Valve Replacement for Aortic Stenosis in Patients With Poor Left Ventricular Function

Comparison of Early Changes With Stented and Stentless Valves

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Background—Long-standing aortic stenosis causes significant left ventricular (LV) dysfunction, which may progress irreversibly. In many cases, LV function can be salvaged by aortic valve surgery, although debate exists regarding the best valve prosthesis to use.

Methods and Results—We studied 33 patients retrospectively who had significant aortic stenosis and impaired LV systolic function, as assessed by transthoracic Doppler echocardiography. Patients were assessed preoperatively and before discharge from the hospital. A total of 20 patients received a stentless (homograft or Toronto) valve, and 13, a stented valve. No patient had significant aortic regurgitation or other valvular disease. Preoperatively, fractional shortening was 18.8±5.5% in the stentless group and 18.6±3.8% in the stented group. Postoperatively, it was 25.6±6.9% (P<0.001 compared with baseline) and 17.0±2.8%, respectively (P<0.001 compared with stentless group). Fractional shortening improved because of a reduction in LV end-systolic and end-diastolic dimensions in the stentless group. Systolic long axis function at the LV free wall also recovered, with an increase in systolic excursion and both peak shortening and lengthening rates. No change was noted in mitral valve Doppler patterns.

Conclusions—Patients who received a stentless valve demonstrated a significantly greater early improvement in LV systolic function compared with those who received a stented valve. (Circulation. 1999;100[suppl II]:II-1–II-5.)

Key Words: echocardiography ■ valves ■ stenosis ■ surgery

Increased left ventricular wall tension due to aortic stenosis may lead to left ventricular dilatation and heart failure; the prognosis for such patients is extremely poor if valve replacement is not performed.2,3 Severe ventricular impairment is not in itself a contraindication to surgery, despite the increased intraoperative mortality, because the outcome of survivors is greatly improved.4,5 Echocardiography is used to measure the transvalvular pressure drop; but in patients with a failing ventricle, the measured gradient alone may underestimate the severity of the stenosis6,7; measurements based on the continuity equation may be more useful in estimating severity in these cases.

For patients with heart failure and aortic stenosis, valve replacement can result in rapid clinical improvement.8 However, the increased pressure drop across stented valves, both metallic valves and tissue, and the concomitant increase in ventricular wall stress may delay improvement in ventricular function after replacement.

Our hypothesis was that stentless biological valves (homograft and the Toronto stentless porcine valve), with a lower pressure drop, may allow more rapid improvement in ventricular function, which may be of clinical importance in the early postoperative period.

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All patients underwent preoperative and predischarge transthoracic echocardiography to assess peak transvalvular gradients, left ventricular end-systolic and end-diastolic dimensions, and fractional shortening. A subgroup of the patients had left ventricular free wall and septal long axis function assessed.

**Echocardiographic Technique**

Doppler echocardiographic examination was performed using a Hewlett-Packard echograph, model 77020 A Sonos 1500, interfaced to a 2.5-MHz phased-array transducer. Two-dimensional guided M-modes were obtained while the patient was lying in the semilateral position; simultaneous ECG and phonocardiogram were also obtained. Standard M-mode echograms of the left ventricular minor axis were obtained with the cursor by the tips of the mitral valve leaflets. From the apical 4-chamber view, we also recorded M-modes of the ventricular long axes, represented by mitral ring motion. The cursor was positioned at the left and septal sites of the ring. Transmital forward flow velocities were obtained using the same transducer in the pulsed-wave Doppler mode, with the sample volume at the tips of the mitral valve leaflets, from the apical 4-chamber view.

M-modes of the left ventricular minor and long axes and Doppler traces were recorded separately on a strip-chart recorder at a paper speed of 100 mm. All M-mode traces were digitized and analyzed using a dedicated computer program.

**Minor Axis**

Left ventricular end-diastolic dimensions were measured using leading-edge methodology at the onset of the Q wave of the ECG and the end-systolic dimension at the onset of the first high-frequency component of the second heart sound of the phonocardiogram (A2). Fractional shortening was calculated as the percentage of systolic decrease in the minor axis divided by end-diastolic dimension. Ejection fraction was calculated using the “cubed” formula: \( \left[ \frac{(\text{end-diastolic dimension})^3 - (\text{end-systolic dimension})^3}{(\text{end-diastolic dimension})^3} \right] \). Left ventricular mass was calculated using the Penn convention method: left ventricular mass = 1.04 × [(end-diastolic dimension + posterior wall thickness in diastole + interventricular septal thickness in diastole) − (end-diastolic dimension)] − 13.6 g.

From the digitized traces, we measured the peak rates of minor axis shortening and lengthening.

**Long Axis**

Two long axis sites were measured: the left ventricular free wall and septum. Systolic long axis excursion was taken as the amplitude of ring movement between the outermost point at the time of the Q wave (end diastole) to the innermost point at A2 (end systole). Peak shortening and lengthening rates were measured from the digitized traces.

**Operative Technique**

Under general anesthesia, cardiopulmonary bypass was routinely established through a medial sternal approach. Stentless porcine valves and aortic homografts were implanted through an oblique aortotomy, using a free-sawn technique and double suture lines. Mechanical or stented valves were selected at the choice of the operating surgeon and implanted by an interrupted single suture line.

**Statistical Analysis**

Values are expressed as mean±SD. Measurements in patients were compared with those in controls using the unpaired Student’s t test. Pre- and postoperative values within the patient groups were compared using a paired t test. A 5% probability was considered significant.

**Results**

**Patients**

A total of 332 aortic valve replacements for aortic stenosis were performed at our institution during this time; 33 (10%) of them (20 stentless and 13 stented) fulfilled the eligibility criteria of the study. Exclusion criteria were as follows: left ventricle fractional shortening >25% or left ventricle not dilated (n=281), atrial fibrillation occurred when assessed (n=12), and no postoperative echocardiogram was available (n=6). The mean age of the patients was 63.5±11 years; 29 (88%) were male. None of the patients had a history of coronary artery disease. No perioperative deaths, myocardial infarctions, or other significant perioperative events occurred in either group of patients. The type of valve substitute used was determined by the operating surgeon; details are shown in Table 1.

**Clinical Data**

The mean age of the stentless group was 63.5±10.9 years; in the stented group, it was 66.5±13.9 years. A total of 12 patients (92%) in the stented group and 17 (85%) in the stentless group were male. No patient in either group had more than mild aortic regurgitation. Preoperative aortic pressure drop was not different in the 2 groups; it was 72±19 and 67±14 mm Hg in the stentless and stented groups, respectively. Postoperatively, the aortic gradient was 12±6 mm Hg in the stentless group and 38±10 mm Hg in the stented group (P<0.001 for difference between groups). No differences in cardiac medications taken by the patients existed preoperatively. Postoperatively, those receiving a mechanical valve took anticoagulants. No patient was taking an angiotensin-converting enzyme (ACE) inhibitor at the time of either assessment.

**Minor Axis**

Left ventricular minor axis dimensions at end-diastole and end-systole were increased at baseline, and fractional shortening was reduced when patients were compared with controls. These values did not differ between the groups (Table 2). Postoperatively, patients receiving a stentless valve showed a significant decrease in mean end-systolic dimension, from 53±6 to 41±7 mm (P<0.001), and in end-diastolic dimension, from 65±7 to 55±6 mm (P<0.001). Fractional shortening increased from 19±6% to 26±7% in this group of patients (Figure 1); at 5 days, it did not differ significantly from controls. In contrast, no statistically significant change was seen in left ventricular dimensions or fractional shortening in the stented group (preoperative and

<table>
<thead>
<tr>
<th>TABLE 1. Valve Substitutes Used</th>
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<tbody>
<tr>
<td>Valve Used</td>
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<tr>
<td>Stentless (n=20)</td>
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</table>

*The mean diameter of stentless values was 27 mm, and of stented values, 26 mm.
Postoperative values for fractional shortening were 19.6±4% and 17.6±3%, respectively. Postoperatively, fractional shortening was significantly higher in the stentless group than the stented group (P<0.001 for the comparison). The improvement in fractional shortening was reflected in the calculated ejection fraction; in the stentless group, it increased from 45.6±10% to 57.6±12%*† (P<0.001). No significant change occurred in the stented group. Left ventricular mass fell from 338±672 to 265±64 g (P<0.001) in the stentless group, but no significant change occurred in the stented group (329±51 g preoperatively and 304±68 g postoperatively). No significant difference in mitral Doppler was seen in either early (E) or late (A) diastolic filling velocities or the E/A ratio.

**Long Axis**
Pre- and postoperative assessments were performed in 9 patients in the stentless group and 10 in the stented group. Preoperative left ventricular free wall and septal excursion,

**TABLE 2. Minor Axis**

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=21)</th>
<th>Stentless (n=20)</th>
<th>Stented (n=13)</th>
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<tr>
<td></td>
<td>Preoperative</td>
<td>Postoperative</td>
<td>Preoperative</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Postoperative</td>
</tr>
<tr>
<td><strong>Left Ventricle</strong></td>
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<tr>
<td>EDD, mm</td>
<td>48±5</td>
<td>64.7±6.6</td>
<td>54.9±6.2††</td>
</tr>
<tr>
<td>ESD, mm</td>
<td>33±5</td>
<td>52.5±5.8</td>
<td>41.1±7.3††</td>
</tr>
<tr>
<td>Peak shortening rate, cm/s</td>
<td>9.0±3.0</td>
<td>5.7±2.6</td>
<td>5.8±2.9</td>
</tr>
<tr>
<td>Peak lengthening rate, cm/s</td>
<td>10.4±2.6</td>
<td>7.0±3.3</td>
<td>6.8±3.3</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>30±10</td>
<td>18.8±5.5</td>
<td>25.6±6.9††</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>68±6</td>
<td>45±10</td>
<td>57±12††</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>190±10</td>
<td>338±72</td>
<td>265±64††</td>
</tr>
<tr>
<td><strong>Mitral Doppler</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>E wave, m/s</td>
<td>0.7±0.1</td>
<td>0.73±0.35</td>
<td>0.75±0.35</td>
</tr>
<tr>
<td>A wave, m/s</td>
<td>0.5±0.1</td>
<td>0.54±0.33</td>
<td>0.51±0.32</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>1.4±0.4</td>
<td>1.85±1.78</td>
<td>1.68±1.22</td>
</tr>
</tbody>
</table>

All values are mean±SD. EDD indicates end-diastolic dimension, and ESD, end-systolic dimension. P<0.01 when all preoperative values (for left ventricle and fractional shortening) were compared with controls.

*P<0.01 compared with preoperative values using paired t-test.
†P<0.01 when stentless values were compared with stented ones using unpaired t-test.

**Figure 1.** Left ventricular M-mode recording of minor axis in patient before (left) and after (right) aortic valve replacement with homograft. Note marked regression in increased left ventricular dimensions and normalization of fractional shortening after surgery. PCG indicates phonocardiogram; EDD, end-diastolic dimension; and ESD, end-systolic dimension.)
peak shortening, and peak lengthening were reduced compared with normal values (Table 3). Postoperatively, no change occurred in septal values for either group, but left ventricular free wall excursion and shortening and lengthening velocities all increased toward normal only in the stentless group (Figure 2). No significant changes occurred in any of the long axis values in the group receiving stented valves.

**Discussion**

We studied a group of patients with aortic stenosis and severely impaired systolic function in the absence of coronary artery disease. These patients represent 10% of all operations for aortic stenosis. Despite similar preoperative values, left ventricular dimensions had decreased after an average of 5 days postoperatively, but only in patients receiving a stentless valve. A corresponding increase occurred in left ventricular systolic fractional shortening and ejection fraction to values approximating the normal range. E/A ratio and transmitral flow velocities in early and late diastole did not alter.

Left ventricular dilatation and dysfunction are often the result of long-term aortic stenosis. Aortic valve replacement can result in an improvement in ventricular function, even in patients with severe heart failure, despite a rate of operative mortality of ~10%. Stentless valves have a beneficial early effect on hemodynamic indices because of their low profile. More recently, a 6-month follow-up of patients (with either preoperative aortic stenosis or regurgitation) receiving the stentless Toronto valve demonstrated an improvement in fractional shortening that was not seen in patients with stented valves. Whether these results reflect any changes in clinical outcome is unclear.

**TABLE 3. Long Axis**

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=21)</th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>Preoperative</th>
<th>Postoperative</th>
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<tbody>
<tr>
<td><strong>Left ventricular free wall</strong></td>
<td></td>
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<tr>
<td>Excursion, cm</td>
<td>1.5±0.25</td>
<td>0.8±0.3†</td>
<td>1.1±0.4*</td>
<td>1.0±0.4</td>
<td>1.0±0.3</td>
</tr>
<tr>
<td>Peak shortening rate, cm/s</td>
<td>8±1.5</td>
<td>3.9±1.6†</td>
<td>5.7±2.4†</td>
<td>4.5±1.5</td>
<td>6.1±2.7</td>
</tr>
<tr>
<td>Peak lengthening rate, cm/s</td>
<td>10±2.5</td>
<td>3.6±2.7†</td>
<td>4.8±1.3*</td>
<td>3.9±1.4</td>
<td>5.1±1.7</td>
</tr>
<tr>
<td><strong>Left ventricular septum</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Excursion, cm</td>
<td>1.5±0.3</td>
<td>0.6±0.3†</td>
<td>0.6±0.2</td>
<td>0.9±0.4</td>
<td>0.6±0.2</td>
</tr>
<tr>
<td>Shortening velocity, cm/s</td>
<td>7.5±1.2</td>
<td>2.6±1.2†</td>
<td>2.4±1.2</td>
<td>3.2±1.3</td>
<td>3.0±1.4</td>
</tr>
<tr>
<td>Lengthening velocity, cm/s</td>
<td>9±1.5</td>
<td>2.7±2.2†</td>
<td>2.9±1.5</td>
<td>2.5±2.3</td>
<td>4.0±1.5</td>
</tr>
</tbody>
</table>

All values are mean±SD. *P*<0.01 when all preoperative values were compared with those of controls. **TABLE 3. Long Axis**

*P*<0.05, †P<0.01 compared with preoperative values using paired t-test.

Figure 2. M-mode recording of left ventricular free wall long axis before and after aortic valve replacement with Toronto stentless valve. Note significant increase in long axis systolic excursion and peak shortening and lengthening rates after surgery. Depth markers represent 1 and 0.2 cm. PCG indicates phonocardiogram, and A2, aortic valve closure component of second heart sound.
A marked reversibility of preoperative left ventricular dysfunction was seen in our study. Therefore, it seems that there may be a subgroup of patients for whom left ventricular dilatation and dysfunction may be rapidly reversible. Improved recovery of function in patients receiving stentless valves compared with those receiving stented valves may occur because ventricular function in these patients is particularly sensitive to even mild obstructive gradients. Thus, a pressure drop of just 30 mm Hg developed by normally functioning mechanical valves (or stented valves) may be enough to delay recovery. The lower afterload associated with stentless valves reduces wall stress, allowing recovery of ventricular function.

In our other studies, seemingly long-term deterioration in ventricular function could be reversed by altering the afterload with ACE inhibitors or successful peripheral vascular reconstruction.17,18 ACE inhibitors cause a decrease in afterload that is associated with recovery of left ventricular dimensions and symptomatic improvement.

The left ventricular dysfunction seen in our patients contained a component that promptly reverted toward normal after aortic valve replacement with a stentless valve. The short time interval involved suggests that this change was the direct effect of a reduction in outflow tract resistance rather than remodeling. The extent of this early change will likely correlate with the degree to which resistance falls, and it is thus greater with a stentless prosthesis. We conclude that the left ventricle, although poorly functioning, can still rapidly improve in function shortly after alleviating outflow tract resistance in patients with aortic stenosis.

The present study is limited by being retrospective, non-randomized, and having a relatively small sample size. A few patients with poor left ventricular function were excluded because a postoperative echocardiogram was not available.

We hypothesized that the use of stentless valves in patients with aortic stenosis and markedly reduced ventricular function may result in more rapid recovery of function compared with similar patients receiving a stentless valve. Larger prospective studies with longer term follow-up are required to determine whether these early changes improve clinical outcomes.

References
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