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Papillary Muscle Traction in Mitral Valve Prolapse: Quantitation by Two-Dimensional Echocardiography

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Previous angiographic observations in patients with mitral valve prolapse have suggested that superior leaflet displacement results in abnormal superior tension on the papillary muscle tips that causes their superior traction or displacement. It has further been postulated that such tension can potentially affect the mechanical and electrophysiologic function of the left ventricle. The purpose of this study was to confirm and quantitate this phenomenon noninvasively by using two-dimensional echocardiography to determine whether superior displacement of the papillary muscle tips occurs and its relation to the degree of mitral leaflet displacement.

Directed echocardiographic examination of the papillary muscles and mitral anulus was carried out in a series of patients with classic mitral valve prolapse and results were compared with those in a group of normal control subjects. Distance from the anulus to the papillary muscle tip was measured both in early and at peak ventricular systole. In normal subjects, this distance did not change significantly through systole, whereas in the patient group it decreased, corresponding to a superior displacement of the papillary muscle tips toward the anulus in systole (8.5 ± 2.6 vs. 0.8 ± 0.7 mm; $p < 0.0001$). This superior papillary muscle motion paralleled the superior displacement of the leaflets in individual patients ($y = 1.0x + 0.8$; $r = 0.93$) and followed a similar time course. The systolic motion of the mitral anulus toward the apex, assessed with respect to a fixed external reference, was not significantly different in the patients and control groups (14.3 ± 4 vs. 15.5 ± 4.4 mm; $p = 0.4$) and therefore could not explain the superior papillary muscle tip motion relative to the anulus in the patients with mitral valve prolapse.

These results demonstrate that normal mechanisms maintain a relatively constant distance between the papillary muscle tips and the mitral anulus during systole. In classic mitral valve prolapse, superior leaflet displacement is paralleled by superior displacement of the papillary muscles that is consistent with superiorly directed forces causing their traction. Two-dimensional echocardiography can therefore be used to measure these relations and test hypotheses as to their clinical correlates in patients with mitral valve prolapse.

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Mitral valve prolapse is characterized by abnormal superior displacement of the mitral leaflets above the level of the mitral anulus during systole. It has been postulated (1–4) that this leaflet displacement may exert abnormal tension on the papillary muscle tips, causing their superior traction or displacement, and that such traction may have adverse pathophysiologic effects. For example, experimental traction of the papillary muscles (5) has been shown to cause electrophysiologic instability that may predispose to ventricular arrhythmia. Angiographic studies (6,7) have demonstrated a hyperdynamic inward "buckling" of the posterior left ventricular wall in patients with severe prolapse and ventricular arrhythmias and have related this buckling to traction of myocardial segments by the superiorly drawn papillary muscles. However, such papillary muscle motion is difficult to assess in ordinary ventriculographic studies (7), and such studies would be difficult to perform routinely in patients referred for evaluation of mitral valve prolapse. The present study originated with the observation that similar motion could be identified during echocardiographic examination of patients with classic mitral valve prolapse. These studies suggested that in such patients the papillary muscles and mitral leaflets move in parallel toward the left atrium in systole, whereas in normal subjects, the papillary muscles move apically during systole in parallel with the mitral anulus, maintaining a relatively constant distance with respect to the anulus.

This observation of altered papillary muscle motion in patients with mitral valve prolapse is of interest for several

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reasons. Even with refined echocardiographic criteria, uncertainty remains as to what constitutes abnormal leaflet displacement relative to the mitral anulus (8). Abnormal superior motion of the papillary muscles may serve to confirm the abnormality of superior leaflet motion in patients evaluated for mitral valve prolapse by demonstrating disturbed motion of other cardiac structures connected to the leaflets. Observations of the motion of the leaflets and papillary muscles might provide additional insight into the mechanical link between the base of the heart and the papillary muscles, which has been proposed to play a role in ventricular function (9–17). Finally, the ability to confirm the observation of papillary muscle displacement and its relation to leaflet displacement by echocardiography would serve as the starting point for exploring the role of such motion in the pathophysiology of mitral valve prolapse.

The purpose of this study was therefore to describe this phenomenon of altered papillary muscle motion by two-dimensional echocardiography and to test the hypothesis that in patients with mitral valve prolapse, exaggerated superior displacement of the papillary muscle tip relative to the mitral anulus occurs and is related in magnitude to the degree of leaflet displacement.

Methods

Selection of subjects. Our hypothesis was tested in patients selected over an 8-month period for having classic mitral valve prolapse by echocardiography, no other structural cardiac abnormalities (such as myocardial or valvular disease that could affect leaflet or papillary muscle motion) and images suitable for quantitative analysis. Classic mitral valve prolapse was defined as leaflet thickening (>5 mm [18,19]) and superior leaflet displacement above the level of the mitral anulus in the parasternal long-axis view (20). These criteria were chosen because the phenomenon was initially observed in such patients and it is well accepted that these changes represent mitral valve prolapse (8,20,21).

Over an 8-month period, studies were performed in 1,792 consecutive patients in the outpatient laboratory of our institution. Forty-one (2.3%) of these patients were diagnosed as having classic mitral valve prolapse. Twenty-eight of the 41 were excluded because they had other significant structural abnormalities, were unable to stay or return for the study examination or had images unsuitable for quantitative assessment. Thus, 13 patients met the selection criteria. To compare papillary muscle tip motion in mitral valve prolapse to that observed in its absence, 18 healthy control subjects who had been recruited for projects in three-dimensional reconstruction because of excellent image quality were also examined.

The patients with mitral valve prolapse consisted of 10 women and 3 men with a mean age of 38.6 ± 17.1 years. All 13 had been referred for evaluation of auscultatory abnormalities. In 10 of these patients, the abnormality was believed to suggest the diagnosis of mitral valve prolapse (that is, a late systolic murmur or mid to late systolic click, or both). The 18 control subjects included 7 women and 11 men with a mean age of 31.2 ± 8.6 years. There was no significant difference in the mean ages of the groups ($p = 0.42$).

Echocardiographic imaging. A Hewlett-Packard phased array cardiac imaging system equipped with a 2.5- or 3.5-MHz transducer was used. To measure papillary muscle tip displacement, an apical long-axis view was obtained with the patient lying in the left lateral decubitus position. The transducer was then tilted medially to image the medial papillary muscle along its axis as well as the mitral anulus. This view was adjusted so that the motion of a particular and consistent papillary muscle tip could be observed throughout the cardiac cycle along with the mitral leaflets and mitral anulus at its greatest diameter. The apical view was used because it allows imaging of the papillary muscle-mitral anulus distance with the axial resolution of the transducer, which is superior to its lateral resolution; the medial papillary muscle could be most consistently imaged in this manner.

Measurements. To describe the relative motion of the papillary muscle tips and mitral leaflets, a common frame of reference was required. Because the mitral annular hinge points are easily defined and have been generally used to describe mitral leaflet motion, they were chosen as this frame of reference.

With a commercially available Sony off-line video analysis system, the distance from the papillary muscle tip to the line connecting the mitral annular hinge points was measured at two points: 1) at full closure of the mitral valve in early systole, and 2) in late systole at the time of maximal superior leaflet displacement (Fig. 1). The change in this distance between these two points in time was calculated and termed the *papillary muscle displacement* with respect to the anulus. This was correlated with *leaflet displacement*, which was defined as the distance from the line connecting the annular hinge points to the ventricular surface of each leaflet at its maximal superior excursion.

The observations leading to this study involved motion of the papillary muscle tip toward the mitral anulus along an apical to basal axis. To explore the logical possibility that this relative motion was caused by abnormally vigorous apical motion of the anulus, the component of annular motion along that axis was assessed relative to a fixed external frame of reference. To accomplish this, a point lying along the axis of apex to base contraction and located apical to the mitral anulus was chosen. The shortest perpendicular distance from this point to the anulus (the line connecting the mitral annular hinge points) was measured at full mitral valve closure and at the time of maximal superior leaflet displacement. The difference in these values was determined and reflected the component of annular motion along this axis in systole. (Because only the difference was desired, the exact position of the reference point along the apex to base axis was inconsequential.)

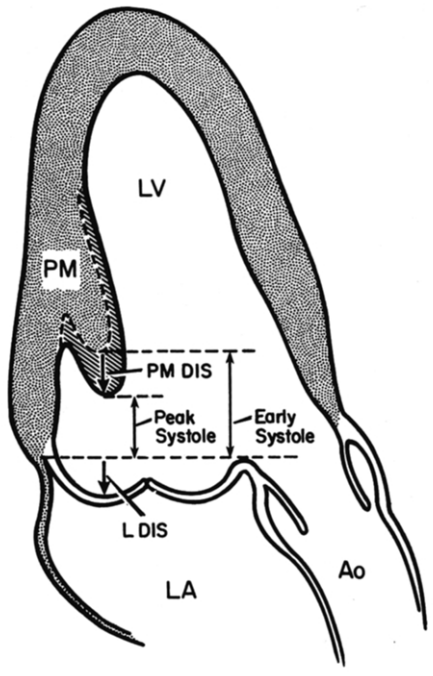


Figure 1. Diagrammatic representation of measurements made from echocardiographic images. The distance from the papillary muscle (PM) tip to the level of the mitral anulus was measured at full closure of the mitral valve in early systole and at peak systole. The difference in these measurements was calculated and termed the papillary muscle displacement (PM DIS). Ao = aorta; L DIS = leaflet displacement; LA = left atrium; LV = left ventricle.

Observer variability. Two observers measured both leaflet displacements and the distance from the papillary muscle tip to mitral anulus in both early and late systole in 10 study subjects (5 patients with mitral valve prolapse and 5 normal control subjects; a total of 30 measurements per observer). Identical video frames were used. Observer variability was assessed as the standard deviation of the differences between the measurements of the two observers. For each type of movement (leaflet and papillary muscle tip), the measurements of one observer were repeated 5 months later to assess intraobserver variability in a similar manner; this also includes the variability due to potential variation in the cycle chosen in the two measurement sessions.

Statistical analysis. Comparisons between the patients with mitral valve prolapse and control subjects were carried out using the two-tailed Student *t* test for unpaired data, with significance assessed as $p < 0.05$. The relation between papillary muscle and leaflet displacement was tested with linear regression analysis.

Results

Observations of papillary muscle motion (Fig. 2 to 4). On real time review of the video images, all normal control subjects displayed motion of the papillary muscle tip toward the apex in systole that paralleled the apical motion of the mitral anulus (Fig. 2). In contrast, patients with mitral valve prolapse displayed some degree of superior motion of the papillary muscle tip in systole that was opposite in direction to the apical motion of the mitral anulus and appeared to parallel the superior displacement of the leaflets (Fig. 3). The difference between normal subjects and patients with mitral valve prolapse is diagrammed in Figure 4.

Quantitative analysis (Fig. 5 to 7). Table 1 lists the measurements of anterior, posterior and maximal leaflet displacement, the distances between the papillary muscle tip and mitral anulus and the superior systolic displacement of

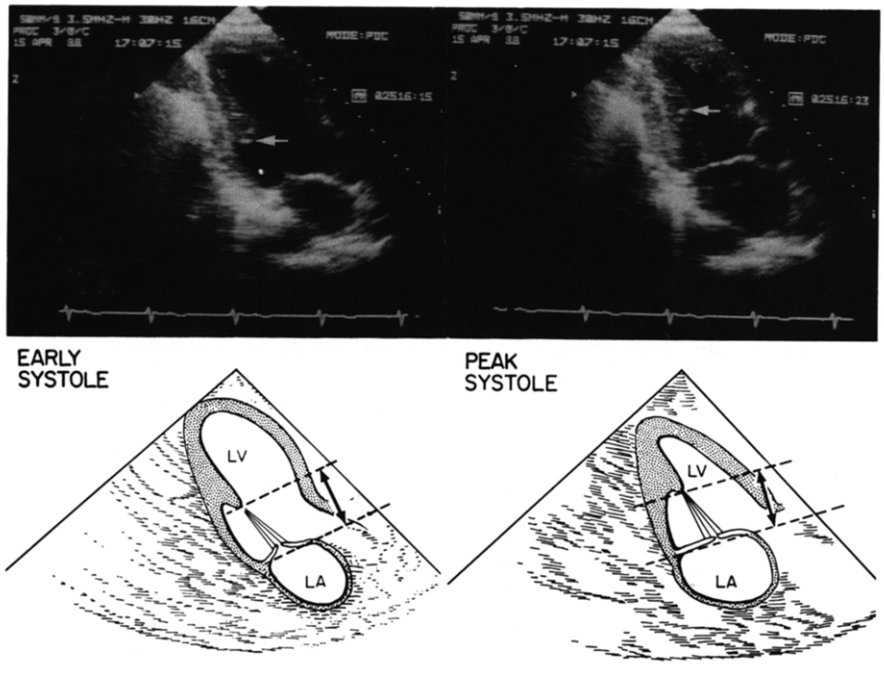


Figure 2. Still frame echocardiographic images from a study in a normal subject taken during early systole (**left**) and peak systole (**right**). The distance from the papillary muscle tip (**arrow**) to the mitral anulus does not change significantly. Abbreviations as in Figure 1.

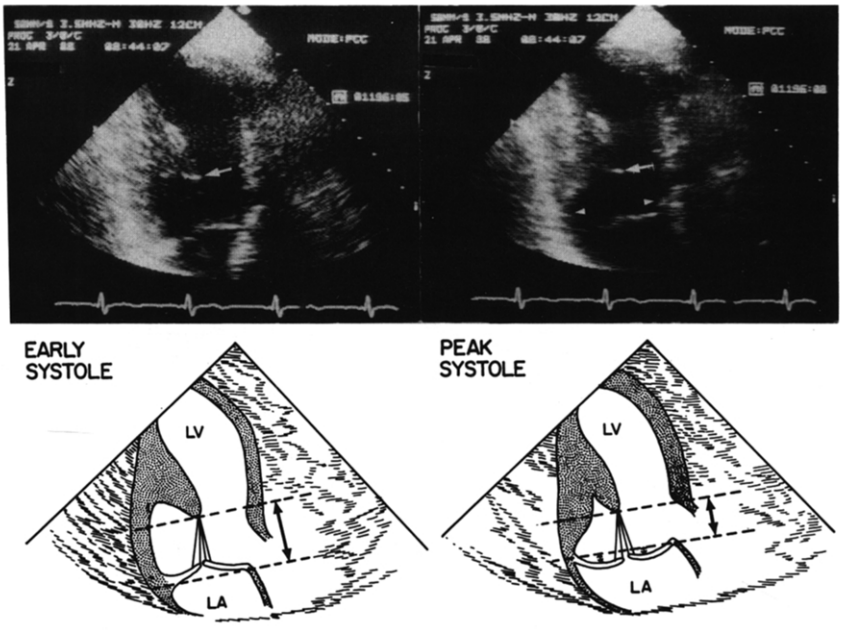


Figure 3. Still frame echocardiographic images taken during early systole (**left**) and peak systole (**right**) in a patient with classic mitral valve prolapse. The distance from the papillary muscle tip (**arrow**) to the mitral anulus decreases from early to late systole. Abbreviations as in Figure 1.

the papillary muscle tip. The distance from the papillary muscle tip to the anulus in early systole was similar in the normal subjects and the patients with mitral valve prolapse. This distance did not change significantly during systole in the normal subjects (25.2 ± 5.9 vs. 24.4 ± 5.7 mm; $p = 0.70$); however, in the patient group, this distance decreased significantly from early to peak systole, resulting in a significantly greater degree of superior papillary muscle displacement with respect to the anulus (8.5 ± 2.6 vs. 0.8 ± 0.7 mm in normal subjects; $p < 0.0001$). These differences are illustrated in Figure 5, which shows that the degree of superior displacement of the papillary muscle tips with respect to the anulus in the patient group uniformly exceeded that in normal subjects and was roughly comparable in range to the degree of superior leaflet displacement.

The relation between these two superior displacements in

Figure 4. Diagram illustrating the difference in systolic motion of the papillary muscles and mitral leaflets in normal subjects and patients with classic mitral valve prolapse (MVP). In both cases, the positions of these structures are similar in early systole. In the normal subjects, both the papillary muscle tip (**upper arrows**) and mitral anulus move apically during systole. In patients with mitral valve prolapse, similar apical annular motion occurs, but the papillary muscle tip (**upper arrows**) moves basally in parallel with the superior leaflet displacement (**lower arrows**).

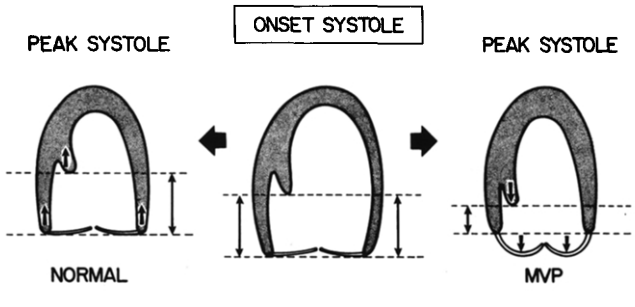
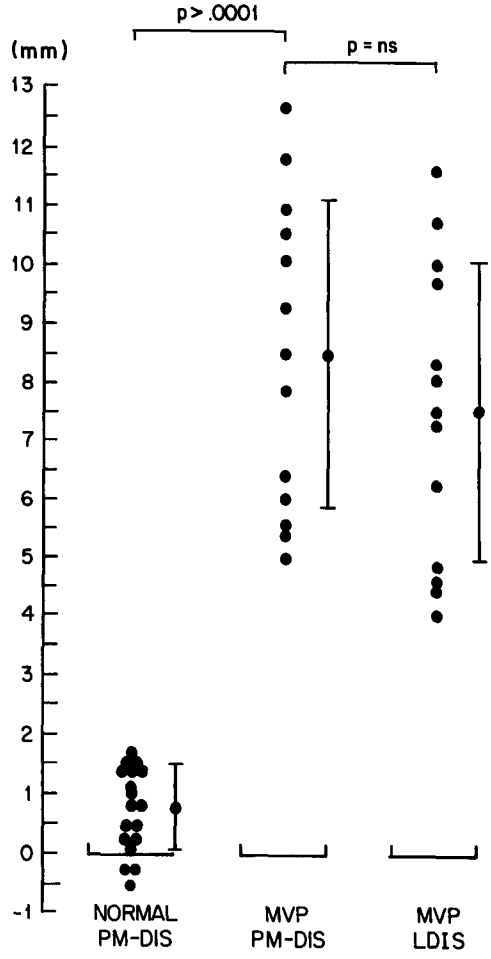


Figure 5. Papillary muscle displacement (PM-DIS) is plotted for all normal subjects (**left column**) and patients with classic mitral valve prolapse (MVP) (**center column**). The **right column** shows values for leaflet displacement (LDIS) in the patients with mitral valve prolapse, which were statistically similar in magnitude to those of papillary muscle displacement.



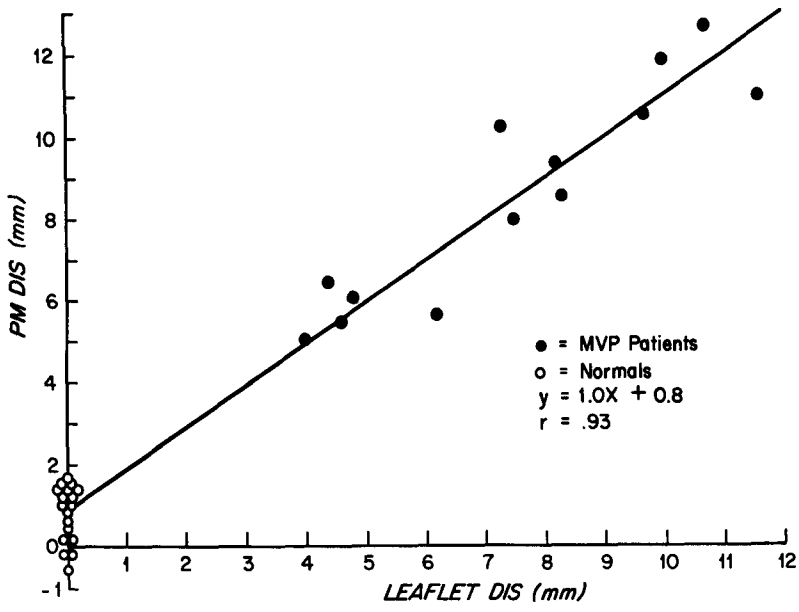


Figure 6. Plot of papillary muscle displacement (PM DIS) versus leaflet displacement (DIS) for 13 patients with mitral valve prolapse (MVP) (**closed circles**) and 18 normal subjects (**open circles**). It can be appreciated that the degree of papillary muscle displacement paralleled the degree of leaflet displacement.

individual patients is shown in Figure 6. Linear regression analysis of the data from the patients with mitral valve prolapse yielded a correlation coefficient of 0.92, a slope of 0.94 and an intercept of 1.4 mm. Including the normal control subjects in the analysis gave a correlation coefficient of 0.93, with a slope of 1.0 and an intercept of 0.8 mm.

These displacements correlated not only at peak systole, but also throughout the cardiac cycle. In the patients studied, the mitral leaflets initially coapted at the level of the anulus; only subsequently after 30% to 50% of systole had elapsed did they abruptly billow into the atrium, with parallel motion of the papillary muscle tip. This pattern is quantitatively illustrated for a patient with moderate mitral valve prolapse in Figure 7.

Annular motion. The component of annular motion toward a fixed apical reference point along the apex to base axis was similar in the normal group and in the patients with mitral valve prolapse (1.43 ± 0.4 vs. 1.55 ± 0.44 cm; p = 0.44).

Observer variability. Interobserver variability was 1.1 mm for papillary muscle tip to anulus distance and 1.2 mm for leaflet displacement measured in the same cycle. Intraobserver variabilities, which included variability resulting from potential variation in the cycles chosen for measurement, were 1.5 and 1.4 mm, respectively.

Discussion

It has been proposed that papillary muscle mechanics influence left ventricular electrophysiology (2,5) and contraction (12–17) in patients with mitral valve disease. Testing hypotheses regarding such effects in human beings requires a method for noninvasively quantitating papillary muscle motion relative to other cardiac structures, for which two-dimensional echocardiography is ideal.

Figure 7. The temporal sequence of changes in papillary muscle (PM) to anulus distance and leaflet displacement in patients with mitral valve prolapse. The two measurements are plotted throughout systole for a patient with classic mitral valve prolapse. It can be appreciated that the timing of the decrease in papillary muscle to anulus distance coincided with the leaflet displacement and, after initial normal coaptation, sudden parallel superior displacement of the papillary muscle and leaflets occurred.

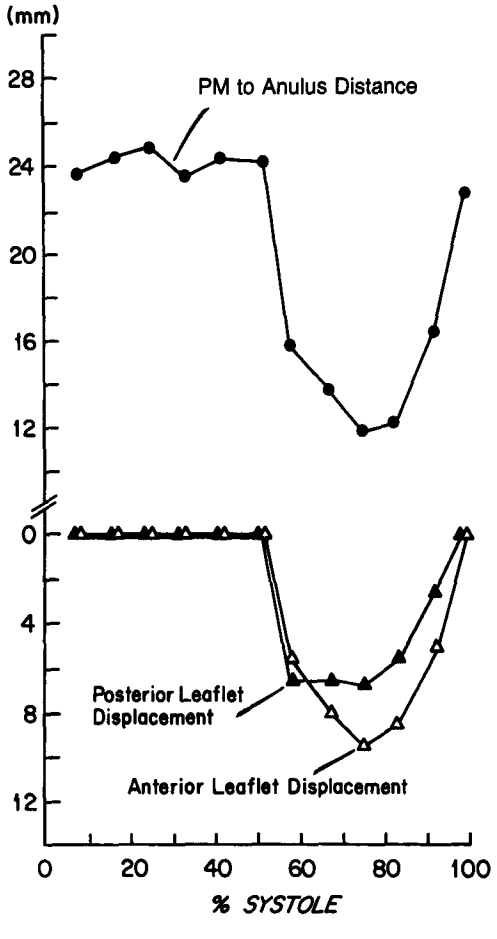


Table 1. Quantitation of Leaflet and Papillary Muscle Displacement in Normal Subjects and Patients With Mitral Valve Prolapse

	Normal Subjects	Patients With MVP
No.	18	13
Leaflet displacement (mm)		
Anterior	0	5.5 ± 2.6
Posterior	0	6.2 ± 3.3
Maximum	0	7.5 ± 2.6
PM-An distance (mm)		
Early systole	25.2 ± 5.9	25.5 ± 6.4
Peak systole	24.4 ± 5.7	16.8 ± 6.3*
PM tip displacement (mm)	0.8 ± 0.7	8.5 ± 2.6†

*$p < 0.002$ versus early systole; †$p < 0.0001$ versus normal subjects. MVP = mitral valve prolapse; PM = papillary muscle; PM-An = papillary muscle tip-anulus.

Findings in normal subjects. Although mitral annular systolic motion has been measured (22,23), the relative motion of the papillary muscle tip and anulus has not been previously described. This study demonstrates that normal left ventricular contraction is attended by parallel apical motion of the mitral anulus and papillary muscle tips. Because leaflet and chordal length are relatively fixed, maintaining a nearly constant distance between the papillary and annular attachments of these structures effectively prevents important prolapse with respect to the anulus.

Findings in mitral valve prolapse. The patients with classic mitral valve prolapse who were studied showed a comparable degree of superior systolic displacement of the papillary muscle tips and mitral leaflets relative to the mitral anulus, both at peak systole and throughout the cardiac cycle. Although these findings may not be surprising, in principle it is also conceivable that mitral valve prolapse could be accompanied by expansion of the redundant leaflets into the atrium without the observed displacement of the papillary muscle tips.

Mechanism of this motion. The motion of the papillary muscle tips clearly indicates that an unbalanced superiorly directed force acts on them, causing their traction. The parallel motion of the mitral leaflets suggests that the same force moves both these mechanically linked structures, namely, the force of left ventricular contraction exerted on the mitral leaflets, and in turn by their chordal connections, on the papillary muscles (1–7,24). Three potential factors can be suggested to explain why a net superior force occurs.

1. Increased superiorly directed force on the mitral leaflets. This force, which is proportional to the product of leaflet area and left ventricular pressure (25,26), will be increased if the area of the leaflets and anulus is increased in patients with myxomatous change (27–31).

2. Failure of coordinated and proportional papillary muscle contraction to restrain the valve. In this study, imaging was not carried out to measure papillary muscle shortening; moreover, experimental studies (32–34) have shown that normally contracting papillary muscles can be lengthened by sufficient external force. The observed normal early systolic coaptation and subsequent abrupt superior displacement of leaflets and papillary muscles (Fig. 7) suggest that at least early in systole, papillary muscle contraction prevents prolapse but subsequently yields to increased stress as left ventricular pressure increases. Prospective experimental studies (for example, with varying afterload) would be required to corroborate this reasoning.

3. Decrease in any small potential Venturi effect of left ventricular outflow. This could help maintain leaflet position away from the atrium by decreasing pressure anterior to the valve. In the prolapse setting, portions of the leaflets are further from the outflow tract and less accessible to such forces.

In any event, the observed superior motion relative to the anulus reflects the response of the papillary muscle tips to an external force that exceeds papillary muscle restraint, not a primarily increased apical excursion of the anulus (its excursion is normal) or the result of active papillary muscle motion toward the anulus (opposite to the normal direction of contraction).

Significance. Papillary muscle traction has been frequently discussed in mitral valve prolapse (2,6,7) but rarely demonstrated or measured. Invasive techniques have been thought to represent this superior motion only indirectly (6,7). To our knowledge, this is the first time that papillary muscle-mitral annular relations have been described and measured noninvasively. The ability to do this should allow testing of hypotheses regarding the significance of findings such as those described, for example, regarding the genesis of arrhythmias in mitral valve prolapse (2,5,7).

The findings of this study also suggest that the mechanical linkage between the mitral valve and left ventricular wall by chordae and papillary muscles can potentially affect the motion of linked myocardial segments. This is of relevance in view of recent studies (12–17) demonstrating the importance of papillary muscle-annular continuity in the maintenance of normal ventricular function after valve replacement.

Limitations. The results of this study cannot be generalized to all patients with superior systolic leaflet displacement. Only patients with classic mitral valve prolapse were selected for study to characterize the phenomenon of papillary muscle tip displacement because the initial qualitative observations motivating this study were made in such patients. This more severely affected group of patients appears to include those who manifest such complications as mitral regurgitation, heart failure and endocarditis (8,19,20). The occurrence and significance of papillary muscle tip displacement in other patient groups will require further prospective study of patients with mitral valve prolapse.

As noted, the demonstration of motion of the papillary muscle tips in a direction opposite to their normal contraction implies the presence of a net superiorly directed force

acting on them, but does not determine the origin of that force. Left ventricular pressure will always generate force acting in that direction because of left ventricular geometry. To what extent that force is increased and the counterbalancing force of papillary muscle contraction is either decreased or fails to increase proportionately cannot be determined from this study, but it might be learned from experimental studies that use echocardiography to observe papillary muscle tip motion.

Measurements of papillary muscle tip to annular distances can vary among observers largely because of individual variability in the choice of reference points. We found this could be minimized by 1) objective identification of the point of chordal insertion into the papillary muscle tip as a point of increased echo density that could be followed throughout systole, and 2) frame by frame review of videotape sequences to identify mitral leaflet hinge points consistently. These methods of landmark identification helped minimize observer variability.

Conclusions. The papillary muscles in normal subjects maintain a relatively constant distance between their tips and the mitral anulus. In contrast, in patients with classic mitral valve prolapse, the papillary muscle tips move superiorly with respect to the mitral anulus in parallel with leaflet displacement. This superior papillary muscle tip displacement is consistent with the existence of net superior forces acting on the papillary muscle in mitral valve prolapse. Such papillary muscle displacement may have important pathophysiologic implications in patients with mitral valve prolapse, and the mechanical continuity it demonstrates may contribute to normal left ventricular function. Two-dimensional echocardiography provides a means of measuring these relations and therefore testing hypotheses as to their pathophysiologic significance.

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