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ON THE INTERPRETATION

OF

CARDIOGRAPHIC TRACINGS,

AND THE

EVIDENCE WHICH THEY AFFORD AS TO THE CAUSATION OF THE MURMURS ATTENDANT UPON MITRAL STENOSIS.

By A. L. GALABIN, M.D.

The cardiograph is an instrument which has hitherto been in the hands of physiologists rather than of clinical physicians, and although it has done great service in demonstrating what is the normal succession of events in a revolution of the heart, is has been turned to but little account in investigating obscure points of cardiac pathology.

On account of the time and the extreme care which are required for its manipulation, and the difficulty in many cases of interpreting the curves which are obtained, it is far less suitable than the sphygmograph for any general clinical use. Yet it seems to be capable of useful application, first, in affording evidence which bears on the general question of the relation of some cardiac murmurs as to which observers are not yet fully agreed; and, secondly, in hospital practice at any rate, to aid in the diagnosis of those occasional obscure cases of heart-

disease, as to which the stethoscopic evidence is doubtful; or in which, without the existence of any definite bruit, the mode of action of the heart appears to be profoundly modified.

The form of cardiograph hitherto used is that invented by Marey, or a slight modification of it. In this the impulse of the heart is transmitted through a flexible tube containing air to a tambour or tympanum, to which the recording lever is attached, and the tracing is then drawn upon a revolving cylinder, called a polygraph. According to the construction ultimately adopted by Marey, a slightly excavated cup is applied to the chest so firmly that the contained air is hermetically enclosed. Within this cup is a spring which depresses the skin immediately over the apex of the heart, and by this means an impulse is communicated directly to the air enclosed in the cup from the motion of the whole surface of the chest which is contained within its circumference. In the cardiograph of Dr. Sanderson a small pad affixed to a spring rests upon the chest, and this is in direct communication with a tambour containing air, the motion of which is communicated through the flexible tube to a second tambour on which rests the recording lever. Thus, in both cases the recording lever is balanced upon a spring of air, the most delicate of all springs, and hence, if any sudden and considerable motion be imparted to it, there must necessarily be a probability that an oscillation may be set up by the effect of acquired velocity. It may readily be seen that this is the case if a sudden impulse be given artificially to the lever.

This liability to error has been already noted by eminent physiologists. Thus Dr. Rutherford, in his "Lectures on the Circulation of the Blood" ('Lancet,' Nov. 25, 1871), points out that a merely momentary touch upon the tambour of the cardiograph causes the lever to describe not a single wave, but a number of waves, these wavelets being due to the pendulum-like oscillation into which the column of air is thrown.

Another result of transmitting the impulse through a tube of air is that the motion actually recorded is the effect of a series of waves which have travelled through the elastic medium, and these waves in their progress are liable to a gradual change, by which their suddenness is lost, and they become more rounded.

These two imperfections do, indeed, to a great extent, neutralise each other, with the result that the cardiogram so

obtained does not differ widely in its main features from the true curve, for the more gradual impulse is less likely to call out oscillations, which would certainly occur if the motions imparted had the suddenness which characterises those of the heart itself.

Dr. Rutherford mentions that the most perfect cardiograms hitherto obtained have been procured by the application of the sphygmograph directly to the chest, and some very complex tracings of this nature have been published by Mr. A. H. Garrod in the 'Journal of Anatomy and Physiology,' for November, 1870. But the sphygmograph cannot be generally applied in this manner in cases of heart disease, for two reasons. In the first place, when the heart is hypertrophied, the extent of motion communicated to the lever is far too great to be recorded; and, secondly, on account of the proximity of the bars on which the instrument rests, when the heart's impulse is diffused, motion is communicated not only to the spring which moves the lever, but to the framework of the sphygmograph. What is recorded is, then, not the absolute motion at any point, but the differential motion between the several parts of the chest wall, and a tracing which should be positive may be converted in this way into one which is wholly or partially inverted.

I have, therefore, had constructed a form of cardiograph,[1] which is simply such a modification of Marey's sphygmograph, that in applying it directly to the apex of the heart these two difficulties may be avoided. In this instrument the knife-edge, through which motion is communicated to the recording lever, is attached by means of a sliding bar which can be fixed by a screw in any position. In this way the amount of amplification given to the motion recorded can be varied from ten to ninety times the original. The frame of the instrument, instead of resting on two parellel bars of ivory, is supported on two transverse rods of steel, and these, again, rest on two pairs of vertical rods, each pair rigidly attached to a bar of wood, covered with leather, which is pressed against the chest. The vertical and horizontal rods are united by such joints that all adjust-

[1] This cardiograph was made for me by Mr. Clark, of 3, Windsor Cottages, High Street, Lower Norwood. Instruments of the kind may also be obtained through Messrs. Krohne and Sesemann, Hawksley, or Weiss.

ments, both vertical and horizontal, are allowed. By horizontal adjustments the width between the wooden bars is varied according to the size of the chest, so that they may be applied beyond the range of the cardiac impulse; by the vertical adjustment the frame of the instrument is raised or lowered until the spring is in a suitable position to rest upon the apex. Lastly, there is a screw by which the pressure can be varied within any required limits, and a small secondary spring to keep the recording lever in apposition with the knife-edge, which can either be used or thrown out of gear as desired.

This cardiograph not only secures greater accuracy by representing the motion without any intermediate stage of transmission, but has the advantage of being more portable and much less costly than the ordinary form. Moreover, since no interval of time is occupied as in the other case, by the conveyance of the wave through a tube, it can be ascertained, by watching the lever at the moment of its application, what is the relation of the chief features of the curve to the sounds heard by the stethoscope, and the shock or thrill felt by the finger. This is sometimes essential for the interpretation of the more complex curves in which some of the usual elevations are partially inverted. It is found that tracings thus obtained have sharper and finer features than those procured by the ordinary cardiograph, and sometimes, when a thrill which accompanies a bruit is very coarse and well marked, it appears in the tracing, and thus the precise relation of the bruit is permanently depicted in the cardiogram.

As to the mode of application, it is often sufficient to rest the frame of the instrument upon the chest, and steady it there by the hand. But frequently advantage is gained by passing round the back two straps which are made partially of elastic material. In consequence of their elasticity they yield somewhat as the chest expands in inspiration, and the effect of the respiratory curve on the tracings is thus diminished, and the recording lever is not thrown beyond the limits of the paper.

The most perfect tracings are obtained when the breath is stopped about the end of a normal expiration, and held during the time that the clockwork is in motion; and in the case of

healthy hearts this is often essential for success. But with many persons it is a condition very difficult to secure, especially when they are suffering from thoracic distress, and when the heart is much hypertrophied it becomes of less importance, because then the amplitude of the cardiac movement is much greater in proportion to that of the respiratory curve.

In many cases also it is of interest to obtain the combined curve, for it is often found that an exact rhythm exists between the cardiac and respiratory movements, each respiration comprising precisely two, three, or four pulsations.

Instances of this are shown in Pl. II, fig. 5, and Pl. III, fig. 18. In such cases, especially when there is much dyspnœa, the beats of the heart rapidly diminish in vigour while the breath is held, and sometimes lose their characteristic features. If, however, the combined curve be procured, it is often found that, if the respiration be not extremely hurried, a single perfect revolution of the heart is shown during the pause which follows the end of expiration. Such a pause extends over about one and a half of the two beats shown in Pl. III, fig. 3.

The majority of the tracings upon which the present paper is founded were taken with the cardiograph here described. Some, however, were obtained with a sphygmograph, which has a similar adjustment for varying the amplitude given to the trace, means being taken in its application to prevent the framework touching the chest within the range of the cardiac impulse. Its action is therefore practically identical with that of the cardiograph, except as regards the slower motion of the clockwork, which occupies eight seconds for the whole transit, the corresponding interval for the cardiograph being five and a half seconds. Tracings taken with this instrument are marked B in the explanation.

The interpretation of the cardiac trace necessarily involves far greater difficulties than that of the arterial pulse. While the latter denotes only the variations of blood pressure, there are at least four conditions of the heart which may affect the cardiac curve; first, its firmness or relaxation; secondly, its state of repletion; thirdly, its locomotion as a whole; and, fourthly, changes in its shape, or the direction of its axis

during its contraction. Thus even now three different theories are held as to the causation of the apex-beat. The first ascribes it to the sudden rigidity and roundness assumed at the commencement of contraction by the heart, which during its state of relaxation is deformed and flattened by pressure against the containing cavity; the second attributes it to the motion of the heart as a whole, causing a literal impulse against the thoracic wall;[1] whilst, according to the third, it is due to the change of shape of the heart in contraction. If the heart of an animal be exposed, it is said that the apex is seen to be jerked forwards and upwards at the end of each contraction, and this is supposed to produce an impact against the chest wall. It is considered to be due to the greater length of the anterior as compared with the posterior muscular fibres of the heart, in consequence of which their contraction is more considerable.[2]

On looking at a normal cardiac tracing (Pl. I, figs. 1, 2, Pl. III, fig. 1), it is seen to differ from a pulse tracing (Pl. I, fig. 3), in the fact that its elevated portion forms a somewhat broad, square summit, commencing in a sudden ascent, and ending in a descent nearly as sudden. If the lever be watched while the finger is placed upon the apex, the pressure against the finger is felt throughout the whole time that the lever is elevated; and if at the same time we listen to the heart sounds, we find that, speaking roughly, the first sound is synchronous with the sudden ascent, and the second sound with the sudden descent of the lever. It is clear, then, that the whole of the square summit corresponds to the apex beat as felt by the hand, and also to the systole of the ventricle. The same thing is shown by a reference to the interesting tracings by which Marey first demonstrated, in the graphic method, the succession of the cardiac movements. He introduced sounds terminating in elastic ampullæ into the right auricle and also the right ventricle of a horse, and placed another ampulla in an opening made in the chest-wall over

[1] "Remarks on the Presystolic Cardiac Murmur." By Dr. Barclay. 'Lancet,' 1871.

[2] *Vide* "Lectures on the Circulation," by Dr. J. B. Pettigrew. 'Edinburgh Medical Journal,' March, 1873.

the apex of the heart. Then by means of the polygraph he obtained simultaneous tracings of the movements of the three, copies of which are shown in Pl. I, figs. 4 and 5. It will be seen that the principal ascent and descent in the apex-tracing and in the curve of ventricular pressure are synchronous.

It will be well to commence by analyzing the elevations into which the systolic eminence is divided. On looking over a large number of tracings from hearts healthy and diseased, it will be found that the most important of these are two; first, a high pointed summit, *d*, which terminates the sudden rise; and, secondly, a swelling, *f*, generally more rounded, which precedes the sudden fall. They are usually separated by one or more other elevations, but the more the heart is hypertrophied the more prominent in comparison do these two become, and it may therefore be inferred that they depend most directly upon the muscular movements of the heart, because, when hypertrophy is great, the effect of any oscillations either of the blood or of any solid structures would become less noticeable in proportion. (See especially Pl. I, fig. 13; Pl. II, figs. 3, 8; Pl. III, figs. 4, 6, 8, 14, 18.) We have therefore first to seek the meaning of *d* and *f*. They have some little resemblance to the so-called percussion and tidal waves of the pulse tracing, but their separation cannot, as in that case, be attributed to the effect of acquired velocity in the instrument, first, on account of the smaller elevations which intervene, and secondly, because they sometimes are separated by a very deep notch, especially in the case of healthy hearts when acting rapidly. (Pl. I, fig. 14, Pl. III, fig. 18.)

Marey's figures prove that the first part at any rate of the cardiac impulse is not due to any stroke against the ribs caused by locomotion of the heart as a whole, which could only commence after the opening of the aortic valves, nor to any shape assumed by it at a late period in its contraction. Pl. I, fig. 4, shows that the cardiac impulse commences at the moment when pressure first begins to rise within the ventricle, and fig. 6 shows that the aortic valves do not open until the ventricular pressure has nearly reached its first maximum, after an interval, which in that particular case was about $\frac{1}{10}$ second.

The theory of the causation of the apex beat now accepted

by the best authorities, is that which attributes it to the sudden rounding and hardening of the ventricles at the beginning of systole. This rests chiefly on the direct experiment of opening the thorax of a living animal, and introducing the hand between the heart and the ribs. It follows that the so-called apex-beat is not produced by the actual apex, but by the surface of the ventricles most closely in contact with the chest wall. Thus, if needles be introduced from without so as to penetrate the living heart of an animal at different points, the one introduced at the centre of impulse has no transverse motion, while the external ends of the others move outwards during systole in proportion to their distance from it, thus denoting a drawing inwards of their points.[1] Hence the spot at which the so-called apex-beat is felt is the centre towards which the muscular mass of the ventricles draws itself together, and the same hardening of the ventricles produces a heaving outward over a limited area, and at the same moment a retraction beyond that area.

The same conclusion may be drawn from the use of the cardiograph. When the heart is dilated and hypertrophied, so that an impulse is felt in several intercostal spaces, the spot of greatest motion, and that which gives the most perfect tracing, is not usually the lowest space in which impulse is felt, nor that which corresponds to the position of the apex when examined after death. Again, if tracings taken from different spaces be compared, the same elements can be recognized in the systolic portion of each, although their relative magnitude is altered; thus showing that the impulse is not caused by contact with any one point of the heart only (compare Pl. II, figs. 3 and 6, and figs. 11 and 12). Beyond a certain area, however, the impulse is replaced by suction, and the tracing becomes inverted.[2] Generally there is an intermediate area in which the inversion is partial; the commencement of systole is marked by a sudden short rise, but this is immediately followed by a rapid fall. The tracing in Pl. II, fig. 2, is of this character, and those in Pl. II, fig. 4, and Pl. III, fig. 5, and perhaps fig. 11, show a similar rapid descent during systole.

[1] *Vide* 'Handbook of the Physiological Laboratory,' edited by Dr. Sanderson, page 254.

[2] Ibid., p. 255, and fig. 232 *b* compared with fig. 232 *a*.

It often happens that when the centre of impulse corresponds to a rib and not to an intercostal space, or when the heart is covered by emphysematons lung, only a partially or wholly inverted tracing can be obtained at the apex. The mixed tracings are often too complex to interpret (Pl. II, fig. 2), but those wholly inverted may be available (Pl. I, figs. 10, 11, 12, 16), although they rarely show the minor waves so distinctly as those that are positive. Negative tracings are obtained most perfectly when the breath is held in deep inspiration, instead of in expiration. To see most clearly their correspondence with positive tracings, they should be turned upside down and read from right to left.

If we consider what sort of curve would be produced by the hardening of the ventricle, it is evident that it would rapidly rise to its maximum height, which would be reached almost immediately upon the closure of the auriculo-ventricular valves, and that from the summit there would be a continuous descent during the rest of systole due to the diminution of size of the heart as it contracts. This point may also be tested by direct experiment. The tracing in Pl. I, fig. 8, was obtained from the heart of a dog whose thorax had been opened. The spring-pad was placed upon the surface of the right ventricle, near the auriculo-ventricular groove, so that no elevation in the trace could be produced by the locomotion of the heart in the direction of its apex. The spring indented the heart during its relaxation, and its elevation was obviously due to the hardening of the ventricle, and commenced from the moment that contraction was first visible. Fig. 7 was obtained from the same heart when its contractions were becoming slow, short, and imperfect, resembling those which may be supposed to occur in a dilated heart. It had then adopted a peculiar rhythm, so that its motions occurred in pairs, each stronger contraction being followed by one more feeble, as sometimes occurs with the human heart in cases of mitral disease, and especially when that valve is contracted. Looking at fig. 8, it is evident that the systolic portion of the curve has exactly the shape already anticipated on *à priori* grounds. Therefore the hardening of the ventricle accounts fully for the main part of the elevation in the curve, and for the first pointed summit *d*, but not for the elevation *f*,

which completes the square top commonly seen in the apex tracing.

For the swelling, *f*, therefore, another explanation must be sought. Its relation may perhaps best be studied by comparing the series of inverted tracings, Pl. I, figs. 10, 11, and 12, obtained at different distances from the apex of a healthy heart. Fig. 11, taken near to the apex, shows all the waves of the normal curve inverted, except that *d*, which is here due to the retraction produced by the hardening of the ventricle, is less sudden and great, in proportion to *f*, than the corresponding elevation in positive traces. Fig. 10 was obtained in the epigastrium to the right of the middle line, at such a distance, therefore, that the effect of change of shape of the heart at its hardening would be lost, and only the suction due to its diminution in size be manifest. Accordingly we find that the depression *d*, has disappeared, and that the curve progresses gradually to a maximum, *f*, reached near the end of systole. Fig. 12, taken at an intermediate point, shows an intermediate character.

Thus we see that *f* corresponds in time to the maximum contraction of the ventricle. Since, however, when it takes the form of an elevation in the positive tracing, it cannot be due to the contraction of the ventricle as such; its cause must be sought in some coincident occurrence, and this can only be the locomotion of the heart as a whole. Such locomotion is produced by two causes, first, the lengthening and slight straightening of the aorta due to the increase of statical pressure within it; this would reach its maximum effect at about the middle of systole or a little later, as may be seen by a reference to the aortic tracings in Pl. I, fig. 6, and Pl. III, fig. 17. The second cause, and probably the most important, is the propulsion of the blood. If the heart were not held by its attachments, it would itself acquire a momentum equal to that impressed upon the blood, but in an opposite direction, according to the same principle as that by which a rocket is propelled, and, as it is held, it will move to some extent by the stretching of those attachments. Since this effect is due to acquired momentum, it will increase throughout the whole time the heart is contracting, and thus fully account for the rounded elevation, *f*, towards the end of systole. This conclusion is

confirmed by a comparison of the curves obtained under different circumstances, for when the heart is most hypertrophied, *f* is most prominent and rounded, and may even rise much higher than *d* (*vide* Pl. II, figs. 5, 10, 12, 13; Pl. III, figs. 3, 4). On the other hand, when dilatation preponderates, *f* may be almost lost (Pl. II, fig. 4; Pl. III, figs. 5, 8). Comparing tracings taken at different points, *d* has much greater proportionate magnitude at the centre of impulse, where the convex surface of the ventricles is most closely in contact with the chest, while the magnitude of *f* is more uniform at different points within the area of impulse. Sometimes *f* terminates in a sharp point as in Pl. II, fig. 16; this may possibly be due to the jerking forward of the apex at the end of systole, which is described by some observers of the exposed heart of an animal.

The smaller waves which often intervene between *d* and *f* are more difficult to interpret, and some of them may be due to the effect of acquired velocity in the chest-wall or the cardiograph. A rounded summit in the middle of systole, found in some cases of hypertrophy, is probably due to the combined effect of the two causes already considered, the hardening of the ventricle then taking place more deliberately, (*vide* Pl. II, figs. 7, 10). When, however, a distinct small elevation, *e*, intervenes between *d* and *f*, it may with some probability be ascribed to a recoil towards the apex of the blood, which had been forcibly impelled against the auriculo-ventricular valves at the moment of their closure. (*Vide* Pl. I, figs. 1, 2; Pl. II, figs. 14, 16; Pl. III, figs. 9, 10, 15).

The double character of the systolic summit is not very evident in Marey's tracings shown in Pl. I, figs. 4, 5, although it is so in some which he has published, taken directly by the sphygmograph. He ascribes the corresponding elevations, *h*, *h'*, *h''*, in the three tracings in fig. 4, to an oscillation in the tension of the auriculo-ventricular valves. Such an oscillation in the valves would raise the pressure on one side at the same moment that it depressed it on the other; and therefore, if this were the cause, it is clear that each elevation in the ventricular trace, and in that of the apex-beat, ought to correspond to a depression in the auricular curve; but on referring to the figure it will be seen that the opposite is the case. Nor can

any such correspondence be found in the similar series of tracings shown in fig. 5. Hence, it seems probable that these small waves in Marey's curves were due to oscillations in the column of air contained in his apparatus, and perhaps the small elevations, such as *b*, *e*, and *g*, shown in the present tracings, are similarly open to the suspicion of being produced in the instrument.

The direct cardiograph, however, is much less liable to such error than the sphygmograph, and *à fortiori* less than the tambour cardiograph. In the latter the trace may deviate from the true curve by a variation in the tension of the air, which may very easily occur; in the sphygmograph by a variation in the indentation of the artery, or in the compression of the elastic tissues covering it. This happens most when the pressure applied is small, and accordingly in such cases the so-called percussion wave of the trace is most manifestly separated. Similarly with the direct cardiograph, there may be a variation in the degree of compression of the structures which lie over the heart; but this is less likely to have any manifest effect, first because, since the moving force is greater, the pressure applied may also be greater; and, secondly, because, at any rate in the case of diseased hearts, the amount of amplification given to the trace is many times less. In some cases error might arise from the recording lever being jerked away from the knife-edge on which it rests, but this may be prevented by the use of the small secondary spring, and it is not found to occur unless its movements are very sudden and ample.

So far as I know, the only attempt by any English writer to explain all the features of the cardiac trace, is that made by Mr. A. H. Garrod, in the 'Journal of Anatomy and Physiology,' for November, 1870, although Dr. Balthazar Foster, adopting Marey's explanation of the normal trace, has discussed the significance of various pathological changes.[1]

Mr. Garrod's paper is founded on a series of tracings from the same healthy heart under varying conditions. Two out of his ten tracings, taken at a little distance from the apex, seem to me to be partly inverted, like that in Pl. II, fig. 2, and their features, therefore, too much obscured for any certain com-

[1] 'Clinical Medicine,' by Dr. B. Foster, London, 1874.

parison with the apex-traces. Mr. Garrod attributes the elevation, *d*, solely to the locomotion of the heart caused by the lengthening of the aorta. The rise, *f*, he considers to intervene between the end of systole, and the closure of the aortic valves, and to be due to the initial relaxation of the ventricle. It appears to me impossible that the relaxation of the ventricle, apart from its repletion, could produce an elevation in the curve, except in those cases in which its hardening produces a depression either at the commencement or towards the conclusion of systole, and the tracing is therefore wholly or partially inverted. Moreover, his view would be quite inconsistent with the great development of the swelling, *f*, in cases of hypertrophy, such as Pl. II, figs. 5, 12, 13; Pl. III, fig. 3; and with the wide space it occupies in Pl. I, fig. 14, and Pl. III, fig. 18; both taken from cases of chronic Bright's disease in which the arterial tension was very high, so that the valves would close rapidly. It is also contradicted by Marey's simultaneous tracings, for both fig. 4 and fig. 5, in Pl. I, show that the principal downstroke in the apex trace, which follows the eminence, *f*, is synchronous with the main descent in the curve of ventricular pressure, indicating the termination of systole.

Having now considered all the important waves seen on the summit of the systolic elevation, there is little difficulty in explaining the small rise, *a*, which precedes the main upstroke in the normal tracing (Pl. I, fig. 1, and Pl. III, fig. 1), and is seen also in many of the morbid curves. It will at once be attributed to the contraction of the auricles, and on reference to Marey's simultaneous tracings in Pl. I, figs. 4, 5, it is shown to be exactly simultaneous with that event. It is not always distinguishable in traces obtained from a healthy heart, and is clearly marked only where the apex-beat is very well defined.

We have next to consider whether any indication is found in the trace of the moment of closure of the valves. On listening to the heart by means of a flexible stethoscope while the eye is fixed upon the motions of the lever, it is found that the upstroke commences a little before the first sound is heard, and the greatest intensity of the first sound appears to correspond with about the upper third and summit of the upstroke. This most intense portion of the sound would seem to be the joint effect of the closure of the auriculo-ventricular valves, occurring

when about two thirds of the ascent are completed, and the full tension of the ventricular walls which coincides with the first summit, *d*. The fact that two constituents not exactly synchronous are thus combined in the first sound will account for its greater length as compared with the second, even without taking into account its third probable component, the sound of muscular contraction.

The closure of the auriculo-ventricular valves, therefore, occurs during the main upstroke. It might be expected to produce first a slight check, and then an acceleration in the ascent; the check, due to the shock of the blood against the valves impelling the heart in the direction of its base; the accleration, in consequence of the increased hardening of the ventricle as soon as it meets with resistance. Such a point is seen at *b* in some of the curves (Pl. I, figs. 13, 14, 15; Pl. II, figs. 6, 7, 19; Pl. III, figs. 12, 13, 15, 19), especially when the heart is hypertrophied, in which case the hardening of the ventricle appears to be less sudden. When, however, the main upstroke is sudden, the point *b* is not distinguishable.

Referring, again, to the evidence of the stethoscope, the second sound is generally heard about the conclusion of the sudden descent of the lever which follows the eminence, *f*. This varies, however, somewhat in different cases, and when arterial tension is high, it seems to occur near the commencement of the descent. In several of the tracings a slight notch, *g*, is seen at or near the bottom of the descent, especially in cases in which the arterial tension is low. (*Vide* Pl. I, figs. 2, 8; Pl. II, figs. 3, 7, 17; Pl. III, figs. 2, 8, 10.) This, if not produced in the instrument, may indicate the moment at which the valves close. In many cases, especially when arterial tension is high, this point appears to be lost in the more sudden portion of the descent. In two such cases, however, Pl. I, fig. 14, and Pl. III, fig. 18, the point, *g*, seems to be visible near the middle of the fall. In other cases, such as Pl. I, figs. 13, 15; Pl. II, fig. 16; Pl. III, figs. 11, 13, it is difficult to determine whether the small rise which follows immediately after the relaxation of the ventricle is to be identified with *g*, or with the larger elevation *k*, which has yet to be considered.

In Marey's tracings in Pl. I, fig. 6, the small rise, *b'*, in the ventricular curve seems to be synchronous with the wave *c*,

indicating the closure of the valves in the aortic trace, and the position of *b'* agrees well with the evidence afforded by the stethoscope as to the instant at which the valves generally close. On comparing this curve, however, with those in figs. 4 and 5, it will be seen that they differ very considerably at this part, and it appears rather uncertain whether the rise *i''* in fig. 4, attributed by Marey to the closure of the semilunar valves, really corresponds to *b'* or to *k* in fig. 6. It seems possible, therefore, that an oscillation in the column of air consequent on the sudden descent of the lever may have had something to do with these elevations in curves obtained with the polygraph.

In the normal tracing there is seen after the sudden fall a distinct elevation, *k*. This is a very constant feature also in the morbid curves, and it cannot be simply due to the recoil of the lever, for it varies greatly in magnitude and position, sometimes occurring later, as in Pl. III, fig. 2, and sometimes being separated from the main descent by the small notch *g*.

Looking at *k* in the healthy curve, Pl. I, fig. 1, we should be inclined to attribute it to the closing of the aortic valves, and it seems nearly to correspond to *i''* in Marey's diagram in Pl. I, fig. 4. On listening with the flexible stethoscope, however, it is found that the instant at which the second sound is heard corresponds to a point somewhat earlier than *k*, and the same conclusion may be drawn from the simultaneous tracings of the left ventricle and the aorta, Pl. I, fig. 6. Moreover, if measurements be taken of the curves by reducing all the points in them to a horizontal line of reference by means of circular arcs of a radius equal to the length of the recording lever, a comparison may be made with the length of the systolic portion of a carotid tracing, measuring this from the commencement of the primary upstroke to that of the dicrotic wave. If, then, a length equal to this systolic portion be measured on the cardiac tracing from a point near to the top of the primary upstroke, which may be taken as representing the moment when the aortic valves open, the end of it will be found not to coincide with *k*, but either with *g* or with some point on the principal downstroke. In Pl. I, figs. 2 and 3, such a comparative measurement is made between the portions of the cardiac tracing and of the radial pulse, which are believed to indicate

the interval between the opening and the closing of the aortic valves. It is better, however, to choose the carotid tracing for this purpose, because the pulse wave has there undergone less modification. The whole breadth of the elevation in the cardiac tracing is always greater than that of the systolic portion of the arterial pulse, because the aortic valves do not open until nearly the end of the principal upstroke of the former, as shown by Pl. I, fig. 6, combined with fig. 4, and, therefore, the systolic eminence in the apex trace includes the earliest part of systole which precedes the opening of the valves, an interval excluded from the systolic portion of the arterial tracing.

The elevation, *k*, then is not synchronous with the closure of the aortic valves, but we can hardly attribute it to anything else than to the reflux of blood which closes those valves, and it probably indicates a slight movement forward of the heart as a whole caused by that reflux. Such a movement would occupy a short interval of time, and hence we may conclude that its effect does not appear in the trace until after the end of the downstroke caused by the relaxation of the ventricle. Moreover, it is to be remembered that the reflux of blood does not cease with the closure of the valves, but continues an instant later, producing a slight second expansion or dicrotic wave in the aorta. This is seen in the aortic tracings, in Pl. I, fig. 6; and Pl. III, fig. 17, and although, in these instances, its magnitude is small, it is probably much greater, as it is in the radial pulse, when arterial tension is lower or the outflow more free. The proportionate magnitude of *k* is found to be much greater in some of the curves, as in Pl. I, fig. 14; Pl. III, fig, 18, and it might be supposed that its height has some relation to the dicrotism of the pulse or the arterial tension. Since, however, it would be the combined effect of the reflux in the aorta and in the pulmonary arteries, such relation appears to be too complex to be traced with certainty.

In some cases the spring-pad can only be applied in such a position that a retraction occurs in the latter part of systole, the tracing being, in fact, for a short space inverted. This is most likely to occur when dilatation preponderates over hypertrophy, so that there is not that prolonged propulsion of the heart against the ribs, which, when the ventricles are acting vigorously, produces a broad eminence in the position of *f*.

In these cases there is often seen, in the position of *k*, a sudden elevation, not followed by a fall, which is then due simply to the relaxation of the ventricle, and is not to be identified as to causation with the elevation, *k*, in the more perfect traces. Such curves may be recognised by the rapid descent during the systolic period from the first summit *d*, so that the eminence *f* is at a lower level than the commencement of the primary ascent. Caution is required in drawing inferences from traces of this kind, an example of which is shown in Pl. II, fig. 4.

It might be supposed that inversion or partial inversion of the trace would be an indication of adhesion of the pericardium, especially when such adhesion affects the base and posterior surface of the heart, and so tends to interfere with its locomotion forwards; and that thus the cardiograph might assist in the discovery of a condition the diagnosis of which is generally very uncertain. A visible suction at the apex has, indeed, been given as one of the signs on which to found such a diagnosis. It is certain, however, that the curve may be inverted in cases where the pericardium is not adherent. Nevertheless, it seems probable that if the heart's action be vigorous, and the centre of impulse distinct, and if at the same time there be a well-marked inversion of the trace in the latter part of systole, adhesion of the pericardium might be inferred. I have not, however, been able as yet to verify this by post-mortem examination. Dr. B. Foster has published a case in which the cardiogram assumed such a form during palpitation of the heart, and after death the pericardium was found adherent.

The curve in this case shows in each pulsation several ample and rapid oscillations,[1] which the author ascribes to the recoil of the chestwall at the early part of diastole, but which, so far as any conclusion can be drawn from so complex a curve, I should rather have supposed to occur during the contraction of the ventricle, especially as it is said that the apex seemed to strike the chest several times with each systole.

The elevation, *k*, as seen in the normal trace in Pl. I, fig. 1, cannot be due to the commencing influx of blood into the ventricle, for it would not then be followed by a fall almost equivalent to the rise. When, however, venous tension is very high, especially when the heart's action is also rapid, the

[1] 'Clinical Medicine,' Dr. B. Foster, p. 285.

eminence *k*, is more or less blended with the ascent, which indicates the repletion of the ventricle; and sometimes the effect of rapid flux into either ventricle, particularly in cases of mitral or tricuspid regurgitation, seems to be combined with an elevation, *k*, enhanced on account of extreme tension in the pulmonary arteries. (*Vide* Pl. II, fig. 12.)

The only portion of the trace yet remaining unexplained is that which intervenes between *k* and *a*. There is here a gradual rise due to the influx of blood into the ventricle, combined probably with its active dilatation, caused by the repletion of the coronary arteries. The ascent is not a straight line, but there is a slight rounding, *l*, which seems to be due to the fact that not simply an increase of statical pressure is indicated, but the influx of the blood with some acquired velocity.

In rapid pulses, *l* is generally merged in *k* or *a*. The rapidity of this ascent seems, as might be expected, to be increased in proportion to the venous pressure, allowance being made for the amount of amplification which has been given to the trace. No estimate of the rapidity of ventricular repletion, however, can be made from a tracing which is in any part inverted, and the first essential in cardiography is to learn to recognise at a glance such curves, and, as far as possible, to avoid drawing any conclusion from them, unless, indeed, it should prove that a diagnosis of pericardial adhesion may in some cases be founded upon this characteristic.

The systolic portion of the tracing varies according to the amount of dilatation or hypertrophy of the heart, and the mode of its muscular action, but in the diastolic part some more direct effects of valvular changes may be found. Thus, in aortic regurgitation the elevation, *k*, is prolonged into a more or less rapid ascent, due to the quick repletion of the ventricle, and by the magnitude of this ascent the freedom of regurgitation may in some measure be estimated. In the somewhat rare cases in which aortic regurgitation produces a marked thrill felt at the apex, this thrill may be depicted as a wavy line in the curve. Instances of this are shown in Pl. II, figs. 4, 9, 10, all taken from cases of very free regurgitation. In Pl. I, fig. 15, and Pl. III, fig. 2, are curves obtained from cases of aortic regurgitation in which the distress was comparatively slight, although hypertrophy was considerable, and in these the diastolic ascent is much less

marked. In some cases of very free regurgitation with, at the same time, great hypertrophy, there is not a gradual ascent leading up to the systole, but a very marked rise followed by a fall (*vide* Pl. II, figs. 17, 19). It seems that the aortic pressure being raised to a very high point in systole causes the blood to flow back into the ventricle with a powerful momentum during diastole, which produces its effect on the trace, not only by raising the ventricular pressure, but by impelling the heart bodily against the ribs.

The case from which the tracing in Pl. II, fig. 17, was taken was of rather a singular character. It is the only one I have met with in which, the apex beat being well marked, I have found that in laying the finger on the apex I mistook the first for the second sound of the heart, and correction was necessary by a reference to the carotid pulse. It seemed that the diastolic rise *k*, being accompanied by a sharper jar, gave a more manifest sensation to the finger than the systolic elevation *d*, *e*, *f*, although the summit of *d* is somewhat higher than *k*. There was heard in this case a systolic bruit at the apex, and a diastolic bruit at the base, so loud and short that it was mistaken by several observers of some experience for a simply intensified second sound. Post-mortem there was found extreme aortic incompetence, in addition to disease of the mitral valve. The narrow and lofty diastolic eminence, *k*, seems to correspond to the short aortic bruit, combined probably with the effect of extreme tension in the pulmonary arteries. From the appearance of the curve only, it might be taken for an increased auricular elevation, and this is, therefore, an instance in which careful comparison of the trace with the sounds heard at the time of taking it was necessary for its interpretation.

In ordinary cases of aortic or mitral regurgitation the cardiograph can add little to the information derived from the stethoscope. But it seems specially adapted to afford some evidence bearing on the mechanism of that cardiac murmur which has perhaps excited the most interest of any, namely, the presystolic. The mode of causation of this is still open to question, for although the majority have adopted the view maintained by Dr. Gairdner, that it is due to the auricular systole, yet some still ascribe it to the passive venous flow; and Dr. Barclay has argued that it is really regurgitant, and caused by the slowly

commencing ventricular contraction. The remainder of this paper will, therefore, be devoted to an attempt to elucidate this point by the aid of the cardiograph, and I have here to express my warmest thanks to the physicians and assistant physicians of Guy's Hospital for the courtesy with which they have allowed me to refer to cases which have been under their care.

I will first give brief notes of a case which appears to show that the natural rhythm of the auricular contraction may sometimes be greatly altered. Richard B—, æt. 34, was admitted into Philip Ward, under Dr. Moxon, on December 2nd, 1873. He had been a soldier, and two years before had been invalided on account of attacks of giddiness or faintness, and it was then noticed that his heart beat very slowly. He had never had rheumatism. Recently he had had three more attacks of faintness. On admission his pulse varied from 25 to 30 in the minute. The cardiac impulse was seen to extend in a wavy manner from the fourth to the seventh intercostal space; the apex being in the sixth space, and one and a half inches external to the nipple. A slight rough systolic bruit at the apex is stated in the report to have been heard at his admission, but in general no murmur was audible. No positive diagnosis was made of the state of the heart, but it was thought that the slow action might be due to aortic obstruction or to some disease of the muscular wall.

A tracing from this patient's heart taken in the fourth intercostal space is shown in Pl. II, fig. 14. The small irregularities in the long diastolic interval might, at first sight, be taken for casual movements, but another tracing, fig. 15, taken on the same day, shows a repetition of them so exact that it might almost be supposed a copy of the first. In fact, the same rhythm, extending over a cycle of two ventricular beats, was at this time constantly repeated, each cycle occupying an interval of somewhat more than four and a half seconds. In listening in this situation there could be heard between the strong pulsations either one or two faint beats which evidently corresponded to the elevations, *a*, in the curve, and which even produced a slight visible wave in the fourth intercostal space. Now, it is common, especially in cases of mitral stenosis, to find each beat of the heart followed by a feeble contraction of the ventricle which gives no apparent pulsation at the wrist, but in such cases

this second contraction is clearly shown in the sphygmographic tracing of the pulse. In this patient, however, nothing of the sort was to be seen in the radial pulse (*vide* fig. 18), and since, moreover, the feeble pulsations could not be heard at the apex, and were scarcely perceptible in the apex tracing, the conclusion seems clear that they were due to the contraction of the auricle only.

The pulse tracing (fig. 18) shows no indication of aortic obstruction, and possesses a long-sustained tidal wave, which is rare in cases of mitral regurgitation. Now the pulse is sometimes irregular and very slow when the mitral valve is contracted. In such cases the sphygmographic tracing shows an amplitude which is considerable, and in some instances much greater than normal, from which, and from the physical signs observed, it may be inferred that when the pulse has this character of extreme slowness, the left ventricle, instead of being small, is dilated and hypertrophied. It is just possible, therefore, that mitral stenosis may have been present in this instance; but, however this may be, we have here a heart the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long pause instead of just before the systole of the ventricle. It is, therefore, *à priori* not improbable that, in mitral stenosis, the auricle, perturbed in its action by the obstruction in front, may adopt an unusual rhythm. The audible auricular pulsation in this case may be compared with the short sharp first sound sometimes met with when the mitral valve is converted into a hard narrow ring, as showing that the click of closing valves is not the sole cause of cardiac sounds.

Of cases of mitral stenosis, the first I shall take is that of which the apex-tracing is shown in Pl. III, fig. 10. There was here evidence of much hypertrophy, the heart's impulse was heaving, its action regular and of moderate rapidity. At the apex was heard a presystolic murmur commencing shortly before the first sound, and running up to and ending in that first sound. Although not very loud it had the rough grinding quality characteristic of presystolic murmurs. The second sound was heard distinctly, and was separated by a clear interval of silence from the commencement of the murmur. The murmur was accompanied by a short presystolic thrill felt

at the apex. The cardiac tracing appears to leave no room for doubt that in this case the bruit was auricular-systolic. The auricular elevation, *a*, is seen to commence somewhat earlier than normal, and to be much increased in magnitude, its relative height being about two and a half times, and its absolute height, allowing for the difference of amplification, about fourteen times that of the normal rise in Pl. I, fig. 1. The reduplication of its summit seems to be an indication of the accompanying thrill.

Dr. Mahomed, in the 'Medical Times and Gazette' for April, 1872, was the first who published cardiac tracings, obtained by the aid of a flexible tube, as evidence of the prolonged auricular systole in cases of mitral stenosis. Of his three figures one resembles that in Pl. III, fig. 7, which has to be discussed hereafter, and which belongs to a case where no hypertrophy of the auricle was found, post-mortem. In the other two the rise, which he regards as auricular-systolic, commences in the situation of *k*, quite at the beginning of diastole. Therefore, although I am disposed to agree with his explanation of the curves; they are perhaps more open to the possibility of another interpretation than the tracing in the present case, in which the widening of the auricular elevation is but slight. Dr. B. Foster [1] has also published two cardiograms taken from cases of contracted mitral, the second of which was verified by post-mortem examination. The first of these shows a gradual rounded swelling commencing soon after the beginning of diastole, which might be taken to indicate the prolonged auricular contraction, although the author does not appear to adopt that interpretation. In the second case, the shape of the systolic portion of the curve, which resembles that in Pl. III, fig. 5, denotes a partial inversion towards the end of systole, and therefore no certain conclusion can be drawn from the ascent which is visible in its diastolic part. It shows, however, undulations corresponding to the presystolic thrill.

It might at first sight be supposed that in mitral stenosis the auricular hypertrophy would produce no increased elevation in the apex-tracing, but that its effect would be neutralised by the obstruction in front. It must be remembered, however, that in such a case as the present, the hypertrophy has to com-

[1] 'Clinical Medicine,' p. 316.

pensate not only for the resistance to the auricular contraction, but for the obstruction to the venous flow during the earlier part of diastole. Moreover, when the mitral constriction is considerable, the auricular contraction would drive more blood backward than forward, and in such case it would tend to impel the heart forward against the ribs. At the same time it is clear that the absence of increased elevation would not disprove auricular hypertrophy, but if the auricular elevation were distinctly seen to be of normal width, we might certainly conclude that the auricular systole was not prolonged. It is also evident that if there be no contraction of the mitral valve, the same degree of auricular hypertrophy will produce a greater effect on the tracing. An instance of this is shown in Pl. III, fig. 6. The case was one of long standing mitral regurgitation, and a systolic bruit only was heard during life. Post-mortem, the mitral valve was found extremely wide, the left auricle dilated and much hypertrophied. It will be seen that the auricular elevation in the trace is much higher, but not much wider than normal. The hypertrophy of the auricle in this case seemed to be due to a cause of an opposite kind to that which generally produces such a result, and to be the effect not of contraction of the mitral valve, but of a dilatation so great that the auricle and ventricle were almost converted into one cavity.

A cardiogram published by Dr. Mahomed, the diastolic part of which closely resembles that in Pl. II, fig. 13, was obtained from a patient in whom it was found, post-mortem, that the left auricle was much hypertrophied, but the mitral valve not contracted. Judging from this result, it may be concluded that the elevation *a*, in fig. 13, is the auricular contraction, although it is not quite so obviously so, as in Pl. III, fig. 6, and if this be the case it is much widened, as well as increased in height. The following is the account of the patient from whom the trace was obtained.

E. S. F—, æt. 12, was admitted into Clinical Ward under Dr. Fagge, on May 13th, 1874. A blowing systolic bruit was heard at the lower part of the sternum; at the usual site of a mitral murmur, the bruit was much less marked. The second sound was much accentuated to the left of the sternum, and pulsation was seen in the veins of the neck. Cardiac impulse

which, it was thought, might be due to the auricular contraction, was felt above the nipple to the left of the sternum. The bruit disappeared in a few days, but was afterwards heard occasionally at the lower part of the sternum. In this instance, therefore, there was tricuspid regurgitation, and considerable suspicion of mitral contraction, although no presystolic bruit was heard.

The next case is one in which the physical signs afforded ground for a confident diagnosis of mitral contraction. There was heard over a limited area at the apex a long rough churning bruit. This commenced immediately from the second sound, became somewhat intensified toward the end, and led almost up to the systole, judging of systole by the apex beat, but at the time when the tracing was taken seemed to be separated from it by a slight interval of silence. There was also a blowing systolic murmur in the same situation, and both murmurs were accompanied by thrill felt at the apex. Thus, in this case, murmur and thrill extended over the whole cardiac revolution, except the short interval just preceding the systolic bruit. The apex tracing is that in Pl. III, fig. 19, and shows the almost continuous thrill. It will be seen that the auricular elevation is separated from the ventricular upstroke by a wider interval than usual, corresponding to the pause between the murmurs. Moreover, this auricular elevation seems not to consist of a single small wave, but to be represented by the rapidly ascending vibratile line, which commences immediately from *k*, and reaches to the summit where the letter *a* is placed. It may be inferred that the auricular contraction began immediately after the second sound, and ceased at a slightly longer interval than usual before the ventricular systole. It could scarcely be doubted that the summit *a*, at any rate, was to be ascribed to the auricular contraction, but it might possibly be maintained that the earlier part of the vibratile ascent in the trace, and the commencement of the murmur, were due to the passive venous flow. It seems, however, unlikely that such a flow through a narrow orifice could cause so rapid an ascent and so coarse a vibration, and there was no evidence of any such increase of tension in the systemic veins as would be likely to cause a very rapid filling of the right ventricle. Moreover, the presystolic bruit was of the same quality throughout.

With the last curve may be compared that in Pl. III, fig. 15, obtained from a patient in whom a bruit was heard, almost exactly corresponding in rhythm to the one just described, for it began immediately from the second sound, became intensified toward the end, but was sometimes separated from the first sound by a short pause, and this was the case when the tracing was taken.

Sarah F—, æt. 19, was admitted into Clinical Ward, on December 31st, 1873. She had rheumatic fever when two years old, and had had five or six attacks in all. She had suffered from shortness of breath as long as she could remember. On admission the cardiac impulse was not heaving; the apex-beat was in the fifth intercostal space in the line of the nipple. At the apex was heard a long loud rumbling presystolic bruit, which generally led up to the first sound, and a thrill synchronous with this was felt over the apex, and a considerable portion of the precordial region. The second sound was heard faintly at the apex, and was reduplicated at the base. A few days later a blowing diastolic bruit after the second sound was heard at the base, and most distinctly at the left edge of the middle of the sternum. In this case it seems the most probable explanation that the whole line of ascent from *k* to *a*, coincident with the loud bruit and coarse thrill, denotes an auricular contraction commencing immediately after the second sound, but becoming more forcible at its conclusion at *a*, which is separated by a slight interval from the principal upstroke. The interpretation is, however, rendered doubtful by the existence of the bruit at the base, which may have been due to disease of the aortic valves. The pulse tracing (fig. 16) has a quality quite opposite to that usual in aortic regurgitation, and the possibility may be suggested that the diastolic murmur to the left of the sternum may have been due to the same cause as that heard at the apex, but modified in character by its situation.

There remain two more typical cases of prolonged presystolic murmur, the cardiogram obtained from the first of which is shown in Pl. III, fig. 13. Matilda A—, æt. 36, was admitted into Petersham Ward under Dr. Moxon on April 8th, 1874. She had rheumatic fever at the age of ten years, and had had two other attacks since. On her admission the heart's action

was tumultuous, and it could only be ascertained that there was a harsh grating murmur at the apex. On the 25th it was found that the bruit at the apex commenced after the second sound, sometimes running up to the first sound and sometimes ceasing before it. The second sound was reduplicated at the base. She was again admitted into Clinical Ward under Dr. Wilks, in November, 1874, and the tracing shown in fig. 13 was then obtained. At that time there was a rough, but not very loud bruit, commencing immediately after the second sound and leading up to the first sound, and this bruit was accompanied by thrill. It will be seen by the figure that there is no trace of the auricular elevation in its usual place. At the end of the principal descent is a small rise which appears to correspond to *k*, although possibly it ought rather to be identified with *g*. This is followed by a sudden rise which is continued in a tremulous line quite up to the succeeding ventricular systole, evidently, therefore, corresponding to the murmur and thrill. In this case there is no trace of imperfection in the curve due to a retraction at the latter part of the systolic period, but on the contrary the eminence *f* appears at its usual position, although the ventricular systole is somewhat short. The elevation which immediately follows *k* cannot, therefore, be explained as due to the relaxation of the ventricle, as it may be when the curve resembles those in Pl. II, fig. 4, or Pl. III, fig. 5, nor as caused by the passive venous flow, for then it would not be sudden at its commencement. It can hardly, therefore, be doubted that it represents the auricular contraction commencing almost immediately after the second sound and continuing quite up to the next ventricular systole.

The following case is a very similar one. George M—, æt. 19, was admitted into Stephen Ward under Dr. Fagge, on November 19, 1873. Fifteen months before he had been under the care of Dr. Rees, and a presystolic murmur had been heard then. On admission the apex-beat was in the fifth intercostal space, and half an inch external to the nipple. Impulse was also felt in the sixth intercostal space, as low as three inches below the nipple, and again one and a half inches below the ensiform cartilage. Pulsation was seen in the veins of the neck. There was a long, very loud presystolic bruit, of rough grinding quality, commencing immediately after the second

sound, and leading up to the first sound. At the lower part of the sternum a systolic murmur was heard. On Decemher 29th a blowing systolic murmur at the apex became also audible. In the cardiogram obtained from this patient (Pl. III, fig. 11), there is some suspicion of partial inversion towards the end of systole, on account of the rapid descent of the curve from the first summit *d*, and the low position of the eminence *f*. A sudden rise at the commencement of diastole might therefore be due simply to the recoil which is produced by the relaxation of the ventricle, when there has been retraction in the latter part of systole. An elevation of this kind, however, immediately succeeds the principal downstroke without the intervention of any other wave, as shown in Pl. II, fig. 4. In the present figure, however, we see that the main descent is followed by a slight rise, *k*, and after this is an ascent, *a*, commencing suddenly, and continued in a vibratile line, which represents the presystolic murmur and thrill, quite up to the succeeding systole. The diastolic portion in this figure so nearly resembles that in the curve last considered that the same interpretation must apply to both, and we must conclude that in this case also the ascent commencing at *a* represents the prolonged auricular contraction.

All the cardiograms of mitral stenosis hitherto considered seem to me to require for their explanation the supposition of abnormally early commencement of the auricular systole. But a totally different causation of murmurs in such cases is mechanically possible, namely, from the simple influx of the blood through a rough narrow orifice during diastole, rendered more forcible by the high pressure in the pulmonary veins. Although the murmur of mitral stenosis has often been spoken of as diastolic, especially by the earliest writers, at the time when the systole of the auricle was supposed to follow immediately upon that of the ventricle, and to be the cause of the second sound of the heart, yet some authors who have most forcibly insisted that the murmur is auricular-systolic, seem scarcely to recognize a diastolic, as distinguished from a presystolic bruit, to be a possible occurrence in such cases. But that it may occur, at least theoretically, is proved by Marey's experiment. In the schema of the circulation by means of which he reproduced the various sounds and murmurs of the heart, the auricle of

which was not contractile, he introduced a plug perforated by a hole between the auricle and ventricle, and then found that a diastolic murmur was produced, but only when the auricular pressure exceeded a certain point. Hence, he concludes that the murmur of mitral contraction may be either diastolic or auricular-systolic according to circumstances.[1]

In this experiment the diastolic murmur must have extended through the whole interval up to the next systole, or until the ventricular pressure had risen nearly to the level of that in the auricle. If, however, there be a roughened but not very closely contracted mitral orifice through which regurgitation takes place, the regurgitant flow might suddenly raise the venous pressure to such a point that the first part of the direct flow would cause a murmur, while the latter part, after the pressure had fallen somewhat in the auricle and risen in the ventricle, would take place silently. I think that such a murmur ought to be called diastolic and not post-diastolic or post-systolic, even though the first part of it be lost in the second sound, for its rhythm is precisely the same as that of an aortic regurgitant murmur, when heard in conjunction with a second sound. I am here adopting that sense of diastolic as applied to murmurs which though usual is not strictly accurate; for in common use it denotes not the whole, but only the earlier part of diastole, during which the murmur of aortic regurgitation commonly occurs, commencing from the moment at which the second sound should be heard, but extending over a wider interval than that occupied by the normal second sound. In the stricter sense of the word diastolic, according to which it would include the whole diastole of the ventricles, the pre-systolic is only a special variety of diastolic murmur, and thus by those authors who observed it before the introduction of the term presystolic, it is described simply as diastolic. I am disposed to object altogether to the term post-diastolic frequently applied to direct mitral murmurs; for, speaking accurately, a murmur which is post-diastolic can be nothing else than systolic, since any cavity of the heart must be either in systole or diastole.

There is another case in which it is possible that, even though no perceptible regurgitation took place, there might be

[1] 'Physiologie Medicale de la Circulation du Sang,' Paris, 1863, p. 521.

a diastolic murmur at the apex due to the venous flow through the narrow orifice into the ventricle, and separated by an interval of silence from the succeeding first sound; for if the heart's action were very slow, and systole did not begin until some time after the ventricle had been filled or nearly filled, then the murmur might cease as soon as the pressure in the ventricle had become nearly equalised with that in the auricle.

It has seemed to me possible that some observers, not admitting theoretically that a blowing diastolic murmur, separated by a pause from the succeeding first sound, can be the result of mitral contraction, may perhaps sometimes, when they meet with such cases, mistake them for aortic regurgitation, especially since murmurs of this kind are often audible near the base to the left of the sternum.

I have met with several instances in which the cardiogram has appeared to favour the interpretation which I have suggested of the causation of the murmur. I will here give brief notes of one of these which seems to me the clearest.

Eliza P—, æt. 16, was admitted into Addison Ward under Dr. Habershon, on October 27th, 1874. She had acute rheumatism when six years old. In July, 1870, she had been in Clinical Ward under Dr. Fagge. The report of the case at that time was headed "Morbus Cordis, Aortic. Dropsy. Enlarged Liver." A diastolic bruit was then heard at the base, and a systolic bruit not very distinct anywhere. When she left the hospital in September, there was only a to-and-fro murmur at the apex. She was again in the hospital under Dr. Pavy in November, 1873. There was then a systolic bruit at the apex, and the second sound was prolonged into a murmur. At the base the second sound was reduplicated. The pulse was small and feeble. Mitral contraction was at this time diagnosed. On December 1st there was heard a third short murmur at the apex between the diastolic murmur and the first sound, and separated from the former by a short interval of silence.

On her admission in October, 1874, the apex beat was in the sixth intercostal space, two inches below and two inches external to the nipple. A characteristic but not prolonged presystolic bruit was heard at the apex; no systolic murmur was audible. The second sound was reduplicated. On November 2nd the sounds at the apex had entirely altered their character.

There was a short systolic murmur followed by a longer blowing diastolic murmur, and then a long pause. When the heart beat quicker the diastolic murmur ran up to the first sound, and covered the pause. The murmur had occasionally a rumbling quality. On December 13th there was heard at the apex a loud first sound accompanied by a short murmur. The second sound appeared reduplicated, and a long blowing diastolic murmur was continued from its second part. This did not reach the first sound, but left a considerable pause between them. The murmurs at this time were not audible at the base or down the sternum, and the character of the pulse, both as judged by the finger and in the sphygmographic tracing, was entirely that of mitral and not of aortic disease. It may be inferred therefore that the murmur was not due to aortic regurgitation.

The tracing in Pl. III, fig. 12, was obtained from this patient at a time when only the diastolic and no presystolic bruit was audible. No auricular rise is visible, and the systolic elevation is narrower than usual at the summit, indicating a somewhat short contraction, yet it does not commence by a single high-pointed summit, as is the case when dilatation preponderates over hypertrophy. It would therefore correspond well with a comparatively small ventricle, and its general shape is in this respect similar to that in fig. 13. It has been supposed by Dr. Fagge that the rhythm of the auricle may be so far inverted that its contraction may habitually follow instead of preceding that of the ventricle.[1] It might be suggested, therefore, that *k* in the figure indicates the auricular beat and the murmur. This cannot be, however, because the elevation *k* is much too narrow to correspond to the duration of the bruit, and it seems to be the same as *k* in the normal trace, only somewhat greater than usual, probably from increased pressure in the pulmonary arteries. The quality as well as the rhythm of the bruit in this case seems to distinguish it from that due to the auricular systole, and it will be noted in the history that on one occasion both murmurs seem to have been audible at the same time, but separated by a pause. It may be compared, therefore, with the case reported by Dr. Barclay,[2] in which, while the heart was acting very slowly, there was a short soft murmur at the apex,

[1] 'Guy's Hospital Reports,' 1871, p. 334 and p. 337.

[2] 'Lancet,' March 2nd, 1872.

beginning almost immediately after the second sound, then an interval of complete silence, and lastly a longer harsh grating murmur leading up to the first sound. In both cases it seems that the two murmurs must be attributed to two different causes, although not necessarily, as Dr. Barclay maintains, to two different directions of blood-current.

The next case is one to the obscurity of which I had my attention directed, being at the time house-physician, from the fact that the patient passed successively under the care of three physicians, of whom the first diagnosed mitral contraction, the second aortic regurgitation, while at her last admission there was no evidence for a while of anything except mitral regurgitation.

Sarah N—, æt. 50, was admitted into Addison Ward, under Dr. Habershon, on April 22nd, 1873, with left hemiplegia, apparently the result of embolism. She had rheumatic fever at the age of twenty, and again at the age of thirty-one, and ever since the latter illness she had had cardiac symptoms. A year before she had dropsy of the abdomen and legs. She had been in the hospital two months before, and a systolic bruit at the apex had then been heard. On her admission the apex beat was in the fifth intercostal space, an inch internal to the nipple; the impulse was not heaving. Under the influence of digitalis the heart's action became slow but irregular. There was then a long blowing diastolic murmur audible at the base, especially to the left of the sternum, but also audible and somewhat louder at the apex. The second sound was heard both at apex and base. The murmur always commenced from the second sound, and was sometimes separated by a very long interval of silence from the succeeding first sound. Very rarely there was heard a faint systolic bruit in the axilla.

The sphygmographic tracing of the pulse showed a character very opposite to that of aortic disease. It had a high tension, requiring a pressure of four ounces, a small amplitude, a prolonged tidal wave, and no sign of collapse in the diastolic portion. The curve was similar to that in Pl. III, fig. 16, except that the amplitude was somewhat greater and the rate slower. It is a form of pulse which has nothing in itself characteristic of any cardiac affection, but it is rather common in mitral stenosis, at a stage at which the heart is acting

vigorously, and is very rare in any other form of heart disease. In mitral regurgitation the tension may become pretty high and the tidal wave considerable under the action of digitalis, but then at the same time the amplitude becomes great. It seems as if sometimes in mitral stenosis the arteries, accustomed to receive a pulse wave of small amplitude but propelled by considerable force, assume a habitual state of contraction.

This patient was quite unable to hold her breath, but the cardiac tracing, including the effect of a single respiration, is shown in Pl. III, fig. 14. No auricular rise can be distinguished, the elevation, *k*, is less pointed and more prolonged than usual, but there is no rapid ascent in the diastolic portion, such as occurs generally in aortic regurgitation or when the mitral orifice is wide and the valve incompetent. The development of the eminence, *f*, like the tidal wave in the pulse, shows a strong action of the ventricle.

The same patient was again admitted into Clinical Ward, under Dr. Moxon, on February 21st, 1874, with greatly increased cardiac distress. There was then a systolic murmur at the apex, and strong pulsation in the veins of the neck.

On the 26th the systolic murmur was no longer heard, but a diastolic or presystolic murmur at the apex.

On the 28th the diastolic murmur was heard over the sternum about the level of the nipple.

On March 9th the diastolic or presystolic bruit was best heard to the left of the sternum at the level of the nipple, and was lost at the base and in the axilla.

On April 27th the patient had much improved; there was heard only a slight systolic murmur at the apex, and the second sound was reduplicated. Mitral contraction was diagnosed on this occasion, and the case seems to me to be one in which a diastolic murmur due to this cause resembled at times that which is produced by aortic regurgitation. The only alternative is to suppose that the diastolic murmur was caused by aortic regurgitation too slight in degree to have any effect on the pulse or system generally. But I believe that no one who examined the case was led to infer the existence of more than one valvular lesion, although at one time opinions differed as to which valve was affected.

In these two cases the evidence of the cardiograph is simply

negative as to increased auricular contraction. In the following instance, however, the auricular rise seems to be shown in its natural position, and neither increased in magnitude nor prolonged (Pl. II, fig. 7).

John F—, æt. 25, was admitted into Stephen Ward, under Dr. Wilks, in October, 1874. On his admission a short rough murmur was heard at the apex, which was considered to be presystolic. This soon disappeared, and was inaudible when the cardiogram was taken. There was then a short soft diastolic murmur, heard only at the apex. It should be stated, however, that this murmur was very faint, and that there was some difference of opinion as to its existence.

It is to be observed that for the causation of such a diastolic murmur followed by a pause, while the heart's action is of moderate rapidity, it is necessary that there should be sufficient regurgitation to have a manifest effect on the pressure in the pulmonary veins, and, therefore, that the contraction of the mitral orifice should not be extremely close. Accordingly in such cases, if a presystolic bruit distinct from the diastolic is heard, it is generally short, often quickly disappears, and usually a systolic bruit can also be heard at times. When the diastolic murmur is short it is also soft, and resembles in quality that of aortic regurgitation, because the venous pressure is only briefly and slightly raised above the level necessary to produce a sound. As, however, it becomes longer it becomes rougher and louder, and is assimilated in quality to the presystolic murmur, especially when the intervening pause is obliterated, because then a more considerable excess of venous pressure is indicated, and the sound is increased as the force producing it becomes greater. A diastolic murmur distinct from the presystolic, and caused by the venous flow in mitral contraction, has been recognised by Marey, and also by Dr. Balfour, Dr. Barclay, and other writers; but I am not aware that any one has discussed the mechanical conditions which are implied when it is blowing in quality and succeeded by a pause.

I will add very brief notes of four other cases in which it appeared to me that a direct mitral murmur was to be attributed with greater probability to the venous flow than to the auricular systole. In all of them a systolic murmur at the apex was heard in addition.

Hannah G—, æt. 47, was admitted into Bright Ward, under Dr. Moxon, on December 19th, 1874. She had suffered from occasional shortness of breath more than ten years before. At the age of forty-two she had acute rheumatism, and again a year later. On the latter occasion some hæmoptysis occurred. On admission there was strong pulsation at the left side of the ensiform cartilage; a thrill during diastole was perceptible one inch below and one inch internal to the nipple, and sometimes faint impulse was seen in the normal position of the apex. Pulsation was visible in the jugular veins. There was a short, blowing, systolic murmur at the apex, which was audible at the back. The second sound was reduplicated at the base. Generally there was another murmur also audible at the apex, the quality of which was usually soft and purring, but occasionally became somewhat rumbling. This murmur began at the commencement of diastole, and almost always ran up to the succeeding systole, but became, if anything, fainter rather than more intense towards its conclusion. Once or twice, when the action of the heart had become slow under the action of digitalis, the murmur was separated by a very distinct interval of silence from the succeeding systole.

Two tracings obtained from this patient are shown in Pl. II, fig. 1, A and B. Tracing A was taken at the apex, and tracing B over the epigastrium; the latter is amplified in vertical height nearly twice as much as the former. The epigastric trace, therefore, shows a systolic eminence very much higher and distinctly wider than that seen in the apex curve. This difference of width seems to denote that the systole of the right ventricle was more prolonged than that of the left, a fact indicated also by the reduplicated second sound. In the apex tracing A the auricular elevation *a* is seen in its normal position and scarcely prolonged, and in the earlier part of diastole there seems to be an indication of the thrill which was felt in this position. The cardiographic evidence, therefore, tends to show that the murmur could not be due to the auricular systole in this case.

Jessie H—, æt. 11, came under my care as out-patient at the Hospital for Sick Children, Great Ormond Street, on April 4th, 1874. She had never had rheumatism, and the symptoms of illness had only attracted attention three weeks before.

The apex beat was found to be in the sixth intercostal space, and one inch external to the line of the nipple. It was violent in character, and strong pulsation was felt also in the epigastrium. There was a systolic thrill and rough murmur at the apex, followed by a distinct musical murmur during the latter part of systole. The same double quality in the murmur could be distinguished posteriorly. The second sound was reduplicated. On September 22nd there was a diastolic or presystolic murmur accompanied by thrill at the apex, and a systolic murmur heard at the apex and at the back. On January 19th, 1875, there was a thrill with systole at the apex, and a more distinct one throughout the whole of diastole. In this situation a systolic murmur was audible, and also another murmur, not harsh in quality, which extended through the whole of diastole and ran up to the following systole, but seemed fainter towards its conclusion than at its commencement. The heart's action was always very rapid. The apex tracing obtained from this patient showed the thrill which extended over the whole revolution of the heart. No increased auricular eminence was visible, but on some occasions the auricular systole seemed to be shown in its normal position and not prolonged, while on others it could not be distinguished.

The next case is that of Henry H—, æt. 24. He had acute rheumatism at the age of ten, and chorea at that of thirteen. Since that time he had had acute rheumatism four times. In September, 1871, he was in Clinical Ward, under Dr. Fagge. The apex beat was then in the fifth intercostal space, and a quarter of an inch internal to the nipple. There was a harsh bruit at the apex, commencing immediately after the second sound, and running up to and blending with the first sound. On February 10th, 1875, he was again admitted into Philip Ward, under Dr. Moxon. On admission the apex beat was in the sixth intercostal space, and one inch external to the nipple line. Under the action of digitalis the rate of the pulse was reduced to 40 in the minute. A systolic murmur was then heard at the apex, and a second murmur, not very loud or harsh in quality, which began quite at the commencement of diastole, and was separated from the succeeding systole by a considerable interval of silence, thus being distinctly diastolic

in rhythm. Cardiographic tracings were taken at this time, and the curves obtained were found to be inverted at all points within the considerable area over which a wavy impulse could be seen to spread, except precisely at the apex. The apex trace was positive in character, but the auricular elevation could not be distinguished in its usual position. At the commencement of diastole there was a considerable rounded swelling in the apex of the elevation *k*. Therefore in this case the cardiogram would be consistent with the supposition that the auricular systole formed the conclusion, instead of the commencement, of each revolution of the heart. Since, however, retraction during systole occurred over so wide an area, it is more probable that the elevation at the beginning of diastole was due to a recoil of the chest wall at the relaxation of the ventricles.

The last case of this kind is one in which the direct mitral murmur was heard only for a very short time. Mary B—, æt. 38, was admitted into Mary Ward under Dr. Wilks on December 30th, 1874. She had first suffered from short breath and palpitation of the heart four years before, and had never had rheumatism. On admission the apex beat was in its normal position, the impulse was diffused. The heart's action was very rapid and irregular. A soft systolic murmur at the apex, not accompanied by thrill, was heard occasionally. On January 5th a short, harsh bruit at the apex was heard immediately after the accentuated second sound, ceasing before the first sound. On January 13th the rate of the pulse had fallen to 50 in the minute, but it was still very irregular. There was then a somewhat soft murmur at the apex, diastolic in rhythm, and often separated by a considerable interval from the following systole. A faint systolic murmur was heard occasionally. The second sound was reduplicated. A tracing from the apex of the heart, taken at this time, showed no sign of the auricular elevation. In a short time both murmurs disappeared, but the rhythm of the heart always continued very irregular.

Out of the cases collected by Dr. Fagge in his paper on "Mitral Contraction" in the 'Guy's Hospital Reports' for 1871, there are two, namely, Case 67 and Case 68, in which the pulse was at times very slow, and the murmur of unusual

character, having a rhythm which is described as being diastolic rather than presystolic, followed by an interval of silence. Case 67 has some resemblance to the two described above and depicted in Pl. III, fig. 12 and fig. 14, for there was a long wavy murmur, diastolic in rhythm, which was loudest to the left of the sternum at the level of the second costal cartilage, but was not carried along the sternum downwards; and there was also a systolic murmur which appeared loudest between the fourth and fifth costal cartilages. If it be admitted that the venous flow through a contracted mitral orifice may produce a diastolic murmur followed by a pause, and often heard loudly near the base to the left of the sternum, then it would seem that such an explanation may be adopted with some probability for Case 67, and possibly also for Case 68. The only other alternative, assuming that mitral contraction existed, is that which is accepted by Dr. Fagge himself as the explanation in both cases, namely, that each revolution of the heart *ended,* instead of commencing, with a powerful contraction of the auricles.

In the patient whose cardiogram is given in Pl. III, fig. 9, there was a rough murmur at the apex, commencing immediately from the second sound, but becoming very loud and harsh towards its conclusion and leading up to the first sound. The trace shows an auricular elevation greatly increased in magnitude, but only moderately in duration, and the first and third of the revolutions seem to show indications of a thrill continued through the pause. This figure, considered in conjunction with the rhythm of the murmur, seems to me a positive confirmation of the view expressed by Dr. Wilks[1] as to the probable causation of one class of cases of presystolic bruit, namely, that the murmur extends over both the pause and the auricular contraction, the first part of it being due to the venous flow through the narrow orifice.

The following case furnishes evidence of a similar character. Emma C—, æt. 24, was admitted into Mary Ward, under Dr. Wilks, on December 29th, 1874, with left hemiplegia, apparently the result of embolism. She had never had rheumatism, but for three years had been subject to headache and fits of giddiness. On admission the apex beat was in the fifth

[1] 'Guy's Hospital Reports,' 1871.

intercostal space, in the line of the nipple. Very strong pulsation was also seen in the epigastrium. There was a long, somewhat rumbling murmur at the apex, beginning at the commencement of diastole, and becoming much intensified towards its conclusion, which led up to the first sound. A thrill accompanied the systole, and appeared to commence a little before it. On January 5th the murmur had entirely changed. There was a short, distinct, presystolic bruit at the apex, followed by a faint systolic bruit. In the epigastrium a loud systolic murmur was heard, and strong pulsation was seen in the jugular veins. At this time the apex tracing shown in Pl. I, fig, 9, was obtained. It will be seen that the auricular elevation is much increased in height, but not greatly prolonged. At the same time a tracing was obtained from the jugular vein, which showed an elevation due to the auricular systole in the ascending part of the curve, as well as a dicrotic wave in the descent. In technical terms the curve was at the same time anadicrotic and katadicrotic. A few days later the sounds had changed a second time, and the prolonged murmur was again heard through the whole interval of rest, intensified, as before, near its conclusion. The apex tracing still retained the same shape as that shown in fig. 9, and thus proved that the latter part of the bruit only was due to the auricular systole.

I pass on to a case the interpretation of which is somewhat ambiguous. Martha L—, æt. 27, was admitted into Petersham Ward, under Dr. Moxon, on October 22nd, 1873. She had suffered from palpitation of the heart for four months, and had never had rheumatism. On her admission there was a soft blowing systolic murmur at the apex, and strong pulsation in the veins of the neck. After a few days there was heard in addition a murmur at the apex commencing immediately from the second sound and continuing up to the systole. It was rough in quality, but not very loud or grating, and not intensified towards its conclusion. It was considered by Dr. Moxon to be undoubtedly presystolic. While she remained in the hospital sometimes the systolic and sometimes the presystolic murmur was the loudest, but the action of the heart was always exceedingly rapid, so that it was difficult to ascertain whether the murmur belonged in rhythm to the preceding or succeeding

first sound. This patient was again admitted into Clinical Ward, under Dr. Taylor, on May 29th, 1874, and a systolic murmur was then heard at the apex. She died on June 22nd. Post mortem, the heart was found to weigh nineteen ounces; the right ventricle was greatly hypertrophied but not dilated. The cavity of the left auricle was of full size, its lining membrane thick and white; its muscular wall was not hypertrophied. The mitral orifice was contracted, and only admitted two finger tips; its edges were very thick.

The cardiogram obtained in November, 1873, is shown in Pl. III, fig. 7. Its chief characteristic is the rapid ascent during the diastolic period, and it resembles in this respect one of the cardiograms of mitral stenosis published by Dr. Mahomed. This is not unfrequently found in mitral regurgitation when the tension in the pulmonary veins is very great, as shown in some of the tracings of Dr. B. Foster;[1] but in this instance I was at the time disposed to attribute it to the auricular contraction, supposing that the ventricle could not be filled so rapidly by the venous flow through a narrow orifice. Since, however, no hypertrophy of the auricle was found, it would seem rather that the murmur was due to the venous influx through the narrow and indurated orifice. The rapid ascent in the diastolic part of the curve may be explained as the combined effect of two causes; first, the extreme tension in the pulmonary veins, which so far preponderated over the obstruction caused by a contraction only moderate in degree, that the left ventricle was filled quickly; and, secondly, the elevated pressure in the systemic veins, enhanced by the tricuspid regurgitation, which would produce a still more rapid repletion of the right ventricle. It is possible, nevertheless, that although the auricle was not considered to be hypertrophied when the post-mortem examination was made, yet it contracted slowly; and that the long gentle swelling *a* in the figure is due to its effect being added to that of the venous flow.

There are three more cardiograms to be referred to, obtained from patients as to whom a more or less confident diagnosis of mitral contraction was made. Pl. III, fig. 4, is the apex-tracing of a boy, aged 8, in whom there was a loud

[1] 'Clinical Medicine,' by Dr. Balthazar Foster.

systolic murmur at the apex, and the action of the heart was very powerful. A presystolic bruit had previously been heard, but at the time when the tracing was taken, there was only a very faint rumbling sound just preceding the systole. The auricular elevation in the figure is not increased in proportion to that of the ventricle, and seems to be about normal in width and position.

The following is the account of the next case. John R—, æt. 30, was admitted into Stephen Ward under Dr. Wilks, on October 7th, 1874. Six years ago he was discharged from the army on account of palpitation of the heart. He had had rheumatism in the knees, but never acutely. On admission the apex-beat was in a line with the nipple and two inches below it. There was a systolic bruit at the apex accompanied by thrill. On the 13th there was heard at the apex a prolonged systolic murmur. Towards the axilla this disappeared, and was replaced by a thick first sound, and two short rough, post-systolic sounds. There were intervals of silence between the two, and between the last and the systole. On the 19th the systolic murmur had disappeared, and there was a thick rough first sound at the apex, followed by a reduplicated second sound. This was nearly the state of things when the apex tracing in Pl. I, fig. 16, was taken, but the second part of the reduplicated second sound then appeared to me to be prolonged into a faint soft murmur. All the waves in the curve appear to be inverted, and the double depression *k* probably indicates the want of coincidence of the reflux currents in the aorta and pulmonary arteries, although not synchronous with the reduplicated second sound. There seems to be an indication at *a* of the auricular beat, in its normal position and not prolonged, but this is perhaps scarcely distinct enough to be relied upon, considering that the curve is inverted.

The last case is one in which it was considered probable that mitral contraction existed from the history and general condition of the patient, although throughout a space of nine months a systolic murmur only was audible.

Charles H—, æt. 20, was admitted into Philip Ward, under Dr. Moxon, on May 6th, 1874. He had never had rheumatism, but had suffered from shortness of breath for five years. The apex-beat was seen and felt in the fifth and sixth

intercostal spaces in the line of the nipple. There was a loud systolic murmur at the apex, accompanied by thrill, and the murmur was audible in the axilla and at the back. This patient was again admitted on August 10th, and the cardiograms in Pl. II, figs. 11 and 12, were obtained in November. Fig. 12 was taken at the centre of impulse in the fifth intercostal space, fig. 11 in the fourth space. The shape of the systolic eminence in both cases shows a preponderance of hypertrophy over dilatation, but it will be seen that the primary summit *d* has a much less proportionate magnitude in fig. 11 than in the apex trace. There were at this time a systolic murmur and thrill in the epigastrium as well as at the apex, some pulsation was seen in the veins of the neck, and expansive pulsation was felt over the whole surface of the liver, which extended as low as the level of the umbilicus. The chief feature in the cardiac curves is the sudden rise at the commencement of diastole, but this can hardly be attributed to the auricular systole, and is rather the combined effect of a powerful recoil in the pulmonary arteries, and the sudden filling of either right or left ventricle. In the curve obtained from the fourth intercostal space (fig. 11), the auricular elevation *a* seems to be visible in its normal position and not prolonged.

On January 21st an entirely new murmur had made its appearance in this patient. This was a short musical diastolic murmur, having exactly the same rhythm as that produced by aortic regurgitation. This was heard most distinctly at the apex, but was audible also near the base. Occasionally it disappeared when the patient was in an upright position. The pulse was very feeble and so dicrotic that the dicrotism could easily be detected by the finger. It was very unlikely, therefore, that the bruit could be due to incompetence of the aortic valves. Its musical quality also appeared to show that it could not have an exocardial origin. The apex tracing taken at this time, which was obtained too late to be included in the plates, had a very interesting character. At the commencement of the diastolic portion appeared first three rapid vibrations which seemed to correspond to the short musical bruit, then a pause, and, lastly, the auricular elevation, in its normal position, but apparently somewhat higher and wider than usual.

It remains only to refer to the theory which Dr. Barclay has

maintained as to the causation of the presystolic murmur in mitral contraction.[1]

He considers that the systole of the heart commences in a vermicular manner, and that the impact against the ribs does not occur until after the contraction has proceeded to some extent. He believes also that normally the systole of the auricle overlies in part that of the ventricle, and hence, against the view attributing the presystolic murmur to the contraction of the hypertrophied auricle, he argues that the force of the ventricle would overcome that of the auricle, and thus the cnrrent, even during the auricular contraction, would be retrograde. It is proved, however, by Marey's simultaneous tracings in Pl. I, figs. 4 and 5, that normally the systole of the auricle has ceased when that of the ventricle commences. The same conclusion may indeed be drawn from the typical healthy cardiogram in Pl. I, fig. 1, for the auricular elevation *a* does not lead gradually up to the ventricular systole, but is separated from the main upstroke by a slight fall. I have already referred to the demonstration afforded by Marey's tracings that the first part of the apex beat is not due to any impact against the ribs caused by the locomotion of the heart, but to the initial hardening of the ventricles. The actual commencement of the apex beat therefore coincides with almost the beginning of the ventricular systole, and precedes by a very short interval the first sound of the heart.

Moreover, it is evident on watching the exposed heart of an animal that the ventricular systole is not any slow vermicular movement, but is extremely rapid. The same thing is proved by the suddenness of the main upstroke in cardiographic tracings and in those indicating the intra-ventricular pressure. According to Marey's figures, the interval occupied in attaining the first maximum of pressure, which necessarily occurs after the closure of the auriculo-ventricular valves, is at most $\frac{3}{20}$ second (Pl. I, fig. 6), and the smallest such interval is $\frac{1}{20}$ second (fig. 4). On reducing to a horizontal line of reference the various points in the apex-tracings, and ascertaining the corresponding intervals of time, it is found that the time occupied by the main ascent varies from $\frac{1}{12}$ second in the healthy heart to about $\frac{1}{6}$ second in those that are most hypertrophied. In

[1] "Remarks on Presystolic Cardiac Murmur," 'Lancet,' 1871.

Pl. I, fig. 14, the interval is about $\frac{1}{8}$ second. From a comparison with Marey's tracings we learn that the commencement of the upstroke coincides with the first rise of intra-ventricular pressure, and its summit denotes the complete hardening of the ventricle, while the auriculo-ventricular valves seem generally to close when about half or two thirds of the ascent has been accomplished.

It might at first sight be supposed that the walls of the ventricles would not meet with sufficient resistance to cause them to harden until after the closure of the valves, and therefore that this closure would precede the commencement of the systolic upstroke. It is to be remembered, however, that to so rapid a contraction as that of the ventricles, the blood would offer some considerable resistance from the simple effect of its inertia, even though its outflow were not hindered by any valves or by any high pressure in front.

Dr. Barclay's own view is that in mitral stenosis the funnel-shaped valve is only gradually closed by the pressure of blood, and that thus the first sound occurs at a later part than usual of the systole. The presystolic murmur is therefore, he considers, really caused by free regurgitation, and the so-called systolic murmur which sometimes follows it is due to the fact that some small residuum of regurgitation continues after the valve has almost completely closed. In examining after death the small rigid ring, sometimes hardened by calcareous deposit—into which the mitral valve is not unfrequently converted in instances in which a characteristic presystolic murmur has been heard—it has seemed to me impossible that such a valve could produce any valvular click at all. And therefore, although the short, sharp first sound often heard in such cases may partly be due to the closure of the tricuspid valve, yet it would seem rather to constitute a proof, as pointed out by Dr. Fagge, that the sudden tension of the ventricular wall has much to do with the normal first sound of the heart. If Dr. Barclay's view were correct, the closure of the tricuspid valve would occur much earlier than that of the mitral, and therefore we should expect to find the first sound always reduplicated when a presystolic murmur is heard. Perhaps, however, the most crucial test of its soundness is a comparison of the first sound of the heart with the carotid pulse. If a long, grinding presystolic murmur,

such as that depicted in Pl. III, fig. 11 or fig. 13, were due to the ventricular systole, the carotid pulse ought distinctly to precede the first sound of the heart. This is never found to be the case, but, on the contrary, it has its usual relation to the first sound, or is even sometimes a little delayed on account of the lowered arterial pressure and the consequent slowness of transmission.

Finally, it seems to me that Dr. Barclay's theory is absolutely contradicted by almost all the cardiograms of mitral stenosis. The elevation which depicts the thrill and the murmur is in most cases followed by a ventricular upstroke quite as sudden and distinct as usual (*vide* Pl. I, fig. 9; Pl. III. figs. 9, 10, 13, 15, 19), and the systole, measured from that upstroke, is not shorter than might be expected when the ventricle is small, and in many cases its length is not at all diminished in proportion to the pulse rate (*vide* Pl. III, figs. 9, 10, 15). It is when the presystolic murmur is short, as in fig. 9 and fig. 10, that the distinct and separate commencement of auricular and ventricular systole is most plainly manifest, and it is in these very cases that the shape of the curve most distinctly shows the murmur to be auricular-systolic. There are only two of the tracings, namely, fig. 7 and fig. 11, which present in any degree that feature which, according to Dr. Barclay's view, should be present in every case, namely, a progressive ascent, synchronous with the murmur, and leading up gradually to the principal summit, and even these do not by any means require such a theory for their interpretation. It need scarcely be said that his explanation would fail altogether to account for the instances, not uncommonly met with, in which a rough and characteristic presystolic murmur is separated by a short interval of silence from the succeeding first sound. To this class of cases belong those to which Dr. Fagge has drawn attention, in which it is very easy to mistake the presystolic for a systolic murmur, and the short first sound for the second sound of the heart, a mistake which there is reason to think that some observers have habitually made. In Pl. III, fig. 15 and fig. 19, such an interval between the murmur and the ventricular contraction is actually depicted in the trace, and it seems scarcely possible to doubt, from the appearance of the curve, that it really intervenes between the auricular and the ventricular systole.

As an objection to the modern view, Dr. Barclay strongly urges that the auricle, not being protected by any valve behind, would not, even when greatly hypertrophied, have force sufficient to cause a murmur so loud and harsh as the presystolic bruit often is. It is to be remembered, however, that the force of the auricle is added to that of a pressure in the pulmonary veins, which is sometimes extreme, and which there is reason to believe in some cases, such as those represented in Pl. III, fig. 7 and fig. 9, produces a rough murmur even without the aid of the auricular contraction. Moreover, the quality of the murmur probably depends not so much upon its intrinsic loudness as upon the spot at which it is produced. It has often been said that cardiac murmurs are conducted best in the direction of the stream; but in respect of actual conduction the direction of the current could make no apparent difference, since the velocity of the blood would be inappreciable as compared with the velocity of conduction of sound in liquid, about two thousand feet in a second. Thus, even in air the effect of wind upon sound is not due directly to the velocity of the air, but to a change in the shape of the expanding sound wave caused by the different velocities of the wind at different heights above the ground, and nothing corresponding to this can occur in the blood.

The real explanation of the facts observed in relation to cardiac murmurs is that the sound is produced by the eddies and vibrations of the fluid, and these occur chiefly beyond the spot at which there is an obstruction or projection in the stream, or at which it enters through a limited opening into a wider space. Applying this principle to the direct murmur of mitral obstruction, it is evident that the eddies will be directed towards the spot where the convexity of the ventricle comes into closest contact with the chest wall. The sound, therefore, is produced almost immediately beneath the surface, at the point where the apex-beat is felt; and that its loudness is due to this circumstance may be inferred from the fact that it is often audible over only a limited area, although loud and harsh within that area, and that it is generally inaudible at the back, where it would be distinct if it were due to free regurgitation. The only direct positive evidence that Dr. Barclay has adduced in favour of his view is the observation that in

some cases sensible movement can be felt at the apex, coincident in time with the presystolic murmur and preceding the first souud of the heart. In looking at such curves as those in Pl. III, figs. 9, 10, 11, and 13, in which the murmur and thrill are represented by so considerable an elevation preceding the ventricular upstroke, it will readily be believed that this ascent may be perceptible to the finger, although not due to the ventricular contraction. In the case from which fig. 10 was obtained, where the first elevation is perhaps more manifestly auricular-systolic than in any other, it could distinctly be felt as a slight beat, accompanied by thrill, just preceding the main impulse.

It is evident that in cases of mitral stenosis there is no reason why the contraction of the ventricle should be more gradual or prolonged than normal, but rather the contrary, since it is generally small and even thin, and receives but a scanty supply of blood. When, however, the ventricle is much hypertrophied, as especially in cases of aortic, but also in those of mitral regurgitation, its contraction appears to commence rather more deliberately. A measurement of the traces shows that under these circumstances the time occupied in reaching the first summit, which denotes the complete hardening of the ventricle, may be nearly double the corresponding interval in the healthy curve, that is to say, $\frac{1}{6}$ instead of $\frac{1}{12}$ second. The question may therefore arise whether the muscular murmur produced by the more gradually commencing contraction of the hypertrophied cardiac walls, before the closure of the auriculo-ventricular valves, may not in some cases become audible, and resemble a very short and faint presystolic bruit. Dr. Fagge has expressed his belief that no single instance has hitherto been recorded in which a presystolic murmur has existed during life, and in which the mitral orifice has not been found after death to be very decidedly narrower than usual.[1] My own belief is entirely in accordance with this view, so far as regards a manifest presystolic murmur of characteristic rhythm and quality. But, both as student and as house physician, I received the impression that in many cases in which a systolic murmur at the apex was heard, and the heart was much hypertrophied, a more or less confident suspicion has been expressed that the commence-

[1] 'Guy's Hospital Reports,' 1871, p. 317.

ment of the murmur was presystolic, and that in a considerable proportion of such cases no contraction of the mitral orifice has been found after death. This point, however, cannot be exactly verified by statistics, because such suspicions are not generally recorded in reports.

It has seemed to me possible that a mistake may sometimes arise in the way which I have suggested, but there is another mode in which error might occur with regard to the rhythm of a bruit, if the moment of systole be determined not from the carotid pulse, but from the cardiac impulse. In looking at such cardiograms as Pl. II, figs. 5, 12, 13, and Pl. III, fig. 3, in which the eminence *f* near the end of systole rises much higher than the primary summit *d*, it is evident that if we estimate the impulse, as we naturally should do, by its maximum rather than by its commencement, the moment when the apex-beat is felt will correspond in these cases to a much later part of the systole than usual. In this way a murmur really systolic might be supposed to be presystolic.

It should be mentioned that the interpretation of the elevation *a*, as being due to the auricular systole, which has been generally accepted on the evidence of Marey's experiments, and which, in the present paper, has been assumed as undoubted, has been disputed by Fick,[1] who does not, however, give any other explanation for it. His opinion is based on a series of experiments resembling those of Marey, in which sounds were introduced into the aorta and into the several cavities of the heart of a dog. The curves were registered, not by Marey's cardiograph, but by the spring manometer invented by himself. He found no sign of elevation of pressure, even in the auricle itself, during its contraction, and therefore argues that no indication of the auricular systole could appear in the apex trace. This negative result cannot be accepted as invalidating the positive evidence obtained by Marey, and it is contradicted by the venous tracing, which may be obtained in some cases of tricuspid regurgitation. The effect of the auricular systole is then seen as a wave in the ascending curve, and its position and duration, as determined in this way, agrees pretty nearly with those which are inferred from the wave seen in the apex

[1] 'Arbeiten aus dem Physiologischen Laboratorium der Würzburger Hochschule,' 1873.

trace, or in Marey's curves of auricular and ventricular pressure. This "anadicrotic" wave in the venous pulsation is often distinctly visible to the eye in persons having no cardiac disease, when they are placed fully under the influence of chloroform.

The failure of Fick may be explained partly because his experiments were performed, not on horses, but on dogs, the action of whose heart is more rapid and less powerful, but chiefly because the intervening tube was filled with water instead of air. The effect of the inertia of the water would be to render the instrument less sensitive to rapid changes of pressure, and it would also tend to modify the motion. Thus, the tracing obtained by Fick representing the pressure in the aorta shows no secondary waves at all. It has already been seen that the tracing of a dilated aorta (Pl. III, fig. 17) has, allowing for the difference of amplitude, much resemblance to the aortic tracing obtained by Marey (Pl. I, fig. 6), and in both cases the dicrotic wave is distinctly seen, although smaller and narrower than in the radial pulse. But the want of delicacy in Fick's instrument is especially shown by a paradoxical result obtained by him in repeated experiments. In tracings from the aorta and from the left ventricle, taken when the heart's action was rapid, he found that the maximum pressure indicated in the ventricle was considerably less than the minimum pressure in the aorta. Now, it is quite possible that, from the effect of the acquired velocity of the blood, the maximum pressure in the aorta might exceed the maximum in the ventricle, although no trace of such an effect is visible in Marey's curves, or in those of Fick, which were taken from a slowly acting heart. But it seems impossible that the aortic valves could be opened while the ventricular pressure fell far short of that in the aorta, because no considerable onward velocity could be acquired by the blood in the ventricle until after the opening of the valves. It is, indeed, suggested by Fick that this might happen if during diastole the apex of the ventricle only were filled, while the base remained empty, but such a partial reflection appears in the highest degree improbable. The conclusion seems to be clear, although Fick himself is disposed to reject such an explanation that, on account of the inertia of the water in the intervening tube, the

instrument was not sensitive enough to follow the rapid changes of pressure in the ventricle. This is confirmed by the fact that, by the use of an air manometer, Fick obtained results by no means in agreement with the indications of the other instrument, and in some instances the column of water in the manometer was thrown to a higher point when the sound was in the ventricle than when it was introduced only as far as the aorta.

It will have been observed that the cardiographic experience upon which the present paper is founded extends over not more than a year and a half, and therefore there is but a small proportion of the cases in which the diagnosis has been verified by post-mortem examination, since mitral stenosis rarely ends fatally within a short interval from the time at which the characteristic murmur can most easily be detected. It may be assumed, however, that there is no longer any question as to the association of the presystolic murmur with contraction of the mitral orifice, but only as to the mechanism by which the murmur is produced. Of the cases here discussed there are eight in which no physician at the present day would have had any hesitation in making a positive diagnosis of mitral contraction, although opinions might differ as to the mode of causation of the murmur. The cardiograms of the eight cases referred to are those in Pl. I, fig. 9; Pl. II, fig. 1; Pl. III, figs. 9, 10, 11, 13, 15, and 19.

To sum up the inferences suggested by a general review of all the tracings, it appears to me that the evidence of the cardiograph is in favour of the view that two totally distinct murmurs may be caused by mitral contraction—first, the auricular-systolic bruit, which may either run up to the first sound or be separated from it by a short interval; and, secondly, a diastolic bruit due to the venous flow through the narrow and roughened orifice, which in rare cases may be blowing in quality and separated from the succeeding systole by a long pause; and that, thirdly, these two may be merged together into a compound murmur, somewhat rough from its commencement, but much intensified in loudness and harshness towards its conclusion.

The proportionate number out of the cases of mitral contraction here discussed, in which the diastolic murmur was at times audible, would seem to indicate that it is not so rare as it has

been considered to be by the very few authors who have recognised it as distinct in rhythm and causation from the presystolic murmur. In most cases, however, it is heard only occasionally, and sometimes it is very faint.

I have not as yet met with any cardiograms which seemed at all to indicate that the auricular contraction had been transferred in rhythm to the preceding ventricular systole, and so formed the termination instead of the commencement of each revolution of the heart.

EXPLANATION OF THE PLATES.

The tracings have been copied by photo-lithography and are represented of the original size. They were all taken from the apex of the heart, except where it is otherwise specified. The following is the probable explanation of the points to which letters are appended:—*a*, auricular systole; *b*, closure of the auriculo-ventricular valves; *d*, hardening and rounding of the ventricles; *e*, in some cases the reflux, towards the apex, of the blood which had been impelled against the auriculo-ventricular valves; *f*, continued pressure against the chest caused by the locomotion of the heart; *g*, closure of the semilunar valves; *k*, second slight locomotion of the heart forwards caused by the reflux at the end of systole in the aorta and pulmonary arteries; *l*, influx of blood into the ventricles. The number of times the extent of the original motion is magnified in vertical height is indicated by the figures in each trace, which are preceded by the sign of multiplication. The curves to which the mark B is appended were taken with an instrument having a slower clockwork movement.

PLATE I.

Fig. 1. Typical apex tracing from a healthy heart. P. 63, B.

Fig. 2. From a healthy man, aged 25, in whom the arterial tension was low and the heart was acting vigorously. Curved lines are drawn to measure the interval between the opening and closing of the aortic valves. P. 70, B.

Fig. 3. Radial pulse tracing of the man from whom the cardiogram in fig. 2 was taken. Pressure 2 oz. Curved lines are drawn to measure the length of the systolic portion of the curve.

Fig. 4. Series of simultaneous tracings obtained by introducing one sound terminating in an elastic ampulla into the right auricle of a horse, a second similar sound into the right ventricle, and placing a third ampulla in an aperture made in the chest wall over the centre of cardiac impulse. The first curve, therefore, represents the auricular pressure, the second the ventricular pressure, and the third the apex-beat. Vertical lines are drawn at intervals of $\frac{1}{10}$ second. After Marey, 'Physiologie Médicale de la Circulation du Sang.' Page 68.

Fig. 5. Another series of simultaneous tracings obtained from a horse in the same way. After Marey, 'Mouvement dans les Fonctions de la Vie.' Page 142.

Fig. 6. Simultaneous tracings obtained by introducing one sound into the left ventricle of a horse and a second into the aorta. The first curve represents the pressure in the ventricle, the second that in the aorta. After Marey.

Fig. 7 and fig. 8. Tracings taken from the heart of a dog, exposed within the thorax, while artificial respiration was maintained. The pad was rested on the surface of the right ventricle, near the auriculo-ventricular groove. The heart was beginning to fail at the time when fig. 7 was taken.

Fig. 9. Emma C—, æt. 24. Short harsh presystolic murmur at the apex, and loud systolic murmur near the ensiform cartilage. Sometimes a prolonged rough

murmur extended through the interval between the second sound and the pre-systolic murmur. There was strong pulsation in the veins of the neck.

Fig 10, 11, and 12. Inverted tracings taken in different situations from a healthy heart which gave no positive impulse anywhere. Fig. 11 was taken near the usual position of the apex-beat; fig. 12 in the epigastrium, to the left of the middle line; fig. 10 also in the epigastrium, but to the right of the middle line.

Fig. 13. Thomas G—, æt. 56. Chronic Bright's disease with atheromatous arteries. The cardiac impulse was very powerful, but no murmur was heard. P. 64, B.

Fig. 14. Elizabeth A—, æt. 22. Chronic Bright's disease with dropsy. There was a slight systolic murmur at the apex, but the symptoms of the patient were those of renal and not of cardiac disease. The radial pulse tracing required a pressure of five ounces and showed a prolonged tidal wave, thus indicating high arterial tension. P. 76.

Fig. 15. George G—, æt. 33. To and fro aortic murmur; heart much hypertrophied. P. 74, B.

Fig. 16. John R—, æt. 30. A systolic murmur, accompanied by thrill, had previously been heard at the apex, and two separate post-systolic sounds towards the axilla. When the cardiogram was taken the murmur had disappeared, and there was only a rough first sound at the apex and a reduplicated second sound. Diagnosis, mitral contraction. The tracing has every wave inverted. P. 46.

PLATE II.

Fig. 1. Hannah G—, æt. 47. Systolic murmur at the apex, and prolonged purring murmur accompanied by thrill, which generally filled the whole interval from the second sound up to the succeeding systole. There was strong pulsation at the left side of the ensiform cartilage. Tracing A was taken at this spot, and Tracing B at the apex.

Fig 2. John R—, æt. 26. Convalescent after pneumonia. Heart healthy, but apex-beat diffused. P. 97. The tracing is partially inverted, and a retraction occurs soon after the commencement of systole, and cuts short the primary up-stroke at the point where the letter *d* is placed. This partial depression occupies the situation where the summit of the primary elevation *d* is generally seen.

Fig 3. George H—, æt. 19. Aortic and mitral regurgitation. Heart much dilated and hypertrophied. The cardiac impulse extended from the fourth to the sixth intercostal space, the apex-beat being in the fifth space and one inch external to the line of the nipple. The present tracing was taken in the fourth space, and fig. 6 in the sixth space. P. 71.

Fig. 4. Henry B—, æt. 17. Aortic regurgitation. The heart was much dilated, the apex-beat being in the sixth intercostal space and external to the line of the nipple. The pulse tracing showed extreme collapse in the diastolic portion and an almost entire absence of the tidal wave. From this it may be inferred that the regurgitation was very free and the contractions of the heart short and incomplete. The tracing is partly inverted, and a retraction occurs during the latter part of systole, followed by a sudden recoil. P. 60.

Fig. 5. Ellen B—, æt. 10. Mitral regurgitation. Heart much hypertrophied and impulse heaving. The tracing is a compound curve representing the combined

respiratory and cardiac movements, in which each respiration corresponds to exactly two revolutions of the heart. P. 84; R. 42.

Fig. 6. *See* fig. 3.

Fig. 7. John F—, æt. 25. Short, faint diastolic murmur at the apex; a presystolic murmur had been heard previously. Diagnosis, mitral contraction. P. 60.

Fig. 8. Joseph F—, æt. 20. Aortic regurgitation. Heart much hypertrophied. P. 79, B.

Fig. 9. Thomas S—, æt. 45. Aortic regurgitation. The diastolic murmur was very loud, and accompanied by a thrill felt at the apex. P. 74, B.

Fig. 10. Henry B—, æt. 26. Aortic and mitral regurgitation. The heart's impulse was powerful and heaving, and a diastolic thrill was felt at the apex. The tracing was taken during respiration, and two beats of the heart were comprised in each respiration. P. 72, B.

Fig. 11 and fig. 12. Charles H—, æt. 20. Loud systolic murmur at the apex, accompanied by thrill. The cardiac impulse was felt in the fourth, fifth, and sixth intercostal spaces; the centre of impulse was in the fifth space. Fig. 11 was taken in the fourth space, and fig. 12 at the apex in the fifth space. P. 96.

Fig. 13. E. S. F—, æt. 12. Blowing systolic murmur at the lower part of the sternum. P. 74.

Fig. 14 and fig. 15. Richard B—, æt. 34. Heart greatly dilated; apex-beat in the sixth intercostal space and one and a half inches external to the nipple; no murmur audible. The radial pulse of this patient is shown in fig. 18. P. 25, B.

Fig. 16. Rebecca Y—, æt. 11. Mitral regurgitation. Heart's impulse heaving. P. 86, B.

Fig. 17. George P—, æt. 19. Aortic and mitral regurgitation. P. 84, B.

Fig. 18. *See* fig. 14.

Fig. 19. Henry W—, æt. 35. Aortic regurgitation. This tracing was taken during respiration. The quality of the pulse showed the regurgitation to be very free. The heart was greatly hypertrophied. P. 88, B.

PLATE III.

Fig. 1. Healthy heart. P. 60.

Fig. 2. John C—, æt. 25. Aortic regurgitation. Heart much hypertrophied. P. 62.

Fig. 3. Alice E—, æt. 12. Mitral regurgitation. Heart's impulse very powerful and heaving. The tracing was taken during respiration. P. 88; R. 22, B.

Fig. 4. Emily L—, æt. 8. Loud systolic murmur at the apex, preceded by a very faint rumbling sound. A presystolic murmur had been heard previously. Heart much hypertrophied. P. 96.

Fig. 5. Samuel R—, æt. 18. Aortic and mitral regurgitation. Cardiac impulse diffused and thrilling; apex beat one inch below and one inch external to nipple. This patient's pulse was compressible, and dilatation appeared to preponderate over hypertrophy. The tracing is partly inverted on account of retraction in the latter part of systole. P. 86, B.

Fig. 6. William P—, æt. 39. Systolic murmur at the apex. Post mortem, the mitral orifice was found extremely wide, the left auricle dilated and hypertrophied. P. 60.

Fig. 7. Martha L—, æt. 27. Blowing systolic murmur at the apex, and also a rough murmur commencing from the second sound and continuing up to the systole. Post mortem, the mitral orifice was found contracted, but the left auricle not hypertrophied. P. 84, B.

Fig. 8. Rebecca S—, æt. 20. Mitral regurgitation combined with exophthalmic goitre. Heart dilated and hypertrophied. P. 110, B.

Fig. 9. John S—, æt. 9. Rough murmur at the apex, commencing from the second sound and running up to the first sound, becoming loud and harsh towards its conclusion. P. 80.

Fig. 10. Arthur W—, æt. 32. Short but characteristic presystolic murmur running up to the first sound. Heart's impulse very powerful. This patient had also chronic Bright's disease. P. 74.

Fig. 11. George M—, æt. 19. Long, loud, and harsh presystolic murmur, commencing immediately from the second sound and running up to the first sound. P. 60, B.

Fig. 12. Eliza P—, æt. 17. Short systolic murmur at the apex, followed by a longer blowing diastolic murmur, and then an interval of silence. A presystolic murmur had been heard previously. Diagnosis, mitral contraction. P. 64.

Fig. 13. Matilda A—, æt. 37. Long, rough presystolic murmur, commencing immediately from the second sound, and leading up to the first sound. P. 57.

Fig. 14. Sarah N—, æt. 50. Blowing diastolic murmur, followed by a pause, heard at the base to the left of the sternum, but somewhat louder at the apex. A systolic murmur had been heard occasionally towards the axilla. The tracing was taken during respiration. P. 78, B.

Fig. 15. Sarah F—, æt. 19. Long, rumbling presystolic murmur, accompanied by thrill, commencing immediately from the second sound, and separated from the first sound by a very short pause. P. 60.

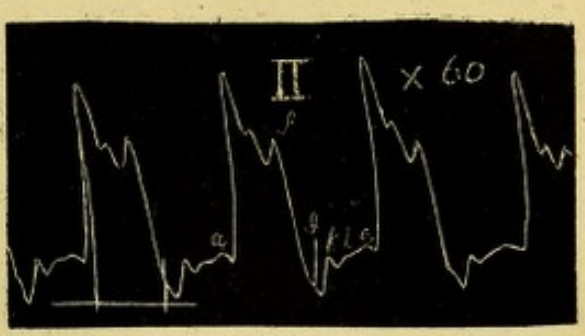
Fig. 16. Radial pulse tracing from the same patient. Pressure 4 oz.

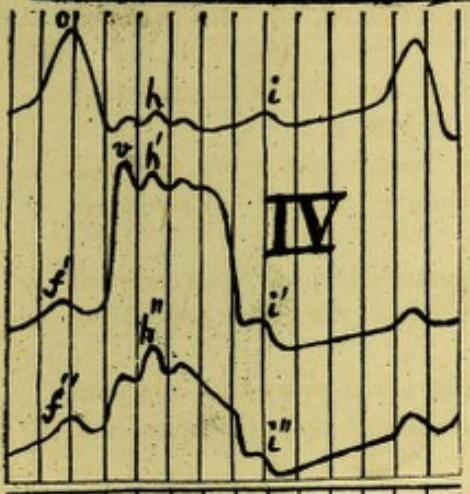
Fig. 17. Sarah M—, æt. 54. Aneurismal dilatation of ascending aorta. The tracing was taken at the spot of greatest pulsation in the second intercostal space to the left of the sternum. The effect of two pulsations is combined with that of one respiration. P. 69.

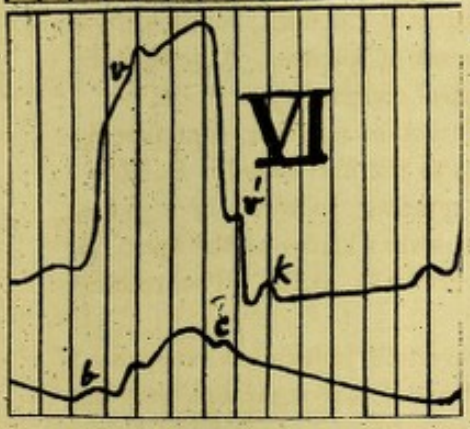
Fig. 18. George C—, æt. 44. Chronic Bright's disease. Heart's impulse powerful. The tracing was taken during respiration, and one respiration comprised exactly three revolutions of the heart. P. 80. The pulse tracing showed a considerable tidal wave, and required a pressure of 5 oz.

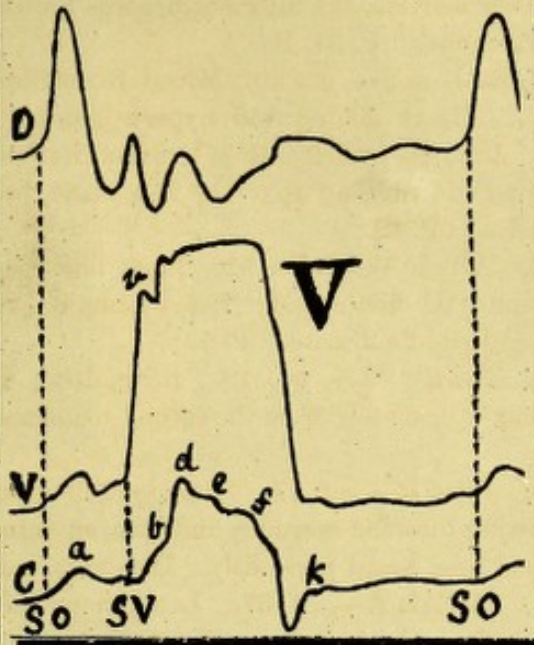
Fig. 19. Henry A—, æt. 8. Systolic, and long, harsh presystolic murmurs at the apex, both accompanied by thrill. The presystolic murmur commenced immediately from the second sound, and was separated by a short pause from the systole. P. 74.

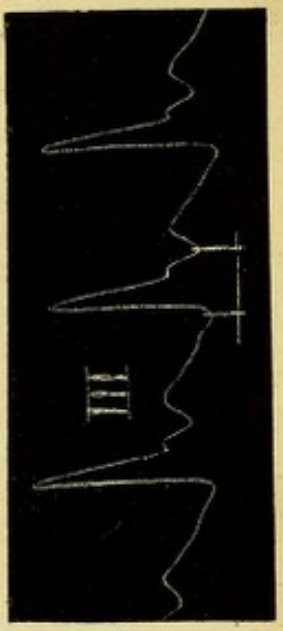
PLATE I.

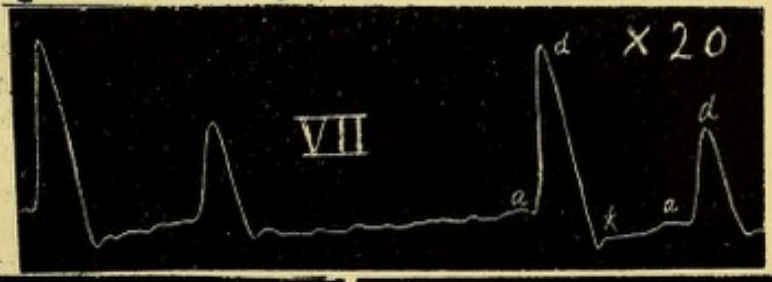


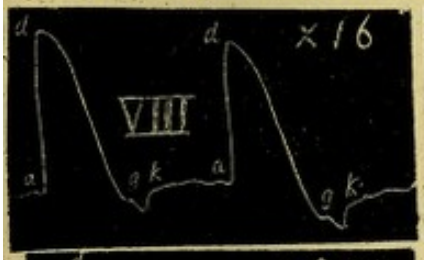


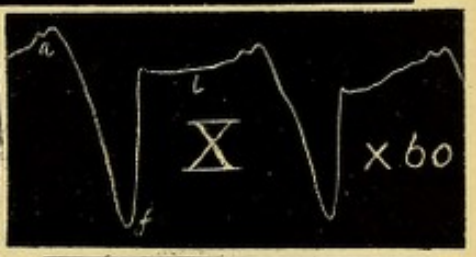


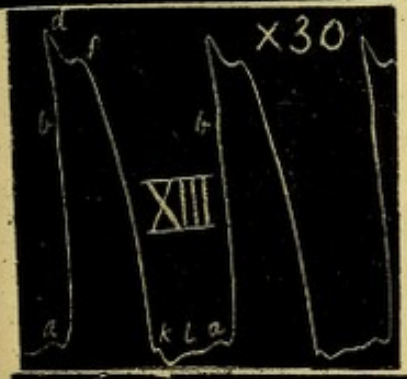


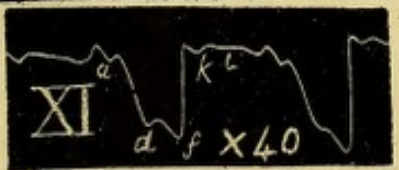


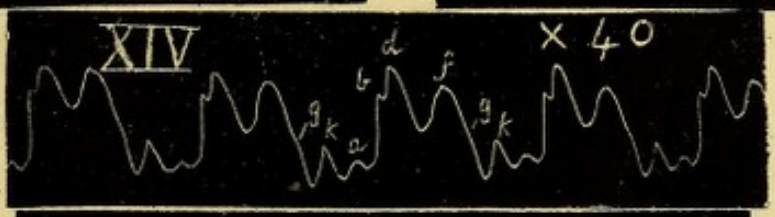












Whiteman & Bass, *Photo Litho's to the Queen*, 236, High Holborn.

PLATE II.

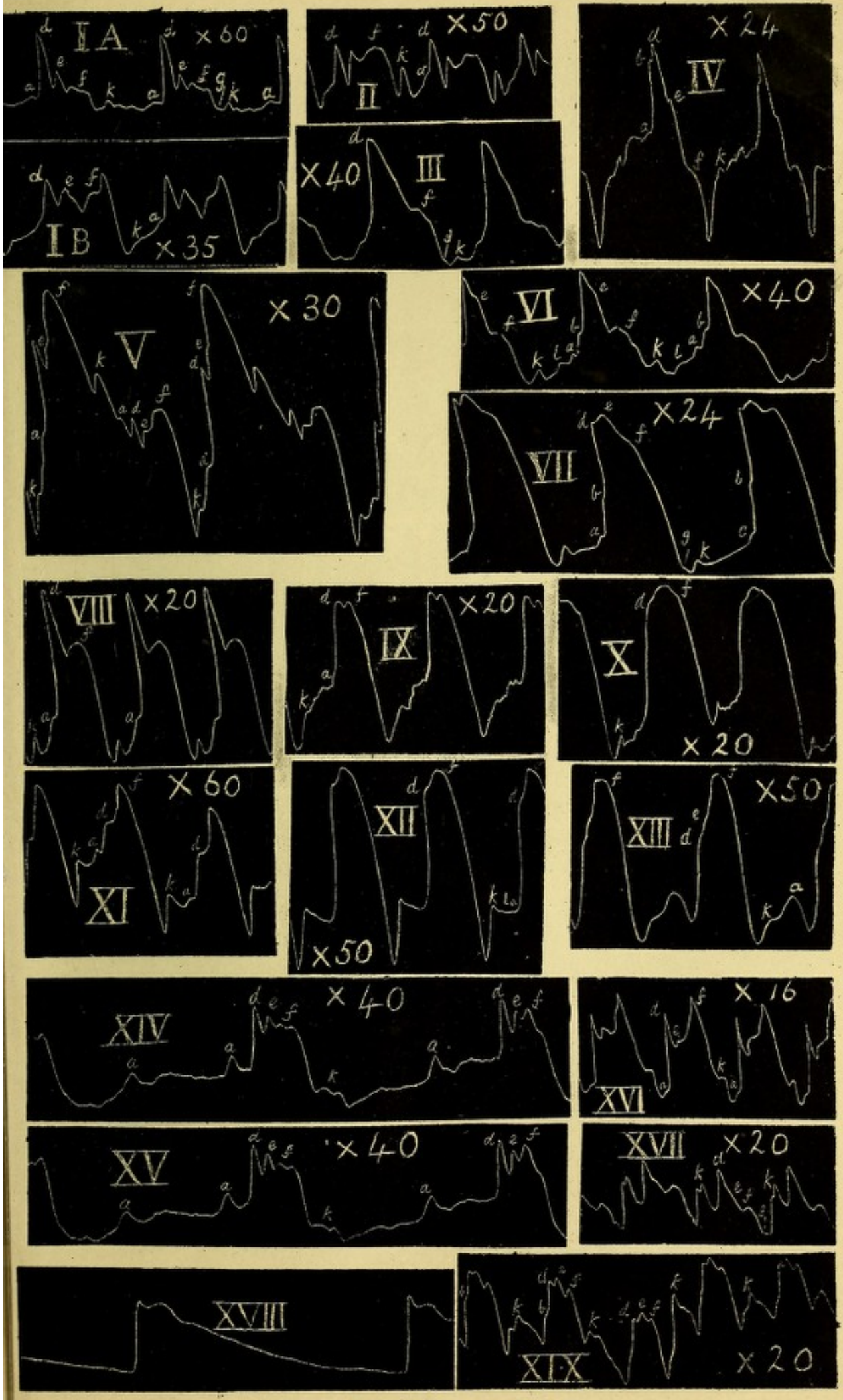


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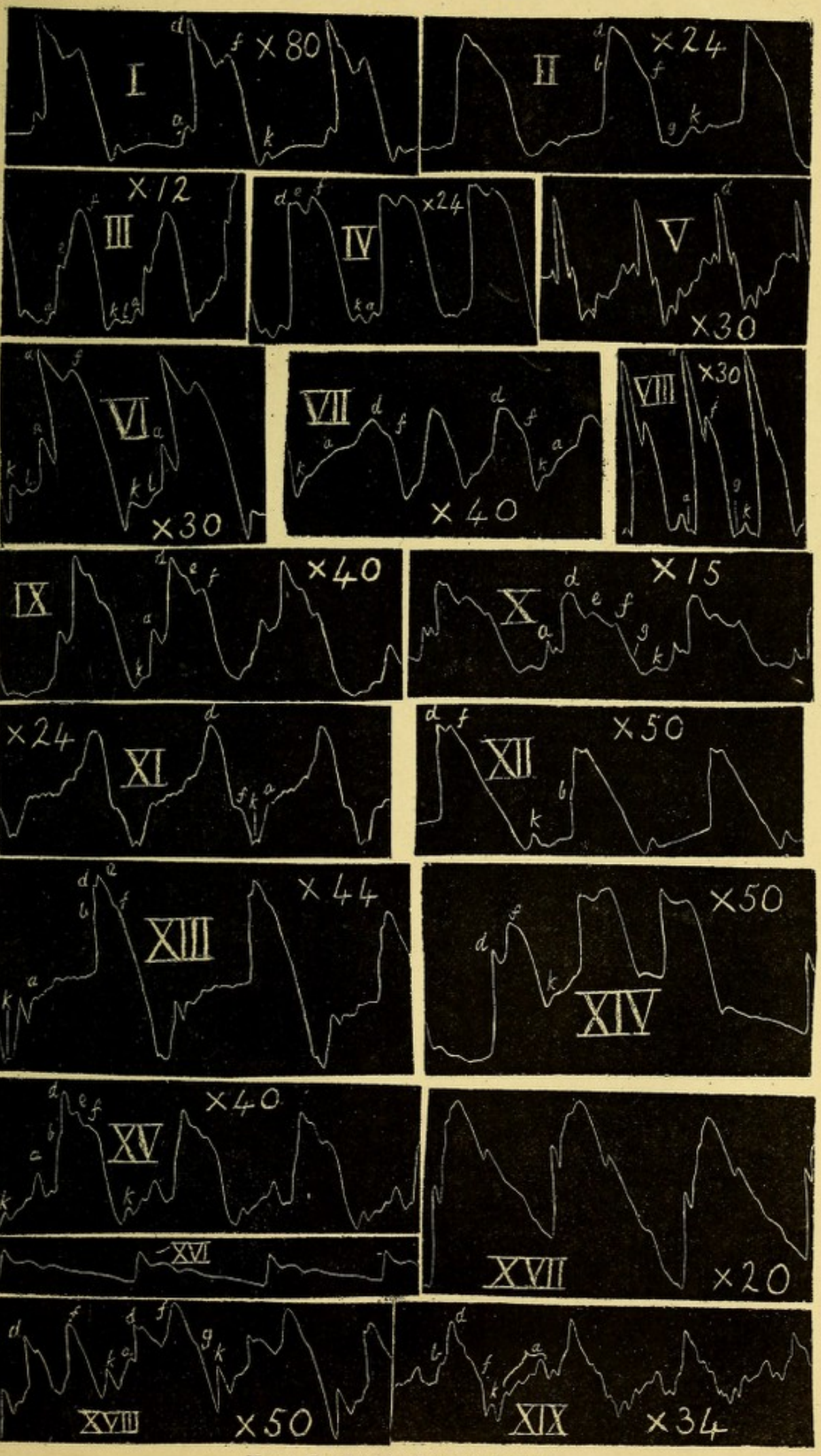
PLATE III.



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