Remarks on Failure of the Heart from Overstrain,

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It has long been known that there is a connection between the work done by the heart and disease of that organ. For example, we may recall the fact that in intra-uterine life, when the right side of the heart is mainly concerned in the circulation of the blood, the valves of the right heart are more liable to disease than those on the left side, while after birth the valves of the left side, which have more work to perform than the right heart, are also more frequently affected.

It is known, also, that certain forms of heart disease, so-called "overstrain" of the heart, are due to the organ being called upon to perform more work than it is capable of compassing without harm to one or other part of its complicated mechanism. The chief recognised cause of this form of overstrain is active or prolonged muscular exertion, which, as we know, involves a higher arterial pressure than the mean normal, and an increased supply of blood to the contracting muscles. It is, moreover, recognised that there are many other conditions besides those mentioned which throw increased work upon the heart, and which may lead to disease of the organ, such as certain forms of acute or chronic renal disease, syphilis, and, in general, all those conditions in which the arterial blood-pressure is higher than normal.

The diseases of the heart which result in such cases are, in some instances, chiefly evident in the valves; while in others the muscular wall of the organ is the part in which the effects of the overwork are most marked. We may mention, also, that in disease of the valves, however produced, the effect of the incompetence or stenosis is to throw more work upon the heart, so that one main result of valvar disease is to increase the work of the organ, or part of the organ. The pathology, therefore, of overwork necessarily underlies the pathology of failure of the heart from disease of its valves.

When, also, from any cause the muscular power of the heart is diminished, whether from imperfect blood-supply, fatty degeneration, or impaired quality of the blood, or from any of the many known causes by which the power of contraction of the heart is impaired, we have a condition in which the relation between the work required of the organ and its power of doing that work is affected in an analogous way to that which occurs when the work of the heart is increased, and its power remains constant.

These considerations induced us to make a series of experiments on the effect upon the heart of variations in the work which it is called upon to perform in conditions of health and disease.

In speaking of the work done by the heart, we refer to the mechanical units of work done by the organ in a certain given time, and which can be found by multiplying the quantity of blood thrown out by the heart by the pressure against which that blood is thrown out: that is, the two factors to be considered are—the quantity of blood expelled by the ventricles in a given time, and the arterial pressure. Thus, in investigating the relation of the work of the heart to the effects of that work upon the organ, it is with these two factors that we have to deal. They can most conveniently be considered separately, and such separate consideration or investigation is, in many cases, desirable, seeing that as causes of disease they do not necessarily go hand-in-hand; indeed, in certain cases one of them may be exclusively active.

Changes in the Blood-pressure.

Let us take, first of all, the effect upon the heart of changes in the arterial pressure, what we have to say applying equally to the left and to the right heart.

It is, of course, easy to vary the arterial pressure experimentally in animals by many methods. For example, by stimulation of vaso-constrictor nerves the arterial pressure can be raised, while by a section of certain of these, or by stimulation of certain vaso-dilator nerves, the arterial pressure can be lowered, in each case within certain limits. Such methods have various objections, which need not be mentioned here, and we preferred to influence the pressure against which the heart has to force out its contents, by narrowing the aorta at one or other part of that vessel, with or without closure or narrowing of certain of its branches. In order to measure the increased resistance against which the heart has to empty its contents in such an experiment, we found it most convenient and accurate to record graphically the intra-ventricular pressure, by means of some trustworthy pressure-gauge. For such a purpose, we have to hand in our laboratory the instrument already described by Mr. Rolleston, in his paper on the Intra-ventricular Pressure Curve, and which gives, we believe, satisfactory tracings of the changes in pressure of any chamber of the heart with which it is placed in communication. It consists, as

Fig. 1.—Intra-cardiac Pressure-gauge. The glass canula shown at the lower part of the instrument is introduced into the ventricular cavity, the side branch of the T tube being closed. The instrument is filled with oil or other fluid, which transmits the pressure within the heart to the piston, b, this latter being in communication with the recording lever (represented broken in the figure).

will be evident from the diagram, of a light piston, which can rise and fall in the carefully bored tube, b, in which it moves without appreciable friction, while the space between the piston and the tube in which it oscillates is so narrow that little or no fluid can escape. This piston is attached to a recording lever, whose movements are restrained by the resistance to torsion of a strip of steel, a, in such a way that the excursions of the piston and the recording point of the lever, which writes on the blackened surface of a revolving cylinder, correspond to the hydrostatic pressures acting on the under surface of the piston. This is not the place to go into the technical details of this method, which are given in Mr. Rolleston's paper, and we need only add that such pressure-gauge may be connected with any cavity of the heart without any difficulty, the heart being exposed by making a window in the thorax, and the respiration of the animal being carried on artificially. In all our experiments on this subject, the animal—usually the dog—has been under the influence, during the experiment, of an anaesthetic, and has, therefore, suffered no pain.

We can only give here a few of the more striking facts obtained in experiments of the kind above indicated. For example, we may say that as the outflow of the blood from the heart is interfered with, the maximum pressure within the ventricle increases,
Under these circumstances the intraventricular pressure in the left ventricle, which in the dog is normally something under 130 millimetres mercurial pressure, can, by gradual narrowing of the ascending aorta, be raised to 250 millimetres, 300 millimetres, or even 400 millimetres, in certain cases. Beyond a certain point further narrowing, and even complete closure (which of course is only possible for a few seconds without causing death) does not raise the pressure within the ventricle beyond the limit above referred to, which limit varies with the animal and with the condition of the heart at the time. With two successive momentary closures of the aorta the maximum pressure obtained in the left ventricle is the same. With fatigue of the heart, produced either by continued slight narrowing, or by closing the ventricles almost completely, or by complete closure of the aorta, this maximum limit gradually falls. It may be added that the curves show a rise in the pressure during diastole, which is relatively less than the rise of systolic pressure. This is well shown by the curve. Such narrowing of the aorta produces a very evident diminution of both ventricles, which, nevertheless, go on contracting and expanding in what to the eye seems a perfectly normal manner. Examination of the large veins, however, shows that in them, with the great or extreme narrowing just referred to, there is a very visible wave proceeding from the heart with each ventricular systole; in other words, this narrowing of the aorta produces regurgitations through both mitral and tricuspid valves. The effect, then, of greatly increasing the resistance which the ventricles have to overcome in the evacuation of their contents, is to raise the intraventricular pressure during systole to a height varying with the individual heart under observation, and to cause great expansion of the chambers of the heart with regurgitation eventually through the auriculo-ventricular valves.

It may further be noted that the supply of blood to the heart muscle is influenced by the height of this limit. This is shown by the fact that narrowing the aorta raises the maximum pressure obtainable in the right ventricle by narrowing the pulmonary artery. The experiment on which this statement is founded runs as follows:—the pulmonary artery is first narrowed (the pressure-gauge being connected with the right ventricle), and the maximum pressure thereby produced is recorded. When this is repeated shortly after the aorta has been greatly narrowed, a very much higher systolic pressure is found to result. We see no reason to doubt that here, with the increased pressure resulting from constriction of the aorta, the coronary arteries receive an augmented blood-supply, which improves the nutrition of the right ventricle.

After overstrain of the heart produced in this way, the valves are found to present certain anatomical changes, to which we will presently have to refer. In the meantime it is more convenient to proceed to the consideration of the second factor which influences the work of the heart, namely, the quantity of blood thrown out by it in a given time.

Changes in the Amount of Blood Expelled.

Ludwig's Strohmehr is capable, it need hardly be said, of giving information on this subject, but it is not specially suited for our purpose, seeing that it gives no indication of the effect on the heart of its own variations in the quantity of blood thrown out by that organ, nor can it be conveniently employed without very serious interference with the circulation in the systemic arteries. The method which we employed is one which permits measurement of the amount of blood thrown out of the heart in a given time, and the effect on the heart itself of variations in that volume. It consists in enclosing the living and working heart in what is in practical purposes a rigid air-tight box, the variations in whose contents can be recorded graphically. In other words, it is a cardiacplethysmograph, or onograph. We will in the following pages refer to it by the name cardiomter.2

The construction of this instrument will readily be understood on looking at the diagram. Here, also, we need not trouble you with technical details, and will only say that round the root of the heart is placed a metal ring, which is made in two pieces, for convenience of adjustment, and which is grooved outside, so as to retain in position a ring of india-rubber, something like an ordinary umbrella ring. This double ring has been placed round the root of the heart, the outer or parietal layer of the pericardium is then drawn tight over it, while against it, and firmly pressing the pericardium, are screwed the two halves of the

3 Both cardiomter and pressure-gauge were made for us by the Cambridge Scientific Instrument Company
lower hemisphere of a spherical box. The upper part of the box is then screwed to the lower half. Attached to the upper hemisphere is a wide cylindrical tube, in which moves a piston, c, connected with a recording lever, a loose, inelastic membrane preventing any passage of air or fluid by the side of the piston, while it allows a free oscillation of the latter. Finally, the interior of the instrument having been filled with warm olive oil, and the piston pulled upwards by means of an india-rubber spring, d, so as to render the pressure outside the heart subatmospheric, as under normal conditions is the case, tracings of the changes in volume of the heart are recorded by the point of the lever on the blackened surface of a revolving cylinder. When the heart contracts, the point of the lever rises, and when the heart expands the point of the lever falls, while the distance between the position of the lever point before, and that at the end of systole expresses the amount of blood thrown out by the heart during its contraction. The volume of blood entering

Fig. 3.—Cardiometer. The separate figure to the right shows the parts (seen from above) composing the lower part of the box, and which are represented detached from one another. The oval ring, a, composed of an inner metal ring surrounded by a circle of india-rubber, is first placed round the root of the heart, as close as possible to the reflection of the pericardium, so that the parietal layer of that membrane is outside the visceral inside the ring. The two halves of the lower hemisphere are then screwed together, the oval opening at the bottom clamping firmly the india-rubber ring and pressing the pericardium against it. The hollow sphere shown at the lower part of the larger figure replaces the parietal pericardium, forming a cavity sufficiently large to allow the heart (shown by the dotted lines at b) to expand and contract freely. The horizontal recording lever rotates round an axis at the top of the support c. The rest of the figure is sufficiently described in the text.

or leaving the heart, which corresponds with any given rise or fall of the lever point, is easily obtained by gauging the instrument before or after the experiment. Our observations lead us to believe that this method of investigating the amount of blood thrown out by the heart, and the size of that organ in systole and diastole, is not only easy of application, but is also very accurate—to us, when we first used it, surprisingly so.

We can in the time at our disposal give only a few of the facts obtained by this method. We may say, first of all, that the quantity of blood thrown out by the heart in a given time is liable to very great variations, independently—that is, without any corresponding change of the arterial blood-pressure.

Let us take, however, first of all, the effect on the heart of changes in the pressure within the arteries. Rise of the arterial pressure produced, for example, by narrowing the descending aorta, a mercurial manometer recording the carotid pressure, does not change the amount of blood thrown out by the heart during the period of raised blood-pressure, as compared with that before or after the period of narrowing. The heart itself, however, is appreciably affected by such change in the blood-pressure. Its size at the end of systole is greater than with normal arterial pressure, that is, there is not so complete an expansion of blood; while, at the same time, its expansion during diastole is also greater. The rationale of this fact appears to be simply that, with a higher resistance to their contraction, the ventricles contract less in systole, while the rise of blood-pressure in the arteries not diminishing the quantity of blood which reaches the heart in a given time, the ventricles must necessarily expand more in diastole, seeing that they contain this increased residual blood in addition to the normal quantity entering from the veins. The result is that, when the arterial pressure rises, the volume of blood in the heart, both at the end of systole and at the end of diastole, is increased. Analogous results are produced when the arterial pressure is raised by other methods. In other words, the effect of

Fig. 4.—Cardiometer tracing, showing the effects of an injection of 50 cubic centimetres of normal salt solution into the external jugular vein of dog: a, before injection; b, after the injection of 30 cubic centimetres; c, after the injection of 50 cubic centimetres (in all) of the solution. The time-curve gives seconds. The increased magnitude of the individual beats, that is, the increased amount of blood thrown out at each contraction, is well seen. The rate of beat is not appreciably altered. The increase in the amount of blood passing through the heart in a given time, resulting from the injection, is about 34 per cent. The figure, moreover, shows that with a greater amount of blood passing into and out of the heart per beat the dilatation of the organ is not purely confined to the diastolic period (rise of curve), but affects the heart in systole as well. For further details of mode of measurement see description appended to Fig. 5.

variations in the arterial pressure on the heart is that increased resistance produces diminution of the systolic contraction of the ventricles; and, the quantity of blood entering the heart in any given time remaining the same, this diminished contraction is, cœperis paribus, necessarily accompanied by increased expansion in diastole.

We have seen that, in the experiments first referred to, the expansion of the heart so produced leads, if extreme, to functional insufficiency of the auriculo-ventricular valves—an effect which is all the more readily produced the more fatigued the heart is.

Let us now consider some of the influences which affect the quantity of blood thrown out by the heart in a given time, and the effects on the organ itself of such variations. It need hardly be said that the larger the quantity of blood which reaches the heart by the veins, the larger the quantity will be which it throws out. In our experiments we find many influences which affect the work of the heart in this way, and which have, curiously
enough, been overlooked by writers on the subject. We may say, first of all, that the quantity of blood within the vascular system influences greatly the volume of blood which the heart has to expel in a given time. This can easily be seen on injecting blood, saline solution, or other appropriate fluid, into one of the veins. For example, we find in one experiment (illustrated by Fig. 4) that injection of 30 cubic centimetres of saline solution produced an increase in the mechanical work done by the heart equal to about 34 per cent.

The caliber of the veins also necessarily affects the quantity of blood which reaches the heart in a given time. This fact can be very well shown by applying gentle pressure to the abdomen, as is well shown by the tracing in Fig. 5. Narrowing of the veins has, therefore, the same effect as increasing the volume of blood in the vascular system.

There are many other ways by which this factor in the work done by the heart can be varied, whether or not change in the arterial pressure, but we do not propose to occupy your time with this subject, seeing that what concerns us here is the effect of change in the work of the heart, however, produced on the organ itself. It need hardly be said that where the rapidity and force of beat remain constant, any increase in the amount of blood which reaches the heart must necessarily increase the diastolic expansion of the organ. Within physiological limits, there appears to be no perceptible harm in such variations in the diastolic expansion of the heart, which, we may note in passing, are very much greater than is generally supposed. We have no reason to believe that, with an increased degree of expansion in diastole, more energy is expended in throwing out a given volume of blood against a given arterial pressure. Supposing the arterial pressure and the force of the muscular contractions to remain constant, any increase in the diastolic expansion of the ventricle necessarily implies a diminished degree of contraction in systole, although the volume of blood expelled by the heart with each contraction may be, and in physiological conditions always is, increased. This is well seen in Fig. 5. When the work of the heart is raised owing to increase in the volume of the blood reaching it by the veins in a given time, the extent and the efficiency of contraction is increased, although the degree of contraction of the individual muscle fibres at the end of systole is diminished.

It is generally asserted that, at the end of each contraction, the ventricle is always completely empty of blood, a view which is evidently opposed to what we have just said as to the varying amount of residual blood in the ventricles at the end of systole. The following experiment, even were there no other evidence, at hand, conclusively shows, we think, that the generally received views upon this subject are erroneous. The experiment consists in introducing the little finger through the apex into the interior of the left ventricle of the living animal, the heart having been exposed. This, at first sight, very difficult operation, is perfectly easy, and need not cause any escape of blood. The finger is introduced in the same way as the cannula of the instrument first described by us, by being pushed through an incision which has penetrated two-thirds of the distance between the pericardium and endocardium. The heart continues to beat, as far as can be seen, quite normally under these circumstances, and by the finger so introduced it can be felt that, at each contraction, the lower part of the ventricular cavity closes completely, the muscular papillares coming into contact with one another; the upper part of the cavity, however, lying between the valves and the papillary muscles does not become emptied.

We must assume that, ceteris paribus, beyond a certain limit, increase in the work of the heart due to increase in the volume of blood thrown out in a given time tends to fatigue or weaken the organ. We find, then, that the work of the heart varies very greatly within physiological limits as a result both of the changes in the arterial pressure, and in the amount of blood which reaches the organ, and that variations in the latter are of even more frequent occurrence than in the former. There is also the fact that increase in the work done, other things being equal, produces diminished completeness of contraction in systole, and therefore an increase in the residual blood in the ventricle. This physiological dilatation of the heart with increased work becomes, when excessive, the cause of failure of the organ from overwork or overstrain, as it is generally called; in other words, the heart goes on contracting and sending out all the blood which reaches it (excepting, of course, the residual blood) until the moment when, either from increase

![Fig. 5.—Cardiometer tracing, showing effect of abdominal compression on the amount of blood thrown out by the heart. The time-curve gives intervals of one second. During the period between the two vertical lines at A, the heart gave 32 contractions, the average height of the movements of the lever-point resulting from these being 27 millimetres. Multiplying 27 by 32 we obtain 864 millimetres of upward movement during that time. Between the vertical lines B, during which the abdomen was being compressed, the heart gave the same number of contractions as during the same period of time before compression. The average height was now 35 millimetres, which number multiplied by 32 gives 1,120 millimetres of upward movement. This is equal to an increase of almost 39 per cent.—more correctly, 24.6. As abdominal pressure does not lower the aortic pressure, this amount represents an equal increase in the work done by the heart. The curve also shows that the increased force to the heart, resulting from abdominal pressure, leads to diminished contraction of the heart in systole, as well as to a relatively greater expansion in diastole. The period of compression begins immediately before the third vertical line, and finishes immediately after the fourth.](image)
or all of the systemic arterioles, as has been supposed by some. Our own observations show that all the blood which reaches the heart is thrown out by it into the arteries until incompetence of the auriculo-ventricular valves results from dilatation of the heart.

We do not, of course, for a moment suppose that the causes of heart failure in man ever act with the rapidity or intensity of those present in this experiment. What most usually takes place apparently is that the mitral valve, whether from primary disease or from overstrain, gives way gradually. This gradually-produced regurgitation is in part compensated by rise of pressure in the pulmonary veins, which is kept up until failure of the right heart the tricuspid gives way.

Let us now consider briefly some of the special pathological conditions of the heart, on which light appears to us to be thrown by what we have stated above. The much discussed question as to the possibility of hypertrophy of the heart in plethenosis without increased arterial pressure appears to us to be answered by the detailed facts and considerations. They appear to us to offer a sufficiently simple explanation of this form of hypertrophy. We have, however, never seen such cases.

Of acute overstrain of the heart from intense muscular exertion, one of us (R.) has on one occasion had personal experience; when, during convalescence from typhoid, he found himself called upon as a medical man to make a fatiguing and rapid journey with a relapsing patient up to the Morzine, and sent him in the course of the ascent to a Chamouni guide who had been severely injured by an Alpine accident. The sensations felt are well described by Clifford Allbutt, with whose observations on overstrain of the heart our own results fully coincide. The feeling of want of breath and fulness in the region of the heart, and the sensation of extreme muscular lassitude, are well-marked subjective phenomena.

With regard to the objective phenomena, it did not occur to the one of us personally involved in this matter to percuss out his heart, as was done by the more intelligent Clifford Allbutt, who found the area of dulness to be an extension of the condition as are appreciable to the non-medical eye the injured guide in question to remark, "Mais vous êtes essoufflé, Monsieur, il faut prendre du cognac," which treatment with rest was found efficacious, mere rest, however, it may be remarked, not giving the immediate relief obtained from it in cases of breathlessness from ordinary exertion. Swiss guides, who are exposed to extreme fatigue in cutting steps on ice in mountain climbing, are well acquainted with the condition, as has previously and independently been noted by Clifford Allbutt; they have found that cognac gives them a fillip to the system at the right time, and with rest gives relief.

The time at our disposal admitted of it we might say a good deal on the light which, we believe, is thrown by our observations on the pathology of heart failure in chlorosis and some other morbid conditions. The question of anemia ofangle of heart failure and the resulting overstrain thereby thrown on the organ, or part of the organ. We need, however, here only mention that dilatation of the heart in such cases, as well as in cases of primary disease of the heart-wall, is recognisable both at the bedside and after death. This dilatation we have shown to be a necessary result of increase in the work of the heart, as a result of increased arterial pressure or of increased output of the organ, and that failure of the heart only takes place when increase in the resistance of the auriculo-ventricular valves from dilatation of the orifices, makes it impossible for the heart to throw into the arteries all the blood that has reached it by the veins.

We have intentionally avoided referring in the above to the influence on the work of the heart of changes in the rate of beat of the organ. We have purposely also avoided entering on the question of hypertrophy, or of the effect on the heart of the vagus and accelerans. These are matters which we hope to deal with in a future communication.

AFFECTION OF THE VALVES FROM OVERSTRAIN.

We must now return to the relation between overstrain and secondary disease of the valves, and we have first to describe the anatomical changes in the valves which we found to result from artificially produced overstrain. Now, it is clear that these appear to us to throw much light on certain forms of valvular disease. If the figure by which the aorta is narrowed be placed round the ascending portion of the aortic arch, and then either repeatedly tightened for a short time, or by slower, or by continued slight narrowing, failure of the heart and regurgitation through the auriculo-ventricular valves, be produced, we find that, in nearly every case (six cases at least out of seven), certain valves are affected, and that these are the aortic and mitral, as well as the tricuspid valves are the seat of oedematous thickening. In the valves this thickening is most marked along the line of insertion of the flaps; these flaps themselves presenting also in some cases a varying degree of thickening. With regard to the mitral and tricuspid valves this thickening is situated chiefly along those parts of the flaps which are normally in apposition during systole. In other words, the oedema occupies those parts of the valves which are specially liable to become thickened by formation of fibrous tissue, in such diseases as rheumatic Bright's or syphilis, with secondary hypertrophy and valvular disease of the heart.

In other cases of overstraining, in which the aorta has been narrowed at a part of its course where there was no possibility of interference with the lymphatics of the heart, there has been no great thickening of the valves, although, in these cases also, we have always found on killing the animal, some slight thickening along the line of insertion of the aortic valves, and a very characteristic roughening of those parts of the mitral valve which are the seat of oedema in those of our experiments where, besides narrowing the aorta, we interfered with a certain extent, to the outflow of lymph from the heart. This roughening we could see, with the aid of a hand lens, was due to the dilatation of the beaded lymphatic vessels of the flaps. Very commonly also a dulness of certain parts of the endocardial covering on the valves led us to suspect a slight thickening of the endocardium of the valves. In all cases there were punctiform ecchymoses along the same parts of the mitral flaps, the rest of the endocardial lining showing only exceptionally any appreciable congestion or ecchymosis, and never being found oedematous by us.

Wherever, we may say, in passing, that the left segment of the tricuspid valve of the healthy dog usually presents a variable degree of thickening.

These anatomical changes in the valves, which result from overstrain, we interpret as indicating an increase in the amount of lymph in certain parts of the valves. The increase or stagnation of lymph tends to formation of fibrous tissue in the affected part, we know the case in many other tissues. Of these we may mention the fibrous thickening of the subcutaneous tissue of the arm, in the case of persons where cancer of the axillary glands obstructs the outflow of lymph from the limb. We are impressed by the fact that the situation of the oedema of the flaps is strikingly identical with that of the fibrous thickening present in the valves of those cases of heart disease which occur in conjunction with abnormal high arterial pressure. We consider the valves and the same phenomena in the same cause, namely, the increase in the quantity of lymph, which is possibly of a purely mechanical nature.

As to the question whether the endothelium be or be not liable to be stripped off the valves in cases of overstrain, and whether such stripping off, if it take place, might not be the cause of deposit of the blood platelets, or of fibrin, and thus lead to so-called "verrucose endocarditis," this is a matter which we cannot discuss here.

Plenty of examples of secondary disease of the valves from overstrain might be given, but we know of none in which the relation between cause and effect appears to us so evident as in the case of the heart changes present in chronic renal disease accompanied by high arterial pressure and hydremia, both of which, as has been seen, increase the work of the heart. This increased strain to hypertrophy of the organ; but, over and above this hypertrophy, disease of the valves is of specially common occurrence; Goodhart, for example, in his valuable paper on "Anemia as a Cause of Heart Disease," remarks that where there has been prolonged regurgitation on the right side of the heart, the tricuspid flaps are generally considerably thickened. Stenosis apparently never occurs in these cases. He says, also, that seldom, in cases due to a primary muscular fault, and to conse-

* St. George's Hospital Reports, vol. v, 1870, p. 29.

9 Reyher, Virchow's Archiv, xxl, 1861, p. 85, states that in thirty-three dogs examined by him and of the experiment of the septal flap of the tricuspid valve, and the other valves much more frequently diseased than has generally been supposed. As regards the tricuspid, it has been shown by Reyher, and there is no reason to believe that we have been led into error by mistaking these for the results of acute overstrain.

quent failure of the mitral valve by stretching, do we find the condition of the mitral one of simple dilatation of the orifices. The chordae tendineae remained unchanged, and the leaflets were thickened. There appears to be no stenosis in these cases where the failure is due to senile weakness of the muscle, or to degeneration.

"Take, next," he remarks, "another fact less well known than it ought to be, namely, that in a large number of cases of chronic renal disease there is some thickening of the mitral valve. Of 192 consecutive cases of chronic renal disease, 72, or more than one-third, had some thickening either of the mitral or the aortic valves, or both. In 49 of these the mitral valve was not thickened either or actually contracted. The mitral valve was thickened, and, therefore, very thick, in 9 cases, and all 9 were from cases of granular or verrucous valvitis. Of 68 of the 192 cases, some thickening of the mitral valve, and of these 49 had more or less thickening of the valve."

Goodhart states as his opinion that much of the mitral stenosis, where there is no history of rheumatism, and which is so common in women compared with men, is the outcome of the chlorate, mitral returgitation which is so commonly met with in girls and young women. With Goodhart's views on this subject we entirely coincide.

Dr. Clifford Allbutt expressed his great interest in Professor Roy and Mr. Adam, and, in the absence of any scientifically explaining clinical observations. He urged that the study of mathematics and physics was one of the chief needs in the education of young physicians, who proposed to devote themselves to original research.—Surgeon-General Maclean asked the attention of the Section to the practical bearings of this question from a military point of view. When the British army was passing through medicine at Chatham, he was struck by the enormous proportion of soldiers of short service who were constantly passing through Fort Pitt suffering from palpitation without cardiac murmur. This was traced to the immense pressure exerted on the chest of the soldier by tight clothing and ill-contrived accoutrements. A committee was formed, chiefly under the guidance of Professor Parkes, which resulted in a very marked demonstration of heart disease in the army, and led to an improved system of accouting the soldier. One other feature remains,—namely, the system of setting-up drill by the drill sergeant. This subject has been brought about by the "setting-up drill" on the drill ground; it has been brought to notice by Dr. Davis, and is likely soon to be remedied by an improved system of drill.—Dr. William Hunter desired to add his testimony to the great value and importance of Professor Roy's paper. He had had the opportunity of seeing Professor Roy and Mr. Adam engaged in many of their experiments on the heart, and nothing in connection with the subject had struck him more remarkable than the fact that the heart could be subjected to an amount of manipulation far in excess of what one could a priori conceive possible without apparently in any way interfering with its action. The condition of ecchymosis and edema of the initial distensibility of the aorta to which the unit had frequently occasion to observe and verify. The condition appeared to him one of no little importance in throwing light on the mode of production of chronic changes in these valves in cases of increased pressure of long standing, such as that met with in chronic Bright's disease—Dr. Boyes Smith (Netley) spoke to the profoundly important bearings of Professor Roy's paper, particularly in its bearings on the efficiency of the British army. There was a vast drain on the national exchequer from the results of heart failure in the person of the young British soldier. Acute "overstrain" of the heart was to the public mind a question, and Dr. Smith, speaking of cases long ago characterised as the "dynamic" heart, it led to great cardiac disturbance (telling apparently at first upon the right heart), to palpitation, extended heart sounds, extended thrill, fugitive murmurs. The physical signs of organic lesions did not exist, and yet the soldier was, for the time at least, altogether unfit for active military duty. His condition was a matter of serious concern, and, without his being invalided from the army, after which, probably from renewed "compensation," or rather from rectification of disturbed dynamic conditions, he sufficiently regained his health to be fit to earn his livelihood in civil life. Dr. Smith ended at some point into the differential diagnosis of conditions of acute heart strain, and noted its causes, and he dwelt with great emphasis on the incalculable value of such researches as Professor Roy had been good enough to lay before the Pathological Section.—Dr. Gordon Hardie observed constantly in India the supervision of func-

palpitation of most severe degree in men in hospital whose only antecedents had been unable insomniac, fever, post-grippe or of malarial neurosis. The palpitation came on in hospital under no conditions of strain, but of repose and loose hospital dress. In his experience of invaliding of the British army the amount of valvular disease, especially aortic, in men apparently in perfect health exceeded anything he had ever encountered in any other army, the Surgeon-General of Addis, of Guy's Hospital, used to tell his pupils that then it was originally the disease of the heart of pavilions and hod-men.—Dr. Finlayson (Glasgow) said that when he was asked by the secretary to take part in this discussion he felt it to be an enormous task to be able to discuss the overstrain of the heart than was quite familiar to all hospital physicians. On reflecting, however, he thought there were perhaps two subjects he could bring forward worthy of remark. A young gentleman had suffered from cardiac symptoms dating from a distinct acute overstrain when a boy at school. The medical attendant took then the somewhat too common opinion of heart disease in young subjects, that he would get all right when he was about 21. After waiting for years very patiently, he found his cardiac symptoms as bad as ever. On examination the heart was found certainly enlarged and apparently could have been in the left ventricle; as the result of the then known causes being discernible. Distrusting his own examination, with such negative results, Dr. Gairdner's opinion was sought, but he found no other explanation than the overstrain some years before. In this case the peculiar internal distress was relieved for a few minutes only without, however, he recovered involuntarily that the distress passed away for the day. In this relief some explanation of the symptoms in this case might be sought. Dr. Finlayson thought the overstrain of the right ventricle, from bad attacks of whooping-cough, seemed at times the origin of the apparatus for registering the volume of the kidney under varying circumstances. Dr. Coats desired to find some physiological explanation of the symptoms of overstrain, more particularly of palpitation. He had not been able to form a proper physiological conception of palpitation, which is the most constant result of overstrain. The promptness with which palpitation occurs, when patients suffering from heart disease ascend a few steps of a stair, is striking, especially in view of the fact that such persons can often walk considerable distances on a level. Dr. Coats also referred to the great variety in ability in the heart in different persons. He believed that the power of survival in acute diseases was often great, and that its longevity in man was more dependent on the ability of the heart than on any other factor.—Dr. John Lindsay Stevnn could not enter upon the physical aspect of this question, but wished to state that, from chemical observations during the past few years, the conviction has been growing upon him that there was a distinct class of heart cases whose origin was distinctly to be attributed to overstrain, the result of laborious occupation of some kind. He referred (in addition to the remarks made at the meeting by Surgeon-General Maclean, of Netley, and Professor Boyes Smith, of Netley) to the observations of Peacock on the Cornwall heart, and to Dr. Forster on the Russian heart, to the occurrence then described of case of heart disease from overstrain, in which he had made a post-mortem examination. In conclusion, he referred to the influence of the coronary arteries on the muscular diseases of the heart.—Dr. William Russell (Edinburgh) joined in expressing his appreciation of the communication made to the Section of Heart Disease, and not attempted to discuss the immediate points after hearing it. Dilatation of the ventricle, and the consequent mitral regurgitation, was well-known now to physicians, and it was important to have it confirmed in this way. The confirmation also of the observations of pathologists as to the site of the early changes in valvar affections was of great interest, and very suggestive.