Systolic Anterior Motion of the Mitral Valve in the Absence of Asymmetric Septal Hypertrophy

GARY S. MINTZ, M.D., MORRIS N. KOTLER, M.D., BERNARD L. SEGAL, M.D., AND WAYNE R. PARRY

SUMMARY Asymmetric septal hypertrophy (ASH) is considered the unifying link in the spectrum of hypertrophic cardiomyopathies. ASH and mitral valve systolic anterior motion (SAM) are the two most important echocardiographic criteria for the diagnosis of idiopathic hypertrophic subaortic stenosis (IHSS). Ten patients found to have SAM without ASH were studied. Septal thickness, thickening, and excursion were normal.

Seven patients had left ventricular ejection times (LVET) measured before and after amyl nitrite inhalation. In six of them, corrected LVET increased by more than 40 msec. Four patients underwent diagnostic cardiac catheterization. Resting or provokable left ventricular outflow tract (LVOT) gradients were demonstrable in all four patients.

The echocardiographic features in patients with SAM alone, ASH and SAM, and ASH alone were compared. LVOT measurements in patients with SAM alone (2.2 ± 0.9 cm) and ASH and SAM (2.1 ± 0.5 cm) were similar and narrower than in patients with ASH alone (2.8 ± 0.5 cm, P < 0.001). Ejection fractions in patients with SAM alone (79 ± 10%) were greater than in patients with ASH and SAM (66 ± 16%, P < 0.02) or ASH alone (60 ± 15%, P < 0.001). Thus, dynamic left ventricular outflow obstruction can exist in the absence of echocardiographic ASH. LVOT width and abnormal LV ejection dynamics may contribute to the LVOT obstruction with or without the presence of ASH.

SYSTOLIC ANTERIOR MOTION (SAM) of the mitral valve, as shown echocardiographically, was first described by Shah et al. in 1969.1 This finding is considered to represent echocardiographic evidence for left ventricular outflow tract obstruction seen in idiopathic hypertrophic subaortic stenosis (IHSS). Henry et al.2 showed that the degree and duration of the systolic septal-mitral valve opposition correlates well with the left ventricular outflow tract gradient. Furthermore, they have proposed that asymmetric septal hypertrophy (ASH) represents the unifying link in a spectrum of symptomatic or asymptomatic and obstructive or nonobstructive hypertrophic cardiomyopathies.8 Echocardiography is a sensitive and reliable technique for detecting ASH. Several studies have characterized the echocardiographic features of the hypertrophied septum as showing a decreased excursion4 and a decreased thickening (less than 20%).4,4 during systole. SAM without ASH has recently been reported in patients without detectable cardiovascular disease.5,9 However, the hemodynamic significance of SAM without ASH is unknown. The purpose of this report is to describe the clinical, echocardiographic, and hemodynamic features in 10 of our patients with SAM who had normal septal thickness.

Materials and Methods

The clinical, echocardiographic, and hemodynamic data on all patients with SAM or ASH or both, documented at our institution, were reviewed. Clinical data based on symptoms, physical findings, and electrocardiographic abnormalities were analyzed.

We performed echocardiographic studies with the patients in the supine or left lateral decubitus position, utilizing a Smith Kline Ekoline 20A ultrasonoscope with a 2.25 MHz medium internally focused transducer. Permanent records were obtained with an Irex 101 Continutrace recorder.

The entire septum, from the aortic root to below the tips of the mitral valve, was scanned in all patients to detect septal hypertrophy. Interventricular septal (IVS) thickness in diastole and systole, percentage of septal thickening, and

References


From the William Likoff Cardiovascular Institute, Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania.

Address for reprints: Morris N. Kotler, M.D., William Likoff Cardiovascular Institute, Hahnemann Medical College and Hospital, 230 North Broad Street, Philadelphia, Pennsylvania 19102.

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septal excursion were measured below the tip of the mitral valve. Diastolic septal thickness was measured from the right ventricular to the left ventricular endocardial surface before atrial systole, but after rapid ventricular filling.\(^6\) Left ventricular posterior wall (LVPW) thickness was measured at the level of the posterior mitral valve before atrial contraction. Left ventricular internal dimensions (LVID) were measured below the level of the mitral valve; the end-diastolic dimension (LVIDd) was measured at the peak of the R-wave, and the end-systolic dimension (LVIDs) at the smallest IVS-LVPW distance. Left ventricular volumes were calculated by the Teichholz formula.\(^9\) The ejection fraction was calculated by EF = (EDV-ESV)/EDV. Normal values in our laboratory are 67 ± 6%. The left ventricular outflow tract (LVOT) was measured from the C point of the mitral valve, or the point of coaptation of the mitral valve leaflets before the onset of SAM, to the left side of the septum (normal values 2.0-3.5 cm).\(^11,12\)

Indirect carotid pulse tracings were recorded, and left ventricular ejection times were measured before and after administration of amyl nitrite in seven of the 10 patients found to have SAM without ASH, in 25 of 45 patients with ASH and SAM, and in 10 of 37 patients with ASH alone. Corrected left ventricular ejection time (LVETc) was calculated according to the formulae of Weissler et al.:\(^13\)

\[
\text{LVETc} = \text{LVET} + 0.0017 \text{ hr (males)}.
\]

\[
\text{LVETc} = \text{LVET} + 0.0016 \text{ hr (females)}.
\]

Cardiac catheterization was performed in four of the 10 patients found to have SAM without ASH, in 14 patients with ASH and SAM, and in two patients with ASH alone. Catheterization was performed for evaluation of symptoms and exclusion of IHSS hemodynamics. Left ventricular and aortic pressures were recorded by means of fluid-filled double-lumen catheters. LVOT gradients were measured at rest and during a variety of provocations, including post-patent ventricular contraction potentiation, amyl nitrite inhalation, isoproterenol infusion, and the Valsalva maneuver.

**Results**

Ten patients (six male, four female; ages 15–81, mean 53) were found to have echocardiographic SAM without septal excursion. Detailed echocardiographic evidence of ASH. These patients had no clinical or echocardiographic evidence of left ventricular hypertrophy, primary pulmonary hypertension, congenital heart disease, mitral valve prolapse, or other structural cardiac abnormality. Forty-five patients (26 male, 19 female; ages 11–80, mean 42) had ASH and SAM. Thirty-seven patients (25 male, 12 female; ages 29–78, mean 57) had ASH alone.

**Clinical Findings**

Dyspnea was present in 40% of patients found to have SAM without ASH, 45% of patients with ASH and SAM, and 54% of patients with ASH alone. Angina was present in 30% of patients found to have SAM without ASH, 30% of patients with ASH and SAM, and 46% of patients with ASH alone. Thirty percent of patients found to have SAM without ASH, 38% of patients with ASH and SAM, and 21% of patients with ASH alone were asymptomatic. Only patients with ASH and SAM complained of lightheadedness. A history of hypertension was more common in patients with ASH (54%) than in patients with ASH alone (30%) or ASH and SAM (23%). Grade II–III systolic ejection murmurs localized to the lower left sternal border or to the apex were present in 80% of patients found to have SAM without ASH and 80% of patients with ASH and SAM. Short soft ejection murmurs, grade I–II, were audible in 70% of patients with ASH alone. Electrocardiographic (ECG) evidence of left ventricular hypertrophy (LVH) was found in an equal percentage of patients with ASH and SAM (38%), SAM alone (30%), or ASH alone (39%); ECG evidence of infarction was not seen in patients with SAM alone, as compared with 13% of patients with ASH and SAM and 7% of patients with ASH alone.

**Echocardiographic Findings**

The echocardiographic data for patients found to have SAM without ASH are listed in Table 1. These measurements are compared with those of patients with ASH and SAM alone in Table 2.

In patients found to have SAM without ASH, septal thickness (0.9 ± 0.1 cm), thickening (46 ± 16%), and excursion (0.7 ± 0.2 cm) were normal (fig. 1). These differed significantly from measurements of septal thickness, thickening, and excursion in patients with ASH and SAM or

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>IVS d(cm)</th>
<th>IVS s(cm)</th>
<th>th(%)</th>
<th>ex(cm)</th>
<th>LVPW d(cm)</th>
<th>LVID s(cm)</th>
<th>EF(%)</th>
<th>LVOT(cm)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>54</td>
<td>0.8</td>
<td>1.1</td>
<td>38</td>
<td>0.6</td>
<td>0.8</td>
<td>5.0</td>
<td>3.3</td>
<td>71</td>
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<tr>
<td>2</td>
<td>46</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
<td>0.7</td>
<td>1.0</td>
<td>4.1</td>
<td>2.2</td>
<td>84</td>
</tr>
<tr>
<td>3</td>
<td>43</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
<td>0.6</td>
<td>1.0</td>
<td>4.3</td>
<td>2.2</td>
<td>86</td>
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<td>4</td>
<td>18</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
<td>0.6</td>
<td>1.0</td>
<td>4.0</td>
<td>2.3</td>
<td>81</td>
</tr>
<tr>
<td>5</td>
<td>41</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
<td>0.6</td>
<td>1.0</td>
<td>4.8</td>
<td>2.6</td>
<td>78</td>
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<tr>
<td>6</td>
<td>71</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
<td>0.6</td>
<td>0.9</td>
<td>4.6</td>
<td>2.9</td>
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<td>7</td>
<td>68</td>
<td>0.9</td>
<td>1.2</td>
<td>33</td>
<td>0.7</td>
<td>0.9</td>
<td>4.6</td>
<td>3.5</td>
<td>56</td>
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<tr>
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<td>81</td>
<td>1.0</td>
<td>1.4</td>
<td>40</td>
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<td>1.1</td>
<td>4.0</td>
<td>2.0</td>
<td>88</td>
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<tr>
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<td>55</td>
<td>0.8</td>
<td>1.5</td>
<td>88</td>
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<td>0.8</td>
<td>5.3</td>
<td>2.6</td>
<td>88</td>
</tr>
<tr>
<td>10</td>
<td>53</td>
<td>1.0</td>
<td>1.6</td>
<td>60</td>
<td>0.6</td>
<td>1.0</td>
<td>5.3</td>
<td>3.0</td>
<td>82</td>
</tr>
</tbody>
</table>

Abbreviations: IVS = interventricular septum; d = diastole; s = systole; th = thickening; ex = excursion; LVPW = left ventricular posterior wall; LVID = left ventricular internal dimension; EF = ejection fraction; LVOT = left ventricular outflow tract.
ASH alone (P < 0.001 for all comparisons). Septal to posterior wall ratios (1.0 ± 0.1; range 0.9–1.1) were normal in all 10 patients with SAM but without ASH.

In patients found to have SAM without ASH, ejection fraction (79 ± 10%) was greater than normal and significantly increased when compared with patients with ASH and SAM (66 ± 16%, P < 0.02) or with patients with ASH alone (60 ± 15%, P < 0.001). LVOT measurements in patients found to have SAM without ASH (2.2 ± 0.4 cm) and in patients with ASH and SAM (2.1 ± 0.5 cm) were similar and smaller than in patients with ASH alone (2.8 ± 0.5 cm, P < 0.001). Examples of ASH alone and ASH and SAM are shown in figure 2.

**Left Ventricular Ejection Times**

LVETc increased by at least 40 msec following inhalation of amyl nitrite in six of seven patients (table 3) found to have SAM without ASH (fig. 3), in 20 of 25 patients with ASH and SAM, but in only one of 10 patients with ASH alone. The typical "spike-and-large-dome" carotid pulse contour was not always seen.

**Cardiac Catheterization Results**

LVOT gradients were demonstrated in four patients found to have SAM without ASH (table 3); one patient had a resting gradient of 35 mm Hg, and three patients had provable gradients of 68, 60, and 64 mm Hg (fig. 4). Six of 14 patients with ASH and SAM had resting gradients. The mean resting gradients in patients found to have SAM without ASH (9 ± 18 mm Hg) were statistically similar to resting gradients in patients with ASH and SAM (22 ± 26 mm Hg). The mean maximum provable LVOT gradients in patients having SAM without ASH (65 ± 6 mm Hg) and in patients with ASH and SAM (62 ± 34 mm Hg) were also

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**Table 2. Comparison of the Echocardiographic Measurements of Patients Found to Have SAM Without ASH, ASH and SAM, and ASH Alone**

<table>
<thead>
<tr>
<th></th>
<th>P*</th>
<th>ASH and SAM</th>
<th>SAM</th>
<th>ASH</th>
<th>Pt</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVS Diastole (cm)</td>
<td>&lt;0.001</td>
<td>1.8 ± 0.3</td>
<td>0.9 ± 0.1</td>
<td>1.7 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systole (cm)</td>
<td>&lt;0.001</td>
<td>1.8 ± 0.4</td>
<td>1.4 ± 0.1</td>
<td>1.8 ± 0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thickening (%)</td>
<td>&lt;0.001</td>
<td>5 ± 10</td>
<td>46 ± 16</td>
<td>5 ± 12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Excursion (cm)</td>
<td>&lt;0.001</td>
<td>0.2 ± 0.3</td>
<td>0.7 ± 0.2</td>
<td>0.3 ± 0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVPW thickness (cm)</td>
<td>NS</td>
<td>1.1 ± 0.2</td>
<td>1.0 ± 0.1</td>
<td>1.0 ± 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>IVS/LVPW</td>
<td>&lt;0.001</td>
<td>1.7 ± 0.4</td>
<td>1.0 ± 0.1</td>
<td>1.7 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVOT Diastole (cm)</td>
<td>&lt;0.02</td>
<td>3.8 ± 0.6</td>
<td>4.6 ± 0.5</td>
<td>4.3 ± 0.7</td>
<td>NS</td>
</tr>
<tr>
<td>Systole (cm)</td>
<td>&lt;0.02</td>
<td>2.6 ± 0.5</td>
<td>2.7 ± 0.5</td>
<td>3.1 ± 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>EF (%)</td>
<td>&lt;0.02</td>
<td>66 ± 16</td>
<td>79 ± 10</td>
<td>60 ± 15</td>
<td>&lt;0.001</td>
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</tbody>
</table>

*The statistical significance of the comparison between patients having SAM without ASH and patients with ASH and SAM.
†The statistical significance of the comparison between patients having SAM without ASH and those with ASH alone.

Abbreviations: IVS = interventricular septal thickness; LVPW = left ventricular posterior wall thickness; LVOT = left ventricular outflow tract.

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**Figure 1. A forty-one-year-old male with systolic anterior motion (SAM) of the mitral valve in the absence of asymmetric septal hypertrophy.** RS and LS define the right and left sides of the septum, respectively. In Panel A a chordal structure (CT) lies anterior to RS; its motion differs from RS particularly at the onset of systole. Septal thickness measured below the mitral valve before atrial contraction is 1.0 cm (panel A). Left ventricular posterior wall thickness measured at the level of the mitral valve before atrial contraction is also 1.0 cm (panel B). The small resting SAM (large arrow in panel B) increases markedly following amyl nitrite inhalation (large arrow in panel C). SAM ends prior to the aortic component of the second heart sound (A2). The scale in panel C differs from the scale in panels A and B. Endo = endocardium, E-P = epicardium, SM = systolic murmur, LSB-HF = left sternal border high frequency.
FIGURE 2. Example (a) is an echogram of a patient with a hypertrophic obstructive cardiomyopathy showing both ASH and SAM; septal and LV posterior wall thicknesses as measured before atrial contraction were 2.0 cm and 1.3 cm, respectively. Example (b) is an echogram of a patient with ASH but without LV outflow tract obstruction; septal and posterior wall thicknesses as measured before atrial contraction were 2.5 cm and 1.3 cm, respectively. IVS = interventricular septum, LGPW = left ventricular posterior wall, MV = mitral valve, CPT = carotid pulse tracing, LSB-MF = left sternal border medium frequency, ACG = apexcardiogram.

Discussion

The original descriptions of IHSS defined the hemodynamic abnormalities.\textsuperscript{14} The most characteristic abnormality was LVOT obstruction, which tended to increase whenever left ventricular contractility was increased or left ventricular preload or afterload was decreased. In most patients the pulse pressure tended to decrease\textsuperscript{15} or the left ventricular ejection time to increase whenever the obstruction increased.\textsuperscript{16} The LVOT obstruction in IHSS is attributed to SAM of the septal leaflet of the mitral valve and a bulge of the septum into the outflow tract.\textsuperscript{14} These characteristic features can be seen easily echocardiographically.

FIGURE 3. In this forty-one-year-old male, whose echogram is shown in figure 1, following amyl nitrite inhalation the uncorrected left ventricular ejection time (LVET) increased from 305 msec to 355 msec. The LVET\textsubscript{c} (LVET corrected for heart rate) increased from 403 msec to 501 msec. Note the appearance of the systolic murmur following amyl nitrite recorded at the left sternal border (LSB) and apex. MF = medium frequency, CPT = carotid pulse tracing.
TABLE 3. Left Ventricular Ejection Time (LVET) Prolongation and Outflow Tract Gradients in 10 Patients with SAM Alone

<table>
<thead>
<tr>
<th>Patient</th>
<th>LVET prolongation</th>
<th>Maximum gradient</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt;40 msec</td>
<td>60 mmHg</td>
</tr>
<tr>
<td>2</td>
<td>&gt;40 msec</td>
<td>64 mmHg</td>
</tr>
<tr>
<td>3</td>
<td>not done</td>
<td>68 mmHg</td>
</tr>
<tr>
<td>4</td>
<td>&lt;40 msec</td>
<td>70 mmHg</td>
</tr>
<tr>
<td>5</td>
<td>&gt;40 msec</td>
<td>not done</td>
</tr>
<tr>
<td>6</td>
<td>not done</td>
<td>not done</td>
</tr>
<tr>
<td>7</td>
<td>&gt;40 msec</td>
<td>not done</td>
</tr>
<tr>
<td>8</td>
<td>not done</td>
<td>not done</td>
</tr>
<tr>
<td>9</td>
<td>&gt;40 msec</td>
<td>not done</td>
</tr>
<tr>
<td>10</td>
<td>&gt;40 msec</td>
<td>not done</td>
</tr>
</tbody>
</table>

The anatomic abnormalities also have been described. 17, 18 The septum was found to be disproportionately thickened, and the papillary muscles were displaced anteriorly. With the advent of echocardiography, asymmetric septal hypertrophy has come to be regarded as the hallmark of IHSS. Obstructive cardiomyopathy is considered to be one presentation of a disease spectrum that also includes nonobstructive cardiomyopathy and the asymptomatic state. 19 Genetic ASH is an autosomal dominant trait, and most patients with genetic ASH are asymptomatic. 20 Disproportionate septal thickening can occur secondary to increases in right and left ventricular wall stresses; some of these patients may also have IHSS hemodynamics. 21 Genetic (usually) or secondary (rarely) ASH is regarded as a prerequisite for the development of the LVOT obstruction of IHSS.

Three theories have been proposed to explain the mechanism of SAM of the mitral valve and outflow tract obstruction. Pridie and Oakley 22 suggested that an abnormally directed and dysynchronous papillary muscle contraction could pull the anterior mitral leaflet into the left ventricular outflow tract during systole. This could cause both the outflow tract obstruction and the mitral regurgitation seen in IHSS.

Henry et al. 23 showed that contraction of the misaligned papillary muscles does not cause the abnormal mitral valve motion. They found that forward displacement of the mitral valve narrows the ventricular outflow tract with the onset of systole. Patients with obstructive ASH had narrower outflow tracts than patients with nonobstructive ASH. Postulated factors contributing to the forward positioning of the mitral valve at the onset of systole included abnormal tethering of the mitral valve by abnormally positioned papillary muscles prior to left ventricular contraction, and distortion of the left ventricular cavity caused by extreme septal hypertrophy. Abnormal hydrodynamic forces may be generated as blood is ejected through this narrowed left ventricular outflow tract. Left ventricular ejection rates are increased in both obstructive and nonobstructive hypertrophic cardiomyopathies; most of ventricular ejection occurs during early systole. 24 Increased flow velocity through the narrowed outflow tract may result in a Venturi effect. The mitral valve may then be sucked up into the outflow tract to produce the obstruction and the gradient. Alternatively, the Venturi effect may generate a pressure gradient as a primary event; the higher pressure behind the leaflet might force it into the outflow tract as a secondary event. With current techniques, it is not possible to tell whether SAM is a primary event or an event secondary to the outflow tract gradient.

FIGURE 4. Simultaneous left ventricular (LV) and aortic (Ao) pressures from the patient shown to have SAM without ASH in figure 6. Resting hemodynamics are shown in the left hand panel; although no resting gradient is present, following a PVC (open arrow), a gradient occurs along with diminution of the aortic pulse pressure (solid arrow). The positive Brockenbrough sign is specific for the dynamic LVOT obstruction typical of IHSS. The right hand panel demonstrates a gradient following amyl nitrite.
Using two-dimensional echocardiography Rodger has presented evidence that SAM, as visualized by unidimensional echocardiography, does not represent mitral valve motion alone. He suggested that SAM is a complex of echoes from, first, the posterior papillary muscle; second, the chordae tendinae; and last and least, the mitral valve. The posterior papillary muscle was found to be displaced superiorly and anteriorly and, in systole, to move upward and in front of the tip of the mitral valve into the left ventricular outflow tract. The abnormal papillary muscle position and motion allowed chordal slackening. Forward and upward motion of the slackened chordae during left ventricular ejection was found to cause the systolic anterior motion of the mitral valve. The valve continued to move anteriorly until it collided with the posterior papillary muscle and the chordae. This collision was seen to occur in the outflow tract, thus causing the obstruction. Rodger did not explain why the posterior papillary muscle moves forward in this disease. Indeed, the posterior papillary muscle motion may be secondary to the abnormal systolic mitral valve motion.

Although the mechanism of SAM has not been proved conclusively, each of these theories is based primarily on the existence of asymmetric ventricular hypertrophy. However, SAM has been shown to occur in a patient in hypovolemic shock without echocardiographic evidence of ASH; the heart was normal at autopsy. SAM has also been reported to occur in other disease states. However, many of these patients did not have true SAM. Rather, they had what has come to be known as pseudo-SAM. True SAM is an abrupt systolic anterior motion of the mitral valve out of proportion to the motion of the left ventricular posterior wall ending at or before the end of systole or before the aortic component of the second heart sound. Pseudo-SAM is an exaggeration of the normal anterior motion of the mitral valve during systole that parallels the left ventricular posterior wall motion during systole and into diastole (fig. 5). Pseudo-SAM may be seen in many disease states, including pericardial effusion, atrial septal defect, mitral valve prolapse, and ventricular aneurysm. Besides hypovolemia, true SAM has been reported to occur in dextroposition of the great vessels with subpulmonic obstruction and membranous subaortic stenosis. However, the significance of true SAM in the absence of ASH is not known.

Our data conclusively show that SAM of the mitral valve, systolic septal-mitral valve apposition, and IHSS hemodynamics can exist in the absence of an echocardiographically demonstrable thickened septum. Septal thickness, systolic septal thickening, and systolic septal excursion in these patients are all normal. Four patients with SAM alone were studied during cardiac catheterization and had resting or provokable gradients. Corrected left ventricular ejection time prolongation of more than 40 msec after inhalation of amyl nitrite was demonstrated in two of these patients and in four more patients. This has been suggested by Cohn et al. as a sensitive and specific marker for the hemodynamics of IHSS; normals may exhibit corrected LVET prolongation following amyl nitrite, but this rarely exceeds 40 msec. In our 10 patients found to have SAM without ASH, the outflow tracts were similar to those in patients with obstructive ASH. These 10 patients were also found to have significantly increased ejection fractions, echocardiographic evidence for increased ventricular dynamics. The influence of outflow tract size and ventricular wall kinetics on the genesis of SAM is most evident in patients without ASH and without a resting SAM. Following provocation, the outflow tract narrows and the wall motion increases concomitantly with the occurrence of SAM (fig. 6). (Admittedly, it is possible that delineation of the right side of the septum may not have been accurate in every instance, and ASH may have been missed in some.)

Thus, ASH cannot be considered a criterion for diagnosing IHSS hemodynamics in all patients. A thickened septum may not be responsible for SAM and the left ventricular outflow tract obstruction. Although the exact mechanism of SAM is uncertain (indeed, it may vary from one individual to the next), the finding of SAM and IHSS hemodynamics in the absence of ASH suggests that the fundamental abnormalities causing the obstruction are abnormal ventricular ejection dynamics superimposed on the narrowed outflow tract.

The causes of outflow tract narrowing and abnormal ventricular ejection dynamics are not known. IHSS is a hemodynamic abnormality. Genetic ASH is a disease that, in approximately 20% of cases, leads to the development of IHSS hemodynamics. Genetic ASH is the most common underlying cause of IHSS hemodynamics. ASH may also

**Figure 5.** Pseudo SAM, indicated by the arrow, is an exaggeration of the normal systolic motion of the mitral valve that parallels the LV posterior wall during systole and into early diastole. RV = right ventricle, IVS = interventricular septum, LVPW = left ventricular posterior wall, MV = mitral valve, Ao = aorta, LA = left atrium.
occur sporadically or as a secondary event. Individuals without ASH may also present with hemodynamics similar to typical IHSS. Attention must, therefore, be focused on the vigorous posterior left ventricular wall motion, the increased left ventricular ejection fraction and ejection rate, the narrowed LVOT, and the small left ventricular volume. The unifying link among the various causes of this dynamic left ventricular outflow obstruction may not necessarily be ASH, but rather abnormal ventricular ejection dynamics.

References
Aortic Stenosis in Children

Experience with Echocardiographic Prediction of Severity


SUMMARY Fifty-six children with aortic stenosis were investigated both by echocardiography and cardiac catheterization. The ratio of end-systolic wall thickness (Ws) to internal diameter (LVES) across the minor axis of the LV as determined by echocardiography and multiplied by a factor of 245 predicts left ventricular pressure (r = 0.83). Subtracting the arm systolic blood pressure from the predicted intraventricular systolic pressure (PISP) gives a predicted systolic pressure gradient (r = 0.91).

The technique is found to be useful for both initial and sequential noninvasive assessment of aortic stenosis with normal LV function irrespective of the level of obstruction. It is shown to apply equally well to a wide range of LV pressures, is independent of volume load, and is useful in predicting small gradients. Some common problems encountered with the measurements are examined in detail.

NONINVASIVE PREDICTION OF LV PRESSURE has a wide application in some forms of congenital heart disease where it cannot be measured from a systemic artery, as with aortic stenosis, d-transposition of the great arteries, or coarctation of the aorta with abnormal brachial arteries due to anatomical variants or previous catheterization.

From a combination of the Laplace relationship, wall stress studies, and the concept of "relative" wall thickness, a simple formula has been developed to predict LV pressure by echocardiography. A similar study by Bennett et al. gave encouraging results for adults. This study of children with aortic stenosis was undertaken primarily to establish that the gradient that would be found at cardiac catheterization could be predicted by echocardiography.
Systolic anterior motion of the mitral valve in the absence of asymmetric septal hypertrophy.
G S Mintz, M N Kotler, B L Segal and W R Parry

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