JACC Vol. 16, No. 7
December 1990:1575–8

Decreased Operative Risk of Surgical Treatment of Mitral Regurgitation With or Without Coronary Artery Disease

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The consecutive 2 year experience with patients undergoing first-time surgery for mitral regurgitation with and without coronary artery disease was reviewed. From January 1988 to January 1990, 127 patients with pure mitral regurgitation undergoing first-time operation were surgically treated. No other valve lesion, no reoperation and no congenital defects were included. The mean patient age was 62 years with 26% of the patients >70 years. Twenty-six percent of the entire group was in functional class IV. Seventy-five patients received mitral valve repair and 52 underwent mitral valve replacement with a St. Jude or Hancock valve. In patients undergoing mitral valve repair, there was a higher incidence of those >70 years old and of coronary artery disease and in patients undergoing mitral valve replacement there was a higher incidence of functional class IV.

The operative mortality rate was 2.3% (3 of 127 patients). No patient failed to be discontinued from cardiopulmonary bypass and all three deaths occurred after mitral valve replacement, with one from complications of chronic renal failure and dialysis. There was no significant difference in patients who either did or did not have a concomitant coronary artery bypass graft and there was no difference related to age or functional class. Postoperative complications occurred in five patients in the valve repair group, including recurrent mitral regurgitation in two necessitating reoperation, and in three patients in the valve replacement group.

With newer operative and postoperative management techniques, especially preservation of the papillary muscle annular continuity, the risk of mitral valve surgery, particularly of valve repair, is considerably lower than in previous years.

(J Am Coll Cardiol 1990;16:1575–8)

The surgical treatment of mitral regurgitation, particularly in patients with concomitant coronary artery disease, has, in the past, been associated with a relatively high hospital operative risk (1–3). With newer operative techniques, better myocardial protection and increasing percentage of patients undergoing repair rather than replacement of the mitral valve, we believe that surgical therapy of mitral regurgitation regardless of degree of associated coronary artery disease is now a lower risk procedure. For the purpose of updating the current in-hospital risk of procedures for mitral regurgitation, we have surveyed our consecutive 2-year experience with patients undergoing first-time surgery for mitral regurgitation with and without coronary artery disease.

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Manuscript received April 16, 1990; revised manuscript received May 29, 1990, accepted June 14, 1990.

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Methods

Study patients. From January 1988 to January 1990, 127 consecutive patients with pure mitral regurgitation undergoing a first-time operation were surgically treated at the Brigham and Women's Hospital. Excluded were patients who had reoperation for mitral regurgitation, those who had mitral regurgitation treated in conjunction with any other cardiac valve lesion including aortic or tricuspid disease or congenital defects or those who were in cardiogenic shock from an acute myocardial infarction. Except for these conditions, no patient was excluded. The diagnosis of mitral regurgitation was made by echocardiogram or cardiac catheterization, or both, using standard techniques. Coronary angiography was done in all patients >40 years old or with a chest pain syndrome.

Table 1 shows the demographic analysis of the overall group of patients divided into those that received mitral valve replacement and those that received mitral valve repair. The mean age was 62, with 26% of patients >70 years old; 26% of the entire group was in functional class IV. Table

0735-1097/90/$3.50

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Table 1. Demographics of 127 Patients

	Total	Valve Repair	Valve Replacement
No.	127	75	52
Sex (M/F)	67/60	40/35	23/29
Age (range, mean)	19 to 86 (62)	17 to 81 (61)	19 to 86 (62)
Age >70 yr (no.)	33 (26%)	22 (29%)	11 (21%)
Mean functional class	3.2	3.1	3.3
Functional class IV (no.)	32 (26%)	14 (19%)	18 (35%)*

*p = 0.05.

2 divides the patient groups into etiologic classes; the most common lesion was myxomatous degeneration, the least common endocarditis. All patients had fixed severe mitral regurgitation; no patient was included who had transient, ischemic regurgitation. Table 3 summarizes the pertinent hemodynamic and angiographic data. Concomitant coronary artery disease was found in 39 patients (31%) with 33 having multiple vessel disease.

Of the entire group, 75 patients (60%) underwent mitral valve repair and 52 (40%) underwent mitral valve replacement with the St. Jude valve (n = 32) or Hancock porcine valve (n = 20). In the group undergoing mitral valve repair there was a slightly higher incidence of patients >70 years old and a higher incidence of patients undergoing concomitant coronary bypass (27 of 75 [36%] versus 12 of 52 [23%]); in the replacement group there was a higher incidence of functional class IV patients.

Operative techniques. These have been reported previously (4–10). For valve replacement the posterior papillary muscle and chordae tendineae at least are preserved. The presence of moderate to severe calcification resulted in valve replacement and valve repair was carried out for every etiology of mitral valve disease including myxomatous degeneration, rheumatic, ischemic and endocarditis as long as adequate noncalcified leaflet tissue, especially of the anterior leaflet, was available. Techniques for valve repair were leaflet resection, chordoplasty, chordal transfer and ring annuloplasty (5–10). Residual regurgitation was evaluated before release of the aortic clamp by volume infusion into the left ventricle and after cardiopulmonary bypass surgery from direct left atrial pressure tracings and transesophageal Doppler echocardiography. Myocardial preservation was carried out with hyperkalemic (30 MEQ/L) cold crystalloid cardioplegia with local *endocardial* and *epicardial* hypothermia, as well as moderate systemic hypothermia. No iced slush was used.

Table 2. Etiology of Mitral Regurgitation in 127 Patients

	Total Group	Valve Repair	Valve Replacement
Myxomatous	80	52	28
Rheumatic	21	6	15
Ischemic	16	14	2
Endocarditis	10	3	7
Total	127	75	52

Patients with coronary disease underwent complete revascularization; the distal anastomosis was performed first and most grafts were gently flushed with 30 ml of cardioplegia after each anastomosis was completed. A pulmonary artery catheter was placed for the measurement of pulmonary pressure and cardiac output postoperatively, as well as a direct left atrial pressure line to evaluate pulmonary and systemic vascular resistance. Postoperative care included balancing of right-sided and left-sided filling pressures, aggressive volume replacement and diuresis and early endotracheal tube extubation.

Results

Operative mortality (Table 4). In the series of 127 patients there was a total of three hospital deaths (operative mortality of 2.3%). No patient failed to be discontinued from cardiopulmonary bypass. All three deaths occurred after mitral valve replacement and there were no deaths in the 75 patients undergoing mitral valve repair. Of the 39 patients that underwent coronary bypass grafting there was one operative death (2.5%) compared with two deaths (2.3%) for those who did not have concomitant coronary bypass surgery. The operative mortality for those with mitral valve repair plus coronary artery bypass was zero.

The operative mortality was due to low cardiac output syndrome in one 80 year old patient an acute myocardial infarction in one 55 year old patient and complications of chronic renal failure in a 38 year old patient on long-term dialysis. There was no correlation of hospital mortality with age; of the 33 patients >70 years old, there was one death (3%) compared with two (2%) in the 94 patients <70 years old.

Postoperative complications (Table 5). These occurred in five patients in the repair group and three in the valve replacement group. In the repair group, two patients had residual mitral regurgitation necessitating reoperation within 2 months, two had mediastinal bleeding requiring reoperation and one had a transient stroke. In the replacement group two patients had additional surgery for mediastinal bleeding and one developed endocarditis but was treated successfully with antibiotics alone. Intensive care unit days, as well as the length of stay, were generally shorter in the mitral valve repair group.

Discussion

The safety of surgery for mitral regurgitation with or without coronary disease has improved markedly in the

Table 3. Catheterization and Coronary Angiography in 127 Patients

	Total (n = 127)	Valve Repair (n = 75)	Valve Replacement (n = 52)
Coronary artery disease	39 (31%)	27 (36%)	12 (23%)
1 VD	6	3	3
2 VD	15	8	7
3 VD	18	16	2
Total	39	27	12
Mean pulm. art. press. (n = 106) (range, mean)	11 to 55 (31)	11 to 54 (30)	16 to 55 (33) p = NS
LV EF (n = 99) (range, mean)	20 to 90 (58)	20 to 87 (60)	28 to 90 (57) p = NS

LV EF = left ventricular ejection fraction; pulm. art. press. = pulmonary artery pressure; VD = vessel disease.

modern era of cardiac surgery. Certainly, a decade ago the combined operation of mitral valve replacement, especially with coronary bypass surgery, was a higher risk procedure in many institutions including our own (1–3). There has been a gradual realization that great importance must be given to the preservation of as much as possible of the mitral anulus and chordal structures with particular attention to myocardial protection including the installation of cardioplegia through coronary bypass grafts after completion of anastomoses. This concept of anulus-chordal continuity was first suggested by Lillehei et al. (11) but never substantiated because of the laboratory and clinical evaluation techniques available in the 60's and 70's (12,13). It was only after the development of two-dimensional echocardiography and experimental crystal ultrasonography that clinical and laboratory confirmation of this important concept was established (14–17).

Table 4. Operative Mortality in 127 Patients

	Total	Valve Repair	Valve Replacement
Deaths (no.)	3/127 (2.5%)	0/75	3/52
With CABG	1/39 (2.5%)	0/27	1/12
Without CABG	2/88 (2.3%)	0/48	2/40
Age (yr)			
≥70	1/33 (3%)	0/22	1/11
<70	2/94 (2.1%)	0/53	2/41
Mortality	Preoperative LV EF		
1 Low output	0.40		
1 Acute MI	0.48		
1 Chronic renal failure	0.60		

CABG = concomitant coronary artery bypass graft; LV EF = left ventricular ejection fraction.

Valve repair versus valve replacement. Though the numbers are relatively small and the clinical profile of our two surgical groups is not perfectly matched, the valve repair group did better in terms of operative mortality than the patients undergoing valve replacement even with the preservation of at least some part of the chordal attachment, a concept we have reported before (5). In our patients undergoing valve repair for mitral regurgitation, there was a higher incidence of patients >70 years old and those with coronary disease compared with those who underwent valve replacement. The replacement group, however, had a higher number of patients in functional class IV. Both groups had similar hemodynamic function including pulmonary artery pressure and left ventricular ejection fraction. In our opinion, this is related to the total preservation of the intact valve chordal papillary muscle-annular continuity that appears to be increasingly important in the preservation of left ventricular function after mitral valve surgery. Our total series of primary mitral valve repair for mitral regurgitation now includes approximately 200 patients operated on since 1984, with an overall mortality rate of <3%; in the present series of 75 patients there were no deaths.

For many years it was thought that patients with mitral regurgitation did poorly postoperatively because of the acute increase in systolic wall stress prompted by the abrupt closure of the "pop-off" valve to the left atrium, first suggested by Kirklin (18). Although an increase in left

Table 5. Postoperative Complications

	Valve Repair	Valve Replacement
Residual MR	2	0
Postop. bleeding	2	2
TIA	1	0
Endocarditis	0	1

MR = mitral regurgitation; Postop. = postoperative; TIA = transient ischemic attack.

ventricular wall stress does occur to a certain extent, the main reason patients failed after mitral regurgitation surgery in that era was loss of papillary muscle-annular continuity and the natural ovoid shape of the left ventricle. With the increasing number of mitral valve repairs for regurgitation or mitral valve replacement, preservation of annular-chordal structures and left ventricular shape and thus left ventricular dynamics allows for optimal configuration and function postoperatively. This alleviates the chronic low cardiac output commonly seen after mitral valve surgery in previous years when the chordal attachments, as well as the tips of the papillary muscles, were often removed in the course of a Starr-Edwards valve replacement (18) or other mechanical valve substitutes.

The etiologic diagnoses in the patients operated on in this series are fairly representative of most American centers. There is a large percentage of myxomatous degenerated valves, fewer valves due to rheumatic or ischemic heart disease. In most instances repair has been possible in valve endocarditis, a most desirable scenario obviating prosthetic material in a septic field.

Surgical implications. Although the decision for the timing of mitral valve surgery is still a very complex one based on a combination of clinical, hemodynamic and noninvasive data in an individual patient, it is clear that, if surgery is recommended, the risk of surgery, whether it be valve replacement or repair with or without coronary disease, has considerably decreased in the past years. Furthermore, intermediate term data in this country suggest that survival improves after competent mitral valve repair or when papillary muscle-anulus structures are preserved after replacement, presumably due to a much lower incidence of chronic low cardiac output (3,19–21) and attendant sequelae such as thromboembolism (5,10). With a decreasing risk of surgery, one might give consideration to referral of patients earlier in their course before onset of chronic atrial fibrillation known to be a poor prognostic sign (22,23) before severe pathologic changes occur in the valve requiring replacement or before development of chronic myopathic changes from chronic ventricular volume overload leading to a shortened life span regardless of the competency of the valve (24). Further early and late follow-up of larger numbers of patients will, of course, be important to solidify these conclusions, as will the long-term results of valve repair and replacement for mitral regurgitation.

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