns play a very important part in the formation of the valves, muscle columns, and the tendons to the valves. We have seen that the septum intermedium is formed in the auriculo-ventricular canal and divides it into a right and a left half. When this is complete a section of the heart gives a picture as shown in Fig. 2579. At the same time a portion of the auricle is, as it were, pushed into the ventricle, as the figure shows. This invagination takes with it a portion of the epicardium, to be incorporated in the formation of the valve. Connective tissue is incorporated in the formation of the valve at its beginning, although the great bulk of the valve is at this time composed of muscular fibres. The burrowing of the ventricle walls includes also the valves, and in this way, as the figure indicates, the primitive chordæ tendineæ are formed. Soon, however, the bands lose their muscle fibres, in the portions which form the chordæ, and in other portions, farther away from the valve, the muscle fibres increase to form the papillary muscle. The valves have therefore a double origin: (1) Peripheral, and (2)

from the septum intermedium. According to His, the tendons attached to the valves arising from the periphery, have their permanent attachment to the valve from the beginning, while a portion of those passing to the valve and arising from the septum intermedium may possibly shift their valvular attachment.

Recently MacCallum has made extensive studies on the development of the heart muscle cells as well as of the arrangement of the fibre bundles in the hearts of embryos.* MacCallum has found that the heart muscle cell in its development undergoes certain changes by which younger and newly developed cells can be distinguished from fully developed cells. The difference between the older and younger cells is well shown in Fig. 2580, taken from the heart of a very young pig's embryo. It is seen that the younger cells are immediately under the endocardium and that the older cells have been pushed toward the outside and lie immediately below the pericardium. In the younger cells the nuclei multiply by indirect cell division and the distribution of such cells is shown by the letter *A* in Fig. 2581. Here again the growing point of the muscle of the heart is found to be immediately below the endocardium. It appears then that the growth of the heart after the organ is well formed is on the inside immediately below the endocardium. In some way, not yet definitely known, the heart is then gradually rolled up, becoming more and more complex in the higher animals. MacCallum has succeeded in unravelling the musculature in the embryo pig and the human fœtus before the fibres have become interlaced too much. According to him, the fibre bundles can be divided into four groups: (1) Those arising from the auriculo-ventricular ring on one side and passing to the papillary muscle of the opposite side (Figs. 2582 and 2583); (2) those bundles arising from the papillary muscles on one side and passing to the papillary muscles on the opposite side (Figs. 2584, 2585, and 2586); (3) those fibres arising from the auriculo-ventricular ring on one side and passing to the auriculo-ventricular ring of the opposite side (Fig. 2587); and (4) those fibres which simply encircle the left auriculo-ventricular ring and the aorta (Fig. 2588).

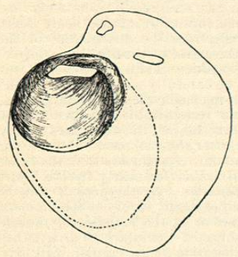

FIG. 2588.—Diagram of the Layer of Muscle Surrounding the Aorta, and Left Auriculo-ventricular Ring. (After MacCallum.) Fig. 2587 is represented in dotted lines.

According to MacCallum, the points to be emphasized in his work are as follows:

The heart consists of several layers of muscle, the course of which is shown in some detail in Figs. 2582–2588. Nearly all begin in the auriculo-ventricular ring of one ventricle and end in the papillary muscle of the other. Those fibres which begin near the outside of one ventricle end near the inside of the other ventricle.

The thin superficial muscle being removed, the left ventricle can be unrolled so that its cavity and papillary muscle are exposed. This shows it to be a flat band of muscle continuous with the muscle fibres which cross over in the septum from the right ventricle.

Grouping these layers together, it is plain that the heart in the embryo is a scroll-shaped band of muscle with tendons at each end. As it grows older the layer of muscle passing over in the septum from the right to the left ventricle remains comparatively thin, while the ventricular cells increase greatly in thickness. The place of most active cell growth is near the inside of the ventricular walls, and they are, therefore, at the two ends of the band of muscles making up the heart.

Franklin P. Mall.

HEART DISEASES: ACTINOMYCOSIS.—Actinomycosis of the heart has been observed a number of times within recent years, and it is very probable that as the reported cases of infection with actinomyces multiply this organ will be found to be not infrequently affected. Since the respiratory tract appears to be one of the most common avenues of entrance of this parasite, secondary involvement of the mediastinum is of frequent occurrence, whereby extension to the pericardium is very likely to take place. Besides infection by direct extension from the mediastinum hæmatogenous metastases in the heart wall may occur, or the heart may be primarily affected, the avenue of entrance being unknown. Paltauf found extensive actinomycosis of the wall of the right ventricle in a tuberculous subject, the condition having been diagnosed as tuberculous pericarditis. In cases observed by Israel, Ponfick, and others, metastases were found in the heart arising from primary foci in the liver, lungs, and other parts of the body (pyæmic actinomycosis). Occasionally a general infection of the body, acute or chronic, occurs, and the heart is involved in common with other organs. Actinomycosis of the heart presents no definite clinical picture. The diagnosis of the condition will rest entirely upon the demonstration of the presence of the parasite within the body. The treatment should be directed along general lines, with the internal use of potassium iodide, which in some cases has appeared to have a specific effect upon the fungus.

Aldred Scott Warthin.

HEART DISEASES: ANÆMIC INFARCTION.—The occlusion of a branch of a coronary artery, if not immediately fatal, is followed by an anæmic necrosis of the part of the heart wall supplied by the occluded vessel. The necrosed area is known as an anæmic or white infarct or as *myomalacia cordis*. The terminal branches of the coronary arteries are true end arteries, and possess but a very limited collateral anastomosis. The area of necrosis produced by the occlusion of a terminal branch corresponds to the physiological distribution of the affected vessel. Infarction occurs most frequently in the portion of the myocardium supplied by the anterior coronary artery, the most common location being in the wall of the left ventricle near the apex.

The occlusion is usually produced by thrombosis or embolism. These events are favored by sclerotic changes; under certain conditions cororonary sclerosis in itself may give rise to infarction without the occurrence of thrombosis or embolism. In such cases the nutrition of the muscle already impaired by the partial obstruction of the vessel caused by sclerosis may be so much further lowered by general circulatory disturbances that the integrity of the muscle can be no longer preserved. Thrombi are of much more frequent occurrence in the coronary vessels than emboli; their formation is directly dependent upon the occurrence of sclerotic changes in the vessel wall. The entrance of emboli into the coronary vessels is of rare occurrence. This fact is explained by the anatomical peculiarities of the coronary arteries, the location of their orifices, angle of divergence with the aorta, force of current, etc. Embolism, however, occasionally occurs; the embolus usually consists of a portion of a thrombus primary in the left heart, either parietal or formed upon the mitral or aortic valves. In other cases the embolus may consist of atheromatous material derived from the vessel wall above the point of occlusion. Septic emboli occur in cases of malignant endocarditis; they are more frequently derived from vegetations on the mitral than

* MacCallum: Anat. Anz., xiii., 1897; Journ. of Experimental Medicine, iv., 1899; and Welch Festschrift, Johns Hopkins Hospital Reports, ix., 1900.

on the aortic valve. In whatever way produced, coronary occlusion leads immediately or very quickly to anæmic necrosis, the slight capillary anastomosis and the Thebesian vessels being inadequate to the task of supplying sufficient nutrition to the affected area.

The infarct is yellowish or grayish-red in color and of a cloudy, parboiled appearance. The consistence is somewhat softer than that of the normal muscle, and the recent infarct is slightly elevated. Subsequently it becomes softer and depressed. The heart infarct is rarely wedge-shaped, as is so frequently the case in infarction of other organs; but the contour is irregular, areas of living muscle alternating with dead tissue. This irregularity of distribution is due to the peculiar overlapping of the coronary branches, as a result of which adjacent muscle bundles may receive their blood supply from terminal branches arising from different main trunks of the third or fourth degrees. This fact is of great importance in the heart economy in so far as the occurrence of infarction is concerned. Occlusion of the smaller terminals does not lead to an infarction extending entirely through the heart wall, and the chances of rupture or aneurismal dilatation are thereby much lessened. Around the periphery of the infarcted area there is almost always a narrow zone of hemorrhage and congestion outlining the yellowish dead muscle with a border of red. This is to be regarded as being of the nature of a narrow zone of hemorrhagic infarction in the limited area of collateral anastomosis between the occluded vessel and the neighboring terminal. Large hemorrhages occasionally occur about the infarct; these may extend in between the dead muscle bands of the anæmic area, giving it the appearance of a hemorrhagic infarction. Infarcts of small size do not usually involve either the peri- or the endocardium, but larger ones are accompanied by the formation of thrombi on the endocardium and localized areas of pericarditis.

On microscopical examination the muscle fibres in the central portion of the infarct show a condition of simple necrosis, their nuclei having completely disappeared, and the muscle protoplasm presenting a more or less homogeneous appearance, staining deep red with eosin. Occasionally extensive fragmentation or Zenker's necrosis may be present. Around the periphery of the completely necrosed area there is usually found a zone of diffused chromatin, the muscle nuclei at this point showing beginning or partial disintegration. Bordering upon this zone is the area of partially damaged muscle with greatly congested vessels and extensive interstitial hemorrhage. If the infarct is not of very recent occurrence extensive leucocyte infiltration may be found around the periphery, or evidences of repair in the formation of granulation tissue. In older infarcts the dead muscle is almost or wholly replaced by new connective tissue at the periphery, in which numerous hypertrophic muscle fibres may be present. Scattered throughout infarcts of large size small islands of living heart muscle are usually found corresponding to the distribution of neighboring branches. The boundary between such areas and the necrosed muscle usually shows a narrow intermediate zone of partial necrosis, diffused chromatin, and hemorrhage. In the majority of infarcts there is usually present a narrow line of living muscle just beneath the peri- and endocardium, which is to be explained by the presence of small collateral branches running beneath these surfaces.

The regeneration of heart-muscle fibres in or around healing infarcts takes place only to a very limited extent. Evidences of this are seen in greatest degree in week-old infarcts. The division figures are never very numerous, and soon disappear. It is probable that the new cells formed quickly degenerate. Hypertrophic muscle fibres are often very numerous in the immediate neighborhood of a healing infarct. This must be regarded as being of the nature of a compensatory hypertrophy. Whether an increase in the number of fibres takes place or not cannot at present be positively stated. Ultimately infarcts of small size are completely replaced by scar tissue, and the area of induration thus formed appears as a white tendinous patch or band in the heart muscle. The designation of fibroid heart or fibrous myocarditis is applied to this condition as well as to the areas of sclerosis due to obliterating endarteritis.

Septic infarction may occur in cases of pyæmia, the smaller branches of the coronary arteries may be blocked with septic emboli. Purulent inflammation of the vessel wall and the neighboring tissue results, and the area of infarction becomes converted into an abscess. Usually death results from the general condition before these develop into abscesses of large size. In the majority of cases they appear as pinhead abscesses, rarely as large as a pea. Very rarely they may reach such a size as to perforate into the heart cavity or into the pericardium, forming what has been termed acute cardiac ulcer. Embolic aneurismal dilatation of the affected coronary branch may result at the point of embolism, and rupture of this may lead to the formation of a dissecting aneurism of the heart wall.

As a result of anæmic infarction of the heart death may occur instantaneously from the disturbance of the cardiac circulation and the arrest of the fibrillary contractions. In all cases of sudden death occlusion of the coronary vessels should be carefully sought for. Particularly is this the rule in medico-legal examinations. Death may occur before recognizable changes in the structure of the muscle take place. In other cases it may not result for some time after the occlusion of the vessel. It has been shown by animal experiments that the heart muscle is capable of contraction for nearly twelve hours after ligation of the coronaries. At the present time it is impossible to say whether death occurs at the moment of occlusion of the vessel or at the moment of muscle failure. With the softening of the infarcted area either rupture or aneurismal dilatation of the cardiac wall may take place. Sudden death may be caused by the former event. After healing of an infarct the cicatricial tissue may give before the blood pressure and produce an aneurism. In many cases there is a progressive occlusion of the coronary branches with the production of multiple infarcts. A very extensive degree of fibroid heart may result from the healing of these.

The symptoms of anæmic infarction are usually those of angina pectoris. The direct relation between the anginal attack and the moment of infarction has not been sufficiently emphasized. The cases of sudden death are quite frequently preceded by an intense but brief attack of this nature. In other cases there are no definite cardiac symptoms. After healing of the infarcted areas the clinical picture is that of fibrous myocarditis. Prognosis and treatment will be considered under that head.

Aldred Scott Warthin.

HEART DISEASES: ANEURISM.—The term aneurism of the heart is applied to three very different conditions: aneurisms of the coronary vessels, pouch-like projections of the valves, and local dilatations of the heart wall. The original use of the expression cardiac aneurism was that of Avenbrugger and Corvisart, who applied it to all cardiac enlargements of the nature of hypertrophy or dilatation, distinguishing the two as *aneurysma passivum* and *aneurysma activum*. This conception continued up to the time of John Hunter and Matthew Baillie with whom the term took on its modern significance of a partial dilatation of the cardiac wall. In accordance with this idea the condition is often spoken of as partial aneurism, though in the majority of cases cardiac aneurism is taken as referring to aneurism of the wall alone, coronary and valvular aneurisms being considered under separate heads.

Aneurism of the Coronary Arteries.—Aneurismal dilatations of the coronary branches of the second and third degree occur as the result of sclerosis or embolism. They are usually multiple, but may be found singly. They vary in size from a mustard seed to a large cherry, and occasionally form small nodular enlargements scattered over the heart, lying just beneath the epicardium. They are found in greatest numbers and of largest size when due to septic emboli. The symptoms are those of

myocarditis. They may rupture either into the pericardial cavity or into the heart wall, in the latter case forming dissecting aneurisms which may be mistaken for true aneurisms of the cardiac wall.

Aneurism of the Valves.—(See article next beyond this.)

Aneurism of the Heart Walls.—Localized dilatation of the heart wall may be congenital or acquired. In the latter case it is due either to trauma or to some pathological condition diminishing the resistance of the heart muscle in localized areas.

Congenital Form.—In defective development of the heart muscle aneurismal or hernial projections of the endocardium may occur between the muscle bundles. As a result of congenital abnormal thinness of the cardiac wall aneurismal dilatations may occur in any part of the heart, most frequently in the membranous portion of the ventricular septum. The occurrence of congenital aneurisms in individuals reaching maturity is, however, very rare, the majority of the cases being found in infants or young children.

Acquired Form.—In the majority of cases this is due to fibroid changes in the heart muscle following coronary obstruction or syphilitic infiltration, less frequently to anæmic infarction, fatty degeneration, mural endocarditis and wounds of the heart. It is not of common occurrence, though cases have been reported with increasing frequency in late years. In 1867 Pelvet collected the cases published up to his time, and in 1883 a similar collection was made by Legg, who found in the literature the reports of 90 cases. Since his article new observations have been constantly made, and the number of recorded cases is now in the neighborhood of 300. According to Williglk, 170 cases of cardiac aneurism were found in 34,000 autopsies. The acquired dilatation may be acute or chronic; according to its manner of formation it may be a pouch-like dilatation communicating with the heart cavity through a large opening, or it may be saccular with a very small opening, or the aneurism may be of the dissecting variety. Rarely it may be multilocular, consisting of a number of intercommunicating sacs.

The majority of cases occur in males of advanced age, but a number of cases in young men have been reported, and one in a young girl of twelve years. In the younger cases the condition is almost always the result of syphilitic weakening of the wall. The usual seat of the aneurism is in the anterior wall of the left ventricle near the apex, occurring in this location in 59 of the 90 cases reviewed by Legg. The septum is involved next in point of frequency, while the auricles and right ventricle are only very rarely affected. Usually the aneurism is single, but very rarely localized dilatations have been found in all four of the heart cavities. In the auricles the whole wall may be involved in the dilatation. Occasionally a number of dilatations, in one of the reported cases four, have been found side by side, their walls in apposition. The aneurismal dilatation of the septum usually protrudes into the right ventricle, but one case having been recorded of its projecting into the left.

The size of the aneurism may vary greatly, from the size of a fist to a man's head. The larger the sac, the thinner the wall. In very large sacs it may be as thin as paper without any traces of heart muscle remaining. The sac may be filled with a soft or firm thrombus. This may become calcified and the sac thus obliterated. Induration and calcification of the sac wall may also occur. Usually the pericardium becomes adherent to the epicardium over the sac, so that eventually the wall of the sac may consist of the endocardium and both the visceral and the parietal portions of the pericardium.

The acute dilatation of the heart wall is usually due to anæmic infarction or septic myocarditis, rarely to direct trauma of the heart, as in the case of stab wounds. In some cases localized degeneration or necrosis of the endocardium may make the latter the place of least resistance. The softened membrane yields to the blood pressure, is torn, blood passes through the rent and gradually undermines the heart muscle. When the area of softening is primary in the muscle the endocardium overlying the weakened spot is stretched or torn, forming a cavity into which the blood forces its way. In this manner the weakened area gradually bulges out more and more until there is a definite pouch formed. Acute aneurisms usually increase very rapidly in size, in some cases becoming as large as the heart, so that the condition has been regarded as one of double heart. Though the sac may become filled with blood clot, the wall never acquires a sufficient power of resistance, and the dilatation usually proceeds until rupture occurs.

Chronic aneurism in the great majority of cases is the result of fibrous changes in the heart muscle. The connective tissue lacking the power of contraction against

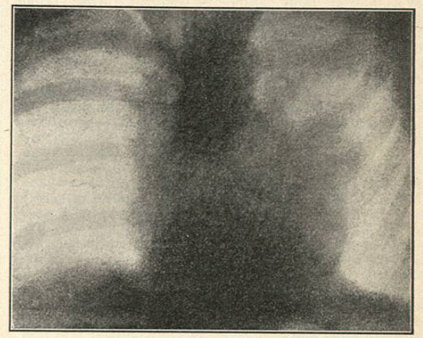

Fig. 2589.—Skiagram of a Case of Cardiac Aneurism. (After Walsham: *Edinb. Med. Journal*, April, 1901.)

the blood pressure during systole gradually becomes stretched outward or a cavity may be formed in the wall connected with the ventricle by a narrow opening. By many writers syphilitic myocarditis is regarded as the most common cause of cardiac aneurism of the slowly progressive type. That the fibrous changes are directly the result of syphilis cannot be proved in the majority of cases; much rather is the process to be looked upon as being of the nature of a chronic anæmic infarction due to the progressive narrowing of the coronary arteries through sclerotic changes, which may be due to syphilis or to other causes. In a number of cases, however, the dilatation has been found to be the direct result of gummatous changes in the cardiac wall. Further, the occurrence of coronary sclerosis and fibrous changes in the heart muscle in young individuals may be quite safely regarded as being due to syphilis. Anæmic infarction plays a much greater part in the causation of chronic aneurism than is accorded to it by the majority of writers. Small infarcts are replaced by connective tissue, and up to certain limits the tone of the wall may be preserved, but if the general tone be diminished by fatty degeneration, atrophy, etc., the area of connective tissue offering the least resistance gives way and dilatation occurs at this point. Localized areas of fatty degeneration, atrophy, etc., may of themselves be the primary factor in the production of aneurism.

Dissecting Aneurism.—The dissecting variety of cardiac aneurism deserves especial mention. Vestberg has made the most complete study of this condition. He defines it as a pathological cavity communicating with the heart or beginning of the aorta, caused by a separation of the layers of the cardiac wall by the blood. Four varieties are distinguished: parietal, septal, valvular, and interparietal. To these might be added a fifth variety when the cavity is formed between the layers of pericardial adhesions. According to Vestberg, the total number

of dissecting aneurisms is 60. Of these 47 were interparietal or septal. In 30 cases the left ventricle was the site of the aneurism. The interparietal form occurs in the peri-aortic space of Vestberg, from which the blood may dissect into the auricular or ventricular septum. In 16 cases the point of origin was in the sinus of Valsalva. Acute ulcerative endocarditis is the cause in the majority of cases; in other cases abscess, atheroma, and trauma were the causal factors. Dissecting aneurisms of the aorta may extend into the walls of the heart and through secondary rupture become converted into dissecting cardiac aneurisms.

Diagnosis.—The diagnosis of cardiac aneurism is in the majority of cases impossible; it is usually made at the autopsy table. In the case of acute aneurism the symptoms are those of acute myocarditis; in the chronic form they cannot, as a rule, be separated from those of chronic myocarditis. The aneurism is usually so small as to present no characteristic physical signs. In rare cases the sac has caused erosion of the thoracic wall, forming a visible tumor. The results of percussion and auscultation are very uncertain guides, and though in individual cases unusual murmurs may be produced, they are not constant or characteristic enough to be of diagnostic significance. The Roentgen ray has recently been applied to the differential diagnosis of this condition, and the location, size, and form of the aneurismal dilatation have been shown by this means.

Prognosis.—The prognosis in the majority of cases is not favorable. In early and undoubted syphilitic cases it is relatively better. The terminations are very variable. After reaching a certain degree of dilatation the process may remain stationary. The sac may be obliterated by thrombosis; calcification of the thrombus may occur. Death usually results from cardiac insufficiency to which other factors than the aneurism may contribute. In other cases gradual increase of size with resulting rupture takes place.

Treatment.—The treatment in suspected cases is along the general lines indicated in myocardial and valvular diseases. The possibility of syphilis should always be considered. *Aldred Scott Warthin.*

HEART DISEASES: ANEURISM OF VALVES.—A valvular aneurism is a cavity in the substance of the valve itself, containing pus and other inflammatory products, or blood clots and débris. The origin seems to be always in endocarditis of a more or less acute form, of which this is really only one of the possible results. If the process starts on the surface of the valve, ulceration may perforate one layer, after which the blood pressure dissects away the other, and so dilates the cavity that it may be as large as a pigeon's egg. On the other hand, the process may begin in the tissue of the valve and result in perforation of one of the layers from within, after which the blood pressure acts precisely as in the former case. These aneurisms are generally single, but may be multiple, and seem to be confined to the left heart. Though many authorities state that aneurisms are most commonly found in the aortic valves, the review of the literature made by Drasche shows that they are more frequently located in the mitral valve and that the anterior leaflet is more often affected than the posterior. Of the 23 cases collected by Pelvet, in 16 the aneurism was on the mitral, in 7 on the aortic valve. The perforation is almost always found on the side of the valve which is exposed to the highest pressure—the ventricular surface of the mitral, the distal surface of the aortic. The cavity may be lined by a laminated clot. The layer of the valve opposite to that containing the perforation may rupture, and the valve then suddenly become insufficient. The symptoms in such a case might be precisely similar to those of valvular rupture from any other cause.

Aneurisms have also been found, though rarely, in the valves of the right heart. As may be readily understood, valvular aneurism is of pathological rather than clinical interest, inasmuch as it betrays its presence by no distinctive diagnostic mark. Acute endocarditis can usually be detected, and so also valvular incompetence, but the precise steps which have led from one to the other in any given case must remain highly uncertain. This being the case there is, of course, no special treatment to be recommended other than that suitable to acute endocarditis. It would seem that the affection occasionally becomes chronic when seated in the mitral curtains, and is, therefore, somewhat less serious than in the semilunar cusps.

There is one other and more chronic condition which is sometimes classed as valvular aneurism. A portion of, or the whole valve, namely, may become so modified by inflammatory changes that its tissue generally yields and becomes stretched by the force of the blood pressure—both layers of the valve remaining in apposition, but being bellied out with the convexity toward the left auricle or ventricle respectively, according as the changes are seated in the mitral or in the aortic valve.
Frederick C. Shattuck.

HEART DISEASES: ANIMAL PARASITES.—Animal parasites are but rarely found in the human heart. The echinococcus is the most frequent and the only one likely to give rise to serious symptoms. The heart is involved in about one per cent. of all cases of echinococcus infection. In a large per cent. of cases the heart alone was involved. The cysts are found much more frequently in the right side of the heart. They may vary in size from a pinhead to an orange or even larger. In one case in which the hydatid was situated in the left ventricle the enlargement of the heart was so great as to displace and compress both lungs. The cysts may be single or multiple; in one case eighty were found forming a grape-like cluster extending from the right auricle into the ventricle through the tricuspid orifice. As a rule the hydatid develops within the wall, but it may occasionally project as a pedunculated cyst into the heart cavity or into the pericardial sac. In both cases the origin of the cyst is in the myocardium, but as the hydatid increases in size the muscle is either pushed aside or becomes atrophic, and may finally disappear, the cyst then coming to project either upon the pericardial or upon the endocardial surface. The pedicle consists of a portion of the overlying pericardium or endocardium. In rare cases the cyst may extend entirely through the wall, forming a fluctuating tumor covered on one side by pericardium and on the other by endocardium. The large hydatids may contain numerous daughter cysts, varying in size from a pea to a pigeon's egg. The heart muscle in the immediate neighborhood of the cyst may show no changes or the muscle fibres may be atrophic. The thickness of the cyst wall varies from 1 to 5 mm., but this may be much increased by fibroid changes in the overlying peri- or endocardium.

Small hydatids of the myocardium not projecting into the pericardial sac or heart cavity produce no symptoms and are usually discovered only at autopsy. When the cyst projects into the cavity of the pericardium, pericarditis may be produced, and this may in turn give rise to a pleuritis by direct extension. Subpericardial hydatids, however, do not always excite pericarditis. In neither case are the symptoms characteristic. Hydatids projecting into the heart cavity are very likely to rupture. In this event sudden death may result from the embolism of the daughter cysts. In the great majority of cases the perforation is into the right heart, the cysts blocking either the tricuspid orifice or the pulmonary artery. Smaller cysts may pass through the main pulmonary branches and lead to embolism of the smaller arteries and the formation of multiple hydatids throughout the lung. Death may result in such cases from pneumonia, slowly developing pleuritis, general cachexia, etc. Partial obstruction of the pulmonary orifices may give rise to symptoms of pulmonary stenosis and insufficiency. Rupture of an echinococcus cyst into the left heart may lead to blocking of the aortic orifice with resulting stenosis and insufficiency, or smaller cysts may cause occlusion of peripheral arteries, leading to local gangrenous conditions. In one case rupture occurred into both the right and the left heart. Embolism of echinococcus cysts in the right heart or pulmonary vessels may also occur as the result of the rupture of primary hydatids into the large veins. This occurs most frequently in the case of liver hydatids.

The differential diagnosis of heart hydatids is in the majority of cases impossible. The condition may be suspected when hydatids are present in other parts of the body, or from the nature of the emboli when embolism occurs. In one of the reported cases, in which there were numerous metastases throughout the lung, small cysts were found in the sputum. Aspiration of the pericardial sac and microscopical examination of the fluid thus obtained may lead to the recognition of the condition. In those cases in which the cysts do not rupture or project into the pericardial sac the symptoms are of a very indefinite nature and may suggest either myocarditis or valvular lesions. The treatment is entirely symptomatic. In certain cases surgical interference may be of avail. Goodhart claimed to have cured a case of hydatid cyst of the cardiac wall.

The cysticercus cellulosæ is of rare occurrence in the heart, only about thirty cases having been reported. The left ventricle appears to be most frequently affected. The cysts are usually few in number, the size varying from a bean to a hazelnut, rarely larger. They may be found in the muscle or just beneath the endo- or pericardium. As in the case of the echinococcus hydatids they may project from the endocardial surface into the blood stream, being attached to the wall by a slender pedicle formed by the endocardium. The layer of endocardium over the cyst may become very much thickened. The condition cannot be diagnosed during life, the symptoms being of a very indefinite character or entirely wanting.

Trichina spiralis is stated by many writers never to occur in heart muscle, but cases of such occurrence have been observed by Leuckart, von Zenker, and Fiedler. Pentastoma denticulatum has also been found in the heart wall. In the case of both of these parasites the affection of the heart is of pathological interest only.
Aldred Scott Warthin.

HEART DISEASES: DISPLACEMENTS.—Definitions and Varieties.—The heart, in a state of health, is subject to certain changes of position, caused, first, by the respiratory movements, and, second, by alterations in the bodily posture. Abnormal displacements of the organ may be either congenital or acquired. The latter only—or those that occur as the result of disease—will be considered.

In studying the displacements of the heart it is important to remember that the organ is held *in situ* directly by the great vessels, and only indirectly, through the pericardium, by the diaphragm. The attachments, fixing only the base of the heart, permit free play to the body of the organ, which hangs loosely in the pericardial sac, and very easily undergoes displacement laterally or vertically, while its dislocation either forward or backward is exceedingly rare. This statement, however, points only to a broad general classification of the disorders in question, since the heart is very seldom displaced in an absolutely horizontal, or in an absolutely vertical, plane.

Morbid Anatomy.—It is noteworthy that the amount of dislocation actually found after death is usually less than the physical signs during life have led one to expect. The most obvious, though less important, change which the heart undergoes is a disturbance of its normal relations to adjacent structures. Compression of the cardiac walls, which may occur to a greater or less degree in the pressure class of cases, may be the more important condition. The pericardium is sometimes displaced with the heart, or greatly stretched, and the great vessels at the base of the heart and at the root of the neck may be stretched and twisted, their change in position varying with the direction and extent of cardiac displacement. In some instances the circulation in the vessels is seriously impeded. Occasionally the pericardium and heart become adherent, and adhesions between the pericardium and pleura fix the heart in its abnormal position.

The functional disturbances resulting from these alterations vary with the cause of the displacement. In those which are due to pressure, the heart, if healthy, suffers little or no real compression, even when the dislocation is effected rapidly; and if it is displaced in front of, and in contact with, a solid tumor, such as an aneurism, a solidified lung, or the spinal column, its impulses may be so vigorous and diffused as to be mistaken for cardiac hypertrophy. But if the heart is diseased, and especially if its walls are degenerated, or its cavities much dilated, sudden compression, although moderate, may cause embarrassment of the cardiac action, and even induce a fatal syncope. In those displacements which are due to retraction of either lung, the heart is displaced toward the retracted side, partly by traction, and partly by extra-distention of the opposite lung, and the effects upon the functional activity of the heart are comparatively slight. In very rare cases the heart, when displaced, becomes involved in the inflammatory process of the lung or pleura, which leads to the displacement, and fibroid changes in its walls, occurring secondary to adhesions, ultimately interfere greatly with the heart power.

Etiology.—The causes of acquired displacement of the heart are most conveniently arranged in two classes: first, conditions that exert pressure, and second, conditions that exert traction upon the heart. On the same principle all cardiac displacements have been placed under the two heads of "excentric" and "concentric." In the former of the above classes, the heart may be pressed or pushed out of position by fluid effusions into either pleural cavity. This is by far the most frequent cause of lateral displacement. Pneumothorax will also push the heart to one side, and when accompanied by perforation and inflammatory effusion, will carry the displacement to its utmost possible limit. Hydrothorax being usually double, and, therefore, pressing equally on both sides of the heart, does not displace the organ laterally, but downward. Intrathoracic tumors, aneurisms, and abscesses sometimes press the heart to the right or the left. Hypertrophy and emphysema, or other causes of enlargement of the lungs, occasionally operate in the same way, as also does extensive peumonic consolidation. Pericardial effusion, when considerable, but not extensive, may push the apex into a position corresponding to the left nipple, or even above it. Cardiac displacement may also depend upon various abdominal conditions, as gaseous distention of the stomach and intestines, enlargement of the liver or spleen, the pregnant uterus, abdominal tumors or ascites. Diaphragmatic hernia or abscesses in extremely rare cases cause displacement of the heart upward.

In the second class of cases the displacement is concentric, that is, toward the origin of the displacing force. This movement takes place during absorption of pleuritic effusions, with imperfect expansion of the lung; and in consequence of a fibroid phthisical process or cirrhosis of the lung causing a gradual decrease in its volume. The heart also suffers traction in some forms of deformity of the chest dependent upon curvature of the spine. It must be observed, however, that in all these instances the displacing force is in reality one of pressure, resulting in displacement from removal of the normal counter-pressure. In certain cases, however, actual traction undoubtedly occurs, as when the pericardium becomes involved in a cirrhotic process in the lungs, or in an adhesive pleurisy, and it and the heart are displaced by the subsequent retraction.

Since displacement of the heart is a purely mechanical process, its motion will be subject to the universal law of moving bodies; that is, it will take the direction of least resistance, or of the resultant of all the acting forces. The more common elements of this resultant are: