Is repair of aortic valve regurgitation a safe alternative to valve replacement?

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Objective: To assess outcome of valve repair in patients with aortic valve regurgitation with emphasis on incidence and risk of reoperation.

Methods: We retrospectively reviewed 160 consecutive patients (127 men) who underwent aortic valve repair between 1986 and 2001. Ages ranged from 14 to 84 years (mean 55 ± 17 years). Patients were categorized according to the main etiology of valve disease: 63 patients (39%) had annular dilation leading to central leakage, 54 (34%) had bicuspid valve, 34 (21%) with tricuspid valve had cusp prolapse, and 9 (6%) had cusp perforation. Repair methods included commissural plication (n = 154, 96%), partial cusp resection with plication (n = 47, 29%), resuspension or cusp shortening (n = 44, 28%), and closure of cusp perforation (n = 10, 6%).

Results: There was 1 early death (0.6%). Two patients required repair of the aortic valve during initial hospitalization. During a mean follow-up of 4.2 years, there were 16 late deaths. Overall, 16 of 159 hospital survivors had late reoperation on the aortic valve (mean interval 2.8 years) without early mortality. Risks of reoperation on the aortic valve were 9%, 11%, and 15% at 3, 5, and 7 years, respectively.

Conclusions: Aortic valve repair can be performed with low risk and excellent freedom from valve-related morbidity and mortality. Late recurrence of aortic valve regurgitation led to reoperation in 8.8% of patients, but mortality associated with subsequent procedures is low. Aortic valve repair appears to be a good option for selected patients, particularly young patients who wish to avoid chronic anticoagulation with warfarin.

Aortic valve replacement has been the standard surgical procedure for treatment of aortic valve regurgitation since reliable prosthetic valves became available. In most centers, aortic valve repair has been reserved for patients with valve leakage due to aortic disease (dissection or dilatation) or associated ventricular septal defect. Although the early and late outcomes of valve replacement have steadily improved, there are important limitations and complications of prosthetic valves, especially for younger individuals. Thus, valve repair may be a useful option for selected patients.

Indeed, data from several centers indicate that survival and functional outcome after mitral valve repair are superior to outcomes following mitral valve replacement; however, overall experience with repair of aortic valve regurgitation is
relatively small, and reported series include patients having primary valve disease as well as aortic valve regurgitation secondary to disease of the ascending aorta or ventricular septal defect.4-7,9 As is true for the mitral valve, etiology of valve regurgitation would be expected to have a strong influence on outcome of aortic valve repair, especially on late risk of reoperation.10-12 The aims of this study were to assess the early and late outcomes of valve repair in patients with aortic valve regurgitation particularly as regards incidence and risk of reoperation.

Patients and Methods

Patients

From February 1986 through December 2001, 160 consecutive patients underwent aortic valve repair procedure at the Mayo Clinic: this cohort represents 13% of all primary operations for severe aortic valve regurgitation during the study interval. They were operated on by 7 staff surgeons. Our investigation specifically excluded the patients who developed aortic valve regurgitation associated with acute or chronic aortic dissection or ventricular septal defect; also excluded were patients with annuloaortic ectasia who underwent aortic valve-sparing operations.

We reviewed Mayo Clinic charts and operative records to identify the patient characteristics, etiology of valve disease, operative techniques, and surgical results. Late outcomes were determined from Clinic records when available or from written correspondence with patients’ physicians and direct patient contact with mailed questionnaire or telephone interviews when necessary. Clinical and echocardiographic data at latest follow-up were collected by contacting referring physicians. This study was approved by the Mayo Foundation Institutional Review Board, and patients or families gave informed consent.

Patient Demographics

Of the 160 patients, 127 (79%) were men, and ages ranged from 14 to 84 years (mean 55 ± 17 years). Important associated cardiovascular problems included systemic hypertension (n = 56, 35%) and coronary artery disease (n = 33, 21%), congestive heart failure (n = 27, 17%), infective endocarditis (n = 12, 8%), and prior myocardial infarction (n = 9, 6%). Eight patients (5%) were found to have systemic diseases including Takayasu’s arteritis in 3, systemic lupus erythematosus in 2, giant cell arteritis in 1, Kawasaki’s disease in 1, and juvenile rheumatoid arthritis in 1. Nine patients (6%) had undergone cardiovascular operations prior to aortic valve repair including repair of aortic coarctation (n = 3) or other procedures. As regards functional status preoperatively, 44 patients (28%) were in New York Heart Association (NYHA) functional class I, 50 patients (31%) were in class II, 63 (39%) were in class III, and 3 (2%) were in class IV.

Surgical Indications and Echocardiographic Findings

Most patients were referred for operation because of severe aortic valve regurgitation. Other indications for operation included aortic valve regurgitation in patients referred for repair of severe mitral valve leakage and moderate or severe aortic valve regurgitation in patients with severe coronary artery disease who required revascularization. In the latter patients, the decision for aortic valve repair was made intraoperatively with information from transesophageal echocardiography. Transesophageal Doppler echocardiography was used to judge adequacy of repair intraoperatively, and transthoracic Doppler echocardiographic studies were performed routinely prior to hospital dismissal.

Operative Techniques and Classification of Main Etiology of Aortic Valve Regurgitation

All patients underwent operations via median sternotomy with cardiopulmonary bypass established through ascending aortic cannulation and single or bicaval venous cannulation with normothermia or mild hypothermia. Profound hypothermia was used in 4 patients who required circulatory arrest for proximal aortic arch reconstruction. The mean aortic crossclamp and cardiopulmonary bypass times were 46 ± 20 and 64 ± 33 minutes, respectively.

The patients were categorized into 4 groups according to etiology of aortic valve regurgitation:

1. Annular dilation (n = 63, 39%). Annular dilation without cusp prolapse or perforation can cause central valve leakage due to inadequate cusp apposition. Mean age of patients with this mechanism was 54 ± 17 years. Repair was accomplished by commissural plication; placement of 1 or 2 pledgetted, horizontal mattress stitches with 3-0 braided polyester suture through the aortic wall at each commissure so as to narrow the angle of the commissures, reduce the circumference of the annulus, and increase the surface area of cuspal coaptation (Figure 1, A). Care was exercised to avoid excessive narrowing leading to functional aortic stenosis. Early after repair of severe aortic valve regurgitation, patients often had higher than normal aortic valve gradients because of a large left ventricular stroke volume, and if there was any question of adequacy of the aortic valve area after plication, the orifice was calibrated with a dilator to confirm adequate annular size.

2. Bicuspid valve (n = 54, 34%). In most patients with congenital bicuspid aortic valve disease, regurgitation is caused by retraction and/or prolapse of the conjoint cusp; the commonest anatomical finding is a conjoint cusp beneath the right and left coronary sinuses. Mean age of this group was 41 ± 14 years. Valves were repaired by limited triangular resection and suture repair of the median raphe with continuous 5-0 polypropylene suture. This maneuver shortens and elevates the free edge of the cusp, permitting apposition with the noncoronary cusp (Figure 1, B). The length of the resection from the free edge toward the annulus should be no more than 30% to 50% of the entire length to preserve the dimension of the cusps and to avoid suture of the thinner area of the central cusp. In a few patients with very pliable cusps, prolapse of the conjoint cusp can be corrected by simple plication suture without resection. Because some degree of annular dilation almost always accompanies regurgitation of a bicuspid aortic valve, commissural plication was combined with cusp repair (Figure 1, B).

3. Cusp prolapse of tricuspid aortic valve (n = 34, 21%). Regurgitation of tricuspid aortic valves may be due to prolapse of 1 or more cusps, usually with elongation of the free edge. Mean age of patients in our study with this mechanism was 62 ± 15 years. The most commonly used technique to
resuspend the cusp was cusp plication near the commissure with 5-0 polypropylene suture as described by Trusler and colleagues. Other methods of repair for isolated cusp prolapse included limited triangular resection and plication of the prolapsing cusp and resuspension of the free edge of the cusp by weaving with 5-0 polytetrafluoroethylene suture along the free edge and anchoring it to the commissure.

4. Cusp perforation (n = 9, 6%). Cusp perforation resulting from infective endocarditis was repaired by patch closure utilizing autologous pericardium secured with interrupted or continuous 6-0 polypropylene sutures (Figure 1, C); mean age of this group was 46 ± 17 years.

Surgical methods are summarized in Table 1. The other technique used was cusp shaving; hypertrophied or calcified nodules of

Figure 1. A, Annular dilation of aortic valve causes central regurgitation. Plication stitches are placed in the aortic wall at each commissure. B, Bicuspid aortic valve with prolapse of the conjoint cusp. Triangular resection is made in the midportion of the conjoint cusp and then repaired. Commissural plication stitches are also inserted. C, Perforated cusp is repaired with patch closure using pericardium.
TABLE 1. Operative methods of aortic valve repair and concomitant procedures (n = 160)

<table>
<thead>
<tr>
<th>Operative techniques</th>
<th>Patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Commissural plication</td>
<td>154 (96%)</td>
</tr>
<tr>
<td>Partial cusp resection with plication</td>
<td>47 (29%)</td>
</tr>
<tr>
<td>Resuspension or cusp shortening</td>
<td>44 (28%)</td>
</tr>
<tr>
<td>Closure of cusp perforation</td>
<td>10 (6%)</td>
</tr>
<tr>
<td>Cusp shaving</td>
<td>7 (4%)</td>
</tr>
<tr>
<td>Concomitant procedures</td>
<td>98 (61%)</td>
</tr>
<tr>
<td>MV repair</td>
<td>42 (26%)</td>
</tr>
<tr>
<td>CABG</td>
<td>29 (18%)</td>
</tr>
<tr>
<td>Repair of ascending aorta</td>
<td>27 (17%)</td>
</tr>
<tr>
<td>Septal myectomy</td>
<td>9 (6%)</td>
</tr>
<tr>
<td>TV repair</td>
<td>5 (3%)</td>
</tr>
<tr>
<td>MVR</td>
<td>5 (3%)</td>
</tr>
<tr>
<td>Closure of ASD/PFO</td>
<td>5 (3%)</td>
</tr>
<tr>
<td>Maze procedure</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>Membranectomy of SAS</td>
<td>3 (2%)</td>
</tr>
<tr>
<td>Tumor resection</td>
<td>2 (1%)</td>
</tr>
<tr>
<td>Left ventricular aneurysmectomy</td>
<td>1 (1%)</td>
</tr>
</tbody>
</table>

ASD, Atrial septal defect; CABG, coronary artery bypass grafting; MV, mitral valve; MVR, mitral valve replacement; PFO, patent foramen ovale; SAS, subaortic stenosis; TV, tricuspid valve.

Arantius may prevent complete coaptation, and this can be improved by thinning the area to restore pliability of the free edge (7 patients, 4%). Concomitant procedures were performed in 98 patients (61%). The most common associated operations were mitral valve repair (42 patients, 26%) and coronary artery bypass grafting (29 patients, 18%). Repair of dilated ascending aorta was performed in 27 patients (17%), including graft replacement in 19 and partial wedge resection and primary closure in 8; all patients were found to have near normal diameter of sinotubular junction.

Statistical Analysis
Postoperative survival and freedom from reoperation were estimated by the Kaplan-Meier method. Overall survival was compared with the expected survival of persons of the same age and sex, as derived from vital statistics for the west north central region of the United States. The statistical significance of observed versus expected survival was assessed with a 1-sample log-rank test. The associations of potential risk factors to survival and reoperation were assessed with log-rank tests and the Cox proportional hazards model. Data are expressed as mean ± standard deviation. Early operative mortality was defined as death occurring within 30 days of operation or at any time during the index hospitalization.

Results
Early Results
There was 1 early death (0.6%); this occurred in an 80-year-old man who underwent aortic valve repair combined with mitral valve repair, coronary artery bypass grafting, and extended left ventricular septal myectomy. The patient was separated from cardiopulmonary bypass with minimum inotropic support; however, he subsequently developed left ventricular free wall rupture requiring multiple attempts to repair and died in the intensive care unit due to cardiac failure on the fifth postoperative day.

Two patients had reoperation for aortic valve re-repair during initial hospitalization. In each patient, routine pre-dismissal transthoracic echocardiography identified significant new aortic valve regurgitation compared with that present intraoperatively at the conclusion of repair. Both patients had bicuspid aortic valves and were found to have dehiscence at the plication sutures on the conjoint cusp. Re-repair by resuturing the cusp was successful with satisfactory late results.

Other nonfatal complications included exploration for bleeding in 3 patients (1.9%), neurological event in 3 (1.9%) and respiratory failure in 3 (1.9%).

Preoperative and postoperative echocardiographic evaluations of the aortic valve are summarized in Table 2. Notice that in 11 patients, significant aortic valve regurgitation was missed by the prebypass echocardiographic study.

Late Results
Clinical follow-up was obtained in 152 of 159 hospital survivors (96%), and the mean follow-up period was 4.2 ± 2.6 years (maximum follow-up 14 years). There were 16 late deaths, and half were known to be due to cardiovascular causes including myocardial infarction in 3, aneurysm rupture in 2, and stroke in 1. Importantly, there was only 1 late death due to congestive heart failure and 1 sudden death. There were 4 other noncardiovascular deaths and 4 unknown deaths. Overall survival estimates at 3, 5, and 7 years were 96%, 92%, and 89%, respectively, and as seen in Figure 2, survival of study patients was similar to that of an age- and sex-matched population. Estimates of freedom from known cardiac-related death (myocardial infarction, congestive heart failure, stroke, sudden death) at 3, 5, and 7 years were 97%, 96%, and 96%, respectively.

On univariate analysis, preoperative variables associated with late death were coronary artery disease (P = .005) and older age (P = .002). A multivariate analysis was not done because of the small number of late events.
Late Reoperation and Other Valve-Related Complications

Seventeen patients required late cardiovascular operations, and 16 of these had aortic valve replacement (Table 3); 1 patient had thoracoabdominal aortic aneurysm repair. Mean interval between initial operation and late aortic valve reoperation was 2.8 ± 2.5 years. Among patients having late aortic valve reoperation, the etiologies of aortic valve regurgitation at the initial operation were bicuspid valve in 6 patients, annular dilation in 5 patients, and cusp prolapse of tricuspid valve in 5 patients. No patients having repair of cusp perforation required late reoperation.

The primary indications for late reoperation were severe aortic valve regurgitation in 13 patients, severe mitral valve regurgitation in 1, aortic valve stenosis in 1, and ascending aortic aneurysm in 1. Thus, late failure of the original aortic valve repair was the primary indication for reoperation in 14 of 16 patients. Of note, there were no early deaths related to reoperation. Cumulative risks of aortic valve reoperation at 3, 5, and 7 years were 9%, 11%, and 15%, respectively (Figure 3). Risk of aortic valve reoperation due to severe aortic valve regurgitation at 5 years was 10%, and for patients who had aortic valve repair without mitral valve procedure, risk of reoperation at 5 years was 6%.

Occurrence of late reoperation was also stratified according to the main etiology of aortic valve regurgitation at initial repair (Figure 4). Because the numbers of patients in each group were relatively small, there were no significant differences in rates of reoperation. However, the highest rate of reoperation was seen in patients who had repair of tricuspid aortic valves with cusp prolapse, and, again, there were no reoperations in patients who had repair of cusp perforations.

The late occurrences of other complications generally considered to be valve-related were low. During follow-up, 6 patients developed gastrointestinal bleeding, 5 had stroke or transient ischemic events, and 1 patient had intracranial bleeding. No infective endocarditis was noted. Estimates of freedom from stroke, bleeding, and endocarditis at 5 years were 98%, 94%, and 100%, respectively. The linearized rate of these complications (combined) was 1.0% per patient-year.

Among the 143 late survivors, 27 patients (19%) were receiving warfarin for various reasons, including thromboembolism prophylaxis for a mechanical prosthesis (aortic or mitral position, n = 14), chronic atrial fibrillation (n = 6), history of deep venous thrombosis (n = 3), and history of stroke (n = 2). The indication for anticoagulation was unknown in 2 patients. Of the 130 patients who at most recent follow-up were alive with their original aortic valve repair, only 11 (8.5%) were on warfarin.

Follow-up Doppler echocardiographic data were obtained from 104 of 130 survivors (free from late death and aortic valve reoperation). The mean interval between the date of operation and the date of most recent transthoracic echocardiogram was 2.8 years. Degrees of aortic valve regurgitation were severe in 2, moderately severe in 3, moderate in 26, trivial to mild in 62, and none in 11. There were no patients with hemodynamically important mitral stenosis (Table 2).

At last contact, 107 patients (75%) were in NYHA class I, 30 (21%) were in class II, and 5 (3%) were in class III.

Discussion

Management of patients with valvular regurgitation has changed markedly over the last 2 decades. This is particularly true for mitral valve regurgitation, due to several factors including the accuracy of echocardiography in defining the severity and mechanism of valve leakage, a better understanding of the natural history of mitral valve disease, and the refinements and predictability of repair of degenerative mitral valve disease. Less progress has been made in management of patients with chronic aortic valve regurgitation, but recent studies do suggest that the traditional

| TABLE 2. Echocardiographic assessment of aortic valve before and after repair |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| AR grade | 0, n (%) | 1, n (%) | 2, n (%) | 3, n (%) | 4, n (%) | 0, n (%) | 1, n (%) | 2, n (%) | 3 or 4, n (%) |
| Pre-CPB* | 1 (1) | 10 (6) | 49 (31) | 40 (25) | 51 (32) | 62 (40) | 31 (21) | 26 (17) | 6 (4) |
| Post-CPB† | 40 (26) | 106 (70) | 6 (4) | 0 (0) | 0 (0) | 94 (65) | 43 (31) | 6 (4) | 0 (0) |
| At dismissal‡ | 23 (15) | 120 (77) | 13 (8) | 0 (0) | 0 (0) | 57 (60) | 29 (31) | 9 (10) | 0 (0) |
| Follow-up§ | 11 (11) | 62 (60) | 26 (25) | 3 (3) | 2 (2) | 57 (60) | 29 (31) | 9 (10) | 0 (0) |

AR: Aortic regurgitation; AS: aortic stenosis; CPB: cardiopulmonary bypass; grade 0: none; grade 1: trivial or mild; grade 2: moderate; grade 3: moderately severe; grade 4: severe.

*Pre-CPB data were obtained from intraoperative transesophageal echocardiography before cardiopulmonary bypass, available in 151 patients of 160.
†Post-CPB data were obtained from intraoperative transesophageal echocardiography after cardiopulmonary bypass, available in 152 patients of 160.
‡At dismissal, data were obtained from transthoracic echocardiography at the time of hospital dismissal, available in 156 patients for aortic regurgitation and 143 patients for aortic stenosis.
§Follow-up data were obtained from transthoracic echocardiography of the patients who were free from late death and late aortic valve reoperation, available in 104 patients for aortic regurgitation and 95 patients for aortic stenosis.
conservative strategy of deferring valve replacement until there is clear-cut evidence of ventricular dysfunction, progressive left ventricular enlargement, or symptoms may result in excess mortality. Indeed, earlier intervention for aortic valve repair or replacement may prove to be the best strategy for patients who have well-documented severe aortic valve regurgitation.

In contrast to the situation with degenerative mitral valve disease, where repair is the rule rather than the exception, operation for chronic aortic valve regurgitation usually leads to prosthetic replacement, and concerns of clinicians regarding late complications of prostheses only reinforces a conservative clinical approach to patients.

Most previous reports of aortic valve repair have included patients with acute and chronic aortic dissection, annuloaortic ectasia, or congenital heart problems and the role of repair for primary aortic valve disease is not well defined. The present study provides information on the safety and durability of aortic valve repair in such patients. Although patients in this series were highly selected, the very low operative mortality compares very favorably with contemporary reports on risk of aortic valve replacement with biological and mechanical heart valves. Surely, there is no suggestion that early mortality is increased with valve repair. Our study population did not include those patients in whom an initial attempt at valve repair was

### Table 3. Summary of late aortic valve reoperations

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (y)</th>
<th>Interval (mo)</th>
<th>Etiology</th>
<th>Concomitant procedure</th>
<th>Post-repair AR (TEE)</th>
<th>Dis-TTE AR</th>
<th>Primary indication</th>
<th>Redo procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68</td>
<td>31</td>
<td>Dilation</td>
<td></td>
<td>1</td>
<td>1</td>
<td>Severe AR</td>
<td>AVR</td>
</tr>
<tr>
<td>2</td>
<td>75</td>
<td>12</td>
<td>Dilation</td>
<td>Asc.Ao Rep/CABG</td>
<td>1</td>
<td>1</td>
<td>Severe AR</td>
<td>AVR/CABG</td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>51</td>
<td>Bicuspid</td>
<td></td>
<td>1</td>
<td>1</td>
<td>Asc.Ao Anerysm</td>
<td>CG</td>
</tr>
<tr>
<td>4</td>
<td>59</td>
<td>131</td>
<td>Dilation</td>
<td>CABG</td>
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<td>AVR/CABG</td>
</tr>
<tr>
<td>5</td>
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<td>Prolapse</td>
<td>MV repair</td>
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<td>1</td>
<td>Severe AR</td>
<td>AVR/MVR</td>
</tr>
<tr>
<td>6</td>
<td>65</td>
<td>11</td>
<td>Prolapse</td>
<td>MV repair</td>
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<td>1</td>
<td>Severe AR</td>
<td>AVR with RE</td>
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<tr>
<td>7</td>
<td>21</td>
<td>25</td>
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<td>AVR</td>
</tr>
<tr>
<td>8</td>
<td>62</td>
<td>68</td>
<td>Bicuspid</td>
<td>MV repair/maze</td>
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<td>1</td>
<td>(AS1) Severe MR</td>
<td>AVR/MVR</td>
</tr>
<tr>
<td>9</td>
<td>74</td>
<td>36</td>
<td>Prolapse</td>
<td>MV repair</td>
<td>1</td>
<td>1</td>
<td>Severe AR</td>
<td>AVR/MVR/CABG</td>
</tr>
<tr>
<td>10</td>
<td>53</td>
<td>32</td>
<td>Dilation</td>
<td>MV repair</td>
<td>1</td>
<td>1</td>
<td>Severe AR</td>
<td>AVR/MVR</td>
</tr>
<tr>
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<td>21</td>
<td>9</td>
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<td>1</td>
<td>Severe AR</td>
<td>AVR</td>
</tr>
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<td>12</td>
<td>52</td>
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<td>2</td>
<td>Severe AR</td>
<td>AVR</td>
</tr>
<tr>
<td>13</td>
<td>73</td>
<td>43</td>
<td>Dilation</td>
<td>MV repair</td>
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<td>1</td>
<td>Severe AR</td>
<td>AVR/MVR/CABG</td>
</tr>
<tr>
<td>14</td>
<td>57</td>
<td>35</td>
<td>Prolapse</td>
<td></td>
<td>1</td>
<td>2</td>
<td>Severe AR</td>
<td>AVR</td>
</tr>
<tr>
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<td>Severe AR</td>
<td>AVR</td>
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<td>22</td>
<td>12</td>
<td>Prolapse</td>
<td>MV repair</td>
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<td>1</td>
<td>(AS2) Severe AS</td>
<td>AVR with RE/MVR</td>
</tr>
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<td>34</td>
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</table>

**Notes:**
- AR, Aortic regurgitation; AS, aortic stenosis; Asc.Ao, ascending aorta; Asc.Ao Rep, ascending aortic replacement; AVR, aortic valve replacement; CABG, coronary artery bypass grafting; CG, composite graft aortic root replacement; MS, mitral stenosis; MR, mitral regurgitation; MV, mitral valve; MVR, mitral valve replacement; RE, aortic root enlargement; grade 0, none; grade 1, trivial or mild; grade 2, moderate; grade 3, moderately severe; grade 4, severe; Age, age at initial operation; Interval, interval between initial and redo operation; Etiology, main etiology of aortic regurgitation at initial operation; TEE, transesophageal echocardiography; Dis-TTE, transthoracic echocardiography at the hospital dismissal.
unsuccessful and immediate conversion to prosthetic valve replacement was undertaken. There were few such patients during the study interval, and there were no operative deaths related to an initial attempt at aortic valve repair.

The late results of aortic valve repair that have been presented should be interpreted in the context of expected results from prosthetic valve replacement. Certainly, replacement with a mechanical or a biological prosthesis would be expected to have a lower rate of reoperation during the first 5 to 7 years postoperatively, but this durability comes at the expense of valve-related complications, which, for mechanical valves, occur at a rate of approximately 5% per patient-year.21 In this series, the linearized rate of thromboembolism, anticoagulant-related bleeding, and infective endocarditis combined was 1.0% per patient-year.

Thromboembolism and anticoagulant-related bleeding after aortic valve replacement are lower with biological valves than with mechanical valves, but structural valve deterioration is predictable with heterografts, and rates of valve failure at 10 years postoperatively are 13% to 30% for patients in their fifth decade of life and 18% to 25% for patients in their sixth decade.22-24 Although our follow-up is not sufficiently long to allow formal comparison, durability of valve repair appears similar to that of porcine heterografts in younger patients and offers some hope of function beyond 15 years. In the present series, the mean age of the patients was 55 years with a freedom from reoperation of 85% at 7 years; additional observation will be necessary for secure conclusions regarding durability in comparison to heterograft prostheses.

Also, it should be recognized that these results represent the learning curve for this procedure, and experience in selection of patients and operative methods might be expected to improve subsequent results. For example, early failure resulting from suture dehiscence at the repair site of the bicuspid valve has not occurred since 1997, and late breakdown at the repair site was the cause of recurrent valve leakage in only 1 of the 14 patients who had late aortic valve replacement for aortic valve regurgitation.

The important influence of the learning curve on late outcome is illustrated in Figure 5, which shows the cumulative risk of reoperation after mitral valve repair at our institution during 2 decades. Rates of late reoperation have been reduced by half in the latter portion of this experience. Indeed, the risk of late reoperation after aortic valve repair is very similar to the risk of late reoperation following mitral valve repair involving the anterior leaflet in the 1980s.25

Congenital bicuspid aortic valves are present in approximately 2% of the general population,26,27 and up to 63% of people with a bicuspid aortic valve will have no significant valve dysfunction with normal life expectancy.28 Valvular regurgitation is a frequent presentation of bicuspid aortic valve disease in young patients.26,27 These younger patients might be the best candidates for valve repair. Cosgrove and colleagues4,10,11 have reported good late durability of repair of bicuspid valves with freedom from reoperation estimates at 5 and 7 years of 87% and 84%, respectively, very similar to those in the present study (91% at 5 years).

In conclusion, aortic valve repair in patients with aortic valve regurgitation can be performed with very low mortality and morbidity. Intermediate-term follow-up suggests that valve-related complications are very low and risk of reoperation is acceptable, particularly when compared with the anticipated risk of structural valve deterioration of heterograft valves in young patients.

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assistance with data collection and analysis. Also, we thank Nobuhiko Handa, MD, who initiated this project and collection of the clinical data.

References


Discussion

Dr Lawrence H. Cohn (Boston, Mass). This is a very interesting article, very well presented by Dr Minakata, who is a member of one of the premier valve surgery groups in the United States, if not the world, the Mayo Clinic. They took a look at 15 years of data and accumulated 160 patients; that is about 10 to 12 patients per year. My first question to you is this. Is this rate of repair increasing, decreasing, or staying the same, and what might be the denominator per year, approximately, that this fraction represents?

Dr Minakata. During the same time range, approximately 4000 patients underwent first-time aortic valve replacement due to aortic valve disease. Of those, 1250 patients underwent aortic valve replacement due to severe aortic valve regurgitation. The repair rate is increasing for probably the last 5 years.

Dr Cohn. At the Brigham last year we probably did something in the range of 5 to 8 such operations, and I certainly agree that in any noncalcified valve in any form, whether it be bicuspid or tricuspid, aortic valve repair should certainly be considered, but what is not clear to me and maybe to you is your degree of comfort in the repair prior to closing the aorta?

Dr Minakata. If there is any significant aortic stenosis, we prefer not doing repair of the aortic valve. In terms of the degree of calcification of the valve, if we resect the calcified portion of the conjoining cusp and still have enough tissue to sew and plicate, I think we can still try the repair.

Dr Cohn. Another question is, how do you test for competence of your repair prior to closing the aorta?

Dr Minakata. Literally, there is no way to evaluate the repair appropriately before closing the aortic wall.

Dr Cohn. No way to do that? Would Dr Schaff like to comment on that maybe?

Dr Hartzell V. Schaff. Evaluation of the repair is more difficult with the aortic valve than with mitral valve repair because the aortic root is relaxed and it is not possible to inspect the valve under physiologic pressure. After repair of a bicuspid valve, you can displace the 2 commissures and check for good central appo-
sition of the cusps, but often you have to wait until the aorta is closed to assess valve competence.

Dr Cohn. My final question is, do your good results suggest to your team that in patients in whom you are doing mitral valve repair that oftentimes have moderate aortic regurgitation, should you be more, shall we say, “aggressive,” in repairing those aortic valves along with the mitral valves?

Dr Minakata. For the last question, interestingly, if we exclude the patients who had both mitral and aortic valve repair, the risk of reoperation at 5 years would be about 6% versus 11% in entire series of our paper. If patients have both diseases, mitral and aortic valve regurgitation, they might not be good candidates for this repair.

Dr. Robert A. Dion (Leiden, The Netherlands). I congratulate you for this magnificent paper, and I would like to ask you which technique you would prefer in the presence of a prolapse of 1 of the cusps of a tricuspid aortic valve. We favor the reinforcing of the free edge with a continuous suture of 7-0 polytetrafluoroethylene (Gore-Tex; W. L. Gore & Associates, Inc, Flagstaff, Ariz) above the triangular resection. What would you recommend for the monocusp failure?

Dr Minakata. There were only a couple of patients who had a triangular resection in the tricuspid aortic valve patients. We usually plicate using the commissural plication under each commissure of the prolapsing cusp so that we could decrease the length of the free edge and increase the coaptation area, and oftentimes we also do what we call a “Trusler stitch” to shorten the cusp. Those are the main techniques we usually use.

Dr Schaff. Let me add to that, Dr Dion. We have used primarily 3 methods: 1 would be a Trusler stitch to shorten the free edge of the cusp near the commissures; another would be a very limited triangular resection; and the third maneuver would be to support or resuspend the free edge of the cusp, as you describe, with a suture passed from outside the aorta inward and then along the free edge; we usually use 5-0 polytetrafluoroethylene for cusp resuspension.

Dr Christophe Acar (Paris, France). Congratulations, Dr Minakata. I wish to ask you a question concerning the identifica
tion of the mechanics of aortic regurgitation. In your article you mentioned that you had 40% of patients with annulus dilatation. We find it very difficult to identify, especially on the aortic valve. So could you tell us, how do you know that the mechanics of aortic regurgitation were precisely annulus dilatation rather than cusp retraction or rather than dilatation of the sinotubular junction, and did you evaluate this using transesophageal echocardiography or any other method?

Dr Minakata. Essentially we excluded the patients who had dilatation of the sinotubular junction or annuloaortic ectasia from this series. With patients who had tricuspid valve and annular dilatation, usually we felt that the main reason for regurgitation was spreading out the cusps toward the outside of the annulus. This is what we usually see in the elderly patients. The regurgitation was almost always central and due to essentially dilatation of the annulus.

Dr Schaff. I might add that, as Dr Minakata mentioned, this series does not include patients with dilatation of the sinotubular junction. Those are easily repaired by inserting a tube graft or narrowing that segment of the aorta. For patients with tricuspid valves and annular dilatation, we don’t have specific formulas to determine the extent to which the annulus can be reduced. In practice one often has to place those plication sutures and see if you then have good central apposition of the three cusps. We don’t use any formulas or specific guidelines of measurement.

Dr A. Sampath Kumar (New Delhi, India). Were there any cases in which the aortic repair failed on the table and how did you detect and treat this?

Dr Minakata. We encountered 2 early re-repairs. Both patients had bicuspid valves that were found to have dehiscence in the plication sutures on the conjoining cusps. We always try to make a smaller resection in the conjoining cusps with probably 30% of length of the height of the cusp so that we could avoid the tension of the suture lines. And we also had 2 patients who had to go back on cardiopulmonary bypass to have a better repair after initial repair, but all patients had satisfactory results of the additional repair or re-repair of the aortic valve.

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