of the apex, the absence of forcible, heaving impulse, and of any obvious cause of hypertrophy will afford satisfactory criteria for a diagnosis. The reverse condition exists in some cases in which emphysema masks moderate cardiac hypertrophy. The area of dulness may be normal, or even diminished, and the pulse and character of the sounds will help in the diagnosis; but it is sometimes a difficult matter.

Prognosis.—The course of any case of cardiac hypertrophy may be divided into three stages:

(*a*) The period of development, which varies with the nature of the primary lesion. For example, in rupture of an aortic valve, during a sudden exertion, it may require months before the hypertrophy becomes fully developed; or, indeed, it may never do so, and death may follow from an uncompensated dilatation. On the other hand, in sclerotic affections of the valves, with stenosis or incompetency, the hypertrophy develops step by step with the lesion, and may continue to counterbalance the progressive and increasing impairment of the valve.

(*b*) The period of full compensation—the latent stage—during which the heart's vigor meets the requirements of the circulation. This period may last an indefinite time, and a patient may never be made aware by any symptoms that he has a valvular lesion.

(*c*) The period of broken compensation, which may come on suddenly during very severe exertion. Death may result from acute dilatation; but more commonly it takes place slowly and results from degeneration and weakening of the heart-muscle.

The breaking or rupture of cardiac compensation may be induced by many causes, among which the most important are: (1) Failure of the general nutrition. In many instances of heart-disease, exposure, poor food, and alcohol combine to bring about disturbance of a well-balanced heart lesion. Acute illnesses, particularly the fevers, may induce general debility and with it weakening of the heart-muscle. (2) Disturbance of the local nutrition of the heart, owing to gradual sclerosis of the coronary arteries, is a common cause. (3) Very severe muscular exertion, which may disturb a compensation, perfect for years, and induce death in a few days (Traube). (4) Mental emotions. Severe grief or fright may bring on failure of compensation.

The prognosis is largely, as already stated, a matter of maintained compensation. Once established, the hypertrophy rarely, if ever, disappears, inasmuch as the cause usually persists. Occasionally, perhaps, the hypertrophy associated with neurotic palpitation from tobacco, or other causes, or the hypertrophy following muscular over-exertion, may disappear.

Dilatation of the Heart.

Two varieties are recognized, dilatation with thickening and dilatation with thinning. The former is the most common, and corresponds to the dilated or eccentric hypertrophy.

Etiology.—Two important causes combine to produce dilatation—increased pressure within the cavities and impaired resistance, due to weakening of the muscular wall—which may act singly, but are often combined. A weakened wall may yield to a normal distending force, or a normal wall may yield under a heightened blood-pressure.

(1) Heightened endocardiac pressure results either from an increased quantity of blood to be moved or an obstacle to be overcome, and is the most frequent cause. It does not necessarily bring about dilatation; simple hypertrophy may follow, as in the early period of aortic stenosis, and in the hypertrophy of the left ventricle in Bright's disease.

A majority of the important causes of increased endocardiac pressure have already been discussed under hypertrophy. One or two may be considered more in detail.

The size of the cardiac chambers varies in health. With slow action of the heart the dilatation is complete and fuller than it is with rapid action. Physiologically, the limits of dilatation are reached when the chamber does not empty itself during the systole. This may occur as an acute, transient condition in severe exertion—during, for example, the ascent of a mountain. There may be great dilatation of the right heart, as shown by the increased epigastric pulsation, and even increase in the cardiac dulness. The safety-valve action of the tricuspid valves may here come into play, relieving the lungs by permitting regurgitation into the auricle. With rest the condition is removed, but if it has been extreme, the heart may suffer a strain from which it may recover slowly, or, indeed, the individual may never be able again to undertake severe exertion. In the process of training, the getting wind, as it is called, is largely a gradual increase in the capability of the heart, particularly of the right chambers. A degree of exertion can be safely maintained in full training which would be quite impossible under other circumstances, because by a gradual process of what we may call physical education the heart has strengthened its reserve force—widened enormously its limits of physiological work. Endurance in prolonged contests is measured by the capabilities of the heart, and its essence consists in being able to meet the continuous tendency to overstep the limits of dilatation.

We have no positive knowledge of the nature of the changes in the heart which occur in this process, but it must be in the direction of increased muscular and nervous energy. The large heart of athletes may be due to the prolonged use of their muscles, but no man becomes a great runner or oarsman who has not naturally a capable if not a large heart. Master McGrath, the celebrated greyhound, and Eclipse, the

race-horse, both famous for endurance rather than speed, had very large hearts.

Excessive dilatation during severe muscular effort results in heart-strain. A man, perhaps in poor condition, calls upon his heart for extra work during the ascent of a high mountain, and is at once seized with pain about the heart and a sense of distress in the epigastrium. He breathes rapidly for some time, is "puffed," as we say, but the symptoms pass off after a night's quiet. An attempt to repeat the exercise is followed by another attack, or, indeed, an attack of cardiac dyspnœa may come on while he is at rest. For months such a man may be unfitted for severe exertion, or he may be permanently incapacitated. In some way he has overstrained his heart and become "broken-winded." Exactly what has taken place in these hearts we cannot say, but their reserve force is lost, and with it the power of meeting the demands exacted in maintaining the circulation during severe exertion. The "heart-shock" of Latham includes cases of this nature—sudden cardiac breakdown during exertion, not due to rupture of a valve. It seems probable that sudden death in men during long-continued efforts, as in a race, is sometimes due to over-distention and paralysis of the heart.

Examples of dilatation occur in all forms of valve lesions. In aortic incompetency blood enters the left ventricle during diastole from the unguarded aorta and from the left auricle, and the quantity of blood at the termination of diastole subjects the walls to an extreme degree of pressure, under which they inevitably yield. In time they augment in thickness, and present the typical eccentric hypertrophy of this condition.

In mitral insufficiency blood which should have been driven into the aorta is forced into and dilates the auricle from which it came, and then in the diastole of the ventricle a large amount is returned from the auricle, and with increased force. In mitral stenosis the left auricle is the seat of greatly increased tension during diastole, and dilates as well as hypertrophies; the distention, too, may be enormous. Dilatation of the right ventricle is produced by a number of conditions, which were considered under hypertrophy. All circumstances, such as mitral stenosis, emphysema, etc., which permanently increase the tension of the blood in the pulmonary vessels, will cause its dilatation.

(2) Impaired nutrition of the heart-walls may lead to a diminution of the resisting power so that dilatation readily occurs.

The loss of tone due to parenchymatous degeneration or myocarditis in fevers may lead to a fatal condition of acute dilatation. It is a recognized cause of death in scarlatinal dropsy (Goodhart), and may occur in rheumatic fever, typhus, typhoid, erysipelas, etc. The changes in the heart-muscle which accompany acute endocarditis or pericarditis may lead to dilatation, especially in the latter disease. In anæmia, leukæmia, and chlorosis the dilatation may be considerable. In sclerosis of the walls, the yielding is always where this process is most advanced, as at the left apex.

Under any of these circumstances the walls may yield with normal blood-pressure.

Pericardial adhesions are a cause of dilatation, and we generally find in cases with extensive and firm union considerable hypertrophy and dilatation. There is usually here some impairment as well of the superficial layers of muscle.

Morbid Anatomy.—The condition usually exists with hypertrophy in two or more chambers. It is more common on the right than on the left side. The most extreme dilatation is in cases of aortic incompetency, in which all the cavities may be enormously distended. In mitral stenosis the left auricle is often trebled in capacity, and the right chambers also are very capacious. The auricles may contain from eighteen to twenty ounces of blood. In chronic lesions of the lungs the right chambers are chiefly involved. In great distention of one ventricle the septum may bulge toward the other side. The auriculo-ventricular rings are often dilated, and there may be an increase in the circumference of an inch and a half or even two inches. Thus, the tricuspid orifice, the circumference of which is about four and a half inches, may freely admit a graduated heart-cone of over six inches; and the mitral orifice, which normally is about three and a half inches, may admit the cone to five and a half inches or even more. Great dilatation is always accompanied with relative incompetency of the valves, so that free regurgitation into the auricles is permitted. The orifices of the venæ cavæ and of the pulmonary veins may be greatly dilated.

The endocardium is often opaque, particularly that of the auricles. The muscle substance varies according to the presence or absence of degenerations. The microscope may show marked fatty or parenchymatous change, but in some instances no special alteration may be noticeable. There is much truth in Niemeyer's assertion "that it is not possible by means of the microscope to recognize all the alterations of the muscular fibrillæ which diminish the functional power of the heart." Of the changes in the ganglia of the heart we know very little. As centres of control they probably have more to do with cardiac atony and breakdown than we generally admit. Degeneration of them has been noted by Putjakin, Ott, and others.

Symptoms and Physical Signs.—Dilatation causes weakness of the cardiac walls, diminishes the vigor of their contractions, and is therefore the reverse of hypertrophy. So long as compensation is maintained the enlargement of a cavity may be considerable. The limit is reached when the hypertrophied walls in the systole can no longer expel all the contents, part of which remain, so that at each diastole the chamber is abnormally full. Thus, in aortic incompetency blood enters the left ventricle from the aorta as well as the auricle; dilatation ensues, and also hypertrophy as a direct effect of the increased pressure and increased amount of blood to be moved. But if from any cause the hypertrophy

weakens and the ventricle during systole fails to empty itself completely, a still larger amount is in it at the end of each diastole, and the dilatation becomes greater. The amount remaining after systole prevents the blood from entering freely from the auricle. Incompetency of the auriculo-ventricular valves follows, with dilatation of the auricle and impeded blood-flow in the pulmonary veins. Dilatation and hypertrophy of the right heart may compensate for a time, but when this fails the venous system becomes engorged and dropsy may result. The consideration of the symptoms of chronic valvular lesions is largely that of dilatation and its effects. Acute dilatation, such as we see in fevers or in sudden failure of a hypertrophied heart, is accompanied by three chief symptoms—weak, usually rapid, impulse, dyspnœa, and signs of obstructed venous circulation. Cardiac pain may be present, but is often absent.

The *physical signs* of dilatation are those of a weak and enlarged organ. The impulse is diffuse, often undulatory, and is felt over a wide area, and an apex beat or a point of maximum intensity may not exist. When it does exist, it may be visible and yet cannot be felt—a valuable observation made by Walshe. An extensive area of impulse with a quick, weak maximum apex beat may be present. When the right heart is chiefly dilated the left may be pushed over so as to occupy a much less extensive area in front of the heart, and the true apex beat cannot be felt; but the chief impulse is just below, or to the right of, the xiphoid cartilage, and there is a wavy pulsation in the fourth, fifth, and sixth interspaces to the left of the sternum. In extreme dilatation of the right auricle a pulsation may sometimes be seen in the third right interspace close to the sternum, and with free tricuspid regurgitation this may be systolic in character. Whether the pulsation frequently seen in the second left interspace is ever due to a dilated left auricle has not been determined. I have sometimes thought it was presystolic in rhythm, though it may be distinctly systolic. Post mortem, it is rare in the most extreme distention to see the auricular appendix so far forward as to warrant the belief that it could beat against the second interspace. The area of dulness is increased, but an emphysematous lung or the fully distended organ in a state of brown induration may cover over the heart and greatly limit the extent. The directions of increase were considered in connection with hypertrophy.

The first sound is shorter, sharper, more valvular in character, and more like the second. As the dilatation becomes excessive it gets weaker. Reduplication is not common, but occasionally differences may be heard in the first sound over the right and left hearts. The sounds are frequently obscured by murmurs, which are produced by incompetency of the valves due to the great dilatation, or are associated with the chronic valve disease on which the condition depends. The aortic second sound is replaced by a murmur in aortic regurgitation. The pulmonary sound is accentuated in mitral regurgitation and pulmonary congestion, but

with extreme dilatation it may be much weakened. The heart's action is irregular and intermittent, and the pulse is small, weak, and quick.

On auscultation both the sounds may be free from murmur. Often there is the condition known as embryocardia or fœtal heart-rhythm, in which the first and second sounds are very alike, and the long pause is shortened. In other instances there is the typical and characteristic gallop rhythm, rarely found apart from conditions of dilatation. With the various valvular lesions the corresponding murmurs may be heard. Murmurs, however, which have been present may disappear, as in the case of mitral stenosis. In other instances a loud systolic murmur may be heard at the apex, and when the case first comes under observation it may be impossible to say whether this is due to organic mitral lesion. The murmur may be confined to the apex region, or propagated well to the back. It is extremely common in the dilatation which follows the hypertrophy of the left ventricle in arterio-sclerosis. Under treatment, with the gradual disappearance of the dilatation, a murmur of this kind, even though most intense, may completely disappear, showing that it has been due to a relative insufficiency, not to a valvular lesion. All varieties of arrhythmia may occur in dilatation of the heart. The pulse, as a rule, is small, weak, quick, and often irregular.

Dilatation and Hypertrophy due to Overexertion and Alcohol.—There is a group of cases of dilatation and hypertrophy dependent upon prolonged overexertion, which rarely comes under observation until compensation has failed, and which then may be very difficult to distinguish from the similar conditions produced by valvular disease. The patients are able-bodied men at the middle period of life, and complain first of palpitation or irregularity of the action of the heart, shortness of breath, and subsequently the usual symptoms of cardiac insufficiency develop. On inquiring into the history of these patients none of the usual etiological factors causing valve disease are present, but they have always been engaged in laborious occupations and have usually been in the habit of taking stimulants freely. This is the affection which has been specially studied by McLean, Clifford Albutt, Seitz, and others, and in its earlier condition by Da Costa, in what he termed the irritable heart It is met with very frequently in soldiers. These cases may return to hospital three or four times with cardiac insufficiency, sometimes with slight anasarca, hæmoptysis, and signs of pulmonary engorgement. The condition is by no means infrequent. Bollinger has called attention to the common occurrence of dilatation and hypertrophy in beer-drinkers, particularly in the workers in the German breweries, who drink twenty or more litres in the day. Strümpell, at his Erlangen clinic, told me that this condition was very common in the draymen and workers in the breweries of that town, very few of whom pass the forty-fifth year without indications of hypertrophy and dilatation of the heart. On post-mortem examination the valves may be quite healthy, the aorta smooth, and no extensive arterio-

sclerosis or renal disease. The heart weighs from eighteen to twenty-five ounces; the chambers are dilated. The condition has been met with also in animals, and Houghton states that the heart of the celebrated greyhound Master McGrath weighed 9·57 ounces, just threefold in excess of the normal proportion of heart-weight to body-weight.

Idiopathic Dilatation.—And, lastly, there are other cases in which dilatation of the heart occurs without discoverable cause. In some instances there has been a history of sudden exercise or of mental emotion, but in other cases the condition seems to have come on spontaneously. In some the condition is acute and the patient has dyspnœa, slight cyanosis, cough, and great cardiac distress. Death may occur in a few days, or dropsy may supervene and the case may become chronic. Delafield has reported an interesting series of cases of this group.

Treatment.—The treatment of hypertrophy and dilatation has already been considered under the section on valvular lesions. I would only here emphasize the fact that with signs of dilatation, as indicated by gallop rhythm, urgent dyspnœa, and slight lividity, venesection is in many cases the only means by which the life of the patient may be saved, and from twenty-five to thirty ounces of blood should be abstracted without delay. Subsequently stimulants, such as ammonia and digitalis, may be administered, but they are accessories only to the bleeding in the critical condition of acute dilatation, which is so frequently met with in cardiac lesions.

IV. AFFECTIONS OF THE MYOCARDIUM.

1. **Lesions due to Disease of the Coronary Arteries.**—A knowledge of the changes produced in the myocardium by disease of the coronary vessels gives a key to the understanding of many problems in cardiac pathology. The terminal branches of the coronary vessels are end arteries. The blocking of one of these vessels by a thrombus or an embolus leads to a condition which is known as—

(*a*) *Anæmic necrosis*, or white infarct. This is most commonly seen in the left ventricle and in the septum, in the territory of distribution of the anterior coronary artery. The affected area has a yellowish-white color, sometimes a turbid, parboiled aspect, at others a grayish-red tint. It may be somewhat wedge-shaped, more often it is irregular in contour and projects above the surface. Microscopically the changes are very characteristic. The nuclei disappear from the muscle fibres, the condition of fragmentation is present, and the fibres present a homogeneous, hyaline appearance. In some instances there is complete transformation, and even to the naked eye a firm white patch of hyaline degeneration may appear in the centre of the area. Sudden death not infrequently follows the blocking of one of the branches of the coronary

artery and the production of this anæmic necrosis. *In medico-legal cases it is a point of primary importance to remember that this is one of the common causes of sudden death.* This condition should be carefully sought for, inasmuch as it may be the sole lesion, except a general, sometimes slight arterio-sclerosis. Rupture of the heart may be associated with anæmic necrosis.

(*b*) The second important effect of coronary-artery disease upon the myocardium is seen in the production of *fibrous myocarditis*. This may result from the gradual transformation of areas of anæmic necrosis. More commonly it is caused by the narrowing of a coronary branch in a process of obliterative endarteritis. The sclerosis is most frequently seen at the apex of the left ventricle and in the septum, but it may occur in any portion. In the septum often there are streaks of fibroid degeneration which do not reach the endocardium, and it may be necessary to divide the muscle in order to see them. Hypertrophy of the heart is commonly associated with this degeneration. It is the invariable precursor of aneurism of the heart.

Complete obliteration of one coronary artery, if produced suddenly, is usually fatal. When induced slowly, either by arterio-sclerosis at the orifice of the artery at the root of the aorta or by an obliterating endarteritis in the course of the vessel, the circulation may be carried on through the other vessel. Sudden death is not uncommon, owing to thrombosis of a vessel which has become narrowed by sclerosis. In the most extreme grade one coronary artery may be entirely blocked, with the production of extensive fibroid disease, and a main branch of the other also may be occluded. A large, powerfully built imbecile, aged thirty-five, at the Elwyn Institution, Pennsylvania, who had for years enjoyed doing the heavy work about the place, died suddenly, without any preliminary symptoms. The heart, which is in my collection, weighed over twenty ounces; the anterior coronary artery was practically occluded by obliterating endarteritis, and of the posterior artery one main branch was occluded.

(*c*) *Septic Infarcts.*—In pyæmia the smaller branches of the coronary arteries may be blocked with septic emboli and cause infarcts in the myocardium in the form of miliary abscesses, varying in size from a pea to a pin's head. These may not cause any disturbance, but when large they may perforate into the ventricle or into the pericardium, forming what has been called acute ulcer of the heart.

2. **Acute Interstitial Myocarditis.**—In the fevers and in pericarditis the intermuscular connective tissue is swollen and infiltrated with round cells and nuclei, the vessels are dilated, there are minute extravasations, and the muscle fibres may be granular or fatty, with indistinct striæ and nuclei. These instances have been met with in typhoid fever, small-pox, and diphtheria. The muscle substance is pale, soft, and easily torn, and the condition has been described either as inflammatory or degenerative.