It is probable that there are varieties in the quality and degrees of virulence in the provoking toxin of rheumatism. As I have just remarked, we are not justified in accepting the views of Singer to the effect that the disease merely represents an attenuated form of pyaemia due to various pyogenic organisms, staphylococcal and streptococcal.

Perfect conviction on this question may perhaps only be reached after a continued and careful study by bacteriological methods of many fatal cases of chorea. Examination during life of the cerebro-spinal fluid by means of lumbar puncture, made early, may possibly reveal the specific micrococci, since they have already been found in that fluid.

In the meantime many of us must admit that the evidence in favour of the rheumatic nature of chorea is stronger, if possible, from the clinical than from the bacteriological side. Time will not fail, in my belief, to settle the question from both sources of inquiry in the direction just indicated.

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An Address

ON

DILATATION AND HYPERTROPHY OF THE HEART AND SOME OTHER POINTS IN CARDIAC PATHOLOGY.

DELIVERED BEFORE THE EASTBOURNE MEDICAL SOCIETY.

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In a recent textbook I found a murmur defined as a new sound, replacing, partly or entirely, the heart-sounds. Such a definition is clearly inadequate in respect of the second sound, and it is equally inaccurate in respect of the first.

NORMAL CARDIAC CYCLE.

If the heart's action be represented diagrammatically as a circle, that circle is interrupted by two sounds—one long, the other short—the first marking the commencement of systole and the other that of diastole. These two sounds divide the circle roughly into two parts, the longer, or diastolic, being half as long again as the shorter, or systolic. In order that two sounds should be heard distinctly they must be separated by intervals of silence. If in these intervals of silence in the normal heart's action new noises are heard, they are called murmurs. This is the essential character of a murmur. It thus stands distinguished clearly from the heart sounds, which may be audible, and apparently normal, even when murmurs are present. This is all clear on a diagram.

Fig. 1.—A is the diagram for the normal heart; B is the diagram for a heart with simple systolic and diastolic murmurs, the sounds being normal.

The heart sounds may, of course, be altered too, and may even be inaudible, the murmur taking their place more or less.

The diagram I have given of the normal heart differs from that which may be found in many books. I have drawn the first sound as not occupying more than half of the systolic period, whereas it is often made to occupy the whole, fading gradually away until the second is heard. Now, such a diagram does not represent what the ordinary ear hears, or what, in my opinion, any ear can hear. Anyway, there are many cases in which a systolic murmur is heard, and yet the two sounds appear perfectly normal in every respect, while the murmur appears to be

as long as the first sound—that is, the two divide the systole equally between them. In this case, at any rate, the first sound cannot be drawn as lasting beyond the middle of the systole. Of course it is impossible for one man to say what another can or cannot hear. Some persons cannot hear the grasshoppers on the Swiss mountains, while to another their noise is almost painful.

As there was for a long time no means of actually measuring it, the length of the first sound remained a matter of opinion. By an ingenious arrangement the following tracings of the heart sounds have been obtained by Einthoven and Geluk,¹ which seem to show that the diagram as I have drawn it is correct:

Fig. 2.—Cardiogram of Man: I, II, the first and second heart sounds.

Careful clinical observation could hardly, I think, lead to any other conclusion.

The next question I should like to discuss is the cause of the second sound at the apex. The second sound at the base is produced by the closure of the aortic and pulmonary valves. It is, I believe, commonly taught that the second sound at the apex is the base-sound transmitted to the apex. Clinical observation shows that this explanation is inadequate, and that there is no such simple relation between the second sound at the base and at the apex as that theory would require. The second sound may be loud at the apex when it is faint at the base, or it may be loud at the base and unusually faint at the apex. Lastly, it may be completely absent at the apex when it is loud at the base, and this is what happens in a typical case of mitral stenosis. At the apex the long rumbling pre-systolic murmur leads up to a long and accentuated first sound, but the second is absent, while at the base the pulmonary second sound is markedly accentuated, and the aortic is distinct and often apparently not less loud than in the normal heart. The pathological lesion suggests an explanation, the mitral orifice is reduced to a narrow slit, the so-called button-hole. With a healthy orifice the blood passes readily through into the ventricle with some force, which when the valve is highly stenosed it cannot do. The impact of the blood, then, in health upon the ventricular wall might be sufficient to cause a sound, but in mitral stenosis the blood cannot rush in, but trickles in, and the impact is inaudible.

I do not know to whom this explanation is due, but it seems to me the only one possible, and if correct we must conclude that the second sound at the apex is not a transmitted sound only, but is in great part produced in the ventricle itself.

It is important in auscultating the heart to distinguish clearly between the heart sounds and the murmurs, and to study each carefully, for it may be stated in general terms that where with murmurs the heart sounds are present, the lesion is less and the prognosis therefore better, than in cases in which the heart sounds are greatly modified or absent.

Even where there are no murmurs the characters of the heart sounds have great clinical importance. At the base an accentuated pulmonary second sound points to increased pressure or tension in the pulmonary vessels. In the aorta it indicates increased arterial tension, and may give a useful hint as to the disease, as in granular kidney or thoracic aneurysm.

The character of the first sound over the ventricle is of greater importance, as it gives an indication of the strength of the cardiac contraction. In fever it is of special significance, as Stokes long ago pointed out, for it may be weak or even almost inaudible in cases of fever which might otherwise appear mild. This indicates the need of stimulants, cardiac or general, and is of even more value in this respect than is the pulse. As an illustration I may refer to a case of typhoid fever, so mild as to cause no anxiety if it had not been for the great enfeeblement, almost absence, of the first heart sound. A severe relapse

occurred, in which the patient with difficulty escaped with his life.

In acute pneumonia, again, more can be learnt from the first sound of the heart than from feeling the pulse.

I do not wish to pursue this subject further now, and will pass on to another—namely, that of dilatation of the heart and the murmurs associated with it.

The amount of work which the heart in health is called upon to do varies within very wide limits, but so long as the heart is healthy it remains equal to the work required of it. If P be the power of the heart and W the work it has to do the equation of health would be:

$$P = \text{and } \propto W.$$

Where the increase in work is permanent the heart responds by growth and becomes larger or hypertrophied. If W becomes W , P becomes P , but the equation is still preserved. This is what occurs in training. Similarly, if W returns to W , P must return to P —the equation still being preserved. This happens on going out of training.

Hypertrophy or growth takes place at the expense of the cardiac reserves, so that a large heart has diminished reserves, and the greater the hypertrophy the less is reserve. By and by the limit might even be reached and all the reserves be exhausted by growth, and thus is explained the relation which exists between hypertrophy and dilatation. As soon as the work required of the heart is greater than the heart can do, the equation

$$P = W$$

is destroyed. P becomes less than W . The equation, then, for a weak heart is

$$P < W.$$

A weak heart dilates, and therefore this is also the equation for dilatation.

This may serve to render clear what seems to present a difficulty—namely, to understand how it is that hypertrophy so easily passes into dilatation. Hypertrophy is really a condition of disease, because though the heart is large the reserves are small.

A diagram again will make this clear. In any given case the force of a heart and its reserves at any given

time may be regarded as fixed. Under ordinary conditions the reserves R are large; but all hypertrophy encroaches on these reserves as the diagrams indicate, until where hypertrophy is excessive the reserves may almost or entirely disappear.

For growth time is necessary. Training consists in getting the heart and muscle ready for the supreme effort of the race. To strike the balance between the growth and the re-

serves constitutes the difficulty of proper training, which has, moreover, to take into calculation the personal peculiarities of each individual. To bring a boat's crew of eight men up to the scratch all in their fittest condition is a task which obviously requires much skill and care. Over-training means unduly diminished reserves. Under-training, diminished growth.

HEART STRAIN.

Sudden and prolonged effort without preparation for it is very likely to prove more than the heart can stand, and explains the cardiac trouble which may follow the running or rowing a race without training at all. This is what is often called *heart sprain* or *heart strain*. The symptoms are those of cardiac weakness—pain, palpitation, and shortness of breath on exertion—and the physical signs are those of dilatation. A heart once severely strained may take a long time to recover. If this is true of an ordinary muscle, how much more likely is it to be

the case with the heart, which cannot be rested to the same extent. In disease the best illustration of the relation between hypertrophy and dilatation is found in granular kidney. Here the gradual increase of work leads to hypertrophy, which in time reaches its limit, or cannot be maintained, and then the signs of cardiac failure appear which mark so often the later stages of that disease.

Of course in all diseases of the heart, if the lesion throws extra demands upon any part of it, that part hypertrophies in the same way, and is liable to similar risks of dilatation.

I do not propose to consider further those cases in which the cardiac failure is due to increase of work. I will turn now to the more interesting group where the result is due to failure of the heart's power.

To make the matter simple I will assume that the work is of normal or ordinary amount, so that W is constant and when the equation $P < W$ is arrived at by diminution in the value of P , it matters not what the actual values of P and W are, that is, whether there be previous hypertrophy or morbus cordis or not. In any case as soon as P , becomes less than W dilatation occurs. The evidence of it will be the symptoms of cardiac weakness—namely, pain, palpitation and shortness of breath, and the physical signs of which the chief is increase in size of the cardiac dullness.

The first group of cases of this kind I will refer to is that in which the muscular power P has been reduced by previous illness, that in which the dilatation is met with during convalescence. This is especially common in children after some specific fever, notably diphtheria, and that, too, in cases where the heart has not been in any way involved during the acute illness. Symptoms may or may not be present, but the increase of cardiac dullness is manifest. This may be obvious when the child is still in bed, but if not, is easily produced by the child being up for an hour or two, the mere extra effort due to the erect position being sufficient to demonstrate the cardiac weakness which exists.

I have more than once been asked to see a child in consultation, because although there had been no other signs of cardiac disease the increase in cardiac dullness was so marked. The child has been pale and poorly, and disinclined to move about, though it did not complain of any cardiac symptoms. Besides the increased cardiac dullness the heart's action may be excitable and somewhat irregular. As convalescence proceeds and health improves the dullness diminishes, and the heart recovers itself.

This weakness of the heart may often be easily demonstrated in the adult too. I recall two cases of rheumatic fever, in both of which the heart had escaped mischief. The cardiac dullness was marked out in each before they were allowed to get up. The next day both were found with a much increased cardiac area, and the apex displaced outwards, without, however, other definite symptoms.

The one patient was kept in bed; the dilatation immediately disappeared, and when the patient was allowed to get up again, a week later, the dilatation did not recur. The other was allowed to remain up. In this patient, too, the dilatation slowly passed away, but only after several days. These cases are interesting as showing the undesirability of hurrying convalescents up, especially after diseases such as diphtheria, influenza, or rheumatic fever, in which there is special tendency for the heart to be affected.

Another interesting group of cases is that in which morbus cordis has been known to be present for a long time, yet no symptoms have shown themselves, till all of a sudden they appear, and never disappear again. A case I often refer to is that of a gentleman of middle age who was known for years to have aortic regurgitation, and had been warned to be careful, but having no discomfort he disregarded the warning. He was an athlete for his age, played football with his boys, was a great swimmer and walker. One day he saw two boys in the river out of their depth and drowning. He swam out, and brought them both to shore. He arrived greatly exhausted, and had an attack of angina on the bank. From that day he was a cardiac cripple, and died a few months later of gradual cardiac failure.

In other cases the onset of symptoms may date from a nervous shock and emotional disturbance, or from some acute illness such as influenza, pneumonia, or typhoid.

Fig. 3.

In all these cases the explanation is simple. The previous disease had drawn largely on the reserves which were sufficient for ordinary needs, but not enough for any sudden call, whether muscular effort, or nervous shock, or the strain of illness. The equation was destroyed, P became less than W, and the cardiac failure once started could not be recovered from. Cases of this kind could be easily multiplied.

DILATATION MURMURS.

Where the heart is dilated murmurs are often heard. They are not organic, for they come and go with the dilatation. How are they produced?

It is often stated that the mitral and tricuspid orifices are too large to be completely closed by the valves unless they are narrowed by the contraction of the muscles which surround them. Thus weak muscular contraction would render the valves incompetent. The muscle is weak when the heart is dilated, therefore it follows that the murmurs with dilatation are due to regurgitation. There are grave clinical objections to this explanation, which prevent it from being regarded as satisfactory. *A priori* it is contrary to what we know of other organs, and above all things unlikely in the case of so important an organ as the heart, to believe that the margin between health and disease is so narrow. Half the kidney, half the liver, and half the lungs at least may be useless and yet life be carried on without much distress. Is it likely, when we consider the serious consequences of mitral or tricuspid incompetence, that the heart alone should have such a narrow margin to work on as the theory assumes?

A priori objections are, however, of no great value, and my objections are clinical. The so-called "dilatation murmurs" are systolic in time, are heard at the apex and over the precordium, and but rarely outside its limits. Any way, they are not propagated in the direction they ought to be. An organic mitral regurgitant murmur is heard at the apex, is propagated some distance out into the left axilla, and is audible behind. This, at any rate, is so in the great majority of cases. So, again, a tricuspid regurgitant murmur is audible beneath the lower part of the sternum, and is propagated horizontally across the sternum to the right. We may fairly ask, Why is this so with organic disease and not so with dilatation if the cause of the murmur in each case is the same—namely, incompetency of the valves, sufficient to allow of regurgitation?

I cannot, therefore, believe that this theory is true. It is rash, perhaps, to question statements which rest on the authority of Ludwig, but I must say that the experiments upon which they are based are of a violent and unnatural kind and hardly justify so sweeping a generalization. I therefore believe that dilatation murmurs are not due to regurgitation, but to intraventricular causes—that is, to the dilatation of the ventricle, just as the systolic murmurs are produced over a dilatation or aneurysm of the aorta.

Before leaving this subject I should like to say a word upon the diagnosis of tricuspid regurgitation. The signs of dilatation of the right side of the heart with marked venous pulsation in the neck is often thought sufficient for the diagnosis even in the absence of a murmur, and if a systolic murmur is present as well the diagnosis is regarded as certain. Without a systolic murmur propagated in the right direction I do not consider the diagnosis justified. Tricuspid incompetence the result of organic disease is rare, but it has been my fortune on a few occasions to make the diagnosis during life, and on *post-mortem* examination to find the lesion present. Now I must repeat the question—Why is the murmur propagated in the characteristic way with organic disease of the valves and not so propagated with simple dilatation if in both cases the cause of the murmur be regurgitation?

The three cardinal symptoms of heart affection are palpitation, pain, and shortness of breath.

I wish to conclude with a few remarks on palpitation and pain.

PALPITATION.

It is remarkable how extensive the organic lesions of the heart may be and how long they may continue without causing much or any discomfort to the patient. It is with affections of the heart especially that

the contrast is striking between the amount of disease or discomfort the patient suffers and his disease in the morbid anatomy sense of the term. Palpitation is, of course, common enough with a weak or failing heart, whether it be organically diseased or not; but where palpitation is complained of as the prominent trouble by the patient the heart is more often than not free from organic disease. Conversely, if with organic disease of the heart palpitation be an unusually pronounced symptom, the question would arise whether there were not some other condition besides the mere heart lesion to account for it—for example, exophthalmic goitre or some neurosis.

Exophthalmic goitre is often selected as the disease to prove that long-continued palpitation may lead to valvular disease, the condition resulting being usually mitral incompetence. The value of this disease for the purpose is discounted by its frequent association with rheumatic fever. In going through the cases of exophthalmic goitre some years ago at St. Bartholomew's Hospital I stumbled quite accidentally upon this association, which I found as frequent as 11 per cent., and the frequency with which valvular disease is met with is not greater than this association with rheumatic fever would explain. Severe palpitation may be sometimes associated with organic disease, and if it be present may aggravate it, but I do not think there is any satisfactory evidence to prove that palpitation alone can produce it.

TACHYCARDIA.

Palpitation is usually associated with rapid action of the heart, and these cases are now often described under the heading of tachycardia. Yet so far as the patient is concerned it is not the rapid action but the palpitation which causes distress. Tachycardia is a term which has come to be used so widely and indiscriminately that it has ceased to have any special meaning. Any patient whose pulse beats more rapidly than normal may be said to have tachycardia, but this does not convey much information. The use of a technical term like tachycardia would seem to imply some peculiar clinical condition. There is such a condition, but it is often lost sight of in the crowd of unimportant conditions included under the term. There is a group of cases in which the rapidity of the pulse is extraordinary, 200, 250, or 300 in the minute, and in which this constitutes apparently the whole disease.

Some of the earliest cases were recorded by Bristowe under the name "attacks of recurrent palpitation in the apparently healthy." The objection to this title is that it is not so much the palpitation as the rapidity of the heart beats that is so characteristic, and, further, there is good reason to believe that, however healthy the heart may seem, it is often diseased; at any rate, many of the cases recorded ultimately died as heart cases often do. I suggested some time later, with the view of giving a definite name to the group, "paroxysmal hurry of the heart," but that has now become "paroxysmal tachycardia," a good term if only it had not been so indefinitely extended. The most remarkable case I can recall of this kind was in a robust policeman who came complaining of his heart making such a noise that his neighbour in the dormitory was kept awake by it. He had a loud musical systolic murmur which could be heard at some distance from the chest walls, although I never myself heard it at a greater distance than 18 in. The murmur was occasional, and occurred only during the attacks of tachycardia. Then the heart would beat 250 in the minute, faster, indeed, almost than one could count. The heart's apex would then be a couple of inches outside the nipple, and the cardiac dullness much enlarged, especially to the left. The murmur came and went with the dilatation, and there was no evidence whatever, from shortness of breath or cough, that there was any mitral regurgitation. Indeed, it was extraordinary how little discomfort the attacks produced, and how little attention the patient paid to it. He had many attacks while under observation, but ultimately got quite well and returned to active service.

CARDIAC COLIC.

The last subject upon which I have a few remarks to offer is cardiac pain. This varies in degree from mere discomfort, the mere sense of having a heart, up to the most severe attacks which are termed angina. The great characteristic of all cardiac pain is the anxiety with which

it is associated, the patients seem conscious that the pain, even when slight, is of serious import. The emotional side of heart disease is a subject upon which much might be said. Cardiac pain may be regarded as the outcry of a weak or overtaxed heart. It expresses the struggle of an over-distended heart to empty itself. Consequently dilatation will be the factor common to them all. The struggle will be greatest and the resulting pain most severe in those parts of the heart where the muscles are biggest. The right auricle may be very greatly distended with little more than discomfort. Pain will be more marked where the right ventricle is concerned, but all the most severe attacks of pain are associated with the left heart, and especially with the left ventricle. One other factor must be borne in mind. It is this, that the call upon the heart and the consequent distension must be more or less sudden—gradual distension may occur without pain, and reach even a higher degree; but sudden distension leads to an attack of pain. This, of course, is a matter of daily experience in left ventricle affections. The first attack of angina may follow exertion, as in the case I described above. Afterwards an attack may be produced by very slight effort, even that of walking a short distance fast or uphill.

The nature and cause of angina has been much discussed, but I think all we really know about it may be summed up in the term "cardiac colic." I do not know of anything observed in angina which cannot be seen in other forms of colic. The pallor, faintness or collapse, the small arteries, dilated pupils, and various conditions supposed to stand in some causal relation with angina may all be equally seen in biliary, renal, and intestinal colic.

The pain of a distended urinary bladder or of a pregnant uterus at the commencement of parturition is strictly comparable with the pain of angina. In all cases alike it is the struggle of the muscular organ to get rid of its contents, and the spasmodic contraction of the muscles is attended with the paroxysms of pain. As soon as the organ has emptied itself the pain ceases. If it cannot get rid of its contents, the paroxysms become less and less severe as the muscles become feeble, and finally a condition of paralytic distension develops in which the pains cease. So it is also in the heart, the most violent attacks of angina occur generally when the heart muscle is still fairly strong. As the heart fails the attacks may become less severe, and when extreme dilatation occurs and the other signs of cardiac failure appear, the tendency to severe angina disappears.

The term "cardiac colic" removes angina from the little niche in which it stood all by itself, and places it in the general group of colic about which we know a good deal, though much remains still to be learnt.

REFERENCE.

¹ Arch. f. Ger. Physiol., 1904, lvii, 607.

A Lecture

ON

CONGENITAL HEART DISEASE.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
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LADIES AND GENTLEMEN,—There is much that might be said upon this subject which would interest the pathologist and embryologist, but the time at our disposal compels me to restrict myself to the more purely practical side. We can readily group the morbid changes in congenital heart disease under two headings:

1. Those due to malformation.
2. Those due to intrauterine endocarditis.

The first group can then for convenience sake be classified into three subgroups:

Subgroup I.—Arrest of development in early fetal life: Such results follow as the formation of hearts with two chambers only, a ventricle and an auricle; or possibly there may be two auricles and one ventricle. These conditions are incompatible with life.

Subgroup II.—Arrest later in fetal life: In such cases the auricles and ventricles though developed are imperfect, and the aorta and pulmonary artery may be partially developed or the large vessels displaced.

Subgroup III.—Defects at the end of fetal life; for instance, premature closure of the foramen ovale, or premature obliteration of the ductus arteriosus.

The second group of cases—those resulting from intrauterine inflammation—are of more interest, and in this group are to be found remarkable examples of fetal endocarditis, probably rheumatic in origin, of which the following case from the Museum Catalogue of University College Hospital is a striking instance:

In a child who lived for thirty days there was discovered the day after birth a loud systolic murmur which was heard all over the precordium and at the back. It was a small, quiet, and pale infant, but, except for occasional blueness of the lower eyelids, showed no cyanosis. The mother had suffered twice from rheumatic fever, and was attacked a third time during her pregnancy. Her three other children were healthy. The necropsy showed that the interventricular septum was deficient at the upper and back part, and to the margins of the aperture the cusps of the mitral and one cusp of the tricuspid valve were adherent. Both valves were beaded by vegetations. The foramen ovale was patent, as also the ductus arteriosus. The other valves were natural.

Another striking example which came under my own observation was that of a child born cyanosed and in an almost asphyxiated condition. Death followed on the third day. During pregnancy the mother had suffered from a severe attack of rheumatic fever. The necropsy showed extensive and recent mitral endocarditis.

The diagnosis of this form of heart disease has been made even in intrauterine life, as in those cases reported by Peters and others in which a murmur has been detected in the fetal heart.

In the second group, then, the important lesions are connected with fetal endocarditis and consequent anomalies of the valves and cardiac septa.

The relation of congenital heart disease to a family history of rheumatism is a point of some interest, and I have come upon several examples of this occurrence.

CASE I.—In 1905, a boy, aged 2, was brought here with congenital heart disease, and a history of rheumatic arthritis in pregnancy was given by the mother.

CASE II.—E. D., aged 9 months, suffered from congenital heart disease, the mother from rheumatic arthritis, and the maternal grandmother died from heart disease consequent on rheumatic fever.

CASE III.—An infant of 2 years with congenital heart disease, whose maternal grandmother suffered from severe rheumatic fever, and his uncle also from rheumatic fever.

CASE IV.—H. S., aged 3 years. Congenital morbus cordis. Maternal grandfather died of rheumatic fever with heart disease. The mother of the child had five married sisters, each with a child who was cyanosed on exertion and short of breath.

CASE V.—G. R., aged 2 months. Congenital heart disease. His mother had repeatedly suffered from swollen joints.

These cases, with the two I have alluded to more fully, will serve as a reminder of the importance of the rheumatic infection in intrauterine life. Congenital syphilis is also a cause of congenital cardiac disease, although, as it so happens, I have rarely met with a convincing example of its occurrence.

The most important clinical cases in this group are those in which there is narrowing of the pulmonary artery. This is the form of congenital heart disease in which life is more likely to be prolonged to adult age than in any other.

There is frequently associated with this stenosis a patent interventricular septum. This opening is, as a rule, a small one at the upper part of the ventricular septum, in the membranous undefended space. In addition, the foramen ovale may be patent and the ductus arteriosus incompletely closed.

Stenosis of the aortic or mitral valves are also met with occasionally, and the tricuspid valve may be damaged, but such conditions are far less common than the pulmonary lesion.

The symptoms of congenital heart disease, where they are well marked, are remarkable ones, but in infancy they are sometimes somewhat indefinite. The most frequent of all manifestations, cyanosis, usually attracts attention at birth, but it would be an error to suppose that infants