Recurrent mitral regurgitation after repair: Should the mitral valve be re-repaired?

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Objective: We sought to evaluate the clinical and echocardiographic outcomes of reoperation for failed mitral valve repair.

Methods: One hundred forty-five patients with recurrent mitral regurgitation after primary mitral valve repair of degenerative leaflet prolapse underwent mitral valve reoperations between January 1, 1970, and January 1, 2005. The mean age was 66 years, and 102 (70%) were men.

Results: The mean duration from initial repair to reoperation was 4.1 years (standard deviation = ± 5.1 years). Indications for reoperation were regurgitation alone (n = 109 [75%]), hemolysis (n = 27 [19%]), obstruction from systolic anterior motion (n = 3 [2%]), endocarditis (n = 3 [2%]) and stenosis-other (n = 3 [2%]). New pathology was found in 80 (55%) patients, and failure of the initial repair was found in 61 (42%) patients. The mitral valve was re-repaired in 64 (44%) patients and replaced in 81 (56%) patients. Early operative mortality was similar after re-repair and replacement (1.6% vs 4.9%, $P = .38$). Independent predictors of improved survival on multivariate analysis were mitral re-repair (hazard ratio = 0.44, $P = .03$), younger age (hazard ratio = 1.06, $P = .001$), and an operative indication of mitral regurgitation alone (hazard ratio = 0.31, $P = .005$). Seven patients had a third mitral operation (all replacements), 6 after re-repair and 1 after replacement. At last follow-up echocardiogram (n = 96), ejection fraction was greater ($P < .001$) and left ventricular end-systolic dimension was smaller ($P = .009$) in patients undergoing re-repair compared with values in those undergoing valve replacement.

Conclusion: Recurrent mitral regurgitation after prior repair is frequently caused by new valve pathology. Mitral re-repair is performed in almost half of patients and is associated with superior survival, improved ejection fraction, and greater regression in ventricular dimension compared with valve replacement.

Mitral valve (MV) repair is the standard of care for the correction of significant mitral regurgitation (MR) caused by degenerative valve disease.¹⁻⁵ Valve repair is associated with superior survival and equivalent, if not better, durability compared with valve replacement. MV reoperation after primary repair has been reported to occur with a linearized hazard rate of 0.5% to 1.5% per year.⁶ There has been hesitance to perform a second mitral repair because

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Abbreviations and Acronyms

AL	= anterior leaflet
LV	= left ventricular
MR	= mitral regurgitation
MV	= mitral valve
PL	= posterior leaflet

of concern that patients with a failed initial repair might have poor tissue quality, limiting the durability of re-repair. Also, there has been speculation that secondary MV repair might have greater risk than valve replacement.

Little is known regarding the effects of re-repair versus replacement on survival, durability, and normalization of left ventricular (LV) size and function. The objective of this study was to examine patients presenting with recurrent regurgitation after primary MV repair of leaflet prolapse, to analyze causes of technical failure after the first repair, and to determine factors predicting survival and durability after reoperation.

Patients and Methods

This was a retrospective review of patients undergoing surgical correction (repair or replacement) of recurrent MV regurgitation after primary MV repair for regurgitation caused by degenerative leaflet prolapse. The study was reviewed and approved by the Mayo Clinic College of Medicine Institutional Review Board. The need for individual patient consent was waived because relevant identifiers were not included in the database. Included were those having concomitant coronary bypass (coronary artery bypass grafting) or closure of a secundum atrial septal defect/patent foramen ovale. We excluded patients who declined involvement in clinical research, those who underwent conversion to replacement at the time of the initial operation, and those who had an initial diagnosis of MR caused by congenital or ischemic heart disease or other forms of cardiomyopathy. Also excluded were patients with significant MV stenosis or those with endocarditis causing leaflet defects/subvalvular abscess at the time of primary MV repair.

Between January 1, 1970, and January 1, 2005, a total of 145 patients underwent mitral reoperations for recurrent MR at Mayo Clinic Rochester. One hundred seven patients had their first MV repair at Mayo Clinic Rochester (5% of 2122 patients undergoing primary MV repair for degenerative leaflet prolapse during the study interval), and 38 had their first operation elsewhere. A total of 64 (44%) patients underwent MV re-repair, and 81 (56%) underwent replacement of the previously repaired valve. The mean interval between the initial operation and reoperation was 4.1 ± 5.1 years. The date of reoperation occurred within the first 3 months after the primary repair in 26 (18%) patients. A total of 6 patients undergoing re-repair and 1 undergoing replacement had a third mitral operation (second mitral reoperation). All 7 of these had valve replacement. The indications for reoperation were recorded from preoperative clinical assessments and echocardiograms. Findings at the time of the operation were determined from a review of the operative notes. The mean duration of follow-up in survivors was $3.3 (\pm 4.1)$ years.

Surgical Procedure

Both the indications for primary MV repair and the surgical techniques used evolved over the 35-year period. During the third decade of the study (1990s), we adopted a strategy of earlier primary MV repair for regurgitation caused by leaflet prolapse on the basis of quantitative echocardiographic criteria.² The most frequent lesion for which patients underwent initial surgical correction of MR at Mayo Clinic was isolated posterior leaflet (PL) prolapse of the middle scallop caused by either chordal elongation or chordal rupture. The most common method of correction during the initial operation was triangular resection and suture repair of the involved portion of the PL supplemented by a standard-length (63-mm) flexible posterior annuloplasty band.^{7,8} During the first 2 decades of the study, anterior leaflet (AL) prolapse was corrected by means of chordal shortening, chordal transfer, or commissural annuloplasty. More recently (after 1990), we used artificial polytetrafluoroethylene* neochordae⁸ for repair of AL prolapse and, in selected patients, leaflet plication. Indications for reoperation were recurrent MR with or without associated complications. Techniques used for MV re-repair were generally similar to those used in primary MV repair.

Statistical Analysis

Group statistics were expressed as means $\pm$ 1 standard deviation. Categorical variables were compared between groups by using the χ^2 test for independence or the Fisher exact test. Two-sample *t* tests were used to compare continuous variables between groups. Survival and reoperation end points were estimated by using the Kaplan-Meier method (actuarial). Multivariate models to identify potential risk factors for these end points were investigated by using Cox proportional hazards and were constructed with stepwise selection techniques. *P* values of .05 or less were considered significant.

Results**Baseline Characteristics**

Baseline characteristics of patients are presented in Table 1. Patients undergoing MV replacement were slightly more symptomatic and had greater left atrial dimensions before reoperation than patients undergoing valve re-repair. All other aspects of the preoperative risk profiles were similar comparing those who underwent MV re-repair versus those who underwent prosthetic replacement.

Indications for Reoperation

The indications for a second MV procedure are shown in Table 2. A total of 109 (75%) patients underwent reoperations for significant regurgitation alone. The remaining factors responsible for a decision to reoperate were hemolysis in 27 (19%), dynamic outflow tract obstruction caused by systolic anterior motion of the anterior mitral leaflet in 3 (2%), endocarditis in 3 (2%), MV stenosis in 1 (1%), and "other" in 2 (1%). Patients were more likely to have valve replacement at reoperation if the preoperative indication

*Gore-Tex neochordae, registered trademark of W. L. Gore & Associates, Inc., Newark, Del.

TABLE 1. Baseline characteristics

	Re-repair	Replacement	P value
N	64 (44%)	81 (56%)	
Age (y)	64 ± 13	67 ± 11	.24
Sex			.15
Male	49 (77%)	53 (65%)	
Female	15 (23%)	28 (35%)	
Atrial fibrillation	7 (11%)	10 (12%)	.79
NYHA class			.01
I	7 (11%)	0 (0%)	
II	29 (45%)	40 (49%)	
III	25 (39%)	32 (40%)	
IV	3 (5%)	9 (11%)	
Preoperative MR (n = 132)			.20
Trivial	2 (3%)	1 (1%)	
Mild	3 (5%)	2 (3%)	
Moderate	14 (23%)	8 (11%)	
Severe	42 (68%)	60 (85%)	
Preoperative EF (%) (n = 136)	57 ± 12	56 ± 11	.23
Preoperative LVEDD (mm) (n = 109)	56 ± 7	57 ± 8	.47
Preoperative LVESD (mm) (n = 103)	37 ± 8	38 ± 8	.57
Preoperative LA size (mm) (n = 83)	56 ± 13	60 ± 12	.04
Decade >1990 vs <1990	57 (89%)	68 (84%)	.38

NYHA, New York Heart Association; MR, mitral regurgitation; EF, ejection fraction; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LA, left atrial.

was MR alone and were more likely to have re-repair if hemolysis was the cause. Of the 27 patients with hemolysis, the mean MR grade at reoperation was 3.4, and the mean interval from first operation to reoperation was 1.1 years.

Reoperation

The findings at reoperation are shown in Table 3. Overall, the surgically determined factor or factors contributing to recurrent MR included the AL in 65 (45%), the PL in 61 (42%), ring annuloplasty dehiscence in 42 (29%), and chordal rupture in 20 (14%). New valve pathology was determined to be the responsible cause of recurrent MR in 80 (55%) patients, and technical failure of the primary

TABLE 2. Indications for reoperation

Variable	Overall	Re-repair	Replacement	P value
Regurgitation alone	109 (75%)	40 (63%)	69 (85%)	.002
Hemolysis	27 (19%)	20 (31%)	7 (9%)	<.001
SAM obstruction	3 (2%)	2 (3%)	1 (1%)	.43
Endocarditis	3 (2%)	0	3 (4%)	.12
Other	2 (1%)	1 (2%)	1 (1%)	.87
Stenosis	1 (1%)	1 (2%)	0	.26

SAM, Systolic anterior motion of the anterior mitral leaflet.

TABLE 3. Findings at reoperation

Abnormal findings	Overall	Re-repair	Replacement	P value
Anterior leaflet	65 (45%)	27 (42%)	38 (47%)	.57
Posterior leaflet	61 (42%)	27 (42%)	34 (42%)	.98
Annuloplasty	42 (29%)	30 (47%)	12 (15%)	<.001
Chordal rupture	20 (14%)	7 (11%)	13 (16%)	.38
Mode of failure				
New pathology	80 (55%)	27 (42%)	53 (65%)	.005
Technical	61 (42%)	36 (56%)	25 (31%)	.002
Unclear	4 (3%)	1 (2%)	3 (4%)	.43

repair was most responsible in 61 (42%) patients. The cause of MR at reoperation was unclear in 4 (3%) patients. Patients having MV re-repair were more likely to have a specific anatomic cause for recurrent MR identified by the surgeon at reoperation, such as annuloplasty dehiscence or technical failure of the prior repair. Discovering new pathology was associated with a tendency to replace the MV.

At reoperation, 64 patients underwent MV re-repair, and 81 underwent valve replacement. During re-repair, a modification of the annuloplasty was carried out in 52 of 64, an AL procedure was performed in 33 of 64, and the PL was addressed in 32 of 64. Concomitant procedures were performed in 38 patients and included coronary artery bypass grafting in 14 (10%), tricuspid valve repair in 12 (8%), aortic valve replacement in 8 (6%), the Cox maze procedure in 4 (2.8%), atrial septal defect closure in 1 (0.7%), and patent foramen ovale closure in 1 (0.7%). The operative mortality after valve replacement was 4.9% (4 patients), and that after mitral re-repair was 1.6% (1 patient). Where data were available on predismittal echocardiography, 97% (59/61) of patients undergoing re-repair and 98% (64/65) of those undergoing valve replacement had no greater than mild MR.

Univariate and Multivariate Predictors of Late Mortality

Univariate analysis and multivariate models were constructed to determine the influence of preoperative and intraoperative variables on late mortality (>30 days) after reoperation. The univariate survival advantage associated with re-repair is shown in Figure 1. On multivariate analysis, 3 independent factors were associated with improved survival: MV re-repair, younger age, and the preoperative indication of pure MR (Table 4).

Univariate Predictors of Third Mitral Operation

A total of 7 patients (6 undergoing re-repair and 1 undergoing replacement) underwent a third MV operation at a mean of 5.7 years after the second operation (operative mortality, 0%). The actuarial survival free of subsequent reoperation after the first MV reoperation was 93% at 5

Figure 1. Kaplan-Meier probability of late survival (>30 days or after discharge from the hospital) after mitral valve re-repair or replacement at reoperation for recurrent mitral regurgitation. The late survival associated with mitral valve re-repair is greater than that seen after mitral replacement (mortality hazard ratio for re-repair, 0.49; $P = .05$ vs valve replacement).

years overall. The 5-year actuarial freedom from reoperation after re-repair was 83%, and the linearized risk of reoperation was 3.4% per year. The only univariate predictor of a third MV operation was performance of an AL procedure at the time of re-repair. The number of events was insufficient to perform multivariate modeling.

Follow-up Data

At the date of last contact, 43 (100%) of 43 patients undergoing re-repair were in New York Heart Association class I/II versus 31 (91%) of 34 after replacement. The final available follow-up echocardiograms (between 1-5 years, $n = 96$) were analyzed to assess LV size and function after MV re-repair versus replacement. These were performed a mean of 946 days after re-repair and 915 days after valve replacement (Table 5). Patients undergoing MV re-repair at the second operation had significantly better ejection fraction and smaller LV end-systolic dimension during follow-up compared with those undergoing MV replacement.

Discussion

This is the first study to compare the outcomes of MV re-repair versus replacement for recurrent MR after primary repair of degenerative MV prolapse. Our results indicate that reoperation was most often required for recurrent regurgitation and that the cause was similarly distributed between failure of the previous repair and new valve pathology. MV re-repair was performed in just under half of

all patients in our series. Importantly, we found that re-repair is clearly beneficial, conveying improved survival, along with better recovery of LV function and greater LV regression, compared with valve replacement. If MV re-repair is technically possible, it should be the favored choice at reoperation.

The indications for reoperation and intraoperative findings both influenced the choice of surgical procedure. Significant MR alone was the most frequent indication for reoperation after initial MV repair, as has been shown in prior reports,⁹ and was associated with a tendency to perform valve replacement over re-repair (85% vs 63%, $P = .002$). Technical reasons for failure of the primary MV repair were discovered to be important in 42% of patients, which more frequently led to be re-repair (56%) over replacement (31%, $P = .002$). Discovering a specific anatomic cause for disruption of the prior repair and, specifically, annuloplasty dehiscence was more prevalent in those undergoing re-repair (47% vs 15%, $P < .001$). This observation underlines the fact that the ability to discern an anatomic reason for failure of the first repair is important if a second repair is to be considered. It also reaffirms the importance of reliable intraoperative and pre-discharge echocardiography to identify those with early repair failure so that surgical reintervention can be undertaken before leaving the hospital, as others have suggested.⁹ Finding new valve pathology at reoperation might have influenced the decision to replace the MV (42% vs 65%, $P = .005$). This tendency might have been associated with a concern that disease progression in remaining leaflet tissue would increase the likelihood of a third MV operation.

The observation that roughly half of all patients (55%) have new MV pathology causing recurrent MR after primary repair differs from the reports of others, who have suggested that technical failure was the most common finding at reoperation.^{9,10} It is possible that the prevalence of rheumatic and ischemic MV disease in these studies might have influenced both the cause of repair failure and the stated preference for valve replacement at reoperation.

The incidence of hemolysis after MV repair is unknown, and the optimal clinical management is still a topic of debate. In this series hemolysis as a cause for reoperation after initial MV repair of degenerative leaflet prolapse was unusual (19%) but was also a factor favoring re-repair. A recent report suggested that 90% of patients presenting with hemolysis after prior MV repair required MV reoperations for surgical correction.¹¹ The operative techniques used to eliminate the regurgitant jet and to prevent the recurrence of hemolysis while preserving the native MV have been described elsewhere.¹¹⁻¹⁴ Several groups have published their experience with reoperation for this condition,^{11-13,15,16} and there has been a suggestion that MV replacement might be

TABLE 4. Univariate and multivariate predictors of late mortality

Variable	Univariate HR	Univariate <i>P</i> value	Multivariate HR	Multivariate <i>P</i> value
Age	1.06	.002	1.06	.001
Male sex	0.58	.10		
Atrial fibrillation	1.43	.34		
NYHA class	1.55	.06		
Diabetes	4.36	.05		
CHF	2.27	.04		
Decade >1990 vs <1990	0.85	.65		
Indications				
MR alone	0.62	.19	0.31	.005
Hemolysis	1.28	.55		
Findings				
MR alone	3.30	.10		
Anterior leaflet	0.65	.21		
Posterior leaflet	0.81	.54		
Annuloplasty	1.11	.76		
Ruptured chordae	0.63	.38		
Mode of failure				
Technical	0.75	.38		
New pathology	1.15	.65		
Reoperation thromboembolism	2.94	.08		
Reoperation hemolysis	1.36	.41		
Follow-up MR grade	0.82	.44		
Preoperative EF (%)	1.01	.62		
Preoperative LVEDD (mm)	1.01	.64		
Preoperative LVESD (mm)	1.03	.17		
Preoperative LA size (mm)	1.01	.47		
Time to reoperation	1.00	.91		
Re-repair vs replacement	0.49	.05	0.44	.03
CABG	2.22	.08		

NYHA, New York Heart Association; CHF, congestive heart failure; MR, mitral regurgitation; EF, ejection fraction; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension; LA, left atrial; CABG, coronary artery bypass grafting.

the treatment of choice.¹¹ Our current data indicate that re-repair for hemolysis is effective and is not a risk factor for a third mitral operation.

Operative mortality in our study was 1.6% after re-repair and 4.9% after replacement. El Asmar and colleagues¹⁰ found an overall reoperative mortality of 1.4% in a series in which 85% of repair failures were followed by replacement. Gillinov and associates⁹ studied a mixed population of patients (degenerative, 59%; rheumatic, 20%; ischemic, 16%;

endocarditis, 4%; and congenital, 1%), reporting an early mortality after re-repair of 8.6%. A similar operative risk, 8.9%, was reported by Niederhauser and coworkers.¹⁷ Our study examines a cohort of patients who are relatively homogeneous. In limiting our review to degenerative MV disease, we have attempted to focus on the actual reoperative risk associated with re-repair versus replacement in this population.

MV re-repair was an independent predictor of improved late survival in our study, despite similar preoperative risk profiles to those undergoing MV replacement. Moreover, re-repair led to superior recovery of ejection fraction and more complete regression of LV dimensions toward normal, as has been shown after primary valve repair.⁶ It has also been demonstrated elsewhere that greater LV function and smaller LV dimensions after primary MV surgery are associated with a survival advantage during long-term follow-up.¹⁸⁻²⁰ This might explain the observed benefit in late survival that we have seen in our current series after re-repair. The findings in this report challenge the assumption that MV

TABLE 5. Follow-up echocardiographic data

Variable	Re-repair	Replacement	<i>P</i> value
Time to last echocardiogram (d)	946	915	.65
EF	55 (39)	44 (35)	<.001
LVEDD	55 (43)	57 (32)	.30
LVESD	38 (36)	44 (29)	.009

EF, Ejection fraction; LVEDD, left ventricular end-diastolic dimension; LVESD, left ventricular end-systolic dimension.

replacement is the conservative choice for correction of recurrent MR. The previously established benefits of primary MV repair on survival, LV size, and function persist after re-repair.

Although the durability of MV replacement was slightly greater than that of re-repair at reoperation, the difference between the 2 groups could not be statistically differentiated. Surgical correction of the AL during MV re-repair was performed in 33 (52%) of 64 patients undergoing re-repair and was the only univariate risk factor to be associated with the need for a third mitral operation. Because the majority of the procedures in this report were carried out between 1980 and 2000, many patients had correction of AL prolapse with methods that are now known to be less durable than chordal replacement.^{5,21} Our recent results concur with those of others who have found no significant difference in MV reoperation rates after primary repair of isolated PL prolapse versus that involving the AL in the current era.^{6,22,23} The improved durability of primary AL repair will also likely influence the results of re-repair in the future.

Limitations

This is a retrospective review and is associated with the traditional limitations. It is possible that we selected a group of patients who were more likely to be followed for some distinguishing feature. Although we captured all patients presenting for reoperation at our institution, regardless of the location of the first repair, it is conceivable that this group differs from those who had reoperations elsewhere. Finally, because of the length of the period over which this study was completed, there are variations in the frequency and extent of echocardiographic follow-up.

Clinical Implications

Delaying surgical reintervention for recurrent severe MR after primary MV repair can be deleterious. Identification of MV-related hemolysis, an anatomic abnormality of the prior repair, or technical failure at reoperation all increase the likelihood of performing a second MV repair. Just under half of all patients in this study underwent re-repair at reoperation. The proportion of those who have anatomy amenable to re-repair is likely higher than this. Re-repair frequency might be limited either by surgeon bias or patient concern regarding a third operation. Whereas patients have previously been counseled to accept MV replacement as the more conservative choice after failure of primary MV repair, the survival advantage, durability, and improvements in LV size and function associated with re-repair should motivate clinicians to offer this opportunity to patients.

Conclusions

Mitral re-repair is currently performed in almost half of all patients with recurrent MR after failed primary repair of degenerative MV disease. The reoperation rate after re-repair is 3.4% per year, and will likely improve as the results of more recent valvuloplasty techniques for AL repair become available. MV re-repair should be offered as the preferred approach to preserve longevity along with LV size and function at reoperation.

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Discussion

Dr A. Marc Gillinov (Cleveland, Ohio). In this excellent and very clear review, you describe the treatment for recurrent MR after a primary operation for degenerative disease or prolapse, and in your series you re-repaired nearly half of the valves. This is an important topic because although failure of a repair is uncommon, as the number of repairs increase, we will see these cases. I have 3 questions for you.

The first concerns operative technique. When you go in for a re-repair, do you generally find it necessary to take down the entire previous operation and start over, or do you use a more directed approach and fix whatever appears to be broken?

Dr Suri. Thank you, Dr Gillinov. At the time of re-repair, the pathology found by the operating surgeon influences the valvuloplasty technique used. Generally, we address the specific anatomic feature requiring correction. Additionally, we always ensure that we maintain adequate posterior annular support by anchoring a posterior annuloplasty band between the left and right fibrous trigones.

Dr Gillinov. My second question concerns any potential changes during the time frame of this study, which spans more than 3 decades. Has there recently been a greater tendency to re-repair the valves as the surgeons have gained more experience?

Dr Suri. That is a good question. There is no doubt over the course of the 35-year study that trends and techniques have evolved. In the 1990s, we benefited from the data from yourself and others using new valvuloplasty techniques for correction of AL prolapse, including the placement of artificial polytetrafluoroethylene neochordae. As the comfort level with these techniques increased, so also did the rate of MV re-repair. That said, the frequency with which one encounters a patient with a re-repairable MV is still greater than the number that currently undergo re-repair. The reason for this can be broadly divided into 2 categories: patient-related factors and surgeon-related factors. Some patients present to the office with recurrent MR after failure of initial repair, convinced that they require replacement either because they have been counseled elsewhere to request it or they believe for some reason that because the first repair failed, replacement might be the more conservative option. We suggest, based on the data we present here today, that patients should be assured that there are distinct benefits of re-repair that warrant its attempt at reoperation.

Regarding surgeon-related factors, there is no doubt that the volume of primary MV repair that an individual surgeon performs influences how patients are treated when presenting with recurrent MR. This affects how a patient is counseled preoperatively by the surgeon, the breadth of re-repair techniques attempted intraopera-

tively, and the outcomes attained. The final adjunct important to a surgeon's comfort with re-repair of the MV is the availability of reliable intraoperative echocardiography to ensure adequate quality control of re-repair before leaving the operating room. In summary, as we accumulate data confirming the clear advantages of mitral re-repair, patients, referring physicians, and surgeons will be comforted by the realization that the benefits of primary MV repair persist after re-repair.

Dr Gillinov. I have one final question. Patients undergoing a re-repair had superior clinical outcomes. Why do you think this is? Might it be related to preservation of the subvalvular apparatus with repair versus replacement?

Dr Suri. There is certainly good evidence, as others have discussed already at this meeting, regarding the benefit of preservation of the mitral subvalvular apparatus in the maintenance of left ventricular function and regression of left ventricular dimensions toward normal values. The philosophy at our institution is to maintain chordal integrity whenever possible. Over the 35-year period of this study, 93% of patients undergoing replacement for whom data were available had some degree of chordal preservation. Nonetheless, the extent of chordal preservation during valve repair might be more complete, and this might partially explain the advantages in survival and ventricular recovery over valve replacement.

Dr Gillinov. Thank you.

Dr Paul Kurlansky (Miami, Fla). I want to congratulate you on a very clear and beautifully presented study, as we have come to always expect from the Mayo Clinic. I have a couple of brief questions.

You mentioned that approximately half of the re-repairs were due to technical problems and the other approximately half to new pathology, with a certain amount being caused by ring dehiscence. I was wondering whether you could help teach us—even though understanding that this represents only 3% per year, so it is a relatively small amount—but what you could teach us about what you learned from the technical problems that you encountered. Is there anything that you learned that could help us to prevent problems the first time around, specifically in regard to the technical issues that you confronted at re-repair?

Second, I wonder whether there is any technical advice that you can give us regarding ring dehiscence, ways that might predispose to it and ways that might help prevent it.

Third, regarding new pathology, was this an issue of progressive valve degeneration in the setting of degenerative disease or was this a new pathology, a new ischemic MR in a patient who did not have ischemia at the time of the original operation, is this progression of rheumatic disease, et cetera? Were you able to tease any of that out?

Thank you.

Dr Suri. Thank you. Those are important questions. It was a privilege to review these data because it was a true historic summary of the evolution of MV repair. Reviewing the revolutionary techniques used by Professors Kirklin and McGoon in their early operative notes, it was fascinating to follow how initial attempts were modified to attain the current permanence of MV repair. As an example, the initial technique for correction of segmental leaflet prolapse involved what was called a "McGoon plication." However, it was subsequently discovered that merely

plicating redundant prolapsing leaflet tissue led to excessive bulk, which might have predisposed to early senescence of the repair. Understanding that a plication essentially removed a triangular-shaped portion of the leaflet from the coaptation area led to the evolution of a simple triangular resection of the prolapsing segment, followed by suture reconstruction instead. Because the leaflet redundancy caused by chordal or rupture is most problematic near the coaptation point, resection toward the base of the leaflet toward the annulus was determined to be unnecessary. Similarly, early use of bicommissural annuloplasty stitches was often found to be ineffective at reoperation, allowing progressive dilation of the posterior annulus. This observation led to the practice of anchoring posterior annuloplasty bands in the fibrous skeleton of the heart between the right and left trigones. Those were some of the interesting lessons from the early days of MV repair at the Mayo Clinic.

Regarding the current modes of failure, I do not think that there are necessarily any novel insights that we could offer beyond those that are currently published. Even with annuloplasty band placement, one factor that was common in those with either annuloplasty dehiscence or progressive annular dilation was the failure to attain or maintain anchoring of the band of the initial repair in the fibrous trigones.

The issue of new pathology was also interesting. There was a proportion of the population in our study in whom repair failure could be accounted for by progression of Barlow's-type myxomatous leaflet degeneration. However, there were certain patients in whom it seemed that correction of the segmental prolapse might have led to the redistribution of forces and failure of adjacent leaflet segments. Even though a ruptured chord might not have been identified on a bordering segment initially, there were new ruptured chords identified at reoperation. It might be that structurally important cords of questionable integrity might be best addressed at the time of primary repair.

Finally, the issue of ischemic pathology leading to recurrent MR is worthy of further examination. In a study we presented earlier this year at the Society of Thoracic Surgeons, coronary artery bypass grafting turned out to be an independent predictor of reoperation after primary MV repair. Initially we were puzzled by this, but we hypothesize that there might be a component of evolving ischemic disease that is responsible for subsequent ischemic MR. This issue will be studied further in future studies.

Thank you for those questions.

Dr Khalid Rasheed (*Islamabad, Pakistan*). Congratulations on this very elegant presentation. You did mention that in most of your patients who needed MV replacement, you did preserve some of the subvalvular apparatus. Do you generally preserve the entire subvalvular apparatus or only that of the posterior mitral leaflet? Now that it has generally been shown that the entire annulus dilates, including the intertrigonal area, what were your reasons for not selecting a complete ring, either rigid or flexible?

Thank you.

Dr Suri. Those are good questions and probably extend beyond the time we have allocated today. Just quickly, the subvalvular apparatus was preserved to differing extents over the 35-year period, but in those for whom data were available, 93% had some degree of chordal preservation.

Regarding progressive dilation of the annulus, although there are new data suggesting that the anterior annulus might elongate between trigones, there is still debate regarding the category of patients at risk, along with the quantitative and qualitative elements of the phenomenon. Although we did not specifically measure the anterior intertrigonal distance, what I can say is that in the absence of leaflet pathology, recurrent MR at the time of reoperation was unusual in the setting of a posterior annuloplasty band that remained anchored between the left and right fibrous trigones.

Dr David H. Adams (*New York, NY*). Rakesh, it is just a follow-up to what we have just been talking about. I believe you said you use a standard-length 63-mm band in everyone, and then you said one of the reasons for failure was an inability to secure it at the trigones. Are you sure that part of the problem here is not a methodologic one? You do not have enough of a downsized annuloplasty? And we did not really have time to talk about it, but how many failures were caused by the annuloplasty being revised as part of the treatment?

Also, when you say you could not secure it at the trigones, maybe you did secure it and the anterior annulus did dilate. Is that possible, do you think?

Dr Suri. Thank you, Dr Adams. I appreciate your comments. Fifty-two of the 64 required some manipulation, placement, or replacement of an annuloplasty ring for differing reasons. Over the 35-year period, the most homogenous trend that we can speak to in terms of the discovery of an annular cause of recurrent MR at the time of reoperation was either the absence of a prosthetic annuloplasty device or finding that the initial annuloplasty band was not anchored into one or both fibrous trigones.

Regarding sizing, we most commonly use a standard 63-mm band for degenerative MV repair. We initially chose that size based on pathologic cross-sectional data derived from our institution in the 1980s, which demonstrated that the average posterior annular length between fibrous trigones in the normal human heart was 63 mm. Now while placing that band, there is a slight amount of tapering that is possible based on stitch width and the degree of cinching performed while tying the knots down. This allows us some adjustment for heart size and systolic anterior motion risk. Finally, I understand that there is still significant debate among centers regarding partial versus complete rings and ring size. As Dr Duran stated this morning, there might be—and I am offering this as a hypothesis—more controversy about bands and rings in purely degenerative MV disease than is necessary or warranted based on the science we have available. I merely offer that for consideration until we have the data from randomized studies to understand the concept further.