Echocardiographic Recognition of the Mitral Valve–Posterior Aortic Wall Relationship

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SUMMARY
Aortic-mitral valve discontinuity has previously been described in double outlet right ventricle, endocardial cushion defect, single ventricle, tetralogy of Fallot, and prolapse of the mitral valve. We are reporting two additional examples of aortic-mitral valve discontinuity including 15 cases of gross left ventricular dilatation and a case of acute pneumococcal bacterial endocarditis with a large subannular erosion. While nonspecific, aortic-mitral valve discontinuity is a clinically important sign that should be sought with slow-M-mode scanning and strip chart recording.

Additional Indexing Words:
Mitral-semilunar valve discontinuity
Congestive cardiomyopathy
Slow M-mode scanning

The Anterior Mitral Leaflet (AML) is anatomically continuous with the posterior aortic wall (PAW) as these two structures lie at the same depth from the anterior chest wall during systole. Formerly this relationship was best defined on a lateral or left anterior oblique left ventricular angiogram, and continuity assured the origin of the aorta from the systemic ventricle. Echocardiography can now be employed to demonstrate aortic-mitral continuity or discontinuity when the ultrasonic transducer is swept along the aortic-mitral valve axis. For example, in double outlet right ventricle the echocardiogram demonstrates disruption of the normal aortic-mitral continuity, with the AML displaced posterior to the plane of the PAW. This paper reports our experience with echocardiography in the study of the PAW-AML relationship and shows that aortic-mitral discontinuity can be found in conditions other than double outlet right ventricle, endocardial cushion defect, single ventricle, tetralogy of Fallot, and prolapse of the mitral valve.

Materials and Methods

Three groups of patients were studied. Group 1 consisted of 30 normal subjects. Group 2 consisted of 15 patients with left ventricular enlargement (table 1): there were two patients with rheumatic mitral and aortic regurgitation, one with ischemic heart disease, and 12 patients with congestive cardiomyopathy. The mean age of the 12 patients with congestive cardiomyopathy was 42 years, with a range of 12 to 62 years. Ten of the 12 patients had a history of severe, chronic alcoholism, one patient (age 21) presented with congestive heart failure following a viral illness, and in one patient no etiology was suggested. Four of the 12 patients with congestive cardiomyopathy, both patients with rheumatic valvular involvement and the patient with ischemic heart disease underwent cardiac catheterization. Group 3 consisted of a patient with acute pneumococcal endocarditis causing acute aortic regurgitation with severe congestive heart failure. At the time of surgery his aortic valve had a large fenestration of the noncoronary cusp with a large subannular ring-shaped erosion extending beneath the noncoronary cusp around to the junction of the left and right coronary cusps, thus effectively separating the anterior leaflet of the mitral valve from its attachment to the annulus. After removal of the diseased aortic valve, the base of the anterior mitral valve was reattached to the aortic annulus, closing the excavation with 3-0 Tevdek sutures backed with felt pledges. A Starr-Edwards prosthetic aortic valve was then inserted.

The echocardiographic examinations were carried out using a Unirad Model 100 ultrasonoscope and a 2.25 MHz transducer, 1.6 cm in diameter with a repetition rate of 1000 impulses/sec. Records of M-mode scan were made with a Polaroid photograph or a Honeywell 1856 Visicorder strip chart recorder. The patients were examined in the recumbent position with the torso elevated approximately 30° above the horizontal or in the partial left lateral decubital position. Airless contact between the transducer and the chest wall was achieved by the use of aquasonic gel. The transducer was placed in the third or fourth intercostal space along the left sternal border until the typical pattern of the anterior mitral leaflet was identified. The transducer was slowly rotated superiorly and medially toward the patient’s right shoulder until echoes from the aortic root and left atrium were recorded. The transducer was then slowly rotated inferiorly and laterally until the characteristic mo-
Echocardiographic Data in Normals and in Patients with Left Ventricular Enlargement

<table>
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<th>Group</th>
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<th>Ds (cm)</th>
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<th>LV posterior wall thickness (cm)</th>
<th>Septal thickness (cm)</th>
<th>AML displaced post. to PAW (cm)</th>
<th>Septum displaced anterior to ant. aortic wall (cm)</th>
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Abbreviations: Dd = end-diastolic dimension; Ds = end-systolic dimension; LV = left ventricular; AML = anterior mitral leaflet; PAW = posterior aortic wall.

*Coronary artery disease.
†Rheumatic heart disease.

Results

Group 1

The amount of posterior displacement of the AML from the PAW was negligible, ranging from 0 to 3 mm with a mean of 1 mm (fig. 1).

Group 2

In all patients with gross left ventricular dilatation, echocardiographic evaluation of the aortic-mitral sweep demonstrated discontinuity between the PAW and the AML with posterior displacement of the AML and no cases of anterior displacement (fig. 2). The degree of posterior displacement of the systolic portion of the AML from the PAW varied between 1.0 cm and 2.8 cm (mean 1.8 cm) as shown in table 1. Six of the 15 patients also demonstrated anterior displacement of the septum in relation to the anterior aortic wall. The degree of anterior septal displacement varied between 0.5 and 2.0 cm.

Four of the 12 patients with congestive cardiomyopathy underwent cardiac catheterization. The cardiac indices ranged between 1.5 and 1.9 L/min/m², and the left ventricular end-diastolic pressures (LVEDP) varied between 12 and 39 mm Hg. All had significantly increased left ventricular end-diastolic volumes (313–550 ml) and end-systolic volumes (217–264 ml). Two of the four patients had minimal mitral regurgitation. The two patients with rheumatic heart disease had significant mitral regurgitation. In both of these patients the LVEDPs were modestly elevated at 15 and 20 mm Hg, and the cardiac indices were 3.0 and 3.43 L/min/m². End-diastolic volumes were 182 and 107 ml. The patient with coronary artery disease had no angiographic evidence of mitral regurgitation. The LVEDP measured 33 mm Hg, cardiac index 4.1 L/min/m², end-diastolic volume 280 ml, and end-systolic volume 200 ml.
Group 3

The preoperative echocardiogram as well as an artist’s view of the surgeon’s findings in the patient with acute pneumococcal endocarditis and aortic insufficiency are shown in figure 3. With the onset of ventricular diastole the mitral valve barely opened to the E point and there was premature closure well before the QRS complex, a previously well-described finding in acute aortic regurgitation. An unexpected but important echocardiographic finding was the AML-to-PAW discontinuity with the systolic portion of the AML displaced 4 mm anterior to the PAW (fig. 3 left). In addition there was absence of recordable echoes and a distinct echo-free zone between the base of the anterior mitral leaflet and the posterior aortic wall suggesting destruction of supporting tissue. This was confirmed by the surgical findings (fig. 3 right). Postoperatively, the echocardiogram demonstrated normal PAW-AML continuity.

Discussion

During M-mode scanning the direction of the transducer is changed minimally while continuous recordings are displayed on a strip chart recorder. One of the most important and fundamental landmarks in echocardiography is the aortic-mitral sweep or axis. The importance of demonstrating anatomic continuity of the posterior left ventricular wall with the ativoventricular ring and left atrium in the diagnosis of pericardial effusion has previously been emphasized. Normally, the anterior mitral leaflet is attached to the mitral ring, which is contiguous with the posterior aortic wall. During systole when the anterior and posterior leaflets have coapted, the anterior mitral leaflet lies at the same depth from the transducer as the posterior wall of the aorta (fig. 1). We are using the term discontinuous to mean an abrupt change in distance from the transducer while going from the PAW to the AML while not necessarily meaning a complete separation of the structures, although this was obviously the case in the patient with pneumococcal endocarditis and in certain congenital heart defects.

Discontinuity between an ativoventricular valve and the posterior wall of an outflow vessel has previously been documented in double outlet right ventricle, endocardial cushion defect, single ventricle, tetralogy of Fallot, endocardial fibroelastosis, and prolapse of the mitral valve. This paper adds two more conditions, left ventricular enlargement and

Figure 1

Strip chart recording showing the normal continuity between the posterior aortic wall (PAW) and the anterior leaflet of the mitral valve (AML). The anterior aortic wall (AAW) is continuous with the septum (IVS). RCC = right coronary cusp, NCC = noncoronary cusp, LA = left atrium.

Figure 2

Strip chart recording showing posterior displacement of the systolic position of the mitral valve in relation to the posterior aortic wall (PAW) in a 45-year-old patient with congestive cardiomyopathy. The anterior aortic wall (AAW) is continuous with the septum (SEPT). AM = anterior mitral leaflet, PM = posterior mitral leaflet, LA = left atrium, ANT = anterior, POST = posterior.
destructive endocarditis of the aortic valve, in which this discontinuity is also found (figs. 2 and 3).

Our examples of left ventricular enlargement consisted of cardiomyopathies and rheumatic valvular disease. The echocardiographic features of congestive cardiomyopathy have previously been well described.\textsuperscript{10-12} These include increased left ventricular internal dimensions, increased left ventricular outflow tract dimension, and reduced excursion of septal and posterior left ventricular wall motion. Diminished amplitude of motion of the mitral valve\textsuperscript{10} and abnormal mitral valve motion have been reported in those patients with elevated left ventricular end-diastolic pressures.\textsuperscript{13}

This paper documents the additional findings of posterior displacement of the AML relative to a normally positioned PAW, which was observed in all 12 of our patients with congestive cardiomyopathy. The exact mechanism of this discontinuity is not clear. It is possible that the observed phenomenon may be artificially produced by the sudden rotation of the transducer during the recording of the aortic-mitral valve echoes. This phenomenon can be produced to some extent in normals if the transducer is placed more cephalad than usual, for example in the second intercostal space, and rotated rapidly. When the transducer was placed in the third or fourth intercostal space along the left sternal border and rotated slowly from a superior and medial position to an inferior lateral position, negligible discontinuity between the PAW and AML was observed in every normal control. Thus the PAW-AML displacement simply reflects gross left ventricular enlargement secondary to various etiologies rather than any specific disease process. In patients with left ventricular dilatation, the position of the papillary muscle with its chordal attachments may play a role in producing the discontinuity (fig. 4). The tension developed in the normally situated papillary muscle maintains the closed mitral leaflets in an almost continuous plane with the posterior aortic wall. However, in the dilated left ventricle the abnormal cephalad and lateral position of the papillary muscles may have a tendency to pull the leaflets in a more posterior direction. It is important to realize that the posterior displacement of the mitral valve was seen not only with congestive cardiomyopathy, but also when the left ventricle was enlarged as a result of rheumatic valvular or ischemic heart disease.

In the patient with bacterial endocarditis, the AML was displaced anteriorly to the PAW, as a result of erosion of the attachment of the anterior mitral leaflet to the aortic ring subsequent to acute pneumococcal endocarditis (fig. 3 right). The loss of echoes between the AML and PAW was caused by the large subannular ring-shaped erosion extending beneath the non-

\begin{figure}
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\caption{Left: Echocardiographic recording as the transducer is angled in a superior and cephalad direction from the mitral valve (AMV) toward the aorta in the patient with acute aortic insufficiency secondary to bacterial endocarditis. The arrow points to discontinuity and absence of echoes between the mitral valve (which is now anterior) and the posterior wall (PAW). LPW = left ventricular posterior wall, LA = left atrium, AAW = anterior aortic wall. Right: Artist’s impression of the surgeon’s findings in the same patient. There is a large subannular ring-shaped erosion extending beneath the non-coronary cusp and separating the anterior leaflet of the mitral valve from the attachment to the annulus. Fenestration of the non-coronary cusp and vegetations on all the cusps are clearly shown.}
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coronary cusp and separating the anterior leaflet of the mitral valve from its attachment to the aortic annulus. Because of the extensive pathologic destruction of the aortic annulus, the mitral valve was free to assume an anterior position. Buchbinder and Roberts have recently emphasized the invasive and destructive nature of acute pneumococcal endocarditis involving the aortic valve and described semilunar aortic valve destruction with the subannular erosion. This preoperative echographic finding was of clinical importance to our patient since it alerted the surgeons to the possibility of mitral valve repair as well as aortic valve replacement.

Thus, this report emphasizes the potential value of careful echocardiographic study of the PAW-AML relationship in patients with left ventricular cavity dilatation, in patients with a variety of complex congenital cardiac disorders, and in patients with bacterial endocarditis.

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References


Figure 4

Left) This diagram shows the normal relationship between the posterior aortic wall and the closed position of the mitral valve (C). Right) In the dilated left ventricle, the abnormal cephalad and lateral position of the papillary muscle exerts its traction in a posterior direction during systole. The mitral valve closure, position C (position 2), is posterior to the posterior aortic wall (position 1). The septum bulges anteriorly in relation to the anterior aortic wall in the dilated left ventricle. RV = right ventricle, LV = left ventricle, Ao = aorta, LA = left atrium, T = transducer, COCM = congestive cardiomyopathy.
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