SURGERY FOR ACQUIRED HEART DISEASE

REOPERATION FOR FAILURE OF MITRAL VALVE REPAIR

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Background and Objective: Mitral valve repair is the procedure of choice to correct mitral regurgitation of all types. Up to 10% of patients who undergo mitral valvuloplasty require late reoperation for recurrent mitral valve dysfunction. To determine the causes of failed mitral valve repair, we examined the surgical pathology of patients who underwent reoperation for failed mitral valve repair. **Patients and Results:** From 1986 to 1994, 81 patients had 86 reoperations for recurrent mitral regurgitation after mitral valve repair. Mean age was 59.2 ± 1.4 years; 55 were men. Primary valve disease was degenerative in 48 patients (59%), rheumatic in 16 (20%), ischemic in 13 (16%), endocarditic in 3 (4%), and congenital in 1 (1%). Mean time interval between initial mitral valve repair and reoperation was 15.6 ± 2.5 months. Causes of repair failure were procedure-related (50 cases, 58%), valve-related (33 cases, 38%), or unknown (3 cases, 3%). Procedure-related valve failure was caused by suture dehiscence (21 cases), rupture of previously shortened chordae (19 cases), or incomplete initial correction (10 cases). Valve-related repair failure was caused by progressive primary valve disease (27 cases), endocarditis (5 cases), or extensive leaflet retraction (1 case). Repair failure was procedure-related in 70% of patients with degenerative valvular disease versus only 13% of patients with rheumatic valvular disease ($p = 0.0001$). At reoperation, mitral valve replacement was performed in 64 patients (79%) and repeat mitral valve repair in 17 (21%). **Conclusion:** We conclude that (1) most mitral valve repair failures are procedure-related in degenerative disease and valve-related in rheumatic disease; (2) rupture of previously shortened chordae is a common cause of late failure in patients with degenerative mitral valve disease; and (3) repeat mitral valve repair results in successful treatment for a minority of patients. (J Thorac Cardiovasc Surg 1997;113:467-75)

Mitral valve repair is the procedure of choice for mitral regurgitation (MR) of all etiologies.¹⁻⁷ Numerous studies have documented that mitral

valve repair performed by standardized techniques is reproducible and associated with low operative morbidity and mortality.¹⁻⁷ Advantages of mitral valve repair over mitral valve replacement (MVR) include lower operative mortality, better preservation of left ventricular function, and higher freedom from thromboembolism, anticoagulant-related hemorrhage, and endocarditis.^{4, 8-12} Given these advantages, an increasing proportion of surgeons is using mitral valvuloplasty to treat mitral insufficiency.

Although the feasibility of mitral valve repair extends to 95% of patients with degenerative valvular disease and up to 75% of patients with rheumatic or ischemic valvular disease, nearly all reports describe patients who have required reoperation for recurrent mitral valve dysfunction.¹⁻⁷ The longest

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Table I. Repair techniques used at initial operation

Repair technique	No.	%
Annuloplasty	78	91
Carpentier	39	45
Pericardial	25	29
Cosgrove	5	6
Duran	5	6
Other	4	5
Resection of posterior leaflet	27	31
Resection of anterior leaflet	4	5
Chordal shortening	25	29
Chordal transfer	12	14
Débridement	9	10
Commissurotomy	8	9
Leaflet patching	4	5
Suture repair of cleft leaflet	2	2

follow-up to date is reported by Deloche and associates,¹ with a 15-year actuarial freedom from reoperation of 87.4% after mitral valvuloplasty. Thus reoperation to treat recurrent MR is a significant late morbidity associated with mitral valve repair.

Although the incidence of repair failure and risk factors for recurrent valve dysfunction are well documented, little data exist concerning the mechanisms of recurrent mitral valve dysfunction after mitral valve repair or success of subsequent treatment modalities. Such information could aid in the selection of patients and techniques for mitral valvuloplasty. This study was undertaken to determine the causes of failed mitral valve repair.

Patients and methods

Between January 1986 and December 1994, 81 patients had 86 operations at The Cleveland Clinic for recurrent mitral valve dysfunction after initial mitral valve repair for MR. Patients having unsuccessful mitral valve repair followed by MVR at the same operative procedure were not included in this analysis. Nineteen of the 81 patients had initial mitral valvuloplasty at another institution and were referred to The Cleveland Clinic for treatment of recurrent valve dysfunction. Mean age at the time of reoperation was 59.2 ± 1.4 years (range 18 to 79 years); 55 patients were men. The primary valve disease was degenerative in 48 patients (59%), rheumatic in 16 (20%), ischemic in 13 (16%), endocarditis in 3 (4%), and congenital in 1 (1%).

MR was the indication for initial valve repair in all 81 patients. Severity of MR was determined before the operation by echocardiogram or cardiac catheterization, or both.¹³ Before mitral valvuloplasty, all patients had grade 3+ or 4+ MR on a scale of 0 to 4+; mean degree of MR was 3.5 ± 0.1 . Mean New York Heart Association (NYHA) functional class was 2.7 ± 0.1 , with 57 patients (70%) in class III or IV. Left ventricular function was normal or mildly impaired in 62 patients.

At the time of reoperation, all patients had recurrent

Table II. Causes of failed mitral valve repair

Cause of failed repair	No.*	%
Procedure-related	50	58
Suture dehiscence		
Annuloplasty	15	17
Leaflet repair	6	7
Ruptured chordae (previously shortened)	19	22
Incomplete repair	10	12
Valve-related	33	38
Progressive disease	27	31
Endocarditis	5	6
Leaflet retraction	1	1
Unknown	3	3

*Results were tabulated for 86 procedures in 81 patients.

MR that was grade 3+ or 4+ (mean MR grade 3.4 ± 0.1). All 81 patients underwent reoperation for failed mitral repair at The Cleveland Clinic. Five patients underwent a third operation at The Cleveland Clinic after a second mitral valvuloplasty failed; thus there were 86 operations for failed mitral valve repair. All patients had echocardiograms before reoperation. The predominant cause of recurrent mitral valve dysfunction was determined by echocardiogram and the surgeon's findings at operation.

Techniques used at initial mitral valve repair are summarized in Table I. Seventy-eight of 86 operations included annuloplasty. Most annuloplasties were performed with either a Carpentier-Edwards ring (Baxter Healthcare Corp., Edwards Div., Santa Ana, Calif.) or a strip of glutaraldehyde-treated bovine pericardium.¹⁴ An average of 1.9 repair techniques were used in each patient. Forty-two patients had additional cardiac procedures: coronary artery bypass grafting in 24 patients, aortic valve repair or replacement in 13, and tricuspid valvuloplasty in 7. Intraoperative echocardiography was used to assess mitral valve repair in 56 patients (transesophageal echocardiography in 41; epicardial echocardiography in 13; transesophageal and epicardial echocardiography in 2). After mitral valvuloplasty, mean grade of MR by intraoperative echocardiography was 0.6 ± 0.1 ($p < 0.0001$ vs preoperative degree of MR).

Follow-up data were available for 72 of 74 hospital survivors (97%). Follow-up information was obtained during outpatient clinic appointments or by telephone interview with the patient or referring cardiologist, or both. Mean duration of follow-up was 45.9 ± 3.3 months (range 1 to 115 months). Late echocardiograms were obtained on all patients who underwent re-repair of the mitral valve.

Statistical analyses. Unless otherwise indicated, all values are expressed as mean $\pm$ standard error of the mean. Primary valvular disease and mode of failure were analyzed for their relationship to time to repair failure. When normality or variance homogeneity was questionable, results of nonparametric tests were compared with the analysis of variance. Nonparametric results were used when significant discrepancies occurred. For time to event analyses, Kaplan-Meier curves were generated. Fisher's exact test was generated to test for effect of primary valvular disease on cause of repair failure; to preserve

Table III. Causes of failed mitral valve repair and primary valve disease

Cause of repair failure	Primary valve disease* (%)				
	Degen (N = 48)	Rheum (N = 16)	Isch (N = 13)	Endo (N = 3)	Cong (N = 1)
Procedure-related					
Ruptured chordae (previously shortened)	36	0	8	0	0
Suture dehiscence	22	13	23	100	100
Incomplete surgery	12	0	23	0	0
Valve-related					
Progressive disease	22	74	31	0	0
Endocarditis	2	13	8	0	0
Leaflet retraction	2	0	0	0	0
Unknown	2	0	8	0	0

Degen, Degenerative; Rheum, rheumatic; Isch, ischemic; Endo, endocarditis; Cong, congenital.

*Results were tabulated for 81 patients; in patients having two operations for failed mitral valve repair only the initial procedure was tabulated.

independence between observations, only the first repair failure was analyzed in patients having two failed mitral repairs. A significance level of 0.05 was used for all testing.

Results

Mean time interval between initial mitral valve repair and reoperation was 15.6 ± 2.5 months (range 3 hours to 100 months). Ten patients underwent reoperation during the same hospitalization as the initial mitral valvuloplasty; in 7 of these, the operation was performed on an emergency basis for acute hemodynamic compromise.

Causes of failed mitral valve repair are depicted in Table II. Recurrent valve dysfunction was caused by either procedure- or valve-related factors. Overall, 50 of 86 failures were procedure-related, 33 were valve-related, and 3 were of unknown cause. Procedure-related causes of valve dysfunction included rupture of previously shortened chordae (19 cases), suture dehiscence of an annuloplasty ring (15 cases) or leaflet repair site (6 cases), and incomplete initial repair (10 cases). Incomplete initial repair was judged to be the cause of repair failure when MR was 2+ or greater by postrepair echocardiogram in the operating room or in the early postoperative period. Two patients with incomplete initial repair had systolic anterior motion of the mitral valve and left ventricular outflow tract obstruction as the cause of their MR. Valve-related causes of recurrent MR included progressive native valve disease (27 cases), endocarditis (5 cases), and excessive leaflet retraction (1 case).

Table III shows the relationship between mechanism of repair failure and primary valve disease. In patients with degenerative disease, 70% of valve failures were caused by procedure-related factors.

Table IV. Operative morbidity at reoperation

Event	No.	%
Reoperation for bleeding	7	8.1
Respiratory failure	5	5.8
Stroke	4	4.6
Sternal infection	3	3.5
Intraaortic balloon pump	3	3.5
Myocardial infarction	2	2.3
Gastrointestinal hemorrhage	2	2.3
Renal failure	1	1.2
Pulmonary embolism	1	1.2

Rupture of previously shortened chordae was the most common cause of recurrent MR in patients with degenerative valvular disease. In contrast, valve-related factors were responsible for recurrent MR in 87% of patients with rheumatic disease ($p = 0.0001$ vs patients with degenerative disease); progressive native valve disease was the predominant cause of repair failure in patients with rheumatic valvular disease, and this was characterized by leaflet fibrosis and retraction. In patients with ischemic MR, recurrent MR developed for a variety of reasons; procedure-related failure occurred in 54% of patients and valve-related failure in 39%. One patient who had mitral valvuloplasty for endocarditis had recurrent endocarditis in the repaired valve.

Timing of reoperation was dependent on the mechanism of repair failure. Mean time interval between initial mitral valvuloplasty and reoperation was 22.5 ± 4.5 months for patients with valve-related failure versus 12.8 ± 3.2 months for patients with procedure-related failure ($p = 0.002$). In addition, there was a trend toward longer time to reoperation in patients with rheumatic valvular dis-

Fig. 1. Kaplan-Meier curve of freedom from reoperation after repeat mitral valve repair ($n = 17$).

ease than in patients with degenerative valvular disease (median time intervals of 14 months and 6.5 months, respectively); however, this difference was not statistically significant ($p = 0.08$).

Reoperations included MVR in 64 patients (79%) and repeat mitral valve repair in 17 patients (21%). Associated procedures were performed in 18 patients and included coronary artery bypass grafting ($n = 7$), aortic valve repair or replacement ($n = 4$), tricuspid valve repair or replacement ($n = 6$), and closure of patent foramen ovale ($n = 2$). There were 7 operative deaths, for an operative mortality of 8.6%. All operative deaths occurred in patients undergoing MVR. Causes of operative mortality included sepsis (3 patients), left ventricular failure (2 patients), and stroke (2 patients). Significant operative morbidity is summarized in Table IV.

The 17 patients treated by repeat mitral valve repair included 10 patients with degenerative valvular disease, 3 patients with rheumatic valvular disease, 2 patients with ischemic valvular disease, 1 patient with endocarditis, and 1 patient with congenital mitral insufficiency. Six of these 17 patients subsequently required MVR (Table V). After repeat valve repair, 5-year freedom from reoperation on the mitral valve was $60.5\% \pm 13.3\%$ (Fig. 1). Of 10 patients with degenerative mitral disease undergoing a second mitral valve repair, only 1 required subsequent MVR. At mean follow-up of 56.8 ± 6.1 months, 11 patients with successful repeat mitral valve repair are in NYHA functional class I and none has had any valve-related morbidity; mean degree of MR in these patients is 0.5 ± 0.2 .

There have been 14 late deaths at a mean follow-up of 45.9 ± 3.3 months (range 1 to 115

Table V. Causes of failed mitral re-repair

Patient	Valve disease	Cause of first failure	Cause of second failure
1	Degenerative	Dehiscence (leaflet)	Dehiscence (leaflet)
2	Rheumatic	Progressive disease	Inadequate surgery
3	Rheumatic	Progressive disease	Progressive disease
4	Ischemic	Endocarditis	Chordal rupture*
5	Ischemic	Dehiscence (annuloplasty)	Unknown
6	Endocarditis	Dehiscence (annuloplasty)	Endocarditis

*Chordae were previously shortened.

months) (Table VI). Five-year survival was $84.6\% \pm 4.6\%$ (Fig. 2). Of 60 long-term survivors, 59 are in NYHA functional class I or II. Four patients who underwent MVR required repeat MVR, 2 for prosthetic valve endocarditis and 2 for structural deterioration of a bioprosthetic valve.

Discussion

Since the introduction of standardized techniques for mitral valve reconstruction by Deloche,¹ Carpentier,² Duran,⁷ and others,³⁻⁶ mitral valve repair has become the surgical treatment of choice for MR. Numerous retrospective studies have demonstrated significant benefits of mitral valve repair over MVR in patients with mitral insufficiency.⁸⁻¹² Deloche and associates¹ have stated that mitral reconstruction is feasible in 95% of patients with degenerative valvular disease, 70% with rheumatic valvular disease, and 75% with ischemic valvular disease. Others have confirmed this experience.^{5,7}

Although mitral valve reconstruction has several significant advantages over MVR, the incidence of late reoperation is equal after the two procedures.^{4,8-10} Mitral valve reconstruction is followed by a high initial instantaneous risk of valve failure and a subsequent low constant risk of late valve failure.^{5,11,12} Most authors report a 5-year freedom from reoperation of approximately 90%.^{4-6,15-17} Deloche and coworkers¹ report a 15-year actuarial freedom from reoperation of 87% after repair of insufficient mitral valves.

Several factors increase the risk of late reoperation after mitral valve repair. These include rheumatic disease, advanced myxomatous changes of both leaflets, chordal shortening procedures, failure to perform an annuloplasty, residual MR at the completion of repair, NYHA functional class III or IV, and performance of concomitant cardiac procedures.^{1,16-21} Most of these risk factors are related

Table VI. Causes of late death

Cause of late death	No. of patients
Congestive heart failure	3
Myocardial infarction	2
Operative mortality (redo MVR)	2
Pneumonia	2
Pulmonary embolism	1
Lung cancer	1
Unknown	3

either to the surgical procedure or to the patient's native valve disease.

Rheumatic disease is the most well-established risk factor for late failure after mitral valvuloplasty.^{1, 7, 11, 21-25} Deloche and associates¹ found that 15-year actuarial freedom from reoperation after repair was 76% in patients with rheumatic mitral disease versus 93% in those with degenerative mitral disease. Duran,²²⁻²⁴ Galloway,⁶ and others²⁵ have confirmed this observation. Duran and colleagues²²⁻²⁴ have emphasized that younger patient age and active rheumatic carditis are additional risk factors for reoperation.

Although series from Europe contain large proportions of patients with rheumatic disease, degenerative valvular disease is the most common cause of MR in North America.^{3, 15-17} Cohn and associates¹⁷ found that failure to perform a ring annuloplasty increased the risk of recurrent MR in patients with degenerative mitral disease. Recent data demonstrate that chordal shortening is an additional risk factor for recurrent operation in patients with degenerative mitral disease.¹⁸ For patients with anterior leaflet prolapse, Smedira and coworkers¹⁸ reported 5-year freedom from reoperation of 74% in patients having chordal shortening versus 96% in patients undergoing chordal transfer. Recurrent MR in patients having chordal shortening was caused by chordal rupture at the site of insertion into the papillary muscle. Others have also observed this complication associated with chordal shortening procedures.²⁶

Although risk factors for reoperation after mitral repair have been identified, few studies have systematically examined the precise mechanism of recurrent valve dysfunction. In a review of 72 patients undergoing late reoperation after mitral valve repair, El Asmar and coworkers²⁷ proposed that failure could be classified as surgeon-related or valve-related. In this experience, most repair failures were surgeon-related in degenerative disease and valve-related in rheumatic disease. Devising a similar

Fig. 2. Kaplan-Meier curve of late survival after reoperation for failed mitral valve repair ($n = 72$).

classification system, Duran⁷ proposed four possible causes of failure necessitating reoperation after mitral valve repair: wrong indication, technical error, instability of the repair technique, and progression of the disease process.

The purpose of this study was to identify the mechanisms of failed mitral valve repair. As noted previously, other authors have determined the incidence of recurrent mitral valve dysfunction and have identified risk factors for repair failure. This study of 86 reoperations for failed mitral repair is the largest reported series. Analysis revealed that the mechanism of valve failure could be classified as either procedure-related or valve-related, similar to the observations of El Asmar's group²⁷ and Duran.⁷ Unlike reports of these two authors, the majority of patients in this series had degenerative mitral valve disease. Procedure-related factors were the most common cause of recurrent valve dysfunction necessitating reoperation, confirming the findings of El Asmar and colleagues.²⁷ Rupture of previously shortened chordae was the single most common cause of valve failure in this group. Because of this observation, chordal transposition is favored over chordal shortening for correction of anterior leaflet prolapse.

Other important causes of valve failure in patients with degenerative disease included suture dehiscence, progressive native valve disease, and incomplete initial operation. Suture dehiscence occurred at sites of leaflet resection and at the annuloplasty ring. Dehiscence of the annuloplasty was unpredictable and occurred with all techniques. Progression of degenerative valve disease usually presented as elongation or rupture of previously untreated chor-

dae. Six patients with degenerative valvular disease had incomplete initial correction of MR. Three of these cases occurred before the routine use of intraoperative echocardiography. In the remaining 3 cases, intraoperative echocardiogram revealed residual MR that was 2+ or greater after repair. A previous study demonstrated that this is a risk factor for late reoperation.¹⁹

As in large series reported by Duran,²²⁻²⁴ Deloche,¹ Carpentier,² and others,^{25, 27} repair failure was attributable to valve-related factors in the majority of patients with rheumatic valves. Progressive rheumatic changes were the most common cause of reoperation in patients with rheumatic mitral disease. None of the patients had active rheumatic carditis at initial operation. At reoperation, most of these patients had progressive valve sclerosis with leaflet retraction. Others have noted similar post-rheumatic changes in the absence of active rheumatic carditis.²³⁻²⁵ Duran²²⁻²⁴ and others²⁵ demonstrated that mitral valve repair is less durable in those with rheumatic disease. Patients with rheumatic disease undergoing mitral valvuloplasty therefore require careful postoperative follow-up.

Ischemic mitral insufficiency has traditionally been a surgical challenge. Until recently, confusing classification schemes and an incomplete understanding of pathophysiology have made repair of such valves difficult.²⁸ Kay and associates²⁹ have documented the largest experience with mitral valvuloplasty for ischemic MR, and they report 94% freedom from reoperation at 10 years. In the current series, 13 patients with ischemic MR required reoperation after failed repair. Causes of failed repair were varied, with procedure- and valve-related factors accounting for nearly equal numbers of reoperations. Lack of a predominant cause of recurrent MR in this group may reflect the wide variety of pathophysiologic mechanisms responsible for ischemic MR.

Operative mortality for patients undergoing reoperation after failed mitral reconstruction was 8.6%, which agrees with the 8.8% operative mortality reported by Niederhauser and colleagues.³⁰ Seventy-nine percent of patients underwent MVR, and 21% had re-repair of their native mitral valves. Surgical treatment of recurrent MR after mitral valvuloplasty is controversial. El Asmar and associates²⁷ performed a second mitral valve repair in 15.3% of patients with failed mitral valvuloplasty. Others suggest that failed mitral valve repair should always be treated by MVR.³⁰ Of 17 patients having

a second mitral valvuloplasty, 6 required a third operation for recurrent mitral dysfunction. Of note, in only 1 of 10 patients with degenerative disease was a second mitral valve repair unsuccessful. These results suggest that select patients with degenerative disease can undergo successful repeat mitral valve repair when initial mitral valvuloplasty fails.

Mitral valve repair is the procedure of choice for most patients with mitral insufficiency. Although morbidity is low, previous studies demonstrate that after 5 years approximately 10% of patients will require reoperation for recurrent mitral valve dysfunction. As surgeons take a more aggressive approach toward repair and the number of mitral repair procedures increases, the incidence of unsuccessful mitral repair might be expected to increase. However, greater operator experience, echocardiographic monitoring, elimination of unsuccessful techniques, and decreasing prevalence of rheumatic disease should minimize the incidence of repair failure.

This study identifies mechanisms of failed mitral valve repair. In a group of patients with predominantly degenerative mitral valve disease, procedure-related factors were responsible for most cases of recurrent MR. This suggests that careful patient selection and precise application of surgical techniques can reduce the rate of late repair failure. By eliminating incomplete repair and abandoning the technique of chordal shortening, 34% of repair failures could be avoided. Routine use of intraoperative echocardiography to assess mitral repair should virtually eliminate incomplete correction as a cause for late reoperation. When reoperation is necessary after failed mitral repair, a small percentage of patients with degenerative disease can be treated successfully by repeat mitral valve repair.

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Discussion

Dr. Tirone E. David (Toronto, Ontario, Canada). The authors concluded that the principal cause of failure of mitral valve repair was related to the operation in patients who had MR as a result of degenerative disease; in those patients who had ischemic MR, the cause of failure was due to the progression of the valve disease. They point out that the rupture of previously shortened chordae tendineae was the principal cause of failure in patients with degenerative disease. We agree with their conclusions, and our experience is similar. What Dr. Gillinov did not mention is the frequency of these problems.

I have a few questions for Dr. Gillinov. First, how often does the mitral valve repair fail in each subgroup? How often do shortened chordae tendineae rupture in patients with floppy mitral valves? Finally, should chordal shortening be abandoned altogether if it is the cause of the failures in one third of the cases?

We reviewed our experience with 512 patients who had mitral valve repair from 1979 to 1993. In 309 the repair was done for degenerative disease of the mitral valve, and during a mean follow-up of 42 months, the failure rate was 3%. The actuarial freedom from failure was 96% at 10 years. All these failures occurred in the first year, indicating again that Dr. Gillinov is correct in stating that failure after repair of myxomatous mitral valve is largely related to technical problems. A regression analysis in this study showed that bileaflet prolapse and advanced myxomatous change in both leaflets were the two independent predictors of failure. In patients with ischemic MR, all failures in our experience were procedure-related. The failures occurred very early, in the first 7 months after the operation, indicating the complexities in repairing ischemic MR. Finally, like the authors' experience in rheumatic mitral valve repair, most of our failures were due to a progression of the disease, but they happened much later, after a mean time of 6 to 7 years, as opposed to the 25 months observed by Dr. Gillinov.

Dr. Alain F. Carpentier (*Paris, France*). I basically agree with the conclusions expressed in this paper, with some differences that I would like to underline. The 3.1% incidence of reoperation in your paper relates to the global number of valve operations. We would like to know what the incidence is in relation to the number of mitral valve repairs. The incidence in our experience accounted for 3.9% in a series of 3000 consecutive mitral valve repairs.

With regard to the annuloplasty, you have been using two basic types of approaches, and I notice that failure represented the third most frequent cause of reoperation; in our series, using exclusively remodeling annuloplasties, ring dehiscence was the least frequent cause of reoperation, with an incidence of 0.6%. Is this due to a higher incidence of failure in the group having pericardial reinforcement? If not, how do you explain this difference?

With regard to chordal rupture after chordal shortening, I do agree that this is one of the main causes for reoperation, actually the second cause for us after progressive valve disease. However, again, we should correlate the number of reoperations for this cause to the number of chordal shortenings performed. In our series chordal rupture accounted for only 4% of the chordal shortenings performed. Thus, before advising that this technique be abandoned, we must be sure that the alternative techniques can challenge this result over the long term. I cannot present a new approach, a new technique we have developed to minimize the drawbacks associated with the chordal shortening, because it is too difficult to explain without a slide.

Another new approach we have been exploring is valve repairs through a minithoracotomy with video assistance and peripheral extracorporeal circulation. Including the first operation, which took place last February, four patients have been operated on successfully. We were struck by the rapid recovery of these patients, but also by the greater difficulty of the operation; therefore we should be extremely cautious in the development of this approach.

Dr. Lawrence H. Cohn (*Boston, Mass.*). This manuscript is full of important details for those who do a lot of mitral valve repairs. I think the issue here is primarily how to handle the anterior leaflet of the mitral valve for the floppy mitral valve that has very elongated chordae.

We have also observed ruptured chordae in the papillary muscle trench. I think this complication is due to the fact that when the surgeon ties down the suture on the trench, he or she may actually tie the suture onto the chordal structure itself, leading to late rupture in these particularly fragile chordae. Therefore our current technique is to use polytetrafluoroethylene (PTFE) chordae, namely, the CV-5 Gore-Tex PTFE chordae.* We find that technique has several advantages. It is easy to use, and we think that the other chordae should also be left intact should anything happen. I am told that the symposium conducted at this meeting showed that there has been relatively minimal morbidity with the use of this technique now for 10 years. We believe that the PTFE chordae might be a little bit simpler and extremely reliable rather than the chordal transfer. What is the favorite technique

for chordal elongation shortening at the Cleveland Clinic at the present time?

Dr. Carlos G. Duran (*Missoula, Mont.*). This is an important paper because now that everybody agrees that repair is the preferred modality for the surgical treatment of mitral disease, it is good to hear about problems.

After listening to the paper, I still do not know what the incidence of failures was according to etiology and pathology. Apparently 59% of the cases were degenerative, but how many of these repairs failed and how many of the repairs for rheumatic disease? I am also surprised at the large number of ring dehiscences reported. Did you use different techniques and rings? In our experience this is a rare complication. My third question is whether you have tried to correlate these reoperations with the quality of repair immediately after the initial operation as evaluated by transesophageal echocardiography. Not all patients leave the operating room with the same degree of success.

I agree with the previous comments about the increased use of PTFE. I think it might prove to be the solution for anterior mitral prolapse. Finally, I have a comment that probably does not apply to this country but that we have found germane in the very young patient in the developing world. There is a very rapid rate of change in the echocardiographic left ventricular dimensions after surgery. After a few days these dimensions are reduced by nearly 30%, and consequently the shortened chordae to the anterior leaflet become too long, resulting in MR.

Dr. Gillinov. We would like to thank the discussants for all of their thoughtful comments. In response to Dr. David's questions, he and the other discussants asked what the incidence of repair failure is for each type of valve pathology at the Cleveland Clinic. We are currently conducting a long-term follow-up study to answer this question, so I do not yet have that data for you. Dr. David noted that the freedom from reoperation after repair for degenerative mitral valve disease among his patients is 96% at 10 years, which certainly is excellent and compares favorably with other reports. With the longest follow-up to date, Dr. Carpentier's group has reported a 13% incidence of reoperation after mitral repair at 15 years' follow-up; the incidence of reoperation is higher for those with rheumatic mitral disease.

Dr. David also asked about the frequency of chordal rupture after chordal shortening. In a study published in *The Journal of Thoracic and Cardiovascular Surgery* (1996; 112:287-92), we report a 74% 5-year freedom from reoperation after chordal shortening for anterior leaflet prolapse in degenerative mitral disease. In contrast, we found a 96% 5-year freedom from reoperation after repair of anterior leaflet prolapse using chordal transfer techniques, and this difference was statistically significant.

Finally, Dr. David asked if we would therefore recommend abandoning the chordal shortening technique. Like Dr. Carpentier, we do not recommend abandoning any particular technique until we have something that we can demonstrate is clearly superior. At 5 years we show that chordal transfer is superior to chordal shortening, and there is now emerging data that replacement of chordae with synthetic materials, like PTFE, may also be better.

Dr. Carpentier also asked what our incidence of repair failure was by pathology. We will have that information

*Gore-Tex prosthetic tissue, registered trademark of W. L. Gore & Associates, Inc., Elkton, Md.

soon. Similarly, I do not yet have the data for the incidence of annuloplasty dehiscence according to annuloplasty technique. Certainly annuloplasty dehiscence was an important cause of failure, causing 17% of repair failures in our series. Dr. Carpentier agreed that failure is more common after chordal shortening than after other techniques of repair for anterior leaflet prolapse. Again, I agree that we must test other methods before recommending the abandonment of this technique. We currently have no experience with minimally invasive approaches to mitral valve repair but certainly look forward to future presentations on this topic.

Dr. Cohn commented on treatment of anterior leaflet prolapse, agreeing with the other discussants. The precise cause of rupture of previously shortened chordae is not completely understood. It certainly has something to do with the fact that these are structurally abnormal, weakened chordae and is probably also related to difficulty in judging the appropriate length of these chordae. At The

Cleveland Clinic we favor chordal transfer over chordal shortening for correction of anterior leaflet prolapse.

Finally, Dr. Duran asked about our incidence of ring dehiscence, as it is an infrequent cause of late failure in his series. Approximately 50% of the patients in this series had a remodeling annuloplasty using a Carpentier-Edwards ring, and the second most common technique was posterior annular plication with a pericardial strip. The latter technique we have since abandoned because it is difficult to achieve a measured plication of the annulus. This technical difficulty may have contributed to repair failure in some patients. Dr. Duran asked us to relate the incidence of reoperation to the degree of MR at the completion of repair. We do have data to demonstrate that at the completion of repair, if MR is 1+ or 2+, the incidence of late reoperation is increased threefold over those who have trace or no MR at the end of repair. Thus incomplete initial repair is an important cause of repair failure.