Use of Aortic Valve Homografts for Aortic Valve Replacement

By ROBERT B. WALLACE, M.D., EMILIO R. GIULIANI, M.D., and JACK L. TITUS, M.D.

SUMMARY

One hundred sixty-nine patients underwent replacement of the aortic valve with an aortic valve homograft; of these patients 17 had concomitant commissurotomy or annuloplasty for an associated mitral valve lesion. Homografts were prepared by sterilization with beta-propiolactone solution and stored in Hanks' solution, or were prepared by sterilization with irradiation and stored at —70 C. Operative mortality was 5.3%. Thirty (19%) of the 160 surviving patients had an aortic diastolic murmur at the time of hospital dismissal, and five of these patients had hemodynamically significant homograft incompetence.

For 156 patients, follow-up was longer than 6 months; among the 147 patients who survived operation, there were 12 late deaths. Eighty-four per cent (132) of these 156 patients were alive and well 6 to 54 months after operation. Six patients have required reoperation for valve incompetence. An aortic diastolic murmur was present in 43% of patients. Five patients had significant valve incompetence; four of these patients are markedly improved over their preoperative status. Thromboembolism has been a rare complication despite the fact that anticoagulant therapy was not used. Late calcification of the homograft valve has been noted in only one patient.

Additional Indexing Words:
Aortic valve Homograft preparation

could function in this position for several years. The use of aortic valve homografts for aortic valve replacement was begun at the Mayo Clinic in 1965. Since that time, the early and late results of this experience continue to support the use of this type of valve replacement in many patients requiring aortic valve replacement.

Methods

Patients

Between May 1965 and January 1970, 169 patients (125 males and 44 females) have had replacement of the aortic valve with an aortic valve homograft at the Mayo Clinic*; their ages ranged from 12 to 69 years, with the majority of patients in the fifth and sixth decades of life (table 1). Several patients who had associated

*Many of the operations done early in this series were performed by Dr. John W. Kirklin.
congenital intracardiac defects repaired at the time of aortic valve replacement are excluded from this series.

All patients had symptomatic aortic valve disease. In 92 patients the predominant lesion was aortic stenosis; two patients, both less than 30 years old, had congenital valve stenosis, and one patient had undergone a previous attempt at surgical relief of the stenosis. In 39 patients, aortic valve incompetence was the predominant lesion; six had undergone a previous valvotomy that had resulted in significant valve incompetence, and eight others had a history of subacute bacterial endocarditis. Eighteen patients had combined aortic stenosis and valve incompetence of equal severities. Seventeen patients had an associated mitral valve defect requiring commissurotomy or valvuloplasty at the time of aortic valve replacement. In three patients, an aortic valve homograft was used to replace a prosthetic valve; two of these patients had infection of a ball valve prosthesis resulting in incompetence, and one patient had incompetence of a prosthetic cusp.

Aortic valve homografts were not used in patients requiring prosthetic valve replacement of either the mitral or the tricuspid valve or in patients with aortic dissection and aortic valve incompetence. An enlarged aortic root was not in itself a contraindication to the use of a homograft.

In 57 patients in this series, an enlarged aortic root was tailored to conform to the size of the graft. Because the tailoring procedure was used only with enlarged aortic roots, it did not produce stenosis of the aortic root.

Where adequate coronary perfusion was not possible, a homograft valve was not used because it requires a longer period of bypass for insertion than does a prosthetic valve.

**Preparation of Valves**

The homografts used in the first 92 patients of this series were sterilized with beta-propiolactone solution and stored in Hanks' solution. On the basis of laboratory work by Malm and associates\(^6\) which indicated that sterilization by irradiation better preserved the aortic wall strength and valve architecture of the homograft, this technique was adopted and used in the last 77 patients in this series. It is the current technique we use for homograft sterilization.

Aortic valve homografts were obtained at autopsy, in a nonsterile manner, within 18 hours after death. Valves were not used from cadavers in which there was gross evidence of disease affecting the ascending aorta or the aortic or mitral valve. We also excluded valves from patients with known connective tissue disease. Septicemia was not a contraindication to valve use if the tissues were normal.

The valve was excised with a portion of the ascending aorta, the upper portion of the left ventricle, and part of the anterior leaflet of the mitral valve. Excess muscular and fibroareolar tissues were trimmed from the external surface of the graft. The graft was rinsed in water to remove all blood, and the diameter of the valve was measured with a calibrated obturator. The graft was then sealed in a plastic bag, and that bag sealed in two others. Glass beads were placed in the outer bag, and an appropriate identifying tag was inserted. Because the glass beads turn black when irradiated, they serve to indicate that the graft has been treated; they do not, however, reflect quantitatively the dose of radiation energy.

The bags were stored in a carbon dioxide freezer at \(-70\) C. Later they were sterilized while still frozen with a 6 Mev electron beam extracted from the linear accelerator which is routinely used for supervoltage X-ray therapy in our institution; this provided an absorbed dose of ionizing radiation of approximately 2.5 megarads in the 25 min of exposure. The effectiveness of the sterilization procedure has been verified by irradiating numerous samples which had been deliberately heavily contaminated with a variety of microorganisms.

After irradiation, the valves were catalogued and stored at \(-70\) C until they were used. It is not known how long homografts might be stored, but valves have been discarded arbitrarily after a 12-month period.

**Operative Technique**

A median sternotomy incision is used, and the right atrial appendage and ascending aorta are cannulated for extracorporeal circulation. The left ventricle is vented by a vent inserted through a stab wound. The aorta is cross-clamped, and an oblique aortotomy incision is made extending down into the sinus of the noncoronary cusp. The

### Table 1

**Age Distribution of Patients Undergoing Homograft Replacement of Aortic Valve**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-19</td>
<td>11</td>
</tr>
<tr>
<td>20-29</td>
<td>11</td>
</tr>
<tr>
<td>30-39</td>
<td>24</td>
</tr>
<tr>
<td>40-49</td>
<td>41</td>
</tr>
<tr>
<td>50-59</td>
<td>54</td>
</tr>
<tr>
<td>60-69</td>
<td>28</td>
</tr>
<tr>
<td>Total</td>
<td>169</td>
</tr>
</tbody>
</table>

*From Circulation, Volume XLIII, March 1971*
coronary arteries are cannulated and perfused throughout the procedure.

The aortic valve is excised, and calcium deposits are debrided completely. The internal diameter of the valve ring is measured with a graduated obturator, and a valve of the same size is selected from the valve bank.

If it is necessary to decrease the diameter of an enlarged aortic root, tailoring is accomplished by excising a wedge-shaped segment of the aortic root in the area of the noncoronary sinus. The defect thus created is closed with interrupted fine silk sutures. Minor decreases (2 or 3 mm) in the diameter of the aortic root can be accomplished by sutures placed at the commissures.

The valve is removed from the three plastic bags in turn in a sterile manner; as each bag is removed, it is soaked in 95% alcohol colored with thimerosal (Merthiolate), which allows detection of any defect in the plastic bag. If a defect is noted, the valve is discarded. The valve is then thawed by immersion in saline at 37 C, and

**Figure 1**
Trimmed inverted valve, which has been turned inside out, is mounted on valve holder, and inferior suture line is placed.

trimmed of all excess tissue in such a manner as to provide a straight-line sewing margin beneath the valve leaflets. The aorta is trimmed above the leaflets to leave a 1-mm scalloped margin of aorta which conforms to the attachment of the valve leaflets.

Early in our experience we used the technique of Barratt-Boyce for inserting the valve. This technique has been modified, and currently a valve holder is used (fig. 1). The valve is inverted, turned inside out, and placed on the valve holder. Interrupted sutures for the inferior suture line are then placed through the valve and through the recipient root on a horizontal plane beneath the margins of excision of the excised valve. The valve is removed from the valve holder and lowered into the left ventricle. The sutures are tied and the valve is inverted again to return it to its normal position.

The superior suture line is placed by using three double-arm sutures in a continuous technique (fig. 2). Each suture is started at the base of a coronary sinus, and each end is continued up the commissures and tied to the adjacent suture at the apex of the commissures.

It is of great importance that the commissures be adequately suspended and properly spaced (fig. 3). It is important that the commissures be spaced in relation to the homograft rather than to the line of excision of the diseased valve. This is particularly true when the root has required tailoring, since tailoring alters the distance between the commissures of the host root. The

**Figure 2**
After sutures of the inferior suture line are tied, valve is returned to its normal position and continuous sutures are placed for superior suture line.
At completion of insertion of valve, commissures of graft should be equidistant.

Aortotomy is closed, and the operation is completed in the usual manner.

Results

Early Results

Mortality

There were nine hospital deaths in this series of 169 patients, for an operative mortality of 5.3% (table 2). Three deaths were directly related to hemorrhage, one at the time of operation and two in the early postoperative period. Three patients died as a result of myocardial infarction in the early postoperative period. One patient with coronary artery disease and systemic hypertension died on the second postoperative day from ventricular arrhythmia. One patient died from a massive cerebral embolus from an Aspergillus endocarditis of the graft; postmortem examination revealed an Aspergillus infection of a preoperative pulmonary infarction. One patient, in whom there was dissection of the aorta from a femoral artery cannulation site, died from massive infarction of the intestine.

Valve Incompetence

Of the 160 patients surviving operation, 30 (19%) had an aortic diastolic murmur at the time of dismissal from the hospital (table 3). Five patients (3%) had hemodynamically significant incompetence of the homograft valve as evidenced by an aortic diastolic murmur, peripheral signs of aortic valve incompetence, and a systolic blood pressure greater than two times the diastolic pressure. Of the 30 patients with a murmur, 22 had aortic valve incompetence as the original lesion; eight of the 22 had had aortic root tailoring, and 10 of the 22 were less than 25 years of age and had a noncalcified aortic valve. Three patients had had previous aortic valve surgery.

Late Results

Mortality

There were 12 late deaths (7.5%) among the 160 patients surviving operation, occurring 2

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Table 2

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Lesion</th>
<th>Postoperative day</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>37</td>
<td>AS; AI</td>
<td>2</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>58</td>
<td>AS</td>
<td>1</td>
<td>Bleeding</td>
</tr>
<tr>
<td>50</td>
<td>AS</td>
<td>3</td>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>38</td>
<td>AS</td>
<td>*</td>
<td>Bleeding</td>
</tr>
<tr>
<td>46</td>
<td>AI; hypertension</td>
<td>2</td>
<td>Ventricular fibrillation</td>
</tr>
<tr>
<td>56</td>
<td>AI; MS</td>
<td>17</td>
<td>Cerebral infarct</td>
</tr>
<tr>
<td>57</td>
<td>AI; MI</td>
<td>3</td>
<td>Aortic dissection; intestinal infarction</td>
</tr>
<tr>
<td>65</td>
<td>AS</td>
<td>4</td>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>46</td>
<td>AS</td>
<td>4</td>
<td>Myocardial infarction</td>
</tr>
</tbody>
</table>

Abbreviations: AS = aortic stenosis; AI = aortic incompetence; MS = mitral stenosis; MI = mitral incompetence.

*In operating room.
to 43 months after insertion of the homograft valve (tables 4 and 5).

Of the six patients who died of a myocardial infarction, four patients (who died, 2, 4, 5, and 7 months, respectively, after operation) had no clinical evidence of valve dysfunction, and postmortem examination in three revealed a pliable and competent homograft valve. The two other patients (who died 8 and 9 months, respectively, after operation) had clinical evidence of aortic valve incompetence, and, in both, postmortem examination showed a recent myocardial infarction. In one of these patients there was a tear in one of the cusps of the homograft; in the other there was prolapse of a cusp.

One patient died of multiple myeloma 26 months postoperatively. Postmortem examination was not performed, but there was no evidence of valve dysfunction prior to death.

Two patients died of bacterial endocarditis and homograft incompetence. In both cases the organisms were streptococci of the viridans group. One of these patients manifested aortic valve incompetence 23 months postoperatively but refused reoperation and died 15 months later. The other patient was asymptomatic for 36 months. He developed bacterial endocarditis after dental surgery and died 4 months later.

Another patient died 43 months postoperatively from valve incompetence and congestive heart failure. After having had an excellent result for 12 months, she developed valve incompetence with a clinical picture suggesting subacute bacterial endocarditis,

### Table 4

**Late Results After Homograft Replacement of Aortic Valve**

<table>
<thead>
<tr>
<th>Time of operation (no.)</th>
<th>Patients (no.)</th>
<th>Deaths (no.)</th>
<th>Death Operative</th>
<th>Death Late</th>
<th>Reoperation of patients (no.)</th>
<th>Diastolic murmur</th>
<th>Significant valve incompetence (no.)</th>
<th>Asymptomatic or improved</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1969 (Jan.-June)</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>4</td>
<td>50</td>
<td>0</td>
<td>8</td>
<td>90</td>
</tr>
<tr>
<td>1968</td>
<td>38</td>
<td>0</td>
<td>3</td>
<td>1</td>
<td>12</td>
<td>34</td>
<td>1</td>
<td>34</td>
<td>89</td>
</tr>
<tr>
<td>1967</td>
<td>43</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>18</td>
<td>46</td>
<td>2</td>
<td>37</td>
<td>86</td>
</tr>
<tr>
<td>1966</td>
<td>43</td>
<td>2</td>
<td>6</td>
<td>2</td>
<td>13</td>
<td>37</td>
<td>2</td>
<td>34</td>
<td>79</td>
</tr>
<tr>
<td>1965</td>
<td>23</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>11</td>
<td>58</td>
<td>0</td>
<td>19</td>
<td>83</td>
</tr>
<tr>
<td>Total</td>
<td>156</td>
<td>9</td>
<td>12</td>
<td>6</td>
<td>58</td>
<td>43</td>
<td>5*</td>
<td>132†</td>
<td>84</td>
</tr>
</tbody>
</table>

*Based on number of living patients.
†Includes one patient with significant valve incompetence and unimproved.
‡Includes six patients reoperated on; excludes two patients with neurologic deficits.
Table 5

Late Deaths After Homograft Replacement of Aortic Valve

<table>
<thead>
<tr>
<th>Postoperative interval (month)</th>
<th>Valve incompetence present*</th>
<th>Cause of death</th>
<th>Condition of homograft valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>0</td>
<td>MI</td>
<td>Excellent</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>Pneumonia (lupus)</td>
<td>Thickened</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>MI</td>
<td>Excellent</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>MI</td>
<td>Excellent</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>MI</td>
<td>Excellent</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td>MI</td>
<td>Cusp prolapse</td>
</tr>
<tr>
<td>9</td>
<td>+</td>
<td>MI</td>
<td>Cusp tear</td>
</tr>
<tr>
<td>26</td>
<td>0</td>
<td>Multiple myeloma</td>
<td>-</td>
</tr>
<tr>
<td>27</td>
<td>0</td>
<td>Unexplained</td>
<td>Excellent</td>
</tr>
<tr>
<td>38</td>
<td>+</td>
<td>SBE</td>
<td>SBE</td>
</tr>
<tr>
<td>40</td>
<td>+</td>
<td>SBE</td>
<td>SBE</td>
</tr>
<tr>
<td>43</td>
<td>+</td>
<td>CHF</td>
<td>-</td>
</tr>
</tbody>
</table>

Abbreviations: MI = myocardial infarction; SBE = subacute bacterial endocarditis; CHF = congestive heart failure.

*Clinical assessment prior to death.

although this was not proved bacteriologically. One patient with lupus erythematosus died of bronchopneumonia 4 months postoperatively; at autopsy the leaflets of the homograft were focally thickened by vegetations of the Libman-Sack type. Another patient died from an unexplained cause 27 months postoperatively; the valve leaflets were pliable and competent at postmortem examination.

Valve Incompetence

One hundred eleven patients who did not have an aortic diastolic murmur at the time of hospital dismissal were followed for 7 to 54 months. In 41 patients (37%), an aortic diastolic murmur developed. Two of these patients died; both had significant valve incompetence secondary to bacterial endocarditis. Two other patients required reoperation for valve incompetence. None of the remaining 37 patients who developed diastolic murmur during the follow-up period had hemodynamically significant valve incompetence, and all were asymptomatic or improved over their preoperative status.

Twenty-eight patients who had a diastolic murmur at the time of hospital dismissal were followed for 6 to 51 months. Four of these patients have required reoperation for valve incompetence, at 9, 11, 15, and 20 months, respectively, after the initial operation. There were three late deaths, occurring at 8, 9, and 40 months postoperatively. Of the remaining 21 patients, all are asymptomatic or improved

Table 6

Patients Reoperated on for Valve Incompetence

<table>
<thead>
<tr>
<th>Interval after first operation (month)</th>
<th>Valve preparation*</th>
<th>Early murmur (grade†)</th>
<th>Status of valve</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>X-ray, frozen</td>
<td>1</td>
<td>Ruptured cusp</td>
</tr>
<tr>
<td>11</td>
<td>Chem., Hanks’ sol.</td>
<td>0</td>
<td>Central leak</td>
</tr>
<tr>
<td>11</td>
<td>Chem., Hanks’ sol.</td>
<td>3</td>
<td>Central leak</td>
</tr>
<tr>
<td>15</td>
<td>Chem., Hanks’ sol.</td>
<td>2</td>
<td>Central leak</td>
</tr>
<tr>
<td>20</td>
<td>X-ray, frozen</td>
<td>3</td>
<td>Perforated cusp</td>
</tr>
<tr>
<td>26</td>
<td>Chem., Hanks’ sol.</td>
<td>0</td>
<td>Ruptured cusp</td>
</tr>
</tbody>
</table>

*Abbreviations: X-ray, frozen = sterilization by irradiation and preservation by freezing; Chem., Hanks’ sol. = beta-propiolactone sterilization and preservation in Hanks’ solution.
†Graded on the basis of 1 to 6.
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over their preoperative status except for one patient who is unimproved and who thus far has declined reoperation. Five of these patients have hemodynamically significant valve incompetence.

Reoperation

Six (3.7%) of the 160 patients surviving operation required reoperation for homograft incompetence 9 to 26 months after the initial operation (table 6). Four of these six patients had had an aortic diastolic murmur at the time of hospital dismissal. All patients had evidence of significant aortic valve incompetence within 6 months of the initial operation, except one patient in whom it developed, secondary to cusp rupture, at 14 months. In no patient was the homograft valve incompetence due to a paravalvular leak. Three patients had a central leak producing incompetence, and in each instance the valve preparation had been by the beta-propiolactone technique. Two of these three patients had evidence of homograft valve incompetence early postoperatively.

In two patients, incompetence was secondary to cusp rupture; one of the valves had been prepared by the beta-propiolactone technique and the other by freezing and irradiation. One of these patients had mild aortic valve incompetence early which progressed rapidly; he required reoperation 9 months after his initial operation. The other patient developed aortic incompetence 14 months postoperatively; it progressed, and he required reoperation 26 months postoperatively.

One patient who had replacement of an infected ball valve prosthesis with a homograft had valve incompetence early postoperatively. He required replacement of his homograft 20 months after insertion, and a perforation of a cusp was found.

A ball valve prosthesis was used for replacement of the homograft in five patients and a homograft was used in one patient. All patients survived reoperation.

Thromboembolism

None of the patients in this series was placed on anticoagulant therapy postoperatively. Four patients (2.5%) who survived operation had definite or probable embolic complications. One patient with a severely calcified valve had a cerebral embolus 2 days postoperatively resulting in hemiparesis. Another patient with atrial fibrillation developed hemiparesis 1 month postoperatively. Two other patients had transient neurologic deficits thought to be secondary to embolism; both of these patients had had concomitant mitral valve surgery.

Bacterial Endocarditis

Bacterial endocarditis was responsible for two late deaths, at 38 and 40 months postoperatively. In both instances, streptococci of the viridans group was cultured. A third patient developed aortic valve incompetence 18 months postoperatively, and a clinical diagnosis of subacute bacterial endocarditis was made; however, this was not proved bacteriologically. This patient died 52 months postoperatively from congestive heart failure, having refused reoperation. In one patient, in whom an infected ball valve prosthesis was replaced with a homograft that subsequently required replacement because of a perforated cusp, the perforation was suspected to be secondary to infection of the valve.

Calcification of Homografts

Calcification of the homograft valve was looked for roentgenographically at the time of follow-up and was noted in only one patient 31 months postoperatively; it was minimal, and the patient was asymptomatic without evidence of valve dysfunction. Calcification of valve leaflets was not present in any of the patients who required reoperation or in any of the valves examined post mortem.

Symptoms

There were 156 patients who had been operated on at least 6 months to 54 months prior to the time of this report (table 4). One hundred thirty-two (84%) were alive and asymptomatic or markedly improved over their preoperative status; six of these patients have required reoperation. Fifty-eight (43%) of the patients
had an aortic diastolic murmur. However, there were only five patients who had significant valve incompetence, and four of these patients were asymptomatic or improved. One patient was unimproved. Two patients had no cardiac symptoms but were considered to be disabled because of neurologic deficits secondary to embolic episodes.

Twenty-three patients were operated on at least 4 years ago. Nineteen (83%) of these patients are alive and well 48 to 54 months postoperatively. Eleven of them had an aortic diastolic murmur, but none of these patients has significant homograft valve incompetence.

**Discussion**

Although the ball valve prosthesis is an effective replacement for the aortic valve, certain disadvantages are associated with its use. Some of these disadvantages are overcome by the use of an aortic valve homograft.

Experience has shown that an aortic valve homograft can be inserted with a low operative mortality, one similar to that of any other type of aortic valve replacement. The operative mortality in this series of patients with isolated aortic valve disease was 5%, with only one death in the last 69 cases.

The operative technique has become standardized. The bypass time required for the insertion of an aortic valve homograft is 60 to 75 min. This is approximately 15 to 20 min longer than that required for the insertion of a prosthetic valve. For this reason we have not used a homograft in a few cases where advanced coronary artery disease precluded the use of coronary artery perfusion.

An enlarged aortic root can be tailored to conform to the size of available grafts, as was done in 57 (34%) of the patients in this series. However, in certain cases of aortic valve incompetence—those, for instance, associated with aortic dissection, or those with markedly dilated roots in which the incompetence is secondary to disease of the aortic root as in Marfan's syndrome—a homograft is not used. In such situations, a ball valve prosthesis is used because the rigid sewing ring prevents recurrent dilatation of the aortic root. Aortic valve homografts are not used when a prosthetic valve is used to replace the mitral or tricuspid valve; a primary advantage of the homograft is lost in that these patients require anticoagulant therapy postoperatively.

There may be advantages to using a fresh homograft in preference to a preserved graft. These advantages would, however, appear to be offset by the problem of procurement of fresh valves and the difficulty in maintaining a valve bank with a variety of sizes.

Although the data from this series are insufficient to permit comparison of those homografts sterilized in beta-propiolactone and stored in Hanks' solution with those sterilized by irradiation and preserved by freezing, experimental work suggests that the latter technique is preferable. In the three patients who required reoperation because of a central valve incompetence without disruption of the cusps, the valves used had been sterilized in beta-propiolactone; it is possible that shrinkage of leaflet tissue, reported to occur with this preparation, may have been a factor in allowing this incompetence to occur. Valves preserved by freezing have a better consistency and are easier to work with than are valves preserved in solution.

Five of the six patients who required reoperation had evidence of significant valve incompetence within 6 months of the initial operation. This suggests that the technique of insertion, improper sizing of the valve, and insufficiency of leaflet tissue surface might be more important factors in valve dysfunction than degeneration of the leaflet tissue.

The incidence of early diastolic murmurs had decreased to less than 10% in the last 50 patients in the series, as compared with an overall incidence of 19%. This decrease in incidence of early diastolic murmurs may be a result of improved technique with increased experience, although here, too, it may be significant that valves preserved by freezing were used in the last half of the series whereas valves preserved in solution were used in the first half. It was unusual for a patient to develop significant valve incompetence if there was no diastolic murmur at the time of

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hospital dismissal. Only two of 111 patients without a diastolic murmur at the time of hospital dismissal and followed for at least 6 months developed significant valve incompetence requiring reoperation.

A major advantage of a homograft valve is that anticoagulant therapy is not required postoperatively, and the incidence of thromboembolic complications is minimal. Four patients in this series had probable or definite embolic episodes. In one patient, who had a cerebral embolus immediately postoperatively, the embolus was thought to be a calcific fragment. The three other patients had had concomitant mitral valve surgery or had atrial fibrillation.

It was initially thought that bacterial endocarditis would be an insignificant problem with homograft valves; this, however, has not been our experience. Two deaths were a direct result of bacterial infection of the valve, and one other patient died as a result of fungal infection of the valve. Two other patients were suspected of having subacute bacterial endocarditis although this was not proved bacteriologically. It is recommended that prophylactic antibiotic therapy be used during periods of increased susceptibility to bacteremia.

There was one unexplained death in the 12 patients who died after hospital dismissal. Of the five patients who had incompetence of the homograft that was thought to be a contributing cause of death, three could have been reoperated on; however, they refused reoperation. The other two patients had bacterial endocarditis of the valves, and probably should have been operated on earlier in the course of their illness.

Eighty-four per cent of the patients who have been followed for 6 to 54 months are asymptomatic or markedly improved over their preoperative status, although six have required reoperation. It is of interest that, of the 23 patients followed for more than 48 months, 19 are alive and well (one has required reoperation). In none of these patients is there evidence of significant valve incompetence. Of the four deaths, two were operative deaths and two were late deaths. One of these late deaths was caused by bacterial endocarditis and the other by a myocardial infarction.

This experience supports the continued use of aortic valve homografts in most patients requiring replacement of the aortic valve. Long-term results are comparable to those of prosthetic valve replacement, and homograft use has a distinct advantage in that anticoagulant therapy is not required postoperatively.

References

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