Results of triple valve replacement in 91 patients: perioperative mortality and long-term follow-up

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ABSTRACT Between 1961 and 1984, 91 patients underwent simultaneous triple valve replacement at the Mayo Clinic. Of the 273 prosthetic valves used, 77% were Starr-Edwards. Perioperative (30 day) mortality was 24% to 27% between 1962 and 1974 and 7% between 1975 and 1983 (p = .17). In patients with NYHA class IV symptoms, perioperative mortality was 44%, and in those with milder symptoms, it was 8% (p < .0001). The median follow-up was 7.5 years (range, 6 weeks to 20 years). Cumulative survival, which was calculated taking into consideration perioperative mortality, was 64% at 1 year, 55% at 5 years, 40% at 10 years, and 25% at 15 years. Multivariate analysis identified preoperative functional class and age as predictors of late survival. Among causes of late mortality were sudden death in 32.5%, congestive heart failure in 15%, thromboembolism in 12.5%, prosthetic valve dysfunction in 7.5%, and infective endocarditis in 5%. Late complications included systemic emboli in 42% (embolic rate, 12.3 events per 100 patient-years), bleeding in 22%, myocardial infarction in 16%, and infective endocarditis in 6%. Eight patients required reoperation for prosthetic valve dysfunction, and 12 patients had permanent pacemakers. Of the 29 patients still alive, 79% are in NYHA class I or II. In summary, perioperative mortality after triple valve replacement appears to be declining; long-term survival in 30 day survivors is similar to that after single valve replacement and excellent symptomatic improvement can be obtained, although morbidity is high.


OVER the past 10 years, reports from many centers have defined the early and late results of cardiac valve replacement for most categories of patients.1-9 The outcome of triple valve replacement (aortic, mitral, and tricuspid valve replacement) is less well defined, primarily because of the relatively small numbers of patients who have undergone the operation. With the trend toward conservative tricuspid valve surgery coupled with the development of more predictable methods of valve repair and the declining incidence of rheumatic heart disease in the United States, triple valve replacement is less frequently required than it was previously. Nonetheless, rheumatic heart disease with involvement of multiple valves remains a serious problem in many countries.10 Accordingly, we report our experience in 91 consecutive patients with rheumatic heart disease who underwent simultaneous triple valve replacement at the Mayo Clinic. This is the largest reported series and is particularly notable for the long follow-up period, which spanned 20 years (median duration of follow-up, 7.5 years in perioperative survivors). During this period, major changes in the preoperative, intraoperative, and postoperative practice of valve surgery have occurred. Particular attention has been focused on factors influencing operative risk and late survival, on the causes of late death and late complications, and on the accumulated knowledge derived from this prolonged learning experience. Our earlier experience with triple valve replacement has been reported.11,12

Material and methods

We reviewed the records of all 91 patients with rheumatic heart disease who underwent aortic, mitral, and tricuspid valve replacement during a single operation at the Mayo Clinic from 1962 through 1984. For perspective, this cohort represents only 1% of the total 8359 patients who underwent cardiac valve replacement during the same period. At our institution, the frequency of triple valve replacement has progressively declined: 78 operations were performed from 1962 through 1974, whereas only 14 triple valve replacements were done on 13
patients from 1975 through 1984. One patient had undergone triple-valve replacement in 1970, but all three cloth-covered Starr-Edwards valves were replaced in 1984 because of hemolysis.

Clinical data on the 91 patients are summarized in table 1. The severity of preoperative symptoms appears to have changed. Between 1962 and 1974, 50% of patients were in New York Heart Association (NYHA) class IV before surgery and 50% were in NYHA class II or III; of those undergoing operation from 1975 through 1984, only 8% were in NYHA class IV and 92% were in class II or III (p = .024). The predominant valve lesion was classified according to clinical findings, preoperative and intraoperative intracardiac pressure recordings, and operative inspection (table 2). Preoperatively 86% of patients were in atrial fibrillation, one patient was in paced rhythm, and one patient had episodic sinus arrest. The remaining patients (14%) were in sinus rhythm.

All patients had rheumatic heart disease, and 23 patients had 35 previous cardiac operations. Eight prior operations involved the aortic valve, 24 involved the mitral valve (21 mitral commissurotomies), and three were performed for tricuspid valve disease. Four patients had undergone previous valve replacement (including the patient with a prior triple valve replacement). During the interval of this study, a variety of prosthetic heart valves were used, and these are listed in table 3. Most (77%) were Starr-Edwards prostheses.

Surgical techniques. Methods of extracorporeal circulation and surgical techniques evolved throughout the two decades of this study. All operations were performed through a median sternotomy, and high-flow (2.0 to 2.4 liters/min/m²) hypothermic extracorporeal circulation was used. For the first 84 patients in this series, myocardial protection was achieved by continuous coronary artery perfusion; in the last seven patients, cold potassium cardioplegia was used during aortic cross-clamping. Anticoagulation with sodium warfarin was begun in all patients as soon as possible after operation, generally on the second or third postoperative day.

Follow-up. Current information on all patients was obtained by questionnaires to both patients and referring physicians, by telephone contact, or, in patients who had been seen before the end of the present review, by Mayo Clinic records. If possible, the cause of death was ascertained from copies of death certificates or from information supplied by the patient’s physician or family.

The diagnosis of thromboembolism was made by a history of stroke, transient cerebrovascular ischemic attacks, or myocardial infarction in patients younger than 45 years or in those with documented normal coronary arteries. Splenic, renal, and pulmonary emboli were diagnosed in a few patients in whom adequate documentation was present, including those in whom a diagnosis was made at autopsy. All patient contacts included specific questions related to possible thromboembolic episodes.

### TABLE 1

<table>
<thead>
<tr>
<th>Preoperative characteristics of patients</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>91</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>23</td>
<td>25</td>
</tr>
<tr>
<td>Female</td>
<td>68</td>
<td>75</td>
</tr>
<tr>
<td>NYHA class</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>III</td>
<td>47</td>
<td>52</td>
</tr>
<tr>
<td>IV</td>
<td>41</td>
<td>45</td>
</tr>
</tbody>
</table>

Mean age of the patients was 48 years, range 19 to 68.

The amount of data available regarding the adequacy of anticoagulation was insufficient for further analysis.

#### Statistical analysis

**Perioperative mortality.** Univariate analysis of discrete variables was performed by means of a chi-square test or, when needed, Fisher’s exact test, and continuous variables were analyzed by the two-sample t test or Wilcoxon rank-sum test. Multivariate analysis of prognostic variables related to perioperative death was by a logistic multiple regression model.

**Late survival.** Survival was calculated actuarially by the Kaplan-Meier method. Univariate analysis of discrete variables was by the log-rank test or Gehan-Wilcoxon test. Both univariate and multivariate analyses of continuous variables were performed with the Cox proportional-hazards model.

### Results

**Perioperative mortality.** Perioperative (30 day or hospital) mortality was 24% (22 patients). There was a trend toward improved mortality in recent years. Among the 14 operations between 1975 and 1984, there was only one perioperative death (mortality 7%; p = .17). This death occurred in a patient who had undergone a previous mitral commissurotomy and subsequent mitral and tricuspid valve replacement. At the time of triple valve replacement, she underwent replacement of her malfunctioning tricuspid prosthesis, replacement of a malfunctioning mitral prosthesis, and an additional replacement of a stenotic native aortic valve.

The following factors were analyzed by univariate and multivariate logistic multiple regression analysis to evaluate their influence on perioperative mortality: sex, age, NYHA class, presence of atrial fibrillation (pure tricuspid insufficiency as opposed to mixed stenosis and insufficiency), previous surgery, duration of aortic cross-clamping, duration of cardiopulmonary bypass, use of cardioplegia, and year of operation. Univariate analysis identified only preoperative NYHA class, duration of cardiopulmonary bypass, and pure tricuspid insufficiency as significant predictors of hospital mortality (p < .0001, < .002, and < .01). By the multiple regression model, the only significant variable influencing hospital mortality was preoperative NYHA class IV (p < .02). Perioperative mortality was 44% among patients with class IV disability but was only 8% in patients with milder symp-
toms (p < .0001). Preoperative catheterization data and measurements of ejection fraction were available in insufficient numbers of patients to enable those variables to be subjected to statistical analysis. Cardioplegia was used in only seven patients during triple valve replacement.

**Causes of perioperative death.** Low cardiac output was the principal cause of perioperative death in the patients, including seven patients who could not be weaned from cardiopulmonary bypass and died in the intraoperative period. Four died in the early postoperative period because of inadequate myocardial function. Multiorgan failure, hemorrhage, and arrhythmia accounted for the hospital deaths of the remaining 11 patients.

**Long-term survival.** Figure 1 illustrates the cumulative survival of all patients and all 30 day perioperative survivors. The high attrition rate in the first year was not sustained, so that between years 1 and 10 the rate was about 3.5% per year. Overall survival was 64% at 1 year, 55% at 5 years, 40% at 10 years, and 25% at 15 years. For 30 day survivors, late survival was 54% at 10 years and 33% at 15 years. In perioperative (30 day) survivors, the median duration of follow-up was 7.5 years (range, 6 weeks to 20 years). The longest survivor is alive and well (NYHA class II) 20 years after operation, and one patient (previously reported) had a successful pregnancy after triple valve replacement.13

In patients with class II or III disability before surgery, overall 5 and 10 year survival rates were 73% and 54%, respectively; in contrast, survival rates for patients with preoperative class IV disability were 33% and 25%, respectively (figure 2). As expected, most of the difference between the two groups formed according to preoperative disability were noted in early mortality. In perioperative survivors 5 and 10 year survival rates were 80% and 59% in those with class II or III disability before surgery compared with 59% and 45% in those with class IV disability.

**TABLE 3**

<table>
<thead>
<tr>
<th>Types of prostheses used in 91 patients undergoing simultaneous replacement of three valves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Starr-Edwards</td>
</tr>
<tr>
<td>Bjork-Shiley</td>
</tr>
<tr>
<td>Braunwald-Cutter</td>
</tr>
<tr>
<td>Smeloff-Cutter</td>
</tr>
<tr>
<td>Bioprosthesis</td>
</tr>
<tr>
<td>Magovern-Cromie</td>
</tr>
<tr>
<td>Hall-Medtronic</td>
</tr>
</tbody>
</table>

**FIGURE 1.** Cumulative survival of all patients undergoing simultaneous triple valve replacement and that of perioperative survivors. Number of patients at risk is shown below the horizontal axis. Expected survival, determined on the basis of age and sex distribution, is from the 1970 life table for the West North Central states.
We compared the late survival of patients discharged from the hospital after triple valve replacement between 1962 and 1971 with that of a previously reported group of patients at the Mayo Clinic who underwent single valve replacement during the same period (figure 3). The survival curves are remarkably similar during the first 10 to 15 postoperative years. For 11 patients undergoing triple valve replacement since 1975, the cumulative 5-year survival rate was 81%; in comparison, the rate was 52% for patients who underwent triple valve replacement before 1975.

With use of the Cox proportional-hazards model, only preoperative NYHA class IV symptoms and advanced age were related to poor survival (\( p < .0002 \) and \( p < .01 \), respectively).

Causes of late death. Causes of late death among 67 perioperative survivors are listed in table 4. The most common cause was sudden death (death within 1 hr of symptoms). Three patients had fatal strokes, one patient died of pulmonary embolism presumed to be related to the Starr-Edwards tricuspid prosthesis, and one patient had thrombosis of a Starr-Edwards aortic valve and extension of the thrombus to the left main coronary artery. Progressive congestive heart failure was believed to be the cause of death in 15% of patients, and three other patients died as a result of prosthetic valve dysfunction (including reoperation in one patient). Two patients died of infective endocarditis.

Late complications. Major late complications are listed in table 5. Thromboembolism and hemorrhagic complications of anticoagulation produced serious morbidity during the follow-up period. Eight patients required reoperation on 11 cardiac valves. One patient underwent successful replacement of all three cloth-covered Starr-Edwards valves (6300 series) because of cloth wear and severe hemolysis. Isolated tricuspid valve malfunction requiring reoperation developed in two patients with Starr-Edwards prostheses and in one patient with a Björk-Shiley prosthesis. One patient required replacement of both Starr-Edwards aortic and tricuspid prostheses, and two patients underwent reoperation for mitral periprosthetic leaks, which required valve replacement in one patient and suture repair in the other. The remaining patient underwent reoperation for a malfunctioning Braunwald-Cutter aortic valve. The actuarially determined risk of reoperation due to valve malfunction or periprosthetic leak was 4% at 5 years, 12% at 10 years, and 17% at 12 years.

Thromboembolism occurred in 29 patients, and 12 of these had multiple episodes of thromboembolism (table 6). The thromboembolic rate (reflecting all thromboembolic episodes, regardless of severity) was 12.3 per 100 patient-years. The central nervous system was the most commonly involved site, and in three patients, cerebrovascular thromboembolism was fatal. Most episodes (30 of 43) were not associated with permanent neurologic deficits. Figure 4 illustrates the actuarially determined survival free of thromboembolism. Only the first thromboembolic event was considered in the actuarial analysis, but all first episodes were included, regardless of severity. Embolism-free survival rates were 86% at 1 year, 70% at 5 years, and 43% at 10 and 12 years after surgery.

Twelve patients in this series required permanent

Infective endocarditis
Permanent reoperation
Cerebrovascular episode

TABLE 4
Causes of late death in 40 of 69 perioperative survivors

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden death</td>
<td>13</td>
<td>32.5</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>6</td>
<td>15</td>
</tr>
<tr>
<td>Thromboembolism</td>
<td>5</td>
<td>12.5</td>
</tr>
<tr>
<td>Prosthetic valve dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(including reoperation in one patient)</td>
<td>3</td>
<td>7.5</td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
<td>7.5</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Other</td>
<td>5</td>
<td>12.5</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Septicemia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Acute pulmonary edema (cause unknown)</td>
<td>1</td>
<td>2.5</td>
</tr>
<tr>
<td>Noncardiac death</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

Pacemakers. In two patients, permanent pacemaker implantation was performed in the perioperative period. In the other 10 patients, permanent pacemakers were required for symptomatic bradyarrhythmias, and the most frequent indication was atrial fibrillation with complete heart block or symptomatic pauses. In these patients, the interval from operation to pacemaker implantation ranged from 3 to 18 years (mean, 9.4 years) after initial valve replacement.

Functional outcome. Figure 5 illustrates the functional class of the 29 patients (32% of the total population) still alive at the end of follow-up. The median duration of follow-up was 10.7 years. Clinical improvement was striking. Before surgery 96% of patients were NYHA class III or IV, and at the time of last follow-up 79% of patients were NYHA class I or II.

Discussion
At our institution, the frequency of triple valve replacement has decreased markedly during the past decade. This may be explained by a decrease in the incidence of rheumatic heart disease and by recent trends toward conservative reparative procedures for patients with functional tricuspid valve insufficiency. Between 1975 and 1984, 59 patients have undergone aortic and mitral valve replacement with additional tricuspid annuloplasty at our institution.

Early mortality. The reported risk of triple valve replacement varies widely — from 10.5% to 50%. 11, 12, 14-25 This probably reflects the lack of uniformity in operative techniques, choice of prostheses, and patient selection criteria over the past 20 years as well as the small number of patients in most published series. In our own experience, operative mortality appears to have declined since 1975. It is likely that the relatively small number of patients undergoing this procedure since 1975 precludes statistical significance in comparisons of mortality, but many improvements in the management of patients with triple valve disease are believed to have been implemented during this experience. Most important is the improved preoperative clinical status of patients referred for operation. The higher operative risk for patients with class IV

TABLE 5
Late complications in 69 perioperative survivors

<table>
<thead>
<tr>
<th>Complication</th>
<th>n</th>
<th>%</th>
<th>No. in whom complication was fatal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embolism</td>
<td>29</td>
<td>42</td>
<td>5</td>
</tr>
<tr>
<td>Bleeding</td>
<td>15</td>
<td>22</td>
<td>3</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>11</td>
<td>16</td>
<td>1</td>
</tr>
<tr>
<td>Cerebrovascular episode</td>
<td>11</td>
<td>16</td>
<td>5(^{a})</td>
</tr>
<tr>
<td>Reoperation</td>
<td>8</td>
<td>11.5</td>
<td>1</td>
</tr>
<tr>
<td>Permanent pacemaker</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>implantation</td>
<td>10</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>4</td>
<td>6</td>
<td>2</td>
</tr>
</tbody>
</table>

Some patients had more than one complication.

\(^{a}\)Including two with hemorrhage.

FIGURE 4. Cumulative survival free of a first thromboembolic event in perioperative survivors after triple valve replacement. Number of patients at risk is shown below horizontal axis.

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disability before surgery is similar to findings in other reports, and is emphasized in our study by the results of the univariate and multivariate analyses, which show that preoperative functional status is the most powerful predictor of perioperative mortality. It is relevant that the percentage of patients with preoperative class IV disability decreased from 50% in the period before 1975 to 8% in the 1975 through 1984 period (p < .02).

Significant changes in surgical techniques occurred during the latter part of our experience. Although the small numbers of patients undergoing triple valve replacement since 1975 precluded further statistical analysis, the reduction in perioperative mortality could also reflect improved methods of intraoperative protection (only seven patients had cardioplegia) and subtle technical advances, including those in the areas of methods for suturing prosthetic valves, methods for removing intracardiac air at the conclusion of extracorporeal circulation, and routine placement of epicardial temporary pacing electrodes for control of postoperative arrhythmias. Before 1975, temporary pacing was used in 24% of patients; this rate increased to 54% from 1975 onward.

Operative mortality was 34% in patients with pure tricuspid insufficiency, which is significantly higher than the 13% in patients with mixed tricuspid valve disease. In some of the former group, tricuspid insufficiency might have been functional and a manifestation of poor right ventricular function and pulmonary hypertension, whereas in patients with mixed disease, tricuspid valve involvement presumably was organic.

Late mortality. Late attrition — that after the first postoperative year — is relatively small but consistently observed. The overall 5, 10, and 15 year survival rates in this series are similar to rates in other studies. Five year survival rates were reported to range from 42% to 67%, and a 15 year survival rate of 23% was reported by Teply et al. Again, patients with advanced heart failure and class IV disability have a much higher long-term mortality. This fact and the relatively good results of triple valve replacement in our patients with milder symptoms (all but three of whom were NYHA class III) argue strongly for earlier operation. Five year survival in perioperative survivors operated on since 1975 is 90% and is at least partially related to their better preoperative status; only one patient had class IV symptoms. Furthermore, the date of surgery did not appear as an independent predictor of long-term mortality.

For perspective, we compared the long-term results of triple valve replacement with those of single valve replacement over a similar time span (figure 3). Other authors have emphasized that late survival after triple valve replacement is significantly worse than that noted in patients who have undergone single valve replacement. However, most of this difference relates to the high perioperative mortality in the multiple valve replacement group. For example, our data (figure 3) indicate that the outcome for perioperative survivors after triple valve replacement is similar to that of patients surviving single valve replacement. Since the severity of symptoms is such an important predictor of perioperative mortality, the case for earlier operation is further strengthened.

Causes of late death. Approximately one-third of the late deaths in this series were determined to be related to thromboembolism, bleeding, or prosthetic valve dysfunction. The incidence of valve-related deaths is similar to the 43% reported by Macmanus et al. after triple valve replacement and higher than that noted after single valve replacement.

The high frequency of late sudden deaths after triple valve replacement is disturbing. In other long-term follow-up series of patients undergoing surgery for valvular heart disease, sudden death was the primary cause of late mortality in 20% to 43%. Some of the deaths in this study were due to documented ventricular arrhythmias. The frequency of complex ventricular arrhythmias after tricuspid and triple valve replacement has been alluded to previously but not systematically evaluated. Other possible causes of sudden death are thromboembolism, intracerebral hemorrhage, sudden prosthetic valve dysfunction, and intracardiac conduction disease. Sudden death after valve replacement is clearly a cause for concern; the scope of the problem and the approach to management, however, need further evaluation.
An important finding of this review was the significant incidence of bradyarrhythmias requiring permanent pacemaker implantation. The causes are multifactorial, including intraoperative damage to the conduction system, fibrosis secondary to chronic compressive effects of the rigid mitral and tricuspid sewing ring, and rheumatic heart disease. During the course of this experience, indications for permanent pacing have become more liberal, and this change could have contributed to the late incidence of permanent pacemaker implantation. Nonetheless, all the patients undergoing pacemaker implantation, except one in whom implantation was performed intraoperatively, did have symptoms of major conduction disease.

Our data suggest that late survivors after valve replacement should be regularly evaluated (including Holter monitoring) for ventricular arrhythmias and conduction disturbances. A high index of suspicion should be maintained, since the presentation of conduction disease and arrhythmias may be subtle.

Currently, we advocate implantation of permanent epicardial leads at the time of triple valve replacement to avoid a pacemaker lead being placed across the prosthetic tricuspid valve.

**Thromboembolism.** Comparing the incidence of thromboembolism in different series is difficult. The incidence varies widely and depends on criteria used to define a thromboembolic event, frequency and accuracy of follow-up data, type of valve used, and the inherent limitations of retrospective analyses. Clearly, thromboembolism is a frequent cause of serious morbidity and mortality after triple valve replacement; the embolic rate of 12.3 events per 100 patient-years in this study is similar to the 11.4 events per 100 patient-years reported by Macmanus et al. Embolism-free survival at 5 years is 70%, similar to that in other early series of single valve replacements with Starr-Edwards valves. The 10 year embolism-free rate is lower and the overall embolic rate higher in patients with three valves than in those with single or double valves. Several reports have documented a recent reduction in incidence of thromboembolism after valve replacement. This may be related to improved methods of maintaining anticoagulation, the addition of antiplatelet agents, earlier operation, improved prosthetic valve design, or a combination of these.

**Valve durability.** The durability of the Starr-Edwards valve is underscored by the relatively low incidence of reoperation or death due to prosthetic valve dysfunction. The vulnerability of the mechanical prosthesis (particularly tilting-disk prostheses) in the tricuspid position to thrombosis and sudden malfunction has been noted by others. Five of the 11 valves requiring replacement were in the tricuspid position, and two deaths were due to tricuspid valve thrombosis, an indication that the Starr-Edwards valve is also prone to dysfunction in this position. There are lessons to be derived from this experience. The use of certain prosthetic valves subsequently found to be associated with a high incidence of problems, for example, the Braunwald-Cutter valve and the cloth-covered Starr-Edwards valve, was discontinued later. Improved techniques for tricuspid anuloplasty have undoubtedly diminished the requirement for tricuspid valvular replacement, and when replacement is still found to be necessary, the knowledge that mechanical prostheses tend to do less well in the tricuspid position than in the mitral or aortic position has led to our current policy of using tissue valves for tricuspid valve replacement.

**Symptomatic status.** The symptomatic status of patients currently alive is encouraging, and the results of comparisons with preoperative status are quite striking. That 79% of patients after a median duration of follow-up of almost 11 years should be in NYHA class I or II is indicative of the value of the procedure, but these results must be construed in light of the fact that 68% were dead at the time of last follow-up. Furthermore, the impact of surgery on these patients is not confined to the procedure of triple valve replacement. Twenty-three patients had had prior cardiac surgical procedures, and some of these patients were still alive and well more than 30 years after an initial mitral commissurotomy.

In conclusion, in our experience operative mortality for patients undergoing triple valve replacement appears to have been reduced. The frequency of use of the procedure has declined markedly in parallel with an increase in the number of patients undergoing double valve replacement and tricuspid anuloplasty. Early and late results can be expected to improve further if operative intervention is carried out before the onset of class IV symptoms. The frequency of late sudden death is of concern, and periodic evaluation for ventricular arrhythmias and conduction disturbances is recommended. The increased rate of thromboembolism and the significant incidence of bleeding after triple valve replacement with mechanical prostheses mandate meticulous anticoagulation for life.

Because most late mechanical prosthetic failures were of valves in the tricuspid position, the use of tricuspid anuloplasty when possible is recommended; if tricuspid valve replacement is required, a biologic prosthesis should be considered.
In patients who survive the perioperative period, the favorable long-term outcome and excellent symptomatic relief after triple valve replacement establish a continued role for the procedure in patients with severe multivalvular rheumatic heart disease.

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References


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