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Some Observations ON DISEASES OF THE MYOCARDIUM.*

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THE lecture which I have the honour of delivering this afternoon was founded by Lady St. Cyres. It was her intention that the subject of the lecture should be definitely connected with the myocardium, so I have decided to give you some account of the way in which the muscle of the heart becomes diseased, what are the manifestations of the morbid condition, and what may be the outlook, together with some suggestions as to treatment.

SOME HISTORICAL CONSIDERATIONS.

It must always seem a strange thing that the ancients should have been under the impression that of all the organs of the body the heart alone could not be diseased; and indeed remarkably little was known about the heart and the circulation generally until the publication of Harvey's great discovery in 1628. Even after that very little was known or written about diseases of the heart for more than a century. Until the discovery of percussion and auscultation by Auenbrugger and Laennec respectively there was almost complete ignorance of valvular disease, but after that so much attention was focused upon the lesions of the different valves that the importance of the myocardium was somewhat neglected. With the advent of the modern cardiology, however, this has all been changed, and for the last thirty years attention has been closely directed to the study of the myocardium.

As far back as 1534, prior to the advent of Vesalius, Nicholas Massa of Venice had observed hypertrophy of the heart muscle, but it was not till more than a century later that Morgagni (1682-1777) called attention to chronic myocarditis, which he regarded in some respects as a degeneration of the heart muscle. Corvisart, in his well-known book¹ upon the heart, lays considerable stress upon the importance of the myocardium; it is, he said, the muscular substance of the heart which most essentially constitutes the central organ of the circulation. He described the gross changes in the size and shape of the various chambers—for example, dilatation and hypertrophy. He was of opinion that a change of form or length of the fibres of the myocardium was visible proof of its disordered action, but apparently he did not realize that, though the myocardium might be fatally weak, it yet might present to the naked eye no change in the general form, nor did he lay much stress upon inflammatory or degenerative conditions of the myocardium, though he seems to have been the first to make use of the term "carditis."

The pupil of Corvisart, the famous Laennec, who first introduced auscultation in 1819, spoke of the condition of the myocardium as "the key to cardiac pathology." Strangely enough Allan Burns (1809), who lived about the same time, considered disease of the myocardium to be less frequent than that of the valves. Later on, in 1834, the great Irish physician William Stokes insisted that the

symptoms associated with evidence of valvular disease depended on the state of the myocardium and not on the valvular lesions themselves; in particular, he studied the softening of the myocardium in typhus fever, correlating it with physical signs. "It is," he said, "in the vital and anatomical condition of the muscular fibres that we find the key to cardiac pathology, for no matter what the affection may be, its symptoms mainly depend on the strength or the weakness, the irritability or the paralysis, the anatomic health or disease of the cardiac muscle." As he could hardly recognize diffuse myocarditis, which is now usually detected by microscopic methods, he conducted his studies mainly on fatty degeneration, of which he described two forms: (1) cases corresponding to what we now know as chronic arterio-sclerosis or chronic interstitial nephritis, but, excellent as were his clinical descriptions of these groups, the pathological basis of fatty degeneration which he attributed to them was erroneous; (2) heart-block, now known as "Stokes-Adams disease." Unlike Corvisart, Stokes realized that important changes may exist in the heart without corresponding physical signs. Fatty degeneration had indeed been first noticed by Corvisart and described by Laennec; in this country Richard Quain was the first to recognize it.

PATHOLOGY OF MYOCARDIAL DISEASE.

Modern cardiology has done much to bring order out of the chaos of cardiac arrhythmias, but now what we are most wanting knowledge about in any given case is the condition of the myocardium. Granted that it may not be difficult to decide in the case of a particular patient that there is some affection of the myocardium, though this is by no means always easy to make out, what we specially require to know is how much of the myocardium is affected and what part of it.

It so happens that in most of the organs of the body—for example, the liver, lung, or kidney—the exact amount or part of the organ which is devitalized and put out of action is not of much material importance; but in the case of the heart we want to know not merely that the myocardium is diseased, but what part of it is affected. Thus if the disease is confined to the auricles, the prognostic outlook is much more favourable than if we have reason to believe that the ventricles are also affected. Again, if the ventricles are affected, it is of greater importance if the lesion is situated near the apex or at the interventricular septum than if it is situated in other parts of the ventricular wall.

Now the muscle of the heart may be affected pathologically in two different ways: (a) by fatty changes; (b) by fibroid changes; and these are often combined together in varying proportions.

Fatty Infiltration.

When the changes take this form, the fat infiltrates the heart muscle, spreading along the main fibrous septa and even between individual muscle fibres, but there is no substitution of fatty material for muscular substance as occurs in fatty degeneration; it is rather a degeneration from the overlying layer of fat around the heart. It is more common in males and rare before middle age. It is invariably accompanied by a certain amount of fatty degeneration, but the converse is not always true. It is always greater on the right side of the heart and is usually associated with general obesity. In fatty infiltration more or less dilatation of the cardiac chambers is usually present,

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often accompanied by hypertrophy. The symptoms specially to be noted in fatty infiltration are cyanosis with dyspnoea on exertion, together with giddiness or faintness; there may also be some oedema about the ankles and along the tibiae at night.

Fatty Degeneration.

Here the heart substance is paler and softer than normal, and the heart muscle has a faded leaf colour. The musculi papillares will usually have a streaky appearance—the so-called “tabby-cat” striation—due to the interspersions of pale strands of fattily degenerated muscular substance among healthy fibres. Without the microscope it can hardly be recognized satisfactorily. Muscle lying closely beneath the endocardium tends to be more affected than that which is nearest to the pericardium. When advanced the fatty degeneration leads to a disintegration of the muscle fibres of the heart and their consequent replacement by fibrous tissue. Most commonly it is situated in the left ventricle and is rare in the auricles. In the left ventricle it is the papillary muscles, the adjacent muscle on the posterior walls, and the septum which most often show fatty degeneration. In cases of anaemia and aortic disease it is the left ventricle which is most affected, while in chronic pulmonary disease and in mitral disease it is mainly the right ventricle. Fatty infiltration and fatty degeneration differ in their site rather than in their essential nature. Fatty degeneration is most common in severe anaemias, chronic alcoholism, chronic renal disease, severe cases of typhoid fever, and in fatal phosphorus and arsenical poisoning. The primary change in these cases is atrophy of the muscle substance, invasion of the fibres by fatty matter being secondary.

There are no characteristic symptoms of fatty degeneration, with the possible exception of angina pectoris, which is often associated with fatty degeneration of the heart, though by no means always; more common symptoms, perhaps, are syncope and apoplectic attacks due to defective pressure in the arterial system. Nor can it be said that there are any physical signs which are characteristic of fatty degeneration; it is rare to find much oedema, and probably it never occurs in a case of uncomplicated fatty degeneration. Until the disease is far advanced the diagnosis of fatty degeneration of the heart is far from easy; yet it is a condition which is frequently associated with sudden death, it may be during exertion or excitement or after a full meal. Rupture of the heart is one mode of termination, and may take place on very slight provocation. In the obese, cardiac failure is due to the relative smallness and weakness of the heart muscle—that is, the incapacity to meet the demands made upon it by the condition of general corpulence. In some fat but muscular individuals the heart is correspondingly large and muscular, while symptoms of cardiac insufficiency do not appear; as a rule, however, the heart becomes incapable of that driving power necessary to force the blood through the extensive system of capillaries which have been created for the supply of new adipose tissue. Sometimes fatty degeneration may signify simply an excess of physiological activity. In old hearts with coronary sclerosis even the bundle of His may be thoroughly fatty, and yet there may be no apparent diminution of conductivity; even well-marked fatty deposits may appear without giving rise to any clinical evidence of heart failure.

Fibroid Heart.

We now turn to the more important form of destructive disease of the heart muscle—namely, myofibrosis, or fibroid disease of the myocardium. Probably all individuals who after middle age have shown some cardiac symptoms have hearts in which fibroid changes in the myocardium would be found after careful microscopic examination. Like fatty degeneration, fibrosis of the heart muscle is the lesion most commonly associated with sudden death. Of course, the vast majority of patients with an impaired myocardium do not die during the early stages of the affection, so we do not have the opportunity of recognizing the nature of the changes which at the outset cause the impairment. By the time death has supervened the original changes

have become greatly modified, either by becoming more pronounced or by the introduction of secondary alterations.

Pathologically, in fibrosis of the myocardium we find the connective tissue increased in amount and gradually replacing the muscle tissue. As a rule it is not distributed uniformly, but is scattered in little islets or bands throughout the myocardium. These fibroid patches are generally situated deeply, and do not always extend to the surface; they may be of any size or shape, and are most commonly found in the left ventricle, the parts specially affected being in the following order: (1) musculi papillares; (2) the apical third of the left ventricle; (3) the lower part of the septum ventriculorum; (4) the posterior wall of the left ventricle about the junction of the upper and middle third. Fibrosis rarely begins in the right ventricle, but may invade it by extension from the left. Foci of fibroid degeneration, when not very extensive, may not seriously interfere with the functional integrity of the heart, but in some instances they lead to a localized dilatation.

In the hearts of children fibrous tissue is scanty; not till about the age of 30 is connective tissue apparent, and it reaches no considerable development until the decade 40–50. This gradual increase of connective tissue may be a strengthening rather than a weakening of the chamber of the heart; it may, in fact, be a regenerative or auxiliary fibrosis in which a substantial part of the fibrous tissue is laid down in support of the muscular tissue. More usually, however, there is a substitutive fibrosis in which the genuine muscular fibres are supplanted by fibrous tissue. In the same heart both kinds of fibrosis, the substitutive and what might be called the protective, are sometimes mingled in varying degrees. It may be noted that connective tissue is a less resistant tissue and its elastic limit narrower, so that when once it is outstretched it cannot recover, but it is tougher. When the circulation is slowly interfered with by sclerosis of the coronary arteries, atrophy of the muscle fibres takes place without destruction of the connective tissue, and the shrunken and atrophied fibres are gradually replaced by fibrous tissue. It is the condition of the coronary circulation which, of necessity, dominates the nutrition of the heart.

Coronary Obstruction.

The most usual pathological cause of fibrosis is obstruction of the coronary arteries, which impairs the nutrition of the heart muscle. The sudden and complete closure of a main branch may lead to instant death; short of this it produces an infarct, though the lesion may be smaller than the area which is anatomically supplied. It is still possible for the cardiac muscle to be nourished through the foramina Thebesii, which are present in all the chambers of the heart, though they are more numerous on the right side; generally they are thought to be venous in function, and proceed from the muscular substance of the heart. Even though gross infarcts of the myocardium may occur, healing may still ultimately take place. If the obstruction of the coronary arteries is gradual the onset of symptoms is necessarily insidious and the course of the disease is essentially chronic. In consequence of the alteration and slow obliteration of the coronary vessels two forms of sclerosis of the myocardium appear: (1) *Hard sclerosis*, which is characterized essentially by fibroid tissue poor in cellular elements, containing at most a few leucocytes in the lymphatic spaces. (2) *Soft sclerosis*, which is distinguished from the former by its great vascularity. This sclerotic condition is the consequence of the alteration and slow obliteration of the vessels—that is, an arteritis obliterans in the fine arteries of the myocardium followed by ischaemia of the corresponding areas.

Coronary occlusion, however, need not always imply myocardial decay; even considerable hypertrophy of the heart may coexist with coronaries obliterated by atheroma. The myocardium, too, may remain quite normal even in a condition in which the coronary arteries are calcified. We must assume in such cases that the vessels of Thebesius have been able to carry enough blood from the interior of the ventricles into the myocardium to prevent any very serious loss of its nutrition. On the other hand, there may be a very severe and extreme change of the whole heart muscle, chiefly of the left ventricle, in which the part is

converted into a dense, hard, fibrous structure, diffuse, knotty, and nodular, without any noticeable dilatation or disease of the coronary arteries. The coronary arteries are so often affected because of (1) their high origin in the aorta, where blood pressure is at its maximum, (2) their structure, which is mainly muscular, (3) their numerous and acute curves and their continued activity in supplying blood to tissues constantly in action, and (4) their relationship to the constantly contracting heart, which exposes them to jerks and contusions.

OTHER CAUSES OF MYOCARDIAL DISEASE.

Though coronary sclerosis is an important cause of myocardial degeneration it is by no means the only one. Thus, valvular lesions may bring about degenerative changes of the myocardium by perverting cardiac metabolism and giving increased work to the heart. The disturbance of the nutrition of the heart results from lack of healthy blood on the one hand and defective removal of waste products on the other. Thus, aortic stenosis lessens the amount of blood sent into the coronary arteries, while mitral stenosis, through the stasis which it induces in the right heart, interferes with an adequate flow from the coronary veins. Regurgitant lesions on the left side of the heart also cause myocardial degeneration; they exert their effect upon the heart walls largely through the increased strain upon them. In heart strain there is a disproportion between the demands made upon the heart and its supply of nutrition. When hypertrophy exists the heart requires more nutrition than normal, and if dilatation at the same time prevents it from getting this supply then there is sure to be myocardial degeneration.

Apart from these more intrinsic causes of myocardial disease there are also various extrinsic ones. These include (a) excessive physical toil and hardship; (b) *luxus consumption*, combined with too little exercise and too much alcohol and tobacco; (c) raised blood pressure from the production of toxins within the alimentary canal and their absorption into the circulating medium; these toxins may also exert a directly injurious effect upon the myocardium; (d) exhausting diseases—for example, cancer, chronic dysentery, chronic suppurating diseases, and pernicious anaemia; (e) chronic infection arising in connexion with such diseases as cholecystitis and appendicitis; (f) poisoning by phosphorus and arsenic; (g) most of the diseases which give rise to acute myocarditis; and (h) syphilis.

Syphilis, quite apart from its effect on the coronary arteries, is a most important cause of myocardial disease. It may affect the heart either by forming gummata, which vary in size and number, and which may break down and give rise to aneurysm of the heart or undergo cicatrization; or by causing the replacement of cardiac muscular tissue by a round-celled fibroid tissue which, whether it occurs as nodules only or as a more widespread change, interferes with the heart's contraction, though it cannot be said to give rise to any distinctive symptoms. After death it is not possible to tell whether the changes in the myocardium are or are not syphilitic, except in those cases where there are actual gummata.

Myocardial disease is rarely if ever universal, and the local distribution of the fibroid or fatty changes is dependent upon local and, most commonly, on vascular causes. It so happens, however, that the visceral vessels may be most seriously affected without any evidence of change in the arteries, which are accessible to examination. It is our ignorance of the condition of the coronary arteries in any given case which makes it almost impossible to estimate this, the most important factor, at its true value.

DIAGNOSIS.

The recognition of myocardial disease is by no means easy, but amid much diversity of detail there is always the same background—namely, cardiac incompetence; it is, in fact, doubtful whether there can be any cardiac incompetence without some myocardial disease. From the clinical standpoint there are two main groups of myocardial disease.

1. Latent Myocardial Disease.

In this group the symptoms referable to the heart are latent, or so disguised as to escape recognition, and the existence of heart disease is not realized till sudden death

has occurred. To these latent cases belong the sudden and unexpected deaths which not infrequently occur among middle-aged or elderly and apparently robust men. They are sometimes reported as apoplexy, but they are really instances of cardiac paralysis or sudden heart failure. This latency is due to the insidiousness of the changes going on in the heart muscle and to the position in which these changes occur—for the seat of the disease may lie in the septum, which has been called the "vital centre." It is in these cases that a bulging of the heart wall may occur, producing what is known as "aneurysm of the heart." Examination by a competent physician would probably reveal signs of general cardiac hypertrophy, together with some raised tension of the pulse. The myocardium may, for a time, respond to the heavy demands made upon it by an ever-increasing thickness of its walls; not, however, by a true hypertrophy of its muscular elements, but by a false hypertrophy due to the growth of connective tissue.

2. Cases with Cardiac Insufficiency.

In this group the symptoms of cardiac inadequacy are more or less conspicuous—namely: limitation of the field of cardiac response as shown by breathlessness on moderate exertion; palpitation; dizziness, which may be slight and transient or may amount to an annoying vertigo; epigastric fullness or pain, which the patient attributes to indigestion; and slight oedema about the ankles.

General myocardial weakness may be diagnosed when, in middle life, sometimes later on, manifestations occur of a chronic, gradually increasing cardiac weakness, a feeble pulse, sometimes very rapid, sometimes very slow, a tendency to syncope, and an extreme feeling of general weakness with increasing languor and apathy. The question naturally arises, Can myocardial degeneration be suspected before any symptoms have shown themselves?

In arriving at an answer the following points may be helpful.

1. The presence of distinctly sclerotic arteries, not only the radials but other accessible vessels.
2. The existence of an abnormally high blood pressure.
3. Increased area of deep cardiac dullness, especially towards the left, with often a triangular outline.
4. An aortic second sound, which is accentuated, and has a peculiar ringing and metallic quality.
5. At the apex a first sound, which may be high pitched and valvular, and apparently less intense than the second sound in the same area.
6. A distinct, usually harsh, systolic murmur in the aortic area, and occasionally after exercise a soft systolic murmur accompanying the first sound at the apex.
7. Urine increased at night, and giving evidence of nephritic changes.
8. An abnormal pulse rate; but the varying rate depends on so many factors that it is not easy to recognize which factor is the cause in any given case.
9. Abnormal rhythms, as in auricular fibrillation, auricular flutter, and *pulsus alternans*. Not infrequently extra-systoles are due to some damage to the conducting mechanism of the heart.
10. Lastly, an electro-cardiogram may be helpful. As a rule, electro-cardiographic tracings are mainly useful in cases of arrhythmia; when the rhythm is normal, apart from showing the preponderance of the right or left ventricle, they do not give much definite information. An inverted T wave in the first or second lead is a clear indication of myocardial degeneration; unfortunately, however, a normal T wave in these leads does not give any guarantee that the myocardium is sound. Some observers attach significance to the size of the ordinary T wave; in my opinion not much reliance can be placed on this sign in the diagnosis of the condition of the myocardium.

Diagnosis of the Severity of the Lesion.

Chronic myocardial disease is hardly ever observed alone; it is nearly always associated with different manifestations of arterio-sclerosis, and above all with renal sclerosis. A complexity of symptoms thus arises, in the midst of which it is difficult to distinguish which actually belong to chronic myocardial disease; indeed, this condition in the greater number of cases may be regarded as one of the manifestations of a general arterio-sclerosis.

So far no definite means have been discovered for estimating the strength or the weakness of the myocardium. The tests for measuring the contractile force of the heart are mostly fallacious. Physiology and pathology have shown

that the acceleration of the pulse rate after effort has no relation to the contractile force of the heart or the quality of the cardiac affection. True, indeed, some useful information may be derived by making observations on the pulse, respiration, and blood pressure just before and just after exercise, noting the time each takes to return to normal; these methods, however, give only a very general idea of the efficiency of the heart muscle. At present we have no definite mechanical device for testing the strength of the myocardium.

With regard to the functional capacity of the heart muscle, it is the degree of exhaustion of contractility which determines the nature of the subjective phenomena—for example, breathlessness, anginal symptoms, and cardiac asthma. When, however, the tonicity of the heart muscle becomes exhausted some dilatation of the heart ensues and we have the symptoms which result from this dilatation—namely, dropsy, oedema of the lungs, and so forth. When conductivity is damaged we then have either some degree of heart-block or auricular fibrillation; it is the position and character of the invasion of the myocardium which determine whether it will end in heart-block or auricular fibrillation. Even then when there is no doubt about the existence of myocardial degeneration it is still extremely difficult to make a certain prognosis.

PROGNOSIS.

The duration of life may vary from a few months to many years. In spite of the immense amount of work which has been done upon the heart we do not seem to have come much nearer to a knowledge of the strength of the cardiac muscle in any given case. The seriousness of the condition does not depend upon the total amount of muscle tissue which is converted into fibrous tissue, but rather upon the part of the heart which is affected.

In any given case could we be reasonably sure that the disease was confined to the auricles we could give a much more favourable prognosis. Probably this is why the condition of auricular fibrillation has such a varying outlook; it is essentially a lesion of the auricles, but, unfortunately, we have no means of being certain that the lesion is confined to them and has not extended to the ventricles, and it is this uncertainty which makes it necessary to give such a doubtful prognosis. I have had under my care for fourteen years a woman aged 61 with auricular fibrillation, and she does not seem very much worse now than when she first came under my care; I think one may feel fairly sure in this case that the ventricles have been but little affected. Often the history of the affection is a more useful guide to the prognosis than are the conditions present at the moment. The younger the patient the better as a rule is the prognosis, as there is less likelihood of serious degenerative changes having set in; after middle age such changes are usually present and compensatory hypertrophy is seldom re-established after it has once badly given way. The collection of numerous clinical details, both signs and symptoms, is comparatively useless in affording a prognosis unless we can form some adequate picture of the cardiac muscle at its work. For instance, is the income and output of the heart just balanced or is there a substantial reserve? If there is a reserve, is it disappearing, and how fast is it disappearing? We are too much in the habit of taking one lesion of the heart—it may be aortic regurgitation, or mitral stenosis, or auricular fibrillation—and basing on it our diagnosis, instead of considering each of these separate structural lesions as a part of the cardiac apparatus which is out of action, and estimating how much of the remainder is intact and healthy and able to carry on the necessary requirements of the circulation.

Heredity and the family history often give a valuable guide to the prognosis of myocardial disease. There are some families whose members seem to have a myocardium which begins to fail soon after middle age, without any definite antecedent disease. The outlook will usually be better when the myocardial impairment appears to be due to some rheumatic antecedent, because in these cases the lesion is more likely to remain stationary; when it is due to arterio-sclerosis the prognosis is definitely worse,

because here the lesion is likely to be progressive. Intermediate between these two causes is syphilis; here, too, the lesion is apt to be progressive and to infect a large part of the myocardium. On the other hand, if the diagnosis is made early and active antisyphilitic treatment begun, the prognosis may be much better than that of the arterio-sclerotic cases.

Dilatation of the heart has been considered as evidence of myocardial impairment; it is not so necessarily, for dilatation is merely evidence of the exhaustion of the tonicity of the heart muscle, and is no exact measure of the quality of the myocardium. We have no measure of cardiac insufficiency; the pulse is generally accelerated in conditions of myocardial disease, but this is not an absolute guide. On the other hand, great slowing of the pulse, indicative of Stokes-Adams disease and *pulsus alternans*, is definite evidence of myocardial degeneration and of grave prognostic significance; so also are syncopal attacks. The presence of angina pectoris is of bad prognosis, since in the majority of cases there is more or less interference with the coronary circulation, and thus the possibility of sudden death. Also, when the myocardial degeneration is associated with chronic nephritis the outlook is very bad; above all, the development in these cases of a cantering rhythm, implying that the ventricular wall is subjected to a strain to which it must inevitably yield, indicates that death is not far off. On the other hand, a myocardium, though greatly weakened, may continue to perform its functions fairly well, provided no extra burden is put upon it. Any illness, especially acute infections, such as influenza, may prove to be the last straw.

When the changes in the heart muscle are fatty rather than fibroid the prognosis is very serious; the more corpulent the individual the worse is the outlook, since it is hardly likely that the obesity can be materially reduced without damaging the nutrition of the heart muscle. What makes prognosis so difficult is that the heart may be performing its work with but slight evidence of anything wrong, while all the time a process of degeneration is going on which is slowly undermining its resistance.

Finally, we might quote the saying of Huchard with regard to cases of myocardial disease: "Their evolution is latent, their beginning insidious, their course paroxysmal, their progress interrupted, their visceral complications various, and their explosions of cardiac insufficiency are sudden."

TREATMENT.

The first thing to consider is the state of the heart as a whole rather than the individual lesion which has presented itself. These patients require guidance rather than definite treatment. At the outset not only physical, but mental exertion must be reduced. We have to ask ourselves how much of the heart muscle is intact; whether there are causes at work which are likely to have a constantly weakening effect upon the myocardium; or whether the lesion is practically confined to the mitral or aortic valves or auricles and likely to be stationary. The problem before us is how to prevent any further impairment of the cardiac reserve and how to make the best of the sound heart muscle which is left. Obviously we cannot in the least modify the condition of the muscle fibres of the heart when their disease is far advanced, nor can we do much to arrest the progress of the deterioration when it has reached a stage at which it is recognizable through symptoms or physical signs. In the treatment of myocardial degeneration we have to consider climate, diet, and exercise.

Climate.

It is most important to find a climate which is mild and equable, sheltered from winds and rain, and having as much sunshine as possible; on the whole, the sea coast, except of the Mediterranean, is best avoided. The patients should not go to high altitudes, especially if they have high blood pressure. The effect of a rarefied atmosphere is to increase the rate and diminish the volume of the pulse, and to increase the depth and frequency of the respirations. But very few patients have a free choice in the matter of climate, so we have to make the best of their immediate neighbourhood. We should, then, look out for a gravel or sandy soil at a moderate elevation, where the rainfall is

below and the sunshine above the average and the water not hard, conditions best realized in England in Kent, Surrey, and Sussex. The house should face towards the south, and there should be protection from the north and east; obviously it should not be situated on the top of a hill, and the immediately surrounding country should not be too hilly.

Diet.

If the patient has exercised moderation in eating and drinking there is no reason to alter his habits because some myocardial weakness has been discovered. But most patients have not been moderate, and for them there should be definite dietetic restrictions. In patients past middle age, with some degree of arterio-sclerosis and a somewhat high blood pressure, we should naturally reduce the intake of meat and enjoin abstention from meat soups and rich gravies; with advancing years a more or less vegetarian diet is better for most people, though we have to beware of flatulence and the trouble incident thereto, such as abdominal distension and palpitation. It is important that the food should be eaten slowly and thoroughly masticated. Drinking large quantities of fluid increases the labour of the heart, and should be stopped; fluids should be taken at the end of meals. Tea and coffee are permissible, but in cases of raised blood pressure their use must be sparing; they tend to excite the myocardium, and whatever puts unnecessary work upon it helps to produce its ultimate enfeeblement.

It is well to disabuse the patient's mind of the idea that alcohol will increase or sustain his strength, but if he has been in the habit of taking it in moderation there is no reason why he should not be allowed to continue to do so. In so far as it helps him to digest his food it is beneficial, but it is better taken in the form of light wine than as beer or spirits. With some people the habit of smoking has become so deeply rooted that life without it seems almost unbearable; here again we should certainly not be too rigid, for anything which promotes the attainment of mental and moral calm is most important. It is important always to inquire into the strength of the tobacco; strong tobaccos, as in Havana cigars, must be banned, for they often accelerate the heart, and sometimes seem to augment the blood pressure. It is, however, essential not to fuss the patient unduly by a too meticulous restriction of diet; it was wisely said by Montaigne, "C'est ennuyeux de conserver la vie par un régime trop sévère."

Exercise.

It is well known that the strength of the skeletal muscles is increased by exercise; no doubt the heart muscle is not strictly analogous, for the mere fact of its continued beat involves some exercise; still, if the whole body is quiescent it is hardly possible to maintain the tone of the cardiac muscle, and a certain amount of exercise, provided it is kept within strict limits and there is no dyspnoea or oedema, is therefore essential for myocardial health.

Gentle walking exercise is beneficial in the early stages of myocardial incompetence, but patients must begin their walk at a slow pace and increase their speed only as they find exercise and breathing grow easier. In particular, they should be warned against walking when there is a strong or cold wind, and forbidden to carry heavy parcels. The plan of moderate hill-climbing, as originated by Oertel, is often helpful in cases where the myocardium retains a fair measure of integrity. The principle underlying this line of treatment consists in the ascent of gentle inclines at a speed that does not cause dyspnoea or palpitation. Only when such activity can be surmounted with ease is a steeper grade to be allowed. Golf in very strict moderation may be allowed.

Whatever outdoor exercise is permitted certain restrictions should be imposed. (1) Patients must not take exercise immediately after eating; in most cases they should remain quiet for at least an hour. (2) Walking, or indeed any other exercise, should not be indulged in to the point of fatigue. (3) In cases showing some indications of incompetence rest in the recumbent posture must be insisted on at the close of exercise. Some cases are certainly helped by "resistant exercises" and Nauheim baths, or by breathing exercises and light gymnastics. These medical

gymnastics are specially helpful for patients who are obese or have abdominal fat in excess, and whose heart muscle is consequently flabby and lacking in tone; but it is essential that the myocardium should be fairly sound. Such exercises are intended to ease the work of the heart; whenever they cause palpitation or actual dyspnoea we know that the strength of the myocardium is being taxed, and not lightened of its labours. They should be performed with the assistance of some person who is trained to this work and will know how to avoid carrying the various movements to the point of causing embarrassment to the respiration and circulation. These exercises may be practised even when the patient is confined to bed, but they should stop short of producing any cardiac distress as manifested by increased frequency of the pulse. Almost any form of muscular exercise may be beneficial which promotes easier breathing and a more active circulation without producing great fatigue. Clothing should be warm, but tight clothing is specially to be avoided by women with high tension, since whatever occasions pressure upon the abdomen tends to raise blood pressure by acting on the splanchnics.

When the stage of incompetence is reached there are no measures for the treatment of myocardial insufficiency different from those applicable to the broken compensation of valvular disease.

REFERENCE.

¹ *Essai sur les maladies et lésions organiques du cœur et des gros vaisseaux.*

HYDATID DISEASE AS A CLINICAL PROBLEM:

SOME NEW ZEALAND EXPERIENCES.

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As is well known, *Taenia echinococcus*, the cestode parasite responsible for hydatid disease, completes its life cycle in two stages, which are passed in animals of separate species. As a rule the taenia stage is passed in the dog (definitive host), and the cystic or hydatid stage is very frequently passed in the sheep (intermediate host), but since the cystic stage may also occur in man, and since shepherds require dogs for the management of their flocks, "hydatid disease" in man is of fairly frequent occurrence in pastoral countries such as New Zealand, the personnel of the sheep stations becoming infested through their dogs.

Nevertheless, even in pastoral countries hydatid disease frequently presents a clinical problem of great difficulty. In a review of 90 cases in Dunedin Hospital it was found that an incorrect diagnosis had been made in 45.¹ Fairley, in a paper advocating the complement fixation test, reported in 1922 that of the cases admitted to Melbourne Hospital in ten years only 40.7 per cent. were diagnosed correctly before operation.² The likelihood of the disease is constantly impressed upon students; in any routine lecture upon haemoptysis, for instance, the common causes will be given as "phthisis, mitral stenosis, chronic bronchitis, and—in this country—hydatid of the lung"; and whereas in England the injunction is given: "In cases showing symptoms or physical signs of obscure origin—think of syphilis," in New Zealand the injunction is rather "Think of hydatid." Nevertheless, people frequently fail to think of it. This paper gives some account of the reasons for the obscurity of the condition, and also of some of the forms in which hydatid disease has been found to occur.

One reason for the difficulty in diagnosis is that the disease, formidable as it is, is not really very common. Sir Louis Barnett, a very close student of the subject, estimated twenty years ago that the incidence in New Zealand was about 100 new cases a year; he now estimates it as approaching 150, which is at much the same rate, allowing for the increase in population.³ The number, therefore, that comes under the observation of any single medical man is not great; the 90 cases in Dunedin Hospital, of about 300 beds, represent the admissions for