# Several New Considerations in Mitral Valve Repair

J. Scott Rankin, Ricardo E. Orozco, Theodore R. Addai, Tracey L. Rodgers, Robert H. Tuttle, Linda K. Shaw, Donald D. Glower

Centennial Medical Center, Nashville, TN, Vanderbilt University, Nashville, TN, Duke University Medical Center, Durham, NC, USA

Background and aim of the study: A retrospective evaluation was made of a small personal series of patients undergoing mitral valve repair in order to address four contemporary questions: (i) What is the best method of achieving a stable repair in mitral valve prolapse?; (ii) How should patients with pure annular dilatation without prolapse or antecedent ischemia be categorized?; (iii) Are valve procedures in ischemic mitral regurgitation (MR) still associated with less satisfactory early and late outcomes?; and (iv) Is prophylactic amiodarone therapy safe and effective in reducing postoperative arrhythmias? Methods: Between 1993 and 2002, a total of 118

patients with non-rheumatic MR undergoing isolated mitral valve repair with or without coronary bypass was analyzed retrospectively: of these patients, 66 had prolapse (Group I), 21 had pure annular dilatation (Group II), and 31 had ischemic MR (Group III). All three groups routinely underwent Carpentier ring annuloplasty. Twenty-three patients in Group I were managed with leaflet resection and reconstruction (LRR), but in 1996 the technique for Group I was changed to uniform artificial chordal replacement (ACR) and no leaflet resection (n = 43). Also in 1996, prophylactic amiodarone therapy was first used routinely, and postoperative arrhythmia data were compared to those from prior patients. Baseline and outcome variables were assessed for each group and compared between the three groups. Survival data were evaluated using the Cox proportional hazards model.

Results: Significant differences in baseline characteristics were observed: Group II was predominantly

female; Group III more often experienced acute presentation; and Groups II and III had more comorbid disorders and left ventricular dysfunction (all p <0.01). ACR was highly successful for repair of prolapse, and no ACR patient exhibited significant residual MR or outflow tract obstruction. Operative mortality and morbidity were low in all groups, and ischemic etiology failed to be an independent predictor of early or late adverse outcome (p >0.10). Cox model analysis to nine years of follow up (median 4 years) identified only advanced age and number of comorbidities as influencing late mortality (both p <0.03). Over the follow up period, 8.7% of LRR patients required reoperation for valve failure due to late chordal rupture, whereas none of the ACR patients failed. Finally, prophylactic amiodarone significantly reduced postoperative arrhythmias (p = 0.03) with no observed complications, and also eliminated death due to arrhythmia.

Conclusion: Ischemic etiology may be diminishing as an independent risk factor in Group III, at least partially because of uniform valve repair. Group II comprised a distinct entity of females with higher comorbidity, and prophylactic amiodarone therapy seemed useful as a routine measure. Finally, ACR appeared to produce a stable repair in virtually all Group I patients, suggesting that prolapse might be appropriately managed with ring annuloplasty and uniform ACR. However, future studies are suggested for further consideration of these hypotheses.

The Journal of Heart Valve Disease 2004;13:399-409

At present, it is an established principle that mitral valve repair provides superior clinical outcomes for the treatment of non-rheumatic mitral regurgitation (MR). It is gratifying that a category of valve disease in which results were suboptimal three decades ago (1) has been transformed by valve repair into a relatively low-risk group (2-8). However, problems still exist; for

Address for correspondence:

J. Scott Rankin MD, 2400 Patterson Street, Suite 103, Nashville, TN 37203, USA

e-mail: jsrankinmd@cs.com

example, it may be difficult to apply valve repair in more complex prolapse pathologies, and with large posterior leaflet resections, left ventricular outflow tract obstruction (LVOTO) can occur (9). Late failure of repair has been a small but persistent occurrence, and procedures for repair of post-infarction ischemic MR, while providing better results than valve replacement, have been associated with poorer results (10-14). Finally, postoperative arrhythmias have been a persistent and bothersome problem since the beginning of mitral valve surgery.

In a recent small personal series of patients undergoing isolated mitral valve repair with or without coronary artery bypass (CAB), several relevant concepts seemed evident. Therefore, a study was designed to evaluate these issues using retrospective observational methods. Based on clinical impressions, the following hypotheses were generated: (i) Early and late outcomes of valve repair for prolapse are better with uniform artificial chordal replacement (ACR) than with leaflet resection and reconstruction (LRR); (ii) Patients with pure annular dilatation and no prolapse or antecedent ischemia are perhaps a distinct clinical entity with definable characteristics; (iii) Early and late results for ischemic MR continue to improve with routine application of mitral valve repair; and (iv) Prophylactic amiodarone therapy reduces postoperative arrhythmias.

## Clinical material and methods

#### **Patients**

For the purpose of this study, 'non-rheumatic MR' was defined as secondary to mitral valve prolapse, pure annular dilatation, or post-infarction ischemic MR. This definition excluded patients with rheumatic valve disease, bacterial endocarditis, or annular dilatation due to acute viral cardiomyopathy.

Over a nine-year period from August 1, 1993 to August 31, 2002, 118 patients had isolated mitral valve repair for 'non-rheumatic MR' with or without coronary artery bypass (CAB); these patients constitute the primary study group. Group I had valve repair for prolapse (n = 66), Group II had pure annular dilatation without prolapse or antecedent ischemic events (n = 21), and Group III had post-infarction ischemic MR (n = 31). Of the 118 patients, all had received confirmation of their clinical status by personal contact or telephone in September and October of 2002. Echocardiographic findings within two years of the follow up date were documented. Maximal follow up was nine years (median four years).

This study was approved by the Institutional Review Boards of Centennial Hospital (Nashville, TN, USA) and Duke University (Durham, NC, USA).

# Surgical approach

In Group I (n = 66), 23 patients underwent LRR, and 43 patients ACR. Between 1993 and 1996, quadrangular resections or 'sliding plasty' reconstructions (9) were performed for posterior leaflet prolapse. During this period, ACR was used selectively for anterior leaflet prolapse. In 1996, LRR was abandoned completely, and all subsequent patients (anterior *and* posterior leaflet prolapse) were treated with ACR from a papillary muscle to the prolapsing segment without leaflet resection. Single chords were placed in 33 patients (77%) and multiple chords in 10 (23%). In 29 patients (69%), chords were posterior only, in nine (20%) the chords were anterior only, and in five (11%) the chords were placed to both leaflets.

The 'adjustable' ACR technique employed in these patients had been refined during the late 1980s for repair of anterior chordal rupture, and in the present series the method was generalized to all types of prolapses. Artificial chords to the left aspect of the anterior or posterior leaflet were anchored to the anterior papillary muscle, and those to the right half of either leaflet were anchored to the posterior papillary muscle (as with normal anatomy). Pre-bypass imaging with transesophageal echocardiography (TEE) provided a good impression of the location of the prolapse, but after cardiotomy and retractor placement, the valve was tested by forcefully injecting cold saline across the

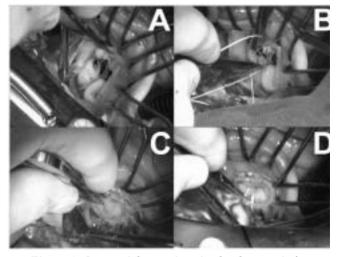


Figure 1: Sequential steps in mitral valve repair for generalized prolapse (Barlow's) using two artificial chords to the anterior leaflet and two to the posterior leaflet. A) Typical Barlow's valve showing generalized bileaflet prolapse with a superimposed chordal rupture. B) Two artificial chords have been placed through the pledgeted anchor suture in the anterior papillary muscle. C) The artificial chord has been passed through the free-edge of a posterior leaflet segment. D) Note complete competence of the valve at the conclusion of the procedure (saline has been injected into the ventricle).

leaflets and into the ventricle using a bulb syringe.

After confirmation of the prolapsing segments (Fig. 1A), pledgeted anchor sutures (horizontal mattress sutures of 4-0 Prolene buttressed with Teflon felt pledgets) were placed longitudinally into the papillary muscles, passing one arm through the fibrous tip. Each anchor suture was tied firmly, but care was taken not to injure the papillary muscle. A double-armed Gore-Tex vascular suture (2-0 or CV-3 is recommended) was then passed through the anchor suture but not tied (Fig. 1B). A metal clip was engaged across both arms of the suture behind the needles, and the Gore-Tex chord was placed into the ventricle in an organized way, with the needles last, so that they could be easily retrieved later. The annuloplasty ring then was implanted. All patients underwent a standard Carpentier ring annuloplasty (Carpentier-Edwards Classic, Model 4400), sized according to the intertrigonal distance and sutured to the mitral annulus with interrupted horizontal mattress sutures of 2-0 Polydek (Deknatel, Inc.) buttressed with supra-annular Teflon felt pledgets.

With the ring in its final position, the Gore-Tex chords were retrieved from the ventricle, and the clip was removed. Both needles of the double armed suture were passed into the free edge of the prolapsed segment, straddling the point of maximal prolapse. The sutures were then woven with two or three bites up through the coaptation surface to the line of coaptation, with near full-thickness bites being taken to prevent future disruption (Fig. 1C). If the leaflet tissue was fragile, then pericardial pledgets were used. The two arms of the suture were tied with a slip-knot on the atrial surface of the valve, just tight enough to bring the leaflet to the annular plane, and a clip was placed lightly across the knot. By leaving the Gore-Tex suture untied in the papillary muscle, both arms were free to move and assume equal length and tension. Further chords were retrieved (if multiple chords were placed), and similarly tied with slip knots after passage through their designated prolapsing segments.

Once the first estimate of leaflet position had been achieved, the valve was again tested by injecting cold saline across the leaflets, similar to techniques used previously (15-17). Almost uniformly, the first estimate of chordal length was too short or long, and the leaflets would not seat properly in the annular plane. The clip then was removed, the slip knot lengthened by 1 cm (or tightened if the length was too long), and another slip knot and clip were placed. The process was repeated until all segments seated well into the ring, and the valve was completely competent. Six to eight more knots then were tied *tightly* against the clip, the suture was cut, and the clip removed. Without tying against the clip, it was difficult to make the final knot tight, creating a possibility for the knot to untie later. At the

final testing, the leaflets should have seated well back into the ring, and the chordal length should have been adjusted just short enough to make the valve competent (Fig. 1D). After normal filling and outward displacement of the papillary muscles, the chords always tended to appear shorter by TEE, and this phenomenon was considered as chord lengths were determined. The 'adjustability' technique described above *always* allowed optimal chordal length and leaflet competence to be achieved, irrespective of the complexity of the prolapse.

In the most difficult generalized prolapse, such as with a Barlow's valve, pledgetted anchor sutures were placed in both papillary muscles, after which two Gore-Tex sutures were positioned in the anterior papillary muscle and two in the posterior papillary muscle. The anterior papillary chords were placed to the left aspect of both leaflets, and the posterior papillary chords to the right aspects (Fig. 1D). Prolapsing commissural cusps or intervening segments of the posterior leaflet were sutured to a segment supported by the ACR, using interrupted 4-0 Prolene sutures. This maximal procedure was used to repair even the most serious of generalized prolapsing valves. Pre- and postoperative echocardiograms of a patient with Barlow's syndrome undergoing this procedure are shown in Figure 2.

None of the 21 pure annular dilatation patients had antecedent infarctions, symptoms of ischemia, or regional wall motion abnormalities, and all received ring annuloplasty only. All of the 31 ischemic MR patients had prior infarctions (17 were acute), and 25 had ring annuloplasty only. Two additional patients

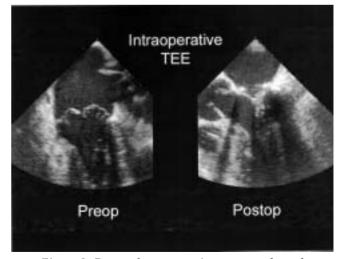


Figure 2: Pre- and postoperative transesophageal echocardiograms of a patient with Barlow's syndrome. Note the good postoperative leaflet position, and the large surface area of leaflet coaptation. The valve was completely competent on color flow mapping.

with acute ischemic symptoms had components of myxomatous posterior leaflet prolapse, and underwent LRR of the prolapsing posterior leaflet in addition to ring annuloplasty. Four other patients had ischemic MR associated with large posterior wall aneurysms. These patients were managed with transventricular mitral valve repairs, through the open ventricle, as the posterior wall aneurysm was resected.

All patients underwent a median sternotomy, standard cardiopulmonary bypass, and received St. Thomas' Hospital myocardial protection. Before 1996, arrhythmia management consisted primarily of combinations of procainamide and beta-blocking agents. Through 1996, a routine amiodarone protocol was developed, with intravenous loading for at least 12 h prior to surgery, multiple intraoperative bolus doses, and aggressive intravenous and then oral amiodarone therapy for at least six weeks postoperatively. If atrial fibrillation continued beyond six weeks, interval cardioversion was performed. Coumadin (warfarin) anticoagulation was used only in patients with atrial fibrillation. No Maze operations or atrial ablations were employed.

Positive comorbidity of chronic lung disease was defined as severe emphysema requiring daily pharmacological therapy; preoperative renal insufficiency was defined as a serum creatinine level  $\geq 1.5$  mg/dl; postoperative renal insufficiency was defined as a rise in serum creatinine >1.0 mg/dl; severe left ventricular dysfunction was an estimated ejection fraction (EF)  $\leq 0.35$ ; a stroke was a permanent neurological defect; reversible or transient neurological dysfunction used standard definitions; and operative mortality was defined as a patient dying in hospital within 30 days of surgery.

# Data analysis

Preoperative baseline characteristics were abstracted from hospital chart reviews, along with operative and postoperative data, and follow up telephone and echocardiographic information. After transfer of data into SAS files for analysis, the appropriate time-toevent variables were derived for subsequent survival analyses. Baseline characteristics were reported as percentages for discrete variables and compared using chi-square tests; continuous variables were described by median, 25th and 75th percentiles, with differences assessed by Kruskal-Wallis tests. Cumulative survival rates as a function of time after date of surgery were generated using the Kaplan-Meier method. The Cox proportional hazards model was used to examine both univariable and multivariable relationships between baseline characteristics and mortality. At the sample size and event rates observed in this study, a power analysis was performed to evaluate therapeutic differences that were statistically definable. A p-value < 0.05 for statistical results was considered to be significant.

### Results

Baseline characteristics for the three groups are presented and compared in Table I. Group III patients were slightly older, though the difference did not reach statistical significance. Approximately 95% of Group II patients were female, as compared to the minority in Groups I and III (p <0.0001). Co-morbidity was higher in Group II, and highest in Group III (p <0.0001). Severe left ventricular dysfunction (EF <0.35) occurred in a minority of prolapse patients, but was observed in 33% of annular dilatation patients (always global dysfunction), and in 39% of ischemic patients (p = 0.0013).

Table I: Pre- and postoperative patient data.

Variable	Group I (n = 66)	Group II (n = 21)	Group III (n = 31)	p-value
Preoperative				
Age (years)*	62 (55, 70)	60 (52, 64)	65 (56, 71)	0.2009
Female gender (%)	40.9	95.2	35.5	< 0.0001
Comorbidities (n)*	0 (0, 1)	1 (0, 2)	2 (1, 3)	< 0.0001
Severe LV dysfunction (%)	9.1	33.3	38.7	0.0013
Coronary disease (%)	27.3	23.8	100.0	< 0.0001
Acute presentation (%)	27.3	33.3	61.3	0.0050
Postoperative				
Complications (n)*	0 (0, 0)	0 (0, 0)	0 (0, 1)	0.1063
30-day mortality (%)	1.5	0.0	6.5	0.2540
Days to discharge*	7 (6, 8)	7 (5, 9)	7 (5, 11)	0.9060
Complications (n)*	0 (0, 0)	0 (0, 0)	0 (0, 1)	0.1063

\*Values are median and 25th, 75th percentiles.

Severe LV dysfunction =  $EF \le 0.35$ ; other definitions are given in the text.

Table II: Preoperative comorbidity.\*

Condition	Group I (n = 20/66)	Group II (n = 15/21)	Group III (n = 28/31)	Overall (n = 63/118)
Hypertension	9	7	4	20
Diabetes	4	4	4	12
Renal	1	4	6	11
Pulmonary	1	5	7	13
CVD	1	0	4	5
PVD	1	0	2	3
Acute MI	0	0	17	17
Abdominal	2	1	1	4
Reoperation	3	0	2	5

\*Patients often had more than one comorbidity (numbers of patients having comorbidity is given in parentheses).

CVD: Cerebral vascular disease; MI: myocardial infarction (precipitating hospital admission); PVD: Peripheral vascular disease; Reoperation: Previous cardiac surgery.

Definitions of other variables are given in the text.

Approximately one-quarter of prolapse and annular dilatation patients had asymptomatic coronary disease requiring bypass, whereas all ischemic patients had symptomatic coronary disease, prior infarctions, and regional wall motion abnormalities (p <0.0001). Around one-third of Group I and II patients were admitted to the coronary care unit with acute presentation (predominantly congestive heart failure), but almost two-thirds of ischemic patients presented acutely (combinations of acute infarctions and congestive heart failure) (p = 0.005).

Preoperative comorbidity is illustrated in Table II for each of the three groups. Over half of the patients had significant comorbidity, most commonly hypertension, severe chronic lung disease, diabetes, and renal insufficiency. Approximately half of Group III patients presented with an acute myocardial infarction; the remainder had congestive heart failure and/or chronic ischemia after a prior infarct. In total, 33 patients (28%) were aged over 70 years, and one patient was in his nineties (Table III). All patients but five had severe MR on pre-bypass TEE, and all but two had NYHA class III-IV congestive heart failure preoperatively. Twenty patients (17%) had atrial fibrillation preoperatively, and four ischemic MR patients had episodes of preoperative ventricular tachycardia.

Details of acute residual MR assessed by TEE at the conclusion of the surgical procedure are shown in Table IV. Only two of the 43 ACR patients had mild MR, whereas the incidence was higher with LRR. One LRR patient demonstrated LVOT which resolved with conservative management. No prolapse patient required prosthetic valve replacement after converting to uniform ACR in 1996. Acute residual MR rarely occurred in Groups II or III. Postoperative variables for the three groups are detailed in Table I.

The 30-day, in-hospital operative mortality was 2.5% overall (Group I, 1.5%; Group II, 0%; Group III, 6.5%), and was not significantly different among the three groups (p = 0.2540). Postoperative complications, which occurred in one-quarter of patients, and times to discharge were not different for the three groups (Table I). The one patient in Group I who died experienced thrombosis of the entire heart and circulation (documented by TEE) at 30 min after a successful valve repair, and was thought to have developed a hyperacute form of heparin-induced thrombosis syndrome. No patients in Group II died, and the two deaths in Group III were the result of postoperative sustained ventricular tachycardia in patients with documented preoperative monomorphic ventricular tachycardia secondary to prior infarctions. Both of these patients

*Table III: Distribution of advanced age* (≥70 *years*) *among patients.* 

Age (years)	Group I (n = 66)	Group II (n = 21)	Group III (n = 31)	Overall (n = 118)
70-79	10	3	12	25
80-89	6	-	1	7
≥90	1	-	-	1

underwent surgery before the routine use of high-dose perioperative intravenous amiodarone, and no patient has experienced postoperative death from arrhythmias since institution of the amiodarone protocol. Among the 92 patients managed with amiodarone (A), as compared to the 26 patients receiving procainamide (P) and beta-blockade, new postoperative arrhythmias (any atrial fibrillation (AF) or ventricular tachycardia) also were reduced significantly (A = 13%; P = 31%; p = 0.03). No serious complications of amiodarone therapy were observed in this series, and only one surviving patient was in AF at late follow up. However, acute conversion from preoperative AF was not improved with amiodarone (preoperative AF: A = 17%; P = 15%) (discharge AF: A = 11%; P = 12%), and excellent late conversion rates seemed to reflect persistent amiodarone therapy, late reversal of cardiac dilatation, and aggressive interval cardioversion protocols.

Approximately one-quarter of patients developed postoperative complications (Table V), most commonly pulmonary infections requiring intravenous antibiotics. Rates of transient renal insufficiency and stroke were low, as were those of other complications. No sternal infections were observed. Six patients underwent treatment with postoperative balloon pumps, and four of these were placed preoperatively in Group III. Late valve-related complications were rare. One patient underwent late reoperation and valve replacement for prosthetic endocarditis of the annuloplasty ring (0.8%). Two of 23 LLR patients were reoperated on for failure of the repair (8.7%) due to late chordal rupture (one valve replaced and the other repaired with ACR), and no late failures were observed in the ACR group over the follow up period. There were no confirmed thromboembolic episodes. Eight patients experienced undefinable late complications (two sudden deaths, three strokes, and three unwitnessed deaths

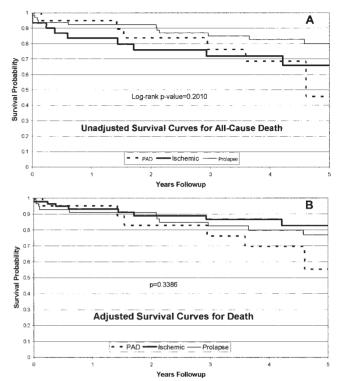


Figure 3: A) Unadjusted survival curves for the three study groups. Follow up is truncated at five years because no deaths were observed between five and nine years of follow up. B) Survival characteristics adjusted for baseline differences in age and comorbidity. See text for details. PAD: Pure annular dilatation.

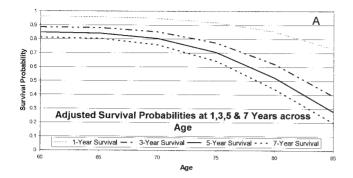
from unknown causes). Two of the stroke patients had prior histories of cerebrovascular disease and remote strokes prior to mitral valve repair, and none of these eight patients had documented problems with his or her valve at the time of the event.

The incidence of recurrent MR at late follow up (median four years) is detailed in Table IV.

Table IV: Transesophageal echocardiography findings.\*

MR finding	Group I (ACR) (n = 43)	Group I (LRR) (n = 23)	Group II (n = 21)	Group III (n = 31)
Acute residual				
Mild	2	3	0	2
Moderate	0	1	0	0
Severe	0	0	0	0
LVOTO	0	1	0	0
Late recurrent				
Mild	6	2	0	2
Moderate	3	4	0	1
Severe	0	2	0	0

<sup>\*</sup>See text for description of procedures and definitions.



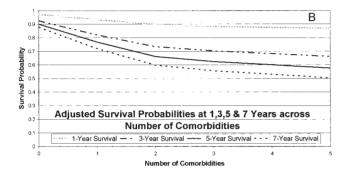


Figure 4: A) Effects of age, and (B) number of comorbidities on patient survival in the overall study group (n = 118). See text for details.

Approximately one-third of the patients had no late echocardiograms because of an absence of symptoms and murmurs. The remainder were studied primarily with surface echocardiography, and therefore were subject to potential inaccuracies. However, moderate recurrent MR occurred in only 7% of the ACR group,

compared to 25% of the LRR patients. Recurrent valve dysfunction was uncommon in Groups II and III.

Causes of late mortality are shown in Table VI. In general, the etiology of late death was distributed evenly among the organ systems, with a negligible cardiac death rate. Long-term Kaplan-Meier raw survival data for the three groups are shown in Figure 3A. Cox multivariable model analysis identified only the number of comorbidities (p = 0.0020) and advanced age (p= 0.0288) as determining late mortality in this population. The etiology of regurgitation, severe left ventricular dysfunction, and acute presentation were not statistically significant determinants of long-term survival (all p >0.25). However, it should be noted that, with only 26 deaths in this population of 118 patients, the multivariable model may have been underpowered to assess adequately all covariate relationships with survival. For example, at a power of 90%, this sample size could only define therapeutic differences of 20% or greater. However, understanding these limitations and when survival data were adjusted for differences in the two significant baseline characteristics (Fig. 3B), no statistical difference in long-term survival between groups was observed (p = 0.3386). Specifically, the average adjusted eight-year survival was approximately 70-80% in both the prolapse and ischemic patients, even though 28% of patients were aged over 70 years and over half had significant comorbidity at baseline. While operative mortality and late survival were slightly worse in Group III, this may have reflected higher baseline comorbidity and advanced age in the ischemic MR subset, at least within the power limits of the analysis.

Modeled effects of age and comorbidity on survival are illustrated in Figure 4. While advanced age significantly diminished survival (Fig. 4A), the outcomes in

*Table V: Postoperative complications.* 

Complication	Group I	Group II (n = 4/21)	Group III (n = 11/31)	Overall (n = 26/118)
	(n = 11/66)			
Pulmonary	8	4	2	14
Renal	0	2	4	6
Stroke	1	0	1	2
TIA/RIND	1	0	1	2
Periop. MI	1	0	0	1
Pacemaker	2	0	0	2
Abdominal	1	0	1	2
Reop. bleeding	1	0	1	2
IABP	1	0	5	6

Some patients had more than one complication; numbers of patients experiencing complications are shown in parentheses. IABP: Intra-aortic balloon pump used postoperatively; Periop MI: Postoperative myocardial infarction by criteria of ECG evolution; Reop bleeding: Reoperation for mediastinal hemorrhage; RIND: Neurological symptoms resolved within 24 h; TIA: Transient ischemic attack, with neurological symptoms completely resolved within 30 min. See text for other definitions.

the older patients were surprisingly good. The average five-year survival rate for 80-year olds was 50%. The effects of number of comorbidities on survival are shown in Figure 4B. Patients with serious preoperative non-cardiac illnesses experienced higher late mortalities, usually from progression of preoperative conditions (Table VI).

# Discussion

Although numerous individuals have contributed to the development of mitral valve repair (18), Carpentier must be given credit for accelerating applications by combining the techniques of measured ring annuloplasty with LRR, chordal shortening, and chordal transfer (19). As a result, reparative methods are now highly refined and successful (20). In mitral valve prolapse, however, LRR is based on the quality of residual chordal support, and inadequate chords due to generalized chordal elongation or multiple chordal ruptures can limit repair in some patients. Additionally, late deterioration of valve competence due to late chordal rupture necessitates eventual reoperation in 5-10% of LRR patients in most series.

These issues and prior investigations into chordal replacement (21) prompted the introduction of the Gore-Tex ACR by Frater and associates in 1986 (22). ACR has several theoretical advantages over LRR. First, chordal support is significantly augmented by Gore-Tex sutures, and repair is not based solely on residual myxomatous chords. Second, ACR does not require leaflet resection, and by preserving a greater leaflet surface area, ACR could reduce the incidence of acute residual MR. Third, any prolapsed leaflet or segment can be repaired with ACR, making it convenient to repair more complex types of prolapse, such as large anterior leaflet chordal ruptures, bileaflet defects, or generalized prolapsing Barlow's valves. Fourth, the added strength and chordal support of Gore-Tex artificial chords could reduce the incidence of late chordal rupture, valve failure, and reoperation. Finally, by maintaining posterior leaflet surface area, the anterior

leaflet coapts well down into the ventricle, and LVOTO is avoided.

The testing of these concepts began in the first author's practice in 1996, when LRR was abandoned in favor of uniform ACR in all patients with prolapse. While the number of LRR patients in the present series was small, it represents the last of an LRR practice that extends well back into the 1980s, and the incidence of late valve failure in this report is representative of results obtained earlier, as well as in most publications. Concern with late chordal rupture after chordal shortening (23) prompted the refinement of 'adjustable' ACR in the late 1980s for use in anterior leaflet prolapse. This application was consistent with reasoning also published by others (24-32). The difficulty with ACR was obtaining an optimal length (one centimeter too short or long and the valve would still leak), and the 'adjustability' method seemed to solve this problem. When ACR to the anterior leaflet was performing well, and with persistent problems with LRR as described above, a transition was made in 1996 to using ACR without leaflet resection for all types of prolapse, even those involving the posterior leaflet. While the method seemed excellent from the beginning, it was important to allow at least a four-year median follow up before reporting the series, as well as to obtain experience with sufficient numbers of patients and different pathologies to be certain of uniform utility. In the present analysis, ACR does seem to improve applicability, to have better stability, and to eliminate LVOT as compared to LRR at intermediateterm follow up. It is therefore reasonable to expand the use of ACR, although more experience with larger numbers of patients and longer follow up times will be necessary in order to draw definitive conclusions.

Severe MR from pure mitral annular dilatation without prolapse or ischemia has been repaired with simple annuloplasty for decades. However, as noted in the results of the present study, annular dilatation patients in this series were slightly younger and predominantly female (Table I). Moreover, the majority had small valves (15 patients sized to #26, four to #28, and two to #32). Two-thirds of the patients had serious preopera-

Table VI: Causes of late mortality

Cause	Group I (n = 11/66)	Group II (n = 6/21)	Group III (n = 9/31)	Overall (n = 26/118)		
Pneumonia	2	0	3	5		
Renal failure	0	3	1	4		
Neurological	1	1	2	4		
Sudden death	2	0	0	2		
Abdominal	2	0	1	3		
Old age	1	1	0	2		
Unknown	2	1	0	3		
Operative	1	0	2	3		
-						

without reservation.

tive comorbidity, one-third had severe left ventricular dysfunction, and one-third presented with acute illness. Almost half (10/21) had significant hypertension, chronic renal insufficiency, or renal failure requiring dialysis - conditions that probably predisposed to ventricular and annular dilatation. Thus, the average profile in this group was a younger female with a small valve, with a higher comorbidity, and presenting with a greater incidence of acute illness and left ventricular dysfunction. These valves could be repaired simply with ring annuloplasty, and the patients' prognoses were determined primarily by their underlying medical disorders. In the entire annular dilatation group, including the seven patients with severe left ventricu-

lar dysfunction, all had complete relief of congestive

heart failure, left ventricular function recovered rou-

tinely, and the five late deaths were from non-cardiac

causes. Thus, even though this group seems to be high-

risk at baseline, the outcomes are excellent and these

patients should be considered for valve repair, almost

Patients with post-infarction ischemic MR have presented a difficult management problem for decades. As exemplified by several early series (10), patients undergoing mitral valve replacement for ischemic MR carried a much worse early and late prognosis than for myxomatous (prolapse) or rheumatic valves. Most ischemic MR patients develop valve regurgitation after a right or distal circumflex coronary infarct (33-35), and almost 90% have annular dilatation at the posterior commissure, usually combined with a commissural cusp or cleft. The decision to repair the valve is made in the operating room, based on the pre-bypass TEE, sometimes combined with volume loading after aortic cannulation (34). Most display a central regurgitant jet on TEE (with no leaflet prolapse or tethering) and can be repaired with a high degree of long-term success by ring annuloplasty. A complete coronary revascularization also should be performed, routinely using an internal mammary artery graft. In the present series, none of the ischemic patients repaired by ring annuloplasty displayed significant early or late valve dysfunction, and mitral repair should be performed on any patient undergoing coronary bypass, who exhibits moderate-to-severe MR on pre-bypass TEE (36).

It is interesting that an aggressive approach to valve repair in ischemic MR resulted in an eight-year adjusted survival of 70-80% - a prognosis that was similar to that for mitral valve prolapse (Fig. 3). This is a significant change from most previous data (10-14). The conversion from valve replacement to repair in the 1980s reduced the predicted operative mortality for ischemic MR from 50-60% in 1980 to around 10-15% by 1990 (33,39). During the past decade, this mortality seems to have been halved again by consistent use of valve

repair, and perhaps by better pre- and postoperative care. Theoretically, the combination of lower operative mortality, restoration of excellent long-term valve function, and complete coronary revascularization could restore the ischemic MR subset to a prognosis which differed little from that for prolapse. While the Cox model analysis in this study (Fig. 3) was consistent with this concept, the low number of events (26/118) potentially reduced the power of the model to detect any but the very strongest covariate relationships with mortality, and further assessment will be necessary.

Another interesting outcome of the present study was the lack of statistical significance of acute presentation (versus elective procedure) as a risk factor. Operative mortality seems to be reduced by valve repair because of the more physiological effects of repair on left ventricular function (40), and because of the extremely low incidence of valve-related complications. Also of importance are improved techniques of postoperative care. Pulmonary, renal, gastrointestinal, nutritional, antibiotic and arrhythmia management are all steadily advancing, and there is little doubt that these improvements have contributed to lower mortality in the acutely ill patient. Especially important was the change in arrhythmia management in 1996, when all patients undergoing mitral repair began to be loaded preoperatively, and were maintained both intra- and postoperatively with high-dose intravenous amiodarone. While further, perhaps randomized, studies might be considered, short-term (6- to 12-week) amiodarone therapy: (i) was associated with no appreciable complications; (ii) reduced postoperative arrhythmias significantly; and (iii) eliminated ventricular arrhythmias as a cause of mortality. At late follow up, sinus rhythm was maintained in all but one patient irrespective of the preoperative rhythm, and the routine amiodarone protocol very likely contributed to improved early and late outcomes in the mitral valve subset that is so prone to arrhythmias.

In the present population, severe left ventricular dysfunction also was not a significant risk factor for long-term mortality. An advantage of repair is maintenance of ventricular-valvular continuity (41) and preservation of left ventricular function in the perioperative period. Repair also provides the best chance for ventricular remodeling and recovery of function in the long term. Again, it was difficult in the present study to quantify this phenomenon, because most of the late echocardiograms were office-based, transthoracic procedures. However, reduced end-diastolic volume and improved ejection fraction were encountered routinely. Thus, valve repair for MR provides an excellent chance for recovering good cardiac function in the long term, optimizing prognostic potential.

The two significant factors predicting late mortality

in the present series were patient age and comorbidity. Of course, these patient-related variables cannot be controlled by surgical technical innovation, but efforts still can be made to reduce mortality - and especially morbidity - even further. Better cardiopulmonary bypass methods, with biocompatible surfaces, pulsatile perfusion, and minimization of pump suckers offer the potential for less postoperative blood activation, strokes and renal insufficiency. Since pulmonary infection requiring intravenous antibiotics was the most common complication in this series, meticulous attention to nosocomial infection sources, as well as to better antibiotic protocols, offer the potential for improving pulmonary complications and reducing postoperative hospital stay, even in the sickest patients. The recent introduction of port access and robotic approaches for mitral repair offer great potential for further improvement in this regard (42,43). Even though age is a significant risk factor, the good survival data obtained in the elderly (Fig. 4) justify continued application of valve repair in the advanced age group, as reported by others (44,45).

From multiple publications over many years, it is clear that mitral valve repair has many advantages, and these were evident in the present series. Operative mortality is low, coumadin (warfarin) anticoagulation is avoided, and thromboembolism is rare. Late bacterial endocarditis occurs infrequently, and left ventricular function is optimized. All of these factors contribute to the excellent long-term outcomes achieved in nonrheumatic MR in this and previous studies. At this point in time, ischemic etiology seems to diminishing as a significant risk factor, and patients with pure annular dilatation can be restored to a prognosis determined primarily by underlying comorbidity. Finally, it also appears that uniform ACR in prolapse may allow successful repair of virtually all pathologies with a low incidence of late valve failure. Undoubtedly, the future appears bright for mitral valve repair.

### Acknowledgements

These studies were supported by a Grant Stipend from Edwards Lifesciences, LLC to the Duke Clinical Research Institute. Demonstration videos are available by contacting J. S. Rankin MD.

### References

- 1. Kouchoukos NT. Problems in mitral valve replacement. In: Kirklin TW (ed). Advances in Cardiovascular Surgery. Grune & Stratton, New York,1973:205-216
- 2. Gillinov AM, Cosgrove DM, III, Shiota T, et al. Cosgrove-Edwards annuloplasty system: Midterm results. Ann Thorac Surg 2000;69:717-721
- 3. Grossi EA, Galloway AC, LaPietra A, et al. Minimally invasive mitral valve surgery: A 6-year

- experience with 714 patients. Ann Thorac Surg 2002;74:660-664
- 4. Angell WW, Oury JH, Shah P. A comparison of replacement and reconstruction in patients with mitral regurgitation. J Thorac Cardiovasc Surg 1987;98:665-674
- 5. Cohn LH, Kowalker W, Bhatia S, et al. Comparative morbidity of mitral valve repair versus replacement for mitral regurgitation with and without coronary artery disease. Ann Thorac Surg 1988;45:284-290
- 6. Galloway AC, Colvin SB, Baumann FG, et al. A comparison of mitral valve reconstruction with mitral valve replacement: Intermediate-term results. Ann Thorac Surg 1989;47:655-662
- 7. Mohty D. Orszulak TA, Schaff HV, et al. Very longterm survival and durability of mitral valve repair for mitral valve prolapse. Circulation 2001;104(12 Suppl.1):11-17
- 8. Braunberger E, Deloche A, Berrebi A, et al. Very long-term results (more than 20 years) of valve repair with Carpentier's techniques in non-rheumatic mitral valve insufficiency. Circulation 2001;104(12 Suppl.1):18-21
- 9. Jebara VA, Mihaileanue S, Acar C, et al. Left ventricular outflow tract obstruction after mitral valve repair: Results of the sliding leaflet technique. Circulation 1993;88(5 Part 2):II30-II34
- 10. Miller DC. Ischemic mitral regurgitation redux to repair to replace? J Thorac Cardiovasc Surg 2001;122:1059-1062
- 11. Grossi EA, Goldberg JD, LaPietra A, et al. Ischemic mitral valve reconstruction and replacement: Comparison of long-term survival and complications. J Thorac Cardiovasc Surg 2001;122:1107-1124
- 12. Gillinov AM, Wierup PN, Blackstone EH, et al. Is repair preferable to replacement for ischemic mitral regurgitation? J Thorac Cardiovasc Surg 2001:122:1125-1141
- 13. Tavakol R, Weber A, Brawner-la Rocca. Results of surgery for irreversible moderate to severe mitral valve regurgitation secondary to myocardial infarction. Eur J Cardiothorac Surg 2002;21:818-824
- 14. Seipeli RG, Schoendube FA, Yazquez-Jimenez JF. Combined mitral valve and coronary artery surgery: Ischemic versus non-ischemic mitral valve disease. Eur J Cardiothorac Surg 2001;20:270-275
- 15. Kasegawa H, Kamata S, Hirata S, et al. Simplified method for determining proper length of artificial chordae in mitral valve repair. Ann Thorac Surg 1994;57:237-238
- 16. Revuelta J, Garcia-Rinaldi R, Gaite L, et al. Generation of chordae tendineae with PTFE stents. J Thorac Cardiovasc Surg 1989;97:98-103
- 17. Sarsam MA. Simplified technique for determining the length of artificial chordae in mitral valve repair. Ann Thorac Surg 2002;73:1659-1660

- 18. Rankin JS. Mitral and tricuspid valve disease: Historical aspects. In: Sabiston DC, Jr. (ed). Textbook of Surgery. WB Saunders Company, Philadelphia, 1986:2345
- 19. Carpentier A. Cardiac valve surgery the "French correction". J Thorac Cardiovasc Surg 1983;86:323-337
- 20. Duran CMG, Pomar JL, Revuelta JM, et al. Conservative operation for mitral insufficiency: Critical analysis supported by postoperative hemodynamic studies of 72 patients. J Thorac Cardiovasc Surg 1980;79:328-348
- 21. Frater RWM, Gabbay S, Shore D, et al. Reproducible replacement of elongated or ruptured mitral valve chordae. Ann Thorac Surg 1983;35:14-28
- 22. Vetter HO, Burack JH, Factor SM, et al. Replacement of chordae tendineae of the mitral valve using the new expanded PTFE suture in sheep. In: Bodnar E, Yacoub M (ed). Biologic bioprosthetic valves. Yorke Medical Books, New York, 1986:772-784
- 23. Koutlas TC, de Bruijn N, Sheikh KH, et al. Chordal rupture as a late complication after mitral valve reconstruction. J Thorac Cardiovasc Surg 1991;102:466-468
- 24. Phillips MR, Daly RC, Schaff HV, et al. Repair of anterior leaflet mitral valve prolapse: Chordal replacement versus chordal shortening. Ann Thorac Surg 2000;69:25-29
- 25. Adams DH, Kadner A, Chen RH. Artificial mitral valve chordae replacement made simple. Ann Thorac Surg 2001;71:1377-1379
- 26. Kumar AS. Chordal replacement or repair. Ann Thorac Surg 2001;71:2084
- 27. Zussa C, Frater RWM, Polesel E, et al. Artificial mitral valve chordae: Experimental and clinical experience. Ann Thorac Surg 1990;50:367-373
- 28. di Gioia CRT, Brancaccio G, Sinatra R, et al. Longterm histologic features of synthetic chordal replacement for mitral valve repair: A case report. Cardiovasc Pathol 2001;10:87-89
- 29. Kobayashi J, Sasako Y, Bando K, et al. Ten-year experience of chordal replacement with expanded polytetrafluoroethylene in mitral valve repair. Circulation 2000;102(suppl.III):III-30-III-34
- 30. Tomita Y, Yasui H, Tominaga R, et al. Extensive use of polytetrafluoroethylene artificial grafts for prolapse of bilateral mitral leaflets. Eur J Cardiothorac Surg 2002;21:27-31
- 31. Duebener LF, Wendler O, Nikoloudakis N, et al. Mitral-valve repair without annuloplasty rings: Results after repair of anterior leaflet versus posterior-leaflet defects using polytetrafluoroethylene sutures for chordal replacement. Eur J Cardiothorac Surg 2000;17:206-212

- 32. David TE, Omran A, Armstrong S. Long-term results of mitral valve repair for myxomatous disease with and without chordal replacement with expanded PTFE. J Thorac Cardiovasc Surg 1998;115:1279-1286
- 33. Rankin JS, Livesey SA, Smith LR, et al. Trends in the surgical treatment of ischemic mitral regurgitation: Effects of mitral valve repair on hospital mortality. Semin Thorac Cardiovasc Surg 1989;1:149-163
- 34. Rankin JS, Feneley MP, Hickey MSJ, et al. A clinical comparison of mitral valve repair versus valve replacement in ischemic mitral regurgitation. J Thorac Cardiovasc Surg 1988;95:165-177
- 35. Hickey MSJ, Smith LR, Muhlbaier LH, et al. Current Prognosis of ischemic mitral regurgitation. Circulation 1988;78(suppl.I):I-51-I-9
- 36. Sheikh KH, Bengtson JR, Rankin JS, et al. Intraoperative transesophageal Doppler color flow imaging used to guide patient selection and operative treatment of ischemic mitral regurgitation. Circulation 1991;84:594-604
- 37. Johnson WD, Pedraza PM, Kayser KL. Preservation and restoration of the mitral mechanism during inferior wall aneurysmectomy. In: Duran CMG, Angell WW, Johnson AD, Oury JH (ed). Recent progress in mitral valve disease. Butterworth & Co, London, 1984:410-415
- 38. Menicanti L, Di Donato M, Frigiola A, et al. Ischemic mitral regurgitation: Intraventricular papillary muscle fimbrication without mitral ring during left ventricular restoration. J Thorac Cardiovasc Surg 2002;123:1041-1050
- 39. Carpentier A, Didier L, Deloche A, et al. Surgical anatomy and management of ischemic mitral valve incompetence. Circulation 1987;76(suppl.II):II76-II83
- 40. Boncheck LI, Olinger GN, Siegel R, et al. Left ventricular performance after mitral reconstruction for mitral regurgitation. J Thorac Cardiovasc Surg 1984;88:122-127
- 41. Hansen DE, Cahill PD, Derby GC, et al. Relative contributions of the anterior and posterior mitral chordae tendineae to canine global left ventricular systolic function. J Thorac Cardiovasc Surg 1987;93:45-55
- 42. Glower DD, Siegel LC, Frischmeyer KJ, et al. Predictors of outcome in a multicenter port-access valve registry. Ann Thorac Surg 2000;70:1054-1059
- 43. Chitwood WR, Jr., Wixon CL, Elbeery JR, et al. Video assisted minimally invasive mitral valve surgery. J Thorac Cardiovasc Surg 1997;114:773-782
- 44. Jebara V. Mitral valve repair in the elderly. Ann Thorac Surg 2001;71:1752
- 45. Grossi EA, Zakow PK, Sussman M, et al. Late results of mitral valve reconstruction in the elderly. Ann Thorac Surg 2000;70:1224-1226