Role of Echocardiography in Diagnostic and Hemodynamic Assessment of Hypertrophic Subaortic Stenosis

By Pravin M. Shah, M.D., Raymond Gramiak, M.D., Allan G. Adelman, M.D., and E. Douglas Wigle, M.D.

SUMMARY

An abnormal sharp anterior motion of the anterior mitral leaflet has been previously described as a diagnostic abnormality in hypertrophic subacute stenosis (HSS). The present prospective study explored the diagnostic sensitivity and specificity of this finding in 26 patients studied without knowledge of clinical diagnosis. When the abnormality observed was complete and persistent, a correct diagnosis of HSS was made in every case. In the absence of systolic abnormality at rest and on provocation, the diagnosis of HSS could be excluded. When the abnormality was small and inconstant (nine patients) two thirds of the patients were shown to have a latent or labile form of outflow obstruction whereas the remaining third had no evidence of HSS.

In an extended part of the study on 31 patients with known HSS, comparisons of resting pressure gradients across left ventricular outflow tract were made with the echocardiographic findings at rest. Those with persistently abnormal mitral valve movement had an average pressure gradient of 78 mm Hg, those with inconstant abnormality had an average gradient of 24 mm Hg, and those with no resting abnormality had no gradients at rest.

Additional Indexing Words:
Mitral valve
Left ventricular outflow obstruction

The clinical and hemodynamic characteristics of hypertrophic subaortic stenosis (HSS) have been studied over the last decade.1-4 No single test short of cardiac catheterization and angiography has proved to be of reliable diagnostic value. Even these have been less than satisfactory in the estimation of the severity of left ventricular (LV) outflow obstruction, owing to its labile and variable nature. The observed pressure differences across the LV outflow tract have varied in the same subject from one examination to the next, from one moment to the next, from one phase of respiration to the next, and even within the same systolic segment of a cardiac cycle. Further refinement through multiple cardiac-catheterization studies in the same subject is neither practical nor universally desirable. Development of a noninvasive and nontraumatic method to aid in the diagnosis and in the assessment of the severity of LV outflow obstruction would be of considerable use.
Moreyra and associates, using pulses ultrasound cardiology, observed diastolic apposition of the anterior mitral leaflet to the interventricular septum and explained it on the basis of narrow LV outflow space. No attempt was made to correlate the findings with the systolic features of the disease. Shah et al. first described an abnormal mitral valve motion in systole in the form of an abrupt anterior movement of the anterior leaflet in midsystole with resultant narrowing of the LV outflow space. This abnormal mitral valve motion was associated with the dynamic LV outflow obstruction and possibly the accompanying mitral regurgitation. Validation of the relation of the mitral leaflet to LV outflow narrowing in HSS was obtained by Gramiak et al., who used ultrasonic contrast techniques to identify cardiac structures. These observations have subsequently been confirmed by Popp and Harrison and also by Pridie and Oakley in England.

When the mitral leaflet is detected from the left parasternal position, the ultrasonic "beam" traverses the anterior wall and cavity of the right ventricle, interventricular septum, LV outflow space, the anterior mitral leaflet, and generally the left atrium posteriorly. In normal subjects the anterior leaflet occupies a posterior position away from the interventricular septum throughout ventricular systole (fig. 1). When the mitral valve opens in early diastole, the leaflet moves anteriorly toward the interventricular septum. Observations made in our laboratory have identified an abnormal systolic movement of the anterior mitral leaflet in patients with HSS. It begins with the early peak of the carotid artery pulse and with the onset of the murmur and results in narrowing of LV outflow space lasting for up to 60% of

**Figure 1**

Left panel is a schematic drawing of the course taken by pulsed ultrasound beam with the transducer held to obtain anterior mitral leaflet motion from left of the sternum. Right panel shows reflected ultrasound record obtained in a normal subject simultaneously with the carotid pulse (CP), the phonocardiogram (PCG), and the electrocardiographic lead (ECG).

Abbreviations: RV = right ventricle; IVS = interventricular septum; LVO = left ventricular outflow space; MV = mitral valve (anterior leaflet); LA = left atrium. In this and all subsequent figures an upward deflection reflects anterior motion and downward deflection posterior motion.
The ejection period (fig. 2). Toward the latter part of systole, the leaflet returns to the posterior position. The free edge of the mitral leaflet produces the most strikingly abnormal ultrasonic patterns and must be sought in every examination to avoid false-negative results.7

The purpose of the present study is to assess in a prospective manner the diagnostic accuracy of abnormal mitral valve movement as detected by ultrasound. In addition, an attempt has been made to estimate the severity of LV outflow obstruction on the basis of pressure gradients and to correlate this with the echocardiographic abnormality.

**Methods and Material**

Simultaneous recordings of the echocardiogram of the mitral valve, the phonocardiogram, the external carotid pulse, and the electrocardiogram were obtained in consecutive beats in over 100 cardiac cycles. The detailed method of ultrasound recordings of the anterior mitral leaflet in our laboratory has been reported elsewhere.7,11

Briefly, the transducer was placed in the fourth or the fifth left interspace and angled so as to obtain the typical free and snapping movement of the mitral leaflet. The ultrasonic "B" mode display was presented on the upper beam of Tektronic 565 dual-beam oscilloscope, and continuous recordings were made on a 35-mm film by means of a Fairchild camera (type 321-A). The lower beam on the oscilloscope was used in multitrace operations by means of a four-trace amplifier to record additional physiologic data.

The phonocardiogram was obtained through a Maico contract microphone held over the apex. The output was passed through a Sanborn heart sound preamplifier 350-1700 B) and filtered through a high-pass filter over 100 Hz. The carotid artery pulse was obtained with the help of a crystal pick-up and a D-C amplifier (Model 350-3200).

The observations were made with subjects in supine resting state, and were subsequently repeated with Valsalva maneuver and amyl nitrite inhalation, since these interventions are known to provoke LV outflow obstruction in HSS. The clinical diagnosis was confirmed by hemodynamic and angiographic studies independent of the echocardiographic examination. A few patients

**Figure 2**

Ultrasound tracing of anterior mitral leaflet (MV) in a normal subject (left panel) compared with a patient with HSS (right panel). The specific abnormality in systole (arrow) develops during early ejection and consists of a sharp anterior motion obliterating the outflow space. In the latter part of systole the leaflet returns to a posterior position. The strikingly flat diastolic segment is nonspecific and reflects reduced ventricular filling rate. Note the timing of the murmur and double-peaked carotid pulse with the systolic abnormality. Abbreviations: HOCM = hypertrophic obstructive cardiomyopathy; MSS = muscular subaortic stenosis; IHSS = idiopathic hypertrophic subaortic stenosis.
were restudied at cardiac catheterization and echocardiography, and left ventricular and brachial artery pressures were recorded simultaneously with the ultrasonogram.

The patient material studied will be reported under two sections.

Section I of the study was concerned with diagnostic evaluation of the method and included 26 patients with an age range of 10 to 56 years. Patients with nonvalvular heart disease were examined, since prior experience of echocardiography in over 200 cases with valvular heart disease had in our hands failed to show a pattern of mitral valve abnormality that could be confused with HSS. The patients were selected by two authors in Canada (A.G.A. and E.D.W.). The patients were selected from among those with known myocardial disease studied independently by cardiac catheterization and angiocardiography earlier in their laboratory. Arbitrary preselection included several patients with HSS intermixed with those having nonobstructive cardiac hypertrophy. The Canadian investigators also included family members of one patient with HSS and two without, all of whom had underlying myocardial disease. The echocardiograms were obtained and interpreted by two authors (P.M.S. and R.G.) without any knowledge of the underlying heart disease in these patients. Section II was concerned with an attempt to establish criteria of severity of LV outflow obstruction on the basis of echocardiography in 31 patients with known HSS (age range, 19 to 59 years). Hemodynamic assessment of these patients was obtained separately; the majority were studied in Canada (by A.G.A. and E.D.W.). The echocardiographic interpretation was independently made (P.M.S. and R.G.) without prior knowledge of the hemodynamic findings.

Echocardiographic findings were broadly classified on the basis of the systolic segment of the mitral leaflet motion:

1. Normal mitral valve motion both at rest and on provocation (negative for HSS).
2. Abnormal systolic anterior motion with narrowing of LV outflow space noted.
The ultrasound recording shows a somewhat unimpressive, small anterior motion (shown by an arrow) of the mitral leaflet (MV). This is inconstant from one beat to the next and results in only partial narrowing of the outflow space. Pressures obtained simultaneously revealed absence of outflow gradient and lack of a significant murmur at rest. In this patient Valsalva maneuver and isoproterenol infusion resulted in the characteristic pressure gradient, a prominent systolic murmur, and the systolic abnormality in mitral leaflet motion. Abbreviations: SAM = systolic anterior movement.

Consistently at rest (positive for HSS) (fig. 3).
3. The systolic abnormality seen as a small anterior movement present inconstant at rest and/or on provocation (suggestive for HSS) (fig. 4).

Results
Section I: The results are summarized in table 1. On the basis of echocardiographic findings of mitral valve motion, the patients in this section were placed in three groups. Nine of the 26 patients in this study showed a normal pattern of mitral valve motion in systole at all times (shown as symbol • in table 1.). Eight patients had consistently abnormal mitral valve motion and were reported as positive for HSS (symbol ☐ in table 1.). The remaining nine patients had inconstant abnormality and were reported as suggestive for HSS (symbol ☐ in table 1.). Thus, all the patients reported as negative for HSS on echocardiographic assessment did not have HSS. Eight patients reported as positive all had confirmed HSS with significant resting pressure gradients across the LV outflow tract. The third group of nine patients with findings interpreted as suggestive for HSS consisted of two patients with resting outflow gradient, four with latent outflow obstruction, and three with no evidence of HSS. The last three cases represent false-positive results in this study.

Section II: The pressure gradients across LV outflow tract measured in resting state were compared with echocardiographic appearance of mitral valve motion obtained...
separately. Figure 6 depicts the observed results in 31 patients with hemodynamic and angiographic evidence of HSS. Eighteen patients with a persistently abnormal systolic mitral valve motion and almost total narrowing of the LV outflow tract (represented by closed circles) had peak systolic gradients of 20 to 140 mm Hg (average, 78 mm). Five patients in whom the echocardiographic abnormality of the mitral valve motion was inconstant, with partial narrowing of the outflow tract, had peak systolic gradients of 10 to 52 mm Hg (average, 24 mm Hg). Seven of the eight patients with no demonstrable systolic abnormality of mitral valve motion at rest (represented by open circles) had no pressure gradient at rest and in one it was 10 mm Hg.

Discussion

Bjork\textsuperscript{11} was the first to postulate that asymmetrical hypertrophy of the interventricular septum may result in an abnormal rotation of the anterior mitral leaflet which balloons into the LV outflow tract to produce obstruction in HSS. This view was not generally accepted because of lack of supportive evidence. More recently, Dinsmore and associates\textsuperscript{12} demonstrated angiographically an abnormal anterior motion of the mitral leaflet in systole. These observations were further extended by Simon and associates\textsuperscript{13} and by Adelman and associates,\textsuperscript{14} who implicated this movement with obstruction angiographically.

The present study tests our earlier echocardiographic observations\textsuperscript{6,7} in a prospective evaluation of the method. It was pointed out

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**Figure 5**

Ultrasound recording in a patient with hypertrophic cardiomyopathy which showed high artificial pressure gradients from catheter entrapment on advancing the catheter tip to the apex of the left ventricle. There was no systolic pressure gradient between inflow and outflow of LV. This patient had idiopathic left ventricular hypertrophy with cavity obliteration rather than outflow obstruction. The abnormal systolic motion of the mitral leaflet could not be demonstrated at rest or on provocation.
Table 1

<table>
<thead>
<tr>
<th>No. of patients</th>
<th>Mitral valve motion</th>
<th>Hemodynamic and angiographic evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>○</td>
<td>No evidence of HSS*</td>
</tr>
<tr>
<td>8</td>
<td>●</td>
<td>All proven HSS with LV outflow gradient 40-110 mm Hg.</td>
</tr>
<tr>
<td>9</td>
<td>○</td>
<td>6, proven HSS†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3, no evidence of HSS‡</td>
</tr>
</tbody>
</table>

*4 patients had idiopathic hyperkinetic circulatory state, 3 idiopathic hypertrophy without demonstrable LV outflow obstruction but with cavity obliteration (fig. 5), and 1 with restrictive and 1 with congestive form of cardiomyopathy.

†4 with HSS had no outflow gradient at rest but developed it on provocation (latent obstruction; 2 had resting gradients of 40 and 50 mm Hg respectively).

‡1 with ostium primum and cleft mitral valve, 2 with nonobstructive cardiomyopathy with accompanying mitral regurgitation.

In our previous communication that a negative evaluation by echocardiography may be obtained by directing the ultrasonic transducer over the mitral leaflet near its annular attachment. Thus, false-negative results can be avoided by careful search for the free edge of the mitral leaflet with a more lateral and caudal angulation of the transducer. No false-negative results were noted in the present study, probably because our system allows continuous recording of several hundred consecutive beats. In our experience, this technique is highly sensitive in detecting HSS, even in the absence of other clinical data. The proper interpretation of all echocardiographic data needs reemphasis that this technique permits display of structures in only one plane. Hence, the comments pertaining to narrowing of LV outflow space relate to the space observed in the echocardiogram and as illustrated in figure 1. When the abnormal systolic anterior motion of the mitral leaflet was present consistently and the abnormality almost completely obliterated the LV outflow space, the diagnosis of HSS with resting obstruction was made with confidence. No false-positive result was obtained with this type of abnormality. When the diagnostic evaluation was extended to include an abnormal motion in the occasional beat with partial narrowing of the outflow tract, it was possible to diagnose those patients with latent and labile obstruction. This was achieved with some loss of specificity since the same abnormality was noted in a patient with cleft mitral valve and in two patients with nonobstructive forms of cardiomyopathy. Similar inconstant abnormalities have been observed in some patients with mitral valve prolapse, but these can be distinguished from the HSS as the abnormality occurs near the annular margin of the valve cusp and not its free margin.

Since the systolic anterior motion of the mitral leaflet appears to make an important contribution to the LV outflow obstruction in HSS, the second objective of this study was to examine the role of echocardiographic findings in prediction of hemodynamic severity of obstruction. Results of hemodynamic assessment in resting state correlated well with the

![Figure 6](image)

Comparison of the types of echocardiographic abnormalities of mitral valve motion observed at rest with the peak systolic pressure difference between left ventricle and aorta also obtained at rest but on a separate occasion in 31 patients with HSS. Symbols: ● = complete and constant SAM, as in figure 3. ○ = partial and inconstant SAM, as in figure 4. ○ = no abnormality in systole.
separately obtained echocardiographic findings under resting conditions. Those having no obstruction at rest had no detectable abnormality on the echocardiograms. Those with persistent obstruction at rest and a relatively high persistent pressure gradient across the LV outflow tract generally had the persistent and complete type of abnormality. In patients with variable outflow gradients, there were only minor and inconsistent abnormalities on echocardiograms. Considering the variable nature of obstruction in this disease, the overall correlation between the two methods used on separate occasions is good. Hence, a fairly gross prediction of the presence and severity of LV outflow obstruction may be made in the majority of cases of HSS.

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References


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