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.

3. **Parenchymatous Degeneration.**—This is usually met with in fevers, or in connection with endocarditis or pericarditis. It is characterized by a pale, turbid state of the cardiac muscle, which is general, not localized. Turbidity and softness are the special features. It is the softened heart of Laennec and Louis. Stokes speaks of an instance in which "so great was the softening of the organ that when the heart was grasped by the great vessels and held with the apex pointing upward, it fell down over the hand, covering it like a cap of a large mushroom."

Histologically, there is a degeneration of the muscle fibres, which are infiltrated to a various extent with granules which resist the action of ether. Sometimes this granular change in the fibres is extreme, and no trace of the striæ can be detected. It is probably the effect of a toxic agent, and is seen in its most exquisite form in the lumbar muscles in cases of toxic hæmoglobinuria in the horse. It is met with in cases of typhoid, typhus, small-pox, and other infectious diseases, particularly when the course is protracted. There is no definite relation between it and the high temperature.

A form of myocarditis has been described, characterized by fragmentation of the fibres owing to softening of the cement substance. According to von Recklinghausen this is a post-mortem change.

4. **Fatty Heart.**—Under this term are embraced fatty degeneration and fatty overgrowth.

(*a*) *Fatty degeneration* is a very common condition, and mild grades are met with in many diseases. It is found in the failing nutrition of old age, of wasting diseases, and of cachectic states; in prolonged infectious fevers, in which it may follow or accompany the parenchymatous change; associated with acute and chronic anæmias. Certain poisons, such as phosphorus, produce an intense fatty degeneration. Local causes: Pericarditis is usually associated with fatty or parenchymatous changes in the superficial layers of the myocardium. Disease of the coronary arteries is a common and important cause. Lastly, in the hypertrophied ventricular wall in chronic heart-disease fatty change is by no means infrequent. This degeneration may be limited to the heart or it may be more or less general in the solid viscera. The diaphragm may also be involved, even when the other muscles show no special changes. There appears to be a special proneness to fatty degeneration in the heart-muscle, which may perhaps be connected with its incessant activity. So great is its need of an abundant oxygen supply that it feels at once any deficiency, and is in consequence the first muscle to show nutritional changes.

Anatomically the condition may be local or general. The left ventricle is most frequently affected. If the process is advanced and general the heart looks large and is flabby and relaxed. It has a light yellowish-brown tint, or, as it is called, a faded-leaf color. Its consistence is reduced and the substance tears easily. In the left ventricle the papillary columns and the muscle beneath the endocardium show a streaked or

patchy appearance. Microscopically, the fibres are seen to be occupied by minute globules distributed in rows along the line of the primitive fibres (Welch). In advanced grades the fibres seem completely occupied by the minute globules.

(*b*) *Fatty Overgrowth.*—This is usually a simple excess of the normal subpericardial fat, to which the term *cor adiposum* was given by the older writers. In other instances the fat infiltrates the muscular substance and, separating the strands, may reach even to the endocardium. In corpulent persons there is always much pericardial fat. It forms part of the general obesity, and occasionally leads to dangerous or even fatal impairment of the contractile power of the heart. Of 122 cases analyzed by Forchheimer there were 88 males and 34 females. Over eighty per cent occurred between the fortieth and seventieth years.

The entire heart may be enveloped in a thick sheeting of fat through which not a trace of muscle substance can be seen. On section, the fat infiltrates the muscle, separating the fibres, and in extreme cases—particularly in the right ventricle—reaches the endocardium. In some places there may be even complete substitution of fat for the muscle substance. In rare instances the fat may be in the papillary muscles. The heart is usually much relaxed and the chambers are dilated. Microscopically the muscle fibres may show, in addition to the atrophy, marked fatty degeneration.

5. **Other Degenerations of the Myocardium.** (*a*) *Brown Atrophy.*—This is a common change in the heart-muscle, particularly in chronic valvular lesions and in the senile heart. When advanced, the color of the muscles is a dark red-brown, and the consistence is usually increased. The fibres present an accumulation of yellow-brown pigment chiefly about the nuclei. The cement substance is often unusually distinct, but seems more fragile than in healthy muscle.

(*b*) Amyloid degeneration of the heart is occasionally seen. It occurs in the intermuscular connective tissue and in the blood-vessels, not in the fibres.

(*c*) The hyaline transformation of Zenker is sometimes met with in prolonged fevers. The affected fibres are swollen, homogeneous, translucent, and the striæ are very faint or entirely absent.

(*d*) Calcareous degeneration may occur in the myocardium, and the muscle fibres may be infiltrated and yet retain their appearance as figured and described by Coats in his Text-book of Pathology.

Symptoms of Myocardial Disease.—These are notoriously uncertain. A man with advanced fibroid myocarditis may drop dead suddenly, while doing heavy work, without having complained of cardiac distress. On the other hand, a patient may present enfeebled, irregular action and signs of dilatation; he may have shortness of breath, œdema, and the general symptoms believed to be characteristic of cases of fibroid and fatty heart, and the post-mortem show little or no change in the myocardium.

Cardio-sclerosis or fibroid heart is in some cases characterized by a

feeble, irregular, slow pulse, with dyspnœa on exertion and occasional attacks of angina. Irregularity is present in many, but not in all cases. The pulse may be very slow, even 30 or 40 per minute. Ultimately the cases come under observation with the symptoms of cardiac insufficiency. The arrhythmia, which may have been present, becomes aggravated and, according to Riegel, may not only precede, but also persist after the cardiac insufficiency has passed away. This certainly does not hold in all cases, for a patient recently under observation had the most marked arrhythmia, which persisted after recovery from a severe attack of cardiac insufficiency in which he nearly died. Upon his return, a few weeks ago, with dilatation and arrhythmia, we agreed that the condition was probably one of cardio-sclerosis; but the autopsy showed simple dilatation without either fibroid or marked fatty change in the heart.

Fatty degeneration of the heart presents the same difficulties. Extreme fatty changes, as in pernicious anæmia, may be consistent with full, regular pulse and a regularly acting heart. In some of these cases the fat does not appear to interfere seriously with the function of the organ. The truth is it may exist in an extreme grade without producing symptoms, so long as great dilatation of the chambers does not occur. The cardiac irregularity, the dyspnœa, palpitation, and small pulse are in reality not symptoms of the fatty degeneration, but of dilatation which has supervened. The fatty *arcus senilis* is of no moment in the diagnosis of fatty heart. The heart-sounds may be weak and the action irregular. When dilatation occurs, there is often the gallop rhythm, shortening of the long pause, and a systolic murmur at the apex. Shortness of breath on exertion is an early feature in many cases, and anginal attacks may occur. There is sometimes a tendency to syncope, and in both fibroid and fatty heart there are attacks in which the patient feels cold and depressed and the pulse sinks to 40 or 30, or even, as in one case which I saw, to 26. The patient may wake from sleep in the early morning with an attack of severe cardiac asthma. These "spells" may be associated with nausea and may alternate with others in which there are anginal symptoms. These are the cases, too, in which for weeks there may be mental symptoms. The patient has delusions and may even become maniacal. Toward the close, Cheyne-Stokes breathing is met with in a number of cases.

Fatty overgrowth of the heart is a condition certain to exist in very obese persons. It produces no symptoms until the muscular fibre is so weakened that dilatation occurs. These patients may for years present a feeble but regular pulse; the heart-sounds are weak and muffled, and a murmur may be heard at the apex. Attacks of cardiac asthma are not uncommon, and the patient may suffer from bronchitis. Dizziness and pseudo-apoplectic seizures may occur. Sudden death may result from syncope or from rupture of the heart. The physical examination is often difficult because of the great increase in the fat, and it may be impossible to define the area of dulness.

For practical purposes we may group the cases of myocardial disease as follows:

(1) Those in which sudden death occurs with or without previous indications of heart-trouble. Sclerosis of the coronary arteries exists—in some instances with recent thrombus and white infarcts; in others, extensive fibroid disease; in others again, fatty degeneration. In many cases there is never any complaint of cardiac distress, but, as in the case of Chalmers, the celebrated Scottish divine, may enjoy unusual vigor of mind and body.

(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms. These are the cases in which the condition may be strongly suspected and, in some instances, diagnosed. It is rarely possible to make a distinction between the fatty and fibroid heart.

(3) Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart. Dropsy is often present, and with a loud murmur at the apex it may be difficult, unless the case has been seen from the outset, to determine whether or not a valvular lesion is present.

Prognosis.—The outlook in affections of the myocardium is extremely grave. Patients recover, however, in a surprising way from the most serious attacks, particularly those of the second group.

Treatment.—Many cases never come under treatment; the first are the final symptoms.

Cases with signs of well-marked cardiac insufficiency, as manifested by dyspnœa, weak, irregular, rapid heart, and œdema, may be treated on the plan laid down for the treatment of broken compensation in valvular disease. Digitalis may be given even if fatty degeneration is suspected, and is often very beneficial.

Much more difficult is the management of those cases in which there is marked cardiac arrhythmia, with a feeble, irregular, very slow pulse, and syncope or angina. Dropsy is not, as a rule, present; the heart-sounds may be perfectly clear, and there are no signs of dilatation. Digitalis, under these circumstances, is not advisable, particularly when the pulse is infrequent. Complete rest in bed, a carefully regulated diet, and the use of the aromatic spirits of ammonia, sulphuric ether, and stimulants are indicated. For the restlessness and distressing feelings of anxiety morphia is invaluable. From an eightieth to a sixtieth of a grain of strychnia may be given three times a day. If, as is sometimes the case, the pulse is hard and firm, nitroglycerin may be cautiously administered, beginning with one minim of the one per cent solution three times a day and increased gradually.

In certain cases of weak heart, particularly when it is due to fatty overgrowth, the plan of treatment recommended by Oertl is advantageous. It is an invaluable method in those forms of heart-weakness due to intem-