Mitral Valve Reconstruction in Elderly, Ischemic Patients*

Steven F. Bolling, MD, FCCP; G. Michael Deeb, MD; and David S. Bach, MD

The role of mitral valve reconstruction is controversial in elderly patients with concurrent ischemic heart disease owing to technical difficulty, prolonged operative times, high mortality, and possible residual mitral regurgitation. However, mitral reconstruction could be most beneficial in this age group due to preservation of left ventricular function, avoidance of anticoagulation, or repeat operation for bioprosthetic degeneration. We studied the outcome of mitral valve reconstruction in 100 consecutive elderly ischemic patients 65 years or older (mean=73 years; range, 65 to 86 years) operated on between October 1990 and May 1995. Preoperatively all patients were New York Heart Association (NYHA) class III or IV with an ejection fraction of 32±2%. All patients underwent primary coronary bypass grafting (2.7±0.2 grafts) and had a flexible mitral annuloplasty ring inserted. Additionally, 54 patients required further complex mitral repairs. All patients had 4+ mitral regurgitation by transesophageal echocardiography prior to operation. After mitral reconstruction, no patient had more than 1+ regurgitation, while most had none and no systolic anterior leaflet motion was noted. There were 4 early (30 day) deaths (4%) and 6 late deaths (6%) at a mean follow-up of 25 months. Patient morbidity has included episodes of mild congestive heart failure (nine), transient ischemic attack (one), endocarditis (one), and respiratory failure (five). There have been one early and two late reoperations for mitral valve replacement. All remaining patients are in NYHA class I or II. While longer-term follow-up is mandatory, coronary bypass grafting and mitral valve reconstruction in the elderly can be accomplished with acceptable surgical mortality and morbidity, yielding reliable improvement in symptoms and quality of life.

(CHEST 1996; 109:35-40)

Key words: elderly; ischemia; mitral repair

Mitral regurgitation is a significant complication of ischemic heart disease and may be due to existing degenerative disease or annular-ventricular dilation, ischemic papillary muscle dysfunction, or altered ventricular geometry from prior infarction. Mitral regurgitation leads to volume overload of the ischemic ventricle, progressive annular dilation, worsened mitral regurgitation, and congestive heart failure. Severe mitral regurgitation as a complication of ischemic disease predicts a poor survival, particularly in the elderly. While surgical correction of mitral valve regurgitation at the time of revascularization could be most beneficial, replacement mitral valve surgery has a prohibitive operative mortality in these ill patients and is associated with a postoperative loss of left ventricular (LV) systolic function due to interruption of annular-chordal-papillary muscle continuity. This added loss of ventricular function, poorly afforded in ischemic elderly patients with regurgitation and prior functional compromise, may be responsible for their poor late survival. Treatment options for this age group are problematic as valve replacement distorts ventricular-annular geometry and function, mechanical mitral valve replacement puts elderly patients at risk from anticoagulant complications, bioprosthetic mitral valve replacement may increasingly subject these patients to future redo valve operations in an age group that continues to survive longer, and revascularization alone often results in continued mitral regurgitation, heart failure, and a poor outcome. However, reconstruction of the mitral valve with preservation of annular-chordal-papillary muscle continuity results in maintenance of LV systolic function, lower LV volumes and end-systolic wall stress, and has a lower mortality than mitral valve replacement.

Coronary revascularization and correction of mitral regurgitation preserving mitral annulus-chordal integrity by mitral reconstruction could yield acceptable operative and postoperative mortality. This present study was designed to assess the feasibility of mitral valve reconstruction for ischemic mitral regurgitation in the elderly and monitor the early outcome in terms of survival, impact on heart failure, and ventricular function.

MATERIALS AND METHODS

Between October 1990 and May 1995, 100 consecutive, pro-
spective nonrandomized patients at the University of Michigan Medical Center, 65 years of age and older, with coronary artery disease resulting in significant ischemia and severe mitral regurgitation were studied. All patients had angina or equivalence on functional testing and New York Heart Association (NYHA) class III or IV congestive heart failure despite receiving maximized medical regimens. No patients were excluded from this study on the basis of preoperative status, function, age, or any other criteria.

Echocardiographic Imaging

All preoperative and postoperative echocardiographic imaging was performed using standard transesophageal windows with commercially available equipment. Intraoperative echocardiography was performed using echocardiography (Hewlett-Packard Sonos 1500; Hewlett-Packard Co; Waltham, Mass) and biplane transesophageal probe. Analysis was performed by blinded experienced echocardiographers. Mitral regurgitation was assessed using color-flow Doppler; severity was graded as none, trivial, mild, moderate, or severe. Measurements of LV, mitral annular, and outflow tract diameters were made from parasternal long-axis views. LV volumes, stroke volumes, and ejection fraction were calculated using Simpson's method with two apical views. Regurgitant volume was calculated as the difference between mitral inflow and forward cardiac output, and the regurgitant fraction as the ratio of the regurgitant volume to mitral inflow volume. Mitral gradients were calculated using continuous-wave Doppler.

Operation

Coronary artery revascularization and mitral valve reconstruction were performed via median sternotomy with standard hypothermic cardiopulmonary bypass, utilizing hypothermic, combined antegrade, and retrograde blood cardioplegic arrest in all patients. All operations were primary operations (no redo operations) and were performed with institutional approval and informed consent.

Follow-up

Patient follow-up was from a combination of computer database analysis, chart review, and patients contacted by telephone and personal office visit. The patients' personal physicians were also contacted in follow-up. All data are presented as mean ± SD. Comparison between echocardiographic and angiographic ejection fraction was made using a Pearson correlation. Comparisons between preoperative and postoperative NYHA class, LV size, volume, ejection fraction, regurgitant fraction, and forward cardiac output were made using paired Student's t tests. Actuarial survival was performed by the Kaplan-Meier method. Differences were considered significant at a 95% confidence limit (p<0.05; two-tailed).

RESULTS

One hundred patients (58 men and 42 women) were included in the study. Patient ages ranged from 65 to 86 years (mean, 73±2 years; 39 patients were less than 70 years; 7 patients were more than 80 years). Preoperatively, all patients had NYHA class III or IV congestive heart failure, with a mean failure class of 3.6. All patients had at least one hospital admission for congestive heart failure. All patients were receiving maximized medical therapy for congestive heart failure, including combinations of digoxin, diuretics, and afterload reducers. Preoperative ejection fraction from left ventriculography or radionuclide angiography ranged from 15 to 55% (mean, 32±2%). Preoperative coronary angiography and echocardiography were performed on all patients to confirm coronary anatomy, ventricular function, and assess mitral regurgitation. All patients had 3 to 4+ mitral regurgitation, with most having severe regurgitation. On coronary angiography, all patients had significant (>70% lesions) anatomic coronary artery disease. All patients had significant angina or angina equivalent with positive functional testing. These elderly patients had a mean Canadian Cardiovascular Society anginal class of 2.8.

At surgery, all patients underwent mitral reconstruction via a remodeling ring annuloplasty, with implantation of a flexible or semiflexible remodeling ring (Duran Ring; Medtronic Inc; Minneapolis; Physio Ring or Cosgrove Ring; Baxter Inc; Irvine, Calif) as all patients were believed to have annular dilation. At the time of sizing of the mitral remodeling ring, these ischemic patients were slightly undersized in their trigone to trigone diameters if papillary muscle dysfunction was identified on echo. Additionally, 54 patients required a further additional complex repair (leaflet resection, leaflet/chordal turnover, polytetrafluoroethylene chordal replacement or supplementation, etc) at the time of surgery, as these patients were believed to have an element of underlying organic/degenerative mitral disease as well as ischemic annular dilation and/or papillary muscle dysfunction. The average time on cardiopulmonary bypass was 206±8 min, with a mean aortic cross-clamp time of 139±6 min. Coronary artery bypass grafts (CABGs) were placed in all patients with the aim of total revascularization of their coronary artery disease. The average number of CABGs performed was 2.7±0.2 (range, 1 to 6). Eighty-eight patients received left internal mammary artery bypass grafts (88%) and saphenous vein grafts. Additionally, six patients underwent DeVega tricuspid annuloplasty reconstruction, five patients underwent aortic valve replacement, three patients had closure of an atrial septal defect, one patient had placement of epicardial internal cardioverter defibrillator (ICD) patches, and repair of a small LV aneurysm was performed in one patient. Three of the present cases were emergency transfers from the cardiac catheterization laboratory for acute ischemic exacerbation and severe mitral regurgitation.

All patients survived the operative procedure. All patients were weaned from cardiopulmonary bypass using a phosphodiesterase inhibitor, amrinone (Sanofi-Winthrop; Newark, NJ); and other pressors, as needed. Three patients required new placement of an intra-aortic balloon pump (IABP), besides those emergency cases that had come to the operating room with the intra-aortic balloon pump in place. No patient required mechanical support. Intraoperative transesophageal echocardiography (TEE) following revascularization and mitral reconstruction and the discontinuation of
cardiopulmonary bypass with adequate loading revealed no mitral regurgitation in 84 patients, trivial to mild regurgitation in 14 patients, and 2+ regurgitation in 2 patients. At the time of revascularization and mitral reconstruction, one patient required valve repair takedown with porcine valve replacement, due to residual 2 to 3+ mitral regurgitation. The mean mitral regurgitant grade identified by intraoperative TEE was 0.3±0.1. The mean transmural gradient identified by intraoperative TEE was 3±1 mm Hg (range, 2 to 6). No abnormal systolic anterior motion of the anterior leaflet of the mitral valve was noted and no outflow tract gradients were seen. Postoperatively, nine patients required longer than 24 h of mechanical ventilator support following revascularization and mitral reconstruction. There was one late superficial wound infection and no mediastinitis. There were 4 in-hospital (30 day) deaths, 3 patients died from multiorgan failure, and 1 patient died following emergency operation from low cardiac output and a presumed severe brain injury. Three patients were returned to the operating room for postoperative bleeding. The mean duration of hospitalization following surgery was 10±4 days (range, 4 to 54 days).

The duration of follow-up for these patients has been 1 to 54 months, with a 1-year actuarial survival of 90%. At a mean follow-up of 25 months, remaining patients are in NYHA class I or II. Postoperatively, there have been nine patients with hospitalizations for exacerbations of mild congestive heart failure. All patients have continued to receive medical therapy, consisting of combinations of calcium channel blockers, digoxin, diuretics, and afterload reducing agents. However, medical regimens are lower postoperatively, with no patient receiving a higher dose of any medication in the postoperative period than in his or her preoperative regimen. There has been a significant reduction in diuretic dose required. The vast majority of patients have been angina free. All patients have remained in their preoperative rhythm.

All surviving patients reported improvement in their functional status following surgery, citing activities performed postoperatively for which exertional dyspnea was previously prohibitive. The NYHA failure class significantly fell from a mean of 3.6±0.2 to 1.7±0.4 for the group. The angina class significantly fell for every patient individually and from a mean of 2.8±0.4 to 0.4±0.3 for the group. Follow-up echocardiography at 20 to 24 months was available for 34 patients. The mean transmural gradient on follow-up was 3±1 mm Hg (range, 1 to 5 mm Hg). Measurements of LV size, Doppler-derived flows, and volumes denoted that patients had a marked reduction in end-systolic and end-diastolic diameters, regurgitant volume, and regurgitant fraction, which fell from 64 to 15%.

There have been six late deaths in this elderly group following revascularization and mitral valve reconstruction. One late death occurred related to complications following a motor vehicle accident. Two late deaths were presumed cardiac ischemic deaths, and three other late deaths occurred from continued congestive heart/pulmonary failure.

**Discussion**

Mitral regurgitation is well known to be a poor prognostic sign for patients with coronary artery disease and may affect up to 15% of patients undergoing coronary bypass surgery. If mitral regurgitation is not corrected, it profoundly worsens hospital mortality and late survival, even with good myocardial revascularization. In a 10-year study of 601 nonsurgical patients, Proudfoot et al reported survival rates of only 20% in patients having diffuse coronary artery disease and LV impairment, with the most important detriment to survival being mitral regurgitation. Presently, the mechanism of ischemic-related mitral regurgitation remains unclear. Some investigators classify it into regurgitation derived from (1) myxomatous degeneration with unrelated coincidental coronary artery disease, or from (2) reversible ischemic disease and papillary muscle dysfunction, or from (3) irreversible ischemic disease and altered geometry due to prior infarction. However, while ischemic mitral regurgitation is closely associated with right coronary and/or circumflex disease, resulting in restricted leaflet motion and papillary muscle dysfunction/coordination, many authors have also noted a high incidence of coexisting degenerative mitral disease with coronary artery disease in this age group. As in the present series, mitral annulus dilatation is believed to be present in all cases and may be the only mechanism of regurgitation in up to 50% of patients. Interestingly, Fishbein has shown that the ischemic mitral valve demonstrates histologic changes that are indistinguishable from primary myxomatous degeneration, both in the chordae and valve leaflet. Therefore, exclusion or inclusion of patients into those having myxomatous degeneration with unrelated coronary artery disease vs “pure” ischemic regurgitation may be somewhat arbitrary. However, in this present series, 46 patients were believed to have ischemic mitral regurgitation (class I, Carpentier), with no underlying organic mitral valvular disease. These patients appear to have chordae that are functionally “too short,” as papillary muscle dysfunction and/or altered ventricular geometry does not allow for adequate leaflet coaptation and patients were treated with “over-correction” of their annular dilation, by undersizing the annuloplasty ring. No instances of systolic anterior motion or LV outflow tract obstruction were noted.

Whatever the underlying mechanism of ischemic mitral regurgitation, the published mortality for surgi-
cal intervention in this subgroup of elderly, ischemic patients remains high, varying from 10 to 48%. In a recent report,22 the most profound effect on survival after mitral replacement was in patients older than 60 years whose survival was decreased by 50% if they required CABG at the time of mitral valve replacement. Furthermore, those patients older than 70 years had another 50% reduction in their median survival following CABG and mitral replacement. This finding agrees with two other studies24,25 in which the operative mortality rate for patients 70 years and older undergoing CABG and mitral replacement was more than 20% and predictive factors for poor outcome included age and worse ischemia. A further study26 examined late results of combined mitral replacement and CABG in 28 patients who had ischemic mitral regurgitation believed to be unsuitable for valve reconstruction and therefore underwent mitral replacement. There were six intraoperative deaths (21%) and the 5-year survival was only 43%. These data are confirmed by other studies27,28 in which 63 and 50 patients underwent CABG combined with mitral valve surgery. Hospital mortality was 9.5% and 8% and the 5-year survival rate for both series was only 40%. Interestingly, however, survivors improved by at least one functional NYHA class. Many other surgical studies have denoted that greater functional and hemodynamic impairment and reduced ejection fraction such as seen in the elderly ischemic patients equates with poor operative and inhospital survival in patients operated on for mitral regurgitation by mitral valve replacement.8,13-15,24 This is in accordance with many experimental studies demonstrating that chordal transection results in significant reductions in rest and exercise ejection fraction, caused by a significant increase in end-systolic circumferential wall stress and geometric distortion.9,12 One study notes that loss of annular-chordal-papillary muscle continuity immediately decreases LV systolic function by 30%, alters the twist mechanics of myocardial contraction, and results in a globular, inefficient geometry of the heart.9,12 all of which can be ill afforded in this elderly, ischemic group.

Despite this, surgical correction of mitral regurgitation and accompanying volume overload has been shown to be warranted for patients with mitral regurgitation and could be most beneficial in elderly patients suffering from ischemic disease with secondary mitral regurgitation. In a study of 249 patients with mitral regurgitation, patients with surgical correction had significantly improved survival as compared with medically treated patients. Those patients with moderate or greater impairment of ejection fraction accounted for the majority of improved survival in patients having surgery, despite greater preoperative functional and hemodynamic impairment.14 Unfortunately, surgical options for correction of mitral regurgitation in this patient group are problematic, as even when the subvalvular apparatus is left intact, mitral valve replacement distorts ventricular-annular geometry and function. Valve replacement in situ does not allow for the normal three-dimensional flexion of the mitral annulus during systole and diastole or for the normal change in annular size during the cardiac cycle. Additionally, valve replacement in situ is often performed using a large prosthesis, which unfortunately “stents” the ventricular-annular apparatus in an open or enlarged configuration and does not allow for ventricular remodeling or healing. Furthermore, mitral valve replacement using a mechanical prosthesis puts elderly patients at indefinite risk from anticoagulant complications. Conversely, when using a bioprosthesis, patients increasingly face redo valve operations from valve degeneration, in this age group that continues to survive for longer time periods and may “outlive” their bioprostheses. Finally, revascularization alone, in elderly patients, has been almost universally noted to result in continued mitral regurgitation, heart failure, and a poor outcome. However, reconstruction of the mitral valve with the aim of preservation of annular-chordal-papillary muscle continuity could subject the patient to a prolonged operation with significant technical difficulty and possible residual mitral regurgitation. Despite this, many studies have shown that mitral reconstruction, as opposed to replacement, results in improved rest and exercise ejection indexes, primarily due to a marked reduction in end-systolic stress and maintenance of a more ellipsoidal chamber geometry.28,29 Previous clinical studies have compared the results of mitral valve reconstruction against those following mitral valve replacement and have concluded that the preservation of the annular-chordal-papillary muscle continuity results in maintenance of LV function and geometry,30 leading to better patient outcome.17-20,31

Technically, in this study, mitral reconstruction effectively corrected mitral regurgitation, confirmed both on intraoperative TEE and on follow-up transthoracic studies. Furthermore, despite high operative risks and longer operative times, there were no operative deaths and an actuarial survival of 90% at 1 year. Not only was survival improved, but functional status was better in surviving patients. Additionally, echocardiographic analyses in those patients available at longer follow-up revealed a trend toward decreased LV volumes postoperatively. While loss of low impedance left atrial “pop-off” might be expected to lead to further impairment, LV function improved in many patients postoperatively, implying that some of the preoperative ventricular decompensation was due to volume overload associated with severe mitral regurgitation superimposed on ischemic dysfunction. Postulated
mechanisms for the improvement in ventricular function include stabilization of the mitral annulus and LV unloading which induced a more favorable ventricular geometry. A recent study denotes that remodeling of LV geometry may be rapid in patients with resultant regurgitant fractions of less than 30% after correction of mitral regurgitation. A second mechanism may involve decreasing the volume overload of the LV and establishing a better position on the Frank-Starling curve.

In this study, while there are not large enough groups to statistically analyze subpopulations by 65-, 70-, 75-, and 80-year-old age groups, mortality and morbidity did increase with age and higher preoperative risk factors. Although it was believed that no patient had incomplete revascularization and all patients had good initial resolution of mitral regurgitation, most patients who died of progressive cardiopulmonary failure got significantly better, then within a relatively short time, their conditions deteriorated. These patients appeared to have ventricular function that was “too far gone” for them to be good operative candidates. This is perhaps reflective of the previous prevailing attitude associated with mitral valve replacement, that these elderly, ischemic patients do not do well at surgery and are therefore only referred as a last resort. However, even in the elderly, this becomes a self-fulfilling prophecy, as had these patients been operated on somewhat earlier with mitral repair, at a time when the ventricle could still sustain recovery, they may have done better, reflected by the excellent outcome of the remaining patients. While longer-term follow-up with greater number of patients and perhaps case-matched control subjects is necessary, this study demonstrates the feasibility of revascularization and operative repair of mitral regurgitation, with an excellent technical outcome and a low operative mortality. Other aspects of follow-up, including assessment of exercise oxygen consumption and LV remodeling, are being investigated. Finally, we are encouraged by these early results and believe that mitral reconstruction offers a new strategy for elderly, ischemic patients.

REFERENCES
6 Lee SJ, Bay KS. Mortality risk factors associated with mitral valve replacement: a survival analysis of 10 year follow-up data. Can J Cardiol 1991; 7:11-8
26 Ashraf SS, Shankat N, Odom N, et al. Early and late results following combined coronary bypass surgery and mitral valve