Hemodynamic Determinants of Prognosis of Aortic Valve Replacement in Critical Aortic Stenosis and Advanced Congestive Heart Failure

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SUMMARY Fourteen patients with critical aortic stenosis (valve area ≤ 0.4 cm²/m²), a history of advanced congestive heart failure, left ventricular ejection fraction less than 0.45 (mean 0.28 ± 0.03) and no other valvular lesions or obstructive coronary artery disease were studied to assess prognosis with aortic valve replacement. Eleven of 14 (79%) survived surgery; 10 of these 11 showed major clinical improvement postoperatively and formed group 1. The three patients who died and the patient who did not improve formed group 2. Although group 2 had higher preoperative values for aortic valve area and left ventricular end-diastolic volume and lower ejection fraction and cardiac output than group 1, none of these factors alone reliably predicted outcome. The mean systolic gradient was an important predictor of outcome: No patient with a mean systolic gradient ≤ 30 mm Hg had a good outcome, irrespective of valve area or other hemodynamic variables.

Ejection fraction was plotted against left ventricular wall stress for both groups. For group 1, there was a close linear relation that could be extrapolated back to normal wall stress and normal ejection fraction. This suggested afterload mismatch as a major cause for this group’s depressed ejection fraction. In group 2 ejection fraction was lower for any given wall stress, suggesting depressed contractility, rather than afterload mismatch, as the cause of the left ventricular dysfunction. Thus, either afterload mismatch or depressed contractility may result in depressed ejection fraction in patients with aortic stenosis; which one predominates may have major prognostic importance.

THE NATURAL HISTORY of critical aortic stenosis is well defined.1,2 Patients with this disease who have symptoms of congestive heart failure and remain untreated by aortic valve replacement have an average life expectancy of approximately 2 years. Although recent studies3,4 have shown that the majority of patients with critical stenosis and congestive heart failure respond well to corrective surgery, some patients with this condition are not helped by aortic valve replacement. Patients with aortic stenosis and congestive heart failure who responded well and those who responded poorly to aortic valve replacement may represent two distinct groups, rather than opposite ends of a spectrum. To test this hypothesis and to identify the defining characteristics of these two groups, we examined our experience at the Peter Bent Brigham Hospital in patients with pure aortic stenosis and severe congestive failure who underwent cardiac catheterization and aortic valve replacement over a 5-year period.

Materials and Methods

All catheterization reports from January 1, 1974 to January 1, 1979 were examined. Eighty-six patients with aortic stenosis (aortic valve areas less than 0.4 cm²/m²) who had no evidence of other hemodynamically significant valvular disease at catheterization were identified. Of this group, 28 patients (33%) had an angiographically determined left ventricular ejection fraction less than 0.45 and a clinical presentation of severe congestive heart failure (New York Heart Association [NYHA] functional class III or IV). All 28 patients underwent coronary arteriography during catheterization. Ten patients had normal coronary arteriograms and four had minor, non-obstructive coronary narrowing (less than 40% decrease in luminal diameter). These 14 patients without significant coronary disease or other valvular lesions, who had critical aortic stenosis, congestive heart failure and depressed left ventricular ejection fraction, constitute the study population.

Each patient underwent right- and retrograde left-heart catheterization, including left ventriculography and coronary arteriography. Thirteen patients were in sinus rhythm at catheterization, and one patient was in atrial fibrillation. The left ventricle was entered retrogradely via the brachial approach in all patients. Systemic arterial pressure was measured by means of a PE 160 catheter placed percutaneously in the right femoral artery. Left ventricular pressure was measured by standard fluid-filled angiographic catheters in nine patients and by micromanometer-tipped, high-fidelity catheters in five. Left ventricular and systemic arterial pressure were recorded simultaneously and mean systolic gradients were measured by planimetry. All gradients were confirmed by recording pressures during pullback of the catheter from the left ventricle to the central aortic position. Although the catheter may contribute to the effective stenosis in patients with aortic valve areas less than 0.6 cm², the degree of this contribution could not be
assessed, and therefore valve area was calculated using
the measured left ventricular and aortic pressures in
the manner described by Gorlin. Cardiac output was
measured by the Fick method, simultaneously with
the gradient recording. Left ventriculography was
done in single-plane right anterior oblique projection
and recorded on 35-mm cine film at 60 frames/second.
Left ventricular volumes were determined by the
area-length method in 13 patients using a regression
equation derived in our laboratory from the study of
postmortem left ventricular casts. In the remaining
patient the absence of a grid for calculating the
angiographic magnification correction factor allowed
only for determination of ejection fraction. Left ven-
tricular mass was calculated by methods previously
described. The ratio of left ventricular wall
thickness (h) to the minor-axis radius (r) was also
calculated. Left ventricular circumferential midwall
stress (σ) was calculated by methods described
previously, using the formula of Mirsky:

\[ σ = \frac{Pb}{h} \left( 1 - \frac{h}{2b} - \frac{b^2}{2a^2} \right) \]

where \( P \) = left ventricular pressure, \( h \) = wall
thickness, \( a = \) midwall semimajor axis \((L/2 + h/2)\)
and \( b = \) midwall semiminor axis \((D/2 + h/2)\). Left
ventricular ejection fraction was calculated as
(EDV - ESV)/EDV, where EDV and ESV are left
ventricular end-diastolic and end-systolic volumes,
respectively.

Significant aortic regurgitation was ruled out by
aortography in two patients. In 11 other patients in
sinus rhythm, the regurgitant flow was calculated as:

\[ \text{FCO} = \left( \frac{\text{EDV} - \text{ESV}}{\text{AHR}} \right) - \text{FCO} \]

where EDV = angiographic end-diastolic volume,
ESV = angiographic end-systolic volume, AHR =
heart rate during angiography and FCO = cardiac
output determined by the Fick method. Regurgitant
flow was less than 3 l/min in all patients, suggesting
the absence of significant aortic or mitral
regurgitation. In the last patient the presence of
atrial fibrillation precluded calculation of regurgitant
flow. In this patient the absence of a diastolic murmur,
widened pulse pressure and fluttering of the mitral
valve leaflet on the echocardiogram served to rule out
significant aortic regurgitation.

All patients underwent aortic valve replacement
within 1 month of catheterization. Eleven patients
received a porcine heterograft valve and three received
a Björk-Shiley prosthesis. Follow-up information was
obtained from office records. Four patients underwent
radionuclide ventriculography postoperatively to
assess ejection fraction directly.

Statistical inference was made using the exact
probability method for attributes and the \( t \) test for
variables.

Results

The clinical data for all patients are shown in table
1. Thirteen patients were male, one was female. Eight
patients were in NYHA class III and six were in class
IV preoperatively. Of the eleven survivors, eight
became class I, two became class II and one remained
in class IV postoperatively (fig. 1). Patient follow-up
averaged 28 ± 5 months.

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Symptoms</th>
<th>Preoperative NYHA class</th>
<th>Postoperative NYHA class</th>
<th>Follow-up (months)</th>
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<tbody>
<tr>
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<td>M</td>
<td>Angina, DOE</td>
<td>III</td>
<td>I</td>
<td>57</td>
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<td>2</td>
<td>70</td>
<td>M</td>
<td>Syncpe, DOE</td>
<td>IV</td>
<td>I</td>
<td>45</td>
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<td>3</td>
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<td>M</td>
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<td>III</td>
<td>I</td>
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<td>II</td>
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<td>III</td>
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<td>9</td>
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<td>Angina, syncope, DOE, PND</td>
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<td>I</td>
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<td>8</td>
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</table>

Abbreviations: DOE = dyspnea on exertion; PND = paroxysmal nocturnal dyspnea; OD = operative
death; PD = perioperative death; NYHA = New York Heart Association.
NYHA
CLASSIFICATION  PRE-OP  POST-OP
I  0  8
II  0  2
III  8  0
IV  3  1

FIGURE 1. Preoperative and postoperative New York Heart Association (NYHA) classification of operative survivors who underwent aortic valve replacement for aortic stenosis and left ventricular failure.

Three of 14 patients (21%) died in the operative or perioperative period. This exceeds the overall mortality rate of 3.4% (five of 148 patients) for isolated aortic valve replacement in patients with aortic stenosis at our institution during the same period. Two of the patients could not be weaned from cardiopulmonary bypass. The third patient died of a low cardiac output state 9 days after operation. There were no late deaths.

Ten patients who had a satisfactory result (postoperative class I or II) were grouped together and form group 1. The three patients who died at operation and the one patient who remained in class IV postoperatively form group 2. The two groups were compared to identify preoperative factors associated with satisfactory or unsatisfactory outcome.

Preoperatively, all patients were receiving digoxin and furosemide. All patients complained of at least two-pillow orthopnea and dyspnea with minimal exertion or at rest. There was no statistical difference in age between groups. Angina and syncope were more frequent in group 1, while paroxysmal nocturnal dyspnea and edema were more frequent in group 2 (table 1); neither of these differences was statistically significant.

Two of 10 group 1 patients (20%) and all four (100%) group 2 patients were in functional class IV preoperatively (p < 0.05).

Hemodynamic and angiographic data are presented in tables 2 and 3. The mean pulmonary capillary wedge pressure was similar in both groups. The average aortic valve area index for group 2 was 0.30 ± 0.02 cm²/m², which was significantly larger than for group 1 (0.21 ± 0.02 cm²/m², p < 0.05). Cardiac index was significantly lower for group 2 (1.5 ± 0.3 l/min/m²) than for group 1 (2.1 ± 0.1 l/min/m², p < 0.05). The average end-diastolic volume index for group 2 was 174 ± 9 ml/m², compared with 119 ± 9 ml/m² for group 1 (p < 0.005). The end-systolic volume index for group 2 was 140 ± 9 ml/m², compared with 80 ± 7 ml/m² for group 1 (p < 0.005). The average ejection fraction for

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**TABLE 2. Hemodynamic Data from Patients with Severe Aortic Stenosis and Left Ventricular Failure**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Left ventricular end-diastolic pressure (mm Hg)</th>
<th>Aortic valve area index (cm²/m²)</th>
<th>Peak systolic gradient (mm Hg)</th>
<th>Mean systolic gradient (mm Hg)</th>
<th>Cardiac index (l/min/m²)</th>
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</tr>
<tr>
<td>1</td>
<td>32</td>
<td>0.16</td>
<td>110</td>
<td>90</td>
<td>2.4</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
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<td>60</td>
<td>38</td>
<td>1.7</td>
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<td>40</td>
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<td>74</td>
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<td>40</td>
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<td>138</td>
<td>93</td>
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<tr>
<td>Mean ± SEM</td>
<td>39 ± 2</td>
<td>0.21 ± 0.02</td>
<td>80 ± 9</td>
<td>61 ± 6</td>
<td>2.1 ± 0.1</td>
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<td>35</td>
<td>0.26</td>
<td>32</td>
<td>25</td>
<td>1.3</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>38 ± 1</td>
<td>0.30 ± 0.20*</td>
<td>28 ± 5*</td>
<td>22 ± 1*</td>
<td>1.5 ± 0.3*</td>
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</table>

*p < 0.05.
TABLE 3. Angiographic Data from Patients with Severe Aortic Stenosis and Left Ventricular Failure

<table>
<thead>
<tr>
<th>Pt</th>
<th>LVEDVI (ml/m²)</th>
<th>LVESVI (ml/m²)</th>
<th>LVEF</th>
<th>LVMI (g/m²)</th>
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<td>—</td>
<td>0.17</td>
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<td>5</td>
<td>157</td>
<td>118</td>
<td>0.25</td>
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<tr>
<td>Mean</td>
<td>119</td>
<td>80</td>
<td>0.32</td>
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<tr>
<td>± SEM</td>
<td>± 9</td>
<td>± 7</td>
<td>± 0.03</td>
<td>± 16</td>
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<tr>
<td>Group 2</td>
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<tr>
<td>Mean</td>
<td>174</td>
<td>140</td>
<td>0.20</td>
<td>251</td>
</tr>
<tr>
<td>± SEM</td>
<td>± 9</td>
<td>± 9</td>
<td>± 0.02</td>
<td>± 33</td>
</tr>
</tbody>
</table>

*p < 0.05.
†p < 0.005.

Abbreviations: LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; LVEF = left ventricular ejection fraction; LVMI = left ventricular mass index.

group 2 was 0.20 ± 0.02, which was less than that for group 1 (0.32 ± 0.03 p < 0.05) (fig. 2). The peak systolic aortic valve gradient for group 2 was 28 ± 5 mm Hg, compared with 80 ± 9 mm Hg for group 1 (p < 0.005). A similar striking difference existed for mean systolic gradient, which averaged 22 ± 1 mm Hg for group 2 vs 61 ± 6 mm Hg for group 1 (p < 0.005) (fig. 3).

Four group 1 patients had postoperative determination of left ventricular ejection fraction by radionuclide ventriculography 6–13 months after surgery. Because ejection fraction determined in this manner correlates well with angiographically determined ejection fraction,16,17 we compared preoperative angiographic ejection fraction with postoperative radionuclide ejection fraction in these four group 1 patients. The preoperative ejection fraction was 0.33 ± 0.03, compared with 0.59 ± 0.03 postoperatively (p < 0.001).

Mean circumferential wall stress was plotted against ejection fraction for the five group 1 patients not previously studied by this method, as well as for the four previously analyzed11 (fig. 4). A linear relationship (r = 0.93) similar to that reported by Gunther and Grossman15 was found. Values for preoperative wall stress in group 2 all fell below and to the left of the line developed from coordinates for group 1 (fig. 5). Thus, for any given wall stress, ejection fraction was lower in group 2.

Left ventricular wall thickness/radius ratio (h/r) was higher in group 1 patients (0.42 ± 0.03) than in group 2 patients (0.35 ± 0.03), although the difference was not statistically significant (p = 0.08).

FIGURE 2. Individual preoperative ejection fractions (EF) for patients in groups 1 (•) and 2 (X). Although mean EF was greater for group 1 than for group 2, three group 1 patients had EF similar to that of group 2. Thus, EF alone was not a good predictor of outcome.

FIGURE 3. Mean systolic aortic pressure gradients for patients in group 1 (•) and group 2 (X). Group 2 patients had lower systolic gradients in every case.
Discussion

The main finding of this study is that patients with critical aortic stenosis and advanced congestive heart failure apparently fall into two distinct subgroups. One subgroup (group 1) had left ventricular failure apparently on the basis of excessively high wall stress and afterload mismatch. This group uniformly had a good surgical result. A second subgroup (group 2) had more severe left ventricular failure despite less severe aortic stenosis (larger valve area) and a lesser afterload, suggesting depressed myocardial contractility as a major contributing factor producing left ventricular failure. These patients had a poor prognosis.

Cohn and associates stressed the importance of preoperative left ventricular function as a prognostic guide to both valvular and coronary artery bypass surgery. Forty percent of their patients with an ejection fraction less than 0.50 died at surgery. However, recent studies by Croake et al., Smith et al., and Thompson et al. suggest that this relationship may not hold true for patients with severe aortic stenosis and depressed left ventricular function. These studies, however, did not identify preoperative factors that predicted outcome, possibly because many patients in these studies had coronary artery disease. Because coronary artery disease is believed to be a strong negative prognostic factor, it may have obscured other prognostic factors. Our series of patients, although not large, only included patients with severe aortic stenosis and no obstructive coronary disease.

That patients with aortic stenosis and depressed left ventricular function should do well after surgery is not altogether surprising. Many studies have shown postoperative improvement in left ventricular function after aortic valve replacement in patients with aortic stenosis and depressed preoperative ejection fraction. Gunther and Grossman recently presented evidence supporting the concept that some patients with aortic stenosis and depressed left ventricular performance may have this depression on the basis of excessive wall stress (afterload mismatch) rather than on the basis of depressed contractile state. Relief of this excessive afterload by aortic valve replacement allows for immediate improvement of ventricular performance; hence, even patients with severely depressed preoperative left ventricular function may have an excellent surgical result. Further support for this concept comes from the elegant studies of Strauer, who reported that in patients with left ventricular pressure-overload hypertrophy from hypertension, as well as aortic stenosis and regurgitation, left ventricular ejection fraction and peak systolic wall stress correlate inversely.

Our study confirms that many patients with
depressed left ventricular function and aortic stenosis have a good prognosis with aortic valve replacement. Ten of 14 such patients (72%) returned to class I or II postoperatively. When we analyzed preoperative factors we thought might have had prognostic importance, we found a significant difference in preoperative left ventricular function between the patients who had a satisfactory surgical result (group 1) and those who did not (group 2). Group 2 patients had more advanced clinical class, lower cardiac index, higher end-diastolic volume index and lower ejection fraction than group 1 patients. However, none of these factors alone was highly predictive of outcome. Twenty percent of group 1 patients were in class IV and returned to class I postoperatively. Twenty percent had a cardiac index less than 1.6 l/min/m², 20% had an end-diastolic volume index greater than 150 ml/m² and 30% had an ejection fraction less than 0.25. However, when ejection fraction was plotted against mean wall stress (σ) for both groups, major differences were found. The linear relationship between ejection fraction and wall stress could be extrapolated to a normal ejection fraction and normal wall stress for group 1 patients, suggesting that the major factor in the depressed left ventricular performance in group 1 patients preoperatively was excessive wall stress. In contrast, group 2 patients had a lower ejection fraction at a given wall stress than group 1 patients. Group 2 also had a larger aortic valve area index than group 1. Thus, group 2 had poorer left ventricular performance despite less wall stress and less obstruction to outflow. This suggests that group 2, unlike group 1, had depressed myocardial contractility as a significant component of their left ventricular failure, rather than excessive wall stress.

The etiology of this depressed contractility is uncertain. One possibility is that group 2 patients with depressed contractility represent the end stage of a natural progression of the disease. However, this seems unlikely because there was actually less severe aortic stenosis in group 2 patients. A second explanation is that a concomitant cardiomyopathy of unknown etiology was also present in group 2. As our study was not designed to evaluate this question, data for or against this supposition are lacking. Third, some ventricles simply may not tolerate excessive wall stress as well as others. High wall stress, if produced acutely, may cause myocardial injury in experimental animals. However, the variability in response to chronically elevated wall stress is not known.

The relatively low wall stress in group 2 patients can readily be translated into the low transvalvular gradients and lower left ventricular systolic pressures (less than 150 mm Hg in every case). Thus, a peak systolic gradient less than 30 mm Hg across the aortic valve when associated with congestive failure may identify patients with a significant component of intrinsic myocardial contractile depression. These patients have greater depression of ventricular performance even though afterload is relatively less, and may not be fully identified on the basis of preoperative symptoms, functional class, cardiac output, end-diastolic volume or ejection fraction, because patients with aortic stenosis may have a severe abnormality in any one of these variables and still have a good prognosis. A low transvalvular gradient indicative of a low cardiac output and larger orifice area suggest severe left ventricular dysfunction but less obstruction to outflow. When this is coupled with decreased ejection fraction, despite normal or only slightly increased LV wall stress, the implication is that significant cardiomyopathy is present and the prognosis is poor. Aortic valve replacement in this group carries an extraordinarily high risk.

We found encouraging results for aortic valve replacement in patients with severe aortic stenosis and depressed preoperative left ventricular function. The majority of patients in this series had left ventricular failure because of excessive afterload imposed by the stenotic valve. This group of patients has a good prognosis, because aortic valve replacement decreases afterload and allows for improved ventricular performance. The explanation for the afterload excess or "afterload mismatch" in these patients is uncertain, because many patients encountered in our laboratory during the same years had identical degrees of obstruction (as judged by valve area) but normal left ventricular wall stress and ejection fraction. Because concentric hypertrophy usually develops in aortic stenosis in such a fashion as to maintain normal wall stress,22 we have speculated that inadequate hypertrophy of the left ventricular myocardium may explain the high wall stress and depressed fiber shortening in this subset of patients.11 The lower h/r ratio in group 2 is consistent with this concept, but the number of subjects is too small to permit any firm conclusion to be drawn.

References
9. Kennedy J, Trenholme SE, Kasser IS: Left ventricular volume and mass from single plane cineangiocardiograms: a
comparison of anteroposterior and right anterior oblique methods. Am Heart J 80: 343, 1970


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