Diastolic Vibration of the Interventricular Septum in Aortic Insufficiency


SUMMARY
The echocardiograms of 46 patients with aortic insufficiency which manifested typical high frequency vibrations of the anterior mitral valve leaflet were reviewed. All patients had overt clinical evidence of aortic insufficiency which was confirmed by angiography in 17 cases.

In 17 cases (eight of whom underwent catheterization and angiography), high frequency diastolic vibrations of the interventricular septum of similar frequency to those on the anterior mitral leaflet were observed. This finding was not seen in 100 control echocardiograms from patients without clinical evidence of aortic insufficiency, and represents a previously undescribed echocardiographic manifestation of this lesion.

Additional Indexing Words:
Echocardiography

HIGH FREQUENCY diastolic vibration of the anterior mitral valve leaflet is a common echocardiographic finding in patients with aortic insufficiency.1,2 This report describes an additional echocardiographic finding, high frequency posterior septal wall vibration in patients with overt clinical evidence of aortic insufficiency. To our knowledge, such septal vibration has not been previously reported.

Methods

Patients
The echocardiograms of 46 patients with overt clinical evidence of aortic insufficiency which manifested typical high frequency vibrations of the anterior mitral leaflet were reviewed for evidence of similar high frequency septal wall vibrations. Since echoes arising from the posterior interventricular septum may be nonlinear, particularly in early diastole, septal vibration was defined only when the frequency and amplitude of the septal vibrations were similar to those generally accepted as abnormal for the anterior mitral leaflet in aortic insufficiency. The high frequency of such vibrations is easily distinguishable from the coarse vibrations of the anterior mitral leaflet seen in atrial fibrillation.2

Patients with isolated mitral leaflet vibration were placed in group I, while those with both mitral and septal vibrations were placed in group II. Seventeen of the 46 patients underwent cardiac catheterization for symptoms related to valvular heart disease (congestive cardiac failure, chest pain, or syncope). At catheterization, aortic insufficiency was graded mild, moderate, or severe based on the degree of valvular regurgitation noted on cineaortogram.

In addition, 100 echocardiograms of patients without clinical evidence of aortic insufficiency were reviewed for the presence of high frequency mitral and/or septal vibration.

Technique
Echocardiograms were performed with patients in the recumbent or left lateral positions using a Unirad Series "C" ultrasonoscope and Tektronix graphic recorder. Echocardiograms of the aorta, aortic valve, mitral valve, interventricular septum, and posterior left ventricular wall were obtained in all patients, using a 2.25 MHz, 7.5 cm focused transducer according to standard technique.3

Results
There were 29 patients in group I and 17 patients in group II. A representative echocardiogram from a patient in group I (without septal vibration) is shown in figure 1. This echocardiogram demonstrates the high frequency vibrations of the anterior mitral leaflet seen in aortic insufficiency. It further demonstrates the nonlinear early diastolic echoes of the posterior septal wall that may be confused with the high frequency vibrations seen in patients in group II.

No high frequency septal or mitral valve vibrations were noted in the 100 control patients without clinical evidence of aortic insufficiency.
Nine patients in group I underwent cardiac catheterization. Aortic regurgitation estimated by cineaortography was severe in four patients, moderate in three, and mild in two. Eight patients in group II underwent catheterization; of these, five had severe, one moderate, and two mild aortic regurgitation. Clinical and angiographic data on the patients who were catheterized are shown in table 1. The remaining
Table 1

Clinical and Angiographic Findings in Catheterized Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Etiology</th>
<th>Clinical diagnosis</th>
<th>BP (mm Hg)</th>
<th>Angiographic findings</th>
<th>Other cath findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.H.</td>
<td>F</td>
<td>51</td>
<td>SBE</td>
<td>AI</td>
<td>130/60</td>
<td>AI, moderate</td>
<td>No other valvular lesion</td>
</tr>
<tr>
<td>D.A.</td>
<td>M</td>
<td>17</td>
<td>Congenital</td>
<td>AS, AI</td>
<td>100/80</td>
<td>AI, mild</td>
<td>Severe AS, peak systolic gradient 90 mm Hg</td>
</tr>
<tr>
<td>W.F.</td>
<td>M</td>
<td>41</td>
<td>Unknown</td>
<td>AS, AI</td>
<td>130/60</td>
<td>AI, severe</td>
<td>No AS, no other valve lesion</td>
</tr>
<tr>
<td>F.T.</td>
<td>M</td>
<td>38</td>
<td>Rheumatic</td>
<td>AS, AI</td>
<td>110/50</td>
<td>MI, mild</td>
<td>Severe AS, peak systolic gradient 90 mm Hg</td>
</tr>
<tr>
<td>W.R.</td>
<td>M</td>
<td>65</td>
<td>Unknown</td>
<td>AS, AI heart block</td>
<td>110/70</td>
<td>MI, mild</td>
<td>Severe AS, 90% obstruction of proximal LAD</td>
</tr>
<tr>
<td>E.H.</td>
<td>F</td>
<td>63</td>
<td>Unknown</td>
<td>AS, AI</td>
<td>130/60</td>
<td>AI, moderate</td>
<td>Moderate AS, peak systolic gradient 48 mm Hg</td>
</tr>
<tr>
<td>E.McG.</td>
<td>M</td>
<td>35</td>
<td>Rheumatic</td>
<td>AS, AI MI</td>
<td>140/40</td>
<td>AI, severe</td>
<td>Mild AS, peak systolic gradient 25 mm Hg</td>
</tr>
<tr>
<td>E.E.</td>
<td>F</td>
<td>60</td>
<td>Unknown</td>
<td>AS, AI</td>
<td>140/100</td>
<td>AI, mild</td>
<td>Severe AS, peak systolic gradient 130 mm Hg</td>
</tr>
<tr>
<td>W.W.</td>
<td>F</td>
<td>60</td>
<td>Unknown</td>
<td>AI</td>
<td>150/80</td>
<td>AI, severe</td>
<td>No other valvular lesion</td>
</tr>
</tbody>
</table>

**Group II**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Etiology</th>
<th>Clinical diagnosis</th>
<th>BP (mm Hg)</th>
<th>Angiographic findings</th>
<th>Other cath findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>H.Y.</td>
<td>F</td>
<td>24</td>
<td>Rheumatic</td>
<td>MS, AI</td>
<td>110/55</td>
<td>AI, severe</td>
<td>Mild MS, mild AS</td>
</tr>
<tr>
<td>A.M.</td>
<td>F</td>
<td>52</td>
<td>Rheumatic</td>
<td>MS, AI</td>
<td>130/80</td>
<td>AI, moderate</td>
<td>Moderate MS</td>
</tr>
<tr>
<td>M.C.</td>
<td>F</td>
<td>49</td>
<td>Rheumatic</td>
<td>MS, MI AS, AI</td>
<td>140/80</td>
<td>MI, moderate</td>
<td>Mild AS, moderate MS</td>
</tr>
<tr>
<td>C.M.</td>
<td>F</td>
<td>23</td>
<td>Congenital</td>
<td>AI</td>
<td>135/55</td>
<td>AI, severe</td>
<td>No other valvular lesions</td>
</tr>
<tr>
<td>R.B.</td>
<td>M</td>
<td>32</td>
<td>Rheumatic</td>
<td>MS, AI</td>
<td>100/60</td>
<td>AI, mild</td>
<td>Severe MS</td>
</tr>
<tr>
<td>M.F.</td>
<td>F</td>
<td>63</td>
<td>Rheumatic</td>
<td>MS, AI</td>
<td>120/80</td>
<td>AI, mild</td>
<td>Moderate MS</td>
</tr>
<tr>
<td>M.S.</td>
<td>M</td>
<td>30</td>
<td>Congenital</td>
<td>AI</td>
<td>130/70</td>
<td>AI, severe</td>
<td>No other valvular lesions</td>
</tr>
<tr>
<td>P.D.</td>
<td>F</td>
<td>37</td>
<td>Rheumatic</td>
<td>AS, AI MS, MI</td>
<td>130/55</td>
<td>AI, severe</td>
<td>No AS, moderate MS</td>
</tr>
</tbody>
</table>

Abbreviations: MS = mitral stenosis; MI = mitral insufficiency; AS = aortic stenosis; AI = aortic insufficiency; BP = blood pressure; SBE = subacute bacterial endocarditis; LAD = left anterior descending coronary artery.

patients in the study did not undergo cardiac catheterization, but all had a clinical diagnosis of valvular heart disease including aortic regurgitation.

The echocardiographic findings in three typical cases from group II are described.

**Case 1.** R.M.: The echocardiogram (fig. 2) shows normal mitral valve diastolic slope and excursion and normal motion of the posterior mitral leaflet. There were fine high frequency diastolic vibrations of the anterior mitral leaflet with similar high frequency vibrations of the interventricular septum. Such septal vibrations can be seen in the left ventricular outflow tract opposite the mitral leaflet, as well as lower down into the body of the ventricle. As the patient was asymptomatic, cardiac catheterization was not performed.

**Case 2.** C.M.: Clinical and angiographic findings in this patient are included in table 1. The echocardiogram (fig. 3) shows prominent high frequency diastolic vibrations of the anterior mitral leaflet and interventricular septum. The septal vibrations can be seen to start early in diastole, prior to the opening of the mitral valve. The left ventricle was dilated and hypertrophied, and again the vibrations of the septum could be readily seen in the left ventricular cavity at the level of the chordae tendineae (fig. 4).

**Case 3.** H.Y.: This 24-year-old female had rheumatic heart disease with mild-moderate mitral stenosis and severe aortic insufficiency (see table 1). The echocardiogram (fig. 5) shows a reduced EF slope of the anterior mitral leaflet and abnormal anterior diastolic movement of the posterior leaflet consistent with moderate mitral stenosis. High frequency diastolic vibrations were again seen on the anterior mitral leaflet and on the left ventricular side of the interventricular septum.

**Discussion**

Diastolic fluttering of the anterior mitral valve leaflet in aortic regurgitation was first described by Joyner,¹ and was attributed by him to the regurgitant
jet striking the anterior mitral leaflet or to turbulence created by the opposing aortic regurgitant and mitral inflow streams. All patients in that initial report had an apical diastolic murmur of the Austin Flint type. Winsberg described an additional eleven cases with diastolic mitral leaflet fluttering. His data did not permit any conclusion about the relationship of the Austin Flint murmur and the phenomenon of fluttering. Pridie, in a study of 75 patients with aortic regurgitation, could show no correlation between the presence of the mitral vibrations and the severity of the aortic reflux.

High frequency diastolic vibration of the interventricular septum is presumably due to the effect of the aortic regurgitant jet striking the septum. Such septal vibrations commence in early diastole before the mitral valve opens and therefore are not related to mitral inflow and indeed may occur when the mitral valve is closed. Such septal vibrations are probably a common finding as they were present in 17 out of 46 patients with clinical and/or angiocraphic evidence of aortic insufficiency in this study. The presence of septal vibrations does not necessarily correlate with the severity of the aortic insufficiency although only a small number of patients in this series were catheterized. Of those patients in group I who were catheterized, four had severe, three had moderate, and two had mild regurgitation. Of the patients in group II, five had severe, one moderate, and two mild regurgitation.

Other valvular lesions were frequently present in both groups and did not appear to be clearly related to the presence or absence of septal vibrations. It is of interest, however, that there were no catheterized patients in group I with mitral stenosis, while six of eight catheterized patients in group II had mitral stenosis. Furthermore, five of nine catheterized patients in group I had moderate or severe aortic stenosis, while mild aortic stenosis was seen in only two of eight catheterized patients in group II. Due to the small numbers of patients in each group, the significance of these findings is uncertain.

Auscultatory evidence of an Austin Flint murmur was present in three patients in whom mitral stenosis was excluded at catheterization. All three patients had isolated vibration of the anterior mitral leaflet without accompanying septal vibration. Thus, septal vibration did not appear to correlate with the presence of an Austin Flint murmur. Similarly, Winsberg did not show correlation between the presence of anterior

Figure 3

Echocardiogram of Case 2. The beats marked 1 and 2 in the left panel are enlarged at the right. The high frequency septal vibrations commence at A before the mitral valve opens. B. Beat 2 is a ventricular premature beat which is associated with septal motion and some high frequency septal vibration due to aortic regurgitation, C, although the mitral valve does not open, D.
mitral leaflet vibration and an Austin Flint murmur. Fortuin and Craig6 attributed the murmur to antegrade flow across the mitral valve during rapid closure of the leaflets in mid or late diastole. As they noted, the rumble occurred as flow velocity was increasing through the valve although the actual volume of flow was decreasing.

The mechanism of production of septal vibration may be related to factors such as the direction and velocity of the regurgitant jet. It is possible that variations in the direction of the regurgitant jet might in fact account for the negative correlations with severity of the lesion, especially the presence of septal vibrations in mild aortic regurgitation. The high frequency vibrations are only apparent on the left ventricular surface of the septum, presumably due to attenuation of the vibrations within the septal myocardium. The relatively thick interventricular septum and the relatively thin anterior mitral leaflet appear to vibrate at similar frequencies. It is assumed that this is because the turbulent regurgitant flow is forcing the vibration in both structures, i.e., the vibrations are a direct reaction to the regurgitant flow, not a resonant effect.

It is thus apparent that high frequency vibrations of the interventricular septum are a frequent additional echocardiographic finding in aortic insufficiency.

**Addendum**

Since preparation of this manuscript, it has come to our attention that an abstract describing this phenomenon has been recently published by FriedeValDo VE, FutraH GE, Kinard SA, Phillips B: J Clin Ultrasound 2: 229, 1974

**References**

Diastolic vibration of the interventricular septum in aortic insufficiency.
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