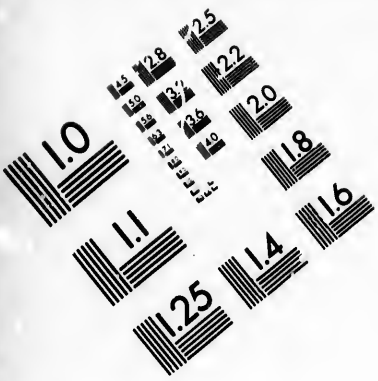

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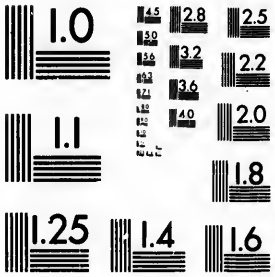

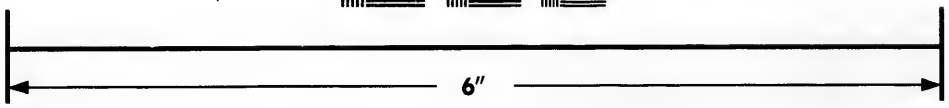

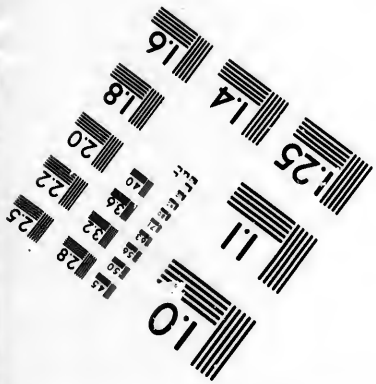

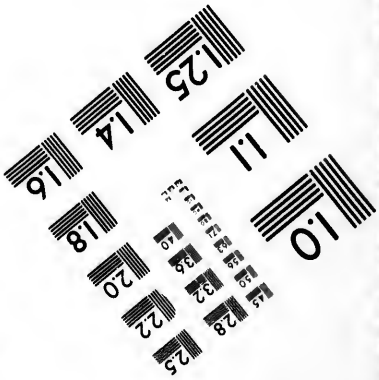

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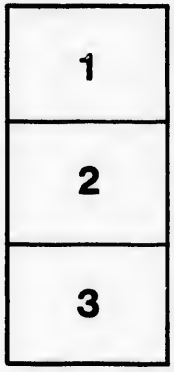

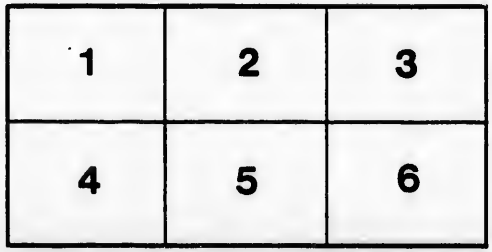

On the action Adami, J. G. 1890

[Extracted from the *Proceedings of the Cambridge Philosophical Society*, Vol VII. Pt. II.]

(3) *On the action of the Papillary Muscles of the Heart*[1]. By J. George Adami, M.A., M.B., Christ's College.

From time to time during the last twenty years continental observers have suggested that, in order to explain certain clinical phenomena, the papillary muscles of the ventricles must be looked upon as either contracting in a different manner to, or later than the rest of the heart, and in this country Ringer has suggested that one form of irregularity of the heart's action is due to a want of synchronism between the contractions of the muscles in question and the ventricular wall. On *à priori* grounds it would seem most unlikely that the papillary muscles contracted absolutely synchronously with the ventricular wall, for, were this the case, they would apparently nullify themselves. Attached as they are by the chordæ tendineæ passing from their apices to the edges and under-surface of the flaps of the auriculo-ventricular valves their main use is to aid in the complete closure of these valves, and thus to prevent regurgitation of blood into the auricles when the ventricles contract. And such is their position with relation to the auriculo-ventricular orifices that did they rapidly shorten contemporaneously with the sharp beginning of the general ventricular contraction, at a time, this is, when the blood pressure in the ventricular chamber has not been greatly raised, in the absence of such sufficient counteracting pressure upon the under surface of the valves they would the rather pull the flaps of the valves apart, and aid regurgitation. Yet up to the present no one has to my

[1] This paper embodies the results of a research that Professor Roy and I have been engaged upon conjointly during the last year. Fuller details will be found in a series of articles upon the 'Heart-beat and Pulse-wave,' published in the *Practitioner*.

knowledge endeavoured to obtain a simultaneous record of the work performed by these two portions of the cardiac muscle—nor has the probability that the two have different periods of action become in any way a part of general medical doctrine. Doubtless physiologists and medical men have voluntarily neglected to attempt exact observations upon this subject, from their knowledge that it is almost impossible satisfactorily to solve what would seem to be a much more simple matter,—the relation in time of the contraction of the muscle of the base and of the apex of the ventricle. And thus it has come to pass that nothing has been accomplished in what Professor Roy and I now find to be a field for research yielding suggestive, if not rich and important results.

Without the aid of diagrams it would be difficult and tedious to describe fully the apparatus employed by us in our work upon this subject, but some idea may be given of the mechanism of our instrument. In order to understand as fully as possible the nature of the papillary contraction it is necessary to gain at the same time a tracing of the contraction of the ventricular wall, so that the relation in time of the different portions of either curve obtained may be interpreted in terms of the other. We found that the most satisfactory means of recording the ventricular wall contraction was as follows. Taking two points upon the anterior surface of the left ventricle, one near to the apex, the other nearer to the base, to the former was attached a light but firm rod, moving easily upon an axis so as not to prevent or modify the general to-and-fro movement of the heart; into the latter was inserted a hook having a long shank, and these two parts of the instrument were connected in such a way with each other, and by means of a fine thread with the recording lever, that approximation of the two points upon the cardiac surface, that is to say, contraction of the muscle of the ventricular surface, resulted in an upward movement of the lever point; separation and cardiac dilatation, in a downward movement.

If now the light rod forming one limb of the above arrangement were attached to the surface of the ventricle over the region of insertion of one of the papillary muscles, a record of the contraction of this muscle could be simultaneously obtained by fixing on to the rod a cross-bar having at its further extremity a pulley, round which passed a fine thread, attached at one end to a second lever, and at the other to a strong hooked wire pulling upon one of the flaps of the mitral valve, and by this means upon the papillary muscle. To gain this attachment it was necessary to clamp off temporarily the left auricular appendix from the rest of the auricle, to make a small incision through its walls, to pass into this incision the hooked wire, to ligature the collar, in which the wire worked, to the wall of the appendix, and

then, removing the clamp, to pass the wire down through the mitral orifice and hook it over the edge of the mid-portion of one of the valve flaps. This operation required a certain amount of practice; the form of the curve shewed when it had been rightly performed. Attached in this way the hook pulled upon the free edge of the valve, and so through the chordæ tendineæ upon the papillary muscles, while the wire moved freely to and fro through the collar inserted in the auricular wall; there was little or no disturbance by clotting. The two levers recorded simultaneously upon the revolving drum, the one the contraction of the ventricular wall, the other the approximation and separation of the base of the columna carnea and the edge of the mitral valve, that is to say, the contraction and expansion of the papillary muscle.

Tracings so obtained shewed that the papillary muscles begin to contract and pull upon the mitral valves at a very definite interval after the commencement of the ventricular systole, indeed the interval between the two is so well marked that the papillary curves frequently exhibit an initial depression, due it would seem to an actual stretching of the papillary muscles and separation of their bases from the edges of the valve, the increased blood pressure within the ventricular chamber acting upon the valve and tending to drive it upwards into the auricular cavity. This is followed by the rapid contraction of the papillary muscles, which in its turn affects the curve obtained from the ventricular wall. Up to the moment when the papillary contraction begins, the lever point registering the heart-wall curve had ascended rapidly and in an almost straight line; but now the ascent is slowed, and at times there may be a slight depression or actual notch on the upstroke. This does not indicate that there has been an interference with the act of *contraction* on the part of the heart-wall, but that the *shortening* of the contracting muscles has been interrupted, and this lessened shortening is due to the sudden increase in the intra-ventricular blood pressure consequent upon the papillary contraction. Small in bulk as they are compared with the heart-wall, the musculi papillares by pulling upon the large flaps of the mitral valve must exert an influence upon a very considerable proportion of the surface of the ventricular cavity, and their contraction must have a distinct effect upon the intra-ventricular blood pressure. That this is the case is rendered evident by a comparison of the heart-wall curve with the curve of intra-ventricular pressure. The slowing or depression upon the upstroke of the former corresponds in time with a very well-marked rise or secondary wave upon the latter, and it is this sharp rise of pressure due to the contraction of the papillary muscles that hinders for the time the shortening of the muscle fibres of the wall of the ventricle: these fibres suddenly receive, as it were, an extra load,

and though there is no stoppage in the act of contraction the rate and extent of their contraction are in consequence diminished.

The sudden powerful contraction of the musculi papillares is followed by a stage in which the shortening of the muscles is slowed and the ascent of the curve more gradual: and simultaneously there is a more rapid ascent of the heart-wall curve. After this both portions of the ventricle remain for a comparatively long period in a state of contraction unaccompanied by further shortening, and the summits of both tracings are more or less flattened. We then find that the papillary muscles begin to expand before the rest of the ventricle.

To sum up the above details: *the papillary muscles begin to contract later than the ventricular walls, and commence their expansion at an earlier period. They act indeed only during that period when upon* à priori *grounds we should expect them to be contracted, not pulling upon the segments of valve until these have been brought into firm apposition by the increased blood pressure, beginning to act also at a time when further increase of pressure would tend to drive the segments upwards into the auricle, and so cause regurgitation.* Their contraction produces a sudden definite increase in the intraventricular blood pressure, well marked upon the blood pressure curves, and this increase causes a diminution in the rate of shortening of the muscle of the heart-wall, indicated by a depression upon the line of ascent of the curve obtained from the ventricular wall.

We hesitate to offer any explanation of this virtually independent action of the papillary muscles: we can only declare that the more we have studied the tracings obtained under various conditions, the more we have been led to conclude that the moment when they begin to contract is not primarily dependent upon the moment of commencing ventricular contraction. We find for example that an overdose of liquor strychnine may lead to complete asynchronism between these two components of the ventricular action; or, again, there may be a ventricular systole unaccompanied by papillary contraction, or *vice versa*. Again, the first effect of strophanthus is to cause rapidly increasing force of the contraction of the papillary muscles as compared with the heart-wall. Further, the period of papillary contraction bears no direct relation to the moment of origin of the pulse wave, to the time that is when the blood begins to pour from the heart into the arteries. Yet under normal conditions the pulse wave would seem to begin almost at the moment when the musculi papillares exert their first sharp strong pull upon the valves, and so act as an additional factor in raising the intraventricular pressure above that in the large arteries. In short, the phenomena of the papillary contraction would appear to supply further proof

as to the automatic, non-nervous action of cardiac muscle-fibres. No nerve ganglia, and, as far as we know, no nerve fibres have been made out as controlling the musculi papillares, and the moment at which they begin to contract, the duration, and the extent of their contraction would appear to be determined in large measure by the intraventricular blood pressure and the quality of the blood.

In conclusion, a few words may be said as to the way in which our observations throw light upon certain peculiarities of the pulse curve, which so far have been very variously explained—and as to which there has been much uncertainty. In tracings of the normal pulse gained by Marey's sphygmograph, or the equally unsatisfactory modification thereof usually employed in this country, or again by Dudgeon's sphygmograph in what may be termed its lucid intervals, there can often be seen two well-marked secondary waves in the first part of the curve previous to the dicrotic notch. The first of these has received the name of 'apex' or 'percussion' wave, the second that of 'tidal' or 'predicrotic.' That the former is not simply due to inertia is shewn by the fact that not unfrequently a small inertia wave may be superposed upon it, removable by proper adjustment of the instrument. By comparing the curves of the contraction of the ventricular wall or of the intraventricular blood pressure with the pulse curve taken simultaneously at the base of the aorta, Professor Roy and I have been enabled to shew that the first of these curves corresponds in time to the first period of contraction of the papillary muscles and the consequent increase in the intra-cardiac (and intra-arterial) blood pressure. This should therefore be termed the *papillary wave.* The second we consider is not by any means a secondary wave, but is really the latter portion of main wave due to the general ventricular systole, the first smaller papillary wave being superposed upon its first portion. This we would call the *systole remainder wave,* or, more shortly, *remainder wave.*

The same series of observations has also given us an explanation of the form of pulse usually termed the *anacrotic,* in which there is a small well-marked wave upon the upstroke of the pulse tracing, not, as in normal conditions, forming the apex of the curve. This form is to be found in cases where there is high intra-arterial pressure, or obstruction to the onward flow of the blood. Where there is high intra-arterial pressure there also the intra-cardiac pressure must be raised to a correspondingly high point before it becomes greater than that in the aorta, and before the valves be thrown open, that is to say, the pulse wave must begin at a later period of the cardiac systole. I have already stated that there is no absolute relation between commencement of the papillary contraction and the moment of opening of the aortic valves.

Hence in this case a fair portion of the papillary contraction has taken place before the blood begins to pass from the ventricle, or to speak more correctly, before the pulse wave can be propelled along the aorta, consequently the papillary contraction is shewn but incompletely upon the pulse wave, only its latter part is represented in the pulse, the papillary wave appears at a lower point on the ascent of the curve than under normal conditions, the greater portion of the blood being expelled by the long continuing systolic contraction; in fact, the *papillary* factor of the pulse is small, the *systolic remainder* considerable. It is interesting to note that where the intra-arterial pressure is greatly increased the intra-cardiac pressure curve shews the same tendency toward anacrotism; the papillary wave, instead of forming the apex of the curve, may be comparatively low down upon the line of ascent.