

Effect of Mitral Valve Surgery on Exercise Capacity, Ventricular Ejection Fraction and Neurohormonal Activation in Patients With Severe Mitral Regurgitation

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- OBJECTIVES** The purpose of this study was to prospectively investigate the effects of surgical correction of mitral regurgitation (MR) on exercise performance, cardiac function and neurohormonal activation.
- BACKGROUND** Little is known about the effect of surgical correction of MR on functional status or on neurohormonal activation.
- METHODS** Cardiopulmonary exercise test, radionuclide angiography and blood samples for assessment of neurohormonal status were obtained in 40 patients with nonischemic MR before and within one year (216 ± 80 days) after surgery. Twenty-four patients underwent mitral valve repair (MVr), and 16 underwent valve replacement (VR) with anterior chordal transection.
- RESULTS** Despite an improvement in New York Heart Association functional class, exercise performance did not change (peak oxygen consumption: 19.3 ± 6.1 to 18.5 ± 5.6 ml/kg/min, percentage of maximal predicted oxygen consumption: $79.5 \pm 18.2\%$ to $76.8 \pm 16.9\%$). After surgery, left ventricular (LV) ejection fraction (EF) decreased ($64.2 \pm 10.3\%$ to $59.9 \pm 11.4\%$, $p = 0.003$) while right ventricular (RV) EF increased ($41.4 \pm 9.6\%$ to $44.7 \pm 9.5\%$, $p = 0.03$). Left ventricular EF did not change after MVr ($64.3 \pm 11.5\%$ to $61.5 \pm 12.2\%$), but RVEF improved ($40.4 \pm 9.2\%$ to $46.0 \pm 10.0\%$, $p = 0.02$). In contrast, VR was associated with an impairment of LV function in the apicolateral area and a decrease in LVEF ($64.1 \pm 8.5\%$ to $57.4 \pm 10.0\%$, $p = 0.01$), whereas RVEF did not change ($42.9 \pm 10.3\%$ to $42.8 \pm 8.6\%$). Moreover, there was only a slight decrease in neurohormonal activation after surgery.
- CONCLUSIONS** Despite an improvement in symptomatic status, exercise performance was not improved seven months after either MVr or VR for MR, and neurohormonal activation persisted. Compared with MVr, VR resulted in a significant impairment of cardiac function in this study. (J Am Coll Cardiol 2000;36:2263-9) © 2000 by the American College of Cardiology

Surgical correction of mitral regurgitation (MR) aims to preserve cardiac function and to improve functional status and survival (1,2). Chronic MR results in a progressive deterioration in left ventricular (LV) contractile function (3,4) although the LV ejection fraction (EF) is maintained over a relatively long period. However, recent data in an animal model of MR (5), as well as in humans with chronic MR (1), suggest that LV contractile impairment recovers toward normal in most patients after mitral valve surgery. Although exercise capacity is an important parameter of quality of life, there is limited information regarding the effect of mitral valve surgery on exercise capacity (6-13), particularly in patients with MR (7-11). Moreover, whether preservation of LV function after mitral valve surgery results in an improvement of exercise capacity is unknown. Periph-

eral factors are important determinants of exercise performance for patients with heart failure (14-17). Patients with heart failure have neurohormonal activation, which may contribute to impairment of exercise performance by limiting exercise-induced vasodilatation or by contributing to maldistribution of peripheral blood flow. However, the effect of mitral valve surgery for severe MR on neurohormonal status is unknown.

Therefore, the purpose of this study was to assess the effects of mitral valve surgery on cardiac function, exercise performance with measurement of gas exchange and neurohormonal activation for patients with pure isolated nonischemic MR.

METHODS

Study patients. Forty consecutive patients who underwent successful mitral valve surgery for pure chronic nonischemic MR were studied. Patients were excluded from the study if they had significant pulmonary or coronary disease, other significant valve disease apart from tricuspid regurgitation, previous thoracic surgery or intermittent claudication severe enough to interfere with the performance of exercise testing. All patients had moderately severe (grade 3, 30 patients) or

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Abbreviations and Acronyms

ANP	= atrial natriuretic peptide
EF	= ejection fraction
LV	= left ventricle
MR	= mitral regurgitation
MVr	= mitral valve repair
NYHA	= New York Heart Association
PRA	= plasma renin activity
PVO ₂	= predicted value of maximal oxygen consumption
RV	= right ventricle
VO ₂	= oxygen consumption
VR	= mitral valve replacement

severe (grade 4, 10 patients) MR for at least six months. All patients gave informed consent, and the study protocol was approved by the Ethics Committee of the University.

Surgery. The decision regarding the type of corrective surgery was made by the cardiovascular surgeon on the basis of preoperative data and after assessment of the anatomic status of the mitral valve during surgery. Sixteen patients (40%) underwent mitral valve replacement (VR) with posterior chordal preservation and anterior chordal resection. St. Jude prosthetic valves were used in 14 patients and bioprosthetic valves (Carpentier-Edwards) in the remaining two patients. Twenty-four patients (60%) underwent mitral valve repair (MVr) using the technique described by Carpentier *et al.* (18). No patient had significant residual MR after surgery.

Radionuclide angiography. All patients underwent radionuclide angiography, cardiopulmonary exercise testing and blood sampling for assessment of neurohormonal status before and within one year (216 ± 80 days) after surgery. Radionuclide angiography was performed at rest in the supine position using red blood cells labeled *in vivo* with 20 mCi of technetium-99m. Data were acquired in the left anterior oblique view at 45° and 70° by means of a gamma camera (DST camera, Sopho Medical, data processing Sophy). All studies were formatted at 16 frames per cardiac cycle. RR intervals and heart rate (beats/min) were recorded. Cardiac cycles with RR intervals that were not within 20% of the average value were discarded. Left ventricle and right ventricle (RV) EF were determined using the equilibrium technique by automated detection of end diastolic and end systolic contours, with manual correction if necessary. The LV was divided into nine regions to analyze regional EF in the left anterior oblique view at 45°. Regions 1 and 9 correspond to the base of heart, regions 2 and 3 to the posterolateral region, 4, 5 and 6 to the lateral, apical and anteroapical region, respectively, 7 and 8 to the anteroseptal and septal region. All radionuclide angiograms were performed by the same investigator (C.F.) who was not aware of the type of surgery performed.

Cardiopulmonary exercise test. Exercise tests were performed in the morning after a light standard meal on an upright electromagnetically braked bicycle (Ergo-metrics

900, Ergoline GmbH, Bitz, Germany) using a continuous protocol (10 W/min). Beta-adrenergic blocking agents were withdrawn 48 h before exercise. Other medications were continued. Patients were asked to cycle at a constant rate of 60 revolutions per minute. Patients were allowed to continue until the respiratory exchange ratio (defined as the ratio between oxygen consumption and carbon dioxide production) was 1. Exercise was terminated at the point when the patient was unable to continue due to fatigue or dyspnea. Heart rate was continuously recorded on a 12-lead electrocardiogram (Case 15, Marquette Electronics Inc., Milwaukee, Wisconsin). Blood pressure was measured every 2 min and at peak exercise with a mercury sphygmomanometer. The data for gas exchange were collected on a breath-by-breath basis using a permanent zirconium oxide electrochemical cell, and the carbon dioxide fraction was measured by using a dual-beam infrared absorption chamber (CPX system, Medical Graphics Corporation, St. Paul, Minnesota). Both analyzers were calibrated by using standard gas mixtures before each exercise test. Anaerobic threshold was determined by the V slope method and by analyzing the ratio of minute ventilation against oxygen consumption (VO₂) and the ratio of minute ventilation against carbon dioxide production. Peak VO₂ and peak carbon dioxide production were defined as the highest value obtained during the last 2 min of exercise. Predicted value of maximal oxygen consumption (PVO₂) was calculated with use of the Wasserman equation, normalizing maximal VO₂ for age, gender, weight and height (19).

As a preoperative, LVEF $\geq 60\%$ is associated with an excellent outcome after surgery and the preservation of postoperative LVEF (20,21), patients were divided into two subgroups according to their preoperative LVEF ($<60\%$ and $\geq 60\%$), and the effect of mitral valve surgery on exercise performance was determined in each subgroup. To evaluate the effect of medications on exercise capacity and LV function, patients were also classified according to their postoperative medication status (unchanged or decreased).

Neurohormonal measurements. Blood samples were obtained at rest from a peripheral vein from patients and controls subjects ($n = 24$). Blood for norepinephrine was collected into heparinized tubes. Samples were collected into chilled tubes containing EDTA for measurement of plasma renin activity (PRA) and aldosterone and into chilled tubes containing EDTA and aprotinin for measurement of plasma levels of atrial natriuretic peptide (ANP) and of endothelin-1. Samples were kept on ice and centrifuged at 4°C within 30 min. All separated plasma samples were stored at -70°C until analysis. Plasma norepinephrine concentrations were measured by high performance liquid chromatography. The normal range was 100 to 600 pg/ml. Plasma renin activity was determined by estimation of the rate of generation of angiotensin I in plasma by radioimmunoassay. The normal range was 0.5 to 3 ng/ml per h. Plasma aldosterone concentrations were measured by radioimmunoassay using a commercially available kit (Behring,

VO₂, EF and Neurohormones in Mitral Regurgitation

Table 1. Baseline Characteristics in Patients Undergoing Mitral Valve Replacement and Patients Undergoing Mitral Valve Repair

	Mitral Valve Replacement	Mitral Valve Repair	p Value
n	16	24	
Men/Women	8/8	16/8	NS
Age (yrs)	65 ± 7	55 ± 13	0.007
NYHA functional class	2.31 ± 0.70	1.79 ± 0.66	0.02
Atrial fibrillation	6 (38%)	4 (17%)	NS
Etiology			
Myxomatous degeneration	8	19	
Rheumatic disease	5	1	0.05
Bacterial endocarditis	3	4	
Treatment			
Diuretics	12	11	
Digoxin	9	11	NS
ACE inhibitors	14	12	
Beta-blockers	1	3	

ACE inhibitors = angiotensin converting enzyme inhibitors; NYHA = New York Heart Association.

France). The normal range was 10 to 160 pg/ml. Plasma ANP and endothelin-1 concentrations were measured by radioimmunoassay after extraction on a Sep-Pak C-18 cartridge using a commercially available kit (Nichols Institute Diagnostics BV, the Netherlands). For determination of plasma endothelin concentrations, the assay was calibrated with endothelin-1. According to the manufacturer, the cross-reactivities to endothelin-2 and endothelin-3 were 52% and 96%, respectively, and the cross-reactivity to big-endothelin was 7%. The normal range of ANP was 5 to 25 pmol/l, and the normal range of endothelin-1 was 1.1 to 2.3 pmol/l.

Statistics. Results are expressed as mean ± standard deviation and as median (95% confidence interval) for neurohormonal values. Comparisons between groups were performed with chi-square tests or with paired or unpaired Student *t* tests, as appropriate. Nonparametric tests (Mann-Whitney *U* and Wilcoxon) were used to compare hormone levels. Comparison of exercise parameters between preoperative and postoperative groups was performed with use of a two-way repeated-measures analysis of variance. The interaction between time and operations were tested. A *p* value ≤0.05 was considered statistically significant.

RESULTS

Clinical data. Ten patients were in New York Heart Association (NYHA) functional class I (25%), 20 in class II (50%) and 10 in class III (25%) before surgery. Patients were more symptomatic before (mean NYHA class: 2.00 ± 0.72) than after operation (1.35 ± 0.53, *p* < 0.001). Ten patients were in atrial fibrillation before surgery and seven patients after surgery.

Table 1 gives the baseline characteristics of the two groups of patients. Patients who underwent VR were older than those who underwent MVr (*p* = 0.007) and were more symptomatic before (*p* = 0.02), but not after, surgery.

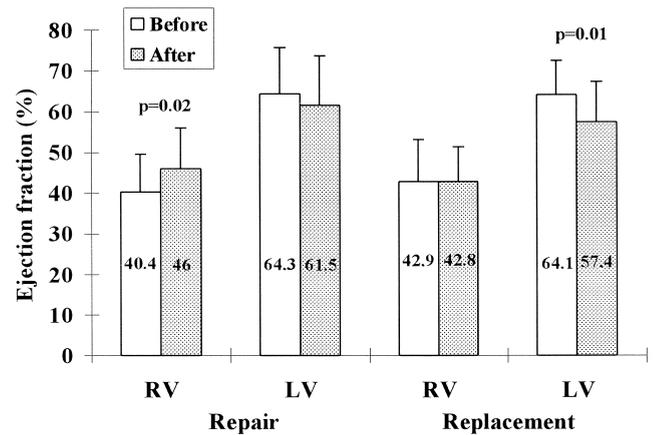


Figure 1. Changes in LV and RV ejection fraction according to the type of corrective surgery performed. LV = left ventricle; RV = right ventricle.

Myxomatous degeneration was the most frequent etiology of MR in the MVr group, whereas rheumatic disease was more frequent in the VR group. Aortic cross-clamp time and total cardiopulmonary bypass time were, respectively, 87 ± 29 min and 106 ± 32 min in the MVr group compared with 57 ± 27 min (*p* = 0.002) and 70 ± 24 min (*p* < 0.001) in the VR group.

Radionuclide angiography. Baseline RV and LV function were similar in the two patient groups (Fig. 1). After surgery LVEF decreased significantly from 64.2 ± 10.3% to 59.9 ± 11.4% (*p* = 0.003), whereas RVEF increased from 41.4 ± 9.6% to 44.7 ± 9.5% (*p* = 0.03). Mitral valve repair was associated with a significant increase in RVEF (*p* = 0.02), whereas LVEF did not change significantly. In contrast, patients who underwent VR had a significant decrease in LVEF (*p* = 0.01), whereas RVEF was unchanged. Moreover, there was a significant decrease (*p* < 0.05) in regional EF 4 and 5 (Fig. 2) of the LV after VR compared with MVr.

Cardiopulmonary exercise test. Despite correction of MR, there was no overall improvement in peak VO₂ (19.3 ± 6.1 ml/min/kg to 18.5 ± 5.6 ml/min/kg) or percentage of PVO₂ (percentage of PVO₂, 79.5 ± 18.2 to

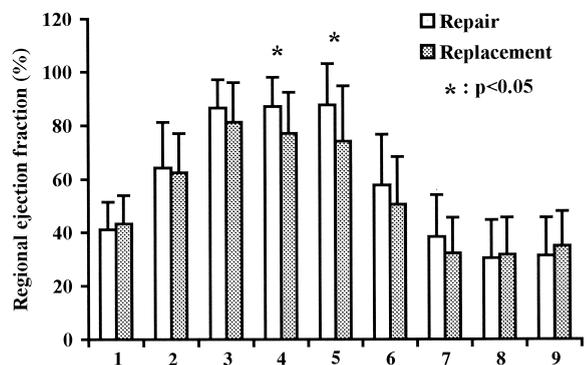


Figure 2. Analysis of regional ejection fraction of the left ventricle: there is a significant decrease in regional ejection fraction after mitral valve replacement in the regions 4 and 5 (apicolateral area) compared with mitral valve repair (*p* < 0.05).

Table 2. Effect of Surgery on Exercise Parameters in Patients Undergoing Mitral Valve Replacement and Patients Undergoing Mitral Valve Repair

	Mitral Valve Repair (n = 24)		Mitral Valve Replacement (n = 16)	
	Before	After	Before	After
Duration of exercise(s)	678 ± 246	605 ± 204*	483 ± 202	452 ± 172*
Peak VO ₂ (ml/min)	1,513 ± 461	1,408 ± 438	1,169 ± 392	1,201 ± 381
Peak VO ₂ (ml/kg/min)	21.5 ± 6.1	19.9 ± 5.9	16.1 ± 4.5	16.2 ± 4.3
% PVO ₂	82.0 ± 17.2	76.5 ± 16.6	75.7 ± 19.5	76.9 ± 17.9
Peak O ₂ pulse (ml/min/beat)	9.5 ± 3.0	9.3 ± 2.1	9.2 ± 3.8	9.2 ± 3.2
% of predicted value of maximal heart rate	98 ± 10	91 ± 11	85 ± 15	86 ± 14
Peak VE (l/min)	62.5 ± 19.1	58.2 ± 17.6	55.9 ± 16.5	54.2 ± 13.3

*p < 0.05 after surgery versus before surgery.
 peak O₂ pulse = peak oxygen pulse; peak VE = peak minute ventilation; % PVO₂ = percentage of predicted value of maximal oxygen consumption; VO₂ = oxygen consumption.

76.8 ± 16.9) after surgery in all patients, as well as in either the valve repair or the valve replacement group (Table 2). Indeed, there was a slight but significant decrease in mean exercise time (600 ± 247 s to 544 ± 204 s, p = 0.02). This decrease in exercise duration was similar in both groups. There was no difference before or after surgery between groups in maximal exercise tolerance assessed by peak O₂ pulse or by percentage of PVO₂. Using two-way repeated-measures analysis of variance, we found no interaction between time and operation type on exercise capacity. Moreover, there was no difference between patients classified according to preoperative LVEF (<60% in 14 patients and ≥60% in 26 patients) with respect to postoperative percentage PVO₂ (74.2 ± 16.5% vs. 77.1 ± 17.7%) or peak VO₂ (18.1 ± 6.9 ml/kg/min vs. 18.1 ± 4.7 ml/kg/min). Nor was there any difference in postoperative exercise performance when the patients were divided into two subgroups based on median postoperative LVEF (<60% and ≥60%).

Effect of medication on cardiac function and exercise tolerance. There was no significant difference in number of drugs prescribed between groups (repair or replacement) before or after surgery. However, there was a significant decrease in the number of drugs prescribed after surgery (p = 0.02). Fewer patients were taking diuretics (13 vs. 23 patients), digoxin (14 vs. 20 patients) or angiotensin-converting enzyme inhibitors (13 vs. 26 patients), whereas more were taking beta-blockers (14 vs. 4 patients). We divided the whole population into two

subgroups as a function of treatment modification (withdrawal of at least one drug compared with no modification). In the subgroup of patients without treatment modification (n = 18), peak VO₂ significantly decreased (19.4 ± 7.1 ml/kg/min to 17.1 ± 5.2 ml/kg/min, p = 0.04), but this decrease was not significant in the percentage of PVO₂ (80.8 ± 22.5% to 73.6 ± 17.6%) or in the absolute value of VO₂ max (1,328 ± 468 ml/kg to 1,209 ± 408 ml/kg). In the second subgroup of patients (n = 22), withdrawal of one or more drug had no effect on exercise capacity, suggesting that treatment status had a marginal effect or no effect on exercise capacity in our population. Moreover, we did not find any effect of treatment modification on LV and RVEF, with a similar decrease in LVEF and a similar increase in RVEF after surgery.

Neurohormonal activation. The results of preoperative and postoperative neurohormonal measurements are presented in Table 3, in conjunction with measurements in control subjects. There was a slight decrease in neurohormonal activation after surgery. The decrease in PRA and in plasma aldosterone levels is probably related to the withdrawal of angiotensin-converting enzyme inhibitors and diuretics after surgery. Plasma levels of ANP decreased after surgery (p = 0.03); however, all but one patient had persistently high concentrations of ANP (>25 pmol/l). Moreover, norepinephrine, PRA, aldosterone and ANP measurements were significantly higher than they were in control subjects both before and after surgery. No significant

Table 3. Neurohormone Levels Before and After Surgery

	Before	After	Controls (n = 24)
Norepinephrine (pg/l)	520 (400-650)†	430 (380-610)†	130 (100-260)
PRA (ng/ml/h)	3.1 (2.0-4.5)†	1.2 (0.9-2.2)*†	0.88 (0.56-1.22)
Aldosterone (pg/ml)	160 (118-180)†	93 (58-133)*†	78 (49-107)
ANP (pmol/l)	75 (47-128)†	62 (48-79)*†	14 (8-21)
Endothelin-1 (pmol/l)	2.1 (1.9-2.4)†	1.8 (1.6-2.2)	1.7 (1.2-2.1)

Data presented are median value (95% confidence interval). *p < 0.05 after vs. before; †p < 0.01 vs. control.
 ANP = atrial natriuretic peptide; PRA = plasma renin activity.

difference was observed between the patients who had VR and those who had MVr.

DISCUSSION

The purpose of this study was to determine whether successful mitral valve surgery for chronic MR affected exercise performance, ventricular EF or neurohormonal activation. The major, and somewhat unexpected, finding of the study was that exercise performance, assessed by cardiopulmonary exercise testing, was unchanged after surgery despite a significant improvement in NYHA functional status. Although MVr preserved cardiac function while VR resulted in a significant impairment of LV and RV function, exercise performance was not improved after either MVr or VR. Moreover, there was only a slight decrease in neurohormonal activation after surgery in our patients. This study also demonstrates the deleterious effect of anterior chordal transection during VR, which resulted in a significant impairment of regional LVEF in the area of chordal insertion.

Exercise capacity. To our knowledge, this is the first prospectively designed study that investigated exercise performance with a cardiopulmonary exercise test before and after either MVr or VR for pure chronic MR. There was previously only limited information regarding the effect of heart surgery on exercise capacity (6-13,22) and particularly the effect of mitral valve surgery in patients with chronic MR (7-11). Previous studies had small numbers of patients, with diverse cardiac pathology. In one study (22), myocardial revascularization was associated with an increase in work capacity after intensive cardiac rehabilitation. However, VO₂ was not measured and angina pectoris limited the preoperative exercise capacity. Most of the patients with valvular heart disease were in atrial fibrillation before surgery, and postoperative exercise capacity improvement may well have been related to conversion to sinus rhythm (8,9). Several studies reported a significant lower postoperative exercise capacity for patients undergoing mitral valve surgery (particularly after VR) compared with patients undergoing aortic valve surgery (6,10,12). Although individual variations were observed in our study, patients did not have an increase in peak VO₂ or percentage of PVO₂ seven months after surgery despite a significant improvement in NYHA functional class. In contrast with a previous study performed in a rehabilitation center without assessment of VO₂ (10), we found no improvement in either exercise duration or in peak workload. Indeed, mean exercise time decreased slightly but significantly after surgery (600 ± 247 s to 544 ± 204 s, $p = 0.02$). However, exercise time is a less accurate parameter than VO₂ to assess objective exercise capacity. Moreover, our results indicate that exercise performance did not improve after either MVr or VR for severe MR (Table 2).

Determinants of exercise capacity. There was no difference regarding peak VO₂ or percentage of PVO₂ between

patients with preoperative or postoperative LVEF values of more than 60% or less than 60%. Based on these results, LV function assessed by LVEF does not appear to be an important determinant of exercise capacity for patients with MR. Indeed, previous studies have demonstrated that peak VO₂ correlates weakly with LVEF in heart failure, leading to the general recognition that systolic function is not a key determinant of exercise capacity (23,24).

Diastolic dysfunction can lead to symptomatic heart failure despite normal systolic function (25). Thus, it is possible that persistent impairment of diastolic function limited the improvement in exercise capacity in our study. Indeed, despite normal postoperative value of peak filling rate, time to peak filling rate (assessed by radionuclide angiography) was slightly increased after surgery compared with normal patients (data not shown).

Pulmonary and peripheral factors may be of particular importance for patients with heart failure (14-17). Patients with heart failure develop skeletal muscle changes (15) and alteration of blood flow distribution (14,16). After cardiac surgery, there is likely to be additional muscle deconditioning and further impairment of lung dysfunction. After heart transplantation, the reversal of skeletal muscle abnormalities or of impaired vasodilatation is delayed for more than a year (26,27) and may contribute to the observed reduction in exercise capacity. Nakamura et al. (7) and Chiba et al. (8) reported in 11 and 23 patients, respectively, a significant correlation between the improvement in vascular function (endothelium-dependent peripheral vasorelaxation) and changes in peak VO₂ several months after mitral or aortic valve surgery.

Neurohormonal activation. Neurohormonal activation may contribute to the impairment of exercise performance in patients with heart failure by limiting exercise-induced vasodilatation or by contributing to maldistribution of peripheral blood flow. In the study of Nakamura et al. (7), the improvement in exercise capacity was associated with an important decrease in plasma ANP levels (167 to 41 pg/ml, with a normal range of 5 to 45 pg/ml). Left ventricular dysfunction is considered to be the main determinant of neurohormonal activation. Indeed, Starling's research on MR (unpublished data) demonstrated a strong inverse correlation between plasma norepinephrine levels and LV contractility ($r = -0.91$) in patients with MR. The persistent neurohormonal activation in this study probably reflects incomplete recovery of LV contractility seven months after surgery. Recent data in an animal model of MR (5) as well as in humans with chronic MR (1) indicate that LV contractile impairment recovers toward normal in most patients after mitral valve surgery despite the decrease in LVEF (2,28,29). Data concerning the timing of recovery of LV function after surgery are conflicting, requiring one to three years. However, patients in this study were evaluated at an average of seven months (216 ± 80 days) after surgery, which is consistent with an incomplete recovery of LV contractility.

Ventricular function. This study also demonstrates the deleterious effect of anterior chordal resection on cardiac function. Despite preservation of the posterior chordae tendinae, anterior chordal transection during VR was associated with a significant impairment of LV regional EF in the area of anterior papillary muscle insertion and a significant decrease in LVEF in this study. This result is consistent with previous studies that indicate that preservation of the mitral valve apparatus during mitral valve surgery for MR improves postoperative LV function (29-35). Impairment of LV regional function after chordal transection has been described previously (29,31,35). We, thus, believe that the significant postoperative differences we observed in ventricular function were likely due, in part, to anterior chordal transection. Moreover, our data demonstrates that RVEF was unchanged after VR but increased after MVr.

Study limitations. First, the lack of randomization between the two types of surgery is the main limitation of this study. The decision regarding the choice of corrective surgery was made by the cardiovascular surgeon on the basis of preoperative data and after assessment of the anatomic status of the mitral valve during surgery. However, since it is well established that MVr is the operation of choice for mitral valve disease (2,21,34), a randomized control trial may no longer be considered ethical. However, the two patients groups (MVr or VR) were similar with respect to preoperative radionuclide angiographic variables, exercise capacity and neurohormonal activation. There were only slight differences concerning age, etiology of MR and subjective functional capacity assessed with the NYHA classification. Second, it is possible that the lack of improvement in exercise capacity in this study was related to the relatively short-term follow-up period. Longer follow-up will be necessary to assess exercise performance two to three years after surgery.

Conclusions. In summary, the results of this prospective study indicate that exercise performance does not improve in most patients within one year of mitral valve surgery for severe chronic MR. The persistent impairment of exercise capacity does not appear to be related to LVEF or to the type of corrective surgery performed. The modest regression of neurohormonal activation in this study may contribute to the impaired exercise capacity. Finally, anterior chordal transection resulted in a significant impairment of regional and global LVEF.

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