Annuloplasty for Mitral Insufficiency

A Five to Six-Year Clinical and Hemodynamic Follow-Up

By Harold E. Aldridge, M.D., Irving H. Lipton, M.D., and Wilfred G. Bigelow, M.D.

Annuloplasty of an incompetent valve by plication of one or both commissures under direct vision was the only available surgical treatment for mitral insufficiency 5 to 6 years ago. At that time, suitable prosthetic valves for the mitral area were not readily available. The simplicity of annuloplasty presented an attractive surgical approach to the correction of mitral insufficiency provided that there was no undue rigidity or calcification of the valve leaflets or loss of valve tissue. While fluoroscopy was of help in excluding significant valve calcification, loss of valve tissue could only be accurately determined on inspection of the valve at operation. While the simplicity of this operation made this an attractive procedure, the long-term results determine whether annuloplasty still has a place in the surgical treatment of mitral insufficiency at a time when prosthetic valves are readily available.

Seventy-three annuloplasties have been done at the Toronto General Hospital. The results in the first 20 consecutive cases on follow-up for 1 to 2½ years were reported by Bigelow and associates.1 This early follow-up suggested that annuloplasty usually led to an acceptable correction of the insufficiency with symptomatic improvement in selected patients. It is now 5 to 6 years since these patients were operated upon. Their assessment at this time forms the basis of this report.

Methods

In the previous report1 preoperative and postoperative studies were presented on 11 patients with mitral annuloplasty. Two of the patients with good results were subsequently killed in automobile accidents and lost to follow-up.

The present report thus deals with nine patients, who were clinically assessed 5 to 6 years after surgery. Eight patients had a second postoperative cardiac catheterization study from 3 to 6 years after surgery, and one patient was studied at autopsy. The New York Heart Association classification was used to grade the clinical disability.2 Preoperative cardiac catheterization data were obtained by transthoracic left-heart catheterization using the Björk technique.3 The mitral insufficiency was assessed by indicator dye-dilution technique.4,5 The second postoperative hemodynamic studies were obtained by transseptal left heart catheterization6 combined with percutaneous retrograde aortic catheterization.7 Insufficiency at mitral, aortic, and tricuspid valves was assessed by indicator dye-dilution technique. Aortic and mitral insufficiency was also assessed by cineangiography.

Results

This 5 to 6 year follow-up of nine patients following mitral annuloplasty showed a significant return of mitral insufficiency in six patients, or two thirds, as might have been expected. Three, or one third, of the patients were still in grade I or II at the time of follow-up. Seven of nine patients were still alive. Two patients died in the fifth year following operation.

Functional Status

One to two years after operation, eight patients were improved to class I or II; at 3 to 4 years five of these patients remained improved in class I or II; at 5 to 6 years three patients were still improved in class I or II, four patients were in class III or IV, and two patients had died (fig. 1). Thus one third of the patients who were in class III or IV before operation were still improved 5 to 6 years after operation.
Cardiac Catheterization and Autopsy Data

(Table 1)

Left Atrial Pressure

The mean left atrial pressure was increased from 5 to 10 mm Hg above the preoperative level and ranged from 7 to 33 mm Hg in seven of eight patients catheterized after operation. In all patients except one, there had been a decrease in the mean left atrial pressure 1 to 2½ years after operation.¹

Left Ventricular End-Diastolic Pressure

The preoperative values ranged from 6 to 15 mm Hg being elevated above the normal (10 mm Hg) in five of the nine patients. Four of the five patients had shown a fall in the end-diastolic pressure at the first follow-up.³ Three patients that had shown no fall in the end-diastolic pressure at the earlier follow-up had further elevation above the preoperative level at 5 to 6 years.

Fate of Annuloplasty

The hemodynamic assessment and autopsy data showed that four of the nine patients had recurrence of severe, and two of moderate, mitral insufficiency. Three patients had developed severe mitral stenosis. Valve calcification was a complicating factor in one of these patients. Two of the three patients showing progression to stenosis had a mixed stenosis and insufficiency lesion preoperatively.

Associated Lesions at Other Valves

Three patients developed severe tricuspid insufficiency, and one severe tricuspid stenosis. The patient with severe tricuspid stenosis also developed severe aortic (and mitral) stenosis. Two patients developed moderate or severe aortic insufficiency while two had mild aortic insufficiency. In all, five of the nine patients had developed both aortic and tricuspid valve lesions (table 1).

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Figure 1

Clinical status of nine patients followed 5 to 6 years after mitral annuloplasty.
### Table 1

**Hemodynamic and Autopsy Data in Nine Patients Three to Six Years After Mitral Annuloplasty**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Time of catheterization or autopsy</th>
<th>Mean left atrial pressure (mm Hg)</th>
<th>Mitral end-diastolic gradient (mm Hg)</th>
<th>Left ventricular end-diastolic pressure (mm Hg)</th>
<th>Assessment of mitral insufficiency</th>
<th>Associated lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.T.</td>
<td>Preop</td>
<td>24</td>
<td>8</td>
<td>12</td>
<td>Moderate</td>
<td>MS, mild; mitral calcium, mild</td>
</tr>
<tr>
<td>49 F</td>
<td>5-6 yr postop</td>
<td>33</td>
<td>11</td>
<td>17</td>
<td>Trivial</td>
<td>AI, trivial; mitral calcium, marked; TI, severe</td>
</tr>
<tr>
<td>J.D.</td>
<td>Preop</td>
<td>10</td>
<td>6</td>
<td>6</td>
<td>Severe</td>
<td>AI, mild; TI, trivial</td>
</tr>
<tr>
<td>42 F</td>
<td>5-6 yr postop</td>
<td>18</td>
<td>3</td>
<td>10</td>
<td>Severe</td>
<td>AI, mild; TI, trivial</td>
</tr>
<tr>
<td>E.O.</td>
<td>Preop</td>
<td>19</td>
<td>0</td>
<td>10</td>
<td>Nil</td>
<td>AI, trivial; mitral calcium, mild</td>
</tr>
<tr>
<td>26 F</td>
<td>5-6 yr postop</td>
<td>23</td>
<td>17</td>
<td>8</td>
<td>Nil</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>D.R.</td>
<td>Preop</td>
<td>22</td>
<td>0</td>
<td>15</td>
<td>Severe</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>38 F</td>
<td>5-6 yr postop</td>
<td>27</td>
<td>0</td>
<td>17</td>
<td>Severe</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>M.C.</td>
<td>Preop</td>
<td>16</td>
<td>2</td>
<td>12</td>
<td>Severe</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>39 M</td>
<td>5-6 yr postop</td>
<td>24</td>
<td>4</td>
<td>19</td>
<td>Severe</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>D.L.</td>
<td>Preop</td>
<td>12</td>
<td>0</td>
<td>10</td>
<td>Severe</td>
<td>AI, moderate; mitral calcium, mild; TI, severe</td>
</tr>
<tr>
<td>22 F</td>
<td>3½ yr postop</td>
<td>7</td>
<td>0</td>
<td>4</td>
<td>Moderate</td>
<td>AI, severe; TI, severe</td>
</tr>
<tr>
<td>L.B.*</td>
<td>Preop</td>
<td>22</td>
<td>4</td>
<td>14</td>
<td>Severe</td>
<td>MS, mild</td>
</tr>
<tr>
<td>17 F</td>
<td>3 yr postop</td>
<td>32</td>
<td>4</td>
<td>14</td>
<td>Moderate</td>
<td>MS, severe; AS, moderate; TS, moderate</td>
</tr>
<tr>
<td>M.P.</td>
<td>Preop</td>
<td>17</td>
<td>3</td>
<td>6</td>
<td>Severe</td>
<td>MS, severe; AS, moderate; TS, moderate</td>
</tr>
<tr>
<td>F</td>
<td>3 yr postop</td>
<td>22</td>
<td>5</td>
<td>12</td>
<td>Severe</td>
<td>MS, severe; AS, moderate; TS, moderate</td>
</tr>
<tr>
<td>C.D.</td>
<td>Preop</td>
<td>19</td>
<td>7</td>
<td>12</td>
<td>Severe</td>
<td>MS, severe; AS, moderate; TS, moderate</td>
</tr>
<tr>
<td>F</td>
<td>Autopsy</td>
<td>5½ yr postop</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Now has Starr-Edwards prosthesis in mitral area.

Abbreviations: MS = mitral stenosis; TS = tricuspid stenosis; AS = aortic stenosis; TI = tricuspid insufficiency; AI = aortic insufficiency.
Late Complications

Subacute Bacterial Endocarditis

In two patients subacute endocarditis developed 2½ to 3 years after annuloplasty. This resulted in breakdown of the annuloplasty with recurrence of moderate insufficiency in one. The infection responded to antibiotic treatment in both.

Systemic Embolization

A cerebral embolus occurred in one patient while clinically well, in class I or II 4 years after operation. Presumably this was of the result of thrombus on the valve or in the left atrium. No residual neurological defect persisted.

Reactivation Syndrome

Two patients presented clinical and laboratory evidence of reactivation of the rheumatic process. This was only partially controlled in one patient with bed rest, long-term steroid and prophylactic penicillin therapy. This patient developed severe mitral, aortic, and tricuspid insufficiency and died 5½ years after operation (see “Late Deaths”). The second patient, a girl, 17 years old, developed recurrence of mitral insufficiency with progressive dilatation of the mitral valve ring. This necessitated further operative intervention and a Starr-Edward’s ball-valve prosthesis was inserted 3 years after the annuloplasty. She has been much improved since.

Late Deaths

Two late deaths both occurred 5½ years after operation. In one patient a myocardial factor appeared to be primarily involved with recurring reactivation syndrome, and the development of severe mitral, aortic, and tricuspid insufficiency. The other patient was well, in grade I or II until the onset of atrial fibrillation, following which rapid deterioration occurred and she died within 3 weeks. At autopsy, severe mitral, aortic, and tricuspid stenosis was demonstrated.

Discussion

This follow-up study shows that mitral annuloplasty can provide long-term symptomat-
ic improvement in seriously disabled patients with mitral insufficiency. Half of the patients maintained improvement for 3 to 4 years, and one third of the patients were still improved 5 to 6 years after operation. Björck and associates have reported improvement in 15 of 19 patients following annuloplasty: seven of these patients have been observed for more than 3 years. Ellis and associates also reported maintained improvement in one half of the patients following annuloplasty observed for an average of 2½ years.

The hemodynamic data shows recurrence of at least moderate insufficiency or progression to stenosis in all of our patients, although one third maintained their clinical improvement. This apparent discrepancy between the symptomatic and hemodynamic findings has been noted before. It points out also the importance of hemodynamic data in the objective assessment of the results of cardiac surgery and suggests that complete hemodynamic correction, although desirable, may not be essential in order to provide symptomatic improvement for a number of years.

The place of active carditis in patients with mitral insufficiency in assessing the significance of the results of this study is difficult to delineate. None of our patients had evidence in the history, clinical findings, or any evidence from the usual laboratory tests of active carditis in the immediate months preceding operation. However, two of the patients, who developed a reactivation syndrome within 1 year of operation with subsequent early deterioration, were suspected on history of having had active carditis within a period of 2 to 3 years preceding operation.

The fact that the mitral lesion in seven of nine patients progressed and that in a number of patients lesions at the aortic or tricuspid valves or both either progressed also or developed de novo must reflect the presence of a continuing subclinical process, which may be clinically suspected, but is difficult to prove by objective evidence.

The incidence of subacute bacterial endocarditis was two in nine patients. There may well be a relationship between the presence

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of foreign body material (Teflon pledgets) and vulnerability of the valve to infection. The infection resulted in the breakdown of repair in one patient, who is still improved in class I or II.

Although this operation was available to only a small percentage of patients with mitral insufficiency, in all cases reported herein the operations were performed before the mitral Starr-Edwards valve or other valve replacements were available. Studies at this center\(^\text{11}\) and other centers\(^\text{12-16}\) of patients in whom the mitral valve has been replaced by the Starr-Edwards ball valve have shown satisfactory hemodynamic correction. However, the incidence of late complications associated with the use of this prosthesis has been disturbing. These complications include systemic embolism necessitating lifetime anticoagulation with its associated morbidity and mortality, sudden death due to ventricular arrhythmia, an increased incidence of bacterial endocarditis, and residual regurgitation between prosthetic ring and mitral annulus, sometimes murmurless, leading to failure of patients to improve and necessitating reoperation.

The late problems in mitral valve replacement, the number of survivors of 5 to 6 years following annuloplasty and the percentage of patients with maintained symptomatic improvement suggest that there is still a place for annuloplasty in the correction of mitral insufficiency. A final assessment of the value of mitral annuloplasty cannot be made until a 5 to 6 year study is available in a comparable group of patients in whom the mitral insufficiency was corrected by prosthetic valve replacement.

**Summary**

Nine patients were followed from 5 to 6 years after mitral annuloplasty. Seven of the nine patients were alive at follow-up, and three of nine, one third of the patients, were still improved in class I or II (New York Heart Association classification). All patients were severely disabled in class III or IV before operation.

Cardiac catheterization and autopsy data at 3 to 6 years showed some progression in the pathology of the mitral valve in all patients including the third who maintained symptomatic improvement.

Severe lesions at tricuspid or aortic valve, or both, had developed in four of the nine patients.

Annuloplasty probably still has a place in the surgical treatment of selected patients with mitral insufficiency.

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**References**


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