Mitral Regurgitation in Ventricular Premature Contractions*

The Role of the Papillary Muscle

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In view of the possibility that the mechanism of mitral regurgitation following premature ventricular contractions may contribute to the understanding of mitral regurgitation in clinically important causes of nonrheumatic mitral insufficiency, we studied 320 premature ventricular contractions in eight dogs. Since the papillary muscle is an important component of the mitral valvular complex, the extent and course of lengthening and shortening of the papillary muscle was investigated with ultrasonic crystals implanted within the anterolateral papillary muscle. Lengthening and shortening of a segment of the free wall of the left ventricle was measured simultaneously with identical crystals in three dogs. During sinus rhythm, lengthening of the papillary muscle always began well before the onset of isovolumic contraction of the left ventricle and continued throughout isovolumic contraction, indicating that lengthening occurred during closure of the mitral valve. When lengthening was prevented by premature ventricular contractions, left atrial v waves occurred. These observations indicate that lengthening of the papillary muscle during the latter part of diastole is required for proper closure of the mitral valve and that inadequate lengthening can result in mitral regurgitation.

Mitral regurgitation during ventricular premature contractions is frequently observed during left ventriculographic studies. Premature ventricular contractions have been shown to produce transient mitral regurgitation when they occur in the midportion of diastole, but not at other times during the cardiac cycle. The mechanism by which the ventricular premature contractions produce mitral regurgitation in these circumstances is not recognized. The clinical association of mitral regurgitation with ischemic dysfunction of the papillary muscle has suggested that failure of the papillary muscle to contract during ventricular systole may cause mitral regurgitation.In view of this suggested mechanism of mitral incompetence, alteration of the normal behavior of the papillary muscle during ventricular premature contractions perhaps may play a role in producing the associated transient regurgitation. The purpose of this investigation is to describe the behavior of the papillary muscle in sinus rhythm and the changes in its pattern of contraction during premature ventricular contractions. The mechanism of mitral regurgitation associated with premature ventricular contractions may be of relevance in understanding mechanisms of clinically important forms of nonrheumatic mitral regurgitation.

Materials and Methods

Eight mongrel dogs weighing 20 to 30 kg (44 to 66 lb) were anesthetized with sodium pentobarbital (30 mg/kg of body weight) and were studied with the chest open. Ventilation was maintained with room air by means of a respirator (Harvard Apparatus Co.) attached to an endotracheal tube. Aortic, left ventricular, and left atrial pressures were measured with a catheter-tip micromanometer (Millar Instruments, Inc.).

Segmental changes of length of the anterolateral papillary muscle were measured by miniature ultrasonic dimension gauges (Schussler). A pair of ultrasonic crystals was inserted blindly through the left ventricular wall into the anterolateral papillary muscle. This was accomplished by inserting the crystals through the left ventricle in a region between the first diagonal branch of the left anterior descending coronary artery and the first marginal branch of the left circumflex coronary artery. The crystals were completely inserted within the papillary muscle and were separated from each other by 0.9 to 2.2 cm. The position of the crystals within the papillary muscle was confirmed at autopsy at the conclusion of each study. Only dogs in which both crystals were shown to be definitely within the anterolateral papillary muscle were included in the study. If the crystals were not properly aligned along the axis of shortening of the papillary muscle, then measured changes of length would be smaller than actually occurred; however, the papillary muscle is a long narrow structure. Therefore, misalignment of only a few millimeters would be possible if both ultrasonic crystals were within the papillary muscle. On the basis of the known

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Manuscript received June 28; revision accepted September 4.

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CHEST, 77: 6, JUNE, 1980
separation of the crystals and the thickness of the papillary muscle, geometric calculations indicate that the maximal error due to misalignment would be about 15 percent.

In six of the eight dogs, autopsy showed the crystals were aligned along the axis of the papillary muscle. In two dogs the crystals inserted at the base were near the endoventricular surface of the papillary muscle. In these dogs, therefore, an estimated error of 15 percent may have occurred in the measurements.

In three dogs an additional pair of miniature ultrasonic crystals was inserted in the free wall of the left ventricle in order to compare simultaneous changes of the length of the segment of the papillary muscle and the free wall of the ventricle. The crystals in the wall were inserted 5 to 8 mm within the myocardium and were separated from each other 1.3 to 1.6 mm.

Segmental length of the papillary muscle, together with left atrial, left ventricular, and aortic pressures and lead 2 of the electrocardiogram were recorded (Electronics for Medicine VR-12) during sinus rhythm and during induced or spontaneous ventricular premature contractions. Premature ventricular contractions were induced by mechanical stimulation of the external surface of the left ventricle at a site well away from the ultrasonic crystals. The appearance of prominent v waves in the tracing of the left atrial pressure was considered evidence of mitral regurgitation.

Elongation of the segment of the papillary muscle was calculated at the ratio of the change in length during one cardiac cycle to the greatest segment length of the papillary muscle. Similarly, elongation of the papillary muscle during premature ventricular contractions was calculated as the ratio of papillary muscular elongation before interruption by premature ventricular contraction to the greatest length of the papillary muscular segment.

RESULTS

During sinus rhythm the free wall of the left ventricle and the papillary muscle showed asynchronous behavior. Lengthening of the papillary muscle occurred out of phase with lengthening of the ventricular wall (Fig 1). Lengthening of the free wall of the left ventricle began shortly after the aortic incisura, continued throughout diastole, and reached a maximal value 39 ± 6 msec before the onset of ejection. The papillary muscle began to lengthen 65 ± 6 msec after the beginning of lengthening of the free wall of the left ventricle. The papillary muscle continued to lengthen throughout diastole and isovolumic contraction of the left ventricle, and lengthening continued 23 ± 2 msec after the onset of ejection. Therefore, it is clear that the papillary muscle was in the process of lengthening at the time of closure of the mitral valve.

Analysis of shortening and lengthening of the papillary muscle was performed during 320 premature ventricular contractions. The percentage of ectopic beats in which a v wave suggestive of mitral regurgitation was observed was highly variable. The ectopic beats associated with v waves showed a consistent pattern. Mitral regurgitation resulted when the premature ventricular contraction prevented sufficient lengthening of the papillary muscle (Fig 2 and 3). No v wave was observed when the ectopic contraction of the left ventricular wall occurred when the papillary muscle was already sufficiently lengthened (Fig 4). During sinus rhythm the papillary muscle was shortest near the end of isovolumic relaxation. This was at or shortly after the crossover of left atrial and left ventricular pressure. During the course of diastole and early systole, the papillary muscle elongated 15 ± 3 percent. If premature ventricular contractions occurred when the average elongation of the papillary muscle was only 3 to 7 percent, then v waves occurred (Table 1). If premature ventricular contractions occurred when
the average elongation was 11 to 15 percent, then v waves did not occur.

DISCUSSION

The mechanism by which abnormal function of the papillary muscle may produce mitral regurgitation can be understood if the normal physiologic function of the papillary muscle is clearly in mind. Observations in this study of papillary muscular behavior during normal sinus rhythm indicate that

![ECG and pressure tracings](image)

**Figure 2.** Lengths of segment of papillary muscle (PAP) and left ventricular (LV) wall and pressures during premature ventricular contraction (PVC) that occurred when papillary muscle was beginning to lengthen. Further lengthening was prevented by premature contraction, and mitral regurgitation was produced as indicated by left atrial v wave (v). AO, Aortic pressure; LV, left ventricular pressure; LA, left atrial pressure; and a, "a" wave.

![ECG and pressure tracings](image)

**Figure 3.** Lengths of segment of papillary muscle (PAP) and left ventricular (LV) wall and pressures during premature ventricular contraction (PVC) that produced premature shortening of papillary muscle. Mitral regurgitation, as indicated by v wave (v), resulted. AO, Aortic pressure; LV, left ventricular pressure; LA, left atrial pressure; and a, "a" wave.
complete mitral valvular closure requires end-diastolic elongation of the papillary muscle. At the time of mitral valvular closure, left ventricular volume is maximal. Therefore, it appears that the observed increase in length of the papillary muscle is necessary to bridge the distance between the left ventricular free wall and the mitral leaflets, allowing their coaptation and subsequent closure of the valve.

The analysis of changes of segment length of the papillary muscle showed that mitral regurgitation resulted from premature ventricular contractions when diastolic lengthening of the papillary muscle was prevented. Occasionally, premature shortening occurred following interruption of lengthening of the papillary muscle, and this may have contributed to the mitral regurgitation.

We observed, as have others,¹-⁷ that not all premature contractions produced transient mitral regurgitation. No mitral regurgitation occurred with premature ventricular contractions if the papillary muscle was already sufficiently lengthened at the time of closure of the mitral valve.

The mechanism of mitral regurgitation during ventricular premature contractions may be relevant to the mechanism of mitral regurgitation in the “papillary muscular dysfunction syndrome.”⁸-¹⁰ Our observations suggest that mitral regurgitation results from insufficient physiologic lengthening of the papillary muscle. This causes an inability of the papillary muscle and chordae to bridge the distance from the insertion of the papillary muscle to the valvular leaflets. In the papillary muscular dysfunction syndrome, two factors may contribute to insufficient lengthening. The first is abnormal and fixed shortening of the papillary muscle due to scarring. Shrinkage of the papillary muscle in chronic ischemic heart disease has been noted by many observers, and a possible role in the production of mitral regurgitation has been repeatedly suggested.⁸-¹² The second factor is an abnormally long distance to be spanned. This would result from dyskinesia or akinesia of the free wall of the left ventricle at the site of attachment of the papillary muscle. Dyskinesia or akinesia of that segment would increase the distance between the left ventricular free wall and the mitral leaflets during systole, thereby requiring more than normal lengthening of the papillary muscle for mitral closure. In support of the possibility that abnormal motion of the wall may produce mitral regurgitation, it has been observed that in the papillary muscular dysfunction syndrome in patients a dyssynchronous, poorly contracting left ventricle is almost always present.¹⁴-¹⁵ In experimental isolated papillary muscle infarction, mitral regurgitation is generally reported not to occur¹⁶-¹⁸ or when observed, it was rarely in the immediate post-infarct period.¹⁹

In conclusion, mitral valvular closure is permitted by end-diastolic lengthening of the papillary muscle. Mitral regurgitation occurred when lengthening was
prevented by premature ventricular contractions. These observations suggest that pathologic circumstances causing abnormal shortening of the papillary muscle, such as scarring, or circumstances causing an elongation of the distance between the left ventricular free wall and the mitral leaflets, such as an aneurysm, would lead to mitral regurgitation.

REFERENCES