Abnormal Left Ventricular Intracavitary Flow Acceleration in Patients Undergoing Aortic Valve Replacement for Aortic Stenosis
A Marker for High Postoperative Morbidity and Mortality

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**Background.** We examined the clinical and echocardiographic characteristics of patients undergoing aortic valve replacement for aortic stenosis whose continuous wave Doppler studies showed abnormal intracavitary flow acceleration.

**Methods and Results.** The clinical and Doppler echocardiographic records of 53 consecutive patients undergoing aortic valve replacement for aortic stenosis were reviewed. Doppler echocardiography was performed at a mean of 6.6 days (range, 0–22 days) after surgery. Thirteen patients (group 1) had a dagger-shaped high-velocity systolic flow signal indicative of abnormal intracavitary flow acceleration on their postoperative Doppler study; group 2 comprised 40 aortic stenosis patients who underwent aortic valve replacement but had no postoperative evidence of abnormal intracavitary flow acceleration. Group 1 postoperative abnormal intracavitary flow velocities ranged from 1.8 to 6.8 m/sec (mean, 4.9±0.9 m/sec): Resulting dynamic gradients ranged from 10 to 184 mm Hg (mean, 104.6±32 mm Hg). Compared with group 2, group 1 patients had a distinctive ventricular geometry with more-pronounced hypertrophy, smaller cavities, and higher ejection fraction. Systolic anterior motion of the mitral valve did not accompany abnormal intracavitary flow acceleration in any patient. Six of 13 group 1 patients suffered postoperative hemodynamic compromise characterized by severe hypotension despite adequate pulmonary capillary wedge pressures; group 1 postoperative mortality was significantly greater than that seen in group 2 patients (38% versus 12%, p<0.05).

**Conclusions.** Abnormal intracavitary flow acceleration after aortic valve replacement for severe aortic stenosis is associated with a distinctive ventricular geometry and supernormal systolic function but not systolic anterior motion of the mitral valve. Such flow acceleration appears to be a marker for increased postoperative morbidity and mortality. Preoperative and postoperative Doppler echocardiography may be useful in risk stratification and guiding therapy. (*Circulation* 1992;86:926–936)

**KEY WORDS** echocardiography, Doppler, stenoses, hypertrophy

Valvular and subvalvular dynamic obstructions may coexist in patients with critical aortic stenosis.1–9 Initial studies have detailed the clinical features of this syndrome3,4,6,7 and demonstrated that the hemodynamic changes after aortic valve replacement may exacerbate preexisting dynamic outflow obstruction with life-threatening consequences. Echocardiography has permitted the preoperative, noninvasive description of left ventricular structure in patients with the syndrome of dynamic subvalvular obstruction complicating aortic valve disease. In some instances, the echocardiographic features appear to be similar to those encountered in patients with hypertrophic obstructive cardiomyopathy and include marked asymmetric hypertrophy of the interventricular septum, narrowed left ventricular outflow tract, and systolic anterior motion of the mitral valve.3 In addition, recent reports8–10 have described a continuous wave Doppler “dagger-shaped” velocity signal (similar to that used to quantitate the intracavitary pressure gradient in hypertrophic obstructive cardiomyopathy11,12) in patients with coexistent aortic stenosis and evidence of postoperative dynamic outflow obstruction. However, neither the prevalence, clinical significance, nor preoperative left ventricular structure of patients with these findings has been examined. We therefore examined preoperative and postoperative Doppler echocardiograms in patients undergoing aortic valve replacement for aortic stenosis to investigate the clinical features and ejection dynamics associated with abnormal intracavitary flow acceleration.

**Methods**

**Patient Population**

The records of 53 consecutive patients undergoing aortic valve replacement for aortic stenosis from July
1987 to October 1990 who also had postoperative two-dimensional Doppler echocardiography were reviewed. Postoperative studies were performed at an average of 6.6 days after aortic valve replacement (range, 0–22 days). Postoperative echocardiographic studies were reviewed to identify Doppler echocardiographic evidence of asymmetric hypertrophy, systolic anterior motion of the mitral valve, chordae tendineae, or abnormal intracavitary late systolic flow acceleration characterized by a dagger-shaped contour of the left ventricular outflow tract signal (Figure 1). Patients with prior aortic valve surgery or concomitant mitral valve replacement or repair were excluded from analysis.

### Clinical Characteristics

Clinical and demographic information was obtained from review of medical, catheterization, and surgical records.

### M-Mode and Two-dimensional Echocardiography

Two-dimensional echocardiography was performed by experienced ultrasonographers using a phased-array system (Hewlett-Packard series 77020A, Hewlett-Packard Medical Products, Andover, Mass.). Standard cross-sectional and long-axis views were obtained from the parasternal and apical windows. Strip-chart recordings of two-dimensionally guided M-mode examination were recorded at 50 mm/sec paper speed for dimensional analysis. M-mode measurements were made according to American Society of Echocardiography standards. Left ventricular mass was calculated from M-mode measurements using the formula described by Troy et al. and corrected according to the method of Devereux et al. Results were indexed to body surface area. The septal-to-posterior wall thickness ratio was calculated, and a value ≥1.3 was used to define asymmetric septal hypertrophy. Diastolic relative wall thickness was calculated as the ratio of twice the posterior wall thickness to left ventricular end-diastolic dimension. Both M-mode and two-dimensional echocardiograms were also reviewed for evidence of systolic anterior motion of the mitral valve or chordae tendineae.

To investigate preoperative left ventricular structure in patients with postoperative evidence of abnormal intracavitary flow acceleration, preoperative M-mode and two-dimensional echocardiograms were reviewed in the 28 patients who had complete preoperative studies. Ten of these patients were from group 1, and 18 were from group 2.
Doppler Analysis

Continuous wave Doppler interrogation of the left ventricular cavity was performed with a 1.9-MHz non-imaging transducer. Left ventricular outflow tract velocities were recorded under two-dimensional echocardiographic guidance from the apical four-chamber view. Abnormal intracavitary flow acceleration was defined as the presence of a dagger-shaped intracavitary flow signal (Figure 1) on continuous wave Doppler. This signal was obtained by positioning the transducer at the apex of the heart and angling the ultrasound beam to obtain the mitral inflow pattern. The Doppler beam was then angled toward the aortic valve until the dynamic flow signal was identified; the peak velocity associated with this signal was recorded. A meticulous attempt was made in each case to distinguish the abnormal intracavitary flow acceleration signal from the jets of mitral regurgitation, the flow velocity across the aortic valve, and the pulsed wave flow velocity recorded immediately under the aortic valve. In general, the velocity of the abnormal intracavitary flow signal was maximal when the mitral inflow pattern was simultaneously recorded; in contrast, the maximum velocity associated with valvular aortic stenosis was frequently obtained when the continuous wave transducer was positioned at the right sternal edge (Figure 2). The pressure gradient associated with the peak intracavitary flow velocity was calculated from the Bernoulli equation.

In eight patients with abnormal intracavitary flow acceleration documented by continuous wave Doppler, pulsed Doppler interrogation of the outflow tract was performed using a 2.5- or 3.5-MHz transducer. The sample volume was moved from apex to base to pinpoint the location of flow acceleration (Figures 3 and 4). In addition, these same eight patients also had color flow Doppler mapping of their left ventricular cavity using the 2.5- or 3.5-MHz transducers with depth and sector width adjusted to maximize frame rate. Turbulent flow indicative of abnormal intracavitary flow acceleration was depicted as a mosaic pattern in the left ventricle on systolic frames (Figure 4).

Cardiac Catheterization

Left and right heart catheterization was performed using standard fluid-filled catheters. In all except one case, dynamic subvalvular obstruction was unsuspected at the time of preoperative catheterization, and studies were not designed to pursue this diagnosis. Pressure measurements were recorded on a photographic recorder (Honeywell Electronics for Medicine VR-12). Cardiac output was determined using the Fick principle for oxygen or by thermodilution techniques. Aortic valve area was determined using the Gorlin equation. Ejection fractions were determined from right anterior oblique ventriculograms using the area–length method. Selective coronary arteriograms were obtained in multiple projections using standard femoral artery techniques. Clinically important coronary artery disease was defined as a >50% stenosis in any major coronary artery.

Aortic Valve Surgery

All patients underwent aortic valve replacement using standard surgical technique. Thirty-eight patients (11 group 1 and 27 group 2) received mechanical prostheses (St. Jude’s Medical), and 15 patients (two in group 1 and 13 in group 2) received bioprostheses.

Pathological Evaluation

Histological evaluation was performed in three patients: two who had undergone septal myectomy, and one whose heart was examined at necropsy. Myectomy specimen and appropriate samples of the left ventricular free wall and septum were fixed in 10% buffered formalin, dehydrated, and embedded in paraffin. Five-micrometer sections were stained with hematoxylin and eosin and examined by light microscopy.

Statistical Analysis

All results are expressed as mean±SD values. Comparison of continuous variables was performed using
FIGURE 3. Pulsed wave Doppler profile obtained from the postoperative examination of patient in the series. The two-dimensional echocardiogram is obtained in the apical two-chamber view with the sample volume located slightly below the level of the papillary muscles. The spectral recording demonstrates the late-peaking systolic flow envelope characteristic of patients in the series.

Results

Thirteen of the 53 patients exhibited abnormal intracavitary flow acceleration on their postoperative continuous wave Doppler study and composed group 1. The clinical characteristics of these patients were compared with those of the remaining 40 patients (group 2) who had undergone aortic valve replacement and postoperative echocardiographic study during the same time period but whose Doppler examinations did not show evidence of abnormal intracavitary flow acceleration.

Clinical Characteristics

There was a higher percentage of women in group 1 than in group 2 (69% versus 42%), but this difference was not significant. There was no significant difference between the two groups with regard to age, prevalence of hypertension, or any of the clinical manifestations of aortic stenosis (Table 1).

Cardiac Catheterization

All patients underwent cardiac catheterization; results are presented in Table 2. Group 1 patients had significantly higher ejection fractions (76±7% versus 56±16%, p<0.0001) and higher mean transvalvular gradients (72±28 versus 54±18 mm Hg, p<0.02). No patient had an intracavitary gradient detected on preoperative catheterization. However, one patient in whom dynamic subvalvular obstruction was suspected before surgery (but not detected at catheterization) underwent repeat left heart catheterization after surgery because of persistent hypotension and Doppler evidence of abnormal intracavitary flow acceleration. A dynamic subvalvular gradient was detected at this time.

The groups did not differ with respect to cardiac index, left ventricular end-diastolic pressure, or calculated aortic valve area. There was a trend toward a higher prevalence of coronary artery disease (eight of 13 [62%] of group 1 versus 19 of 39 [46%] of group 2 patients), but this difference was not statistically significant.

Paired Preoperative Doppler Echocardiography

Preoperative echocardiograms of 10 of 13 group 1 patients and 18 of 40 group 2 patients were available for analysis (Table 3). Comparison of preoperative findings...
shows that group 1 patients had a greater average septal thickness (19.3±2.7 versus 14.2±2.6 mm for group 2, p<0.0002), posterior wall thickness (15.6±4.3 versus 13.2±2.9 mm, p=NS), and relative wall thickness (0.81±0.4 versus 0.54±0.2, p<0.05). Septal-to-posterior wall thickness ratios were similar in the two groups.

Seven patients (six in group 1 and one in group 2) showed asymmetric septal hypertrophy on their preoperative echocardiogram.

TABLE 1. Clinical Features of Study Population

<table>
<thead>
<tr>
<th>Feature</th>
<th>Group 1 (n=13)</th>
<th>Group 2 (n=40)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>70±15</td>
<td>70±10</td>
<td>NS</td>
</tr>
<tr>
<td>Women (%)</td>
<td>69</td>
<td>42</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>62</td>
<td>58</td>
<td>NS</td>
</tr>
<tr>
<td>Dyspnea (%)</td>
<td>69</td>
<td>63</td>
<td>NS</td>
</tr>
<tr>
<td>Congestive heart failure (%)</td>
<td>23</td>
<td>38</td>
<td>NS</td>
</tr>
<tr>
<td>Syncope (%)</td>
<td>38</td>
<td>45</td>
<td>NS</td>
</tr>
<tr>
<td>Angina (%)</td>
<td>46</td>
<td>48</td>
<td>NS</td>
</tr>
</tbody>
</table>

Postoperative echocardiographic results in patients with preoperative studies are also shown in Table 3. In both groups, left ventricular end-diastolic dimension fell from preoperative values, but the difference was significant only in group 1. Fractional shortening increased after surgery in both groups. No significant new wall motion abnormalities were detected after surgery by two-dimensional echocardiography in any study patient.

TABLE 2. Catheterization Data in Study Population

<table>
<thead>
<tr>
<th>Feature</th>
<th>Group 1 (n=13)</th>
<th>Group 2 (n=40)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction (%)</td>
<td>76±7</td>
<td>56±16</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Transvalvular mean gradient (mm Hg)</td>
<td>72±28</td>
<td>54±18</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Cardiac index (l/min/m²)</td>
<td>2.5±0.7</td>
<td>2.3±0.5</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>18±7</td>
<td>19±7</td>
<td>NS</td>
</tr>
<tr>
<td>AVA (cm²)</td>
<td>0.6±0.26</td>
<td>0.6±0.25</td>
<td>NS</td>
</tr>
<tr>
<td>CAD, n (%)</td>
<td>8 (62)</td>
<td>19 (46)</td>
<td>NS</td>
</tr>
</tbody>
</table>

LVEDP, left ventricular end-diastolic pressure; AVA, aortic valve area; CAD, presence of coronary artery disease; values are mean±SD.
Hematocrit

Preoperative hematocrit did not differ significantly between the two groups (39.8±4.7% for group 1 versus 38.3±3.1% for group 2, p=NS). After surgery, there was a significant decline in hematocrit in both groups. However, there was no significant difference between group 1 and group 2 patients with regard to postoperative hematocrit (31.4±4.7% for group 1 versus 31.7±3.7% for group 2, p=NS).

Preoperative and Postoperative Doppler

Doppler evidence of abnormal intracavitary flow acceleration was present in eight of the 10 group 1 patients who had preoperative studies. Pulsed and color flow Doppler mapping of flow acceleration supplemented continuous wave studies in eight patients. The dagger-shaped flow signal was identified in the midventricular cavity in all instances (and at the apex in two instances) with a progressive increase in velocity as the sample volume approached the left ventricular outflow tract (Figures 3 and 4).

Table 4 lists preoperative and postoperative Doppler findings in relation to preoperative echocardiographic results and clinical data of the 13 group 1 patients. The peak intracavitary flow velocities in the preoperative studies ranged from 1.8 to 6.8 m/sec with associated gradients ranging from 13 to 185 mm Hg. In postoperative studies, the peak velocities ranged from 1.5 to 7 m/sec with associated gradients ranging from 9 to 196 mm Hg. Velocities increased from preoperative levels in four patients; two of these four patients died, and the other two patients were discharged after lengthy hospitalizations. The two patients with the highest preoperative velocities (5.0 and 6.8 m/sec, respectively) also had marked septal hypertrophy and underwent septal myectomy to facilitate placement of aortic valve prostheses; their postoperative courses were complicated, and one of these two patients died.

Table 3. Paired Data of Patients With Both Preoperative and Postoperative Studies

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=10)</th>
<th></th>
<th>Group 2 (n=18)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before surgery</td>
<td>After surgery</td>
<td>Before surgery</td>
<td>After surgery</td>
</tr>
<tr>
<td>Septal thickness (mm)</td>
<td>19.3±2.7</td>
<td>18.2±3.4</td>
<td>14.2±2.6</td>
<td>12.2±7.0</td>
</tr>
<tr>
<td>Posterior wall thickness (mm)</td>
<td>15.6±4.3</td>
<td>15.5±4.3</td>
<td>13.2±2.9</td>
<td>11.2±6.5</td>
</tr>
<tr>
<td>Septal-to-posterior wall thickness ratio</td>
<td>1.2±3.1</td>
<td>1.2±0.24</td>
<td>0.54±0.2</td>
<td></td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.81±0.4</td>
<td></td>
<td>0.52±0.20</td>
<td></td>
</tr>
<tr>
<td>LV end-systolic dimension (mm)</td>
<td>16.4±8.8</td>
<td>19.7±11.7</td>
<td>34.2±9.9†</td>
<td>25.7±16.2</td>
</tr>
<tr>
<td>LV end-diastolic dimension (mm)</td>
<td>44.1±7.1</td>
<td>39±9.7*</td>
<td>50.1±8.3†</td>
<td>37.4±7.9</td>
</tr>
<tr>
<td>Fractional shortening</td>
<td>0.46±0.17</td>
<td>0.52±0.20</td>
<td>0.32±0.13</td>
<td>0.35±0.17§</td>
</tr>
<tr>
<td>LV mass index (g/m²)</td>
<td>175±37</td>
<td></td>
<td>165±54</td>
<td></td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>39.8±4.7</td>
<td>31.4±4.7†</td>
<td>38.3±3.1</td>
<td>31.7±3.7§</td>
</tr>
<tr>
<td>Intracavitary flow acceleration</td>
<td>8/10</td>
<td>10/10</td>
<td>0/18</td>
<td>0/18</td>
</tr>
</tbody>
</table>

LV, left ventricular; values are mean±SD.

*p<0.05 compared with group 1 preoperative value. †p<0.01 compared with group 1 preoperative value. §p<0.0001 compared with group 1 preoperative value. §p<0.002 compared with group 2 preoperative value.

Table 4. Echocardiographic and Clinical Data on Group 1 Patients

<table>
<thead>
<tr>
<th>Patient (age/sex)</th>
<th>M-mode echocardiography</th>
<th>CW Doppler</th>
<th>Clinical data</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ST</td>
<td>PWT</td>
<td>LVEDD</td>
<td>LVESD</td>
</tr>
<tr>
<td>79/F</td>
<td>16</td>
<td>43</td>
<td>28</td>
<td>0.60</td>
</tr>
<tr>
<td>76/M</td>
<td>20</td>
<td>52</td>
<td>14</td>
<td>0.46</td>
</tr>
<tr>
<td>71/F</td>
<td>25</td>
<td>30</td>
<td>14</td>
<td>1.67</td>
</tr>
<tr>
<td>80/F</td>
<td>20</td>
<td>24</td>
<td>24</td>
<td>1.8</td>
</tr>
<tr>
<td>72/F</td>
<td>20</td>
<td>35</td>
<td>28</td>
<td>0.86</td>
</tr>
<tr>
<td>79/F</td>
<td>19</td>
<td>42</td>
<td></td>
<td>0.86</td>
</tr>
<tr>
<td>60/M</td>
<td>20</td>
<td>50</td>
<td>50</td>
<td>0.56</td>
</tr>
<tr>
<td>57/F</td>
<td>20</td>
<td>24</td>
<td>24</td>
<td>0.55</td>
</tr>
<tr>
<td>75/F</td>
<td>15</td>
<td>35</td>
<td>16</td>
<td>1.10</td>
</tr>
<tr>
<td>77/M</td>
<td>12</td>
<td>24</td>
<td>24</td>
<td>0.55</td>
</tr>
<tr>
<td>80/M</td>
<td>15</td>
<td>35</td>
<td>16</td>
<td>1.10</td>
</tr>
<tr>
<td>71/F</td>
<td>15</td>
<td>20</td>
<td>20</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Preoperative echocardiography: Measurements were performed in 10 of 13 group 1 patients; dimensions are in millimeters. ST, septal thickness; PWT, posterior wall thickness; LVEDD, LVESD, left ventricular end-diastolic and end-systolic dimensions; RWT, relative wall thickness; ND, not detected; CO, cavity obliteration.

Continuous wave (CW) Doppler: V_{pre} and V_{post}, peak preoperative and postoperative velocities; Dob, dobutamine; Dop, dopamine; Epi, epinephrine; Dig, digoxin; Proc, procanamide; Met, metoprolol; Cap, captopril; D/C, discharged; HD, hospital day.
Of the patients receiving mechanical prostheses, the average valve size for group 1 patients was significantly smaller than that of group 2 (20.1±2.0 versus 21.7±2.2, p=0.05). There was no significant Doppler transvalvular gradient detected after surgery in any study patient.

Seven of 13 patients were receiving digoxin and three additional patients were receiving intravenous inotropic support for hypotension at the time of their postoperative study.

**Hospital Course**

In-hospital postoperative mortality was significantly greater in group 1 (38% versus 12% for group 2, p<0.05). In addition, six of 13 patients suffered important hemodynamic compromise after surgery. Five patients had severe prolonged hypotension characterized by systolic blood pressure <95 mm Hg despite inotropic support and pulmonary capillary wedge and/or left atrial pressures >20 mm Hg. One additional patient with moderate hypotension (systolic blood pressure, <110 mm Hg) had poor cardiac output (cardiac index of 1.7 l/min/m²). Seven patients had protracted hospital courses after surgery (Table 4).

The five group 1 patients who died had complicated courses, and all suffered prolonged hypotension. Two patients had recurrent congestive heart failure and atrial fibrillation; one of these patients developed acute renal failure, presumably related to hypotension. A third patient suffered cardiopulmonary arrest after suffering several days of severe hypotension and atrial fibrillation. She was found to have bronchopneumonia at postmortem examination. The fourth patient developed severe bradycardia (in addition to refractory hypotension) necessitating insertion of a dual-chamber pacemaker and had a 94-day postoperative course; the immediate cause of death was probably bronchopneumonia. The fifth patient suffered a hemispheric cerebrovascular accident and died from related complications.

The hospital course of patient 1 illustrates many of the clinical features encountered in group 1. A 79-year-old woman underwent cardiac catheterization for aortic stenosis. The mean transaortic valve gradient was 66 mm Hg, the aortic valve area was 0.5 cm², and the ejection fraction was 78%. No intracavitary gradient was demonstrable. The patient underwent aortic valve replacement. Inspection at the time of surgery demonstrated a hypertrophied interventricular septum, a small chamber, and a narrowed left ventricular outflow tract. A septal myectomy and an aortic root anuloplasty were performed to allow placement of a no. 21 Carpentier-Edwards valve. The immediate postoperative course was complicated by severe hypotension (central arterial pressure, 90/50 mm Hg) and reduced cardiac index (1.8 l/min/m²) despite a left atrial pressure of 22 mm Hg and treatment with dobutamine, epinephrine, and dopamine. Echocardiography demonstrated hyperdynamic left ventricular systolic function, normal prosthetic valve function, and no evidence of systolic anterior motion of the mitral apparatus. Continuous wave Doppler showed evidence of left ventricular flow acceleration with a peak velocity of 7 m/sec; the corresponding pressure gradient, calculated from the Bernoulli equation, was 196 mm Hg. In view of the excellent systolic function and concern that subvalvular obstruction was exacerbated by enhanced contractility, inotropic therapy was discontinued. A subsequent Doppler study demonstrated abnormal intracavitary flow velocity of 5.2 m/sec and an estimated gradient of 108 mm Hg. However, the patient’s hypotension and reduced cardiac output continued; a third echocardiogram demonstrated peak left ventricular outflow tract gradient of 100 mm Hg. In view of persistent hypotension, cardiac catheterization was performed and demonstrated a left ventricular outflow tract gradient of 80 mm Hg; an esmolol infusion was begun and was associated with a reduction in the outflow tract gradient to 20 mm Hg. Repeat continuous wave Doppler study demonstrated a similar drop in the calculated left ventricular outflow tract gradient.

**Left Ventricular Morphology**

Histological examination of the interventricular septum in three patients revealed hypertrophic myocytes
and interstitial fibrosis (Figure 6). The myocardial fiber disarray characteristic of primary hypertrophic cardiomyopathy\(^2\) was not found in any of the samples.

**Discussion**

We have identified a group of patients undergoing aortic valve replacement for aortic stenosis who had Doppler evidence of left ventricular abnormal intracavitary late systolic flow acceleration. These patients have a distinctive ventricular geometry (small cavities and marked hypertrophy) and hyperdynamic left ventricular systolic function. In contrast to previous echocardiographic studies of patients with the syndrome of subvalvular obstruction after aortic valve replacement,\(^1\)\(^5\)\(^8\)\(^9\)\(^20\) none of our patients exhibited systolic anterior motion of the mitral valve. Patients with these Doppler echocardiographic features suffered markedly increased morbidity and mortality after surgery and often required negative inotropic agents and fluid resuscitation for optimal management.

**Incidence**

Evidence of Doppler flow acceleration was commonly found in our series; 13 of 53 patients (25%) undergoing postoperative study had this finding. This incidence is higher than the clinical occurrence of subvalvular dynamic obstruction complicating critical aortic stenosis, usually estimated to occur in 10% of cases.\(^2\)\(^6\)\(^8\)\(^9\)\(^20\)\(^21\) This discrepancy is most likely due to the fact that several of our patients underwent postoperative study in the absence of significant clinical deterioration; if the five patients who had uncomplicated postoperative courses are excluded, the incidence of abnormal intracavitary flow acceleration in our patients would be reduced to approximately 15%.

**Mechanism of Abnormal Systolic Flow Acceleration**

In classic hypertrophic obstructive cardiomyopathy, echocardiography demonstrates asymmetric septal hypertrophy, left ventricular outflow tract narrowing, and systolic anterior motion of the mitral valve apparatus.\(^22\) Left ventricular structure in patients with coexistent subvalvular obstruction complicating aortic valve disease cited in previous reports, however, appears to vary. Three of the six patients described by Nanda et al\(^20\) were reported to have asymmetric septal hypertrophy and systolic anterior motion of the mitral valve in addition to aortic valve disease. Surprisingly, however, two of those three patients had pure aortic incompetence. In the individual aortic stenosis cases reported by Schringer et al\(^8\) and Cutrone et al,\(^9\) the left ventricle was described as having severe concentric hypertrophy and systolic anterior motion of the mitral valve. Thus, the subvalvular flow acceleration in these latter instances may be related to the systolic anterior motion of the mitral valve, as has been described in hypertrophic cardiomyopathy.\(^11\)\(^18\)\(^23\)

The mechanism of production of systolic flow acceleration in our patient population cannot be ascribed to systolic anterior motion of the mitral valve. It is our
impression that the flow acceleration and associated gradient we observed in the majority of our patients are epiphenomena of the hyperdynamic state and extremely small cavity\textsuperscript{10,24} and may reflect abnormal ejection dynamics. In this regard, the ventricular structure in our study population bears resemblance to that described for the group of elderly patients with “hypertensive hypertrophic cardiomyopathy”\textsuperscript{25} whose Doppler flow abnormalities were described by Pearson et al.\textsuperscript{26} In these patients with marked left ventricular hypertrophy due to longstanding hypertension, peak continuous wave outflow tract velocities ranged from 1 to 5 m/sec, and the flow velocity contour was similar to that described in both hypertrophic obstructive cardiomyopathy and our patient population. Systolic anterior motion of the mitral valve was uncommon in this series.\textsuperscript{26} Pearson et al speculated that the mechanism of flow acceleration is the near apposition or close approximation of the left ventricular walls during systole\textsuperscript{26}; our data support this speculation. Similar findings have recently been reported by Harrison et al.,\textsuperscript{27} who observed Doppler evidence of midventricular obstruction in a selected group of 10 patients with marked concentric left ventricular hypertrophy (patients with systolic anteriormotion of the mitral valve were excluded from this series) complicating longstanding hypertension. The continuous wave velocity associated with this abnormal intracavitary flow acceleration ranged from 1.9 to 4.5 m/sec.\textsuperscript{27} Our findings of midventricular flow abnormalities in all patients with adequate pulsed Doppler flow mapping further directs attention away from the left ventricular outflow tract and mitral apparatus and toward globally abnormal ejection dynamics.

A second possible mechanism for abnormal intracavitary flow acceleration has been suggested by the study of Bird and coworkers.\textsuperscript{28} These investigators, using a catheter with two laterally mounted micromanometers, demonstrated large subvalvular gradients in a series of 11 patients with aortic stenosis who did not have anatomic evidence of subvalvular obstruction.\textsuperscript{28} The authors postulated that the gradient between the left ventricular cavity and outflow tract helped overcome “blood’s inertia to convective and local acceleration in the tapering subvalvular flow field.”\textsuperscript{28} The authors further hypothesized that flow acceleration following the afterload reduction afforded by aortic valve replacement would lead to increased postoperative ejection velocities,\textsuperscript{28} which was likely enhanced by treatment with positive inotropic agents.

Unfortunately, our data do not permit a precise delineation of the mechanism of flow acceleration in our study population. It is unlikely that the postoperative fall in hematocrit contributed substantially to the abnormal intracavitary flow acceleration since a significant decline was observed in both group 1 and group 2 patients. Nor were there significant differences in frequency of regional wall motion abnormalities between the two groups; in fact, no patients developed a significant wall motion abnormality on their postoperative study. Finally, because on average, group 1 patients received slightly smaller valve prostheses, there is no evidence that this group of patients developed abnormal intracavitary flow acceleration in response to an enhanced drop in afterload following valve replacement. However, it does appear reasonable to speculate that the drop in left ventricular afterload following valve replacement\textsuperscript{29} and concomitant treatment with positive inotropic agents enhanced intracavitary flow velocity and exacerbated abnormal ejection dynamics.

A question raised by the frequency of asymmetric septal hypertrophy in our series is whether the left ventricular hypertrophy and abnormal intracavitary flow acceleration were the result of coexistent aortic stenosis and hypertrophic cardiomyopathy.\textsuperscript{16} Disproportionate septal thickening, while a hallmark of hypertrophic cardiomyopathy, is also seen in secondary forms of pressure-overload hypertrophy. This finding is observed in at least 5% of unselected patients with hypertension\textsuperscript{30,31} and is commonly encountered in older cohorts. Lewis and Maron\textsuperscript{30} recently showed that one third of patients in their series of elderly hypertensives with markedly increased wall thickness were found to have asymmetric hypertrophy, defined as ratio of wall thickness of any two segments of the left ventricle exceeding 1.5:1. Other series have reported an incidence of asymmetric septal hypertrophy of approximately 10% in patients with unspecified aortic valve disease.\textsuperscript{31,32} Hess et al\textsuperscript{2} have shown that asymmetric septal hypertrophy is found in approximately 9% of patients who are found to have critical aortic stenosis at catheterization. This incidence is slightly less than that observed in our series, probably because these investigators used the more restrictive septal-to-posterior wall ratio of >1.5:1 to define asymmetric septal hypertrophy.\textsuperscript{2}

Although it is possible that some of our patients with asymmetric septal hypertrophy (approximately 13% of the total study population) had both aortic stenosis and hypertrophic cardiomyopathy, the coexistence of these two disorders in a consecutive series of patients is unlikely on a statistical basis.\textsuperscript{5} In addition, the pathological data, while limited to only three of the patients in our series, did not demonstrate myocardial fiber disarray characteristic of hypertrophic cardiomyopathy. In the most extensive pathological investigation of this issue reported to date, Hess et al\textsuperscript{2} reviewed histological specimens from 10 patients with critical aortic stenosis and asymmetric septal hypertrophy who underwent septal myectomy at the time of aortic valve replacement; no patient had evidence of myocardial fiber disarray. Panza and Maron\textsuperscript{2} have asserted that the absence of systolic anterior motion of the mitral valve and marked septal hypertrophy (>25 mm) militates against coexistent hypertrophic cardiomyopathy. Septal hypertrophy of this magnitude was present in only one of our patients, and no patient had systolic anterior motion of the mitral valve.

**Clinical Implications of Doppler Echocardiographic Findings**

There appear to be important prognostic and therapeutic implications of the Doppler echocardiographic findings of marked left ventricular hypertrophy and abnormal intracavitary flow acceleration. A striking and somewhat surprising finding of our study was the increased postoperative morbidity and mortality of patients with these findings, arguing that aortic stenosis patients with marked concentric left ventricular hypertrophy and Doppler evidence of abnormal intracavitary flow acceleration may be at high risk for hemodynamic
compromise after aortic valve replacement. Although the immediate cause of death was not necessarily cardiovascular in all instances, prolonged hypotension may have led to either renal failure or prolonged intubation, which appeared to contribute to postoperative mortality. In most instances, hypotension was manifested early in the postoperative period. Hemodynamic compromise in the early postoperative period was most successfully treated by withdrawal of inotropic support, fluid resuscitation, and institution of \( \beta \) - or calcium channel blockers. Furthermore, in several instances, the improvement in clinical status paralleled reduction in the continuous wave intracavitary flow velocity and associated gradient. Thus, Doppler echocardiography identified postoperative patients with hemodynamic compromise who benefited from negative inotropic therapy and fluid resuscitation.

Furthermore, it may be possible to stratify risk in this population according to Doppler echocardiographic findings. Although the study population is small, our data suggest that the magnitude of the intracavitary flow velocity and relative wall thickness on the preoperative study may provide important prognostic information. The two group 1 patients who did not have evidence of abnormal intracavitary flow acceleration before surgery (and who had low values of relative wall thickness) had uncomplicated postoperative courses, and three of the four patients with intracavitary flow velocities of \( > 4 \) m/sec died. When high-velocity intracavitary flow signals (\( > 4 \) m/sec) are present on the postoperative study, the prognosis is equally poor: Two patients died, and two others had complicated, protracted hospitalizations. It is tempting to speculate that abnormal intracavitary flow acceleration is a marker for marked or excessive left ventricular hypertrophy in aortic stenosis; this geometric pattern has long been known to be associated with intraoperative "ischemic contracture" during or after aortic valve replacement.\(^3\) More recently, a similar influence of left ventricular geometry on prognosis in patients with milder degrees of hypertrophy due to hypertension has been reported by Koren et al.\(^4\) Unfortunately, our data do not permit a complete understanding of the pathophysiological link between abnormal ventricular structure, ejection dynamics, and postoperative morbidity and mortality.

Our findings also underscore the usefulness of echocardiography in patients about to undergo aortic valve replacement for aortic stenosis. In our experience, no other clinical variables served to reliably identify patients with normal systolic function who may be at risk for postoperative hemodynamic compromise. It has been suggested that a history of prolonged episodes of dizziness, intermittent spontaneous improvements in symptoms, or exacerbation of symptoms by digitalis administration should alert the clinician to the presence of subvalvular obstruction in the presence of critical aortic stenosis.\(^5\) However, none of our patients had the historical features described by these investigators. Furthermore, neither the ECG nor the chest film furnished clinical clues to the diagnosis as has been previously described.\(^6\) It is also evident that routine left heart catheterization may not necessarily identify patients with this syndrome, particularly if performed with a side-hole (pigtail) catheter.

**Study Limitations**

We acknowledge several limitations to the present study. Because most of the Doppler echocardiographic studies were performed either before surgery to quantitate the severity of valvular aortic stenosis or after surgery to evaluate clinical deterioration, they were not specifically designed to detect abnormal intracavitary flow. Therefore, it is possible that the prevalence of abnormal intracavitary flow acceleration may be underestimated. It is also possible that the postoperative study may not have been obtained at the time of the peak intracavitary flow acceleration and maximal associated gradient. However, the index patient in this series was studied in July 1988. Because that patient provided such dramatic illustration of the features of this syndrome, our laboratory has conducted meticulous examinations to detect intracavitary flow acceleration in aortic stenosis patients.

The distinction among the high-velocity jets associated with aortic stenosis, mitral regurgitation, and abnormal intracavitary flow acceleration may appear to pose a methodological problem. However, in all patients in this series, the jets were distinguishable by careful positioning of the continuous wave probe as described above.\(^13\) Mitral regurgitation was, furthermore, uncommon in group 1 patients. In most cases, the maximal jet associated with the stenotic aortic valve was recorded with the continuous wave Doppler probe positioned at the right sternal edge. In contrast, the jet associated with abnormal intracavitary flow acceleration was maximal when recorded from the apical window; we are, therefore, confident that abnormal intracavitary flow acceleration is a distinct phenomenon. This distinction was confirmed in patient 1, whose postoperative intracavitary gradient at catheterization agreed closely with Doppler results.

**Summary**

We have described the clinical and Doppler echocardiographic features of 13 patients with abnormal intracavitary flow acceleration. These patients had distinct left ventricular geometry characterized by marked hypertrophy and small cavities. After surgery, these patients were more likely to suffer severe hypotension and had markedly increased in-hospital mortality. In general, early postoperative hypotension in these patients responded best to withdrawal of inotropic support, volume resuscitation, and, in some cases, institution of calcium channel and/or \( \beta \)-blockers.

Preoperative echocardiography is superior to clinical evaluation (including routine catheterization) at identifying patients at risk for the development of this syndrome after surgery. Both before and after surgery, Doppler velocity associated with abnormal intracavitary flow acceleration further stratifies patients at higher risk for cardiovascular morbidity and mortality and can be used to guide therapy. Neither the mechanism of abnormal intracavitary flow acceleration nor the precise reason for postoperative morbidity and mortality can be completely determined from our data. However, postoperative compromise does not appear to be caused by outflow tract obstruction caused by systolic anterior motion of the mitral apparatus.
The preoperative echocardiographic findings of marked concentric left ventricular hypertrophy accompanied by abnormal intracavitary flow acceleration should alert the cardiologist, cardiothoracic surgeon, and anesthesiologist that the optimal treatment of hypertension in the early postoperative period may require volume and withdrawal of inotropic support and is best guided by Doppler echocardiography.

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