SURGERY FOR ACQUIRED CARDIOVASCULAR DISEASE

MITRAL VALVE REPAIR AND REPLACEMENT FOR RHEUMATIC DISEASE

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Objectives: Mitral valve repair may be technically feasible in patients with suitable anatomy, but the appropriateness of repair for rheumatic disease remains controversial. We evaluated our late outcomes after mitral repair and replacement for rheumatic disease. **Methods:** Five hundred seventy-three patients underwent mitral valve surgery for rheumatic disease at our institution from 1978-1995. Follow-up was 98% complete (mean, 68 ± 46 months). Survival and morbidity were evaluated by Kaplan-Meier analysis and Cox regression, including propensity score analysis. **Results:** Mean age was 54 ± 14 years, 55% of patients had congestive heart failure, 22% were undergoing redo mitral valve surgery, and 9% also underwent coronary bypass. Mitral stenosis was present in 53%, regurgitation in 15%, and both in 32%. Valve repair was performed in 25%, bioprosthetic replacement was performed in 28%, and a mechanical valve was placed in 47%. Patients undergoing repair were younger and less likely to be undergoing reoperation or to have atrial fibrillation than those undergoing replacement ($P = .001$). The operative mortality rate was 4.2%. Better late cardiac survival was independently predicted by valve repair rather than replacement ($P = .04$) after adjustment for baseline differences between patients. Freedom from reoperation was greatest ($P = .005$) but that from thromboembolic complications was worst ($P < .0001$) after mechanical valve replacement. Twenty-three patients underwent reoperation after initial repair, with no operative deaths. **Conclusions:** Mechanical valves minimize reoperation but limit survival and increase thromboembolic complications. Patients undergoing valve repair had improved late cardiac survival independent of their preoperative characteristics. Rheumatic mitral valves should be repaired when technically feasible, accepting a risk of reoperation, to maximize survival and reduce morbidity. (J Thorac Cardiovasc Surg 2000;119:53-61)

Mitral valve repair is the procedure of choice in patients with mitral regurgitation caused by degenerative disease. Reparative procedures on degenerative mitral valves are associated with low operative mortality and morbidity rates and excellent late survival, free-

dom from reoperation, and freedom from thromboembolic complications.¹⁻³ However, the appropriateness of valve repair for patients with rheumatic mitral valve disease, even when repair appears to be technically feasible, remains controversial.⁴⁻⁶ Our approach has been

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Table I. Patient characteristics

	<i>Repair</i>	<i>Bioprosthesis</i>	<i>Mechanical</i>	<i>P value</i>
Demographics				
No. of patients	142	162	269	
Age (y)	42 ± 1.1	61 ± 1.0	56 ± 0.7	.0001
Sex (% female)	85	77	81	.3
Reoperative mitral surgery (%)	5.6	22	30	.001
Congestive heart failure (%)	53	33	68	.001
Previous CVA/TIA (%)	11	12	20	.02
Preoperative atrial fibrillation (%)	31.7	59.3	67.3	.001
Pathology				
Stenosis (n)	96	82	125	.001
Regurgitation (n)	23	31	33	
Mixed (n)	23	49	111	
Intraoperative				
Coronary bypass (%)	2.1	9.9	13	.002
Tricuspid valve surgery (%)	7.8	14	20	.003
CPB time (min)	54 ± 2	77 ± 2	87 ± 2	.0001
XCL time (min)	38 ± 2	54 ± 2	63 ± 2	.0001
Early outcomes				
Mortality (%)	0.7	5.6	5.2	.057
Myocardial infarction (%)	0	1.2	1.9	.3
Low output syndrome (%)	18	28	16	.012
Stroke (intraoperative/postoperative)	0/0.7	1.2/0	1.5/0.4	.5
Follow-up (mo)	78 ± 4	84 ± 4	54 ± 2	.0001

Continuous variables are presented as means ± SE. CVA, Cerebrovascular accident; TIA, transient ischemic attack; CPB, cardiopulmonary bypass; XCL, aortic cross-clamp.

to repair rheumatic mitral valves when the anatomic substrate appears to permit it. We have reviewed our 17-year single-institution experience with repair and replacement of rheumatic mitral valves to determine the late outcomes and advisability of this strategy.

Methods

Patients. From October 1978 to June 1995, 573 patients underwent mitral valve replacement or repair for rheumatic disease with Carpentier techniques⁷⁻⁹ at our institution. Patients undergoing concomitant aortic surgery were excluded, but patients undergoing concomitant coronary bypass or tricuspid valve surgery were not. Demographic, intraoperative, and perioperative outcome data were recorded prospectively.

Demographics. The mean age of the 573 patients was 54 ± 14 years; 81% were female, 55% had congestive heart failure, 22% were undergoing redo mitral valve surgery, and 9% also underwent coronary bypass. Mitral stenosis was present in 53%, regurgitation in 15%, and both in 32%. Patient demographics, grouped by the type of surgery performed, are listed in Table I.

Procedures. Valve repair was performed in 25%, 28% had a bioprosthesis, and 47% had a mechanical valve. The choice of procedure varied by the underlying valvular pathology (Table I).

Follow-up. Follow-up was conducted by mailed questionnaire or telephone interview and by review of the surgeon's

and cardiologist's office charts between January 1996 and April 1997 and was 98% complete. The mean duration of follow-up was 68 ± 46 months. Late survival and valve-related outcomes were recorded and analyzed as per the specifications of the Joint Society of Thoracic Surgeons–American Association for Thoracic Surgery Ad Hoc Liaison Committee for Standardizing Definitions of Prosthetic Heart Valve Morbidity.¹⁰

Statistical analysis. Data were collected and managed in dBASE IV data sets and analyzed with SAS and BMDP/DYN LR statistical analysis software. Univariate analysis of categorical data was carried out with χ^2 or Fisher exact tests. Univariate analysis of continuous variables was carried out with analysis of variance or the Student *t* test. Late survival and time-dependent morbidity were evaluated univariately by Kaplan-Meier analysis and multivariately by Cox regression.

We also performed a propensity score analysis to quantify the probability that a patient might receive a repair versus a mechanical or bioprosthetic replacement and allow for the evaluation of potential surgical bias in patient selection on late outcomes. To do this, we first performed a logistic regression analysis for the probability that a patient would be selected to have a valve replacement rather than repair. The independent predictors of valve replacement were age, coronary artery disease, type of valve pathology, preoperative atrial fibrillation, and reoperative mitral valve surgery. This model had an area under the receiver-operator characteristic curve of 0.806 and a Hosmer-Lemeshow goodness-of-fit *P*

Fig 1. Survival in 573 patients undergoing mitral valve surgery for rheumatic disease. *Rep*, Valve repair; *Bio*, bioprosthetic valve replacement; *Mech*, mechanical valve replacement.

value of .68. The regression coefficients for each independent predictor were then used to calculate the predicted probability of valve replacement for each patient. The natural log of the probability was calculated as the propensity score.

The Cox regression analyses were then repeated, with the inclusion of the propensity score as a potential predictor of late outcomes, to adjust for the bias in selecting a patient for repair versus replacement. The propensity score did not emerge as an independent predictor of any of our late outcomes, suggesting that differences attributed to type of valve surgery by the initial Cox regression analyses were not explained by surgical bias in patient selection on the basis of their preoperative characteristics.

Results

Hospital outcomes. Operative mortality for all 573 patients was 4.2% but was only 0.7% in patients undergoing repair (Table I). Postoperative low output syndrome was more prevalent after replacement with a bioprosthetic valve ($P = .012$). There was no statistically significant difference between groups in the prevalence of myocardial infarction or perioperative stroke.

Late outcomes

Survival. Overall 5- and 10-year survival was as follows: valve repair, $97\% \pm 1.8\%$ and $88\% \pm 4.5\%$; bioprosthetic valve replacement, $83\% \pm 3.0\%$ and $70\% \pm 4.4\%$; and mechanical valve replacement, $88\% \pm 2.2\%$ and $73\% \pm 6.0\%$ (Fig 1). By Cox regression, the predictors of late survival in all patients with rheumatic mitral valve disease were advanced age (hazard ratio

[HR], 1.07; 95% confidence interval [CI], 1.05-1.09; $P = .0001$), the presence of coronary artery disease (HR, 2.10; 95% CI, 1.15-3.84; $P = .014$), and New York Heart Association symptom class (HR, 1.46; 95% CI, 1.05-2.03; $P = .025$). The complexity of the mitral repair procedure required (commissurotomies for mitral stenosis versus more complex repairs for mitral regurgitation or mixed stenosis and regurgitation) did not appear to directly affect long-term survival; within the 142 patients undergoing mitral repair, the type of valve pathology (stenosis, regurgitation, or mixed stenosis and regurgitation) was not significantly related to late survival ($P = .3$).

Cardiac death. Five and 10-year freedom from cardiac death, including valve-related complications, was as follows: valve repair, $99\% \pm 1.1\%$ and $94\% \pm 3.8\%$; bioprosthetic valve replacement, $90\% \pm 2.6\%$ and $84\% \pm 3.9\%$; and mechanical valve replacement, $95\% \pm 1.5\%$ and $86\% \pm 4.5\%$ (Fig 2). Advanced age was an independent predictor of cardiac death (HR, 2.46; 95% CI, 1.76-3.45; $P = .0001$), whereas mitral valve repair independently predicted improved survival (HR for valve replacement rather than repair, 3.34; 95% CI, 1.32-8.45; $P = .01$).

Nonvalve-related cardiac death. Freedom from cardiac death, excluding valve-related causes, was predicted by advanced age (HR, 1.06; 95% CI, 1.04-1.09; $P = .0001$) and New York Heart Association symptom class (HR, 2.03; 95% CI, 1.27-3.24; $P = .003$). The presence

Fig 2. Freedom from cardiac death in 573 patients undergoing mitral valve surgery for rheumatic disease. *Rep*, Valve repair; *Bio*, bioprosthetic valve replacement; *Mech*, mechanical valve replacement.

Fig 3. Freedom from valve-related death in 573 patients undergoing mitral valve surgery for rheumatic disease. *Rep*, Valve repair; *Bio*, bioprosthetic valve replacement; *Mech*, mechanical valve replacement.

of coronary artery disease (HR, 1.91; 95% CI, 0.89-4.07; $P = .09$) and use of a mechanical prosthesis ($P = .17$) did not emerge as significant independent predictors.

Valve-related death. Five and 10-year freedom from valve-related death was as follows: valve repair, 99% ± 1.1% and 99% ± 1.1%; bioprosthetic valve replacement, 96% ± 1.7% and 92% ± 2.8%; and mechanical valve replacement, 97% ± 1.2% and 95% ± 2.4% (Fig

3). Valve-related death was predicted by advanced age (HR, 1.08; 95% CI, 1.04-1.13; $P = .0001$). Type of valve surgery (repair, bioprosthesis, or mechanical prosthesis) also emerged in the regression model but did not reach statistical significance ($P = .078$).

Reoperation. Five and 10-year freedom from reoperation was as follows: valve repair, 87% ± 3.0% and 72% ± 5.3%; bioprosthetic valve replacement, 94% ±

Fig 4. Freedom from reoperation in 573 patients undergoing mitral valve surgery for rheumatic disease. *Rep*, Valve repair; *Bio*, bioprosthetic valve replacement; *Mech*, mechanical valve replacement.

Fig 5. Freedom from thromboembolic events in 573 patients undergoing mitral valve surgery for rheumatic disease. *Rep*, Valve repair; *Bio*, bioprosthetic valve replacement; *Mech*, mechanical valve replacement.

2.1% and $69\% \pm 5.4\%$; and mechanical valve replacement, $96\% \pm 1.4\%$ and $95\% \pm 1.8\%$ (Fig 4). The risk of reoperation was predicted by bioprosthetic valve replacement (HR, 4.29; 95% CI, 1.74-10.59; $P = .002$), valve repair (HR, 3.40; 95% CI, 1.34-8.60; $P = .01$), age (HR, 0.98; 95% CI, 0.96-0.998; $P = .03$), and active endocarditis (HR, 8.93; 95% CI, 1.16-68.7; $P = .04$).

Of the 142 patients undergoing valve repair, 23 (16%) patients underwent subsequent reoperation, with no deaths. The nature of the mitral pathology at the initial operation and therefore the complexity of the repair required (simple commissurotomy for isolated mitral stenosis versus more complex repairs of regurgitant or mixed lesions) were associated with the risk of late reoperation. Five-year freedom from reoperation was

93% $\pm$ 2.9% for stenotic valves (67% of patients), 82% $\pm$ 8.1% for regurgitant valves (17% of patients), and 94% $\pm$ 5.4% for valves with mixed stenosis and regurgitation (16% of patients) ($P = .012$).

Thromboembolic events. Five and 10-year freedom from thromboembolic complications was as follows: valve repair, 93% $\pm$ 2.3% and 93% $\pm$ 2.3%; bioprosthetic valve replacement, 94% $\pm$ 1.9% and 93% $\pm$ 2.2%; and mechanical valve replacement, 89% $\pm$ 2.5% and 72% $\pm$ 6.7% (Fig 5). Thromboembolic complications were predicted by type of use of a mechanical prosthesis (HR, 6.91; 95% CI, 2.94-16.25; $P = .0001$), advanced age (HR, 1.05; 95% CI, 1.02-1.09; $P = .0006$), and active endocarditis (HR, 17.3; 95% CI, 2.15-139; $P = .007$). The presence of coronary artery disease (HR, 2.27; 95% CI, 0.95-5.46; $P = .066$) and the type of mitral valvular lesion (stenosis, regurgitation, or mixed stenosis and regurgitation; HR, 1.42; 95% CI, 0.98-2.06; $P = .067$) did not reach statistical significance as predictors of thromboembolic events.

Endocarditis. Five and 10-year freedom from repaired native or prosthetic valve endocarditis was as follows: valve repair, 99% $\pm$ 0.8% and 98% $\pm$ 1.3%; bioprosthetic valve replacement, 98% $\pm$ 1.1% and 97% $\pm$ 1.6%; and mechanical valve replacement, 99.6% $\pm$ 0.4% and 97% $\pm$ 2.2% ($P = .8$).

Total valve-related morbidity. Five and 10-year freedom from valve-related morbidity (thromboembolic events, endocarditis, or reoperation) was as follows: valve repair, 86% $\pm$ 3.1% and 71% $\pm$ 5.3%; bioprosthetic valve replacement, 91% $\pm$ 2.5% and 62% $\pm$ 5.4%; and mechanical valve replacement, 87% $\pm$ 2.8% and 64% $\pm$ 6.3% ($P = 0.3$).

Discussion

Over the last 2 decades, our institutional practice in patients undergoing mitral valve surgery for rheumatic disease has evolved from predominant implantation of bioprosthetic valves in the 1970s to a more aggressive strategy of mitral valve repair, starting in the early 1980s, whenever the valvular anatomy appeared to allow correction of the hemodynamic abnormalities. Over time, mechanical prostheses have largely supplanted bioprosthetic valves in the mitral position. However, we have noted a 10-year survival rate of only 73% $\pm$ 6.0% at 10 years after implantation of a mechanical mitral prosthesis, a figure consistent with most published series.¹¹⁻¹³ This late mortality is attributed largely to thromboembolic events and bleeding complications of long-term anticoagulation. Although lower intensity anticoagulation with the St Jude Medical bileaflet valve may be possible, late mortality and morbidity rates remain substan-

tial and have led to persistent interest in defining a subset of patients with rheumatic mitral valvular pathology in whom repair may yield better long-term outcomes.^{5,6,14} These considerations prompted us to review the long-term results of our current operative strategies in patients with rheumatic mitral valve disease.

Late consequences of mitral valve repair. In the carefully selected patients in whom valve repair was performed, we noted an 88% 10-year survival rate compared with a 70% rate for bioprostheses and a 73% rate for mechanical valves. This difference may be partially accounted for by the lower mean age of the patients undergoing repair. We were more aggressive in performing repairs in young patients in order to avoid decades of anticoagulation. However, Cox regression analysis identified mitral repair as a predictor of better cardiac survival independent of age. Therefore the beneficial effect of valve repair is not due to its being performed in younger patients. In addition, patients undergoing mitral repair were less likely to be in atrial fibrillation or to have had a previous stroke or transient ischemic attack. The propensity score analysis was performed to permit evaluation of the effect of type of valve surgery independent of these differences in baseline characteristics, and the results of this analysis suggest that the greater cardiac survival after mitral valve repair was again independent of the differences in age, atrial fibrillation, and previous stroke.

Mitral valve repair was also associated with excellent freedom from thromboembolism but only a 72% freedom from reoperation at 10 years. Reoperation, the major liability of an aggressive strategy of valve repair, was required in 23 patients and was accomplished without mortality. The observation that reoperation did not carry a demonstrably increased risk of death in this series favors a strategy of repair in selected patients. This approach would obviously not be appropriate, however, if reoperation was associated with significant additional mortality or morbidity. At the time of reoperation, however, patients were much more likely to receive a mechanical valve to minimize the chance of requiring a third operation.

Mechanical valves were associated with high and ongoing rates of thromboembolic and bleeding complications. In this series implantation of a mechanical valve was the most significant independent predictor of subsequent thromboembolic events but also the most significant predictor of freedom from reoperation. Mechanical valves were associated with decreased freedom from both valve-related death and nonvalve-related cardiac death, but these associations were not statistically significant.

Selection of patients for repair

Rheumatic mitral stenosis. Patients with rheumatic stenotic mitral valves with low echocardiographic scores are ideal candidates for percutaneous balloon valvotomy.¹⁵⁻¹⁷ Excellent short-term and long-term outcomes have been reported by various groups treating patients with primarily rheumatic mitral stenosis. Our approach would be to palliate these patients with percutaneous balloon valvotomy, reserving operation for patients with more advanced disease.

Patients with moderate distortion of mitral valvular anatomy but in whom the anterior leaflet and chordae tendineae still appear pliable should undergo surgical exploration to determine whether repair is feasible. The thickness of the valve leaflets and the presence of chordae tendineae determine the ability to repair the stenotic rheumatic mitral valve. Patients in whom the papillary muscles are fused directly to the free margin of the valve leaflets should undergo valve replacement rather than repair.

The appearance of a severely distorted valve on preoperative echocardiography, however, portends a nearly certain requirement for valve replacement. In addition, patients with combined aortic and mitral disease have poor outcomes after either aortic valve repair¹⁸ or mitral valve repair,⁴ and in most patients with multiple-valve disease, we would favor valve replacement.

In patients with borderline valvular anatomy, valve repair may also be favored for nonanatomic considerations, primarily contraindications to long-term anticoagulation. Conversely, valve replacement may be favored in patients in whom long-term anticoagulation will be necessary for other indications. However, valve repair even in a subset of these patients may allow lower intensity anticoagulation and perhaps a reduction in bleeding complications.

Rheumatic mitral regurgitation. The risk of reoperation after repair for rheumatic mitral regurgitation may be related to patient age and to the presence of active rheumatic carditis.^{5,19} In young Saudi patients presenting with a dilated anulus, thickened but mobile leaflets, and somewhat thickened and elongated chordae but without severe commissural fusion and subvalvular fibrosis, Gometza and colleagues²⁰ reported an actuarial survival rate of $98\% \pm 2\%$ at 78 months after repair compared with only $75\% \pm 19\%$ at 48 months after replacement. However, 37% of patients undergoing repair required reoperation, with 81% of reoperations required within the first year.

In older patients (mean age, 55 years) with isolated rheumatic mitral regurgitation or mixed regurgitation and stenosis, Grossi and colleagues⁴ reported a 92%

freedom from reoperation at 8 years in patients receiving a St Jude Medical valve compared with 86% for patients undergoing repair. This difference was not statistically significant, but this was felt to be due to the small number of patients followed to that interval.

The failure of mitral valve repair in patients with rheumatic mitral regurgitation appears to be more often valve related (including progressive primary valve disease, endocarditis, or leaflet retraction) than procedure related (ie, suture dehiscence, rupture of previously shortened chordae, or incomplete initial correction).²¹

Mixed rheumatic mitral stenosis and regurgitation. Mixed mitral stenosis and regurgitation caused by rheumatic disease may increase the risk of reoperation after repair. Fernandez and colleagues²² reported that freedom from reoperation after valve repair was 90% at 5 years and 80% at 8 years in patients who had either pure mitral regurgitation or isolated mitral stenosis, but patients with mixed mitral stenosis and regurgitation had only an 80% freedom from reoperation at 5 years and 72% at 10 years. We did not note an increased propensity to failure after repair of valves with a combination of stenosis and regurgitation in our series ($94\% \pm 5\%$ freedom from reoperation at 5 years), but the number of patients in this category was small.

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Discussion

Professor Alain F. Carpentier (*Paris, France*). This is an important paper because if there is little doubt as to the benefit of valve repair in congenital or degenerative valvular disease, recent papers have questioned the interest of valve repair in rheumatic valvular disease.

The authors report a 72% freedom from reoperation at 10 years, and whenever a reoperation was necessary, a 0% operative mortality rate. There was a striking difference in patient survival between valve repair and valve replacement, with 88% versus 72% survival rates at 10 years, respectively. The authors underlined some demographic differences between the 2 groups that favor the valve-repair group. I wonder whether these differences could play a significant role

because my own experience with a larger series and more similar groups confirms that valve repair with the techniques we use is superior to valve replacement. In our experience the freedom from reoperation was 79% at 10 years and 76% at 13 years, a striking difference with other series reported in the literature by authors advising valve replacement rather than valve repair in rheumatic valvular disease. This difference, I think, is not due to different types of patient population but to different types of repair operation.

To see whether we can further reduce the incidence of reoperation, it is interesting to discuss the most frequent causes of reoperation. In our experience they were as follows: annulus dilatation (16%), valve stenosis (16%), leaflet retraction (32%), and leaflet prolapse (36%).

Annulus dilatation is due to a persistent process of annulus distention when no ring was used at the first operation. The simple means to avoid this is to systematically use a prosthetic ring even when a quadrangular resection of the posterior leaflet has been performed. It is important to use large prosthetic rings in rheumatic valvular disease to compensate for the increased rigidity of the leaflets and to reduce the incidence of recurrent stenosis. To do so, one should perform a leaflet extension of either the anterior, the posterior, or both leaflets using glutaraldehyde-treated autologous pericardium. Leaflet extension allows selection of a 1 to 2 size larger ring. I would like to ask the authors whether they have correlated the incidence of recurrent stenosis to the size of the rings used and whether they were able to note that the smaller the ring implanted, the higher the chance of recurrent stenosis in rheumatic valvular disease.

Another cause of reoperation is recurrent leaflet prolapse. How often did the authors find it and what were the mechanisms involved, chordal rupture or failure of a previous chordal shortening? Because I am responsible for having introduced the technique of chordal shortening more than 2 decades ago, I would like to underline a possible cause of failure associated with this technique. The technique consists in burying the extra length of the chords in a longitudinal trench created in the papillary muscle. A secondary chordal rupture can occur whenever the suture used to close the trench is close to the shortened chords, thus leading to chordal abrasion. We therefore recommend placing this suture at a distance from the chords. Since taking this precaution, we have not seen this complication.

Finally, I would like to ask the authors what their current policy is concerning the use of valve repair versus valve replacement in rheumatic valvular disease. In light of the excellent results you presented, do you envision enlarging your indications? In our own experience, particularly at the Vietnam Heart Institute, thanks to a larger use of leaflet extension, the proportion of valve repair versus valve replacement has increased up to 85% in rheumatic valvular disease.

Dr Yau. Thank you, Professor Carpentier, for your kind comments. As to the comparability of these various groups, this is obviously a retrospective analysis of patients operated on over a 17-year time period. We did identify, by means of Cox regression analysis, that late cardiac survival was favor-

ably influenced by mitral valve repair independent of the other potentially confounding factors.

As you have noted, the patients who were undergoing repair were a very select patient population, younger and less likely to undergo reoperative surgery and less likely to have had a stroke or atrial fibrillation preoperatively. The Cox regression analysis did allow us to identify the type of valve surgery as a favorable independent predictor of late cardiac survival. Independent of those things, however, it is clear that in a retrospective analysis such as this, it would be impossible to exclude the effect of other unknown, confounding variables. From the data that we have, I think that we can certainly strongly support the approach that we have had at our institution over the preceding years, that is, to repair a selected subset of these patients with rheumatic mitral valve disease. This in and of itself cannot necessarily support a call for expanded indications for mitral valve repair, although that is certainly our personal bias.

As far as the incidence of stenosis and possible correlations with the size of the annuloplasty ring, in this case not all of our patients had continuing annual echocardiograms, and therefore our primary indication of failure in many of the patients who underwent repair was reoperation. In the small number of patients who underwent reoperation, we were not able to correlate size of the annuloplasty ring with the subsequent development of mitral stenosis.

As far as our current recommendations for mitral repair in this population, for patients with rheumatic mitral stenosis, I think we would certainly agree that patients with low echocardiographic scores and relative preservation of normal mitral anatomy should undergo a percutaneous valvotomy. Patients with moderate distortion of mitral anatomy should, at the time of surgery, undergo exploration for possible repair, and if the anterior leaflet and chordae are relatively preserved and reasonably pliable, then we would certainly favor a strat-

egy of repair in those patients. We would reserve replacement with a mechanical prosthesis for patients in whom we felt the anatomy was unsuitable for repair and less commonly for patients with mixed stenosis and regurgitation. As you saw in our series, mixed stenosis and regurgitation was generally associated with mechanical valve replacement, and valve repair has been reported by some authors, notably Fernandez and colleagues, to result in inferior late outcomes in these patients. The combination of aortic and mitral pathology would generally, in our opinion, also constitute an indication for valve replacement rather than repair.

Dr Lawrence I. Bonchek (*Lancaster, Pa*). I would like to draw attention to your emphasis on the fact that the thromboembolism rate was much higher in the prosthesis group than in the repair group.

You mentioned a lot of confounding variables, including presumably atrial fibrillation, that did not correlate with postoperative death, but did you correlate the incidence of atrial fibrillation with the incidence of thromboemboli in both groups? Although things went by rather quickly, it was quite apparent that the repair group had a much lower incidence of preoperative atrial fibrillation, and if that persisted postoperatively, the lower incidence of atrial fibrillation in the repair group would be associated with a lower incidence of thromboemboli. Thus the prosthesis in the other group would not necessarily be the cause of their higher thromboembolic complication rate.

Dr Yau. Absolutely. As you noted, the incidence of preoperative atrial fibrillation in the patients undergoing mitral repair was about 32% and was significantly higher, about 66%, in the patients undergoing replacement with a mechanical valve. We did, in fact, in the Cox regression analysis for late thromboembolic events enter preoperative atrial fibrillation and preoperative cerebrovascular events into that model, but they did not emerge as statistically significant predictors of late outcomes.