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