Valve Replacement in Aortic Insufficiency
with Left Ventricular Dysfunction

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SUMMARY  Seventeen patients, ages 14–74 years, with severe, isolated aortic insufficiency and reduced ejection fraction (0.25–0.49) underwent aortic valve replacement between January 1973 and July 1977. Three had coronary artery disease and underwent coronary bypass surgery. By New York Heart Association criteria, one patient was functional class IV, seven were class III, seven were class II and two were class I. Left ventricular (LV) end-diastolic pressure was 19 ± 2 mm Hg (mean ± SEM). LV end-diastolic volume index (218 ± 18 ml/m²), end-systolic volume index (124 ± 11 ml/m²) and LV mass index (240 ± 10 g/m²) were increased in all patients. Mean velocity of circumferential fiber shortening (normal Vcf) (0.75 ± 0.05 circ/sec) was depressed in all patients. LV ejection fraction averaged 0.43 ± 0.02.

There were no operative deaths; one patient had a perioperative myocardial infarction and one developed complete atrioventricular block. Five late deaths occurred, including two patients who were not anticoagulated and died within the first year from severe prosthetic valve obstruction. Actuarially determined 3-year survival is 61 ± 15%. Of the late survivors, six are class I, five are class II and one is class III.

Ten patients who were restudied 14.5 ± 3 months after surgery had satisfactory prosthetic function. In these 10 patients, LV end-diastolic pressure decreased from 16 ± 3 to 10 ± 2 mm Hg, LV end-diastolic volume index decreased from 209 ± 15 to 155 ± 17 ml/m², LV end-systolic volume index decreased from 118 ± 10 to 82 ± 14 ml/m² and LV mass decreased from 234 ± 11 to 170 ± 16 g/m². LV volumes returned to normal in only two patients, however, and none had normalization of LV mass. Ejection fraction increased slightly in the restudied patients (0.43 ± 0.03 to 0.49 ± 0.04; 0.05 < p < 0.10); however, it increased more than 15% in five patients and in four of these it returned to normal. Mean Vcf increased from 0.72 ± 0.08 to 0.95 ± 0.11 circ/sec (p < 0.05), but became normal in only two patients.

Our data indicate that aortic valve replacement can be performed with acceptable risk in patients with severe aortic insufficiency and moderately severe impairment of LV function. Functional class was improved or maintained in late survivors, LV systolic pump function improved in 50% of patients, and there was consistent (but usually incomplete) regression of LV dilatation and hypertrophy even in patients who did not improve their ejection fraction.

IN SEVERE AORTIC INSUFFICIENCY (AI), the left ventricle compensates for volume overload by increasing stroke volume, end-diastolic volume, and myocardial mass; resting ejection fraction (EF) is usually normal, but may be reduced.1–3 Recent studies on the results of aortic valve replacement (AVR) in patients with AI have demonstrated improvement in functional class (New York Heart Association criteria) and consistent reductions in left ventricular (LV) volumes and mass, although frequently not to normal.4–6 The reported effects of valve replacement on systolic pump function have varied: one study reported significant improvement6 and others reported no change.4, 5, 7

Depressed preoperative EF has been associated with reduced survival and persistence of symptoms after coronary artery bypass8 and after valve replacement surgery.8, 9 However, we have recently shown a dramatic improvement in symptoms, functional class and LV function in aortic stenosis (AS) with clinical heart failure and impaired LV function.10 Therefore, it seemed important to evaluate the results of valve replacement in the subgroup of patients with severe aortic insufficiency and reduced EF.

We evaluated the results of AVR in 17 patients with isolated, severe AI and reduced EF with regard to survival, functional class, the compensatory mechanisms of LV dilatation and hypertrophy and systolic pump function.

Materials and Methods

All patients who underwent isolated AVR for severe AI at the University of Oregon Health Sciences Center between January 1973 and July 1977 were reviewed. Patients with other valvular heart disease, coexisting congenital lesions or a mean aortic valve gradient ≥ 20 mm Hg were excluded. Of 41 patients identified, 10 were excluded because of inadequate data for analysis and 14 had a preoperative EF ≥ 0.50; 17 patients, had a preoperative EF ≤ 0.49 and form the basis of this report.

All patients underwent initial right- and left-heart catheterization and angiography, performed in the fasting state with mild sedation (diazepam or secobarbital) using techniques described previously.5, 11, 12 LV pressures were recorded before angiography using the retrograde femoral technique. LV cineangiography in the right anterior oblique position and supravalvular cineaoortography were performed in each patient. LV volumes were calculated from the LV cineangiogram using the area-length method.13, 14 Premature and

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Supported in part by a grant from the Oregon Heart Association. Presented in part at the 51st Annual Scientific Sessions of the American Heart Association, Dallas, Texas, November 1978.
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Received April 3, 1979; revision accepted July 26, 1979.
postpremature beats were excluded, and the earliest satisfactory beat (during the fifth cardiac cycle) was analyzed.\textsuperscript{14, 15} In one patient with atrial fibrillation and a slow ventricular response (heart rate 68 beats/min), end-diastolic and end-systolic volumes were calculated as the average of four consecutive cardiac cycles. Preoperative and postoperative ventriculograms were analyzed separately to reduce observer bias. We have previously shown a very high degree of reproducibility of angiographic data when evaluated by the same observer on two occasions.\textsuperscript{16} All ventriculograms were reviewed independently by two of the investigators. LV mass was calculated.\textsuperscript{5, 13} Pericardial effusion, which would result in an overestimation of LV mass, was excluded in all preoperative patients by echocardiography and/or by the absence of effusion at operation (performed within a few days of study) and by echocardiography in all postoperative patients. The mean velocity of circumferential fiber shortening (mean Vcf) was measured as the change in internal circumference between end-diastole and end-systole at the calculated midpoint of the long axis, divided by the ejection time and normalized for end-diastolic circumference.\textsuperscript{5, 14, 17} Ejection time was determined from the simultaneously recorded arterial pressure tracing when available (10 patients)\textsuperscript{14} or from cine frames (seven patients), using the method of Karliner et al.\textsuperscript{17} In each case, pre- and postoperative ejection times were determined by the same method. AI was assessed qualitatively from the supravalvular aortogram using the classification of Hunt et al.\textsuperscript{18} Eleven patients showed complete ventricular opacification, denser than that of the aorta, in three beats or less (grade 5); six patients showed dense opacification of the ventricular cavity after more than three beats (grade 4).

Selective coronary angiography was performed using the Judkins technique in all patients over 35 years of age and in those with a specific clinical indication. Narrowing of the luminal diameter $\geq 50\%$ was considered significant.

Two of our patients denied having symptoms, and seven others were only mildly symptomatic (functional class II). The indications for initial catheterization in these nine patients included a cardiothoracic ratio $\geq 0.55$\textsuperscript{19, 20} (five patients), rapid increase in heart size over the preceding 6 months (two patients), acute AI with increasing symptoms (one patient) and progressive angina pectoris (one patient).

All patients had AVR with Starr-Edwards ball-valve prostheses. No patient went directly to surgery without catheterization. The size and model of prosthetic valve inserted depended on annular size, the surgeon’s preference, the year and the clinical situation. Three patients had simultaneous aortocoronary saphenous vein bypass procedures, one patient required repair of an ascending aortic aneurysm and one required insertion of a tube graft in the ascending aorta with reimplantation of both main coronary arteries. Surgery was performed with moderate systemic hypothermia plus topical hypothermia in each case and intermittent (nine patients) or continuous (eight patients) coronary perfusion. The surgical techniques have been described elsewhere.\textsuperscript{21, 22}

Preoperative functional class (New York Heart Association criteria)\textsuperscript{24} was assessed by review of hospital and clinic charts. Follow-up information was obtained through May 1978. If a patient had not been contacted within the preceding 6 months, information was obtained by telephone conversation between the patient and one of the investigators. One patient was lost to follow-up after 22 months.

Cardiothoracic ratio was determined at the time of initial and follow-up catheterizations; for patients not restudied, the most recent available chest x-ray was used for follow-up.

Repeat cardiac catheterization was performed in 12 patients. Indications for restudy were transient neurologic events or syncope in five patients, clinical and hemodynamic deterioration in four, chest pain in one, and new diastolic murmur in one. One patient consented to elective repeat catheterization. Five patients were not restudied: one had died, three had moved and one asymptomatic patient refused a repeat study. Postoperative catheterization was performed as previously described\textsuperscript{25} and was similar to the initial study except that the left ventricle was entered using the transseptal technique in all patients. Coronary arteriography was repeated only if clinically indicated. Informed consent was obtained from each patient, and no patient suffered a complication at initial or repeat catheterization. In postoperative patients, prosthetic aortic valve areas were calculated by the formula of Gorlin and Gorlin.\textsuperscript{26}

Comparison of pre- and postoperative data was made using the paired $t$ test; cited $p$ values are for a two-tailed test. Survival was evaluated using actuarial techniques.\textsuperscript{26}

\section*{Results}

\subsection*{Initial Clinical Findings}

Etiologies of AI were aortic root disease in seven patients (two with Marfan’s syndrome, one with luetic aortitis and four with unspecified aortic root dilatation), infective endocarditis in three patients (two with documented previous endocarditis 3 months and 9 years before study and one with subacute endocarditis on a previously normal aortic valve, studied at the completion of a 6-week course of antibiotics), a history of rheumatic fever in three, myxomatous degeneration of the aortic valve in two and congenital bicuspid valve in two.

Two patients were in functional class I, seven were in class II, seven were in class III, and one was in class IV. The majority of patients had multiple symptoms.

In addition to the typical blowing, decrescendo aortic diastolic murmur, physical examination revealed peripheral signs of AI in all patients. Pulse pressure was greater than 55 mm Hg in all, with an average of 91 $\pm$ 5 mm Hg (mean $\pm$ SEM); the ratio of pulse pressure to peak systolic arterial pressure was greater
than 0.50 in all patients. Diastolic blood pressure was less than 60 mm Hg in 15 of 17 patients.

The ECGs were abnormal in all 17 patients. Fifteen patients had LV hypertrophy with secondary ST- and T-wave changes and two had right bundle branch block. One patient was in atrial fibrillation. One patient had evidence of an old inferior myocardial infarction (MI).

Chest x-rays were not available for review in the five patients who died. In the remaining 12 patients, cardiothoracic ratio ranged from 0.50–0.70 (mean 0.57 ± 0.02) and was greater than 0.56 in six patients. Two patients had mild-to-moderate pulmonary venous congestion; none had radiographic evidence of pulmonary edema.

Eight patients were taking both digitalis and diuretic, six were taking digitalis alone and three were on no cardiac medications.

Hemodynamics and LV Function (Tables 1 and 2)

Only two patients had systolic gradients across the aortic valve (4 mm Hg and 16 mm Hg, respectively). Cardiac index was less than 2.5 1/min/m\(^2\) in four patients. LV end-diastolic pressure was ≥ 14 mm Hg in 11 patients. LV end-diastolic volume index (EDVI), LV end-systolic volume index (ESVI) and LV mass were increased in all patients.

LVEF was, by definition for inclusion in the study, reduced in all patients. EF was ≤ 0.29 in two patients, 0.30–0.39 in two patients and 0.40–0.49 in 13 patients. Inferior wall akinesis was present in one patient with a prior inferior MI. Preoperative mean Vcf was reduced to a greater degree (average 0.75 ± 0.05 circ/sec, normal ≥ 1.25 circ/sec) than EF (average 0.43 ± 0.02, normal ≥ 0.54).

Associated Lesions

Three patients (18%) had significant obstructive coronary artery disease (three-vessel disease in two and isolated left anterior descending artery stenosis in one); one had had a prior myocardial infarction (MI) and the other two complained of angina pectoris. Both patients with Marfan’s syndrome had ascending aortic aneurysms.

Surgery

No patient died at surgery. One patient developed complete atrioventricular block in the perioperative period and required implantation of a permanent pacemaker. One patient with normal preoperative coronary arteriograms developed a perioperative inferior MI by ECG criteria.

Follow-up

Follow-up has ranged from 4–50 months (average 25 ± 4 months [mean ± SEM]). The follow-up of patients who underwent repeat study is longer (31 ± 5 months) than that of patients who were not restudied (16 ± 3 months) (p < 0.05) (table 1).

Survival

There have been five late deaths. Two patients died with severe prosthetic obstruction 7 and 10 months after surgery; neither had received anticoagulant therapy postoperatively. One patient died suddenly at 9 months; an autopsy was not performed. One patient died of intractable ventricular arrhythmias at 19 months; repeat cardiac catheterization after an episode of syncope 1 month before death showed “normal” prosthetic valve function and moderate LV systolic pump dysfunction, with an EF of 0.41. One patient with Marfan’s syndrome and severely depressed preoperative LV function died of progressive congestive heart failure 29 months after valve replacement; repeat catheterization 8 months after surgery had shown “normal” prosthetic function and persistent severe systolic pump dysfunction, with an EF of 0.27.

The actuarially determined survival at 3 years is 61 ± 15% (mean ± SEM). If the two patients who died with prosthetic dysfunction were excluded, the 3-year survival would be 70 ± 16%.

Late Complications

Two patients had mild periprosthetic AI documented at follow-up catheterization. One patient suffered a cerebrovascular accident with residual hemiparesis 3 months after valve replacement. Her compliance with prescribed anticoagulant therapy was erratic, but her prosthetic valve function was “normal” at restudy. One patient underwent reoperation for prosthetic valve dysfunction with progressive intravascular hemolysis at 41 months; anticoagulation had been consistently adequate, and repeat study at 27 months had shown a “normally” functioning prosthesis.

Three patients experienced transient neurologic events; two of these were receiving no anticoagulant therapy and one was receiving only sulfinpyrazone. All were begun on sodium warfarin and none have had any recurrence of symptoms. One patient required hospitalization for gastrointestinal bleeding as a consequence of excessive anticoagulation.

Clinical Status

Changes in functional class after AVR are shown in figure 1. Functional class was maintained or improved in all late survivors. Cardiothoracic ratio decreased from 0.57 ± 0.02 (mean ± SEM) to 0.55 ± 0.03 (p = NS), and became normal (p < 0.05) in only three patients. The ECG remained abnormal in all survivors.

Four patients are taking digitalis and a diuretic, five are taking digitalis alone, and one is taking a diuretic alone for hypertension. All are receiving anticoagulants. Three patients have required long-term antiarrhythmic therapy and one is pacemaker-dependent.
Table 1. Selected Data from All Patients

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Abbreviations: FC = functional class (New York Heart Association criteria); CT = cardiothoracic; CI = cardiac index; LVEDP = left ventricular end-diastolic pressure; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; Vcf = velocity of circumferential fiber shortening; obs CAD = obstructive coronary artery disease; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; RCA = right coronary artery; OM = obtuse marginal branch of the left coronary artery; CVA = cerebrovascular accident; MI = myocardial infarction; GI = gastrointestinal; ND = not done; LD = late death; V arrhythm = ventricular arrhythmia; periprosth CHF = periprosthetic complete heart block; Asc AA = ascending aortic aneurysm; CHF = congestive heart failure; periprosth AI = periprosthetic aortic insufficiency; prosth dysfunct = prosthetic valve dysfunction; --- = data not available.

**Hemodynamics and LV Function**

Twelve patients underwent repeat catheterization after AVR, including four who subsequently died (see Methods section). Two patients who were not on anticoagulant therapy were suspected of having severe valve obstruction, and were found to have critical prosthetic valve stenosis (calculated aortic valve area indices of 0.1 and 0.4 cm²/m²); their postoperative data are not included in subsequent analyses.

In the remaining 10 patients, repeat study was performed an average of 14.5 ± 2.8 months (mean ± SEM) after surgery. There were no significant differences in age, pre- or postoperative functional class, or initial hemodynamic data between patients who were restudied and those who were not. All restudied patients were receiving digitalis at the time of both the initial and the follow-up catheterizations.

Prosthetic valve function was satisfactory in the 10 restudied patients. Three patients had no valvular gradient, while the other seven had gradients from 4-21 mm Hg (mean 11.5 ± 2.0 mm Hg). Calculated prosthetic aortic valve area was 2.2 ± 0.2 cm² (range 1.6-2.9 cm²) and the calculated valve area index was 1.3 ± 0.1 cm²/m² (range 0.9-1.6 cm²/m²). Two patients had mild periprosthetic AI.
Pre- and postoperative hemodynamic data are shown in Table 2 and figures 2 and 3. Cardiac index did not change significantly. LV end-diastolic pressure became normal in nine of 10 patients. There were significant reductions in LVEDVI, LVESVI and LV mass, but these values usually did not return to normal.

EF increased from 0.43 ± 0.03 to 0.49 ± 0.04 (0.05 < p < 0.10). EF fell slightly in three patients (one with a prior MI and inferior wall akinesis, one with mild periprosthetic AI and one with new postoperative apical akinesis but no ECG or clinical evidence of perioperative infarction) (fig. 3). In five patients, EF increased more than 15% above the preoperative value; in four of these, EF returned to normal (≥ 0.54).20 A cut-off of 15% was chosen because we have previously shown that increases of this magnitude are beyond the range of day-to-day variation in angiographically determined EF.11 Statistical analysis of preoperative factors, including age, functional class and hemodynamic variables, failed to distinguish between patients who improved their EF and those who did not. There was no significant difference in heart rate at the time of preoperative catheterization between patients who increased their EF (79 ± 6 beats/min) and those who did not (72 ± 9 beats/min) (p > 0.10). LV systolic pressure did not change significantly for the restudied group as a whole (153 ± 11 mm Hg preoperatively vs 152 ± 8 mm Hg postoperatively; p > 0.10). Similarly, analysis of prosthetic valve gradients and areas showed no significant correlation with postoperative change in EF.

For the 10 patients who were restudied, we examined postoperative changes in LVEDVI and EF, expressed as a percentage of the preoperative value (fig. 4). The five patients with greater than 15% increase in EF all had simultaneous reductions in LVEDVI. One patient had an 18% reduction in EF.

### Table 1. (Continued)

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<th>LV mass (g/m²)</th>
<th>Ejection fraction</th>
<th>Mean Vcf (cire/sec)</th>
<th>Associated obs CAD</th>
<th>Other lesions</th>
<th>Duration of follow-up (months)</th>
<th>Complications</th>
<th>Duration, surgery to restudy (months)</th>
</tr>
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<tbody>
<tr>
<td>185</td>
<td>0.29</td>
<td>0.31</td>
<td>LAD/LCX/RCA</td>
<td>None</td>
<td>4</td>
<td>CVA</td>
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<tr>
<td>152</td>
<td>0.45</td>
<td>0.75</td>
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<td>0.57</td>
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<td>Prior MI</td>
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<td>279</td>
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<tr>
<td>290</td>
<td>0.48</td>
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<td>0.47</td>
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<td>19</td>
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<tr>
<td>216</td>
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<td>45</td>
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<tr>
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<td>7</td>
<td>LD-prosth dysfunc</td>
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<td>0.47</td>
<td>1.05</td>
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<td>None</td>
<td>10</td>
<td>LD-prosth dysfunc</td>
<td>10</td>
</tr>
</tbody>
</table>

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TABLE 2. Hemodynamic Data in Studied Patients

<table>
<thead>
<tr>
<th>CI</th>
<th>LVEDP (mm Hg)</th>
<th>LVEDVI (ml/m²)</th>
<th>LVESVI (ml/m³)</th>
<th>LV mass (g/m²)</th>
<th>Ejection fraction</th>
<th>Mean Vcf (circ/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>n = 16</td>
<td>Preop mean = 29</td>
<td>SEM = 0.2</td>
<td>Preop mean = 22</td>
<td>SEM = 0.2</td>
<td>Postop mean = 10</td>
</tr>
<tr>
<td>Not restudied</td>
<td>n = 7</td>
<td>Preop mean = 12</td>
<td>SEM = 0.2</td>
<td>Preop mean = 5</td>
<td>SEM = 0.2</td>
<td>Postop mean = 38</td>
</tr>
<tr>
<td>Restudied</td>
<td>n = 9</td>
<td>Preop mean = 29</td>
<td>SEM = 0.2</td>
<td>Preop mean = 20</td>
<td>SEM = 0.2</td>
<td>Postop mean = 15</td>
</tr>
</tbody>
</table>

*p* *Two-tailed t test for paired data, preop vs postop in restudied patients. Abbreviations: CI = cardiac index; LVEDP = left ventricular end-diastolic pressure; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; Vcf = velocity of circumferential fiber shortening.

associated with reduced LVEDVI. There was no significant correlation between changes in EF and LVEDVI (r = -0.28; *p > 0.05*) in these 10 patients. Mean Vcf improved from 0.72 ± 0.08 to 0.95 ± 0.11 (*p < 0.05*). It increased in seven of 10 patients (fig. 3), but returned to normal (≥ 1.25 circ/sec) in only two patients. There was no significant change in LV ejection times between the pre- and postoperative studies (286 ± 14 msec vs 275 ± 13 msec; *p > 0.10*), a finding that has been reported. Changes in mean Vcf and EF were closely correlated (*r* = 0.93; *p < 0.001) (fig. 5). In no instance did mean Vcf and EF change in opposite directions. However, mean Vcf was more reduced preoperatively than was EF, and postoperative increases in mean Vcf were proportionately greater than increases in EF, which may explain why the average increase in mean Vcf was statistically significant and the increase in EF was not.

**Discussion**

AVR in 17 patients with isolated, severe AI and reduced EF resulted in a 3-year survival of 61%. Functional class was maintained or improved in all late survivors. In 10 patients who underwent repeat catheterization, LV dilatation and hypertrophy regressed in all but one patient, although usually not to normal, and LV systolic pump function improved in five.

Operative mortality for patients undergoing AVR is known to be increased in the presence of increased heart size, reduced EF, and advanced functional class. All of our patients had two or more of these factors, and the finding of no operative deaths was, therefore, unexpected. A more reasonable estimate of operative mortality in this type of patient is probably ≥ 6%. The lack of operative mortality in our small group of patients, however, suggests that valve

**FUNCTIONAL CLASS**

![Diagram of functional class]

**FIGURE 1. After aortic valve replacement, functional class was improved in eight late survivors and maintained in four others who had been functional class I or II preoperatively.**
replacement can be undertaken with acceptable risk in such patients.

Overall survival was 61% at 3 years, including two deaths in the first year due to prosthetic obstruction in the absence of anticoagulation. The dangers of thrombotic obstruction in nonanticoagulated ball-valve prostheses have now been clearly defined, and long-term follow-up of caged-ball prostheses suggests that catastrophic prosthetic obstruction is extremely uncommon with adequate anticoagulation. If we assume that these two deaths should be preventable and exclude them from analysis, the 3-year survival is 70%.

Despite major hemodynamic abnormalities in all patients, the degree of associated clinical disability varied widely. Others have noted that AI appears to be well tolerated by most patients for long periods of time, and that the presence or absence of symptoms is not consistently related to the degree of hemodynamic dysfunction. Nine of our patients were in functional classes I and II and eight were in functional classes III and IV. Not surprisingly, the patients in functional classes I and II had less severe hemodynamic impairment than those in functional classes III and IV; cardiac index was higher (3.3 ± 0.2 vs 2.5 ± 0.2 l/min/m², p < 0.01), LV end-systolic volume was smaller (102 ± 10 vs 149 ± 17 ml/m², p < 0.05) and LVEF was closer to normal (0.47 ± 0.01 vs 0.38 ± 0.03, p < 0.05). They also tended to be younger and have lower values for LV end-diastolic pressure and LVEDVI and higher values for mean Vcf, although these differences were not statistically significant. However, all of these patients had severe AI, LV dilatation and hypertrophy and impaired LV systolic pump function at rest.

Repeat hemodynamic evaluation was performed in 10 patients 14.5 ± 3 months after surgery. All restudied patients had satisfactory prosthetic valve function as assessed by valve gradient and calculated valve area and index. Therefore, we believe that the postoperative results in these 10 patients are representative of what may be expected after successful AVR.

In severe AI, the left ventricle compensates for volume overload by increasing end-diastolic volume in order to maintain forward stroke volume. Eccentric hypertrophy develops and permits normalization of systolic wall stress. Correction of volume overload after valve replacement resulted in a significant decrease in LV end-diastolic pressure, and significant but usually incomplete reductions of LV dilatation and hypertrophy. Similar results have been observed in patients with AI and more normal preoperative EF. An important finding of this study is that LV volumes and mass returned toward normal after AVR, despite moderately severe impairment of preoperative LV function.
The reasons for incomplete regression of compensatory changes remain unclear, but several possible contributing causes have been considered.4-6,37-40 A potential additional factor, recently emphasized,29 is that valve prostheses almost uniformly result in less-than-normal effective orifice size and therefore, in some degree of functional "stenosis," even in the presence of a "normally" functioning prosthesis. This problem is evident in our study group. The 10 restudied patients had "satisfactory" prosthetic function, yet seven had a measurable prosthetic valve gradient and five had a calculated aortic valve area index < 1.5 cm²/m². The total impact of residual outflow obstruction on postoperative hemodynamic improvement is not known, but we were unable to demonstrate any correlation between prosthetic valve gradient or calculated orifice size and postoperative changes in hemodynamic parameters in our patients.

Ejection phase indices of ventricular performance such as EF and mean Vcf are useful measures of systolic function;37,41,42 although they are known to be sensitive to acute changes in loading conditions of the heart.14,42,43 Thus, rapid increases in preload or reductions of afterload, as well as a true increase in myocardial function, may contribute to an improvement in LV systolic pump function. However, the effects of chronic alterations in preload or afterload (such as occur after relief of severe volume overload) on various measures of LV function are not well defined. Five of our restudied patients had significant improvement in EF and four of these returned to normal, while mean Vcf increased in seven patients but returned to normal in only two. Significantly improved EF was associated with reduced LVEDVI in each instance (fig. 4); however, no definite conclusions about the relationship of changing EDVI to EF can be drawn from this study because other variables that influence EF (afterload and contractility) were not controlled. Although changes in EF and mean Vcf were concordant in our patients, depression of mean Vcf
preoperatively and improvement in mean Vcf postoperatively were both proportionately greater than the corresponding changes in EF. The reasons for this apparent disparity are not clear.

Indirect evidence from animal†, and human studies suggests that ejection phase indices are not significantly altered during chronic volume overload in the absence of depressed myocardial function. Katz et al. recently performed noninvasive, serial studies of LV function during and after the "natural volume overload" of pregnancy in 19 subjects without preexisting heart disease; the echocardiographically determined EF, percent of fractional shortening and mean Vcf were neither enhanced nor depressed during the development and regression of LV dilatation and hypertrophy. However, ejection phase indices are dependent on the interrelationship of preload, afterload and myocardial function, norm values for EF and mean Vcf may reflect appropriate compensatory alterations in these variables and do not necessarily imply normal contractility. It is not surprising that patients with normal preoperative EF (i.e., who are adequately compensated) maintain their normal LV function (i.e., remain compensated) after correction of AI- or AS.

In patients whose compensatory mechanisms have failed, as indicated by reduced EF and mean Vcf, the relative contributions of excess preload, afterload and depressed myocardial function may be difficult to assess. For example, we have shown consistent and dramatic improvement in LV systolic function after AVR in patients with severe AS, severely depressed LV function and clinical heart failure. In contrast, our present study demonstrates significant improvement in EF for only 50% of patients with AI and moderately severe impairment of LV function, even though preoperative EF was reduced to a greater extent in the AS patients (EF 0.37 ± 0.02 vs 0.43 ± 0.02). Sustained and progressive increases in LV systolic pressure probably play a major role in the decompensation of patients with severe AS. It might, therefore, be expected that reduction of LV systolic pressure after AVR would permit a more normal relationship among the factors that determine ejection phase indices. In patients with severe AI, on the other hand, LV systolic pressure did not change significantly for the restudied group as a whole or for those with improved EF. Moreover, peak LV systolic wall stress is frequently normal and shows no change or only a slight decrease after AVR, a finding presumably related to proportionate reductions of LV mass and volume, as we have demonstrated, and of LV wall thickness and dimension, as demonstrated on M-mode echocardiography by Gaasch and co-workers. Other potential differences between AS and AI patients include the duration of pressure and volume overload, alterations in ventricular shape, myofibrillar slippage and differing degrees of myocardial cellular degeneration and/or fibrosis. However, the interrelationships and importance of these factors remain unknown. End-diastolic volume was reduced and, therefore, it seems probable that sarcomere length would have been reduced or at least unchanged. Therefore, the improvement in EF and mean Vcf in half of our patients may represent an improvement in myocardial function. Conversely, the lack of improvement in half of our patients may indicate a more severe or irreversible degree of myocardial dysfunction. Maron et al. have shown that AI induces degenerative changes in the myocardium. It is reasonable to suppose that the development of ultrastructural changes and impaired LV function occur gradually along a continuum. Consistent with this concept, both afterload stress and exercise appear to unmask LV dysfunction in many patients with AI who have normal resting systolic pump function. Little is known about the development or reversibility of myocardial changes in LV volume overload. Papadimitriou and associates produced aortocaval fistulas in dogs and noted changes in myocardial ultrastructure that regressed to normal after relief of volume overload. The duration of volume overload was relatively brief (28-43 days) in their study, and these results may not be applicable to the more prolonged course of most patients with chronic AI. In this context, we were encouraged to find that half of our patients could still improve their LV pump function despite moderately severe preoperative impairment. In our small group, however, analysis of preoperative factors including age, functional class and hemodynamic data did not distinguish between patients who improved their EF and those who did not.

Clinical Implications

Our data indicate that AVR can be performed with acceptable risk in patients with severe AI and impaired LV function. Functional class was improved or maintained in late survivors. LV systolic pump function improved in 50% of patients. There was consistent, but usually incomplete, regression of LV dilatation and LV hypertrophy, even in patients who did not improve their LV systolic pump function.

Because preoperative factors did not distinguish between patients who improved LV systolic function and those who did not, and because ventricular dilatation and hypertrophy can be expected to regress even in patients who do not improve their EF if there is no perioperative damage, we believe that all patients with severe, isolated AI and moderately severe impairment of LV function (EF 0.25-0.49) should be offered AVR unless there is a specific contraindication to surgery.

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Aortic Valvulotomy in Neonates

L. Henry Edmunds, Jr., M.D., Henry R. Wagner, M.D., and Michael A. Heymann, M.D.

SUMMARY Fourteen consecutive newborns, 1 month of age or younger (mean age 12 days; mean weight 3.5 kg), had aortic valvulotomy for critical life-threatening aortic stenosis. Seven infants died early and seven survived hospitalization. Clinical, laboratory, catheterization, operative and autopsy data were analyzed to determine factors relevant to success or failure.

Left ventricular chamber size and operability of the aortic valve and annulus were the most important determinants of outcome. All nonsurvivors except one had small left ventricles. All survivors had normal-sized ventricles and aortic annuli >5 mm. Left ventricular end-diastolic volumes (LVEDV) were calculated from cineangiograms in 10 infants. Six survivors and one nonsurvivor, who developed ventricular fibrillation before bypass, had LVEDV >35 ml/m². Three nonsurvivors had LVEDV <30 ml/m². Poor peripheral perfusion, severe acidosis (pH < 7.25) and right ventricular hypertension (>60 torr) were significantly more common in nonsurvivors than in survivors. Seven nonsurvivors and only one survivor either required immediate operation or developed ventricular fibrillation during operation before bypass.

Two subgroups of newborns with isolated aortic stenosis are identified. Those with small left ventricular chambers represent an intermediate group between patients with "hypoplastic left-heart syndrome" and normals. Aortic valvulotomy did not save these infants. In contrast, seven of eight newborns with normal-sized left ventricles survived valvulotomy and six remain alive 1.5-6.5 years after operation.

NEWBORNS with symptomatic aortic stenosis require urgent or emergency valvulotomy, and mortality is higher than in older infants and children. Between 1973-1978, 36 children and infants older than 1 month have had operation at Children’s Hospital of Philadelphia for valvar, supravalvar and subvalvar aortic stenosis, and all survived. Others also have reported low mortality. However, in newborns 1 month of age or younger, mortality was 29% and 71% in two series of seven patients each.

This study was undertaken to determine factors that affect survival after aortic valvulotomy in newborns.

Methods

Between November 1970 and October 1977, 14 newborns up to 1 month of age had aortic valvulotomy consecutively by one surgeon. Five infants were operated at the Moffitt Hospital, San Francisco (University of California) and nine were operated at Children’s Hospital of Philadelphia (University of Pennsylvania).

Patients with aortic atresia or associated mitral atresia, coarctation or interrupted aortic arch were excluded.

All patients had a preoperative cardiac catheterization. Cardiac output was estimated by the Fick method in 10 patients using an assumed value for oxygen consumption. Left ventricular angiograms were obtained retrogradely in two patients and via the mitral valve in eight. Left ventricular volumes were estimated in these 10 patients from right anterior oblique views or biplane posterior-anterior and lateral views. In two patients the left ventricle was not entered and in two others left ventricular angiograms were not obtained.

Cardiopulmonary bypass was planned in all patients, but could not be accomplished in two. Surface cooling to 30-32°C was used in three infants who were further cooled to 22-24°C during bypass. Moderate hypothermia to 30°C during bypass was used in nine patients. A variety of anesthetic agents was used; the most common agents were ketamine or nitrous oxide with a muscle relaxant. The valve was approached through a median sternotomy and an oblique incision in the ascending aorta. Valvulotomy was accomplished by incising stenosed commissures and occasionally by removing redundant obstructing tissue attached to thickened leaflets.

Seven infants survived hospitalization and were discharged. Six patients are alive 1.5-6 years later. One


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Received June 18, 1979; revision accepted August 7, 1979.

Valve replacement in aortic insufficiency with left ventricular dysfunction.
D G Clark, J H McAnulty and S H Rahimtoola

Circulation. 1980;61:411-421
doi: 10.1161/01.CIR.61.2.411
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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