Echocardiographic mitral systolic motion in left ventricular aneurysm

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An abnormal, convex systolic anterior motion of the mitral valve, somewhat similar to that seen in idiopathic hypertrophic subaortic stenosis (hypertrophic obstructive cardiomyopathy) and occasionally in atrial septal defect, was found on the echocardiogram in 8 out of 9 patients with angiographically (7 patients) and/or clinically (2 patients) diagnosed left ventricular aneurysm secondary to myocardial infarction. The only patient whose echocardiogram failed to demonstrate convex systolic anterior motion was the only patient who had an inferior wall aneurysm; the rest of the group had anterior and/or apical aneurysms.

In contrast to patients with typical idiopathic hypertrophic subaortic stenosis, the convex systolic anterior motion of the mitral valve observed in our patients tended to 'peak' rather than 'plateau'. Though the basis for this abnormal septal motion in our patients is uncertain, alterations in left ventricular configuration, plus a relatively vigorously contracting posterior left ventricular wall in the presence of abnormal interventricular septal motion, are probable contributory factors. Though non-specific, in the proper clinical and echocardiographic setting convex systolic anterior motion of the mitral valve may be another sign of left ventricular aneurysm.

An abnormal systolic motion of the mitral valve is seen characteristically in idiopathic hypertrophic subaortic stenosis (hypertrophic obstructive cardiomyopathy) and has been described in atrial septal defect. This abnormality can be demonstrated by angiography (Adelman et al. 1969) and non-invasively by echocardiography (King et al., 1973; Popp and Harrison, 1969; Shah, Gramiak, and Kramer, 1969; Shah et al., 1971; Tajik, Gau, and Schattenberg, 1972). A similar motion of the mitral valve was observed on echocardiograms of two patients with post-infarction left ventricular aneurysm. This led us to search for other patients with documented post-infarction aneurysm; one of these, a patient with an apical aneurysm, also had asymmetrical septal hypertrophy. She was, therefore, excluded from the study (Henry, Clark, and Epstein, 1973), even though no demonstrable left ventricular outflow gradient was observed on cardiac catheterization.

Patients and methods

The clinical summary of the 9 patients included in this report is found in the Table. All patients had a documented history of transmural myocardial infarction 4 days to 15 years before this study. None had undergone cardiac surgery. Eight had anteroseptal or anterolateral infarction and one had inferior infarction. Seven of these patients underwent left heart catheterization and selective coronary angiography. All 7 had severe coronary artery disease plus a region of paradoxical ventricular motion. In the 2 patients not catheterized the diagnosis of ventricular aneurysm was based on a history of myocardial infarction, palpable systolic lift over the precordium, and persistent ST segment elevation, and/or progressive left ventricular bulge on serial x-rays.

The echocardiograms were obtained by a Hoffrel Ultrasonoscope Model 101 interfaced with a Cambridge Model 01005 direct strip recorder. With the patient in the semirecumbent or left lateral decubitus position, a 2.25 MHz transducer with a 1.5 cm diameter focused at 7.5 cm was placed along the third or fourth intercostal space near the left sternal border. At least three scans – from aorta to left ventricular apex – were obtained in each patient at paper speeds of 25 and 50 mm per second. One patient had serial echocardiograms at 4 days, 6 weeks, and 11 weeks after infarction. The character of the systolic mitral valve motion was assessed only at points where at least a portion of the posterior mitral leaflet was observed.

Left heart catheterization was performed from 4 weeks to 6 years after infarction. Selective coronary arteriography was carried out by the Sones technique, and left
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TABLE Data summary

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr) and sex</th>
<th>Clinical data</th>
<th>Location of aneurysm by catheterization</th>
<th>Echocardiographic features CSAM</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41 M</td>
<td>Anterior infarction; normal ST segments</td>
<td>Apical, with filling defect</td>
<td>+ +</td>
<td>IVS motion paradoxical; SR: 99/min</td>
</tr>
<tr>
<td>2</td>
<td>47 F</td>
<td>Extensive anterolateral infarction; persistent ST elevation V3-6; visible praeordial systolic impulse</td>
<td>Large apical, with filling defect</td>
<td>+ +</td>
<td>IVS motion decreased; SR: 84/min</td>
</tr>
<tr>
<td>3</td>
<td>57 M</td>
<td>Posteroinferolateral infarction; ST segments normal</td>
<td>Inferior; hypokinetic posterior wall</td>
<td>—</td>
<td>IVS normal; LPW motion decreased; LV dilated; increased RVD; SR: 75/min</td>
</tr>
<tr>
<td>4</td>
<td>40 M</td>
<td>Anteroseptal infarction; persistent ST elevation V1-6</td>
<td>Anterolateral</td>
<td>+ + (×3)</td>
<td>IVS motion decreased; dilated apex, LV dilated; SR: 75-95/min</td>
</tr>
<tr>
<td>5</td>
<td>69 F</td>
<td>Anterolateral infarction; normal cardiac silhouette on repeated chest films; persistent ST elevation V leads</td>
<td>Anteropapical with filling defect</td>
<td>+</td>
<td>IVS motion decreased; SR: 96/min</td>
</tr>
<tr>
<td>6</td>
<td>64 M</td>
<td>Anterolateral infarction; ST elevation V2-6, I, II, L</td>
<td>Not catheterized</td>
<td>+ +</td>
<td>IVS motion decreased; SR: 84/min</td>
</tr>
<tr>
<td>7</td>
<td>66 M</td>
<td>Anteroseptal infarction; no cardiomegaly on repeated chest films; persistent ST elevation V1-5, I, L</td>
<td>Anteropapical</td>
<td>+</td>
<td>IVS motion decreased; LV dilated; SR: 73/min</td>
</tr>
<tr>
<td>8</td>
<td>74 F</td>
<td>Anterolateral infarction; progressive LV bulge on serial posteroanterior chest films; ECGs have become normal</td>
<td>Not catheterized</td>
<td>+ +</td>
<td>IVS motion paradoxical; SR: 80/min</td>
</tr>
<tr>
<td>9</td>
<td>53 M</td>
<td>Anterolateral infarction; slight ST elevation V2-6</td>
<td>Apical</td>
<td>+ +</td>
<td>IVS motion paradoxical; dilated LV; SR: 85/min</td>
</tr>
</tbody>
</table>

CSAM = convex systolic anterior motion of mitral valve (see text).
IVS = interventricular septum. RVD = right ventricular dimension. LPW = left ventricular posterior wall. LV = left ventricle. SR = sinus rhythm. + = prominent. + = moderate. — = absent.

ventriculography was performed in the right anterior oblique projection using meglumine-sodium diatrizoate (Renografin 76%).

Results

For reference, a normal mitral valve echogram is illustrated in Fig. 1. During systole, the normal mitral valve moves anteriorly in a gradual, linear fashion until the onset of diastole, when the mitral leaflets separate. Often the mitral valve in systole shows multiple linear echoes parallel to each other, which possibly reflect portions of the anterior and/or posterior mitral leaflets or chordae (Fig. 2).

In 8 of the 9 patients with left ventricular aneurysm, a distinct convex systolic anterior motion of the mitral valve was noted (Fig. 3, 4). (Only 1 of these 8 patients (Case 4, Table) showed a dilated left ventricular apical segment on echocardiogram. The other 7 patients showed the interventricular septum ‘kissing’ the left ventricular posterior wall, which is the normal systolic relation at or near the apex.) In contrast to typical idiopathic hypertrophic subaortic stenosis (Fig. 5) the convex systolic anterior motion of our patients tended to ‘peak’ rather than ‘plateau’; a normal ratio of septal to posterior wall thickness also distinguished our patients from those with idiopathic hypertrophic subaortic stenosis (Henry et al., 1973). The convex systolic anterior motion of the mitral valve was not notably different from that seen in atrial septal defect (Tajik et al., 1972) but atrial septal defect was not present in any of our patients. The mitral systolic motion associated with the prolapsed mitral valve syndrome is grossly different from that seen here, and is further distinguished by additional echocardiographic features (DeMaria et al., 1974).

Convex systolic anterior motion of the mitral valve was prominent in 6 patients and moderate in 2.
FIG. 1 Normal mitral valve echogram. Gradual, linear anterior systolic motion of the mitral valve is indicated by the arrows. A reference electrocardiogram is above. AML, anterior mitral leaflet; IVS, interventricular septum; LPW, left ventricular posterior wall; PML, posterior mitral leaflet.

The site of the aneurysm in all these patients was in the anterior and/or apical portion of the left ventricle, and their echocardiograms showed either paradoxical or decreased interventricular septal motion. In the single patient who had inferior wall aneurysm, convex systolic anterior motion of the mitral valve was not demonstrable and septal motion was normal (Fig. 6).

Discussion

The evaluation of the phasic motion of the structure depicted in the echocardiogram must be undertaken with care and restraint; otherwise inaccurate, if not erroneous, interpretation may result. A convex systolic anterior motion of the mitral apparatus normally can be seen if the transducer is directed superomedially in the region of the annulus. Therefore, it is essential to evaluate systolic mitral motion in the region where the anterior and posterior mitral leaflets are seen simultaneously (Feigenbaum, 1972).

FIG. 2 Variation of normal mitral valve echogram; multiple echoes are seen in systole (arrows). Abbreviations same as Fig. 1.
FIG. 3 Echocardiogram from Case 2 (Table). Arrows denote a convex systolic anterior motion of the mitral valve, with tendency to peak. The interventricular septal motion is considerably reduced, and nearly paradoxical. By contrast the left ventricular posterior wall moves vigorously. CW, chest wall; AW, anterior wall of right ventricle. Other abbreviations as in Fig. 1.

FIG. 4 Echocardiogram from Case 4. Convex systolic anterior motion of the mitral valve similar to that seen in Fig. 3. Abbreviations as in Fig. 1. Arrows denote main mitral systolic echo.
FIG. 5 Echocardiogram from a patient with idiopathic hypertrophic subaortic stenosis. The convex systolic anterior motion of the mitral valve seen here (arrows) is sustained as a plateau, and almost touches the left side of the interventricular septum, which is hypertrophied. Abbreviations as in Fig. 1.

Usually, this is also the approximate region where the interventricular septum and the posterior left ventricular wall are best seen.

In general, echocardiograms of patients with myocardial infarcts do not reveal convex systolic anterior motion of the mitral valve. Its cause in our patients with left ventricular aneurysm is not certain, but some observations are worth mentioning. Hypothetically, exaggerated motion of the mitral annulus may ‘push’ the closed mitral leaflets anteriorly during systole. However, we did not find any indication of this during cardiac catheterization or echocardiography in our patients. A systolic ‘hump’ of the mitral valve has been described in a patient with mitral stenosis and tachycardia, but it disappeared when the heart rate became normal (Popp and Harrison, 1969). None of our patients had mitral stenosis and all had rates below 100. Moreover, we did not find any correlation between heart rate and the presence or degree of convex systolic anterior motion. Any alteration in left ventricular configuration can potentially affect mitral valve motion (Roberts and Perloff, 1972). Possibly, therefore, the convex systolic anterior motion observed in our patients with left ventricular aneurysm was related to left ventricular asymmetry. Additionally, a vigorously contracting posterior left ventricular wall in association with the observed abnormal interventricular septal motion may have furthered its expression (Fig. 3). In this regard we have also noted the phenomenon in patients with echocardiographically demonstrated concentric hypertrophy of the left ventricle, but only when the septal motion was decreased.

We have not personally seen convex systolic anterior motion of the mitral valve in patients with angiographically documented post-infarction akiness (as opposed to aneurysm) of the left ventricular anterior wall, even when the septum moved abnormally on the echocardiogram. That it may be
associated with akinesis, however, is suggested by an inspection of the mitral valve depicted in Fig. 7b of a study by Jacobs et al. (1973) – despite the fact that their study focused no attention on the mitral echogram per se. We have not seen, nor are we aware of, any reports associating left bundle-branch block with convex systolic anterior motion of the mitral valve, even though abnormal septal motion has been well documented in this condition (Dillon, Chang, and Feigenbaum, 1974; McDonald, 1973). In any case, none of our patients had left bundle-branch block.

Echocardiography is non-invasive, widely available, and helpful in evaluating patients with coronary artery disease as well as the complications thereof (Feigenbaum, 1972; Jacobs et al., 1973). With regard to left ventricular aneurysm per se, two points must be kept in mind: (1) in approximately half of the cases the typical clinical and electrocardiographic features are absent (Abrams et al., 1963; Dubnow, Burchell, and Titus, 1965), and (2) the abnormal systolic motion of the mitral valve described herein is non-specific, and, by itself, insufficient for diagnosis. However, when placed in the proper clinical and echocardiographic context, it should be regarded as a sign pointing to the possible presence of left ventricular aneurysm.

**Addendum**

Since the submission of this paper, Case 4 (Table and Fig. 4) underwent coronary artery bypass. At surgery an 8 x 10 cm aneurysm of the left ventricle, with scarring of the distal two-thirds of the interventricular septum was found and resected. The accompanying echocardiogram (Fig. 7) taken 4 months after operation, shows essentially normal mitral systolic motion.
Fig. 7 Echocardiogram of Case 4 obtained 4 months after resection of a large ventricular aneurysm. Note normal (arrowed) systolic movement of the mitral valve as compared to abnormal motion preoperatively shown in Fig. 4.

References


graphic observations in muscular subaortic stenosis. American Journal of Cardiology, 24, 689.


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