Remarks on failure of the heart from overstrain : being the opening of a discussion in the Section of Pathology at the Annual Meeting of the British Medical Association, held in Glasgow, August, 1888 / by Prof. Roy and J.G. Adami.

With the authors compliments

REMARKS ON

FAILURE OF THE HEART FROM OVERSTRAIN,

BEING THE OPENING OF A DISCUSSION IN THE SECTION OF PATHOLOGY AT THE ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION, HELD IN GLASGOW, AUGUST, 1888.

BY

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ON FAILURE OF THE HEART FROM OVERSTRAIN.

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 valvular 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,[1] 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 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

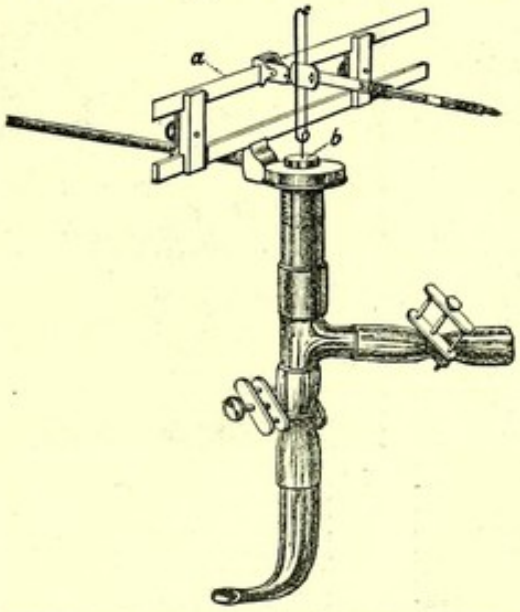

Fig. 1.—Intra-cardiac Pressure-gauge. The glass cannula shown at the lower part of the instrument is introduced into the ventricular cavity, the side branch of the T tube being closed. The instrument is filled with oil or other fluid, which transmits the pressure within the heart to the piston *b*, this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

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 anæsthetic, 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,

[1] *Journal of Physiology*. Vol. viii., 1887, p. 237.

this increase in pressure having a limit, and this limit varying in different animals. The intra-ventricular pressure in the left ventricle during systole, which in the dog is normally something under 130 millimètres mercurial pressure, can, by gradual narrowing of the ascending aorta, be raised to 250 millimètres, 300 millimètres, or even a little more, 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 often repeated great narrowing, or complete closure of the aorta, this maximum limit gradually falls. We may add also 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 distension 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 regurgitation 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 intra-ventricular 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 influences 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.

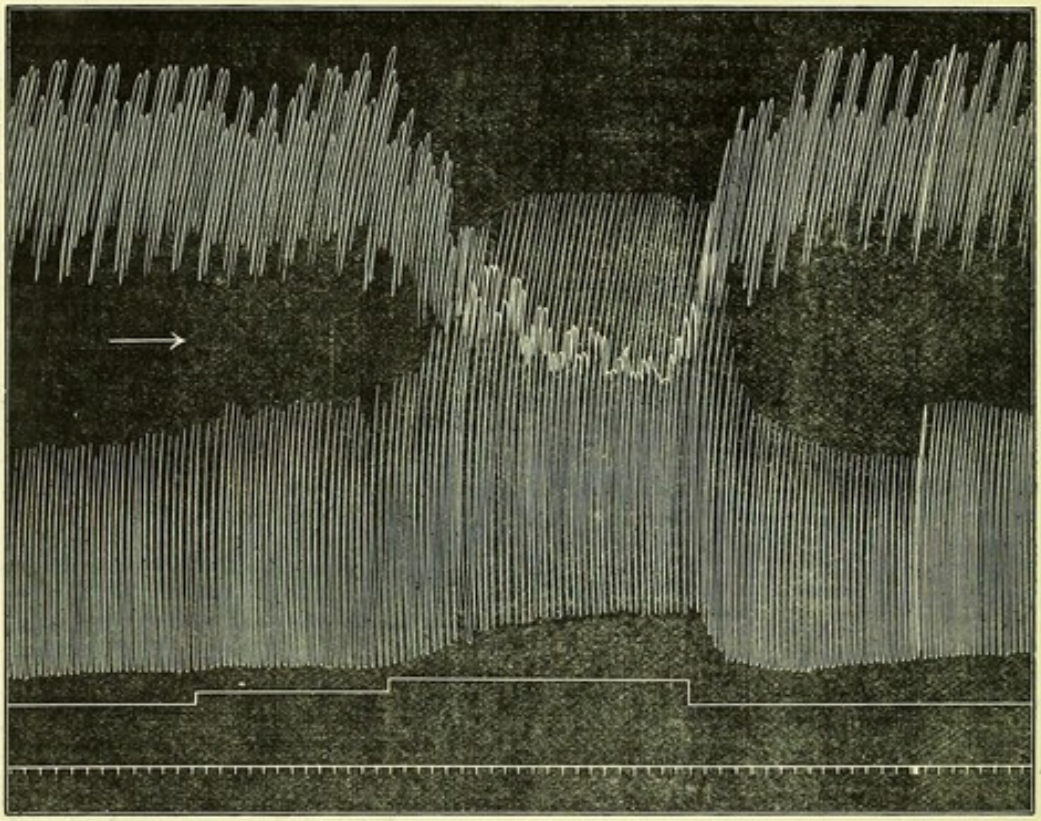

Fig. 2.—Simultaneous tracings of cardiac pressure-gauge and cardiac myograph, applied to left ventricle of dog. The time-curve gives intervals of one second. The signal-curve shows during the slightly raised part the time during which the ascending aorta was slightly constricted, while the middle part of the curve, which is raised higher, gives the time during which the aorta was completely closed. The point of this curve, at which it returns to its original level, indicates the moment when the ligature round the aorta was completely removed. The upper curve was taken by an instrument which permits of a graphic record being obtained of the distance between any two points on the heart wall. [This instrument we hope to describe elsewhere.] The two points on the front of the left ventricle from which the curve was taken lay in a line parallel with the interventricular sulcus, and were situated about halfway between apex and base of the ventricle. Upward movement of the lever point corresponds to approximation of the points chosen—in other words, to contraction of the wall of the ventricle. The individual contractions of the heart, as well as the effect of the respirations, can be readily recognised, together with the great expansion of the ventricle during the period of closure of the aorta. During this time, also, it can be seen that the shortening of the muscular fibres of the ventricle in systole is greatly diminished. The larger curve gives the intraventricular pressure rise of the lower point corresponding to rise of pressure. The slight rise of pressure in systole during the period of slight constriction of the aorta is well shown, as well as the fact that the pressure in diastole remains unchanged. During the period of complete closure of the aorta the systolic pressure soon rises to its maximum limit, which in the case of the heart in question was about 250 millimètres mercurial pressure. During the same period of time the diastolic pressure rises gradually to an extent relatively much less than the rise in systolic pressure.

Ludwig's *Stromuhr*[2] 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 itself of 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 both 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 for all practical purposes a rigid air-tight box, the variations in whose contents can be recorded graphically. In other words, it is a cardiac plethysmograph, or oncograph. We will in the following pages refer to it by the name cardiometer.[3]

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 having 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

[2] Stolnikow, *Die Aichung des Blutstromes*. Dubois-Reymond's *Archiv*, 1886.

[3] Both cardiometer 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

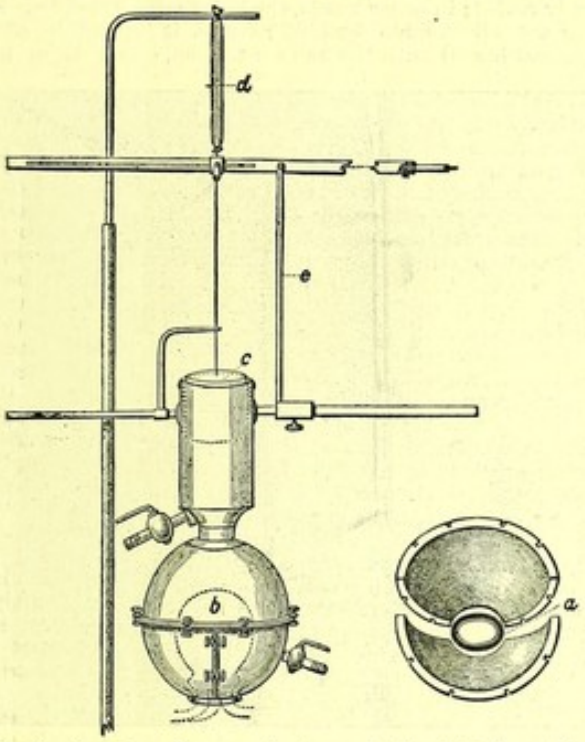

Fig. 3.—Cardiometer. The separate figure to the right shows the parts (seen from above) composing the lower part of the box, and which are represented detached from one another. The oval ring *a*, composed of an inner metal ring surrounded by a circle of india-rubber, is first placed round the root of the heart, as close as possible to the reflection of the pericardium, so that the parietal layer of that membrane is outside and the visceral inside the ring. The two halves of the lower hemisphere are then screwed together, the oval opening at the bottom clasping 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 expulsion 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

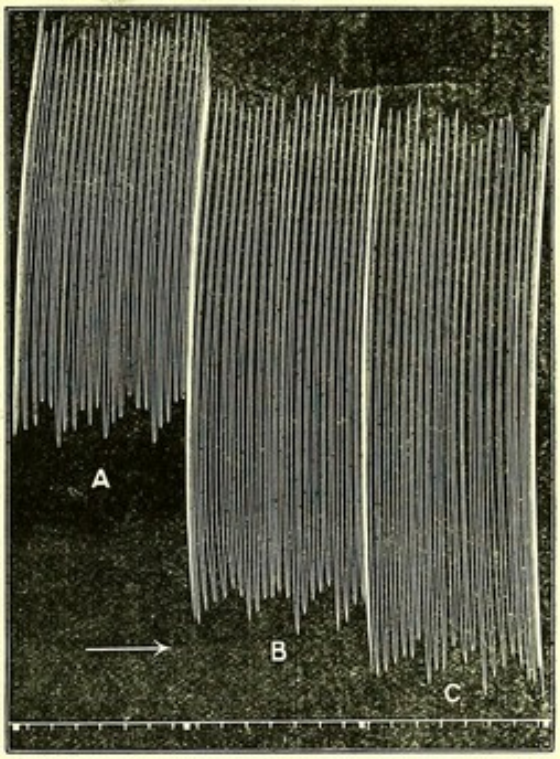

Fig. 4.—Cardiometer tracing, showing the effects of an injection of 50 cubic centimètres of normal salt solution into the external jugular vein of dog. A, before injection; B, after the injection of 30 cubic centimètres; C, after the injection of 50 cubic centimètres (in all) of the solution. The time-curve 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 (base 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, *cæteris 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 incompetence 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, three-quarter per cent. salt solution, or other appropriate fluid, into one of the veins. For example, we find in one experiment (illustrated by Fig. 4) that injection of 50 cubic centimètres of salt solution produced an increase in the mechanical work done by the heart equal to about 34 per cent.

The calibre 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, without any 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 great 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.

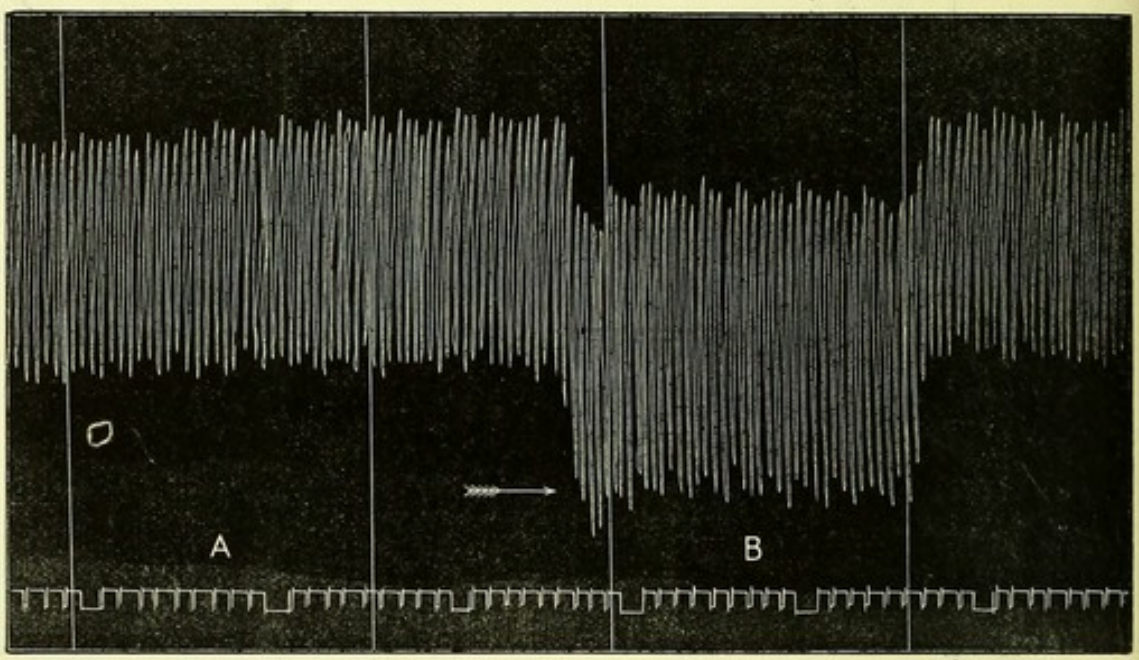

Fig. 5.—Cardiometer tracing, showing effect of abdominal compression on the amount of blood thrown out by the heart. The time-curve gives intervals of one second. During the period between the two vertical lines at A the heart gave 32 contractions, the average height of the movements of the lever-point resulting from these being 27 millimètres. Multiplying 27 by 32 we obtain 864 millimètres 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 millimètres, which number multiplied by 32 gives 1,120 millimètres of upward movement. This is equal to an increase of almost 30 per cent.—more correctly. 29.6. 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 inflow 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.

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—namely, 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 musculi 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, *cæteris 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 in the arterial pressure, or from weakness of the heart muscle resulting from fatigue or from disease of its walls, the muscles at the basis of the ventricles no longer in systole narrow the auriculo-ventricular orifices to a degree which permits of these orifices being closed by their valves, these not being capable of expanding to the same extent as the muscular substance of the heart. This fact is very prettily illustrated by an experiment, we believe, of Cohnheim's, and which we have frequently performed and seen performed; it consists in narrowing the ascending arch of the aorta by a screw clamp while the arterial pressure is recorded by a mercury manometer connected with one of the carotids, the clamp therefore being placed *between* the heart and the point at which the arterial pressure is measured. As the clamp is gradually screwed up there is no corresponding fall in the manometric pressure, such as might *a priori* be expected. What happens is that when the aorta has been narrowed to a considerable extent, which varies in different animals, the arterial pressure suddenly falls, and if the clamp be not removed the animal dies. This continuance of the normal pressure up to the time when the sudden fall occurs is not due to narrowing of any

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 from 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 plethora 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 relieving party over the Mer-de-Glâce to the "Jardin" to attend to a Chamouni guide who had been severely injured by an Alpine accident. The sensations felt are well described by Clifford Allbutt,[4] 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, as well as the sense of extreme muscular limpness, 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 increased; but such characteristics of the condition as are appreciable to the non-medical eye led 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 a fillip to the system at the right time, and with rest gives relief.

Did the time at our disposal admit 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 forms of anæmia and hydræmia. Much might also be said on the effects of disease of the different valves of the heart 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 either of increased arterial pressure or of increased output of the organ, and that failure of the heart only takes place when incompetence 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, seeing that these appear to us to throw much light on certain forms of valvular disease. If the ligature 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 portions both of the aortic and mitral, as well as the tricuspid valves are the seat of œdematous thickening. In the aortic 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 the thickening is situated chiefly along those parts of the flaps which are normally in apposition during systole. In other words, the œdema occupies those parts of the valves which are specially liable to become thickened by formation of fibrous tissue, in such diseases as chronic Bright's or syphilis, with secondary hypertrophy and valvular disease of the heart.

In other of our experiments, 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 œdema in those of our experiments where, besides narrowing the aorta, we presumably interfered, to a certain extent, with the outflow of lymph from the heart. This roughening we could see, with the aid of a hand lens, was due to the distension of the beaded lymphatic vessels of the flaps. Very commonly also a dulness of certain parts of the endocardial covering of the valves led us to suspect a shedding of the endothelium. 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 œdematous by us.

We may note in passing that we are aware that the left segment of the tricuspid valve of the healthy dog usually presents a variable degree of thickening.[5] 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. That such increase or stagnation of lymph tends to formation of fibrous tissue in the affected part, we know is 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 œdema 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 abnormally high arterial pressure. We believe the œdema and the fibrous tissue to be due to 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 socalled "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 hydræmia, both of which, as has been seen, increase the work of the heart. This increased work leads 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 "Anæmia 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."[6] Stenosis apparently never occurs in these cases. He says, also, that

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

[5] Reyher, Virchow's *Archiv*, xxi, 1861, p. 85, states that in thirty-three dogs examined he constantly found thickening of the septal flap of the tricuspid valve, and the other valves much more frequently diseased than has generally been supposed. We are acquainted with the appearances described 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.

[6] *Lancet*, vol. i, 1880.

seldom, in cases due to a primary muscular fault, and to consequent failure of the mitral valve by stretching, do we find the condition of the mitral one of simple dilation of the orifice. The chordæ tendineæ and the edges of the valves are usually thickened. There appears to be no stenosis in thsoe 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 a notable, and sometimes extreme, thickening of the mitral flaps." "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 49of these the mitral was either thickened or actually contracted. The mitral was contracted, and therefore very thick, in 9 cases, and all 9 were from cases of granular contracting kidney; 97 of the 192 cases were granular, and of these 45 had more or less thickening of the valves."

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 chlorotic, mitral regurgitation which is so commonly met with in girls and young women. With Goodhart's views on this subject we entirely coincide.