Echocardiography in the Diagnosis of Idiopathic Hypertrophic Subaortic Stenosis Coexisting with Pericardial Effusion*  

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Of 89 patients with idiopathic hypertrophic subaortic stenosis who had M-mode echocardiograms recorded, seven patients with coexisting moderate to large pericardial effusions were identified. The clinical profile, M-mode echocardiograms, and cardiac catheterization and angiocardiographic studies in two of the seven patients were analyzed. That the two entities were not associated was suggested by the identification of an etiology for the pericardial effusion in four of the patients. Although the “swinging heart” phenomenon was exhibited in the echocardiograms of each patient, the presence of a significant pericardial effusion did not preclude the ability to establish a diagnosis of idiopathic hypertrophic subaortic stenosis by M-mode echocardiography. 

M-mode echocardiography is a reliable tool for the diagnosis of idiopathic hypertrophic subaortic stenosis (IHSS) or hypertrophic obstructive cardiomyopathy (HOCM). The characteristic echocardiographic markers that have been proposed by previous investigators include the following: (1) asymmetric septal hypertrophy (ASH) with a ratio of interventricular septal thickness to posterior left ventricular wall thickness equal to or greater than 1.3;1 (2) systolic anterior motion (SAM) of the mitral valve;2 (3) diminished diastolic E-F slope of the anterior mitral valve leaflet;3,4 (4) decrease in the percentage of systolic thickening and the amplitude of excursion of the interventricular septum (IVS);5 (5) narrowed left ventricular outflow tract during both systole and diastole;6 (6) partial or total premature systolic closure of the aortic leaflets;7 and (7) reduced internal diameter of the left ventricle.8 

Recent observations9-15 have demonstrated that none of the above echocardiographic features are unique to IHSS. In addition, several of the echocardiographic features of IHSS can be mimicked by a moderate to large pericardial effusion. In particular, a large pericardial effusion even in the absence of hypertrophic cardiomyopathy may produce systolic anterior motion of the mitral valve resembling that seen with IHSS.16-17 Furthermore, early systolic closure of the aortic leaflets19 and altered motion of the interventricular septum17-18 have been described in patients with large pericardial effusions. It is postulated that these changes occur as a result of excessive abnormal movement of the heart within the expanded pericardial space—the “swinging heart” phenomenon.9 However, other echocardiographic features such as asymmetric septal hypertrophy and decreased septal excursion do not occur in patients with pericardial effusion alone. Although the simultaneous occurrence of IHSS and pericardial effusion has been reported,20 to our knowledge no critical analysis of the echocardiogram has been described. 

The purpose of this study is to describe our experience with the diagnosis of IHSS coexisting with pericardial effusion and to evaluate the influence of a significant pericardial effusion on the echocardiographic features of IHSS. 

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### Table 1—Clinical Profile

<table>
<thead>
<tr>
<th>Case No</th>
<th>Age, Sex</th>
<th>Presenting Problem</th>
<th>ECG</th>
<th>Chest X-ray Film</th>
<th>Etiology of Pericardial Effusion*</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>53, M</td>
<td>orthostatic dizziness, syncope, chest pain</td>
<td>P-R 0.12 sec; LVH</td>
<td>enlarged cardiac silhouette</td>
<td>viral</td>
</tr>
<tr>
<td>2</td>
<td>55, M</td>
<td>dyspnea, eustolic murmur left parasternal area</td>
<td>first degree A-V block; LVH</td>
<td>enlarged cardiac silhouette</td>
<td>hydralazine</td>
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<tr>
<td>3</td>
<td>56, F</td>
<td>dyspnea</td>
<td>LVH</td>
<td>enlarged cardiac silhouette</td>
<td>unknown</td>
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<tr>
<td>4</td>
<td>51, F</td>
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<td>LVH</td>
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<td>scleroderma</td>
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<td>5</td>
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<td>asymptomatic; apical systolic murmur</td>
<td>LVH</td>
<td>enlarged cardiac silhouette</td>
<td>unknown</td>
</tr>
<tr>
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<td>enlarged cardiac silhouette</td>
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<tr>
<td>7</td>
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<td>asymptomatic; apical systolic murmur</td>
<td>LVH</td>
<td>enlarged cardiac silhouette</td>
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</tr>
</tbody>
</table>

*None had cardiac tamponade
LVH—left ventricular hypertrophy

Echocardiographic Measurements

The analysis of the echocardiograms and the echocardiographic measurements (Table 2) were done according to previously established methods.\(^1\)\(^4\)\(^5\)\(^10\) Furthermore, measurements at end-diastole were made at the onset of the QRS complex of the simultaneously recorded ECG. Normal values for septal and posterior wall thickness and for percentage of systolic thickening, and systolic excursion of the interventricular septum and left ventricular posterior wall were obtained from the author's own laboratory.\(^15\) Finally, the amount of pericardial effusion was estimated according to the criteria proposed by Felner and Schlant\(^15\) and Feigenbaum.\(^21\) An echo-free space present both anteriorly and posteriorly signifies at least a moderate effusion (approximately 100 ml of fluid); if the posterior echo-free space is greater than 1 cm, a large effusion is present (greater than 1000 ml of fluid).

### Results

**Echocardiography**

**Pericardial Effusion:** A significant anterior echo-
free space between the right ventricular free wall and chest wall and posterior echo-free space between the left ventricular epicardium and pericardium was present in six of the seven patients (Fig 1). In the remaining patient, although no anterior echo free space was seen, a large posterior echo-free space was present throughout the cardiac cycle.

It must be emphasized that Feiner and Schlant and Feigenbaum caution that the proposed guidelines for quantitating the amount of pericardial fluid provide only gross estimates. When the posterior echo-free space is greater than 10 mm, in association with an anterior echo-free space the amount of pericardial effusion is estimated to be 1000 ml or more.

Due to the presenting problem of orthostatic dizziness, syncope and chest pain in case 1, approximately 1200 ml of pericardial fluid was withdrawn during cardiac catheterization. Figure 1 shows the echocardiogram obtained before and after pericardiocentesis.

**Interventricular Septal and Left Ventricular Echograms:** A summary of echocardiographic data is tabulated in Table 2. The mean interventricular septal thickness at end-diastole (IVSTₐ) was 19.7 mm with a range of 18-24 mm and the mean posterior wall thickness at end-diastole (PWTₐ) was 12.3 mm with a range of 10-15 mm. The IVSTₐ/PWTₐ ratio exceeded 1.3 (range 1.4-2.40) in all seven patients. The mean end-systolic thickness of the interventricular septum (IVS) was 22.6 mm with a range of 18-28 mm. The degree of systolic thickening of the IVS, expressed as a percentage of the end-diastolic thickness, ranged from 5.29 percent with a mean of 14.1 percent. Maximum systolic posterior excursion of the IVS ranged from 3-9 mm (mean 5.7). Systolic anterior septal (paradoxic) motion was not observed. In contrast to the systolic thickening of the septum, the degree of left ventricular wall systolic thickening ranged from 47-130 percent (mean 65.3 percent). The mean left ventricular internal diameters at end-diastole and at end-systole were 41.8 mm (range 33-34 mm) and 21.6 mm (range 6-24 mm), respectively. The left ventricular outflow tract (LVOT) ranged from 17-30 mm with six of the seven patients 22 mm or less.

**Mitral and Aortic Valve Echograms:** The E-F slope of the anterior mitral valve leaflet was 19-89 mm/sec (mean 45 mm/sec). Systolic anterior movement of the mitral valve was obvious in all seven patients (Table 2). The posterior mitral valve leaflet moved normally in all patients.

Midsystolic notching and/or premature closure of the aortic leaflets was apparent in all seven patients. The aortic leaflets were not thickened in any of the patients. Figure 2 is a series of echograms from case 6 demonstrating characteristic changes in the mitral and aortic valve.

**Cardiac Catheterization and Angiocardiography**

In the two patients (cases 1 and 4) who underwent left and right heart catheterization, there was no gradient at rest across the left ventricular outflow tract. During inhalation of amyl nitrite, a peak

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**Figure 1.** A series of echocardiograms from case 1. Echocardiograms A, B and C were obtained prior to pericardiocentesis. A shows the left ventricle at the level of the posterior papillary muscle on the left and the mitral valve on the right. There is a moderate anterior pericardial effusion, a large posterior pericardial effusion and asymmetric septal hypertrophy (ASH); B shows systolic anterior motion (arrow a) and the decreased E-F slope (arrow b) of the anterior mitral valve leaflet. C shows the aorta, aortic leaflets and the left atrium. Arrows c and d point to the midsystolic notching and gradual premature closure of the anterior and posterior aortic leaflets, respectively. Echograms D, E and F were recorded after removal of approximately 1200 ml of pericardial fluid. D shows the markedly diminished anterior and posterior pericardial effusion, but the same degree of ASH. E shows systolic anterior motion (arrow e) of the mitral valve which is now better visualized, but the height and width do not appear to be different from the pre-pericardiocentesis echo in B. The diastolic E-F slope of the mitral valve has now increased and is no longer closely apposed to the interventricular septum; F shows the mid-systolic notching on the anterior aortic leaflet (arrow f). APE = anterior pericardial effusion; RV = right ventricle; IVS = interventricular septum; LV = left ventricle; PW = posterior wall; PPE = posterior pericardial effusion.
gradient of 60 mm Hg and 40 mm Hg, respectively, was present and catheter entrapment was ruled out. In both patients, there was a small left ventricular cavity at end-diastole with almost complete cavity obliteration at end-systole. Systolic anterior movement of the mitral valve was not seen, but a prominent bulge of the hypertrophied septum into the left ventricle was present. There was no evidence of pericardial constriction and the aortic valve was tricuspid without calcification or regurgitation. The mitral valve was normal in case 1, whereas a calcified annulus without evidence of mitral regurgitation was seen in case 4. Neither patient had a diastolic gradient across the mitral valve and both had normal coronary arteries.

In addition, in case 1, no significant hemodynamic changes were detected after removal of approximately 1200 ml of pericardial fluid. No significant effects were seen on the systolic function of the left ventricle by removal of the pericardial fluid.

**DISCUSSION**

_Echocardiographic Abnormalities involving the Mitral and Aortic Valves_

The systolic anterior movement of the mitral valve in patients with isolated large pericardial effusion and the "swinging heart" phenomenon generally occur simultaneously with excessive motion of the other cardiac structures anterior and posterior to the mitral valve. When exaggerated anterior excursion of the whole heart occurs during systole, the resultant mitral valve echo may display anterior motion simulating the systolic anterior motion seen with idiopathic hypertrophic subaortic stenosis. Vignola et al.\(^2\) have suggested that the patterns of motion exhibited by the mitral valve of either a "pseudo" posterior prolapse or SAM may be a function of the extent and timing of posterior and anterior cardiac swinging respectively, and the heart rate. In eight patients reported "when the heart rate was 120 beats/minute or greater, the posterior cardiac swinging occurred during the beginning or end of systole. When the rate was less than 120 beats/minute, the swinging occurred in midsystole." In our seven patients, mild-to-moderate cardiac swinging was present in five, but the extent and timing of SAM did not demonstrate any consistent correlation with the heart rate.

Since a large pericardial effusion may conceivably affect the position of the heart in the pericardial sac, the systolic anterior motion of the mitral valve in such patients, without IHSS, is considered to be a motion artifact resulting from the exaggerated movement of all cardiac structures along the anterior-posterior axis relative to the transducer stationary on the subjects' chest.\(^1\) In contrast, in hypertrophic cardiomyopathy, the precise mechanism of SAM is not
demonstrate various degrees of premature closure of as an isolated feature, changes on the aortic valve with IHSS. It is well recognized, however, that ing systole were more characteristic of those seen with hypertrophic cardiomyopathy. In IHSS, any condition leading to severely decreased forward cardiac output may demonstrate various degrees of premature closure of the aortic leaflets.

Other studies have suggested that an inspira­tory decrease of the E-F slope of the anterior mitral valve leaflet may occur in patients with cardiac tamponade. This has been postulated to be a reflection of the compromised left ventricular filling during inspiration in patients with cardiac tamponade. Though four of our patients had diminished mitral valve E-F slopes, none clinically had cardiac tamponade. This decrease in mitral valve diastolic slope is consistent with the reported restriction in diastolic filling of the poorly compliant left ventricular cavity of IHSS.

Interventricular Septal Echograms: A spectrum of changes involving the interventricular septum (IVS) including paradox motion or exaggerated systolic posterior motion have been described in patients with large pericardial effusions. However, other echocardiographic features such as asymmetric septal hypertrophy and decreased septal excursion do not occur in patients with pericardial effusion alone. Our observations and measurements (Table 2) of the IVS confirmed that excessive cardiac motion had no significant influence on septal thickness and degree of systolic thickening in patients with hypertrophic cardiomyopathy. In IHSS, the septal-to-posterior wall ratio is reported to be in excess of 1.3 or 1.5. While the latter ratio improves the specificity of this calculation, it is uncertain what effect the higher ratio has upon its sensitivity. In the group examined in this report, this ratio ranged from 1.4 to 2.40 (Table 2).

Other studies have documented that an IVST/PWT ratio greater than 1.3, originally considered as characteristic of IHSS, occurs in infants and young children without heart disease. In addition, patients with a wide variety of cardiac lesions not associated with IHSS, including ventricular septal defect, coarctation of the aorta, tetralogy of Fallot, acromegaly, and pulmonary valve stenosis may demonstrate IVST/PWT ratios in excess of 1.3. None of our patients had any of these conditions (Table 1).

Clinical Implications

It is apparent from our study that the presence of a large pericardial effusion does not preclude the ability to establish a diagnosis of IHSS by M-mode echocardiography. Although a “swinging heart” in patients with pericardial effusion may influence the motion of the heart valves, in the presence of ASH and/or decreased septal excursion, a diagnosis of IHSS may be made accurately in the presence of a large pericardial effusion. It must be stressed, however, that none of the characteristic features of IHSS should be assessed in isolation to avoid erroneous interpretation.

Though the study was not designed to investigate an association between the two entities, our data would suggest that IHSS and pericardial effusion are not etiologically related entities. In four of our seven patients, an etiology for pericardial effusion was identified (Table 1). In the other three patients, the cause of the effusion has not been established conclusively, although pericardial effusion occurring in IHSS might be expected to occur in patients with severe congestive heart failure related to cardiomyopathy or coronary artery disease. There is no evidence to indicate that the two conditions are related in any fashion other than the potential to coexist in a patient. It would be reasonable to postulate that when significant pericardial fluid is detected in a patient with IHSS, another cause must be searched for to explain the pericardial effusion.

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