Echocardiography of Cardiac Valves in Pericardial Effusion

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SUMMARY Nine adult patients with large pericardial effusions (PE) demonstrated echocardiographic motion abnormalities of cardiac valves in systole. In four cases (Group 1), the abnormal findings consisted of prominent systolic anterior movements of the mitral valve resembling those seen in idiopathic hypertrophic subaortic stenosis. In Group 2 (five cases), typical mitral valve prolapse patterns with large posterior midsystolic displacements well below the C point were observed. Additional abnormalities in Group 2 included tricuspid valve prolapse patterns (four cases), early systolic motion of the aortic valve toward closure (three cases), mid-systolic notching of the pulmonary valve (two cases) and abnormal or attenuated motion of the aortic root in all patients. Marked decrease or resolution of PE resulted in complete disappearance of all the observed abnormalities. These findings appeared to be related to large fluid collections behind the left atrium and abnormal movement of the heart in the pericardial space. In the presence of PE, therefore, the echocardiographic observation of abnormal valve motion may not be clinically significant.

THE ROLE OF ECHOCARDIOGRAPHY in the evaluation of pericardial effusion is well established. Edler1 in Sweden was the first to recognize pericardial effusion using this method, but it remained for Feigenbaum and associates2,3 to pioneer its development into a sensitive and specific noninvasive technique when performed by skilled personnel. With large effusions the heart may swing within the pericardial sac and this phenomenon has been shown to be related to the production of electrical alternans on the electrocardiogram.4 Apparent abnormalities of motion of cardiac valves and other cardiac structures have been frequently observed by us during echocardiographic studies in pericardial effusion5 and the purpose of this report is to describe them and discuss their significance.

Material and Methods

An analysis of 34 adult patients with large pericardial effusions, as indicated by echocardiographic findings of anterior and posterior sonolucent spaces measuring more than 15 mm,4 demonstrated systolic motion abnormalities of one or more cardiac valves in twenty-two (65%). Nine of these were restudied by ultrasound following marked reduction or complete resolution of pericardial fluid and form the basis of this study. Thirteen patients who were not restudied by echocardiography were excluded from the present report. The remaining twelve patients with large effusion but no valvar motion alterations formed a comparison group.

None of the nine patients had heart murmurs or other clinical features of valve dysfunction. There was no clinical evidence for pulmonary hypertension, low cardiac output or cardiac tamponade. The electrocardiograms did not show evidence of ventricular hypertrophy. None had roentgenologic evidence of associated pleural effusion or pulmonary hypertension.

All echocardiographic examinations were performed using a commercially available echograph (Picker) and a 2 MHz collimated transducer. Continuous records were made on 35 mm film or a strip chart recorder. Echocardiographic studies of the cardiac valves and heart walls were performed in the standard manner.

Results

Altered motion of all four heart valves, the heart walls and the aortic root was observed in the present study. The patients were divided into two groups based on the predominant pattern of systolic motion of the mitral valve as well as heart wall motion.

Group 1 (four cases)

In this group the abnormal findings consisted of systolic anterior movements of the mitral valve resembling those seen in idiopathic hypertrophic subaortic stenosis.5 The mitral valve closed normally at the beginning of systole, but soon thereafter made an abrupt anterior movement. It remained in this anterior position until approximately the middle of systole and then briefly returned to a posterior position prior to diastolic reopening. This pattern was seen toward the base of the anterior mitral leaflet rather than near its tip (fig. 1). There was no evidence to indicate thickening of the ventricular septum or narrowing of the left ventricular outflow tract. Abnormalities of the other heart valves were not detected.

The anterior right ventricular wall showed early systolic flattening with posterior motion during the remainder of systole. The left ventricular posterior wall moved anteriorly with the beginning of systole, peaked in midsystole, and thereafter executed rapid posterior motion. The aortic root motion was normal in all except one in whom it resembled the altered motion of the left ventricular posterior wall.

All patients had large pericardial fluid collections behind the left atrium with hyperdynamic wall movements. In general, the left atrial wall moved rapidly anteriorly forming a prominent peak in early systole and this was followed by flattening and a rapid posterior motion in midsystole.

Group 2 (five cases)

Typical mitral valve prolapse patterns with large posterior midsystolic displacements well below the C point6,7 were observed in this group (fig. 2). The contour of the mitral

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Tricuspid and mitral valve prolapse patterns associated with pericardial effusion. The upper panel was recorded in the presence of a large effusion while the lower panel shows the same patient after resolution of fluid and return of normal valve motion patterns. AW = anterior right ventricular wall; TV = tricuspid valve; MV = mitral valve; AS = atrial septum.

The anterior right ventricular wall showed rapid posterior motion in systole with a tendency toward flattening in late systole. The left ventricular posterior wall generally showed flattening in early systole with rapid posterior movement in mid or late systole. All patients also showed poor motion of the aortic root. In four cases the peak of anterior motion of the tricuspid valve showed posterior motion throughout systole suggestive of tricuspid valve prolapse (fig. 2). Other valvular abnormalities included early systolic movement of the aortic valve toward closure (3 cases, fig. 3) and mid-systolic notching of the pulmonary valve (2 cases, fig. 4).
the aortic root occurred in mid to late systole while the remaining patient exhibited paradoxical anterior motion in diastole with flattening in systole (fig. 5).

Pericardial effusion could also be demonstrated behind the left atrium in all patients in this group (fig. 6). The left atrial wall exhibited hyperdynamic motion with flattening in early systole and rapid posterior motion in midsystole. In contrast to Group 1 patients, the left atrial wall did not exhibit prominent early systolic anterior motion with peak formation.

In all nine patients studied, the altered motion patterns of the heart valves, walls and the aortic root reverted to normal following marked reduction or complete resolution of pericardial effusion.

The twelve patients with large pericardial effusions and no apparent motion abnormalities of the cardiac valves exhibited increased but normally directed motion of both right and left ventricular walls and no evidence of significant collections of fluid behind the left atrial wall.

Discussion
In the nine patients comprising the body of this report, the observed alterations in valve motion are probably hemodynamically insignificant since none of the patients exhibited any auscultatory or other manifestations of valvular dysfunction, pulmonary hypertension or low cardiac output. There were no patients with tamponade. Certainly, the disappearance of these motion abnormalities of the heart valves and other cardiac structures following partial or complete resolution of the effusion strongly suggests that the abnormal motion patterns do not represent structural or valvular dysfunction.

Swinging of the entire heart in the pericardial sac is known to result in motion artifacts which may distort the normal motion patterns of the heart walls (fig. 7). In Group 2 patients, excessive systolic posterior motion of the swinging heart may be the dominant vector which produces atrioventricular valve prolapse patterns, systolic displacement of the semilunar valves posteriorly and apparent
attenuation of aortic root motion. When systolic anterior motion of the mitral valve predominates (Group 1), the relation-ship to heart wall motion is less clear. However, the presence of significant fluid collections behind the left atrial cavity associated with hyperdynamic movements and systolic peak formation of the left atrial wall probably introduces motion vectors which compound the total swinging motion of the heart in the pericardial sac producing complex variations. There are other factors which may contribute to the images obtained. M-mode echocardiography depicts all movements of the heart as anteroposterior displacements in the pericardial fluid space. Any rocking or rotary motion of the heart about its attachment to the great vessels, believed to occur with large effusions, may add vectors which are superimposed on normal patterns of valve movement. Also to be considered in any attempted analysis are the effects of cardiac swinging on the blood contained within the cardiac cavities. Certainly, this fluid medium will exhibit different acceleration and deceleration characteristics which would also affect the motion patterns of heart valves and walls. Thus, it would appear that the abnormal appearing traces obtained in the presence of pericardial effusion represent a vectorial summation of the magnitude and direction of intrinsic valve motion, movement of adjacent structures, cardiac displacement, and probably alteration in blood flow patterns induced by cardiac swinging.

It is generally recognized that pericardial effusion spaces disappear when the ultrasonic beam is angled from the left ventricular to the left atrial wall at the levels of the mitral and aortic valves since the insertion of the pulmonary veins into the left atrium interrupts the continuity of the pericardial sac in this region. However, pericardial fluid collection behind the left atrium has been shown by ultrasound especially when the beam is directed where the left atrial pericardium is free of attachment, such as the oblique sinus of the pericardium. In our experience it is most readily demonstrated by sweeping the beam from the mitral to the tricuspid valve and delineating continuity of the fluid space from the left ventricular to the left atrial pericardium.

The motion artifacts of cardiac valves and other structures associated with large pericardial effusion may simulate the echo patterns presented by various clinical entities. A false positive diagnosis of mitral or tricuspid valve prolapse may be made on the basis of large posterior displacements of the aortic segment of the valve in the presence of unrecognized pericardial effusion. Abnormal systolic anterior movements of the mitral valve have been seen in idiopathic hypertrophic subaortic stenosis and mitral valve prolapse. Premature closing motion of the aortic valve has been associated with left ventricular leaks (mitral regurgitation, ventricular septal defects), outflow obstruction (subaortic membranous stenosis, idiopathic hypertrophic subaortic stenosis) and low cardiac output states. Midsystolic notching of the pulmonary valve has been reported to be a reli-

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** Pericardial effusion (PE) behind the left atrial wall. Ultrasonic beam scanning from the tricuspid to the mitral valve (MV) demonstrates the presence of pericardial effusion space behind the left atrial wall (LAW) recorded posterior to the atrial septum (AS) (same patient as in fig. 2). VS = ventricular septum.

![Figure 7](http://circ.ahajournals.org/)

**Figure 7.** Altered anterior right ventricular wall motion in pericardial effusion (PE). Complete reversal of the normal motion pattern is observed. The anterior wall (AW) moves anteriorly in systole and posteriorly in diastole. The left ventricular posterior wall (PW) moves normally.
able sign of pulmonary hypertension. Attenuated motion of the aortic root ordinarily indicates low cardiac output and, in the setting of pericardial effusion, could suggest a misleading diagnosis of tamponade when none exists. Since patients with large pericardial effusions may have coexisting cardiac disease, the final determination of the nature of a motion abnormality of a cardiac valve cannot be made until a repeat echocardiogram is obtained following partial or complete resolution of the fluid accumulation.

References

Pseudoejection Sound in Hypertrophic Subaortic Stenosis
An Echocardiographic Correlative Study
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SUMMARY Phonoechocardiographic studies were performed in 23 patients with hypertrophic subaortic stenosis. In ten patients a distinct systolic sound was recorded, usually along the lower left sternal border. In eight of these cases the sound was low or medium frequency. Unlike the ejection click of valvular aortic stenosis, the sound began 40-100 msec after the upstroke of the indirect carotid pulse and occurred close to the initial peak of the carotid pulse.

This sound, termed pseudoejection sound, was associated with systolic anterior movement (SAM) of the anterior mitral leaflet on the echocardiogram. In all six patients studied with simultaneous phonoechocardiograms, the pseudoejection sound coincided with the sudden halting of SAM of the anterior mitral leaflet. Following provocative maneuvers the sound became louder, and its timing, as well as the sharp halting of SAM of the mitral leaflet, occurred earlier in systole.

The pseudoejection sound probably results either from impact of the anterior mitral leaflet against the interventricular septum or more likely from deceleration of blood flow in the left ventricular outflow tract. The echo data support the association of the pseudoejection sound with significant left ventricular outflow obstruction.

THE OCCURRENCE of systolic ejection sounds in idiopathic hypertrophic subaortic stenosis (IHSHS) has been debated. A number of investigators have failed to document such sounds in this disease. Others have noted their presence in a small number of individual cases or as an unusual finding in a large group of patients. Braunwald et al. noted definite early systolic sounds in seven of 64 patients, an incidence of 11%.

Various investigators have commented on atypical features of these sounds. DeJoseph et al. noted that eight of 42 patients (19%) had delayed systolic ejection sounds. In all cases the systolic sound occurred at the peak of the initial carotid pulse rather than at the onset of the carotid upstroke. They concluded that these sounds were different in origin and nature compared with the ejection sounds in other diseases. In an earlier report Snellen denied the occurrence of systolic ejection sounds in IHSS, but noted a similar, if not identical, sound. He observed the frequent occurrence of a loud low-pitched systolic sound which developed with the sudden downstroke following the early peak of the carotid pulse, marking the onset or sudden increase of a systolic murmur. Likewise, Hancock has described a systolic sound of low pitch and delayed onset, simultaneous with the peak carotid pulse. He has termed it a "pseudoejection sound" because of its superficial resemblance to the true ejection sound of valvular aortic stenosis.
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