An Appraisal of Mitral Valve Echocardiograms Mimicking Mitral Stenosis in Conditions with Right Ventricular Pressure Overload

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SUMMARY

Of seven patients with pulmonary hypertension, two had marked and one had moderate retardation of the echocardiographic anterior mitral valve leaflet closing motion, suggestive of mitral stenosis. In each patient, however, mitral stenosis was excluded by hemodynamic or anatomic criteria. The reason for the abnormal mitral valve motion was considered to be a reduced rate of left ventricular diastolic filling. In the group of seven patients it was noted that the ratio of ventricular filling pressures (RVedP/LVedP) was inversely correlated with the anterior mitral valve leaflet closing slope. This observation supports the hypothesis that left ventricular diastolic filling may be secondarily altered by right ventricular overload, and that this impairment of left ventricular filling may result in alteration of the anterior mitral valve leaflet echogram mimicking mitral stenosis.

Additional Indexing Words:
Pulmonary hypertension

MITRAL VALVE echocardiography has proven to be an exceptionally useful technique in the evaluation of patients with suspected mitral stenosis. A reduced diastolic closing slope of the anterior leaflet of the mitral valve is the characteristic finding,\(^1\,2\) and the slope of this motion can be taken as an approximate index of the severity of mitral valve obstruction.\(^1\,3\) Other conditions that may retard the diastolic downslope of mitral valve closing motion and lead to confusion in diagnosis include left atrial myxoma,\(^4\,5\) hypertrophic subaortic stenosis,\(^6\,7\) and severe left ventricular hypertrophy due to aortic stenosis\(^8\) or nonobstructive cardiomyopathy.\(^9,10\) Since it has been suggested that mitral valve echocardiography is a significant tool in the diagnosis of "silent" mitral stenosis,\(^4\) it is important that conditions which may produce mitral valve echograms mimicking mitral stenosis be brought to attention. We report seven patients with pulmonary hypertension, two of whom had mitral valve echograms suggesting mitral stenosis. Mitral valve disease was excluded in each case by cardiac catheterization. A theory relating the abnormal mitral valve motion to decreased left ventricular diastolic filling is proposed.

Methods

Echocardiographic examination of the aortic root, mitral and tricuspid valves, right and left ventricles, and interventricular septum was carried out according to previously described techniques.\(^1,2,14-16\) A Smith Kline Ekoline-20 ultrasonoscope with a 2.25 megahertz transducer of 0.5 inch diameter was used in all examinations. All results for mitral valve motion are the mean value for five measurements. Cardiac catheterization was performed in all patients using standard retrograde techniques.

Results

Echocardiograms from a normal patient and a patient with documented mitral stenosis are illustrated for reference in figure 1. Hemodynamic and echocardiographic data for the seven study patients...
are summarized in Table 1. The anterior mitral valve leaflet diastolic closing motion (E to F velocity) was markedly retarded in patients 1 and 2, and reduced in patient 3. Early diastolic opening velocity of the anterior leaflet (D to E velocity) was decreased in patients 1, 2, and 3.

Right ventricular filling pressure exceeded left ventricular filling pressure by more than 2:1 in patients 1, 2, and 3. Linear regression analysis of the E to F velocity against the ratio of ventricular filling pressures (RVedP/LVedP) for the entire group showed a strong inverse correlation ($r = -0.94$).

Case histories and echocardiograms of patients 1 and 2, in whom the anterior mitral valve leaflet echogram suggested mitral stenosis, are presented in detail below.

### Table 1

**Hemodynamic and Echocardiographic Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>MVE (D to E)</th>
<th>D to E velocity</th>
<th>E to F velocity</th>
<th>RA mean mmHg</th>
<th>RV mmHg</th>
<th>PA mmHg</th>
<th>LV mmHg</th>
<th>RVedP/LVedP</th>
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<tbody>
<tr>
<td>1</td>
<td>15</td>
<td>155</td>
<td>10</td>
<td>14</td>
<td>90/28</td>
<td>90/40</td>
<td>95/8</td>
<td>3.5</td>
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<tr>
<td>2</td>
<td>14</td>
<td>128</td>
<td>10</td>
<td>5</td>
<td>80/12</td>
<td>80/26</td>
<td>105/5</td>
<td>2.4</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>204</td>
<td>44</td>
<td>20</td>
<td>57/24</td>
<td>57/23</td>
<td>125/10</td>
<td>2.4</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>327</td>
<td>77</td>
<td>12</td>
<td>88/14</td>
<td>88/30</td>
<td>138/15</td>
<td>1</td>
</tr>
<tr>
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<td>20</td>
<td>348</td>
<td>78</td>
<td>10</td>
<td>74/16</td>
<td>74/32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>19</td>
<td>315</td>
<td>80</td>
<td>8</td>
<td>82/16</td>
<td>82/33</td>
<td>138/15</td>
<td>1</td>
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<td>352</td>
<td>89</td>
<td>5</td>
<td>63/8</td>
<td>63/29</td>
<td>122/14</td>
<td>.6</td>
</tr>
</tbody>
</table>

Abbreviations: MVE = Mitral valve excursion, RA = right atrium, RV = right ventricle, PA = Pulmonary artery, LV = Left ventricle, edP = end diastolic pressure.

* = pulmonary capillary wedge pressure.
Case Reports

Patient 1

A 40-year-old woman with a history of possible acute rheumatic fever in childhood was hospitalized in late 1972 because of sharp recurrent chest pain, cough, shortness of breath, dizziness, and paroxysmal nocturnal dyspnea. In 1965 generalized sarcoidosis with involvement of lung, liver, spleen, and skin had been diagnosed. In 1970 she developed predominantly right sided congestive heart failure with ascites, hepatomegaly, and peripheral edema which responded to appropriate therapy.

Physical examination revealed fine rales and expiratory wheezes over both lungs. A normal carotid pulse was palpated, but a prominent a-wave was present in the jugular vein pulsations. Along the left sternal edge a prominent systolic heave was present. The first heart sound ($S_1$) was normal. The pulmonic component of the second heart sound ($P_2$) was accentuated. Atrial and ventricular gallop sounds, thought to originate from the right ventricle, were present. A grade 3/6 systolic ejection murmur was maximal at the lower left sternal edge. Hepatomegaly, moderate ascites, and 2+ pitting edema of the lower extremities were present.

The chest X-ray showed a diffuse fibronodular infiltrate throughout both lungs. There was cardiomegaly with predominant right ventricular enlargement. The left atrium did not appear significantly enlarged. Electrocardiogram showed sinus rhythm, P waves suggesting right atrial enlargement, right axis deviation, and large R waves in the right precordial leads compatible with right ventricular hypertrophy.

Echocardiogram

Figure 2 is the mitral valve echocardiogram from patient 1 and appears similar to that from the patient with mitral stenosis, illustrated in figure 1B. The excursion of the anterior mitral valve leaflet, measured from D to E, is 15 mm (normal 18 to 25 mm), and the diastolic downslope of mitral valve

![Figure 2](image_url)

Mitral valve echogram from patient 1 showing absence of the rapid early diastolic closing motion, similar to figure 1B. The systolic portion of the echogram is partially obscured by the dense echo signals from the left atrial wall seen posteriorly. Although the septum appears to move paradoxically in this figure, other echocardiograms obtained in a more appropriate plane for evaluation of septal motion showed it to move normally.
Table 2

Hemodynamic Data

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic blood flow (liters/min)</td>
<td>3.7</td>
<td>4.4</td>
</tr>
<tr>
<td>LV stroke volume (cc/beat)</td>
<td>27</td>
<td>41</td>
</tr>
<tr>
<td>Pulmonary blood flow (liters/min)</td>
<td>3.7</td>
<td>4.0</td>
</tr>
<tr>
<td>Right atrial pressure (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a wave</td>
<td>21</td>
<td>10</td>
</tr>
<tr>
<td>v wave</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>mean</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>Right ventricular pressure (mm Hg)</td>
<td>90/28</td>
<td>80/12</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td>90/40</td>
<td>80/26</td>
</tr>
<tr>
<td>Left atrial pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a wave</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>v wave</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>mean</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Left ventricular pressure (mm Hg)</td>
<td>95/8</td>
<td>105/5</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (dynes-sec-cm⁻²)</td>
<td>1059</td>
<td>848</td>
</tr>
</tbody>
</table>

Closing motion is only 10 mm/sec which is severely retarded. It was not possible to record the posterior leaflet with sufficient diagnostic accuracy. The right ventricular end-diastolic internal diameter is increased to 24 mm (normal in our laboratory is 12 to 18 mm). A diagnosis of mitral stenosis was suggested by the abnormal mitral valve echogram, and the other findings indicated an enlarged, dilated right ventricle.

Cardiac Catheterization

On the basis of the clinical picture and the echocardiogram, the patient underwent cardiac catheterization. Standard right heart, retrograde left ventricular, and transeptal left atrial approaches were used. Hemodynamic data are presented in table 2. Severe pulmonary hypertension was documented, but no diastolic gradient across the mitral valve was found.

A left ventricular biplane angiogram was done after left atrial injection of contrast media. Representative mid-diastolic films are shown in figure 3. A ventricular volume curve calculated using the area-length method of Dodge is illustrated in figure 4. Left ventricular chamber volume is decreased, stroke volume is low, and a slow rate of diastolic filling is present.

Figure 3

Representative anterior-posterior (A-P) and lateral films taken during the angiographic volume study. The films correspond to the mid-diastolic volume measurements. LA = left atrium, LV = left ventricle, RV = right ventricle. Note the large area corresponding to the right ventricle in the lateral film. The vertical interface between LV and RV corresponds to the interventricular septum which is displaced posteriorly so that it occupies a plane vertical to the film.
PATHOLOGY

Four days after cardiac catheterization the patient suffered a cardio-respiratory arrest and could not be resuscitated. The heart weighed 370 grams at post mortem examination. The left ventricle was small and had an average wall thickness of 9 mm. The right ventricle was hypertrophied and enlarged with an average wall thickness of 11 mm. The interventricular septum was hypertrophied, displaced leftward and posteriorly, encroaching upon the usual area of the left ventricle. The mitral valve appeared normal to both gross and microscopic examination, and no myocardial sarcoidosis was found on histological section. The final pathologic diagnosis was generalized sarcoidosis with severe pulmonary involvement and pulmonary hypertension.

PATIENT 2

A 52-year-old woman with a three-year history of progressive dyspnea and intermittent pleuritic pain was hospitalized in 1973 for evaluation of evanescent pulmonary infiltrates. Detailed clinical and laboratory evaluation failed to disclose evidence of either an infectious or collagen-vascular disease.

Physical exam revealed clear lung fields. The carotid pulse was normal to palpation. Prominent a-waves were present in the jugular vein pulsations. A slight left parasternal lift was present. S1 was normal. P2 was increased. A grade 2/6 systolic ejection murmur was present in the pulmonic area. No diastolic murmurs were present. Hepatomegaly and slight edema of the lower extremities were present.

Chest X-ray showed several small cavitary lesions in the right middle and left upper lobes. Moderate right ventricular enlargement was noted. Electrocardiogram showed marked right axis deviation, prominent R waves in the right precordial leads, and ST-T wave changes in leads V1-V6, all compatible with right ventricular hypertrophy.

ECHOCARDIOGRAM

A representative mitral valve echocardiogram is shown in figure 5. Anterior mitral valve leaflet excursion (D to E) is 14 mm, which is significantly decreased. The diastolic downslope (E to F), severely retarded, is 10 mm/sec. Multiple measurements taken on other polaroids which better illustrated the anterior leaflet E to F slope, ranged from 10 to 15 mm/sec. In figure 5, the posterior mitral valve leaflet echocardiogram can be seen to move normally. Figure 6 shows the echocardiogram of the interventricular septum and left ventricular posterior wall. The right ventricular diameter, estimated by measuring from the right side of the septum to a point 5 mm below the nonmoving chest wall echoes at end diastole, is increased to approximately 25 mm. The left ventricular end diastolic and end systolic diameters are decreased to 29 and 18 mm respectively. The diagnosis of mitral stenosis, suggested by the abnormal anterior leaflet echocardiogram, is not substantiated by the posterior leaflet echocardiogram which is normal. The other findings indicate an enlarged, dilated right ventricle and a small left ventricle.

CARDIAC CATHETERIZATION

Hemodynamic data are presented in table 2. The catheter passed easily from right atrium to left atrium and a small right to left shunt was demonstrated by indicator dilution curves. Severe pulmonary hypertension was present. No diastolic gradient across the mitral valve could be demonstrated on multiple continuous pressure recordings during pull back of the catheter from left ventricle to left atrium. A pulmonary angiogram showed changes of severe pulmonary hypertension; a left ventricular angiogram was not done. Diagnoses were severe pulmonary hypertension, etiology undetermined; patent foramen ovale with a small right to left shunt at the atrial level, and right ventricular failure.

PATHOLOGY

One week after pulmonary angiography the patient died. Autopsy showed recent and old pulmonary emboli. The heart was grossly normal with both left and right ventricular wall thicknesses normal. The mitral valve was normal to both gross and microscopic examinations.
Figure 5

Mitral valve echogram from patient 2. The anterior leaflet motion is decreased and the closing slope is retarded to superficially resemble mitral stenosis. The normal posterior leaflet motion (denoted by the arrow) rules out organic mitral stenosis.

Discussion

The rate of diastolic closure of the anterior leaflet of the mitral valve as seen on the echocardiogram depends on multiple factors. The degree of valve thickening and rigidity, along with the extent of shortening of the chordae tendineae, may mechanically retard the rate of diastolic closure in some patients. Hemodynamically, the rate of diastolic closure is a function of the duration of a positive gradient across the valve and the rate of left ventricular filling during diastole. This latter explanation is generally accepted as the more important mechanism. Both of these factors, mechanical and hemodynamic, could be operative in the situation of left atrial myxoma producing a mitral valve echogram mimicking mitral stenosis. The hemodynamic explanation, a decreased rate of diastolic filling, has been used to account for the abnormal mitral valve echograms seen in cases of hypertrophic subaortic stenosis or severe left ventricular hypertrophy. It has been theorized that in these conditions left ventricular compliance is low, the rate of diastolic filling diminished, and the duration of significant filling prolonged. Zaky, Nasser and Feigenbaum, in considering movements of the mitral valve and mitral ring, have pointed out that the two interrelate. A decrease in diastolic filling with a resultant decrease in mitral ring movement would necessarily result in decreased mitral valve excursion as seen in patients 1, 2, and 3, although it should not specifically affect the early diastolic descent of the leaflet.

A similar hemodynamic explanation is likely in the cases presented in this report. In the first patient.
the left atrium and left ventricle can be seen to be quite small (fig. 3); the left ventricular volume curve indicates the absence of rapid early diastolic filling and further illustrates that significant flow across the mitral valve continued throughout diastole (fig. 4). The left ventricle was small in comparison to the right ventricle and left ventricular geometry was abnormal. This change in ventricular geometry, resulting from the enlarged right ventricle displacing the interventricular septum leftward and posteriorly, is illustrated in figure 3.

Although left ventricular angiography was not performed in the second case, the echocardiogram illustrates that the right ventricular internal diameter was significantly increased while the left ventricular internal diameter was abnormally decreased (fig. 6). An unusually close relationship between the interventricular septum and anterior mitral leaflet can be seen in idiopathic hypertrophic subaortic stenosis and in ostium primum atrial septal defect. In both of these conditions the left ventricular outflow tract is narrowed. In patient 2 the heart was normal at autopsy with no evidence of septal hypertrophy or abnormality of the mitral valve. The close relationship of the anterior mitral leaflet and the interventricular septum in patient 2 can be explained by hypothesizing that during life the right ventricle was dilated by the severe pressure overload and this process displaced the septum posteriorly into the usual confines of the left ventricle much as was found in patient 1.

The rate of diastolic opening of the anterior leaflet (D to E velocity) was decreased in the three patients with the lowest closing slopes (E to F velocity), further evidence of a decreased rate of
left ventricular diastolic filling. The factors contributing to this abnormal mitral valve diastolic motion were sought by comparing the hemodynamic data of these patients. A marked difference is apparent if the ratio of right and left ventricular end diastolic pressures in the patients with abnormally moving mitral valves are compared to those with normal mitral valve motion. In all three patients with decreased mitral valve movement, the RVedP/LVedP ratio was 2.4 or greater indicating a significant disparity between the diastolic filling pressures in the two ventricles. These findings suggest the hypothesis that patients with right ventricular pressure overload may develop impairment of early diastolic left ventricular filling, and this impaired filling may result in alteration of the mitral valve echogram mimicking mitral stenosis.

It has been demonstrated in dogs that isolated right ventricular pressure and volume overload will alter the diastolic pressure-volume relationship of the left ventricle in such a fashion as to suggest a decrease in left ventricular diastolic compliance. This change was found only when the right ventricle was overloaded and could be produced acutely by increasing the right ventricular pressure and volume in previously normal canine hearts. This suggests that left ventricular pressure-volume relations in diastole can be altered by changes in ventricular geometry independent of changes occurring in the myocardium. As noted above the patients with abnormal mitral valve diastolic motion had right ventricular diastolic filling pressures significantly higher than the left ventricular filling pressures. In addition each patient had either angiographic or echocardiographic evidence of right ventricular chamber enlargement exceeding any change in left ventricular chamber size. It can be speculated that the rate of left ventricular diastolic filling in the patients with depressed mitral valve motion could have been decreased by the abnormal spatial and hemodynamic relationships between the right and left ventricles during diastole which resulted from the right ventricular overload. Whether or not the reduced left ventricular diastolic filling resulted from a greater degree of distortion of left ventricular geometry or from some alteration in the actual diastolic compliance of the left ventricular myocardium cannot be discerned from the available clinical data.

One criticism that might be directed to the above outlined hypothesis is that patients 1 and 2 had rapid heart rates which could have decreased the E to F slope of the anterior mitral valve leaflet. Although an inverse relationship between E to F slope and heart rate has been documented by Buyukozturk, Kingsley and Segal, marked diminution of the degree seen in patients 1 and 2 was not reported. The rate of mitral valve opening (D to E) was not related to heart rate in their study.

Duchak, Chang and Feigenbaum noted that the echocardiogram of the posterior mitral leaflet could be used to discern the presence or absence of true mitral stenosis in cases where the anterior mitral leaflet had a retarded diastolic closing motion. In mitral stenosis both leaflets show abnormal diastolic movement, whereas the posterior leaflet moves normally during diastole in circumstances where anterior leaflet motion is retarded due to causes other than mitral stenosis. One of the cases they used to illustrate this point had pulmonary hypertension and was similar to the patients of our report.

In summary, of seven patients with severe pulmonary hypertension, two had marked and one had moderate retardation of the anterior mitral valve leaflet closing slope, unassociated with organic mitral stenosis. A theory relating the abnormal mitral valve motion to decreased diastolic flow across the mitral valve is proposed. Caution should be exercised in making the diagnosis of "silent" mitral stenosis based on the echocardiographic appearance of the anterior mitral valve leaflet alone.

References

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