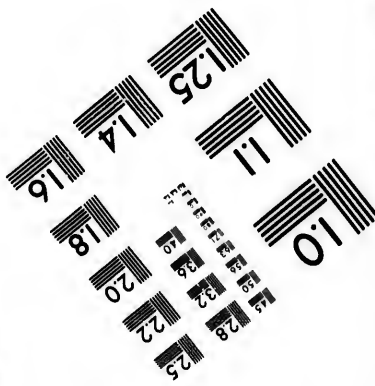
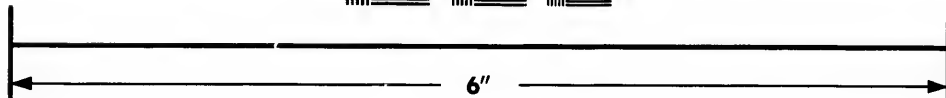
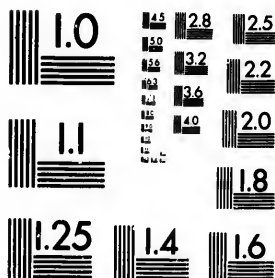


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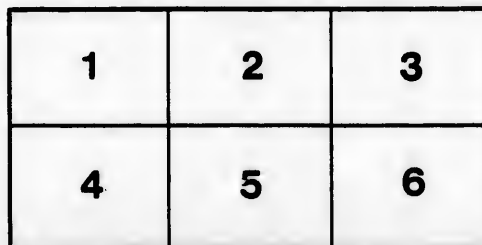
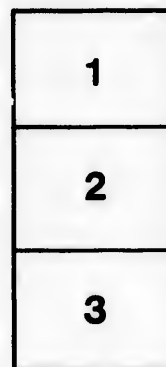
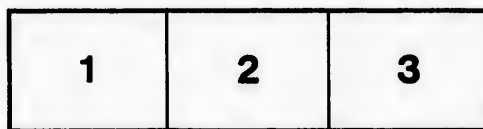
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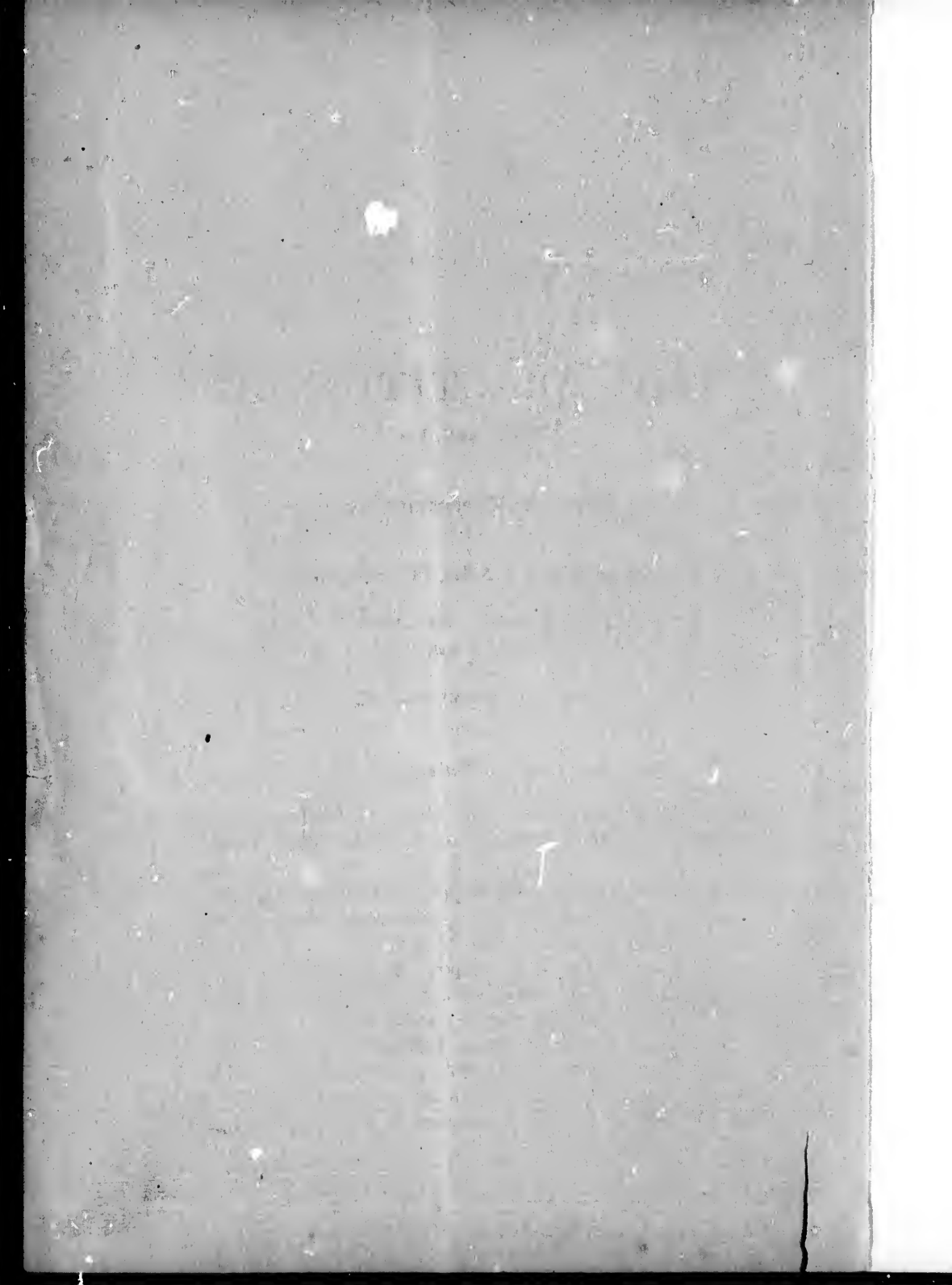
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THE PRACTITIONER.

FEBRUARY, 1890.

Original Communications.

HEART-BEAT AND PULSE-WAVE.

BY C. S. ROY, M.D., F.R.S.,

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[From the *Cambridge Pathological Laboratory.*]

THE clinical importance of an exact understanding of the meaning of graphic records of the heart and pulse, together with the fact that the graphic method is the most convenient for investigating certain physiological and pathological questions connected with the circulatory system, have induced us from time to time to make a few observations on the matters expressed by the title of our paper. In these observations we have employed various new instruments, which must be described in order that our tracings may be comprehended.

We will try, however, to say as little about the methods as is compatible with giving a reasonably intelligible account of our results. These results, we believe, are of a kind which will be found of value at the bedside, although to make them clear it is

necessary for us to refer to some matters which at first sight may seem to be of interest to the physiologist only.

It will be most convenient to consider the heart-beat first, for the simple reason that the characteristics of the pulse-wave are due for the most part to the manner in which the ventricular contraction takes place.

Graphic records of the heart-beat may be obtained in a variety of ways. The contraction and expansion of the muscular wall of the ventricle or auricle, for example, may be recorded. It is possible, also, to obtain a curve of the changes in the intra-ventricular pressure, as was done by Chauveau, Marey, and others, and more recently and accurately by Rolleston. The changes in the diameter of the ventricles, both in the antero-posterior and in the transverse direction, may be graphically determined: and the movements of the apex can in like manner be investigated. We may obtain curves of the contraction and expansion of the *musculi papillares*, showing the movements of the free edges of the auriculo-ventricular valves. Or again we may register the changes in volume of the heart as a whole. The kinds of tracings which we have just mentioned are perhaps among the most important of those by which the characters of the heart-beat can be studied, although there are many others which need not here be referred to.

SECTION I.

CONTRACTION-CURVE OF THE VENTRICULAR WALL.

Let us consider first of all the curve of contraction and expansion of the heart-muscle forming the ventricular wall. In order to obtain trustworthy tracings we must employ a method which will not hinder the movements of the heart as a whole. The method must also give tracings which will not be affected by these movements. The 'myocardiograph' (as we may call it to distinguish it from other forms of cardiograph) shown somewhat diagrammatically in Fig. 1, fulfils these conditions. By its means it is easy to obtain trustworthy graphic records of the variations in the distance apart of any two points on the surface of the heart-wall. Its construction is as follows: The light

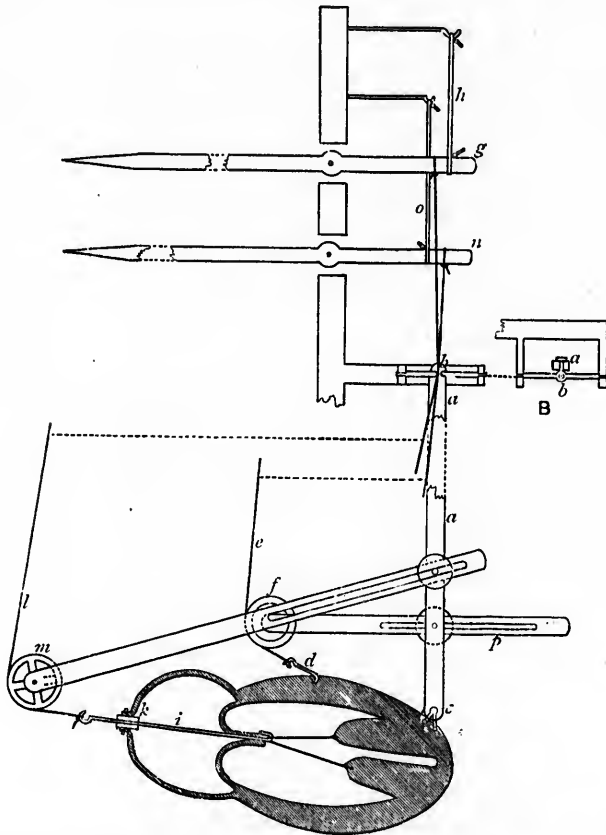


FIG. 1.—Myocardiograph for mammalian heart shown semi-diagrammatically. The light vertical rod *a*, which for convenience of space is shown shortened in the figure, is slung from the pivots which are represented in section as seen from above in B. This arrangement allows the rod *a* to swing freely, the centre of rotation being the small hole at *b* (in B). The lower end, *c*, of this rod is fixed to the surface of the heart-wall as seen in the figure. To obtain tracings of the heart-wall, the small hook *d* is inserted in the visceral pericardium at a convenient distance from the end of the rod *a*. To this hook is attached a strong silk thread *e*, which after passing round the light grooved pulley *f* is conveyed upwards through the small hole *b* to the lever *g*, being kept taut by the fine rubber thread *h*.

To obtain tracings of the contraction of the *musculi papillares*, the fine hooked wire *i* is inserted through the auricular wall and hooked over one of the mitral flaps. It slides easily in the collar *k*, which is tied to the edges of the opening in the auricular wall. To this is attached the thread *l*, which after passing round the light pulley *m* is conveyed upwards through the hole *b* to the lever *n*, being kept taut by the rubber thread *o*.

wooden rod (*a*) is slung on pivots or gimbals (*b*), somewhat after the manner of the ordinary mercurial barometer used on board ship. The lower end (*c*) of the rod is fixed to the heart-wall at any desired point by means of a thread which has been passed under a fold of the visceral pericardium. The end of the rod, from the manner in which it is slung, can follow the complicated movements of the heart without hindering them. Projecting from the rod near its lower end is the horizontal arm (*p*), which carries at its extremity a light vulcanite grooved pulley (*f*). Round this latter runs a strong silk thread (*e*), which is attached to a minute metal hook (*d*) fixed in the ventricular wall at any desired distance from the end of the rod (*a*). After passing round the pulley, the thread is carried upwards through a small hole (*h*) placed at the centre of rotation of the rod, as can be seen from the sketch elevation of the pivoting arrangement B in Fig. 1. From this hole the thread goes upwards to the recording lever (*g*), being kept taut by a fine indiarubber spring (*h*). The object of passing the thread through the hole at the centre of rotation of the rod is to allow free movement of the end of the latter with the heart, to which it is fixed, without any pull on the recording lever being thereby produced. The position of the lever is altered only by alterations in the distance between the point where the hook is fixed, and the point to which the rod is tied. It need hardly be added that in order to make use of this instrument the heart must be exposed by making a "window" in the thorax, the animal (a dog in our experiments) being curarised as well as anæsthetised, and the respiration being, of course, carried on artificially.

On the myographic curve of the auricular wall we do not propose to say anything here. The curves which are represented below in this section of our paper were obtained from the wall of the left ventricle, although, as they differ in no essential particular from curves obtained by the same method from the right ventricle, it must be understood that what we have to say about them applies equally to the two ventricles.

The myographic curve from the ventricular wall varies somewhat in character according to the relative position of the two points to which the instrument is attached. Let us consider first the curve given by this method when the two points

lie in a line running from base to apex, and more or less parallel



FIG. 2.

therefore with the interventricular sulcus. This, for convenience sake, we may call the "longitudinal curve" of the

ventricular wall, to distinguish it from that obtained from a part lying between two points on any line running round the ventricles, parallel to the auriculo-ventricular sulcus, which may similarly be termed the "transverse ventricular" curve.

The curves (A, B, C) shown in Fig. 2 are examples of tracings registering the contraction between two points in the middle third of a line joining the base and apex of the left ventricle at some distance from the ventricular septum. The illustration shows three different tracings, in all of which a rise of the recording lever corresponds to contraction of the ventricle wall, and a descent to expansion. The contraction begins, then, in each at *a*. It can be seen that the heart-wall contracts at first rapidly, until the shortening is suddenly arrested at the point *b*, the height of which on the ascending line varies under different conditions. In curve A (Fig. 2), it is low down, near the commencement of the ascent; in curve B it is about half-way up; while in curve C it is near the top. This arrest of the contraction is often followed by a certain degree of expansion, producing a more or less well-marked notch at *c*. On the other hand, it may (as in Fig. 4, A) be followed by a simple cessation of the contraction, or even by mere slowing, though this, according to our experience, is unusual. After the notch at *c*, the contraction continues, but is more slow than before. What is the cause of this interruption of the shortening? We at first thought that it must be due to the tightening of the auriculo-ventricular valves as a result of the rise of the intra-ventricular pressure, produced by the contraction of the heart-wall. That it results from something more than this will be seen when we come to compare the curve of contraction of the *musculi papillares* with that of the heart-wall itself.

We need only remark here that the time between the commencement of the contraction of the heart-wall and this break in the shortening varies under varying conditions, all of which however affect the amount of blood contained within the ventricle at the commencement of systole. The greater the expansion of the heart in diastole, the sooner does this break in the ascending line of the contraction curve follow its commencement, and *vice versa*. The part of the curve from *c* to *d* shows that the heart contracts more slowly at that part than it did at the beginning of systole. As

we shall see presently this part of the contraction curve corresponds in time to a very high intra-ventricular pressure, whereby greater resistance is offered to the shortening of the fibres of the heart-wall. The top of the curve from *d* to *e* shows that the ventricular wall remains contracted for a certain time after the active shortening has ceased. The duration of this period varies according to the rate at which the heart expels its contents: this rate, as we shall presently show, may vary greatly under different conditions. The expansion of the ventricle wall takes place with varying rapidity, the line from *e* to *f* descending at a fairly even rate. Sometimes the point *f* constitutes the lowest part of the curve (as in A and B of Fig. 2), while in other cases, (as in C of Fig. 2) the line of descent shows a marked shoulder, *g*, the part from *g* to *a* corresponding in time with the inflow of blood resulting from the venous and auricular systole. This shoulder is best-marked under conditions in which the venous pressure is low, and when, therefore, the amount of blood which can enter the ventricle before the auricular contraction is relatively small in amount.

Curve of Contraction taken in a line running round the Ventricle.

In Fig. 3 we give a myographic tracing from the wall of the left ventricle along a line parallel with the auriculo-ventricular



FIG. 3.

sulcus, taken, therefore, in a transverse direction, about half-way between the base and apex of the ventricle.

The tracing does not call for much remark. It resembles generally the myographic curve of the longitudinal fibres shown in Fig. 2. The main difference consists in the greater depth of the notch at *c*, and this, according to our experience, is an invariable characteristic of the transverse ventricular curve, as compared with the longitudinal curve.

Whatever be the cause of this notch, it acts more powerfully on the circular than on the longitudinal fibres.

SECTION II.

CONTRACTION CURVE OF THE PAPILLARY MUSCLES AND COMPARISON OF THIS WITH THE MYOGRAPHIC CURVE FROM THE WALL OF THE VENTRICLE.

To obtain trustworthy records of the contractions of the *musculi papillares* in the living mammalian heart is not such a hopelessly difficult matter as it may appear at first sight. We employed for this purpose a modification of the myocardiograph, by means of which it is possible to obtain curves of the contractions of the *musculi papillares* simultaneously with tracings of the contractions of the ventricular wall, the heart continuing, through the whole course of the experiment, to furnish these two curves without any appreciable abnormality in its action.

Fig. 1 represents the instrument arranged for this purpose. The end (*c*) of the light vertical rod (α) of the instrument is first of all tied to a point on the surface of the ventricle situated not far from the apex, and, as nearly as can be guessed, over the origin of one of the papillary muscles. The wire (*r*) is introduced into the ventricle through the auricular wall and the small hook at one of its ends is hooked over the free edge of one of the mitral flaps. It is perfectly easy to do this, although it may be necessary to move the hook once or twice in order to get it on to the middle of the flap chosen. The characters of the curve show when this has been effected. If it be hooked over the flap at a point where the latter is very narrow, the curve

obtained resembles that of the heart-wall. The middle part of the edge of the flap, on the other hand, is little affected by the contractions of the ventricle-wall, its movements being due almost entirely to the contractions and expansions of the papillary muscles.

The wire hook passes through the wall of the auricle, and, in order that there may be neither escape of blood by the side of the wire, nor interference with its movements by the auricular contractions, it is provided with a collar (*k*), in which it can slide easily, but which fits sufficiently closely to prevent escape of blood between the wire and the collar. The collar pierces the auricular wall, to which it is firmly tied so that no blood can escape. The hook can be inserted and kept in position throughout the experiment without a drop of blood escaping from the auricle. From the extra cardiac end of the wire a thread passes round a light grooved pulley (*m*), and then is carried upwards through the hole (*b*), at the centre of rotation of the rod (*a*), to the recording lever (*n*), being kept taut by a fine indiarubber thread (*o*), by means of which the degree of pull on the *musculi papillares* can be regulated at will. The longitudinal ventricular curve is obtained by means of the same instrument in the way already described.



FIG. 4.

The curves in Figs. 4 and 5 show simultaneous tracings from the ventricle wall A, and the papillary muscles B, obtained in the

manner just described from the left ventricle of a small terrier dog. The lines X, X, give the position of the lever points when the drum is at rest, and points equidistant from them on the two curves correspond in time, so that, by means of a pair of compasses, it is possible to find with exactitude the relation in time of the two curves. These traces show that the contraction of the papillary muscles begins after that of the heart-wall. That part of the contraction of the ventricle wall which lies between *a* and *b* in curve A (Figs. 4 and 5) takes place before



FIG. 5.

the contraction of the *musculi papillares* begins. The point *b* of the heart-wall curve corresponds exactly in time with the sudden commencement of the contraction of the papillary muscles. During the first part of the systole, then, the contraction of the longitudinal fibres of the ventricular wall approximate base and apex before the free edges of the auriculo-ventricular valves are pulled towards the apex by the contraction of the *musculi papillares*. This will of course result in a bulging upwards towards the auricle of the auriculo-

ventricular flaps during the period immediately following their closure. The edges of the valves may even be pushed away from the point of origin of the papillary muscles by the rise of intra-ventricular pressure during this first part of the systole, as is shown in curve B of Fig. 5. This must be due to passive stretching of the papillary muscles and of the *chordæ tendineæ*.

The shortening of the papillary muscles takes place in two successive stages. During the first of these (from *b* to *c* of Figs. 4 and 5) the contraction is rapid. This part of the curve corresponds in time to the arrest or slowing of the contraction of the ventricle-wall (*b* to *c* of the curves A) which is so constant a characteristic of these myocardiographic tracings.

To continue our analysis of these two sets of curves in Figs. 4 and 5—it can be seen that during the second part of the contraction of the papillary muscles (*c* to *d*) the shortening is slower than during the first stage. There are several very obvious reasons why this should be so. (1) When the edges of the mitral flaps are pulled down so that they are more or less in a line with the *chordæ tendineæ*, the resistance to the contraction of the papillary muscles will be greater than when the valves are bulged upwards towards the auricle. (2) If the continued contraction of the ventricular walls keep the intra-ventricular pressure fairly high, after the papillary muscles have nearly reached their maximum shortening, the papillary muscle is placed at a disadvantage, and may even undergo passive stretching, resulting in the appearance of a notch between *c* and *d* of the papillary curve. In other words the papillary muscle, during the first stage of its contraction, is so well placed in relation to the ventricle wall that its contraction usually causes passive arrest or even retrogression of the shortening of the fibres of the latter; while during the second phase of contraction of the *musculi papillares* (from *c* to *d* of Figs. 4 and 5) they are on a more equal footing with the ventricle wall. It need hardly be said that the degree of distension of the ventricle at this part of the systole will influence greatly the degree to which one of these two competing elements of the ventricular systole will be effective at the expense of the other.

The third phase of the contraction of the *musculi papillares* is that of persistent contraction (d to d') unaccompanied by continued shortening. This causes the curve to be more or less flattened at the top.

At the point d' the relaxation of the papillary muscles begins, and this commencement of their relaxation precedes the beginning of the relaxation of the ventricular wall, as can be seen by a reference to Fig. 4, the letters on the two curves of which mark points which correspond in time. The contraction of the papillary muscle, therefore, not only begins later than that of the ventricular wall, but it comes to an end sooner. The period of contraction of the papillary muscles (from b to d) may be about half of that of the ventricle wall, although the difference in duration of the two is usually less than this, the relation being about 5 to 8; the relation at all events is not a constant one. This short duration of the papillary contraction as compared with that of the ventricle wall *might* be explained by a wave of contraction running through the muscle-fibres of the ventricle wall till it reached the papillary muscles, returning as a reflected wave. The facts at our disposal, however, do not permit of our proving or disproving this theory.

It can be seen (in Figs. 4 and 5) that the expansion of the papillary muscles is at first rapid, namely, from the point d to e . During this period the ventricular wall still continues contracted. After e , the expansion of the papillary muscle is more gradual. Immediately after the point e on the papillary muscle curve B there is a more or less rounded shoulder f , indicating that at the commencement of relaxation of the heart-wall there is a slowing of the expansion of the papillary muscle. As the sudden fall of pressure within the heart begins with the commencement of expansion of the heart-wall, and not, to any appreciable extent, with the beginning of expansion of the papillary muscles, it is easy to understand why, while the intraventricular pressure is high, the papillary muscles expand more rapidly than when (after the point e) the intra-ventricular pressure is very much less. The shoulder f is presumably due to the sudden cessation of the pull exerted upon the *musculi papillares* by the pressure of the blood against the mitral valves.

After the point *f* the expansion becomes more and more gradual, ceasing before the expansion of the heart-wall has reached its maximum.

The analysis of the curves which we have given above enables us to divide the ventricular *systole* into five more or less distinct phases.

I.—During this phase the ventricle wall is contracting, but the *musculi papillares* are at rest (*a* to *b*, Figs. 2, 3, 4).

II.—During this the papillary muscles carry out the first rapid part of their contraction, accompanied by slowing, arrest, or retrogression of the shortening of the fibres of the heart-wall, which is most marked in tracings taken along a line running around the heart transversely (*b* to *c*, Figs. 2, 3, 4, 5).

III.—During this the shortening of the papillary muscle is considerably less rapid than during the last phase; the fibres of the heart-wall are also being shortened, although much more slowly than during the first part of its *systole* (*c* to *d*).

IV.—During this phase both papillary muscle and ventricle-wall remain contracted, but do not undergo further shortening (*d* to *d'*).

V.—During this period the papillary muscle expands rapidly, while the ventricle-wall remains contracted (*d* to *e*).

This last phase belongs both to *systole* and *diastole*, the expansion beginning in the papillary muscles.

The *diastole* of the heart-wall may be divided into three phases, namely:—

I.—During this expansion takes place rapidly, and with fairly uniform rapidity (*e* to *f*).

II.—This phase is only well shown on the curves when the amount of blood available to permit the ventricle to expand is not great, so that the expansion becomes slowed or even arrested after the first elastic expansion of the ventricles has drawn into them the greater part of the available blood (*f* to *g*, Fig. 2, C and 5 A).

III.—During this phase the wave of blood which results from the contraction of the veins and auricles reaches the ven-

tricular cavity, and causes or allows the final expansion of the ventricular wall which precedes systole (*g* to *a*).

It will be observed that we have said little or nothing as yet as to the absolute or relative duration of these eight phases of the ventricular cycle. The reason of this is that the relative duration is not by any means a fixed one, and the subject can therefore be best considered when we come to speak of the influences which vary the character of the heart-beat.

(To be continued.)

