Long-Term Results of the Surgical Treatment of Chronic Ischemic Mitral Regurgitation: Comparison of Repair and Prosthetic Replacement

Vittorio Mantovani, Giovanni Mariscalco, Cristian Leva, Claudio Blanzola, Paolo Cattaneo, Andrea Sala

Department of Cardiac Surgery, University of Insubria, Ospedale di Circolo e Fondazioni Macchi, Varese, Italy

Background and aim of the study: **The optimal management of chronic ischemic mitral regurgitation (CIMR) remains controversial. Herein, the authors reviewed the past 10 years of their experience to compare the long-term results of mitral valve repair with prosthetic replacement.**
Methods: **Between January 1993 and January 2003, 102 patients (mean age 67.8 years; range: 51-80 years) with a preoperative diagnosis of CIMR, underwent mitral valve repair (n = 61; 59.8%) or prosthetic replacement (n = 41; 40.2%), along with myocardial revascularization (2.5 ± 1.0 distal anastomoses per patients, internal thoracic artery used in 78.5%). A Carpentier Classic ring was always used in the repair procedures. The two groups were homogeneous for preoperative characteristics and comorbidities.**
Results: **Total operative mortality was 7.8% (repair 8.2%; prosthesis 7.3%; p = NS). The five-year actuarial survival (operative mortality included) was 66.6 ± 7.4% for repair and 73.4 ± 8.7% for prosthesis (p = NS). Cox multivariate analysis showed as independent risk factor for late survival a preoperative left ventricular ejection fraction (LVEF) ≤30% (RR 3.91; 95% CI = 1.47-10.38) and a preoperative pulmonary artery pressure (PAP) ≥35 mmHg (RR 2.74; 95% CI = 1.07-7.02), while the type of mitral procedure was not significant. Patients with annular dilation as a mechanism of regurgitation were significantly more likely to undergo repair rather than receive a prosthesis. Their preoperative LVEF and PAP were significantly worse than patients who had altered leaflet motion as a regurgitation mechanism.**
Conclusion: **Prosthetic mitral replacement and valve repair offer very similar results for CIMR. When a perfect repair is not easily feasible, cardiac surgeons should not hesitate to perform mitral valve replacement, as it is an excellent alternative therapy, though long-term outcome is mainly dependent on preoperative condition.**

The Journal of Heart Valve Disease 2004;13:421-429

The optimal management of chronic ischemic mitral regurgitation (CIMR) remains controversial (1). The surgical approach depends upon the pathophysiological mechanisms and degree of mitral regurgitation. The milder form (grade 1) is generally treated with coronary artery bypass grafting (CABG) alone, whereas the more severe forms (grade 3 and 4) require a valvular or annular procedure along with CABG. The optimal treatment of moderate (grade 2) mitral regurgitation has been debated. Recent studies have also advocated a mitral valve procedure in these patients (2).

Patients with mitral regurgitation (MR) of ischemic origin suffer a higher surgical risk compared with MR of other etiologies, with an operative mortality as high as 20% in some series (3).

Mitral valve repair has shown clearly superior results compared to replacement for degenerative diseases (4,5). In the specific case of CIMR, some reports (6) failed to confirm significant advantages of repair versus replacement, while other series have succeeded in doing so (3,7).

The study aim was to evaluate the long-term results of the surgical treatment of CIMR, comparing mitral valve repair versus replacement.

Clinical material and methods

Patient population

Between January 1993 and January 2003, 102 consecutive patients (61.8% males; mean age 67.8 ± 6.9 years; range: 51 to 80 years) underwent combined CABG and

Presented at the Second Biennial Meeting of the Society for Heart Valve Disease, 28th June-1st July 2003, Palais des Congrès, Paris, France

Address for correspondence:
Vittorio Mantovani MD, Department of Cardiac Surgery, University of Insubria, Ospedale di Circolo e Fondazioni Macchi, Viale Borri 57, 21100 Varese, Italy
e-mail: vitmantovani@hotmail.com

mitral valve surgery for CIMR (grades 2 to 4) at the authors' institution. The mitral valve procedure was a repair in 61 cases and prosthetic replacement in 41 cases, using in 31 cases a bileaflet mechanical prosthesis and in the remaining 10 a porcine bioprosthesis. The preoperative data of the patients (Table I) did not reveal any difference between the two groups. In all patients, MR was assessed by preoperative transthoracic echocardiography in a five-point scale (0 = none; 1 = mild; 2 = moderate; 3 = moderate-severe; 4 = severe). Exclusion criteria from this study included recent myocardial infarction (<21 days), non-ischemic MR associated with coronary disease, presence of significant tricuspid regurgitation, and any associated cardiac procedures other than CABG. The follow up was 100% complete, with a total of 312.6 patient-years (pt-yr). The mean follow up was 36.8 months (median 27.5 months; range: 0 to 117 months)

Surgical technique

A median full sternotomy and moderately hypothermic (28-32°C) cardiopulmonary bypass (CPB) were used in all cases. Myocardial protection was achieved using antegrade and retrograde crystalloid cardioplegia and topical cooling. The distal anastomoses were performed first, followed by the mitral valve procedure and then proximal anastomoses. The mitral valve was exposed through a paraseptal left atriotomy. Whenever technically feasible, a repair procedure was preferred, by inserting a moderately undersized rigid ring (Carpentier-Edwards Classic Ring; Edwards Lifesciences, Irvine, CA, USA).

A hydrodynamic test with saline injection was performed after the procedure in order to assess valvular competence, followed by transesophageal echocardiography after weaning from CPB.

The decision to replace the valve was taken intraop-

Table I: Patient characteristics

Parameter	Repair (n = 61)	Prosthesis (n = 41)	p-value
Age (years)*	67.9 ± 6.9	68.1 ± 6.8	NS
Range	52-80	51 –80	
Gender (%)			
Male	67	54	NS
Female	33	46	NS
Extent of CAD (%)			
Left main disease	16	15	NS
Two-vessel disease	26	24	NS
Three-vessel disease	62	59	NS
LVEF (%)*	44.9 ± 14.1	44.5 ± 13.9	NS
PAP (mmHg)*	39.2 ± 13.4	33.8 ± 10.7	NS
Mitral regurgitation grade*	3.1 ± 0.8	3.0 ± 0.8	NS
Cardiac presentation			
Chronic angina (%)	70	44	NS
Unstable angina (%)	21	20	NS
Heart failure (%)	3	2	NS
Previous MI (%)	64	51	NS
NYHA functional class*	2.7 ± 0.9	2.7 ± 0.8	NS
CCS class*	1.7 ± 1.6	1.9 ± 1.3	NS
Preoperative rhythm (%)			
Sinus rhythm	88	80	NS
Comorbid conditions (%)			
Diabetes mellitus	26	15	NS
Hypertension	54	51	NS
Renal insufficiency	7	7	NS
COPD	13	19	NS
Peripheral vascular disease	41	34	NS
Cerebral disease	15	32	NS
Hypercholesterolemia	33	27	NS

*Values are mean ± SD.
CABG, coronary artery bypass grafting; CAD: Coronary artery disease; CCS: Canadian Cardiovascular Society; COPD, chronic obstructive pulmonary disease; LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; NS: Not significant; PAP: Pulmonary artery pressure.

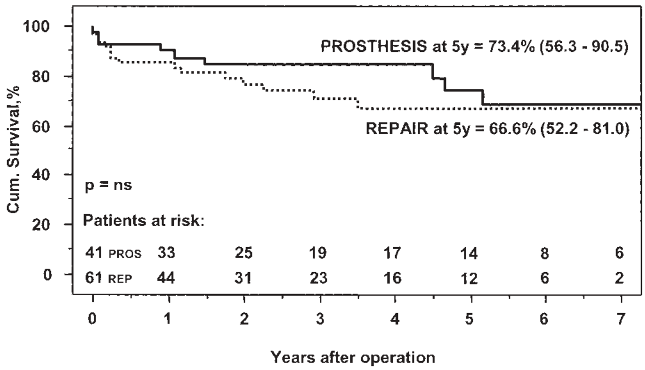

Figure 1: Overall survival of patients receiving either mitral valve repair or prosthetic replacement.

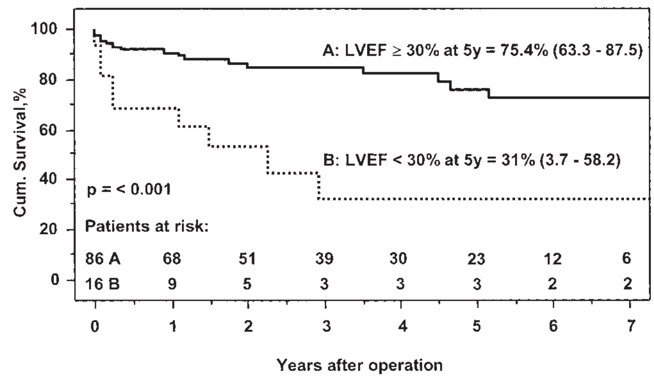

Figure 2: Overall survival of patients grouped for preoperative left ventricular ejection fraction (LVEF) <30% or ≥30%.

eratively at the discretion of the operator, whenever the repair was judged not to be feasible in a short time with a surely predictable result. Valve replacements were performed with posterior leaflet preservation.

Annulus dilation was found to be the main cause of MR in 55% of cases, leaflet abnormal motion was the predominant mechanism in 42%, and papillary muscle dysfunction in 3%. The mitral repair was performed in 92.6% cases of annulus dilatation, in 26.2% cases of leaflet abnormal motion, and in none of the papillary dysfunction cases (p <0.001).

The intraoperative variables are listed in Table II.

After surgery, all patients received warfarin for the first three months. Thereafter, oral anticoagulation was continued only in patients with a mechanical prosthesis or with chronic atrial fibrillation.

Statistical analysis

Continuous variables were compared with a two-sided *t*-test or the Mann-Whitney *U*-test, as appropriate. Categorical variables were compared with chi-square or Fisher exact tests as appropriate. An ANOVA for repeated measures was used to compare sequential measurements. Time-failure curves were constructed with the Kaplan-Meier method and compared with a log-rank test. A multivariate analysis was performed with the Cox proportional hazard method. A p-value <0.05 was considered to be statistically significant.

Table II: Perioperative data.

Parameter	Repair	Prosthesis (n = 61)	p-value (n = 41)
Internal mammary graft (%)	81.9	70.3	NS
No. of grafts*	2.5 ± 1.0	2.6 ± 1.1	NS
Cross-clamp time (min)*	130.6 ± 41.1	121.8 ± 29.3	NS
CPB time (min)*	178.6 ± 54.4	172.7 ± 42.6	NS
Ring/Prosthesis size (mm)+			
25	-	19.5	
26	4.9	-	
27	-	39	
28	19.7	-	
29	-	24.5	
30	34.5	-	
31	-	17	
32	27.8	-	
34	9.9	-	
36	3.2	-	

*Values are mean ± SD.
+Values are percentages.
CPB: Cardiopulmonary bypass.

J Heart Valve Dis
Vol. 13. No. 3
May 2004

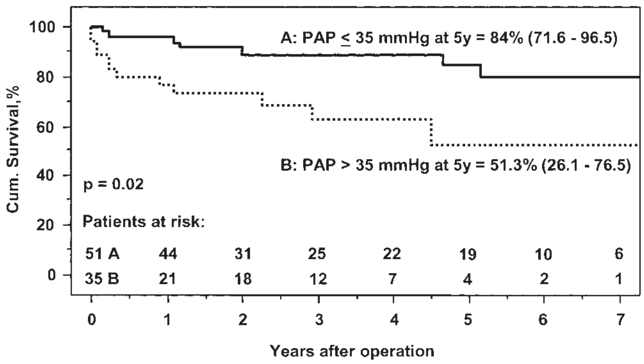

Figure 3: Overall survival of patients grouped for preoperative pulmonary artery pressure (PAP) ≤35 mmHg or >35 mmHg.

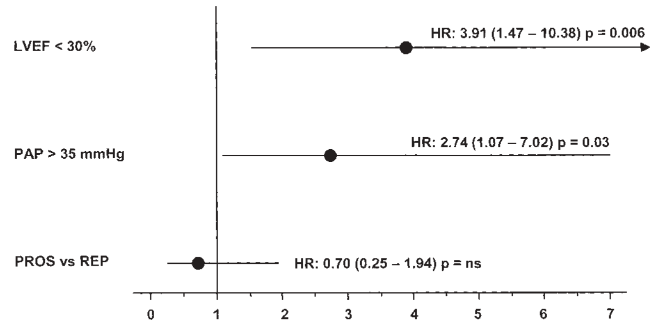

Figure 4: Graphical depiction of Cox regression hazard rates (HR) for overall survival. LVEF: Preoperative left ventricular ejection fraction; PAP: Preoperative pulmonary artery pressure; PROS: Prosthetic replacement; REP: Repair.

Results

Definitions

This report followed the Guidelines for Reporting Mortality and Morbidity after Cardiac Valvular Operations (8). Perioperative myocardial infarction was defined as the appearance of new Q-waves or a significant loss of R-wave forces.

Postoperative bleeding was defined as rethoracotomy for hemorrhage; postoperative renal complications as the need for ultrafiltration; and neurological complications as any transient or permanent neurological deficit that developed after surgery.

Early outcome

The perioperative mortality (repair 8.2%; prosthesis 7.3%) and morbidity did not differ significantly between the two groups (Table III). The results of a logistic regression for risk factors for perioperative death and major complications (myocardial infarction, need for intra-aortic balloon pump, reoperation for bleeding, neurological damage, respiratory failure, renal failure) are listed in Table IV. The mitral procedure, the preoperative left ventricular ejection fraction (LVEF) and the preoperative pulmonary hypertension were not predictors of unfavorable early outcome with multivariate analysis. The preoperative NYHA functional class did not influence the early outcome. Age had no effect on either death or death with major complications.

Late outcome

The long-term survival was not significantly different between the two groups, being 66.6 ± 7.4% for

Table III: Operative mortality and morbidity.

Parameter	Repair (n = 61)	Prosthesis (n = 41)	p-value
Operative (30-day) mortality (%)	8.2	7.3	NS
Perioperative complications (%)			
Cardiac			
Perioperative MI	13.1	10	NS
Postoperative IABP	8.2	2.4	NS
New atrial fibrillation	54	35	NS
AV block	6.5	12.5	NS
Ventricular tachycardia	1.6	2.4	NS
Reoperation for bleeding	1.6	9.7	NS
Cerebrovascular accident	6.5	7.3	NS
Deep sternal wound infection	3.2	2.5	NS
Respiratory failure	11.4	5	NS
Acute renal failure	1.6	2.5	NS
Bacteremia	2.5	0	NS
Death/major complications (%)	39	39.3	NS

AV: Atrioventricular; IABP: Intra-aortic balloon pump; MI: Myocardial infarction.

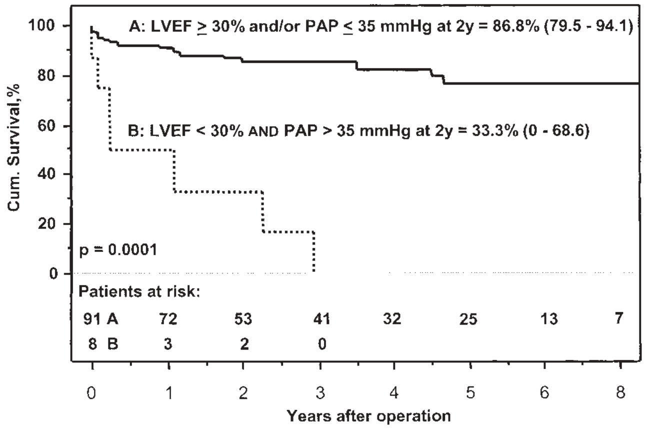

Figure 5: Overall survival of patients grouped for the combination of preoperative left ventricular ejection fraction (LVEF) and preoperative pulmonary artery pressure (PAP).

repair and 73.4 ± 8.7% for prosthesis at five years (p = NS) (Fig. 1). A significant difference was thus evident when the whole population was stratified for preoperative LVEF ≤30% (Fig. 2) or for preoperative pulmonary artery pressure (PAP) ≥35 mmHg (Fig. 3). A Cox regression model showed that these two characteristics were significant independent risk factors for late mortality, while the type of procedure was not (Fig. 4). A dramatic difference in survival was evident in the eight patients who had both low LVEF and elevated PAP (Fig. 5): the two-year survival in this group was 33.3 ± 18.0%, compared with 86.8 ± 3.7% in those patients who had at least one favorable parameter (p <0.0001). Seven of these eight patients had undergone mitral repair. The univariate Kaplan-Meier analysis showed that preoperative NYHA class >2 was significantly associated with lower long-term survival, which at five years was 86.9 ± 6.3% (NHYA class ≤2) compared with 60.3 ± 7.5% (NYHA classes III and IV) (p = 0.041). This association was not confirmed by multivariate Cox regression when preoperative NYHA class was analyzed against the type of procedure, LVEF ≤30% and PAP ≥35 mmHg.

Age less than 65 years was significantly associated with a better survival (88.0 ± 6.6% versus 57.9 ± 8.1% at five years; p <0.04) with univariate Kaplan-Meier analysis, but this was not apparent when data were entered in the Cox multivariate model.

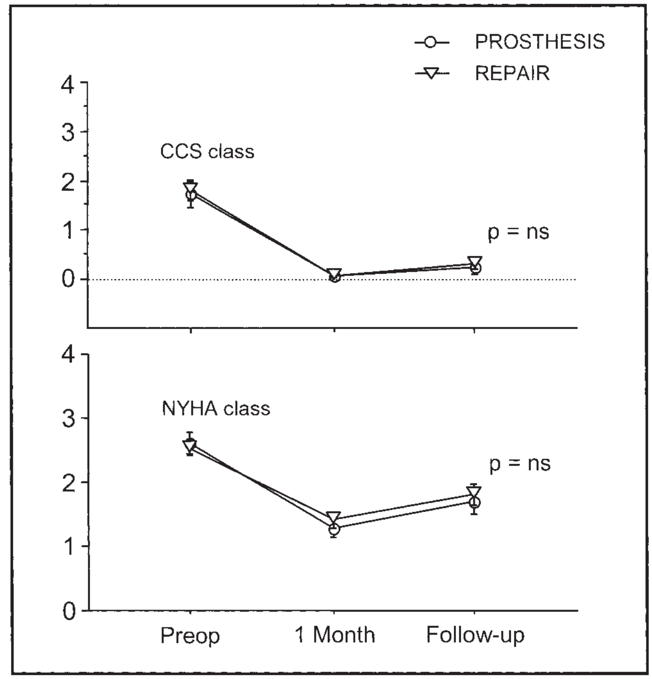

Figure 6: Evolution of Canadian Cardiovascular Society (CCS) and New York Heart Association (NYHA) functional class from preoperative status to one month after surgery and follow up control examination.

Major late complications in prosthesis patients included reoperation for prosthetic endocarditis (n = 1) and thromboembolism (n = 2). Among the repair patients there was one reoperation for ring dehiscence and one case of bleeding. The five-year freedom from death and major complications was not statistically different between the groups (65.8 ± 9.0% for prosthesis; 64.7 ± 7.4% for repair).

Functional outcome

Both NYHA and Canadian Cardiovascular Society class evolved in an almost identical fashion in the two groups (Fig. 6).

The evolution of echocardiographic data is depicted

Table IV: Logistic regression for early outcome: death and major complications.

Parameter	OR	Lower 95% CI	Upper 95% CI	p-value
Repair versus Prosthesis	0.89	0.36	2.23	NS
Preoperative LVEF ≤30%	2.35	0.58	9.49	NS
Preoperative PAP ≥35 mmHg	1.35	0.54	3.40	NS

LVEF: Left ventricular ejection fraction; OR: Odds ratio; PAP: Pulmonary artery pressure.

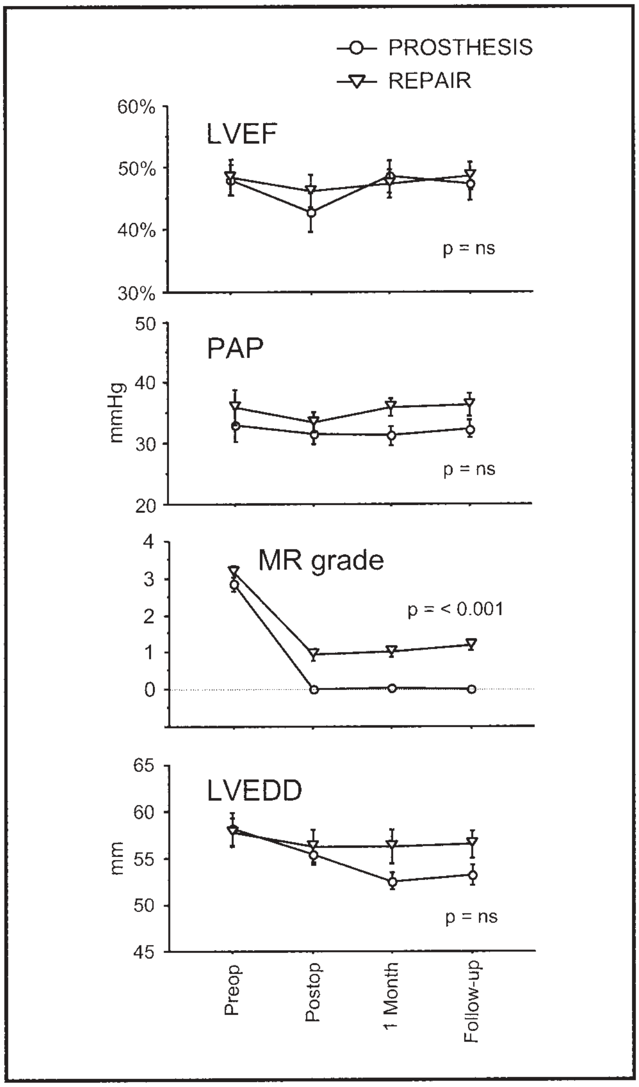

Figure 7: Time course of left ventricular ejection fraction (LVEF), pulmonary artery pressure (PAP), mitral regurgitation (MR) grade and left ventricular end-diastolic diameter (LVEDD) at echocardiographic controls, preoperatively, at hospital discharge, at one month after surgery, and at late follow up respectively.

in Figure 7. LVEF was always almost identical in both groups, whereas PAP tended to be slightly higher in repair patients. Residual MR was significantly higher in repair patients, of whom 15% were found to have MR grade 2 and 10% grade 3 at follow up examination. The left ventricular end-diastolic diameter was decreased more among prosthesis patients, albeit not in any statistically significant manner.

Discussion

Ischemic mitral regurgitation remains the subject of much debate, and important insights have been gained only recently in the underlying pathophysiological mechanisms of the condition. It is now proven that annular dilation is only one of the causes of CIMR, and probably not the main, while leaflet tethering, papillary muscle displacement and dyscoordination and ventricular remodeling play central roles (9-11). This situation was a major cause of uncertainty with regard to the optimal treatment for CIMR. The same uncertainty is apparent regarding the indication, as although there is consensus on treating MR grade 3 or higher (12,13), different opinions have been published about grade 2 (2). Thus, the present authors have adopted a policy of treating CIMR grade 2 and higher.

To the present authors' knowledge, all published papers concerning optimal surgical treatment are retrospective, non-randomized observational studies, and they are well aware that this is a major limit of the present investigation. However, the two groups of patients - repair and prosthesis respectively - were comparable in all preoperative and in most intraoperative characteristics. The two groups were also reasonably balanced in the absolute number of cases, and acute IMR was excluded. One of the main inter-group differences was the mechanism of mitral regurgitation. In most cases of annular dilation, a repair was performed, while prosthetic replacement was predominant in patients with altered leaflet motion. This policy was based on the generally accepted notion that the mitral valve should be repaired whenever technically possible. This is demonstrated for degenerative mitral diseases (4,5), while the lack of such a certainty is one of the major reasons for debate on the topic of ischemic mitral regurgitation. Reports from some of the most respected authors (6) showed no advantage for repair versus replacement for CIMR, while others (3,7) were able to demonstrate some advantages for repair, albeit not in all subgroups of their patients. The operative mortality in the present study was comparable with that in most reported series, and no predictors of unfavorable early outcome could be identified, as in other reports (6).

The main finding of the present study was that long-term survival is dependent upon preoperative left ventricle condition rather than the type of procedure used, and that the LVEF and PAP are independent risk factors. A low LVEF has already been described as major risk factor (14), but a high PAP is a relatively new finding. Some reports have focused on NYHA class (3) which, in the present study, did not appear as important as LVEF and PAP. Clearly, all of these are different ways of examining the same main point - that is, preoperative left ventricular status. A very important consideration made by Cohn et al. (6) was that some mechanisms of MR might be related to poorer left ventricular function. Based on this, in the present study the indicators of left ventricular function were analyzed when stratified for the MR mechanism. In the group of patients with annular dilation, the mean preoperative LVEF was 40.7 ± 12.7% and the mean PAP 40.6 ± 13.5 mmHg, whereas corresponding values for patients with altered leaflet motion as the mechanism of MR were 49.7 ± 13.4% and 34.0 ± 11.1 mmHg, respectively. Both differences were statistically significant ($p < 0.002$ for LVEF, and $p < 0.03$ for PAP). It is important to underline that preoperative LVEF and PAP were not statistically different when comparing the two types of surgical treatment (repair versus prosthesis).

It has been suggested that a bioprosthesis should only be used for CIMR, due to the patients' short life expectancy (1). However, the present authors feel justified in choosing a mechanical prosthesis for younger patients based upon the long-term survival observed herein for prostheses (73.4% at five years). The frequency of long-term complications and the evolution of clinical status and echocardiographic parameters were similar in both the repair and prosthesis groups. The exception was that of residual MR, which eventually became higher than grade 2 in 24% of repair patients. This relatively high number, which is in line with a recent report by Tahta et al. (15), might be due to the difficulty of properly repairing IMR, or to the unfavorable evolution of ventricular remodeling. Carpentier Classic rigid rings were always used in the present repairs; recent studies by Gorman et al. (16) and Tibayan et al. (17) showed the importance of preventing the elongation of the anterior part of the mitral annulus, thus supporting the present choice of complete annuloplasty. In order to improve the present results, smaller ring sizes than before are currently being used, as proposed by Badhwar and Bolling (18). A better understanding of the geometric distortions of the mitral valvular-ventricular complex (19) will surely play a key role in improving the results of repairs. Since the structures below the level of the mitral annulus are heavily involved in the mechanism of regurgitation, it seems unlikely that simple down-sizing of the presently used rings will address this point. Rather, a different ring design may be needed in order to preserve the septal-lateral dimension and the saddle shape of the annulus, and innovative approaches such as the cutting of basal chordae (20) may be needed to hit the true physiopathological target.

In the present series, long-term survival in chronic ischemic mitral regurgitation was related mainly to the patients' preoperative status. The suboptimal long-term performance of mitral repair might offer an explanation for the fact that no survival benefit was associated with this approach. It is important to bear in mind that a residual MR of grade >1 was seen to be detrimental for survival in both surgical (21) and non-surgical coronary patients (22), and thus it is reasonable to affirm that, among ischemic patients, mitral valve repair is an intrinsically better treatment than replacement. However, any advantages in this respect may be outweighed by the fact that the easiest lesion to repair - simple annular dilation - is a consequence of a worse left ventricle. The currently available repair techniques are effective for degenerative mitral diseases, but are probably inadequate for an ischemic etiology. Systematic sparing of the posterior leaflet in the present patients may have further mitigated the disadvantages of prosthesis replacement. The importance of chordal-sparing techniques in order to preserve left ventricular function has been proven over many years (23,24), and a recent prospective randomized study (25) indicated that additional advantages are provided by complete chordal sparing. Thus, it is the intention of the present authors also to spare the anterior chordae. Despite this, mitral valve replacement with chordal sparing remains inferior to repair when preserving left ventricular motion (26). Consequently, it is proposed that, when a perfect repair is judged to be neither easy nor feasible, then mitral valve replacement should be performed a with a chordal-sparing technique, as this offers an excellent alternative therapy.

References

1. Miller DC. Ischemic mitral regurgitation redux - to repair or to replace? J Thorac Cardiovasc Surg 2001;122:1059-1062
2. Aklog L, Filsoufi F, Flores KQ, et al. Does coronary artery bypass grafting alone correct moderate ischemic mitral regurgitation? Circulation 2001;104(12 Suppl.1):I68-I75
3. 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
4. Carpentier A, Chauvaud S, Fabiani JN, et al. Reconstructive surgery of mitral valve incompetence: Ten-year appraisal. J Thorac Cardiovasc Surg 1980;79:338-348
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. Cohn LH, Rizzo RJ, Adams DH, et al. The effect of pathophysiology on the surgical treatment of ischemic mitral regurgitation: Operative and late risks of repair versus replacement. Eur J Cardiothorac Surg 1995;9:568-574
7. 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
8. Edmunds LH, Jr., Clark RE, Cohn LH, et al. Guidelines for reporting morbidity and mortality after cardiac valvular operations. Ad Hoc Liaison Committee for Standardizing Definitions of Prosthetic Heart Valve Morbidity of The American Association for Thoracic Surgery and The Society of Thoracic Surgeons. J Thorac Cardiovasc Surg 1996;112:708-711
9. Otsuji Y, Handschumacher MD, Liel-Cohen N, et al. Mechanism of ischemic mitral regurgitation with segmental left ventricular dysfunction: Three-dimensional echocardiographic studies in models of acute and chronic progressive regurgitation. J Am Coll Cardiol 2001;37:641-648
10. Otsuji Y, Kumanohoso T, Yoshifuku S, et al. Isolated annular dilation does not usually cause important functional mitral regurgitation: Comparison between patients with lone atrial fibrillation and those with idiopathic or ischemic cardiomyopathy. J Am Coll Cardiol 2002;39:1651-1656
11. Gorman JH, III, Gorman RC, Plappert T, et al. Infarct size and location determine development of mitral regurgitation in the sheep model. J Thorac Cardiovasc Surg 1998;115:615-622
12. Harris KM, Sundt TM, III, Aeppli D, et al. Can late survival of patients with moderate ischemic mitral regurgitation be impacted by intervention on the valve? Ann Thorac Surg 2002;74:1468-1475
13. Ryden T, Bech-Hanssen O, Brandrup-Wognsen G, et al. The importance of grade 2 ischemic mitral regurgitation in coronary artery bypass grafting. Eur J Cardiothorac Surg 2001;20:276-281
14. Hausmann H, Siniawski H, Hetzer R. Mitral valve reconstruction and replacement for ischemic mitral insufficiency: Seven years' follow up. J Heart Valve Dis 1999;8:536-542
15. Tahta SA, Oury JH, Maxwell JM, et al. Outcome after mitral valve repair for functional ischemic mitral regurgitation. J Heart Valve Dis 2002;11:11-18; discussion 18-19
16. Gorman JH, III, Gorman RC, Jackson BM, et al. Annuloplasty ring selection for chronic ischemic mitral regurgitation: Lessons from the ovine model. Ann Thorac Surg 2003;76:1556-1563
17. Tibayan FA, Rodriguez F, Langer F, et al. Annular remodeling in chronic ischemic mitral regurgitation: Ring selection implications. Ann Thorac Surg 2003;76:1549-1554; discussion 1554-1555
18. Badhwar V, Bolling SF. Mitral valve surgery in the patient with left ventricular dysfunction. Semin Thorac Cardiovasc Surg 2002;14:133-136
19. Tibayan FA, Rodriguez F, Zasio MK, et al. Geometric distortions of the mitral valvular-ventricular complex in chronic ischemic mitral regurgitation. Circulation 2003;108(Suppl.1):II116-II121
20. Messas E, Pouzet B, Touchot B, et al. Efficacy of chordal cutting to relieve chronic persistent ischemic mitral regurgitation. Circulation 2003;108(Suppl.1):II111-II115
21. Dahlberg PS, Orszulak TA, Mullany CJ, Daly RC, Enriquez-Sarano M, Schaff HV. Late outcome of mitral valve surgery for patients with coronary artery disease. Ann Thorac Surg 2003;76:1539-1487; discussion 1547-1548
22. Grigioni F, Enriquez-Sarano M, Zehr KJ, Bailey KR, Tajik AJ. Ischemic mitral regurgitation: Long-term outcome and prognostic implications with quantitative Doppler assessment. Circulation 2001;103:1759-1764
23. David TE, Uden DE, Strauss HD. The importance of the mitral apparatus in left ventricular function after correction of mitral regurgitation. Circulation 1983;68(3 Pt. 2):II76-II82
24. Hennein HA, Swain JA, McIntosh CL, Bonow RO, Stone CD, Clark RE. Comparative assessment of chordal preservation versus chordal resection during mitral valve replacement. J Thorac Cardiovasc Surg 1990;99:828-836; discussion 836-837
25. Yun KL, Sintek CF, Miller DC, et al. Randomized trial comparing partial versus complete chordal-sparing mitral valve replacement: Effects on left ventricular volume and function. J Thorac Cardiovasc Surg 2002;123:707-714.
26. Okita Y, Miki S, Kusuhara K, et al. Analysis of left ventricular motion after mitral valve replacement with a technique of preservation of all chordae tendineae. Comparison with conventional mitral valve replacement or mitral valve repair. J Thorac Cardiovasc Surg 1992;104:786-795

Meeting discussion

DR. MAREK JASINSKI (Poland): What were the inclusion criteria for the two groups? Was there any difference between them?

DR. CLAUDIO BLANZOLA (Varese, Italy): The criteria were the presence of mitral regurgitation or a proven ischemic region - any other forms of mitral regurgitation were excluded. That is, any patient with mitral regurgitation of other cause was excluded from the preoperative or intraoperative assessment. Acute patients and those with associated procedures, such as tricuspid annuloplasty, were also excluded. So this patient population can be considered quite 'clean'.

DR. JASINSKI: Were there any differences between the groups in that some patients were prospectively scheduled for the operation, while in others the procedure was a 'bailout'?

DR. BLANZOLA: This is an obvious limitation of the study. This was a non-randomized, retrospective study, so on many occasions the choice of procedure was the surgeon's preference. We noted an evolution in the past few years. At the start of the study, the percentage of mitral substitution was greater, but during the years there was a move towards repair. We can say that on many occasions when the valve looked normal - as is often the case in chronic ischemic mitral regurgitation - an attempt was made to repair it. But when the leaflets seemed excessively restricted in their motion, the valve was replaced.

DR. CHRISTOPHE ACAR (France): One comment and one question. In your conclusion, you state that rapid mitral valve replacement can be preferable to a complex repair in ischemic patients. But repair is usually not very complex, and is comparable with prosthetic ring annuloplasty. With regard to the results, one reason why replacement could do better than repair is that there is no recurrence of MR. In your study it doesn't appear that you had a high recurrence of MR in the repair groups. So how do you explain that patients undergoing replacement did better than those undergoing a repair?

DR. BLANZOLA: I think that one explanation for our long procedure times may be the learning curve for many of us who did the repairs. One explanation for the long times is whether the initial intention was to repair the valve - we changed our mind during the intervention six times. The ring annulus we chose was not very tight, and perhaps we should have reduced the size more. I have no simple explanation for this, other than the learning curve of mitral valve repair.

DR. DAVID H. ADAMS (USA): I echo your comments. I noticed that your ring size was 30 or a 32, which is too large, I think. You need to downsize a little. I would also echo what Dr. Acar said - this is not a complex procedure - an annuloplasty is a more simple procedure. In fact, your cross-clamp times were longer in the replacement group than in the repair group, so I agree with your tendency to perform more annuloplasties.

DR. PRAVIN SHAH (USA): Chronic ischemic mitral regurgitation has two pathophysiologies: one is a restricted valve with uniform tethering of both leaflets, and another is where the tethering is more focal, more related to one side, to one papillary muscle more than the other. Did you look at the difference in pathophysiology in relation to which approach you made, and what the results were?

DR. BLANZOLA: I will answer your last question first. Yes, we did look at the pathophysiology, and found that we could stratify patients for dilated annulus or restricted leaflet motion, but occasionally we found a severe restricted motion of the leaflet, so we just changed the valve.

DR. ROBERT FRATER (USA): What technique was used in the replacement cases to preserve annular papillary connection? Was it posterior leaflet only, anterior and posterior, and was it used in every case?

DR. BLANZOLA: All the replacement procedures were performed by using some chordal-sparing techniques for the posterior annuli. This was performed from the very beginning, from about 10 years ago.

DR. MIHAJLOVIC BOGOLJUB (Yugoslavia): You said that patients with additional procedures were excluded from this study, but as we know, in chronic mitral regurgitation we also often have to perform tricuspid annuloplasty. What is your experience in this group of patients?

DR. BLANZOLA: We had some patients who underwent a tricuspid annuloplasty, but they were excluded. Among our chronic ischemic mitral regurgitation patients, there was normally about 20% with tricuspid insufficiency who received treatment. In this study we had two cases of elevated pulmonary artery pressure greater than 60 mmHg, but in those patients the tricuspid insufficiency was grade II, and the surgeons decided not to repair the tricuspid valve.