Henry Lawrence McKisack (1859–1928)

President of the Ulster Medical Society 1911–12

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NON-VALVULAR HEART DISEASE.

Ladies and Gentlemen, I have to thank you very sincerely for the honour you have done me in appointing me to preside over the Society for the ensuing year. With the co-operation of the energetic honorary secretary, Dr. Rankin, the Members of Council of the Society, and the Members generally, I am certain we shall have the advantage of an active and interesting Session.

During the past year the Members and Fellows of the Society have been gratified by the honour paid to one of their number, Sir Alexander Dempsey, to whom we offer our heartiest congratulations and best wishes on his well merited distinction, and I am sure we all wish him long life to enjoy it.

We have, unfortunately, to deplore the loss of a distinguished Fellow, the late Dr. W. B. M'Quitty, of whose eminence in his profession it is unnecessary to remind you. His contributions to the proceedings of the Society were numerous, and were marked by wide and extensive experience, and reasoned insight in the whole field of medicine. His lamented death is a loss to the Society, which touches every Member and Fellow.

The Library of the Society has been well looked after by our honorary librarian, Dr. Storey, and I understand Members have availed themselves largely of its advantages.

The new index which has been inaugurated during the year, under Dr. Storey's superintendence, has proved a great success, and facilitates immensely reference to the volumes on our shelves.

The fabric of our building has been well looked after, and, thanks to the Trustees and other responsible officers, the building is in the best of order.

We are indebted to Mr. John M'Kee for a valuable picture which he presented to the Society in memory of his son, Dr. M'Kee, of Harrogate, who died in February, 1910. His death was much regretted by the Ulster Medical Society, who recollect an interesting contribution which he. made to the proceedings of

the Society, on the Harrogate treatment.

The subject which I wish to bring before you this evening is one of interest to us all, and one which almost daily gives rise to problems not always easily solved. I refer to the well-worn subject of disease of the heart, and I wish especially to draw your attention to the large group of cases of heart disease, which, as to their origin, are, so far as one can judge, independent of valvular disease.

It is of course familiar to every student of medicine that disease of the heart may exist without disease of the valves, but I think too much attention is usually devoted in text books, and at the bedside, to the condition of the valves, and not enough to the condition of the muscle of the heart.

It may be asked if it is worth while conjecturing as to the part played by the various portions of the circulatory apparatus in the production of heart defects. There can, I think, be no doubt as to the answer. It certainly is worth the trouble to investigate the cause, since upon our accurate knowledge of the origin of the defect, must depend our appreciation of

the nature and effects of the diseased process; thereby we obtain a reasonable guide to treatment and prognosis. For example, we know that a case in which cicatricial changes in the valve structures have been the cause of heart-failure cannot hope to get rid of the valvular defect, and we know that the lesion may be progressive; treatment must be directed to assisting the crippled heart to perform its duties unhindered by accidental or removable conditions; while an ineffectual heart, weakened and dilated by some temporary or removable blood-state or strain, offers good hope of more or less perfect recovery when the cause of disturbance is removed and the individual is placed under favourable conditions which may assist him to effect his own cure. Again, certain muscular defects, such as fibrous, fatty or atheromatous degenerations, may tend to progress unfavourably, so the prognosis and treatment of such cases must receive appropriate modification.

In addition to the practical utility of this classification, one has the satisfaction of gaining a clearer conception of the conditions which co-operate to produce the enfeebled heart, and a more rational explanation of the various phases of inefficiency which the heart presents in the course of the complaint.

There is a certain confusion in the use of the term heart-failure, or heart-inefficiency. By some it is taken to indicate syncope and complete prostration; by others to be any degree of impairment of the action of the heart which may be observed by the physician. The normal heart is provided with a generous store of "reserve force," over and above the "working force," which suffices for the needs of the circulation while the body is at rest. Should the heart be damaged by any means, the reserve force may be enough to enable the individual to perform comfortably the usual activities to which his habits and age are suitable; under these circumstances there is no heart-failure. If on the other hand he finds that he is unable to call up such ordinary muscular energy without showing the well-known signs of heart-inefficiency he may be regarded as a subject of heart-failure. Therefore, I would define heart-failure as a condition in which the heart's function is obviously disturbed by an amount of bodily exertion which should cause no inconvenience to a normal individual of the same age and habits.

There are two main groups of conditions in which heart-failure occurs, viz., those originating in disease of the valve structures, and those arising from lesion of the cardiac muscle.

Regarding the first group it is to be remembered that endocarditis which produces such a severe crippling of the organ as to render it ineffectual in spite of its large reserve power, must be very destructive. It has not been shown by *post-mortem* examination that recent simple endocarditis due to rheumatism or other blood poison can produce such extensive valvular lesions as to upset the efficiency of the heart; such destructive effect could only be exercised on the valves by the more active necrotic processes of infective or ulcerative endocarditis. Should, however, the inflammation of the valve structures have commenced or taken place some time previously, the cicatricial contraction, which usually follows the deposition of inflammatory products in the tissues, may result in such deformity of the valve structures as to render the valve mechanism seriously defective. We may, therefore, assume that heart failure occurring in the course of a recent endocarditis (rheumatic or otherwise) cannot be the result of disease of the valves alone, but when an interval of time has elapsed since the onset of endocarditis, the probability of valvular origin of the heart failure is considerable. It must also be borne in mind that definite changes of an inflammatory character have been found in the muscle of the heart in cases of rheumatism, and also of many other fevers, such as influenza, the exanthemata, tuberculosis, etc. Owing to these changes, the heart muscle is unfit to stand the strain of any considerable exertion, and the condition of muscular tone which maintains the normal size and capacity of the heart-chambers is interfered with. This is the chief factor in the cases of heart-failure occurring early in first attacks of acute rheumatism.

The bulk of the cases of heart-inefficiency due to valvular disease are then the result of long-continued and progressive cicatricial changes, rather than active destructive inflammation of the valves chiefly affecting the left side of the heart, and resulting not only in incompetence of the orifice affected, but very frequently, as a result of cicatricial contraction, in narrowing of the orifice. This specially applies to the mitral orifice, but in many cases it also applies to the aortic.

The remaining groups of valvular heart affections, *viz*., those due to disease spreading from the aorta, and those resulting from traumatism or tumour formation need not detain us now, as it is of those instances of heart-failure not the result of valve disease that I wish specially to speak. The chief causes which thus damage the heart-muscle are either inflammatory changes as above mentioned, or degeneration of the muscle fibres. The inflammatory

injury is commonly the result of the toxaemia of fevers, the chief of which is rheumatism, but familiar examples are seen in pneumonia, measles, scarlet fever, typhoid, and other exanthemata. A cloudy swelling of the muscle fibres occurs in almost all pyrexial states, and more definite inflammatory infiltration is common under these circumstances.

The degenerations are even more numerous and striking in their effects. Among the commonest is the fatty change caused by an imperfect blood supply to the muscle. This is found to occur in all cases of severe anaemia, *e.g.*, pernicious anaemia, chlorosis, haemorrhage, inanition from improper or insufficient food, and it is one of the factors in the heart muscle weakness of acute rheumatism and other fevers, in which the blood examination usually reveals a marked anaemia. Mechanical interference with the proper blood-supply to the heart walls is a not infrequent cause of fatty and fibrous degeneration, and occurs in atheroma, thrombosis or embolism of the coronary arteries. Fibrous changes may occur in the heart muscle, analogous to those occuring in the systemic arteries, and probably owing their presence to similar causes, *viz*., strain, toxaemias, especially gout, high tension in the circulation generally, and especially as found in the contracted kidney. This condition of cardio-sclerosis is also likely to result from long-continued efforts of a heart, damaged by valve-defects, to maintain an efficient circulation, and is the usual form of degeneration which attacks a heart hypertrophied by this or similar causes, and hastens the dilatation and breaking down of compensation under unfavourable circumstances.

A brief reference will suffice for a third group of heart failures, those due to nervous lesions. The syncope and cardiac failure occurring after influenza and diphtheria are often of this nature, though the pyrexial changes in the muscle fibres may also be responsible.

It is generally admitted by observers that the decision as to whether a heart lesion is of valvular or of muscular origin is in many cases difficult and at times impossible. Before going farther into the question I should like to quote briefly the opinions of a few authorities. Most of the quotations have reference to that valvular lesion which is so common, and in which it is so difficult to accurately allocate the due proportions of each factor, muscular and valvular, in the production of heart failure – namely, mitral regurgitation.

Sir Clifford Allbutt warns the unwary not to mistake the anaemia of insidious acute rheumatism with heart lesion, for chlorosis with but a temporary relaxation and dilatation of the structures about the orifice.

G. W. Balfour regarded mitral regurgitation as only possibly due to valvular disease – the only proof of actual disease of the mitral valve being mitral stenosis as indicated by an auriculo-systolic murmur. He spoke of "curable mitral regurgitation," which he affirmed was due to muscular relaxation.

Dr. Byrom Bramwell is of the opinion that the murmur of mitral regurgitation is a strong evidence of endocarditis. In the early stages of acute rheumatism the murmur may exceptionally be due to anaemia, and in later stages it may result from endo, or myocarditis, or from anaemia, or from simple muscular relaxation, such as is met with in all fevers.

Sir Wm. Broadbent enumerated the following points as aids to distinguish mitral regurgitation of valvular origin from that due to dilatation:– When a murmur is soft and blowing, heard not only at the apex, but also at the aortic and pulmonary regions, in a patient who is not known to have had rheumatism, it is probably due to dilatation. After acute febrile conditions, including rheumatism, and after chorea, the murmur of mitral incompetence may be only an indication of myocardial change. In the mitral regurgitation of middle or old age, there is commonly considerable dilatation of the orifice, and this often occurs with high arterial tension.

Dr. Lindsay says the distinction between valvular and muscular lesion as a cause for the murmur of mitral regurgitation should depend on (*a*) the history and constitutional state; (*b*) the condition of the left ventricle, and the position and character of the cardiac impulse; (*c*) the site of maximum intensity of the murmur, its conduction, the variations in its characters depending on posture and movement; (*d*) the results of treatment. Those cases in which the murmur under treatment disappears may be assumed to be independent of organic disease of the valves.

Dr. James MacKenzie believes that weakness of the heart muscle is mainly responsible for the cardiac failure in cases of mitral regurgitation, and that the weaknesses not due so much to backward pressure from damaged valves as to inflammatory or degenerative change in the muscles, causing loss of muscular tone. In the course of a febrile attack, even of acute rheumatism, the tonicity of the poisoned heart-muscle may fail, giving rise to incompetence of the mitral orifice.

Sir Wm. Osler states that in cases of hypertrophy and dilatation of the heart, without valvular disease, but associated with heavy work and alcohol, the insufficiency of the mitral valve may be extreme, and

lead to great pulmonary congestion, engorgement of the systemic veins, and a condition of cardiac dropsy which cannot be distinguished by any feature from that of mitral incompetency due to lesion of the valve itself. In chronic Bright's disease the hypertrophy of the left ventricle may gradually fail, leading in the later stages to relative insufficiency of the mitral valve, and the production of a condition of pulmonary and systemic congestion, similar to that induced by the most extreme grade of the valve lesion. It is not always possible to say whether mitral insufficiency is due to lesion of valve segments or to dilatation of the mitral ring and relative incompetency. Here neither the character of the murmur, the propagation, the accentuation of the pulmonary sound, nor the hypertrophy assists the differentiation. The history is sometimes of greater value in this matter than physical examination. The cases most likely to lead to error are those of the so-called idiopathic dilatation and hypertrophy of the heart, and instances of arterio-sclerosis with dilated heart.

Sansom. The effects of endocarditis cause thickening and deformity of the valve structures which render it incompetent. Also some narrowing often results, but the signs may be only those of insufficiency. He quoted Duroziez as saying that if the orifice admits the thumb there will be no signs of stenosis. This, however, depends on whether the internal surface be rough or smooth. The murmur in cases of dilatation is due to imperfect apposition of the valve cusps. The history is the chief guide in distinguishing the dilatation cases from the valvular.

Dr. Graham Steell declares that there is no structural disease of the valves in a very large number of cases of mitral incompetence, but that the want of muscular assistance prevents the valves from performing their function. He points out the importance of muscular efficiency in the maintenance of the mitral valve function, and reminds us that endocarditis is often or probably always accompanied by myocarditis. In some cases of rheumatic endocarditis the damage is so trifling that it cannot cause serious leakage, but the dilatation even in such mild cases is common. Here the dilated muscle is the *cause*, not the *result*, of regurgitation. Cicatricial changes occur slowly and if progressive are likely to end in stenosis, as well as reflux.

Sir Wm. Whitla regards the diagnosis of myocardial degeneration from the dilatation of valvular disease as difficult but points out that the hypertrophy and dilatation of valvular disease are much greater than those of myocardial degeneration or infiltration; also in the latter condition evidence of fibroid change in the systemic arteries will be present. As regards the causation of dilatation, Whitla shows that in cases of acute endocarditis or pericarditis of rheumatic fever, acute dilatation may occur independently of valvular or mechanical causes. He also specially draws attention to the occurrence of acute dilatation after influenza and diphtheria. In these affections as well as in other pyrexial conditions the changes in the muscle fibres are sufficient to cause their stretching and loss of tone even with normal arterial tension.

There is in the main a unanimity of view among these observers: all are of the opinion that mitral regurgitation is commonly produced by dilatation of the walls of the heart, as well as by damage to the valve structures. Some lay more stress on this muscular factor than others: Balfour, Graham Steell, and MacKenzie may be mentioned as apparently regarding the valve element as less important than the muscular. It will, I think, be unnecessary to occupy your time with arguments to prove the importance of properly contracting heart muscle, to ensure the efficient closure of the mitral valve – you are all aware of the part played, not only by the musculi papillares, but also as was shown as long ago as 1882 by M'Allister, by the sphincterlike ring guarding the orifice. What, I think, is not sufficiently realised by us all, is that mitral regurgitation is a not very rare result of pure muscle lesion in rheumatic cases, and is a very common outcome of muscular debility in numerous other morbid states.

On examining a case of heart-failure we generally find some evidence of enlargement of the heart, usually one or more murmurs indicating possible valvular lesions, and then a series of secondary results of defective circulation, shown by the familiar disturbances of liver, lungs, etc., with edema, dyspnoea, and other indications of the inefficient heart. To the observer the most striking abnormality is usually the murmurs, which perhaps unduly occupy one's attention in such circumstances. I will refrain from taking you into the enquiry whether these murmurs are in all cases indications of actual lesion or are only functional or accidental sounds which have no pathological significance, and I shall restrict my remarks to the question whether the various murmurs to be heard in cases of heart-failure are the result of lesions of the actual valve structures, or arise from damage to the. walls of the heart-chambers. The solution of this problem lies at the root of the whole subject of the origin of heart-failure, and I propose to take up as briefly as possible the various murmurs and the lesions which our experience leads us to

believe they indicate.

In illustration I shall briefly refer to a series of cases of heart-failure of which I have examined the records, They are 200 consecutive cases admitted to the wards of the Royal Victoria Hospital as heart disease, and the diagnoses placed the lesions in the following proportions:–

Mitral Stenosis alone, or with Mitral Regurgitation	37 cases	60
Mitral Stenosis, with Aortic disease	23 "	
Mitral Regurgitation alone, or with Tricuspid Regurgitation	82 "	126
Mitral Regurgitation with Aortic disease	44 "	
Aortic disease alone		8
Tricuspid Regurgitation alone		2
No Murmur		4
	Total	200

The milder degrees of heart-failure may not come under the notice of the physician, and are at any rate not likely to be commonly found in the wards of a hospital. In recording one's experience of hospital cases of this description, one must therefore miss a very large proportion of such cases existing among the class of patients who make use of the hospital; hence the figures to be obtained by analysing the records are not very convincing, such as they are, however, they are worth looking into.

There is one lesion which leaves no doubt in one's mind as to its origin, and it may be at once dismissed, and that is mitral stenosis, with which two murmurs, the presystolic, and the earlier diastolic murmur heard best at the apex, are associated. This lesion which, for obvious reasons, is almost always associated with regurgitation occurred sixty times among the two hundred cases. It is invariably the result of cicatricial or adhesive changes in the valve segments or of the orifice itself – indeed some authorities, from the time of G. W. Balfour, till the present, assert that endocarditis if it affect the efficiency of the heart does so by producing an obstruction at the mitral orifice – without, however, restricting itself to this lesion. The corollary to this assertion is that unless mitral stenosis can be diagnosed the case is not one of endocarditic or valvular origin. That is an extreme view which is not in agreement with the experience of most observers.

It is with mitral regurgitation that the chief difficulty rests. This lesion was diagnosed in 126 of the 200 cases, either alone or in combination with other valve lesions (excluding Mitral Stenosis).

First let us consider a case of heart failure in which there is mitral incompetence without evidence of other valve lesion, the lesion being, so far as we can find out, of recent appearance. Such cases are not very commonly met with in the Royal Victoria Hospital, but are I expect more frequent in the Children's Hospitals. It would be easy to furnish from the 200 cases, examples of this as of most of the other varieties of heart failure to be considered, but I do not think I am justified in occupying your time with a mere recital of appropriate cases.

As stated above, simple endocarditis which has been of short duration cannot sufficiently damage a valve so as to produce a reflux which would be extensive or copious enough to prevent the heart accommodating itself promptly to the unfavourable circumstance. It is only in the event of the heart being unable to overcome the defect by means of its liberal supply of reserve force that symptoms of heart failure appear. In this event either the valve lesion is more serious than could be expected from simple endocarditis, or the reserve force is not available; the former condition is found in ulcerative endocarditis, and the latter is fairly common as a result of inflammatory or toxic invasion of the heart-muscle as a result of the same morbid state which had caused the accompanying endocarditis. In such a case the heart-failure is entirely due to muscular causes, though there may be a valve lesion as well.

The latter, however, cannot be looked on as the cause of the heart failure; unless such a valve lesion progress further by cicatricial change or by successive attacks of endocarditis, it may be practically disregarded. It is the lapse of time, bringing with it cicatricial contraction in the inflamed tissues, that works havoc with the valve curtains. In some cases the inflammation has been so slight that the cicatricial changes are trifling and soon come to an end; the heart muscle recovers its power, the heart-failure disappears, but the murmur remains; the case remains stationary. The question then as to whether the valve changes are progressive or stationary has to be decided, and it can only be decided by time.

An even more favourable issue of this class of case less frequently occurs. The toxic influence was mainly exerted on the myocardium, and the endocardium suffered but little or none at all. Here recovery of the muscle's tone results not only in restoration of an efficient circulation, but also in disappearance of the mitral murmur.

Next take the more common instance of

heart-failure in a case of rheumatic mitral regurgitation known to have existed for some time, in which there is no definite sign of any other valvular lesion, but in which the heart is enlarged. Is the heart-failure in this case due to valvular disease alone, to muscular defect, or to both combined?

If one could feel certain that the mitral valve was only incompetent and not contracted, the failure would be the direct result, in my opinion, of the muscular change alone, I do not assert that the valve is without blame, but I believe that the permanent damage to the valve is insufficient to cause inefficiency of the heart, and for this reason: the case is one of mitral regurgitation of some standing, therefore if there had been any considerable inflammatory change in the valves, time would have produced cicatricial changes, and such changes are practically certain to narrow the orifice in time. Further, in such a case where rheumatic poison caused endocarditis, it is probably that myocarditis also occurred; for the heart muscle is more frequently affected by the poison than is generally supposed, and in all probability those instances of moderate valvular lesion with failure of compensation are also cases myocarditis. Here the muscle fibres yielded not only because of the strain imposed on them by mitral reflux, but also because of the toxic injury to their fibres, whereby the muscle tone was impaired. Granting then that there is no obstruction, the inefficiency of the heart must be due chiefly to muscular defect, which not only weakens the contractile power of the heart, but also by dilatation increases the incompetence of the valves. But it may be said that it is the damaged valve which has injured the muscle of the heart, that is, the compensatory hypertrophy has broken down, and the muscle fibres have undergone degeneration. That is possibly but not certainly the case, but the point is that the valve structure is not sufficiently injured by endocarditis to disturb the efficiency of the heart, and many cases of the kind, in which there is mitral regurgitation with moderate hypertrophy remain perfectly efficient during a long lifetime; hence *degeneration with dilatation is no necessary consequence of a leaking mitral orifice*. In these stationary cases the muscle has probably escaped injury from rheumatic poison. When, under treatment, a case of heart failure of the kind we are considering makes improvement, the dilatation grows less, and the "relative insufficiency" of the mitral valve diminishes. Such a favourable course differs only in degree from the more perfect recovery in those cases of mitral regurgitation which are known to be due to muscular relaxation alone (Balfour's "curable regurgitation").

The relation of the remaining valvular inefficiencies to muscular defect need not occupy much time. Tricuspid regurgitation is almost invariably the result of dilatation, while tricuspid stenosis, which, like the similar mitral lesion, is a purely valvular affection, is very rare.

Pulmonary incompetence is uncommon, and is usually the effect of dilatation, though endocarditis may exert its evil influence on both orifices of the right ventricle perhaps more frequently than is generally believed. The rare pulmonary stenosis is the result of this unusual occurrence of right sided endocarditis.

Aortic disease in young subjects is usually the result of endocarditis, in which case it is almost always combined with mitral disease; but incompetence of the aortic valve not uncommonly results from dilatation. In middle and old age aortic disease is as a rule part of an arterial change in which the aorta takes a prominent part, and the valves suffer in consequence. At the same time the ventricle under these circumstances usually becomes dilated, partly as a result of fatty and fibrous degeneration, and partly the effect of mechanical causes set up by the defective aortic valve, so that a vicious circle is established.

In the series of 200 cases above referred to, there were forty-four cases of Aortic disease associated with mitral regurgitation as well as twenty-three cases with mitral stenosis; there were also eight cases of aortic disease without obvious lesion of other orifices.

Excluding the mitral stenosis cases then, there were fifty-two cases of aortic disease, due to either valvular or muscular disease, and the table shows details as to their ages and as to the presence or absence of a history of rheumatism.

52 AORTIC CASES. (EXCLUDING MITRAL STENOSIS).					
Mitral Regurgitation and Aortic disease – 44 cases					
Aortic disease alone–8 cases					
	Mitral and Aortic		Aortic alone		
	Rheumatic		Rheumatic		
Ages	Yes	No	Yes	No	Total
10 to 19	4	0	0	0	4
20 to 29	5	1	0	0	6
30 to 39	10	4	2	0	16
40 to 49	3	6	1	2	12
50 and over	2	9	0	3	14
	24	20	3	5	52

You will observe that the non-rheumatic cases were nearly all middle-aged or elderly, pointing to degenerative arterial and muscular changes as the probable cause of the heart-failure, while the rheumatic cases were doubtless in larger proportion due to valvular disease, and hence were found in younger individuals. We cannot of course rely too implicity on the history.

Pericarditis is commonly regarded as a cause of heart-failure when it causes universal pericardial adhesion. No doubt the difficulty thus imposed on the heart causes it to be subjected to undue strain, and renders it unequal to its task, but here also some myocarditis may I think be assumed to have occurred, so producing an impaired contractility. A case of this description came under my care some years ago in the Royal Hospital, and was published by Dr. Lorrain Smith. The patient was a growing boy, whose pericardial sac was obliterated, and the adherent layers of pericardium formed a dense, tough, fibrous membrane, totally preventing the growth and development of the heart. The need for an increased blood supply as the boy grew could not be met, and debility and inefficiency of the heart was the result.

Considerable help in distinguishing valvular from muscular cases of heart-failure may be obtained from the history, and the general condition of the patient. Previous illnesses may be first considered.

It has been stated that young persons suffering from heart-failure, who have a history of rheumatism, always owe their ill health to endocarditis, but having regard to what I have already stated on this subject, there is I think good ground for doubting the accuracy of this statement. They may owe their valvular defect to endocarditis, but in a certain proportion of cases, especially of mitral regurgitation, their heart-failure is due to myocardial changes.

Chronic bronchitis, especially when it results in emphysema, is a common cause of heart-failure. Here the defect is muscular, the right ventricle becoming dilated, owing to strain, and imperfect oxygenation of the blood, whereby the nutrition of the heart muscle is impaired.

Syphilis by its damaging action on the arteries, on the aorta and coronary arteries in particular, produces both valvular and muscular defects as described below in speaking of atheroma.

Asthma. The connection between asthma and heart-failure is interesting, but more so from the point of view of classification than of pathology. As a cause of heart-failure asthma has but little importance, for the somewhat rare cases of pure spasmodic asthma in which deterioration of the heart muscle with heart-failure supervene, are dependent upon the development of bronchitis or emphysema. The use of the expression "cardiac asthma" must be restricted to those somewhat rare cases where true asthmatic attacks occur in the course of a heart case; the term is neither applicable to true asthma in which heart-failure has supervened, nor to the ordinary dyspnoea of cardiac cases. It is the latter condition that is often mis-named "cardiac asthma."

In this connection may be mentioned congenital heart defects leading to heart-failure. Here the heart may for a time be able to carry on a moderately efficient circulation, but in time, as the individual develops, the needs of the organism are too great for the capability of the heart, and it fails under the strain.

A very large number of instances of heart-failure occur *in the course* of a variety of affections. The diseases so causing heart-failure may be collected into three groups –

(*a*) Anaemias.

(*b*) Toxaemias.

(*c*) Mechanical strain (the overworked-heart).

Some of the affections causing heart-failure might perhaps be placed in more than one of these groups.

(*a*) A very large number of diseases cause a defective blood supply, and this condition readily brings about a degeneration of the heart muscle as well as of the skeletal muscles. A familiar example of this class is Chlorosis. Fatty degeneration results from the anaemia; a feeble and dilated heart is the consequence. It is true that the dilatation in this complaint is usually moderate but the dyspnoea, palpitation, oedema, syncope are mainly the results of heart weakness, though the defective quality of the blood no doubt contributes to the production of these symptoms.

A more marked instance of anaemic heart-failure is found in progressive pernicious anaemia, where similar, but more marked signs of heart-inefficiency are to be found.

Other rarer forms of anaemia may be mentioned, *e.g.*, splenic anaemia, leukaemia, Hodgkin's disease, malaria, bilharziosis.

The various forms of secondary anaemias may give rise to heart-failure. (1) Haemorrhage from any source either sudden or gradual. (2) Long-continued loss of albumin from the blood as in chronic suppuration, prolonged lactation, Bright's disease, cancer, phthisis. (3) Defective nutrition from any cause, as in starvation, improper feeding, obstruction of the oesophagus, imperfect digestion. (4) A number

of blood-poisons may be mentioned as causing anaemia; thus the anaemia of gout, syphilis, rheumatism, lead poisoning, arterio-sclerosis, Bright's disease, and many fevers produce anaemia, and may thus be the origin of degeneration of the heart muscle, though their toxic effect on the heart is in some instances even greater than that of the anaemia which they originate.

(*b*) Toxaemias. As just stated, a variety of blood poisons have to be reckoned with, and in addition to those just enumerated, the infectious and other fevers furnish frequent instances of toxic heart-failure. In pneumonia, for example, the failure of the right heart occurs not only because the right ventricle is overwhelmed by its work in driving the blood through the inflamed lung, but also because the muscle fibres are impaired in contractility by the poison circulating in the blood; in a lesser degree the stronger, but less burdened left ventricle also suffers in the attack. In scarlet fever, rheumatism, diphtheria, typhoid, measles, etc., there is less danger of heart failure during the height of the fever, but decided risk of subsequent dilatation of the softened muscle if the heart is permitted too forcible action during convalescence. Poisonous substances may be swallowed or inhaled, causing heart-failure, usually by their action on the nervous system, *e.g.*, opium, chloroform, nicotin, etc. Also the toxins of disease sometimes exert their effect on the nervous control of the heart, *e.g.*, diphtheria, influenza.

126 Cases of Mitral Regurgitation, alone or in combination with other valve lesions.					
Mitral Regurgitation with Aortic disease – 44 cases					
Mitral Regurgitation alone – 82 cases					
	Mitral Regurgitation with Aortic		Mitral Regurgitation alone		
	Rheumatic		Rheumatic		
Ages.	Yes	No	Yes	No	Total
10 to 19	4	0	4	5	13
20 to 29	5	1	5	8	19
30 to 39	10	4	6	9	29
40 ,to 49	3	6	5	11	25
50 and over	2	9	13	16	40
	24	20	33	49	126

Of the 126 cases in which a mitral systolic murmur was heard, there was no history of rheumatism or chorea in 69. One might be disposed to conjecture that the remaining 57 cases of the 126 mitral regurgitation were probably of valvular origin, seeing that there was a history of rheumatism in each case. No doubt a large proportion of them were the result of valvular disease, but not necessarily all. Thirty-three of them were mitral regurgitation only, and of these a large proportion were middle aged or elderly – more than half of the number were over 40 years old:–

This is in accordance with the experience of most practitioners who are not surprised to meet with mitral regurgitation in elderly people, but very many of them were obviously cases of arterial disease or degeneration of the heart muscle from a variety of causes of which rheumatism may in some cases have been an unimportant factor, or accidental accompaniment.

Of the 69 cases in which there was no rheumatic history, it would be impossible to give an accurate summary of the causes, especially as in many of them no doubt there had been mild rheumatism in childhood which had passed unnoticed.

There was, however, in nearly all the cases sufficient in their condition or history to cause degeneration of the heart muscle:–

Summary of morbid conditions affecting 69 cases of mitral regurgitation without history of rheumatism.			
Bronchitis	9	Stomach affections	7
Arterio-sclerosis Strain Alcoholism	27	Cancer Phthisis Fevers Influenza Starvation	13
Chlorosis	6	Uncertain	3
Bright's disease	4		

I should like to read you summarised notes of a few of the cases of this group, but fear that I have already trespassed too far on your patience. As you see by the list of conditions of which the patients had symptoms or history, the condition of the heart muscle could not have been healthy, but it cannot be confidently affirmed that it was sufficiently affected to produce the symptoms of heart-failure for which the patient was admitted to Hospital. One can realise how small a proportion of the cases of heart-failure the above 200 cases represent when we consider that the exanthemata and phthisis are not admitted into the Hospital, and that the bulk of diseases causing debility and anaemia were quite outside the group of cases enquired into. Thus the case sheets of cases of cancer, Bright's disease, severe anaemias of the primary group, and many other affections commonly

producing heart-failure were not included in the enquiry, which was limited to those grouped together indiscriminately as heart disease.

(c) Mechanical strain is to some extent responsible for heart-failure. In most of the cases, however, there is at least one other factor in the production of muscular debility. Thus Bright's disease in its final stages may present heart-failure, the result of overwork in opposition to high arterial tension, but the heart defect is also due to anaemia, and to the poisonous condition of the blood. Similarly arterio-sclerosis and atheroma may cause fatty or fibrous degeneration of the cardiac walls by obstruction of the coronary arteries, and at the same time a general rise of arterial tension is common in this complaint, thus causing the heart to be hypertrophied and in the end dilated. Gout and chronic lead poisoning damage the heart by anaemia, by strain, and by the poisons carried by blood.

The *habits* of the patient deserve a passing notice as in some cases they favour the production of heart defects. The press and hurry of modern life tend to the production of hardened arteries, and if this condition is combined with an excess of physical exertion the heart may get too much to do and become dilated. Alcoholism, starvation and exposure may all be causes of heart-failure.

The Family History is not of much importance, except that gout, rheumatism and syphilis may be inherited, and thus lay the foundation of heart inefficiency.

There can be no doubt that we must direct our attention chiefly to the condition of the muscle of the heart, for in a proper understanding of the morbid processes affecting its activity we may hope to increase efficacy of treatment, and the accuracy of diagnosis. Of late a great deal has been done by workers in these lines; graphic records in the hands of Wenkebach, MacKenzie, Lewis, and others have certainly helped materially to explain some of the defects of the heart's action due to defects in the function of the heart's muscle. These defects consist in most cases in disturbances of one or more of the three chief functions of the heart-muscle, viz. – excitability, or in the susceptibility of the muscle to the action of a suitable stimulus; conductivity, or the power to transmit impulses causing contraction through the muscle fibres; and contractility or the power to contract. I do not intend to take you at this late hour further into this interesting subject, though I should have liked if time permitted to refer more particularly to the recent results of investigations by means of the ink polygraph and the electro-cardiograph. You are, however, all aware that the subject is being worked up vigorously by many observers, and that our views on the physiology and pathology of the heart's action have been much influenced by these investigations. The recent researches into this question merely emphasise what has been perhaps insufficiently realised that the chief object of our enquiries in cases of heart-inefficiency must be the condition of the heart muscle.