Mean Velocity of Circumferential Fiber Shortening in Prolapsed Mitral Leaflet Syndrome

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SUMMARY In 26 patients with mitral valve prolapse, ventricular function was evaluated by mean velocity of circumferential fiber shortening (MVCF) as measured along the basilar, middle and apical axes. Significantly increased rates of MVCF were found in patients with mitral prolapse along the basilar axis (1.75 ± 0.23 circ/sec) and middle axis (2.09 ± 0.34 circ/sec) (P < 0.025 and P < 0.05, respectively). Patients with mitral valve prolapse and regurgitation demonstrated a significant increase in MVCF along the basilar axes (1.72 ± 0.15 circ/sec) (P < 0.05). Asynergy appears to have a negative effect on the MVCF along the middle axis. The MVCF was found not to be related to clinical findings, symptoms or electrocardiographic changes. The mechanism for the increase in MVCF in patients with mitral valve prolapse remains unsettled.

ABNORMALITIES OF LEFT VENTRICULAR CONTRACTION are commonly found in patients with mitral prolapse. Asynergic patterns of ventricular contraction have been described and were further categorized into five patterns. Functional parameters including left ventricular end-diastolic pressure, cardiac index and ejection fraction have been found to be normal in compensated patients. However, abnormalities of left ventricular function were described in a study of patients who were in more advanced stages of the disease and the presence of abnormal findings in myocardial biopsy further supports the concept of cardiomyopathy being a component of the prolapsed mitral valve syndrome. Whether these ventricular abnormalities are a primary myocardial fault or represent the effects of chordal tugging on the papillary muscles has not been settled.

The purpose of this study was to further characterize left ventricular function by assessment of mean velocity of circumferential fiber shortening (MVCF) in the basilar, middle and apical portions of the left ventricle. The relationship of asynergy and segmental abnormalities on these measurements was also assessed. The amount of mitral regurgitation, the relation of MVCF along the three axes to the location of the insertion of the base of the papillary muscles, electrocardiogram changes and symptoms were investigated.

Subjects and Methods

This study involved 26 patients, 18 females and eight males, whose ages ranged from 15 to 65 years (mean 39 years). They were referred to Deborah Heart and Lung Center for cardiac evaluation of chest pain, dyspnea, dizziness, syncope and/or palpitations. None had a history of decomposition or were receiving cardiac drugs. All patients had systolic clicks or murmurs or both (systolic clicks in 13 and murmurs in 15 patients). The electrocardiogram demonstrated nonspecific ST and T changes in 21, left ventricular hypertrophy in two and was normal in three. In all 26 patients, a diagnosis of prolapsed mitral leaflet (PML) was established by left ventriculography, and seven of these also had tricuspid valve prolapse. Sixteen adults with suspected heart disease who were normal by catheterization and hemodynamic findings were used for comparison. None of the normal patients had clicks or murmurs.

All patients underwent combined right and left heart catheterizations and selective coronary arteriography. Left ventriculography was performed in the right anterior oblique projection at 25–30°. Angiographic recordings were made on 35 mm film at 60 frames per second. All patients were in sinus rhythm at the time of catheterization.

The cine left ventriculogram was projected on a small screen and a left ventricular profile was traced at end diastole and end systole (fig. 1), using the second sinus beat following the ventricular extrasystole if premature ventricular contractions occurred during injection, or using only sinus beats that did not immediately follow a ventricular extrasystole. The ventricular volume was measured by the area-length method of Sandler and Dodge. The long axis was measured in both systole and diastole from the mid portion of the aortic valve to the left ventricular apex. Three equidistant axes were drawn perpendicular to the long axis in systole and in diastole. Thus, the left ventriculogram was divided into basilar, middle and apical axes. The mean velocity of the circumferential fiber shortening was measured along these perpendicular axes as described by Karlner et al. In patients with mitral regurgitation, isovolumic contraction time was not subtracted. Additional hemodynamic parameters obtained included the percent of long axis shortening, ejection fraction, left ventricular end-diastolic pressure, end-diastolic volume, end-systolic volume and stroke volume.

The presence of mitral regurgitation was graded on a scale of 0 to 4+. The presence of asynergy and the insertion of the base of the papillary muscles were assessed by two independent observers. The total extent of shortening and hemi-axial shortening along the basilar, middle and apical axes were measured to assess the effect of asynergy on the MVCF. Comparisons were made in the MVCF between patients with PML with and without mitral regurgitation, and between patients with PML with and without asynergy and normals. Statistical correlations were performed using Student's t-test.

Results

The coronary arteriograms were normal. Eight patients were found to have mitral regurgitation, of whom two had
4+; three had 3+, one had 2+ and two had 1+. The mean values for ejection fraction, end-diastolic volume, end-systolic volume, percent of long axis shortening, left ventricular end-diastolic pressure for patients with and without mitral regurgitation were normal (table 1). The insertion of the base of the papillary muscles was below the basal axis in all cases. The mean stroke volume index was significantly reduced in patients with mitral prolapse, regardless of whether or not mitral regurgitation was present. The MVCF was not related to the clinical findings, symptoms or electrocardiographic changes.

In the total group of 26 patients with mitral prolapse, the basilar and middle axes demonstrated a significant increase of the MVCF (P < 0.025) (fig. 2). For those patients with mitral prolapse without mitral regurgitation, the MVCF in the basilar, middle and apical axes were 1.75 ± 0.23, 2.09 ± 0.34, and 2.15 ± 0.34 circ/sec, respectively (fig. 2 and table 1). The basilar and middle axes demonstrated a significant increase of the MVCF (P < 0.025 and P < 0.05, respectively). This finding occurred in 12 of 18 patients (66%) in the basilar axes and in nine of 18 patients (50%) in the middle axes. A reduced MVCF occurred in four of 18 patients (22%) and six of 18 (33%) of the patients with mitral prolapse in the basilar and middle axes, respectively. For those patients with mitral prolapse and regurgitation, the MVCF in the basilar, middle and apical axes were 1.72 ± 0.15, 2.05 ± 0.23, and 2.16 ± 0.21 circ/sec, respectively. In this group only the basilar axes demonstrated a significant increase of MVCF (P < 0.05). This occurred in five of eight patients (62%) in the basilar axes and five of eight (62%) in the middle axes. A reduced MVCF occurred in 12% and 25% of the patients with mitral prolapse and regurgitation in the basilar and middle axes, respectively. In patients with and without mitral regurgitation, the apical portion did not demonstrate a significant increase in the MVCF (fig. 2).

Asynergy was seen in 10 patients, involving the middle anterior portion of the left ventricle. The patients with mitral valve prolapse without asynergy, when compared to our normals, demonstrated a significant increase in the MVCF in the basilar and middle axes (P < 0.05). In those with both mitral prolapse and asynergy, however, significant increase in the MVCF was only seen in the basilar axes (P < 0.025) (table 2).

The extent of shortening of patients with PML was increased in the basilar axis (P < 0.01), but was not significantly increased in the middle and apical axes. The middle anterior hemi-axes of PML with asynergy did show a significant reduction in the extent of shortening (P < 0.05), but this was compensated by an increase in the extent of shortening in the middle posterior hemi-axes (P < 0.05).

**Table 1. Hemodynamic Parameters in Patients with Mitral Prolapse and in Normals**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>EF (M ± SD)</th>
<th>EDV (range)</th>
<th>ESV (range)</th>
<th>SV index (range)</th>
<th>% Long axis shortening</th>
<th>LVEDP</th>
<th>MVCF basilar axis</th>
<th>MVCF middle axis</th>
<th>MVCF apical axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>.77</td>
<td>49.1-112.1</td>
<td>9.2-21.5</td>
<td>39.9-90.6</td>
<td>27.9</td>
<td>5.9</td>
<td>1.32</td>
<td>1.73</td>
<td>2.03</td>
</tr>
<tr>
<td></td>
<td>±.1</td>
<td></td>
<td></td>
<td></td>
<td>±10.8</td>
<td>±4.1</td>
<td>±12</td>
<td>±14</td>
<td>±15</td>
</tr>
<tr>
<td>Prolapse without MR</td>
<td>.76</td>
<td>41.5-109.5</td>
<td>9-24.2</td>
<td>32.5-85.3</td>
<td>26</td>
<td>5.1</td>
<td>1.75</td>
<td>2.09</td>
<td>2.15</td>
</tr>
<tr>
<td></td>
<td>±.12</td>
<td></td>
<td></td>
<td></td>
<td>±15</td>
<td>±5.5</td>
<td>±23</td>
<td>±34</td>
<td>±34</td>
</tr>
<tr>
<td>Prolapse with MR</td>
<td>.75</td>
<td>40.1-110</td>
<td>8.7-43.3</td>
<td>31.4-66.8</td>
<td>22</td>
<td>7.0</td>
<td>1.72</td>
<td>2.05</td>
<td>2.16</td>
</tr>
<tr>
<td></td>
<td>±.08</td>
<td></td>
<td></td>
<td></td>
<td>±3</td>
<td>±5.6</td>
<td>±15</td>
<td>±23</td>
<td>±21</td>
</tr>
</tbody>
</table>

**Abbreviations:** EDV = end-diastolic volume (cc/beat/m²); EF = ejection fraction; ESV = end-systolic volume (cc/beat/m²); LVEDP = left ventricular end-diastolic pressure (mm Hg); MVCF = mean velocity of circumferential fiber shortening (circ/sec) mean ± sem; SV index = stroke volume index (cc/beat/m²); MR = mitral regurgitation.
**Discussion**

Although left ventricular asynergies are often a feature of prolapsed mitral leaflet syndrome, the functional aspects of systolic contraction have not been fully defined. Nutter et al., in studying 26 patients with mitral valve prolapse, found seven with normal ventricular contraction, eight with inferior deformity and/or anterior asynergy and 11 with hyperkinesia. The results of our study suggest that the basilar and middle axes of the left ventricle in patients with mitral prolapse demonstrated an increased rate of contraction, as determined by the MVCF (P < 0.025). These results are not consistent with those of Liedtke et al., who found a reduction of the MVCF in the basilar segment, nor with Gulotta et al., who, studying patients with advanced disease, found a reduction in the middle axes of the left ventricle. Nakhjavan et al. found a reduction of the MVCF in the basilar and middle axes in 30% of mitral prolapse patients. This is consistent with our findings, although the majority of our patients had a significant increase of the MVCF in these axes. It appears that hyperkinetic component is not uniform for the syndrome, since 30% of the patients had a reduced MVCF. PML syndrome represents a wide spectrum disease and patients may be studied at different stages. The MVCF and the extent of shortening appear to be negatively influenced in patients with mitral valve prolapse having asynergy as compared to normals. This was demonstrated by the significant reduction in the extent of shortening in the middle anterior semi-axes (anterior wall asynergy) and the failure to demonstrate a significant increase in the overall MVCF in patients with PML and asynergy along the middle axes. This influence, however, seems to affect the middle axis predominantly, since significant increases in MVCF in patients with PML occurred in the basilar axes despite asynergy.

The stroke volume index in the patients both with and without mitral regurgitation was significantly less than in our normal subjects. The reason and importance of this finding remain to be determined.

As shown in this study, the mean velocity of circumferential fiber shortening was significantly increased in the basilar and middle segments, as compared to normal subjects. It is not known whether the increase in MVCF is peculiar to patients with PML or whether it may occur in other disease states with or without asynergy.

Whether the increase in MVCF is due to hyperkinetic myocardial component, or is due to tugging of the chordae on the middle axis through the papillary muscles, or due to tugging of the anular attachment of the leaflets on the basilar axes, or whether the increase in MVCF is a combination of these mechanisms remains unsettled.

**Acknowledgment**

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**References**

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