Clinical Investigation

Revascularization Alone or Combined with Suture Annuloplasty for Ischemic Mitral Regurgitation

Evaluation by Color Doppler Echocardiography

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To determine the effectiveness of revascularization alone or combined with mitral valve repair for ischemic mitral regurgitation, we performed color Doppler echocardiography intraoperatively before and after cardiopulmonary bypass in 49 patients (mean age, 70 ± 9 years) with concomitant mitral regurgitation and coronary artery disease (triple vessel or left main in 88%; prior infarction in 90%). After revascularization alone (n=25), the mitral annulus diameter (2.88 ± 0.44 cm vs 2.88 ± 0.44 cm), leaflet-to-annulus ratio (1.44 ± 0.30 vs 1.44 ± 0.29), and mitral regurgitation grade (1.7 ± 0.9 vs 1.8 ± 0.7) remained unchanged (p=NS, postpump vs prepump); mitral regurgitation decreased by 2 grades in only 1 patient (4%). After combined revascularization and mitral valve suture annuloplasty (Kay-Zubiate; n=24), the annulus diameter decreased (to 2.57 ± 0.45 cm from 3.11 ± 0.43 cm), the leaflet-to-annulus ratio increased (to 1.46 ± 0.25 from 1.20 ± 0.21), and the mitral regurgitation grade decreased significantly (to 0.9 ± 0.9 from 2.8 ± 1.0) (p <0.01); mitral regurgitation decreased by 2 grades or more (successful repair) in 75%. The origin of the jet correlated with the site of prior infarction (p <0.05), being inferior in cases of posterior or inferior infarction (67%), and central or broad in cases of combined anterior and inferior infarction (70%). Despite a slightly higher 30-day mortality in the repair group (p=0.10), there was no significant difference in survival between the 2 surgical groups at 5 years or 8 years.

Therefore, in this study of patients with mitral regurgitation and coronary artery disease, reduction in regurgitation grade with revascularization alone was infrequent. Concomitant suture annuloplasty significantly reduced regurgitation by reestablishing a more normal relationship between the leaflet and annulus sizes. The failure rate after suture annuloplasty was 25%; alternative repair techniques such as ring annuloplasty may have a lower failure rate. ***(Tex Heart Inst J 1996;23:270-8)***

Key words: *Comparative study; coronary artery bypass; echocardiography; mitral valve insufficiency/surgery*

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Mitral regurgitation is a common finding in patients with ischemic heart disease, and may occur in more than 30% of patients after myocardial infarction.[1-3] When patients come to surgical attention on the basis of their coronary artery disease, the question of what to do with associated mitral regurgitation becomes an important clinical consideration. Because of the high operative mortality of 20% or greater in patients undergoing combined mitral valve replacement and revascularization for ischemic mitral regurgitation,[4-13] revascularization alone or in combination with mitral valve repair is often considered as an alternative surgical approach.

The purpose of the present study was to evaluate 2 surgical treatment strategies for ischemic mitral regurgitation: revascularization alone, and revascularization combined with suture annuloplasty of the mitral valve. Intraoperative color Doppler echocardiography was used to determine the impact of each surgical procedure on the severity of mitral regurgitation, the mitral annular diameter, and mitral leaflet-to-annulus ratio.

Methods

Patients. The study population consisted of 49 patients (21 women, 28 men) with coronary artery disease (CAD) and mitral regurgitation who were studied in

a prospective manner with Doppler color-flow mapping intraoperatively and up to 64 weeks postoperatively. Combined revascularization and mitral valve repair were performed in 24 patients; revascularization alone was accomplished in 25. Patients whose mitral valve disease was primarily rheumatic, myxomatous, infectious, or congenital were excluded. Patients with mitral regurgitation due to papillary muscle rupture were also excluded. No patient had echocardiographic, angiographic, or hemodynamic evidence of intracardiac shunt or aortic valve disease.

Coronary artery disease was defined as luminal diameter narrowing of 50% or more in at least 1 coronary artery from angiographic studies performed preoperatively in all patients. The extent of coronary disease was classified according to the number of affected major coronary vessel distributions (1, 2, or 3) and the presence or absence of left main stenosis. The presence of mitral regurgitation was confirmed angiographically and was graded semiquantitatively (0 to 4+) on the basis of left atrial opacification during the right anterior oblique left ventricular cineangiogram.[14,15] The left ventricular ejection fraction was calculated by the area–length method from the right anterior oblique left ventricular cineangiogram.[16,17]

Prior myocardial infarction was determined from surgical findings of localized epicardial scarring and thinning and the presence of pathologic Q waves (>80 msec) in 2 or more leads on the 12-lead electrocardiogram (transmural infarction); or from a documented history of prolonged (>30 min) chest pain associated with an abnormal creatine kinase MB level (>20 IU/mL) within 48 hours and ST segment or T wave changes in 2 or more leads on the 12-lead electrocardiogram (nontransmural infarction). The location of the infarction was determined from the surgical or electrocardiographic findings.

Intraoperative Doppler Color-Flow Mapping. Real-time 2-dimensional imaging of intracardiac structure and blood flow was performed with a 3.5-MHz transducer placed directly on the epicardial surface after being coupled with ultrasound gel and then covered with a sterile plastic sheet. Epicardial imaging was performed in views similar to those obtained by transthoracic closed-chest imaging (i.e., parasternal-equivalent long- and short-axis views), so that comparison could be made with the postoperative imaging. Image processing and display were accomplished with an Aloka 880 (Aloka Co., Ltd.; Tokyo, Japan) or HP Sonos 1000 (Hewlett-Packard Co.; Andover, Mass.) color Doppler system, and the images were recorded on 1/2" videotape. Imaging was performed immediately before and after cardiopulmonary bypass; simultaneously, arterial and pulmonary capillary wedge pressures were recorded from radial and pulmonary arterial catheters.[18] Imaging and hemodynamic measurements were repeated with infusion of phenylephrine (mean dose, 135 μg) if the systolic arterial pressure was more than 15 mmHg below the base-line level at preoperative cardiac catheterization. Follow-up closed-chest transthoracic imaging was performed in most patients before hospital discharge and up to 64 weeks postoperatively.

The mitral annulus diameter was measured from still-frames obtained during diastole in a parasternal-equivalent long-axis view. From the same frame, the lengths of the anterior and posterior leaflets of the mitral valve were determined; the length of each leaflet was measured from the mitral annulus to the leaflet tip at maximal opening of the mitral valve (Fig. 1). The lengths of the anterior and posterior leaflets were added together and divided by the diameter of the mitral annulus, to form a leaflet-to-annulus ratio. The left ventricular end-diastolic diameter was measured at the level of the mitral leaflet tips.

Mitral regurgitation was graded semiquantitatively on a scale of 0 to 4+ by using a visual estimation of the regurgitant jet area in relation to the left atrial area, modified from Nanda's criteria.[19] A grade of 1+ was assigned to a regurgitant jet extending only just behind the mitral valve; 2+, up to 20% of the left atrial area; 3+, up to 40%; and 4+, more than 40% of the left atrial area.

Using multiple short-axis views at and just behind the level of the valve, we determined the origin of the regurgitant jet by observing its position in relation to the commissure of the mitral valve. Central jets originated from the middle third of the commis-

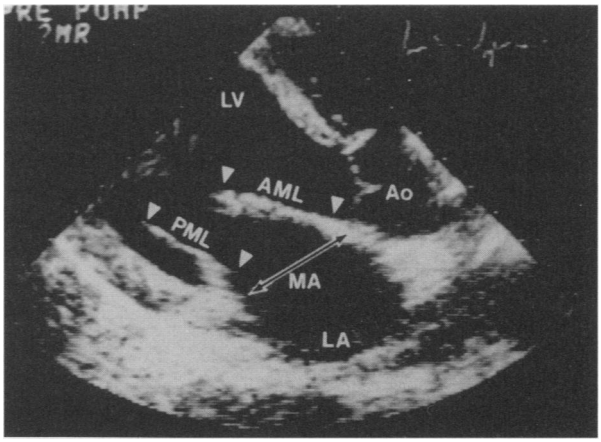

Fig. 1 *Long-axis parasternal-equivalent view during diastole, illustrating measurement of mitral annulus diameter and anterior and posterior mitral leaflet lengths.*

AML = anterior mitral leaflet length; Ao = aorta; LA = left atrium; LV = left ventricle; MA = mitral annulus diameter; PML = posterior mitral leaflet length

sure (Fig. 2); inferior jets from the lower third of the commissure (adjacent to the diaphragmatic wall of the left ventricle); and superior jets from the upper third of the commissure (just below the aortic valve). If the jet originated from at least 2 contiguous sections, or if multiple jets were imaged from different sections, the jet was categorized as "broad."

Surgical Procedure. Coronary artery bypass grafting was accomplished with reversed saphenous vein or the internal thoracic artery. Ischemic mitral regurgitation was repaired by the suture annuloplasty technique of Kay and Zubiate,[5,20] with placement of the sutures at the anterior or posterior (or both) commissures of the mitral valve. Myocardial protection consisted of moderate systemic hypothermia (20-25 °C), topical cooling of the myocardium with electrolyte solution (Normosol) at 4 °C, and intermittent multidose potassium cardioplegia with either crystalloid (St. Thomas Hospital) or a modified blood solution infused into the aortic root at 4 °C.

In patients who underwent mitral valve exploration and suture annuloplasty (n=24), the etiology of valve disease was determined from direct visual inspection of the mitral valve leaflets, annulus, chordae tendineae, and papillary muscles. If the valve was not repaired (n=25), the etiology was determined from echocardiographic findings. An ischemic etiology was presumed if the mitral leaflets and chordae appeared normal but there were findings of papillary muscle infarction or thinning (with or without rupture), papillary muscle ischemia, or mitral annular dilatation associated with left ventricular dilatation and healed infarction(s). Myxomatous degeneration was defined by findings of redundant, scalloped mitral leaflet tissue, elongated chordae, or chordal rupture. A mixed etiology was assigned when findings were consistent with both myxomatous and ischemic causes.

Decisions regarding mitral valve exploration and annuloplasty were made on the basis of preoperative and surgical findings, and at the discretion of the surgeon. Decisions were not made on the basis of the color Doppler findings; the frequency with which surgical practice changed as a consequence of intraoperative Doppler echocardiographic findings could not be determined from this study.

Postoperative Follow-up. After surgery, follow-up was performed by means of a yearly questionnaire, telephone interview, or examination in our offices.[4] All patients received follow-up for 8 years, unless death occurred. No patient was lost to follow-up.

Statistical Methods. Comparisons of continuous variables between 2 groups were made with the unpaired 2-tailed Student's *t*-test, or with the nonparametric Mann-Whitney U-test if the data were from non-Gaussian or heteroscedastic (F test for variance, $p < 0.05$) populations. Comparisons of continuous variables among 3 or more groups were carried out using parametric or nonparametric analysis of variance (ANOVA) techniques, followed by the appropriate multiple comparison statistic if the ANOVA yielded a p value of less than 0.05. Categorical variables were compared using the χ^2 test or Fisher's exact test in instances where expected cell frequencies were inadequate. Continuous variables are summarized as mean ± 1 standard deviation, and categorical variables are summarized as frequency and percent. Survival rates (and standard errors) were calculated from actuarial survival curves using the life-table method, and all deaths were included in the analysis.[4] Differences between survival curves were assessed with the Mantel-Cox statistic.

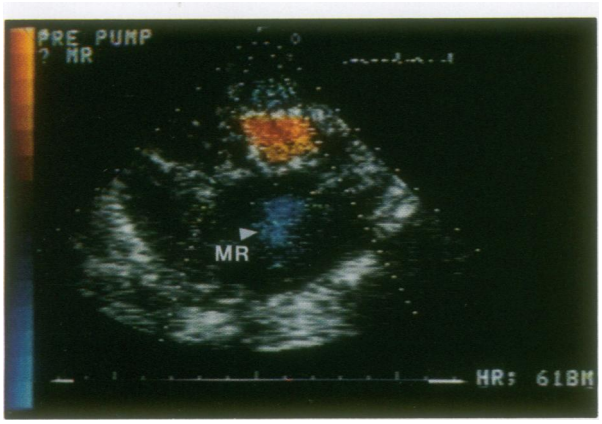

Fig. 2 *Short-axis parasternal-equivalent view of left atrium just behind the mitral valve, demonstrating central blue jet of regurgitation. Antegrade systolic flow through the aortic valve is seen at top of image.*

MR = mitral regurgitant jet

Results

Patients. The clinical features of the 2 surgical groups (revascularization combined with suture annuloplasty of the mitral valve, and revascularization alone) are summarized in Table I. Both groups were elderly, with mean ages of 72 and 69, respectively (p=NS). Gender distribution was not significantly different between the 2 groups. Most patients (54% and 64%, respectively) presented with progressive angina. Smaller percentages, 38% and 24% respectively, presented with heart failure. The extent of coronary disease was similarly distributed within each group, being triple vessel or left main CAD in 88% of both.

A prior infarction was documented by electrocardiography or by enzyme criteria in 92% and 88%, respectively (p=NS). In 86% of both groups, the location of the infarct was inferior or posterior, either alone or in combination with an anterior infarct. The

TABLE I. Clinical Characteristics of Patient Population

Clinical Characteristics	CABG + MVr (n=24)	CABG Alone (n=25)	P Value
Age (years)			
Mean ± SD	72 ± 10	69 ± 8	NS
Range	50-91	50-80	
Sex			
Male	16 (67%)	12 (48%)	NS
Female	8 (33%)	13 (52%)	
Symptoms			
Progressive angina	13 (54%)	16 (64%)	NS
Heart failure	9 (38%)	6 (24%)	
Stable angina	1 (4%)	2 (8%)	
Sudden death	1 (4%)	1 (4%)	
Extent of coronary disease			
Single or double vessel	3 (12%)	3 (12%)	NS
Triple vessel or left main	21 (88%)	22 (88%)	
Prior infarction			
Yes	22 (92%)	22 (88%)	NS
No	2 (8%)	3 (12%)	
Location of infarct			
Anterior	3 (14%)	3 (14%)	NS
Inferior/posterior	9 (41%)	11 (50%)	
Both	10 (45%)	8 (36%)	
Ejection fraction			
Mean ± SD	40 ± 19	49 ± 17	NS
Range	10-85	17-75	
Interval, catheterization to surgery			
>1 week	11 (46%)	10 (40%)	NS
1-7 days	13 (54%)	14 (56%)	
<1 day	0	1* (4%)	

CABG = coronary artery bypass grafting; MVr = mitral valve repair by suture annuloplasty; SD = standard deviation

*Failure of percutaneous transluminal coronary angioplasty

mean ejection fractions were not significantly different between the 2 groups. The surgical procedure was carried out electively or semi-electively in nearly all patients; emergency surgery was required in only 1 patient, due to failure of balloon angioplasty. No patient presented in cardiogenic shock.

Echocardiographic and Doppler Findings. Prior to surgery (prepump), the leaflet-to-annulus ratio was substantially lower in the repair group (p=0.002, Table II), reflecting a somewhat larger mitral annulus and smaller leaflet length in the repair patients. The prepump mitral regurgitation (MR) grade was significantly more severe in the repair group (median 3+ and mean 2.8 ± 1.0) when compared with the revascularization group (median 2+ and mean 1.8 ± 0.7) (p=0.001). Of the 24 repair patients, 6 had 4+ MR, 11 had 3+ MR, 6 had 2+ MR, and 1 had 1+ MR. Of the 25 revascularization-alone patients, 5 had 3+ MR, 11 had 2+ MR, and 9 had 1+ MR.

After suture annuloplasty of the mitral valve, the mitral annulus diameter was significantly reduced, and the leaflet-to-annulus ratio was indistinguishable from that in the revascularization-alone group. No significant changes in the mitral annulus diameter and leaflet-to-annulus ratio occurred after revascularization alone (Table II).

In order to determine whether left ventricular filling conditions were matched between the 2 surgical groups before and after the procedure, the left ventricular end-diastolic diameter (LVEDD) was evalu-

TABLE II. Echocardiographic and Doppler Findings

Clinical Characteristics	CABG + MVr (n=24)	CABG Alone (n=25)	P Value
Mitral leaflet length (cm)			
Anterior + posterior	3.68 ± 0.57	4.09 ± 0.75	0.04
Mitral annulus diameter (cm)			
Prepump	3.11 ± 0.43*	2.88 ± 0.44	0.07
Postpump	2.57 ± 0.45	2.88 ± 0.44	0.02
Leaflet-to-annulus ratio**			
Prepump	1.20 ± 0.21*	1.44 ± 0.29	0.0002
Postpump	1.46 ± 0.25	1.44 ± 0.30	NS
LVEDD (cm)			
Prepump	4.11 ± 0.81	4.11 ± 0.79	NS
Postpump	4.13 ± 0.75	4.12 ± 0.79	NS
MR grade			
Prepump	2.8 ± 1.0	1.8 ± 0.7	0.0001
Postpump	0.9 ± 0.9	1.7 ± 0.9	<0.05

CABG = coronary artery bypass grafting; LVEDD = left ventricular end-diastolic diameter; MR = mitral regurgitation; MVr = mitral valve repair by suture annuloplasty

* Significantly different from postpump value ($p < 0.01$)
** Calculated as (anterior + posterior leaflet length)/mitral annulus diameter

ated. There was no significant change from prepump to postpump, and LVEDD did not differ between the 2 groups. Concomitant myxomatous leaflet changes (redundancy, ballooning, scalloping, or minor degrees of leaflet thinning or thickening) were found in 5 (21%) of the repair group and in 4 (16%) of the revascularization-alone group, not a statistically significant difference. Ancillary leaflet procedures (chordal shortening, leaflet resection, papillary muscle reimplantation) were not performed.

Effect of Surgical Procedure on Severity of Mitral Regurgitation. In patients who underwent revascularization alone (Fig. 3), there was no significant difference in regurgitation grade from prepump (median 2+ and mean 1.8 ± 0.7) to postpump (median 2+ and mean 1.7 ± 0.9), or up to 64 weeks postoperatively (median 2+ and mean 1.6 ± 1.2) (p=NS). As a control, 3 patients with no mitral regurgitation were also studied and demonstrated no new appearance of regurgitation, postpump or postoperatively. Reduction in regurgitation severity by 2 grades or more occurred in 1 patient (4%).

After revascularization combined with suture annuloplasty of the mitral valve (Fig. 4), mitral regurgitation grade was significantly reduced from prepump (median 3+ and mean 2.8 ± 1.0) to postpump (median 1+ and mean 0.9 ± 0.9) (p=0.002). There was no further change, on average, up to 24 weeks postoperatively (median 1+ and mean 1.0 ± 1.0).

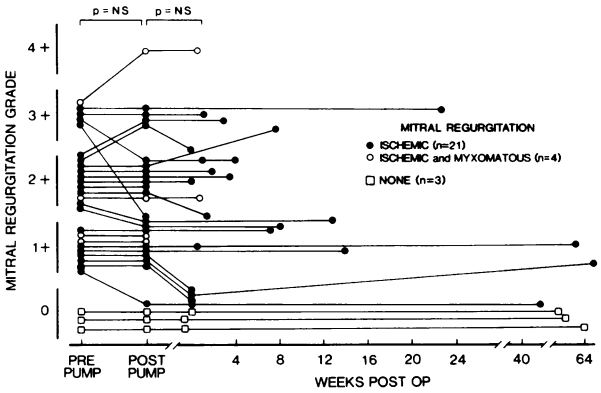

Fig. 3 *Longitudinal study of mitral regurgitation severity before (prepump) and after (postpump, postop) revascularization alone. There was a 2 grade or greater reduction in mitral regurgitation in only 1 patient (from 3+ prepump to 1+ postpump).*

Despite beginning with more severe mitral regurgitation preoperatively ($p < 0.01$), patients undergoing the combined procedure experienced less mitral regurgitation immediately postpump ($p < 0.05$).

Successful and Unsuccessful Repairs. In 18 (75%) of the 24 repair patients, a reduction of 2 or more regurgitant grades (defined as a successful repair) was observed at the latest postoperative follow-up, while 3 patients (12.5%) had a reduction of 1 grade and 3 (12.5%) had no change or worsening of regur-

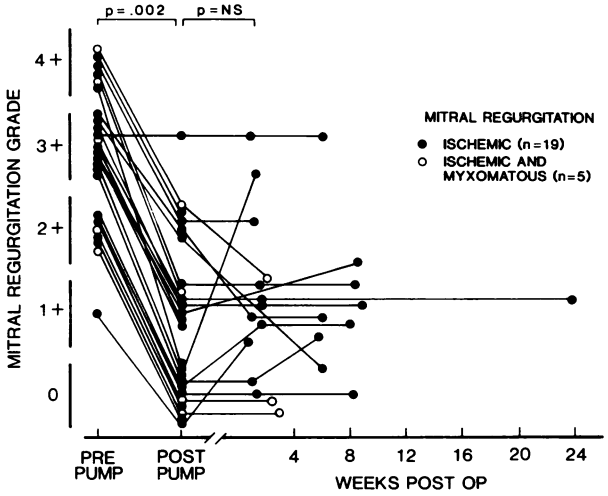

Fig. 4 *Longitudinal study of mitral regurgitation severity before (prepump) and after (postpump, postop) revascularization combined with suture annuloplasty of the mitral valve.*

gitation (failures) when compared with the prepump color Doppler study.

None of the failed cases showed evidence of concomitant myxomatous leaflet changes. Of the 6 failures, 1 case was initially successful (3+ prepump to 0 postpump) but the patient developed a postoperative inferior infarction, resulting in reappearance of regurgitation (3+; Fig. 4). In another case, the patient began with mild (1+) regurgitation, had none postpump, and redeveloped mild regurgitation postoperatively without an intervening cardiac event. In the 3rd case, the patient had no change in regurgitation grade (3+) from prepump to postpump (Fig. 4); this was an elderly patient with poor left ventricular function, for whom a decision was made not to replace the valve. Of the remaining cases, 2 patients began with 3+ or 4+ regurgitation, and 1 had 2+ regurgitation prepump.

Of the 5 patients with concomitant myxomatous leaflet changes, 4 (80%) had regurgitant jets that originated from the center (middle third) of the mitral commissure, and 1 patient had an inferior origin of the jet. After excluding from the repair group these 5 patients with surgically confirmed myxomatous leaflet changes, we had 19 patients with purely ischemic mitral regurgitation.

Correlation of Jet Origin with Site of Infarction. Among the repair patients with purely ischemic regurgitation, the origin of the regurgitant jet correlated with the site of prior infarction (Table III, $p < 0.05$). Seven (78%) of 9 patients with inferior jets had a prior inferior (or posterior) infarction, alone (67%; 6 patients) or in combination with an anterior infarction (11%; 1 patient). Conversely, 8 (80%) of 10 patients with central or broad jets had a prior anterior infarction, alone (10%; 1 patient) or in combination with an inferior (or posterior) infarction (70%; 7 patients). Therefore, patients with inferior jets usually had a prior inferior or posterior infarction, whereas patients with central or broad jets usually had 2 prior infarcts, involving the anterior and inferior (or posterior) territories.

Postoperative Survival. At 8 years, there was a total of 12 deaths in the 24 patients who had revascularization combined with suture annuloplasty of the mitral valve, and 12 deaths in the 25 patients who had revascularization alone. There were 3 deaths (12.5% mortality) within 30 days in the repair group, and survival rates (± standard errors) were 50% ± 10% at 5 years and at 8 years. In the group that underwent revascularization alone, there were no deaths within 30 days ($p=0.10$ by Fisher's exact test, in comparison with the repair group); and the survival rates were 58% ± 10% at 5 years and 49% ± 10% at 8 years ($p=0.56$ in comparison with the repair group). One patient in each group underwent mitral valve replacement during the follow-up period.

Discussion

When mitral regurgitation and coronary artery disease coexist, the regurgitation may be causally related to the coronary disease (i.e., it is ischemic mitral regurgitation); or it may be caused by an independent pathologic process such as myxomatous degeneration, rheumatic disease, a congenital anomaly, or infection. In the latter situations, the pathologic process involves the valve leaflets or chordae tendineae predominantly or solely. In pure cases of ischemic mitral regurgitation, the valve leaflets and chordae are usually structurally normal, and regurgitation is caused by ischemia, infarction, or rupture of papillary muscle, by infarction of the adjacent myocardium, or by mitral annular dilation and left ventricular enlargement as a result of infarction.[21] A mixture of ischemic and nonischemic causes may exist, as occurred in 9 (18%) of the 49 patients in this study.

The surgical approach to ischemic mitral regurgitation has been problematic. In a long-term study of more than 2,000 patients who underwent revascularization alone, uncorrected mitral regurgitation nearly doubled the risk of late death.[22] With mitral valve replacement and revascularization, operative mortality has been 19% to 40% and late (8-year) survival has been only 37%.[4-13] The very high early and late mortality may be related to the drop in ejection fraction after mitral valve replacement, which may be poorly tolerated in patients with coronary artery disease and previous infarctions. Other possible causes of this high early and late mortality include the rigid stiffening of the mitral annulus, which impairs normal basal wall motion; the loss of the teth-

TABLE III. Correlation of Jet Origin with Site of Prior Infarction*

Origin of Jet	Site of Prior Infarction		
	Inferior**	Inferior** and Anterior	Anterior
Inferior (n=9)	6 (67%)	1 (11%)	2 (22%)
Central or broad (n=10)	2 (20%)	7 (70%)	1 (10%)

* Valve repair patients with myxomatous leaflet changes excluded (n=5), leaving 19 patients with purely ischemic mitral regurgitation
** Inferior = inferior or posterior

ering function of the subvalvular apparatus, when the mitral valve is excised; prosthesis-related complications, such as thromboembolism and infection; and the frequent requirement for long-term anticoagulation.[4-13,23-33]

Reparative procedures of the mitral valve, in comparison with valve replacement, have been associated with enhanced survival and with a lower rate of complications, especially thromboembolism and warfarin-related hemorrhage.[12,13,29,30-33] Therefore valve repair, when feasible, offers an acceptable and possibly superior alternative to valve replacement. Experiences with repair techniques for ischemic mitral regurgitation have been limited, however, and have been reported from only a few centers, which used different approaches.[5,12,13,30,34] Common to the repair approaches has been a remodeling of the mitral annulus, for which 2 techniques have been used: Carpentier ring annuloplasty[30,34] and Kay-Zubiate suture annuloplasty.[5,12,13] In the current study, the suture annuloplasty technique was investigated.

In comparison with patients who underwent revascularization alone, the repair patients in this study had a significantly lower leaflet-to-annulus ratio before surgery (Table II), suggesting a disproportionately large mitral annulus in relation to the mitral leaflets. This supports previous observations of a dilated mitral annulus in patients undergoing repair for ischemic mitral regurgitation.[5,12,13] Importantly, the diameter of the mitral annulus was significantly reduced after suture annuloplasty, and the mean leaflet-to-annulus ratio increased, becoming indistinguishable from that of the control group (Table II). Concomitantly, the mitral regurgitation grade by color Doppler was reduced significantly (median 3+ to 1+, Fig. 4). As a consequence, the suture annuloplasty, when successful, accomplishes an effective repair by a significant reduction in the size of a dilated mitral annulus and reestablishment of a more normal relationship between the size of the annulus and the size of the leaflets.

Suture annuloplasty possesses several advantages over ring annuloplasty. The suture technique is simpler to perform, and insertion of a large foreign body is avoided. Antibiotic prophylaxis and a 3-month course of warfarin anticoagulation may not be required. Left ventricular outflow tract obstruction has not been reported with suture annuloplasty, as it has with ring annuloplasty.[35]

Suture annuloplasty may be considered when the valve leaflets and chordae tendineae are structurally normal, as in most patients with ischemic mitral regurgitation in this study. When additional reparative procedures are required, such as leaflet resection, chordal transposition, chordal shortening, or papillary muscle reimplantation, a ring annuloplasty as described by Carpentier[30] may be more suitable. Nonetheless, suture annuloplasty has been reported to yield acceptable results even when these additional reparative procedures are required.[13,36]

When revascularization alone was performed, no significant change in mitral regurgitation grade was observed postpump or postoperatively (Fig. 3). On an individual basis, only 1 patient (4%) experienced a 2 grade or greater reduction in regurgitation. Thus, reduction in ischemic mitral regurgitation with revascularization alone was distinctly unusual.

Papillary muscle dysfunction resulting in severe mitral regurgitation and pulmonary edema has been reported to occur with episodic, reversible ischemia in the absence of infarction.[37] Reduction or elimination of mitral regurgitation may be expected if the ischemia is relieved by revascularization. Reduction in mitral regurgitation may also occur in patients with substantial amounts of "hibernating" myocardium (that is, viable myocardium that is dysfunctional at rest due to ischemia), particularly when the area involves the papillary muscle. Indeed, this appeared to be the mechanism of significant reduction in mitral regurgitation after revascularization in 1 patient in this study (Fig. 3) who presented with recent onset of severe unstable angina but without evidence of acute infarction.

However, many patients with ischemic mitral regurgitation have infarction of the papillary muscle and adjacent myocardium. Experimental studies

have demonstrated that isolated papillary muscle necrosis without infarction of the adjacent myocardium does not produce mitral regurgitation.[34,38-40] This implies that papillary muscle necrosis must be combined with dysfunction of the adjacent myocardium in order to produce regurgitation.[21] In support of this, we observe that 44 (90%) of the 49 patients in this study had evidence of prior myocardial infarction (Table I). Significant reduction in mitral regurgitation severity may not occur, because substantial areas of the myocardium have undergone irreversible necrosis; this may explain the lack of reduction in regurgitation for nearly all (24 of 25) patients who underwent revascularization alone in this study (Fig. 3). Moreover, other mechanisms may cause mitral regurgitation, including left ventricular enlargement (due to infarction) and mitral annular dilation; revascularization alone would not be expected to improve wall motion or mitral regurgitation in these situations. Acute, severe mitral regurgitation with cardiogenic shock is potentially reversible during an evolving acute myocardial infarction, if reperfusion is achieved early;[41,42] however, no such patients were included in the present study.

The results of the present study (showing a 2 grade or greater reduction in mitral regurgitation in only 4% of patients after revascularization alone) therefore suggest that there are populations of patients who do not experience routine reduction in mitral regurgitation after revascularization. Identification of hibernating myocardium in patients with ischemic mitral regurgitation may differentiate those likely to experience reduction in mitral regurgitation after revascularization alone. Further studies are needed to clarify these issues.

There was no significant difference in long-term survival between the repair group and the revascularization group (50% ± 10% vs 58% ± 10% at 5 years, respectively, and 50% ± 10% vs 49% ± 10% at 8 years; p=0.56). Hence, the somewhat higher 30-day mortality in the repair group (12.5% vs 0% in the revascularization alone group; p=0.10 by Fisher's exact test) may be acceptable, and compares favorably with the higher operative mortality of 19% to 40% after mitral valve replacement combined with revascularization.[4-13] The somewhat higher operative mortality in the repair group may be due to the longer ischemic time required for mitral valve exploration and annuloplasty, to an effect of annuloplasty on left ventricular function, or to a reduction in ejection fraction with lessening or elimination of mitral regurgitation.

In the present study, the origin of the regurgitant jet could be located in relation to the mitral commissure. The jet origin correlated with the site of prior infarction in a high proportion of cases (Table III), the jet usually being inferior in cases of prior posterior or inferior infarction (67%), and central or broad in cases of infarctions involving both the anterior and inferior (or posterior) territories (70%). Hence, ischemic mitral regurgitation may be caused by dilation of the portion of the annulus adjacent to the infarcted area.

Nevertheless, in a previous study,[43] success or failure of the repair procedure did not correlate with the origin of the regurgitant jet. None of the failures in the present study involved myxomatous valves. Our failure rate of 25% is therefore not attributable to any factor other than the surgical technique. Alternative surgical techniques of repair, such as ring annuloplasty, may be associated with lower failure rates.[43]

There was no significant change in mitral regurgitation grade between early (postpump) and late (postoperative) studies, regardless of the surgical procedure performed (revascularization alone, Fig. 3; or revascularization combined with suture annuloplasty, Fig. 4). Consequently, excessive regurgitation is reliably detected immediately after termination of cardiopulmonary bypass and completion of rewarming, which allows remedial action prior to chest closure. When there is excessive regurgitation after suture annuloplasty, the repair may be revised by placement of a prosthetic ring (with or without adjunctive procedures such as leaflet resection) or by valve replacement.

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