Muscular Subaortic Stenosis: The Temporal Relationship Between Systolic Anterior Motion of the Anterior Mitral Leaflet and the Pressure Gradient

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SUMMARY Recent studies indicate that in patients with muscular subaortic stenosis at rest, left ventricular outflow tract obstruction is associated with severe systolic anterior motion (SAM) of the anterior mitral leaflet and prolonged SAM-septal contact. We correlated the temporal relationships between echocardiographic and hemodynamic events in 18 patients with muscular subaortic stenosis (gradient of 73 ± 18 mm Hg [mean ± sd]).

After the ECG R wave, aortic ejection began at 72 ± 12 msec and the onset of SAM 23 msec later, at 95 ± 22 msec. The onset of the pressure gradient at 162 ± 22 msec after the R wave was almost simultaneous with the onset of SAM-septal contact at 168 ± 28 msec. SAM-septal contact was maintained for 195 msec and ceased at 363 ± 41 msec after the R wave. Peak posterior left ventricular wall movement occurred at 387 ± 48 msec, 219 msec after peak SAM (the onset of SAM-septal contact). The excursion and mean rate of development of SAM from onset to septal contact (14 ± 2 mm and 208 ± 55 mm/sec, respectively) were almost three times the excursion and mean rate of inward movement of the posterior wall in the same period of systole (5 ± 1 mm and 75 ± 16 mm/sec, respectively).

In terms of the systolic ejection period, SAM began at 6.0 ± 6%, and the onset of the pressure gradient and SAM-septal contact were almost simultaneous, at 23 ± 5% and 25 ± 7%, throughout this period. The end of SAM-septal contact occurred at 76 ± 10% of the systolic ejection period and peak posterior left ventricular wall movement occurred at 82 ± 12%.

We conclude that the onset of SAM is a very early systolic event. The onset of the pressure gradient occurs just before or with the onset of SAM-septal contact, suggesting a cause-and-effect relationship. Posterior wall hyperkinesis plays no part in the genesis of SAM in these patients, judged by the differing rate and extent of excursion of SAM and the posterior wall and the fact that peak left ventricular wall movement occurs 219 msec after peak SAM (onset of SAM-septal contact). Tethering of the anterior mitral leaflet by the papillary muscles is not the cause of SAM, since SAM-septal contact ceases at 76 ± 10% of the systolic ejection period, whereas a tethering effect should last until end-systole. SAM is most likely caused by a Venturi effect related to rapid early systolic ejection.

IN THE 20 years since Bjork et al.1 observed an abnormal movement of the anterior leaflet of the mitral valve in patients with muscular subaortic stenosis (MSS), angiographic2-4 and echocardiographic5-6 demonstrations of systolic anterior motion (SAM) of the anterior mitral leaflet have confirmed this observation.

Much controversy has surrounded the significance and mechanism of SAM in patients with MSS. SAM was originally thought to produce left ventricular outflow tract obstruction in these patients;1-6 others have considered SAM to be the result of "cavity obliteration"7 and hyperkinetic posterior wall movement7-9 and, as such, did not believe that it caused outflow tract obstruction.10, 11 Several investigators2, 3, 12 have suggested that SAM is produced by papillary muscle contraction pulling the anterior mitral leaflet toward the septum. In 1970, Wigle et al.13 suggested that rapid ejection in the early phase of systole could draw the anterior mitral leaflet into the left ventricular outflow tract by a Venturi effect, causing outflow obstruction and mitral regurgitation.

More recent studies14, 15 indicate that all patients with MSS and evidence of left ventricular outflow tract obstruction at rest have prolonged systolic contact between the anterior leaflet of the mitral valve and the interventricular septum (prolonged SAM-septal contact). Thus, in 27 out of 27 cases of MSS with outflow tract obstruction at rest, SAM-septal contact always exceeded 30%, and averaged 54% of echocardiographic systole.15 We have classified this type of SAM as being severe by M-mode echocardiographic criteria,15 and this is equivalent to SAM involving the lower one-third to one-half of the anterior mitral leaflet on two-dimensional (2-D) echocardiographic examination.16

We studied the temporal relationships between these M-mode echocardiographic and hemodynamic events in patients with MSS at rest. In addition, to determine if SAM and posterior wall hyperkinesis in MSS are related, we compared the timing of peak SAM (onset of SAM-septal contact) when it first occurs with the timing of peak posterior wall movement, the excursion of SAM from onset to septal contact with the excursion of the posterior wall in the same phase of systole, and the mean rate of development of SAM from onset to septal contact with the mean rate of posterior-wall inward movement in the same phase of systole.
Finally, to determine whether SAM and the resultant SAM-septal contact is a result of traction of the papillary muscles on the anterior mitral leaflet or a Venturi effect, we observed the time of offset of SAM. If papillary muscle traction is the cause of SAM and, hence, SAM-septal contact, then SAM-septal contact should last as long as papillary muscle contraction, i.e., to end-systole. If the Venturi effect is the cause of SAM and SAM-septal contact, then one would expect SAM-septal contact to cease before end-systole because of a reduced ejection velocity, or because of a decrease in left ventricular pressure (if SAM-septal contact was being maintained by hemodynamic factors).

### Methods

#### Patients

We studied 18 patients with MSS (11 male and seven female, average 39 years). The diagnosis was based on established clinical, hemodynamic and echocardiographic criteria.\(^7\)

#### Hemodynamic Methods

Patients underwent both retrograde and transseptal left-heart catheterization using the percutaneous Sel-dinger technique from the right groin. Simultaneous left ventricular inflow and aortic root pressures were recorded using a transseptal catheter advanced through the mitral valve orifice into the left ventricular inflow tract and a retrograde catheter in the aortic root. In each case, care was taken to ensure that the recorded intraventricular pressure gradient indicated true outflow tract obstruction, and not catheter entrapment.\(^18,19\) Fluid-filled catheters connected to a Statham 23Db transducer were used and pressures were recorded on an Electronics for Medicine recorder. Fifteen patients had a gradient at rest. In three patients with latent obstruction, the timing studies were carried out in the presence of a provoked gradient during isoproterenol stimulation. The mean gradient for all 18 patients was 73 ± 18 mm Hg (mean ± SD).

#### Echocardiographic Methods

All patients underwent standard M-mode echocardiographic studies using a Smith-Kline Ekoline 20 ultrasonoscope interfaced with a Cambridge or an Irex recorder. A 2.25-MHz transducer 1.5 cm in diameter was used. All patients had echocardiographic evidence of asymmetric hypertrophy of the heart with septal–posterior wall ratios greater than 1.5.\(^15\) SAM of the anterior mitral leaflet was considered present when both anterior and posterior mitral leaflet echoes and the posterior left ventricular wall were observed.

#### Timing of Studies

**Group 1**

In patients 1–6 (group 1) (three with latent obstruction and three with resting obstruction), hemodynamic and echocardiographic studies were performed together so that hemodynamic and echocardiographic events could be timed simultaneously (table 1). The measurements for three to five beats were averaged in each patient.

**Group 2**

In patients 7–18 (group 2), the hemodynamic and echocardiographic studies were performed separately, usually a few days apart.

All patients had resting obstruction, characterized by a stable left ventricular outflow tract gradient at rest (table 2). We specifically excluded patients with labile obstruction, in whom the gradient varies markedly at rest. Such cases are rare in our experience. Because of the stability of the obstruction in these 12 patients, the pressure gradients during the hemodynamic and echocardiographic studies were probably similar. To minimize differences in physiologic states at these different times of investigation, we selected cardiac cycles from both studies with identical heart rates (i.e., cycle lengths to within 10 msec of each other).

Patient 3 also had a separate echocardiographic study performed several days beforehand. We chose

### Table 1. Sequence and Timing of Echocardiographic and Hemodynamic Events in Group 1 — Simultaneous Studies

<table>
<thead>
<tr>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset of aortic ejection</td>
<td>56</td>
<td>59</td>
<td>83</td>
<td>69</td>
<td>100</td>
<td>71</td>
<td>73 ± 16</td>
</tr>
<tr>
<td>Onset of SAM</td>
<td>113</td>
<td>77</td>
<td>109</td>
<td>69</td>
<td>128</td>
<td>69</td>
<td>94 ± 26</td>
</tr>
<tr>
<td>Onset of pressure gradient</td>
<td>170</td>
<td>142</td>
<td>187</td>
<td>186</td>
<td>194</td>
<td>211</td>
<td>182 ± 23</td>
</tr>
<tr>
<td>Onset SAM-septal contact</td>
<td>170</td>
<td>160</td>
<td>187</td>
<td>188</td>
<td>225</td>
<td>211</td>
<td>190 ± 24</td>
</tr>
<tr>
<td>Peak LV pressure</td>
<td>325</td>
<td>237</td>
<td>290</td>
<td>295</td>
<td>290</td>
<td>293</td>
<td>288 ± 28</td>
</tr>
<tr>
<td>End SAM-septal contact</td>
<td>382</td>
<td>409</td>
<td>368</td>
<td>400</td>
<td>391</td>
<td>367</td>
<td>386 ± 17</td>
</tr>
<tr>
<td>Peak PLVW movement</td>
<td>N/A</td>
<td>445</td>
<td>389</td>
<td>439</td>
<td>470</td>
<td>N/A</td>
<td>436 ± 34</td>
</tr>
<tr>
<td>End of pressure gradient</td>
<td>442</td>
<td>427</td>
<td>446</td>
<td>427</td>
<td>422</td>
<td>420</td>
<td>431 ± 11</td>
</tr>
<tr>
<td>Diastolic notch</td>
<td>454</td>
<td>445</td>
<td>477</td>
<td>478</td>
<td>452</td>
<td>445</td>
<td>458 ± 15</td>
</tr>
<tr>
<td>RR interval</td>
<td>760</td>
<td>710</td>
<td>930</td>
<td>790</td>
<td>685</td>
<td>821</td>
<td>70 ± 11</td>
</tr>
<tr>
<td>Gradient (mm Hg)</td>
<td>60</td>
<td>80</td>
<td>86</td>
<td>86</td>
<td>71</td>
<td>59</td>
<td>62</td>
</tr>
</tbody>
</table>

All values (except gradient) are in msec.

Abbreviations: SAM = systolic anterior motion; LV = left ventricle; PLVW = posterior left ventricular wall; N/A = not assessable.
Table 2. Sequence and Timing of Echocardiographic and Hemodynamic Events in Group 2 — Nonsimultaneous Studies

<table>
<thead>
<tr>
<th>Patient</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
<th>16</th>
<th>17</th>
<th>18</th>
<th>Mean ± SD</th>
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<tr>
<td>Onset of aortic ejection</td>
<td>67</td>
<td>85</td>
<td>79</td>
<td>69</td>
<td>62</td>
<td>62</td>
<td>67</td>
<td>57</td>
<td>76</td>
<td>77</td>
<td>65</td>
<td>88</td>
<td>71 ± 10</td>
</tr>
<tr>
<td>Onset of SAM</td>
<td>89</td>
<td>85</td>
<td>79</td>
<td>138</td>
<td>82</td>
<td>102</td>
<td>89</td>
<td>91</td>
<td>109</td>
<td>110</td>
<td>117</td>
<td>58</td>
<td>96 ± 21</td>
</tr>
<tr>
<td>Onset of pressure gradient</td>
<td>179</td>
<td>149</td>
<td>178</td>
<td>149</td>
<td>133</td>
<td>154</td>
<td>145</td>
<td>137</td>
<td>153</td>
<td>153</td>
<td>141</td>
<td>151</td>
<td>152 ± 14</td>
</tr>
<tr>
<td>Onset SAM-septal contact</td>
<td>179</td>
<td>128</td>
<td>119</td>
<td>184</td>
<td>164</td>
<td>144</td>
<td>134</td>
<td>182</td>
<td>153</td>
<td>153</td>
<td>164</td>
<td>185</td>
<td>157 ± 23</td>
</tr>
<tr>
<td>Peak LV pressure</td>
<td>235</td>
<td>298</td>
<td>297</td>
<td>229</td>
<td>267</td>
<td>246</td>
<td>307</td>
<td>330</td>
<td>338</td>
<td>263</td>
<td>282</td>
<td>268</td>
<td>280 ± 35</td>
</tr>
<tr>
<td>End SAM-septal contact</td>
<td>335</td>
<td>341</td>
<td>396</td>
<td>413</td>
<td>328</td>
<td>308</td>
<td>268</td>
<td>399</td>
<td>393</td>
<td>329</td>
<td>377</td>
<td>322</td>
<td>351 ± 44</td>
</tr>
<tr>
<td>Peak PLVW movement</td>
<td>358</td>
<td>384</td>
<td>396</td>
<td>459</td>
<td>349</td>
<td>328</td>
<td>313</td>
<td>387</td>
<td>393</td>
<td>351</td>
<td>400</td>
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<td>371 ± 40</td>
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<tr>
<td>End of pressure gradient</td>
<td>425</td>
<td>426</td>
<td>426</td>
<td>396</td>
<td>369</td>
<td>339</td>
<td>380</td>
<td>444</td>
<td>397</td>
<td>439</td>
<td>424</td>
<td>381</td>
<td>408 ± 34</td>
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<tr>
<td>Dicrotic notch</td>
<td>492</td>
<td>469</td>
<td>485</td>
<td>430</td>
<td>415</td>
<td>421</td>
<td>425</td>
<td>467</td>
<td>486</td>
<td>472</td>
<td>471</td>
<td>410</td>
<td>454 ± 31</td>
</tr>
<tr>
<td>RR interval</td>
<td>800</td>
<td>880</td>
<td>1020</td>
<td>760</td>
<td>960</td>
<td>960</td>
<td>800</td>
<td>760</td>
<td>840</td>
<td>840</td>
<td>730</td>
<td>1070</td>
<td></td>
</tr>
<tr>
<td>Gradient (mm Hg)</td>
<td>44</td>
<td>48</td>
<td>71</td>
<td>58</td>
<td>65</td>
<td>70</td>
<td>98</td>
<td>80</td>
<td>102</td>
<td>91</td>
<td>70</td>
<td>104</td>
<td>75 ± 20</td>
</tr>
</tbody>
</table>

All values (except gradient) are in msec.
Abbreviations: SAM = systolic anterior motion; LV = left ventricular; PLVW = posterior LV wall.

10 cardiac cycles from both echocardiographic studies with identical heart rate and compared the timing of the onset of SAM-septal contact after the R wave. During cardiac catheterization this was 181 ± 2 msec. In the study several days beforehand it was 178 ± 2 msec (NS). Thus, this patient, whom we believe to be representative of the whole group, showed no difference in the timing of this echocardiographic event taken several days apart. Finally, to ensure that both echocardiographic and hemodynamic studies were comparable, patients who were taking β blockers discontinued them at least 48 hours before both studies.

In group 2, we could usually find only one cycle per patient for correlation between the hemodynamic and echocardiographic events, because of the requirement of selecting cardiac cycles with identical lengths. When we could find more than one cycle of identical length in the echocardiographic and hemodynamic studies in any one patient, the timing of events correlated to within 10 msec of the other cycles measured in that patient.

Timing of Events

Hemodynamic Events

From the simultaneous left ventricular inflow and central aortic pressure tracings, five events were timed from the peak of ECG R wave (fig. 1): (1) Onset of aortic ejection. (2) Onset of the pressure gradient. This was defined by the point where left ventricular and aortic pressures diverged. This divergence occurred at the peak of the aortic percussion wave, beyond which the left ventricular pressure rose and the aortic pressure fell (fig. 1). In some patients (fig. 2), an early, smaller gradient, the "impulse gradient," was recorded between the left ventricle and aorta, in the period of early systole from the onset of aortic ejection to the peak of the percussion wave. However, in these patients, the main divergence of left ventricular and aortic pressures still occurred at the peak of the percussion wave. Thus, the peak of the percussion wave was taken as the onset of the pressure gradient in all patients. (3) Peak left ventricular pressure. (4) End of the pressure gradient. (5) Dicrotic notch.

Echocardiographic Events

Four echocardiographic events were timed from the peak of the R wave (fig. 3): onset of SAM, the onset of SAM-septal contact (peak SAM), the end of SAM-septal contact, and peak point of posterior left ventricular wall systolic movement.

To permit interpatient correlation all time intervals

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**Figure 1.** Simultaneous left ventricular (LV) and aortic pressure tracing from a patient with muscular subaortic stenosis indicating the hemodynamic events that were timed in this study from the R wave (see text). (1) Onset of aortic ejection. (2) Onset of the pressure gradient. (3) Peak LV pressure. (4) End of the pressure gradient. (5) Dicrotic notch.
were corrected for heart rate by dividing by the square root of cycle length. Both hemodynamic and echocardiographic studies were usually recorded at paper speeds of 50–100 mm/sec.

Extent and Rate of Excursion of Sam and the Posterior Wall

The anterior excursion of SAM was measured from the onset of SAM (fig. 3A) to SAM-septal contact (fig. 3B). The anterior excursion of the posterior left ventricular wall in the same period of systole (onset of SAM to SAM-septal contact) was also determined.

The excursion of SAM and the posterior wall was divided by the time period: onset of SAM to SAM-septal contact, to obtain the mean rate of development of SAM and the mean rate of posterior wall inward movement in this phase of systole.

Statistical analysis was performed using the paired t test. Data are expressed as mean ± SD.

Results

Timing of Events

The sequence and timing of events after the peak of the R wave corrected for heart rate are shown for individual patients in groups 1 and 2 in tables 1 and 2. The sequence and relationship between hemodynamic and echocardiographic events was the same in both groups. Thus, the data for all 18 patients have been grouped together (table 3, fig. 4) and the mean data are considered below.

Onset of Aortic Ejection and SAM

The onset of aortic ejection occurred at 72 ± 12 msec after the R wave, followed 23 msec later by the onset of SAM, at 95 ± 22 msec (table 3, fig. 4). In terms of the systolic ejection period, the onset of SAM was a very early systolic event, occurring at just 6 ± 6% of this period (table 3).

Onset of the Pressure Gradient and SAM-Septal Contact

The onset of the pressure gradient was almost simultaneous with the onset of SAM-septal contact. Thus, the onset of the pressure gradient occurred at 162 ± 22 msec, and the onset of SAM-septal contact occurred at 168 ± 28 msec after the peak of the R wave (table 3, fig. 4). In terms of the systolic ejection period, the onset of the pressure gradient occurred at 23 ± 5% and the onset of SAM-septal contact at 25 ± 7% of this period (table 3).

In eight patients, the time of onset of the pressure gradient and the time of onset of SAM-septal contact were within 10 msec of each other (including four of the patients examined with simultaneous hemodynamic and echocardiographic studies) (table 1, fig. 2); in seven patients, the onset of the pressure gradient began just before the onset of SAM-septal contact, by 31 ± 9 msec (range 18–45 msec); in the remaining three patients, the onset of the pressure gradient began just after the onset of SAM-septal contact, by a mean of 30 ± 25 msec (range 11–59 msec).

Paired analysis revealed that there was no significant difference between the time of onset of SAM-
septal contact and the time of onset of the pressure gradient.

**SAM and the Posterior Left Ventricular Wall Movement**

All patients showed a marked temporal disparity between the onset of peak SAM (SAM-septal contact) and the point of peak posterior left ventricular wall movement. The onset of SAM-septal contact occurred at 168 ± 28 msec and the peak point of posterior left ventricular wall movement at 387 ± 48 msec after the peak of the R wave. In terms of the systolic ejection period, the onset of SAM-septal contact occurred at 25 ± 7% through this period, while peak posterior left ventricular wall movement occurred near the end of this period (at 82 ± 12%).

Paired analysis revealed a significant difference between the timing of the onset of SAM-septal contact and the peak point of posterior left ventricular wall movement (p < 0.001).

**Offset of SAM-Septal Contact**

The offset of SAM-septal contact occurred before the end of the systolic ejection period in all patients. The end of SAM-septal contact occurred at 363 ± 41 msec, and the dicrotic notch 455 ± 26 msec after the peak of the R wave. The end of SAM-septal contact occurred at 76 ± 10% and, by definition, the dicrotic notch at 100% of the systolic ejection period.

**Extent and Rate of Excursion of SAM and the Posterior Wall**

The excursion of SAM from onset of SAM to SAM-septal contact was 14 ± 2 mm, compared with a posterior left ventricular wall excursion in the same period of systole of 5 ± 1 mm (p < 0.01) (fig. 5).

The mean rate of development of SAM was 208 ± 55 mm/sec and the mean rate of posterior left ventricular wall inward movement in this same period of systole was 75 ± 16 mm/sec (p < 0.01) (fig. 5).

**Discussion**

Patients with MSS and a true resting left ventricular outflow tract pressure gradient have severe SAM, defined as SAM with septal contact for 30% or more of echocardiographic systole.15

We examined the temporal relationship between severe SAM and the pressure gradient in patients with MSS. As a group, the onset of SAM-septal contact was almost simultaneous with the onset of the pressure gradient. Thus, the onset of the pressure gradient occurred at 162 ± 22 msec and the onset of SAM-septal contact at 168 ± 28 msec after the peak of the R wave (table 3, figs. 2 and 4). In terms of the systolic ejection period, the onset of the pressure gradient occurred at 23 ± 5% and the onset of the SAM-septal contact occurred at 25 ± 7% of this period (table 3). The accuracy of these measurements may be affected by the
potential measuring error of 10 msec associated with a paper speed of 50 mm/sec and by a delay in the pressure recordings that occurs with a fluid-filled system which, in a previous study, was 8 ± 2 msec.25 However, we believe that such considerations would not change the striking temporal relationship between these two events, and paired analysis revealed no significant difference between the time of onset of SAM-septal contact and the time of onset of the pressure gradient, suggesting a cause-and-effect relationship between these two events. Thus, we suggest that the left ventricular outflow tract pressure gradient in patients with MSS develops as a result of the substantial reduction in left ventricular outflow tract size that occurs just before or with the onset of SAM-septal contact.

In a concomitant study of left anterior oblique left ventricular cineangiograms in patients with MSS at rest,22 the radiolucent line in the outflow tract that indicates angiographic SAM-septal contact occurred at 37.5% of the time period from end-diastole to end-systole. This is almost identical to the time of onset of echocardiographic SAM-septal contact which, in the present study, occurred at 36.9% of the period from the peak of the R wave to the dicrotic notch. The present study and the cineangiographic studies confirm previous work23, 24 that the pressure gradient begins early in systole, and therefore in a ventricle that still has to eject most of its stroke volume. In one angiographic study,22 34% of left ventricular emptying occurred before the radiolucent line of angiographic SAM-septal contact, while 66% of left ventricular emptying occurred in the presence of the radiolucent line.

Based on the present study and the cineangiographic study, we believe that SAM-septal contact produces obstruction to left ventricular outflow, causing approximately 66% of left ventricular emptying to occur in the presence of a systolic overload. These findings are similar to those of Ross et al.24 who found that 70% of forward flow occurred in the presence of the pressure gradient. The significance of this hemodynamic burden in patients with MSS at rest is reflected by the prolonged left ventricular ejection time,25, 26 and the compensatory posterior wall hypertrophy.27 Both of these features of obstruction to left ventricular outflow return toward normal after surgical relief of the MSS.26, 27

Our results are in accord with those of Glasgow et al.,28 who recently performed echocardiographic and Doppler flow studies in 12 patients with MSS. They demonstrated a sharp decrease in aortic flow at the onset of SAM-septal contact and that 47 ± 17% of the total left ventricular diameter shortening occurred after the onset of SAM-septal contact. Retrograde flow was detected into the left atrium (mitral regurgitation).

Murgo et al.29 questioned the importance of the pressure gradient in hypertrophic cardiomyopathy. They reported no difference in the ratio of forward flow time/systolic ejection period between three groups of patients with and without pressure gradients. By using this ratio, however, the direct relationship between the pressure gradient and systolic ejection period is masked.26, 30, 31 Indeed, the data indicate that prolongation of the ejection period is associated with a prolongation in forward flow time and angiographic left ventricular emptying in the group with "resting gradients." Differences between the groups may have been minimized by including two patients with resting gradients less than 20 mm Hg in whom the ejection period was probably normal. In addition, the lack of a significant difference between the groups may partially reflect a beta error due to the relatively small sample size.

This study indicates that severe SAM is unlikely to be the result of a direct pushing action of the posterior left ventricular wall. First, there is a marked temporal disparity between the initial development of peak SAM (onset of SAM-septal contact), which occurs at 25 ± 7% of the systolic ejection period, and peak posterior wall movement, which occurs at 82 ± 12% of the systolic ejection period (p < 0.001). Second, the mean rate of development of SAM (208 ± 55 mm/sec) is almost three times the mean rate of inward posterior wall movement (75 ± 16 mm/sec) (p < 0.01). Third, the extent of excursion of SAM from its onset to SAM-septal contact was 14 ± 2 mm, whereas posterior wall excursion in this same period was only 5 ± 1 mm (p < 0.01). These gross discrepancies between the timing, rate and extent of movement of SAM vs the posterior left ventricular wall refute the suggestion that posterior wall hyperkinesis is the cause of SAM in patients with hemodynamically proved MSS.

Some reports1, 3, 12 have suggested that SAM is caused by a tethering action of the papillary muscles drawing the anterior mitral leaflet into the outflow tract and against the septum. If this were the case, SAM-septal contact should be maintained as long as the papillary muscles are contracting, i.e., until end-systole. The present study, however, indicates that the offset of SAM-septal contact occurs at 76% of the systolic ejection period. This fact casts serious doubt that papillary muscle traction on the anterior mitral leaflet causes SAM. Furthermore, the concept that papillary muscle traction causes SAM has been seriously questioned by two-dimensional echocardiographic studies,16, 32 which demonstrate that the distal one-third to one-half of the anterior mitral leaflet bends at a right angle to both the proximal part of the leaflet and to the long axis of the papillary muscles.

In 1970, Wigle et al.15 suggested that rapid ejection in the early nonobstructive phase of systole could draw the anterior mitral leaflet into the left ventricular outflow tract by a Venturi effect, producing obstruction to ventricular outflow and mitral regurgitation in patients with MSS. The left ventricular outflow tract is narrowed at the onset of systole14-18 and rapid early ejection occurs in these patients.33 The combination of these features in patients with MSS at rest would promote a Venturi effect on the anterior mitral leaflet. We suggest that by this Venturi effect, the anterior mitral leaflet is sucked rapidly toward the septum; it is kept sustained against the septum either by continued Ven-
turi-induced suction or by the left ventricular systolic pressure. In late systole, the anterior mitral leaflet falls away from the septum because ventricular ejection velocity decreases or ventricular pressure decreases or both.

Our results are seemingly at variance with those of Chahine et al., who could find no relationship between SAM and the pressure gradient. However, with the hemodynamic techniques they used, they could not distinguish between true left ventricular outflow obstruction and cavity obliteration with catheter entrapment. Also, SAM was only described as being absent or present, without specifying the degree.

In a recent study of 74 patients with hypertrophic cardiomyopathy, we found that all 27 patient who had MSS at rest had early and prolonged SAM-septal contact (severe SAM) by M-mode echocardiography. In this same study, patients with latent MSS or hypertrophic nonobstructive cardiomyopathy had either no SAM or, at most, mild-to-moderate SAM (without prolonged SAM-septal contact).

One must recognize that SAM may occur in normal persons and in many conditions other than hypertrophic cardiomyopathy. In most of these situations, the SAM is mild or, at most, moderate, and two-dimensional echocardiographic examination of such patients reveals that SAM has a chordal or leaflet tip origin, whereas severe SAM is produced by the lower one-third to one-half of the body of the anterior mitral leaflet. In a few situations other than hypertrophic cardiomyopathy, severe SAM has also been demonstrated, with concomitant left ventricular outflow tract obstruction.

In summary, we suggest that SAM with early and prolonged septal contact in these patients is created by a Venturi mechanism and that septal contact is maintained by either continued Venturi-induced suction or by the left ventricular systolic pressure, resulting in both the left ventricular outflow tract obstruction and the mitral regurgitation that accompanies it.

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Angiocardiography of Multiple Ventricular Septal Defects in Infancy
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SUMMARY  Biplane cineventriculography in 364 infants 1 year of age or younger demonstrated multiple ventricular septal defects (VSDs) in 56 (15%). Among 111 infants with VSDs (with or without patent ductus arteriosus), 18 (16%) had multiple VSDs, whereas 14 of 39 infants (36%) with VSD and coarctation of the aorta had multiple VSDs. The incidence of multiple VSDs in infants with tetralogy of Fallot was 7% (eight of 117), in infants with d-transposition of the great arteries 19% (eight of 43), and in infants with common atrioventricular canal 15% (eight of 54). Perioperative axially angled cineangiography correctly predicted the presence of multiple VSDs in 13 of 15 infants (86%) who underwent operation.

THE MORTALITY RATE for surgery of congenital heart disease when associated with multiple ventricular septal defects (MVSDs) is 14–17%, 1,2 compared with 4% when a single VSD is present. 3 With the trend in many medical centers toward surgical correction of congenital heart defects during infancy, 1,4,5 precise preoperative diagnosis in babies is becoming increasingly important. Our previous experience, in which the presence of MVSDs was identified preoperatively in only 55% of patients operated upon, 1 indicates that preoperative diagnosis must be improved.

This study was undertaken to determine the incidence of MVSDs in infants with ventricular septal defect (VSD), VSD and coarctation (VSD/CoAo), tetralogy of Fallot (T/F), d-transposition (d-TGA) and common atrioventricular canal (CAVC) using cineangiography. A second objective was to evaluate whether the use of axially angled views improved preoperative diagnosis. Additionally, the incidence of spontaneous closure of one or more of these defects was examined among those with VSD and VSD/CoAo by clinical observation and subsequent cardiac catheterization, and among those with d-TGA by repeat cineangiography.

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