A Lecture ON COMBINED AORTIC AND MITRAL DISEASE IN RHEUMATIC CHILDREN.

Delivered at the Hospital for Sick Children, Great Ormond Street.

By F. J. POYNTON, M.D., F.R.C.P.LOND.,

Assistant Physician, University College Hospital; and to the Hospital for Sick Children, Great Ormond Street.

LADIES AND GENTLEMEN.—I have already in three of my former lectures treated of some aspects of rheumatic cardiac disease in early life. In the first I dwelt upon the important part that micro-organisms take in producing endocarditis; in the second, upon damage to the heart wall in rheumatism; and, in the third, I endeavoured to show the parallelism in the course of the rheumatic lesions in various organs, and differentiated three types of rheumatic injury—namely:

1. The simple, as in simple endocarditis.
2. The malignant, as in rheumatic malignant endocarditis.
3. The fibroid, as in mitral stenosis.

In this lecture I shall direct your attention to an exceedingly serious group of rheumatic heart lesions, and illustrate them by a series of clinical cases, giving also the results of a study of twenty-one examples which have been under my care at one time or another during their illness. In all these cases there were evidences of mitral and aortic endocarditis, and it is to this combination of lesions in childhood that I would ask your attention.

Rheumatism, as is well known, damages all parts of the heart—the pericardium, myocardium, and endocardium—sometimes one, sometimes the other; and generally, if it is severe, all three. Recognizing this fact, you will not be surprised that these cases of combined mitral and aortic disease in some instances also present such other lesions as an adherent pericardium. It is, however, useful, in studying so complicated a subject as heart disease, to differentiate a group which shows distinct evidences of mitral and aortic incompetence.

The auscultatory evidence of mitral regurgitation is the presence of a systolic murmur, the maximum intensity of which is over the impulse, and the direction of conduction towards the axilla.

The auscultatory evidence of aortic regurgitation is a diastolic murmur, the maximum intensity of which varies in position. In childhood this murmur is most often at its loudest to the *left* of the sternum in the second or third intercostal space on the left side; and as the child grows up this point generally shifts from the left to the right. This murmur is also often very audible behind the sternum, and can be traced down to its lower end.

The combined lesions will then clearly present two very different murmurs:

One the mitral, systolic in time and conducted towards the axilla; the other the aortic, diastolic in time, and conducted from the base, usually on the left side, downwards.

The occurrence of these two bruits, together with changes in the heart and pulse to which I shall allude later, enable us to diagnose with confidence the combined lesion, for no one should mistake the aortic regurgitant murmur, with a maximum intensity to the left of the sternum, for the rare pulmonary diastolic bruit.

How are we to explain this combination of valvular lesions? Is it that in the first place the aortic valve has been damaged by rheumatism—that as a result the left ventricle has become dilated and hypertrophied, and then in turn mitral incompetence has followed as a result of relative incompetence of the auriculo-ventricular valve? In most cases this is not the explanation, and what actually occurs is that both valves are damaged by rheumatism. In most instances the mitral valve suffers first and the aortic later, either in the same attack of rheumatism or in a subsequent one; and it is clear that if the aortic valve is damaged the later of the two, the mitral incompetence cannot be entirely dependent upon that lesion, although no doubt the aortic incompetence tends to aggravate it.

Multiple valvular lesions are common in childhood—the tricuspid valve, for instance, is often slightly damaged; but this combination of lesions we are considering is of especial importance in actual practice.

From a study of experimental heart disease I believe the infection of the aortic valve is often by direct continuity from the mitral, and in these cases the infection is, as you will see, usually a severe one. In fact, this series of mine convincingly illustrates the severity of the rheumatism. Take first of all that important point—the early age of the first attack. In 7 out of 19 this began before 6 years, and two of the children were under 3 years of age.

Again, many of the first attacks were very severe, and no less than four were fatal; fatal, that is to say, after a long illness, in which there was no real cessation of the symptoms after their first appearance. Two examples will be sufficient:

1. In the first attack there were: multiple endocarditis, pericarditis, pleurisy, and multiple arthritis, and it proved fatal at the age of 3 years 2 months.

2. In the first attack, which extended over many months, there were: polyarthritis, pleurisy, endocarditis, pericarditis, nodules, chorea, and nephritis. In this case the tendency to rheumatism was inherited from both sides of the family.

Yet another proof of the severity of the rheumatism is the high percentage of deaths in this series: 10 out of the 21 children died before reaching the age of 12!

In 7 of these fatal cases necropsies were obtained, and some interesting points concerned with them are worth recording. In 6 of them the “*diplococcus rheumaticus*” was isolated by Dr. Paine.

I. In 1 case the actual cause of death was apparently a rheumatic septicaemia, and I am indebted to my colleague, Dr. A. E. Garrod, under whom the child was a patient while in the hospital, for leave to make use of the notes of the final illness.

This patient, a boy aged 6, came first under my care in 1902 for hemichorea, arthritis, and mitral disease, and when 3 years old had suffered from epistaxis, which, as Dr. Sidney Phillips has pointed out, is not unusual in rheumatic children. He was admitted to the hospital, and there developed aortic regurgitation. After a long illness he again became an out-patient, only to break down two months later with a severe attack of pericarditis. Three months after this he again returned to me and remained tolerably free from rheumatism for some while, then once more signs of the active disease commenced and persisted until his death in November, 1903.

At the necropsy an adherent pericardium and mitral and aortic disease were demonstrated, but the point of especial interest was that a film of the blood in the heart, taken by a pipette after death, showed great numbers of the *diplococcus* in pure growth. This drawing, which is an accurate representation of the film under the microscope (see Fig.), shows the condition. Now rheumatism is essentially a disease with local lesions, and it is in these local lesions that the infective agent is usually found, and not, as a rule, in the blood, which is but a channel of conduction. A general rheumatic blood infection is, so far as is at present known, a rare event. Yet in this case it would appear that this child died in such a condition, for the terminal symptoms pointed to a profound blood poisoning.

II. Another child died of the malignant type of endocarditis. In his case the illness commenced with rheumatism, and mitral and aortic disease as results; next pericarditis developed, from which the boy recovered only to be attacked by arthritis, from which he also recovered. This prolonged illness extended over three months, and during that time there was persistent irregular fever. At length infarction and other signs of malignant endocarditis became apparent, and ended in death. The necropsy proved the occurrence of the malignant type of endocarditis, and this specimen illustrates the disease reproduced by inoculation of a pure growth of the *diplococcus* which was isolated from those cardiac valves.

III. Three of the other children died from rheumatic pericarditis, and in all these cases the *diplococcus* was isolated.

IV. One died with chorea, and the *diplococcus* was isolated from the cerebro-spinal fluid.

V. One died from chronic heart disease, and the cultures were negative.

Here I would emphasize a very important practical point. The cause of a fatal termination in these cases of severe valvular disease is an attack of rheumatism; they rarely die solely from the mechanical difficulties of an injured heart; but almost invariably with the symptoms of another attack of rheumatism.

None of these cases, for example, died from a sudden

syncope such as occurs now and again in the adult suffering from aortic regurgitation.

These combined lesions of the aortic and mitral valves can be grouped clinically into three classes:

In Class I there is no sure evidence of aortic disease, and it is only at the necropsy that some beading or thickening of the aortic segments is discovered. In this group the aortic lesion is of merely academic interest, but the finding of these slight lesions in the aortic valve throws a good deal of light upon a practical point, to which I shall allude again shortly, namely, the disappearance of an aortic regurgitant bruit.

In Class II, the physical signs show plainly enough that mitral and aortic disease exist, but the mitral disease preponderates, and the aortic lesion appears hardly to influence either the symptoms or the course of the disease.

In Class III, both lesions are apparent, but the dominating one is the aortic regurgitation. It is these cases in which one is tempted to explain the mitral incompetence as a secondary result, yet *post-mortem* evidence shows that the mitral valve is almost invariably damaged by endocarditis, and not only relatively incompetent.

It is a curious fact that it is difficult to produce primary aortic endocarditis by experiment, although mitral endocarditis and the combined lesions can be easily produced. In

A film of blood withdrawn in a sterilized pipette from the left ventricle of a child dead of persistent and virulent rheumatism. Neither the illness nor the fever suggested pyæmia, and there were no suppurative lesions. This condition in acute rheumatism is a very exceptional one. Drawn under Zeiss obj. 13th oc. 3.

childhood also, I think, primary aortic endocarditis is rare, although this case I believe to be an example:

M. B., aged 13, had an attack of rheumatic fever last Christmas, in which the knees and ankles were swollen and painful, and later she developed chorea. In May she was brought to me at University College Hospital because there had again been some slight chorea. On examination of the heart I found an aortic diastolic murmur. There were capillary pulsation and a collapsing pulse, and the heart was hypertrophied. The mitral valve, if it is damaged at all, is damaged so little that it has given rise to no physical signs of mitral regurgitation. It is worth mention that the mother had no suspicion that there was any heart disease.

Even more rare than simple aortic regurgitation is, I think, aortic stenosis. The following case, however, I believe to be an example of this condition:

F. C., aged 5½, whose father and mother have both suffered from "rheumatism," had an attack of rheumatic fever following measles; in this there was multiple arthritis, and for it he was laid up eleven weeks. His mother was not, however, aware that the heart was damaged.

There is a loud, grating, aortic murmur, systolic in time, and reaching its maximum over the aortic cartilage. This murmur can be traced upward into the neck, and along the subclavian vessels, and over the base of the heart there is a

systolic thrill. The area of the heart is increased, and the impulse is definite and forcible. There is also a systolic murmur audible over the impulse, which I believe to be conducted from the aortic region.

The pulse is in character deliberate. I do not think this is a congenital condition, for there is nothing to support that hypothesis; but I look upon the case as one of primary aortic stenosis in a child, the result of that prolonged attack of rheumatism.

The next case is a classical example of the combined lesions, and in his case the aortic symptoms are the prominent ones. He first came under my observation four years ago with a history of three attacks of rheumatic fever, in which attacks polyarthritis and profuse sweating occurred.

At that date he had very clear signs pointing to the double lesion—namely, an aortic diastolic murmur at its maximum intensity behind the sternum at the level of the second intercostal space, and a mitral systolic murmur which was conducted into the axilla. There were at that time pallor, a collapsing pulse, great nervousness and breathlessness, and the heart was much enlarged.

For over three years he remained free of any attack of rheumatism, and then last Christmas he had a fourth attack. He is now 13. A very interesting feature in his case has been his intense nervousness. If he goes to school he gets dreadfully flurried and upset. There have not been any clear evidences of the mitral disease in his symptoms, although the lesion is a very obvious one. There has not been dropsy, or cyanosis, or a puffy face, or bronchitis; it is the aortic regurgitation which is troubling him, and that remarkable nervousness and flurry is in accord with that particular valvular lesion.

In the heart disease of childhood the detection of the auscultatory signs of valvular defects in their early stages is clearly an important matter, and in my former lectures I gave the usual physical signs in some detail. It will be remembered that the first result of an attack of rheumatism is to cause an acute dilatation of the heart. The pulse then rises a little in frequency and the tension falls. The impulse becomes diffuse and extends beyond the vertical nipple line. The deep cardiac dullness is increased to the left, and sometimes to the right and also upward. On auscultation the first sound at the impulse is detected as short, and the second sound at the pulmonary base is accentuated. A systolic murmur also may be audible internal to the nipple line.

At first it may be exceedingly difficult to decide whether in addition to the dilatation there is organic mitral disease, but the occurrence of a systolic mitral murmur which is conducted towards the axilla, together with the supervention of hypertrophy and dilatation of the left ventricle, are convincing signs of mitral endocarditis, and this lesion is the more likely when there is persistence of the so-called reduplicated second sound below the left nipple.

Any case of rheumatism in which there is definite dilatation of the heart, and in which the first sound is short or not clear, may also be a case of commencing mitral endocarditis, for it is manifestly impossible to decide without looking inside the heart when the earliest changes of endocarditis commence.

The early signs of aortic regurgitation need even closer attention. The second sound at the base becomes suspiciously faint and blurred, then there appears a systolic murmur, soon followed by the appearance of a diastolic one, or it may be that the diastolic murmur only becomes audible. The second and third intercostal spaces on the left side as well as the classical second right intercostal space must be carefully searched for this bruit.

In some of these cases of combined valvular lesions a pre-systolic murmur also makes its appearance. Most of the children who show this have suffered from the mitral and aortic disease for some considerable period of time before this murmur becomes audible. It may be a result of the aortic regurgitation, an "Austin Flint" murmur, or it may be in some cases due to a slight narrowing of the mitral orifice. The practical importance of this murmur, however, is not great, and it need not detain us longer.

If the morbid changes in the valves are considered, and especially those in the aortic valve, it becomes apparent that they are frequently very slight. If death occurs and the disease is acute, only tiny vegetations may be found. If the disease is chronic, only a slight thickening and puckering of the segments. The changes in the mitral valve, however, are generally more severe. The actual causation of the bruits in heart disease is still rather mysterious, and I have been astonished, when studying experimental heart disease, to find

large vegetations in one case causing a loud murmur, and then in another no murmur at all. If this is the case with large vegetations, it is very reasonable to suppose that during the healing of slight aortic disease—that is, during the transition of the morbid changes from tiny vegetations to fibrous thickening of the valve—a murmur which was originally present may later disappear. There is, I admit, considerable doubt expressed as to the possibility of such a disappearance of an aortic regurgitant bruit, but in two cases under my observation, and here to-day, this gradual disappearance of the murmur has taken place.

CASE I.—This little girl is the child of foreign parents, and the history of her illness very imperfect. When 4 years of age she developed chorea, and this persisted without any absolute cessation for two years. In 1904 this child had well-marked double mitral disease and aortic regurgitation with an aortic diastolic murmur, which was audible at its maximum intensity in the second left intercostal space. These valvular defects I observed repeatedly for two months, and then, after a long rest, she commenced to improve in her health, and in January, 1905, the aortic murmur disappeared. Now the case appears as a case of well-marked double mitral disease.

CASE II.—This is a more recent case, and is remarkable for the wonderful improvement in the child's condition during the last two months. Last November this boy, who is 7 years of age, had a terrible attack of rheumatic fever; chorea, pleurisy, pericarditis, polyarthritides, and endocarditis all occurred in this illness. In April I detected a mitral systolic murmur and an aortic diastolic. At the beginning of May, although the aortic second sound was faint, I could only just detect the aortic diastolic murmur. To-day I cannot find it at all.

This disappearance of an aortic regurgitant murmur should be sharply distinguished from the same occurrence in a patient in whom acute failure of compensation has occurred. In those cases also an aortic diastolic murmur will disappear, but when the heart recovers itself and compensation is restored the aortic murmur again appears. It is never wise to commit yourself to an absolute diagnosis of the condition of a heart when you see it in a condition of acute failure, for both aortic regurgitant and mitral presystolic bruits have a way of disappearing until the heart begins to recover.

A mitral systolic murmur disappears much more often than a diastolic, and there are various reasons for this. It may be that the murmur is the result of a simple dilatation of the left ventricle, and when this dilatation is recovered from the murmur goes. Then, again, in some cases of mitral stenosis a systolic murmur due to regurgitation may disappear, and a presystolic bruit slowly appear. This murmur may also disappear, owing to the slight nature of the original lesion—a natural cure. Thus, in one of my fatal cases both the mitral and aortic bruits disappeared for a while, but the aortic one again became very definite, with a renewed attack of rheumatism.

The necropsy showed considerable aortic disease, but only slight damage to the mitral, and this had been well repaired.

The clinical results of these combined lesions differ considerably. If the mitral regurgitation predominates, the pulse does not show the features of the aortic lesion; it is not sudden or collapsing, and on this account the aortic lesion may be entirely overlooked, for the face need not show any marked pallor. On the other hand, when the aortic lesion is the predominant one, there are the collapsing pulse, the facial pallor, and capillary pulsation which are always associated with descriptions of aortic regurgitation.

The size of the heart is greatly altered, for both aortic and mitral regurgitation cause hypertrophy and dilatation of the left ventricle, and, further, the mitral lesion tells back upon the right side of the heart. In addition, it is not unusual for an adherent pericardium and possibly mediastinal adhesions to complicate the combined valvular lesions. There may be then enormous enlargement of the heart, as in the case of this little boy.

J. N., aged 11 years, suffered from chorea at 8 years. Last August, after a sore throat, he developed very severe rheumatic fever, and was a patient in the London Hospital for five months. His heart is enormous, and I have no doubt that the pericardium is generally adherent in addition to the severe combined valvular lesions. A casual glance at his face would never lead you to suspect his condition, for he has a good colour, and is neither pale nor blue; yet his heart occupies almost all the left axilla, and the left lung must be in great part collapsed.

Several of the other cases also show great enlargement of the heart.

The remarkably natural appearance of this little boy's face leads me to make a few remarks upon the relation of the physical signs to the symptoms in heart disease. That relationship is a very deceptive one. An adult, for instance, may have an attack of angina pectoris, and the heart on examination shows so little that is decisive that the physician is tempted to underrate the gravity of the symptoms. The cardiac sounds may be short or clearer than natural, but there need be no bruit or even enlargement of the heart, and yet the patient may die in another attack of angina within a few months. In childhood also this relationship is deceptive, but the converse is more usual. The symptoms may be very indefinite, and yet the gravest valvular lesions may be present. This peculiarity is well worth special notice, for it is not at all an uncommon occurrence for organic heart disease to be overlooked in early life because of this absence of grave symptoms.

I need not detain you longer with the diagnosis of these combined lesions, for that has occupied most of this lecture. Just one word of warning is needed against mistaking the aortic lesion for pericardial friction, or vice versa.

The prognosis in these cases is very grave. I mean by this the prognosis as to the future of the child and not only the immediate outlook. No doubt with care and every attention much may be done to keep them in fair health, and some, no doubt, grow up, but they are invalids.

When these 21 cases of mine are reviewed (it must be remembered they are children of the poor), it is found that already 10 are dead. I have lost sight of 4, but of these I feel sure 1 is dead. This leaves 7. One is now 14, and can go to school, and keeps in fair health, though he is very pale and easily gets short of breath. Another has reached the age of 13, but has lately had a fourth attack of rheumatism, and the aortic lesion in his case has made him so nervous that he has not been able to attend school.

One, a girl aged 6, has lost the aortic murmur, but the mitral lesion is so severe that her development is much interfered with. One, a boy of 10, probably cannot live another six months. Two are only recovering now from the severe attack of rheumatism that damaged their hearts, and we can have no feeling of confidence that another attack may not follow in the course of a year or two.

It is a very melancholy list, and brings home to us clearly that the great hope in treatment lies in the prevention of rheumatism. This prevention of acute rheumatism is a big problem, full of difficulties, but with great possibilities. It is a disease apparently much more common among the poor than the rich, which in itself suggests that prevention is feasible to some degree. On the other hand, it is difficult to determine whether it is possible to prevent rheumatic fever attacking a rheumatic child, who has already suffered from it severely.

It seems to me that careful mothers—and some of these children have had most careful mothers—do manage to keep their children free for some long periods of time, when these children have made a good recovery from an acute attack, but many cases only partially recover, and the disease seems to lurk about them. These seem to be liable to develop more rheumatism in spite of every care, and I would compare their position to that of a child with partially-healed tuberculous disease. A damp house or a damp neighbourhood are, in my experience, detrimental. I cannot say that the rheumatic infection is actually a result of this damp, for there is no proof forthcoming, but I have observed that several of my most terrible cases have lived in damp surroundings, and I always try to rescue rheumatic children from them if the parents are in a position to change their home.

I cannot bring myself to believe that any remedies have a direct action in preventing severe cardiac rheumatism, or in arresting its progress; salicylates, blisters, iodides, quinine, etc., seem to me powerless to cope with it, and I have seen them all tried in different ways, and by different physicians. A great deal can be done by rest and drugs which tend to improve the child's strength, but such recovery as occurs seems to me to depend rather upon the antidotal juices of the child than upon any particular drugs.

You will not mistake my meaning. I readily admit that a mild attack of cardiac rheumatism with acute dilatation will get well with skilful treatment, but the cure appears to be quite a natural process, for if the same methods are used for a severe case, there seems to be no specific result which is to be accredited to the drugs. I am of opinion that the many

drugs in use for the treatment of virulent rheumatism in childhood are no more specific than the drugs that are used in the treatment of acute tuberculosis, and no more effectual. There is much to be done for a child with valvular disease, much that will help him. Yet it is at the best but a treatment of the third order of merit, for if the first is prevention, and the second cure, this is but palliative.

STRAIN AS A FACTOR IN CARDIO-AORTIC LESIONS.

By H. B. ANDERSON, M.D., L.R.C.P. LOND., M.R.C.S. ENG.,
Associate Professor of Clinical Medicine, University of Toronto.

THE clearest conception of the influence of mechanical stress *per se* in the causation of cardio-aortic lesions is obviously to be obtained by the investigation of cases wherein all other factors can be eliminated. For this reason the effects of strain have been studied, especially in soldiers, athletes, and others, who, by reason of occupation or practice, are subjected to severe sudden exertion or to prolonged over-exertion at a time when the general health is good and the heart normal. Thus Larrabee defines heart-strain as "the untoward effects of over-exertion in the previously normal heart," and Allbutt says, "when we enter upon a discussion of strain of the heart, we are understood to refer to the effects of muscular exertion upon hearts which, before the strain, were either strictly or virtually sound." In other words, the influence of strain has been considered chiefly with reference to persons whose physical condition renders them least liable to show its ill-effects. From a systematic standpoint, the advantages of approaching the subject in this way are readily apparent. In fact, it seems the only way of appreciating its importance as a single, determining, etiological factor. It would also seem a very obvious conclusion that if over-exertion is capable of doing injury to normal structures, much more readily will its untoward effects be produced when these structures are unsound; yet, paradoxical as the statement may appear, the consideration of strain from the view point of the healthy and vigorous has undoubtedly led to an under-estimation of its power to do harm in cases where the myocardium or aorta is already weakened by age or disease. The morbid opportunity offered under these circumstances not only allows of the more ready production of injury by over-exertion, but, what is even more important to the clinician, degrees of strain borne with impunity by the healthy structures may here become factors of the most serious significance. It is the influence of these lesser degrees of strain acting on structures already weakened that is often not fully appreciated. For this reason, it would be better from the clinical standpoint to consider strain in a relative rather than in an absolute sense, to indicate an increase in intracardial or intra-aortic pressure, due to unusual or controllable conditions, out of proportion to the capacity of the tissues concerned, to withstand it. The qualifying phrase "unusual or controllable" is used in order to limit the term "strain" so as to exclude the pressure which is normally incident to the function of the heart and aorta under the most favourable circumstances. I wish to emphasize, however, that to grasp the practical significance of strain it is even more important to appreciate the condition of the tissues exposed than the absolute amount of violence to which they are subjected.

Perhaps another reason for the failure to fully apprehend the ill-effects of strain on the cardio-aortic tissue is due to the general tendency in the past to rely too much on the stethoscope for the detection of cardiac disease. Useful as the stethoscope may be in the recognition of valvular lesions, it is, unfortunately, of very limited service in detecting the much more important myocardial changes. Myocardial changes are rather to be suspected from a knowledge of their association with certain general diseases or of their being the result of certain causes. Therefore a broad view of the pathology of any particular case is a more reliable guide to the recognition of myocardial changes than stethoscopic or other form of physical examination, especially at an early stage, before hypertrophy or dilatation have occurred. This applies particularly to the appreciation of the myocardial trouble associated with the acute infective diseases.

The capacity of the cardio-vascular system, when sound, to withstand severe mechanical stress is very great. Thus Löwy has shown that the heart when exerted to its uttermost is capable of doing thirteen times as much work as when the

body is at rest; and Sherrington states that rupturing strains of the healthy arteries require nearly twenty times as much strain as the body can possibly exert upon them. I can find no experimental data giving any definite statement of the increase of pressure in the chambers of the heart or aorta during severe exertion. An investigation to ascertain this point, desirable as it is, would be beset with great difficulties. Clinical observation has abundantly shown, however, that severe or even irreparable damage may result; and bearing in mind the great reserve strength of these structures in health, the increase of pressure necessary to produce such damage must indeed be enormous. The effect of such degrees of stress on weakened structures may *a priori* be readily imagined.

But apart from this we need only recall how frequently we observe the insufficiency of the cardiac muscle to withstand the requirements of ordinary quiet life, that in consequence dilatation is a common, and rupture not a very rare event, to understand how powerful a cause of mischief severe exertion may be.

Having in mind these well-established facts, it seems remarkable that in special treatises on diseases of the heart, and in systems or textbooks of medicine, the influence of strain is frequently disposed of in a few words, merely mentioned incidentally or in some cases ignored. The relatively unimportant place thus often assigned to this factor in dealing systematically with etiology of cardio-vascular diseases, does not indicate an adequate appreciation of the potency of strain to cause cardiac mischief in the previously healthy, as shown by many contributions by competent observers, to be mentioned later; nor is it in keeping with the frequency with which its influence is seen in the routine of practice. The unrestrained athletics often permitted in schools and colleges, contests or undertakings requiring strength and endurance, entered into by the young, weakly, or untrained, or what is even worse, misguided exercise, laborious holidays, or over-taxation incident to occupation (occasional or continuous) in the sedentary and out-of-trained at a later period of life, or after convalescence from acute infections, all go to show that the possibilities of harm from muscular stress are not generally appreciated. I wish, therefore, within the compass of this paper to deal—of necessity briefly—with the physiological and pathological phenomena of severe exertion with reference to the heart and aorta, to review in a fragmentary way the literature of the subject, to relate a number of illustrative cases which have come within my personal experience or that of some of my colleagues, and to attempt to outline some special circumstances wherein it appears to me the importance of mechanical stress is not kept definitely enough in view in the guidance of the healthy, nor in the management of the sick and convalescent.

The work of the heart is represented by the amount of blood expelled from its chambers during contraction and the resistance to be overcome in forcing it through the peripheral circulation. The energy exerted by the heart during contraction is in part changed into kinetic energy, represented by the momentum imparted to the blood stream, and in part into potential energy stored up as tension by the dilatation of the elastic walls of the arteries. The amount of the latter will depend on the elasticity of the vessel walls. At the beginning of exertion there is a rise of arterial pressure due to compression of the intramuscular vessels by the contracting muscles and to an increased output by the left ventricle. The left ventricle, therefore, has to expel a larger amount of blood against increased pressure. Under the stress of severe exertion the ventricles are unable to empty themselves as completely as under ordinary conditions, so there is a resulting increase of residual blood within them which tends to over-distension, and from this cause also there is increased stress thrown on the muscular walls. The initial effect of exertion is, therefore, to throw increased work on the left ventricle for three reasons, namely, (1) from increased amount of blood to be expelled, (2) from distension of the cavity, and (3) from increased peripheral resistance. It will, therefore, be readily understood that the brunt of sudden exertion and the primary effect of prolonged exertion is borne by the left side of the heart and arterial system. The initial rise of pressure is of short duration. Owing to reflex influences from the muscles, and possibly to the action of fatigue products on the vaso-dilators, dilatation of the intramuscular vessels soon takes place; the cutaneous vessels also dilate, as shown by reddening of the surface and sweating; and the more active respiratory movements increase