Severe Aortic Stenosis with Impaired Left Ventricular Function and Clinical Heart Failure: Results of Valve Replacement

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SUMMARY Nineteen patients, aged 58-80 years, with severe isolated aortic valve stenosis, severely reduced ejection fraction and clinical heart failure underwent aortic valve replacement between January 1970 and April 1977. Ten had concomitant coronary artery disease (all underwent additional coronary bypass surgery), 17 had angina pectoris and four had syncope. Aortic valve area index was 0.32 ± 0.03 cm²/m² (mean ± SEM); left ventricular (LV) end-diastolic volume index was 117 ± 9 ml/m² and LV ejection fraction was 0.37 ± 0.02.

There were four operative deaths and one late death. The follow-up time ranged from six to 74 months (38 ± 6 months). Actuarially determined three-year survival is 74 ± 10%; the expected five-year survival is the same. One patient had a serious cerebrovascular accident. Of the remaining survivors, seven were initially Functional Class IV and six Class III; currently, six are Class I and seven Class II (New York Heart Association classifications). The cardiothoracic ratio has decreased from 0.54 ± 0.03 to 0.49 ± 0.03.

Repeat hemodynamic evaluation has been performed in 10 patients, 22 ± 6 months after surgery. In these 10 patients, the aortic valve gradient decreased from 55 ± 7 to 11 ± 1.3 mm Hg; LV end-diastolic pressure from 22 ± 2.4 to 9 ± 1.9 mm Hg; LV end-diastolic volume index from 119 ± 16 ml/m² to 107 ± 11 ml/m². LV ejection fraction has increased dramatically from 0.34 ± 0.03 to 0.63 ± 0.05 and mean velocity of circumferential fiber shortening from 0.57 ± 0.08 to 1.3 ± 0.18 cir/sec.

The encouraging long-term survival, improved functional class and the marked improvement in left ventricular function that occurred in our patients indicate that all patients with severe aortic stenosis in clinical heart failure should be offered aortic valve replacement.

Patients with severe aortic stenosis have a grave prognosis, with a mortality rate of 8-9% per year.1,2 With the onset of severe symptoms the average life expectancy is three years; that for angina is five years, and for syncope it is three years. The worst prognostic indicator is congestive heart failure, for which the average life expectancy is less than two years.3 Aortic valve replacement for severe aortic stenosis can be performed at a reasonable operative mortality.4,5 This procedure improves hemodynamics,6 and the late survival rate is good.4,6 In the absence of complications, ventricular hypertrophy regresses and ventricular performance improves.7,8,9

The results of valve replacement in patients with aortic stenosis in congestive heart failure have not been defined, and its effects on left ventricular function are not known;8,10,11 therefore, there is uncertainty about the role of surgery in such patients.8,12 We have evaluated the effects of aortic valve replacement on the long-term survival, functional class and left ventricular function in patients with severe aortic stenosis with impaired left ventricular function who were in clinical heart failure.

Material and Methods

All patients with isolated severe aortic stenosis (aortic valve area index ≤ 0.75 cm²/m²),13,14 with or without trivial aortic insufficiency, catheterized at the University of Oregon Health Sciences Center between January 1970 and April 1977, have been reviewed. Patients with other congenital lesions, multivalvular heart disease, moderate or severe aortic insufficiency or who were less than 18 years were excluded. Nineteen patients who had ejection fraction ≤ 0.47 and who had clinical congestive heart failure with varying degrees of effort dyspnea, paroxysmal nocturnal dyspnea and occasional edema were studied for this report.

All patients were felt to be in clinical heart failure. We performed initial hemodynamic investigation after first attempting to control the symptoms of congestive heart failure with digitalis and diuretics and to stabilize the patients' chest pain when necessary. Cardiac catheterization and angiography were performed using standard techniques, and calculations were made. Methods used were exactly the same as those which have been extensively described previously.9,15-21 In brief, right and left heart catheterizations were performed in all patients. The left ventricle was entered using the transseptal technique in 18 patients and by the retrograde femoral technique in one patient. Fick and duplicate indocyanine green dilution curves were used to obtain the cardiac output and calculate valve areas. Left ventricular (LV) function was evaluated from single plane right antero-oblique
cineventriculograms. Left ventricular volumes were measured using the area length method. Ejection fraction and mean velocity of circumferential fiber shortening (mean Vcf) were calculated. Aortic insufficiency was assessed from supravalvular aortography, and extent of coronary artery disease from selective coronary arteriography. All patients underwent coronary angiography and obstructive lesions that narrowed the luminal diameter ≥ 50% were considered significant.

All patients with severe aortic stenosis had valve replacement with Starr-Edwards ball valve prostheses; no patient with severe aortic stenosis was denied surgery after cardiac catheterization. No patient was judged too ill for cardiac catheterization and sent directly to surgery without catheterization. The size and the series of prosthetic valve that was inserted depended on the annular size, the individual surgeon’s preference, the year and the clinical situation. Ten patients had simultaneous aortocoronary saphenous vein bypass surgery. The surgical techniques have been described previously.4,22

Follow-up information was obtained from the clinic charts. If the patient had not been seen in clinic within the previous six months, follow-up information was obtained from telephone conversations between the patients and one of the investigators.

Four patients underwent repeat cardiac catheterization for clinical reasons: one for recurrence of angina, two for suspected perivalvular incompetence and one for clinical-hemodynamic deterioration. One patient who suffered a cerebrovascular accident six months after valve replacement while off anticoagulation and had a dense residual aphasia, was not offered restudy. The remaining patients who had not been restudied were again contacted between May and October 1977; subsequently, they were seen in the clinic and were requested to undergo repeat catheterization. Six patients consented to elective restudy and three refused. The techniques of restudy have been described and were similar to the initial study.20 Informed consent was obtained from all patients. There were no complications from the initial or the repeat studies in any of the patients. Coronary arteriography was repeated only if it was clinically indicated.

Results

Initial Findings

Clinical Findings

Of the 19 patients, 10 were male and nine were female. Their ages ranged from 52–80 years (mean 65 years). Eleven patients were in Functional Class IV (New York Heart Association Classification) from six weeks to 20 years. All 19 had dyspnea on exertion, 13 had paroxysmal nocturnal dyspnea, 12 had orthopnea, seven had pulmonary edema and nine had peripheral edema (table 1). The hemodynamic findings and extent of left ventricular dysfunction assessed at cardiac catheterization were not significantly different in patients who had had peripheral edema compared with those who had not had peripheral edema. Sixteen patients were treated with digitalis and diuretics, and three with digitalis alone.

Angina was present in 17 (89%), and syncope in four (21%). Left ventricular hypertrophy with secondary ST and T wave changes was present in 15 (79%). Three patients had left bundle branch block (LBBB), and one had right bundle branch block. Two (11%) had electrocardiograms that were compatible with previous myocardial infarction (MI). No patient had a normal ECG. Chest x-rays of the five patients who died were not available for review; 10 of the remaining 14 patients had cardiothoracic ratio > 0.5 and the cardiothoracic ratio for the group was 0.54 ± 0.03 (mean ± SEM).

Hemodynamics and LV Function (table 1)

The mean aortic valve gradient ranged from 30–96 mm Hg and averaged 59 ± 5 mm Hg (mean ± SEM). It was < 40 mm Hg in four, from 40–60 mm Hg in 10 and > 60 mm Hg in five patients. The cardiac index was 2.3 ± 0.11 l/min/m²; it was less than 2.01/min/m² in four, between 2.0 and 2.4 in eight, and was ≥ 2.5 in seven patients. The calculated aortic valve area index was 0.56 cm²/m² in one patient and was ≤ 0.4 cm²/m² in the others; it averaged 0.32 ± 0.03 cm²/m². The calculated aortic valve area was 1.15 cm² in one patient and was < 0.79 cm² in the others; it averaged 0.56 ± 0.05 cm². The LV end-diastolic pressure was 20 ± 2 mm Hg. The LV end-diastolic volume index (ml/m²) was 117 ± 9; it was increased in nine (> 110 ml/m²) and was in the upper range of “normal” (> 98 ml/m²) in another five (mean ± 1 sd in “normals”: 86 ± 12 ml/m²).18

The LV ejection fraction was 0.37 ± 0.02 (mean ± 1 sd in “normals”: 0.64 ± 0.05); it was 0.18 in one, 0.19 in one, 0.30–0.39 in nine, and 0.40–0.47 in eight patients. Regional areas of wall motion abnormalities (akinesis)77 were present in only three patients (see below).

Associated Coronary Artery Disease

Ten patients (54%) had significant coronary artery disease; nine of these 10 had angina. Two of the 19 patients had no angina; coronary arteriography revealed normal coronary arteries in one aged 66 years and one-vessel coronary artery disease in the other, aged 58 years. One had akinesis of the inferior wall, disease of the right coronary and left anterior descending arteries, and ECG changes of inferior myocardial infarction, but gave no history of MI. One had akinesis of the anterior wall, disease of the left anterior descending artery and did not have ECG changes of MI, but gave a history of MI. The third patient with akinesis had involvement of the anterior wall, had disease of the right coronary and left anterior descending

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<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (years)</th>
<th>Selected preoperative symptoms</th>
<th>State</th>
<th>Mean AVG (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>LVEDVI (mL/m²)</th>
<th>Ejection fraction</th>
<th>Mean Vaf (circ/sec)</th>
<th>Assoc. OBS, CAD</th>
<th>Duration of follow-up (months)</th>
<th>Complications</th>
<th>Duration, surgery to restudy (months)</th>
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Abbreviations: DOE = dyspnea on exertion; PND = paroxysmal nocturnal dyspnea; ORTH = orthopnea; PULM ED = pulmonary edema; PERIED = peripheral edema; Preop = preoperative; Postop = postoperative; Mean AVG = mean aortic valve gradient; LVEDP = left ventricular end-diastolic pressure; LVEDVI = left ventricular end-diastolic volume index; Mean Vaf = mean velocity of circumferential fiber shortening; OBS CAD = obstructive coronary artery disease; LAD = left anterior descending coronary artery; RCA = right coronary artery; LCX = left circumflex coronary artery; OM = obtuse marginal branch of the left coronary artery; CVA = cerebrovascular accident; LMCAD = left main coronary artery disease; AI = aortic insufficiency; MI = myocardial infarction; CHB = complete heart block; + = present; 0 = absent; --- = not available.
TABLE 2. Clinical Features of Patients Studied

<table>
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<tr>
<th></th>
<th>Operative deaths, preoperative data</th>
<th>Not restudied, preoperative data</th>
<th>Restudied</th>
<th>Postoperative data</th>
<th>P Value</th>
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<tr>
<td>Number of patients</td>
<td>4</td>
<td>5†</td>
<td>10</td>
<td>10</td>
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<td>Mean aortic valve gradient (mm Hg)</td>
<td>66 ± 15</td>
<td>51 ± 8</td>
<td>55 ± 7</td>
<td>11 ± 1.3</td>
<td>&lt;0.001</td>
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<td>Aortic valve area index (cm²/m²)</td>
<td>0.26 ± 0.07</td>
<td>0.32 ± 0.03</td>
<td>0.33 ± 0.05</td>
<td>0.87 ± 0.07</td>
<td>&lt;0.005</td>
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<td>Cardiac index (/min/m²)</td>
<td>2.1 ± 0.24</td>
<td>2.6 ± 0.31</td>
<td>2.4 ± 0.17</td>
<td>2.4 ± 0.1</td>
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<td>Left ventricular end-diastolic pressure (mm Hg)</td>
<td>23 ± 6</td>
<td>16 ± 5</td>
<td>22 ± 2.4</td>
<td>9 ± 1.9</td>
<td>&lt;0.001</td>
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<td>Left ventricular end-diastolic volume index (ml/m²)</td>
<td>104 ± 7</td>
<td>111 ± 16</td>
<td>119 ± 16</td>
<td>107 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>Left ventricular ejection fraction</td>
<td>0.38 ± 0.08</td>
<td>0.41 ± 0.02</td>
<td>0.34 ± 0.03</td>
<td>0.63 ± 0.05</td>
<td>&lt;0.001</td>
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<td>Mean circumferential fiber shortening (mean Vcf) (circ/sec)</td>
<td>-</td>
<td>-</td>
<td>0.57 ± 0.08</td>
<td>1.3 ± 0.18</td>
<td>&lt;0.01</td>
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</table>

*Postoperative: two are Functional Class I, one is Functional Class II, one has cerebrovascular accident and one died.
†Three refused restudy, one has a cerebrovascular accident and one died in the early followup period.
‡Values exclude patient with perioperative myocardial infarction and late heart block.

arteries, gave no history of MI and had LBBB on the ECG.

Four patients had three-vessel coronary artery disease, four had two-vessel disease and two had single-vessel disease. One patient also had left main coronary artery stenosis.

Surgery

Four patients died at surgery (21%), two from technical problems (delayed mediastinal bleed in one and dissection of the left main coronary artery in a patient with occluded right coronary artery and normal left coronary artery), and two from low cardiac output. Two patients had an autopsy; of the two patients with low cardiac output, one had evidence of a perioperative myocardial infarction at autopsy and the patient with mediastinal bleed had no discernible cardiac cause at autopsy for perioperative death. Three of the four patients who died had associated coronary artery disease; all had received aortocoronary bypass grafts. The fourth patient had an isolated totally occluded distal right coronary artery. She developed dissection of the left main coronary artery during surgery and had received coronary bypass grafts.

There was no significant difference in any of the clinical features, hemodynamics, and extent of left ventricular dysfunction between the four patients who died and the 15 that survived surgery (table 2).

Except for the patient mentioned above, there was one other instance of perioperative myocardial infarction (see below).

Follow-up

Follow-up has ranged from six to 74 months, and averages 38 ± 6 months (mean ± SEM). Duration of follow-up in each patient is shown in table 1.

Survival

There has been one late death. A patient died suddenly one month after surgery for unknown causes. Preoperative coronary arteriograms showed normal coronary arteries. The patient had frequent premature ventricular beats in the hospital.

Actuarial analysis shows the survival at three years

![Figure 1](http://circ.ahajournals.org/). Preoperatively, all patients were either Functional Class IV or III. and postoperatively all survivors were either Functional Class II or I.)
and the expected survival at five years are the same, 74 ± 10% (mean ± SEM).

Late Complications

One patient has a moderate periprosthetic aortic regurgitation. Another patient, who developed perioperative myocardial infarction, was not started on anticoagulants for two years, and developed complete heart block late and has a permanent demand ventricular pacemaker. One patient with normal coronary arteries preoperatively redeveloped severe angina. He was found to have iatrogenic left main coronary artery stenosis and subsequently has successfully received two aortocoronary bypass grafts.

Three patients, including the above patient who developed heart block, were initially not started on anticoagulants. One of them has had a cerebrovascular accident and has a severe disability from a dense aphasia. The other two were started on anticoagulants two and three years after surgery.

Clinical Status

The functional class and other indices, such as chest x-ray, of the patient with cerebrovascular accident cannot be evaluated. Of the remaining patients, seven are in Functional Class II and six are in Functional Class I (fig. 1). No patient has syncope, one has angina, and the remaining symptomatic patients complain of fatigue and/or dyspnea on exertion.

The cardiothoracic ratio increased in two patients; in the patient who had a perioperative myocardial infarction and developed heart block late it increased from 0.61 to 0.76, and in the patient with perivalvular aortic regurgitation it increased from 0.45 to 0.51. In one patient the initial value of 0.41 was unchanged and in all the others, the cardiothoracic ratio fell. The postoperative values for the group were 0.49 ± 0.03 and were significantly (P < 0.05) lower than the preoperative value (figs. 2 and 3). Excluding the patients who developed heart block and perivalvular incompetence postoperatively, the cardiothoracic ratio fell in the others from 0.54 ± 0.03 to 0.46 ± 0.02 (P < 0.001).

Hemodynamics and LV Function

Three patients have refused restudy. Their initial hemodynamics were not significantly different from those that died in hospital and from those who have been restudied (table 1).

Ten have been restudied, four for clinical reasons, and six were restudied electively. Of the four patients restudied for clinical reasons, one has moderate periprosthetic aortic regurgitation, one has complete heart block and one developed iatrogenic left main coronary artery stenosis requiring reoperation. The fourth patient had no detectable abnormalities.

Repeat studies, performed 22 ± 6 months after surgery, have generally revealed excellent hemodynamic results and improvement in LV function (tables 1 and 2; figs. 4 and 5). Two patients had no prosthetic valve gradients, two patients had gradients of 21 and 28 mm Hg, and in the others the gradient was less than 20 mm Hg. The cardiac index did not change significantly and the calculated aortic valve area index increased from $0.33 ± 0.05$ to $0.87 ± 0.07 \text{ cm}^2/\text{m}^2$ ($P < 0.005$), (table 2). The aortic valve area increased from $0.6 ± 0.1 \text{ cm}^2$ to $1.5 ± 0.1 \text{ cm}^2$.

The LV end-diastolic pressure fell in all patients and was normal in all but two patients. The left ventricular end-diastolic volume index (LVEDVI) fell from

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**Figure 2.** The preoperative chest x-ray film of one of the patients (left panel). There is considerable cardiomegaly and some pulmonary venous congestion, and blunting of the right costophrenic angle. Postoperatively, (right panel) there is no pulmonary venous congestion and cardiomegaly has decreased.
had postoperative values of < 98 ml/m². Excluding the patient with perioperative myocardial infarction and postoperative heart block, all six patients with preoperative LVEDVI ≥ 98 ml/m² had a fall of volume which decreased from 146 ± 18 to 99 ± 9 ml/m² (P < 0.025).

The LV ejection fraction has increased dramatically, from 0.34 ± 0.03 to 0.63 ± 0.05 (P < 0.001). The patient with perioperative myocardial infarction and late heart block had a reduction of ejection fraction and is the only patient in whom ejection fraction did not increase (fig. 5). The ejection fraction was normal (≥ 0.54) in seven of the 10 patients. Mean Vcf could be calculated both pre- and postoperatively in only six of the 10 patients (fig. 5). It increased in all patients from 0.57 ± 0.08 to 1.3 ± 0.18 circ/sec (P < 0.01); LV ejection time did not change significantly from the preoperative to the postoperative study. This finding is similar to that of other patients with aortic valve disease we have previously reported.³ Postoperatively, mean Vcf was normal (≥ 1.25 circ/sec) in four of the six patients.

Discussion

Aortic valve replacement in patients with severe valvular aortic stenosis in clinical heart failure and reduced ejection fraction resulted in a three-year survival of 74%; the estimated five-year survival is the same. The operative survivors were greatly improved in functional class; severe LV dysfunction was dramatically improved and returned to normal in two-thirds of the patients.

Heart failure is a clinical syndrome that is important to recognize; however, an adequate hemodynamic or biochemical definition is difficult.²² Heart failure produces well-known symptoms and signs.²² These result from an abnormal elevation of systemic venous and/or pulmonary venous pressures, inadequate blood flow to the various tissues of the body, excessive accumulation of sodium and water and from changes produced in the ventricles due to dilatation, myocardial hypertrophy and dysfunction.²² Seventeen of our 19 patients had varying degrees of dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, pulmonary edema or pedal edema. The other two had moderate dyspnea on exertion and severe angina at rest which was recently accelerated; one had an episode of pulmonary edema and the other had pulmonary venous congestion on chest x-ray. All patients were being treated for heart failure. Over 70% had cardiothoracic ratios > 0.5. All had LV ejection fraction of ≤ 0.47. Thus, our patients had moderate to severe impairment of LV systolic pump function in addition to clinical features of heart failure.

The operative mortality of our patients was 21%,

119 ± 16 to 107 ± 11 ml/m² (fig. 5). The patients with perioperative MI and late heart block and postoperative perivalvular regurgitation had an increase of LVEDVI (fig. 4). The change in LVEDVI was not significant probably because there were two subsets of patients — those with initially normal and those with initially increased LVEDVI. Excluding the patient with postoperative perivalvular regurgitation, two patients with preoperative LVEDVI < 98 ml/m² also

![Figure 3. The cardiothoracic ratio for all the patients who have survived surgery. The patient with the cerebrovascular accident is excluded. Postoperatively, one patient developed perioperative myocardial infarction (MI) and late complete heart block (CHB) and one developed peri-prosthetic aortic incompetence. In these two patients the cardiothoracic ratio increased, and in one patient the ratio of 0.41 was unchanged. In all the others, the cardiothoracic ratio fell. In the entire group, cardiothoracic ratio fell from 0.54 ± 0.03 to 0.49 ± 0.03 (P < 0.05). Excluding the patients who developed MI + CHB and aortic incompetence, the cardiothoracic ratio fell from 0.54 ± 0.03 to 0.46 ± 0.02 (P < 0.001).](http://circ.ahajournals.org/lookup/suppl/doi:10.1161/01.CIR.58.2.260/-/DC1/FIG3.png)
FIGURE 4. Left ventricular end-diastolic pressure fell in all patients (left panel). Preoperatively, it was normal in one patient and presumably was the result of excessive diuresis. The patients with perioperative myocardial infarction (MI) and late complete heart block (CHB) and with postoperative perivalvular aortic incompetence had an increase in left ventricular end-diastolic volume index (LVEDVI) (right panel). Excluding the patient with perioperative MI and late CHB, all patients who initially had LVEDVI > 98 ml/m² had a reduction in LVEDVI from 146 ± 18 to 99 ± 9 ml/m² (P < 0.025).
which is higher than that reported for all patients undergoing aortic valve replacement at our institution.4

Half of all the patients had associated coronary artery disease, which is known to increase the operative mortality.22, 26–29 Two operative deaths resulted from technical complications, and the other two from low output states. The patients who died were not significantly different from those who lived with respect to the severity of aortic stenosis and to the degree of LV dysfunction present (table 2). The patients who died had an average age of 68 years and a 100% incidence of coronary artery disease, while those who lived had an average age of 63 years and 40% prevalence of coronary disease; however, these differences are not statistically significant. Myocardial preservation is of critical importance in patients with impaired LV function who undergo cardiac surgery.11 With the use of newer methods of myocardial preservation, the operative mortality in this type of high-risk patients could be lowered in the future.

All our patients had severe aortic stenosis. Rapaport1 and Frank et al.,2 in previous studies on the natural history of isolated severe aortic stenosis, have demonstrated a three-year mortality of 52 and 36%, five-year mortality of 62 and 52%, and 10-year mortality of 80 and 90%. Frank and coworkers5 have also demonstrated that a combination of symptoms is a more ominous sign. Dyspnea was present in 19, angina in 17, and syncope in four of our 19 patients; thus, all our patients had a combination of symptoms. In patients with severe aortic stenosis, the presence of concomitant congestive heart failure reduces life expectancy to less than two years.8 Our patients were in clinical heart failure. In view of what is known about the natural history of severe aortic stenosis, particularly in those with a combination of symptoms and in those with heart failure, the late survival of our patients is most encouraging. Also, the functional result in the survivors is excellent (figs. 1–3).

In aortic stenosis the left ventricle compensates for the chronic pressure load by hypertrophy in order to normalize wall stress and overcome the elevated afterload.9 As a result, LV systolic pump function remains normal, even in the elderly with severe aortic stenosis.30–33 When the effects of chronic pressure overload overwhelm this compensatory mechanism, the heart may use the Frank-Starling mechanism in order to maintain LV systolic pump function. In such instances, parameters of LV systolic pump function such as ejection fraction and mean Vcf may be maintained initially in the normal range,44 but subsequently are reduced in all, and eventually congestive heart failure results. All our patients had reduced LV ejection fraction; in six the ejection fraction was severely reduced (< 0.35). Of our 19 patients, LV volumes were greater than one standard deviation away from the mean of normals in 14. Thus, in our patients compensatory mechanisms were no longer adequate.

Removal of the chronic pressure load resulted in improvement of LV systolic pump function in all patients who were restudied and were in sinus rhythm. The one patient who had a fall in LV ejection fraction sustained a perioperative myocardial infarction and late heart block. LV ejection fraction and mean Vcf returned to normal in about two-thirds of our patients, even though it was severely depressed in some (fig. 5). Although we did not observe it, it is possible that in some patients with severe aortic stenosis and heart failure, myocardial dysfunction is so far advanced that ventricular function may not improve after valve replacement.

The ejection fraction depends on acute changes in loading conditions of the heart (both preload and afterload). Mean Vcf also depends on acute changes in afterload; however, the effect of acute changes in preload on mean Vcf is controversial.21, 36–41 Since end-diastolic volume either fell or did not change in our patients, the observed improvement in ejection fraction and mean Vcf cannot be the result of a decrease in preload because in such instances ejection fraction should be expected to fall.21 Afterload should have been reduced in our patients (aortic valve area increased, LV systolic pressure fell and LV volumes either decreased or stayed the same); therefore, the observed improvement in LV systolic pump function must have been secondary to either a reduction of afterload, or to an improvement in myocardial function, or both. Although acute reduction in afterload has been shown to improve LV systolic pump function,42 chronic reduction in afterload, which would allow compensatory mechanisms to occur and would allow ventricular hypertrophy to regress,6 may not necessarily be associated with an improvement in LV systolic pump function as determined by ejection fraction and mean Vcf. For example, previously we have reported on 10 patients with aortic stenosis restudied 20 months after surgery; eight who had a normal ejection fraction preoperatively showed no significant change following surgery (0.71 vs 0.76).9 If this is also true for patients with impaired LV function, then the improvement in ejection fraction and mean Vcf which we have observed in the present study may indeed represent an improvement in myocardial function. In our patients, ejection fraction was calculated from single-plane right antero-oblique view angiograms. This may over- or underestimate LV function, especially in the presence of segmental disease. However, only three of our patients had localized areas of wall motion abnormalities on their ventriculograms. Single plane LV angiography is widely used. Its limitations are known and understood,43–47 and we believe that this is not a serious limitation of our study. We have restudied 10 of the surviving patients. The average age of those restudied was significantly (P < 0.025) lower than in those not restudied (average 59.5 years vs 70.4 years). However, the preoperative assessment of the severity of aortic stenosis, the extent of LV dysfunction and of the incidence of coronary artery disease in this group of 10 patients was not significantly different from those who were not restudied (table 2). Also, the postoperative improvement in functional class and in the reduction
of the cardiothoracic ratio was not different in the two subgroups. Therefore, it would appear that these 10 patients were probably representative of all the survivors.

In general, our patients had excellent results from valve replacement. The same results, however, should not necessarily be expected in patients in heart failure in association with mild (or, perhaps even moderate) aortic stenosis, because in these patients heart failure would not be expected to be related predominantly to the aortic stenosis. Therefore, the diagnosis of the severity of aortic stenosis is important. Since the clinical estimation of the severity of aortic stenosis is often in error, particularly in the presence of a low cardiac output, complete hemodynamic evaluation is warranted. Also, four of our patients had mean aortic valve gradients of < 40 mm Hg despite the presence of critical aortic stenosis, emphasizing the need for measuring both cardiac output and transvalvular gradient and calculating the aortic valve area. Left ventriculography allows proper evaluation of LV function, measurement of LV volumes, and of estimation of the degree of mitral regurgitation, and is usually essential. Supravalvular aortography allows the evaluation of the degree of aortic incompetence; however, it may not be needed in all patients. Coronary angiography is essential, since about half of the patients have significant obstructive coronary artery disease; angina is not a sufficiently accurate symptom of associated coronary disease, and in any case was present in almost all our patients. Such detailed investigations may be overwhelming for an occasional critically ill patient. The main risks of these studies are related to: 1) duration of the procedure; 2) the injected contrast medium which increases circulating blood volume that may further impair performance of a ventricle that has severe dysfunction; and 3) coronary arteriography which depresses LV performance. Therefore, we may study these patients on two days to lessen the risk of completely evaluating such critically ill patients. We have been able to perform complete studies in these and other critically ill patients with safety. In the patients reported in this study, there were no complications.

Clinical Implications

The encouraging long-term survival, the improved functional class and marked improvement in left ventricular function in our patients indicates that all patients with severe aortic stenosis in clinical heart failure should be offered aortic valve replacement, unless there is a specific contraindication to surgery. Severe aortic stenosis has been variously defined as aortic valve area (AVA) index of $\leq 0.75 \text{ cm}^2/\text{m}^2$, as AVA index of $\leq 0.60 \text{ cm}^2/\text{m}^2$ and as AVA of $\leq 1.0 \text{ cm}^2$. Our data should not be interpreted to indicate that surgical relief of severe aortic valve stenosis should be delayed until the onset of heart failure. It is preferable to operate on such patients before heart failure occurs.

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