Left ventricular remodeling early after correction of mitral regurgitation: Maintenance of stroke volume with decreased systolic indexes

Elena A. Ashikhmina, MD,[a] Hartzell V. Schaff, MD,[a] Rakesh M. Suri, MD, DPhil,[a] Maurice Enriquez-Sarano, MD,[b] and Martin D. Abel, MD[c]

Objective: Mitral valve repair for mitral regurgitation is followed by left ventricle adjustment to new preload and afterload. We evaluated left ventricular geometry and function immediately after mitral valve repair for degenerative prolapse.

Methods: We prospectively studied 25 patients undergoing mitral valve repair; 15 patients undergoing a coronary artery bypass graft served as controls to determine the impact of cardiopulmonary bypass and cardioplegic arrest on left ventricular function. Intraoperative transesophageal echocardiography was conducted after sternotomy before initiation of cardiopulmonary bypass and after termination of cardiopulmonary bypass and protamine infusion. Simultaneous pulmonary catheter data ensured that the images were obtained under similar hemodynamic conditions.

Results: Immediately after mitral valve repair, left ventricular fractional area change decreased significantly from 65% ± 7% to 52% ± 8% ($P < .001$). Left ventricular end-diastolic area decreased minimally (21.3 ± 5.3 cm^2 vs 19.4 ± 4.5 cm^2; $P = .005$), whereas left ventricular end-systolic area increased significantly (7.5 ± 2.3 cm^2 vs 9.3 ± 2.5 cm^2; $P < .001$). Notably, forward stroke volume (thermodilution) remained similar (63 ± 24 mL vs 66 ± 19 mL; $P = .5$). No significant difference was found in controls between pre- cardiopulmonary bypass and post-cardiopulmonary bypass fractional area change (54% ± 12% vs 57% ± 10%; $P = .19$), left ventricular end-diastolic area (16.6 ± 6.2 cm^2 vs 15.7 ± 5.0 cm^2; $P = .32$), and stroke volume (72 ± 29 mL vs 65 ± 19 mL; $P = .15$); they had a slight decrease in left ventricular end-systolic area (7.9 ± 4.4 cm^2 vs 6.9 ± 3.2 cm^2; $P = .03$).

Conclusions: Early after correction of mitral regurgitation, left ventricular fractional area change decreases significantly, primarily as the result of a larger end-systolic dimension. This may be a compensatory mechanism to prevent augmentation of forward stroke volume after mitral valve repair. (J Thorac Cardiovasc Surg 2010;140:1300-5)

Early after correction of mitral valve (MV) regurgitation, systolic function of the left ventricle (LV) declines, but the underlying mechanisms are unknown. Most studies have analyzed LV function at 1 week postoperatively[1-4] or later.[5-12] Although the advantages of MV repair versus replacement have been demonstrated in long-term follow-up,[13,14] the immediate postoperative changes in LV function after MV repair have not been defined. We examined postoperative changes of LV geometry and assessed LV function immediately after correction of mitral regurgitation (MR) caused by degenerative valve disease (leaflet prolapse).

MATERIALS AND METHODS

After institutional review board approval, we prospectively studied 25 patients undergoing MV repair for degenerative leaflet prolapse and 15 patients undergoing coronary artery bypass grafting (CABG). The 15 patients who underwent CABG served as controls for estimation of the possible impact of cardiopulmonary bypass (CPB) and cardioplegic arrest on LV function. Operations were performed with normothermic CPB and hypothermic antegrade blood cardioplegia for myocardial protection.

Transesophageal echocardiography (TEE) with simultaneous Swan-Ganz catheter hemodynamic measurements was performed after sternotomy before initiation of CPB (pre-CPB) and after termination of CPB and 10 to 15 minutes after protamine infusion (post-CPB).

LV size and function were measured off-line. Technical difficulties in obtaining a nonforeshortened LV cavity from the esophageal approach for quantification of LV volumes by TEE sometimes produced inconsistent results similar to those described previously.[15] Schmidlin and colleagues[16] demonstrated good correlation between intraoperative LV area measurements with TEE and conductance catheter LV volume measurements. Thus, we chose to evaluate more reproducible LV linear dimensions and plane areas from the transgastric mid-papillary view that correlate well with LV volumes. The largest (end-diastole) and smallest (end-systole) frames were identified by subjective assessment of cavity size and confirmed with electrocardiographic systole and diastole. The greatest distances between 2 endocardial borders of the anterior and inferior LV walls were measured (LV end-diastolic dimension [LVEDD] and end-systolic dimension [LVESD]), and LV areas were traced (LV end-diastolic area [LVEDA]

From the Divisions of Cardiovascular Surgery,[a] Cardiovascular Diseases,[b] and Cardiovascular and Thoracic Anesthesia,[c] Mayo Clinic, Rochester, Minn.

Disclosures: None.

Received for publication July 20, 2009; revisions received Oct 16, 2009; accepted for publication Dec 14, 2009; available ahead of print March 12, 2010.

Address for reprints: Hartzell V. Schaff, MD, Division of Cardiovascular Surgery, Mayo Clinic, 200 First Street SW, Rochester, MN, 55905 (E-mail: schaff@mayo.edu).

0022-5223/$36.00

doi:10.1016/j.jtcvs.2009.12.031

Abbreviations and Acronyms

CABG = coronary artery bypass grafting
CPB = cardiopulmonary bypass
EF = ejection fraction
FAC = fractional area change
FS = fractional shortening
LV = left ventricle, left ventricular
LVEDA = left ventricular end-diastolic area
LVEDD = left ventricular end-diastolic dimensions
LVEF = left ventricular ejection fraction
LVESA = left ventricular end-systolic area
LVESD = left ventricular end-systolic dimensions
MR = mitral regurgitation
MV = mitral valve
TEE = transesophageal echocardiography
TTE = transthoracic echocardiography

and end-systolic area [LVESA]); the average values of 3 representative cardiac cycles were then recorded. Fractional area change (FAC), a conventional systolic phase index of LV function,[17] correlates well with LV ejection fraction (EF).[18] It was calculated as FAC = [(LVEDA–LVESA)/LVEDA] × 100%, cm^2.[15]

While echocardiographic images were being recorded, we performed hemodynamic measurements with a pulmonary catheter to ensure that they were obtained under similar preoperative and postoperative ventricular loading conditions (pulmonary capillary wedge pressure, 13.9 ± 4.9 mm Hg vs 13.0 ± 2.3 mm Hg [P =.45], and right atrial pressure, 6.9 ± 3.3 mm Hg vs 7.3 ± 3.0 mm Hg [P =.2], in MV repair group; pulmonary capillary wedge pressure, 14.6 ± 5.2 mm Hg vs 14.3 ± 5.1 mm Hg [P =.88], and right atrial pressure, 9.2 ± 4.5 mm Hg vs 8.7 ± 3.8 mm Hg [P =.7], in controls). Tricuspid regurgitation assessed semiquantitatively was absent or trivial in all 40 patients; thus, hemodynamic measurements were affected minimally. No patient received inotropic medication before or after CPB.

MR volume was estimated by calculating the proximal isovelocity surface area of the regurgitant jet.

Descriptive statistics for categoric variables are reported as frequency and percentage; continuous variables are reported as mean ± standard deviation. Categoric variables were compared between cases and controls using the chi-square test. When appropriate, continuous variables were compared using the 2-sample t test or the Wilcoxon rank-sum test. Changes in categoric variables over time were compared using the McNemar test, and changes in continuous variables were compared using the paired t test or rank-sum test. All statistical tests were 2-sided.

RESULTS

Both groups contained more men than women (Table 1). Patients undergoing MV repair were younger than controls. Preoperatively, most patients undergoing MV repair (84%) had mild symptoms (New York Heart Association class I or II), whereas patients from the CABG group had more severe functional limitations (73% New York Heart Association class III). Durations of CPB and aortic crossclamping were significantly shorter for MV repair than for CABG (P < .001).

The most common pathologic finding in the 25 patients who underwent MV repair was posterior leaflet prolapse (92%), typically of the middle scallop, caused by elongation or rupture of chordae tendineae; 11 (44%) of 25 patients had associated anterior leaflet prolapse. Sixteen patients (64%) underwent partial triangular resection and suture repair of the posterior leaflet with insertion of a standard-length (63-mm) flexible posterior annuloplasty band. Two patients (8%) had suture closure of the cleft between scallops of the anterior leaflet with insertion of an annuloplasty band. Seven patients (28%) had MV repair with a posterior annuloplasty band without leaflet repair. The average MR volume was 73 ± 25 mL. Patients undergoing CABG received an average of 3 bypass grafts; none had MR.

Immediate Changes of Left Ventricular Geometry After Mitral Valve Repair

After MV repair, considerable changes in LV size and function were observed with intraoperative TEE shortly after reconstruction (Table 2). LV chamber size decreased slightly at end diastole and increased prominently at end systole (Figure 1). As a result, postoperative versus preoperative fractional shortening (FS) and FAC declined significantly (P < .001) (Figure 2).

Patients undergoing CABG had more heterogeneous echocardiographic data than did patients with MV repair (Figures 1 and 2). In the CABG group (N = 15), FAC increased after CPB in 4 patients (27%) and decreased in 2 patients (13%). Overall, the average FAC in the CABG group did not change (Table 2). LVESD and LVESA decreased slightly.

LV forward stroke volume in patients with MV repair remained normal and was unchanged compared with pre-CPB values. Both groups had an increased heart rate early after CPB (Table 2). Thus, cardiac output and index were slightly increased after MV repair (Table 2). Among patients undergoing CABG, postbypass stroke volumes were also similar to prebypass values; cardiac output and index were slightly higher but not statistically significant compared with prebypass measurements (Table 2).

Early Changes of Left Ventricular Geometry After Mitral Valve Repair

To further evaluate early postoperative changes in LV size and function after MV repair, we analyzed preoperative (admission) and postoperative (predismissal) transthoracic echocardiograms (TTEs) of the 25 patients with MV repair. TTE was performed an average of 4 ± 1 days after surgery. LVEDD decreased from 5.7 ± 0.6 cm preoperatively to 5.2 ± 0.4 cm postoperatively (P < .001), but LVESD was basically unchanged (3.7 ± 0.7 cm before vs 3.6 ± 0.5 cm after surgery; P = .41). LVEF declined significantly (64% ± 6% vs 57% ± 9% postoperatively; P < .001). There was no difference in mean blood pressure from preoperative to postoperative evaluations (83 ± 8 mm Hg vs 85 ± 7 mm Hg; P = .44), but heart rate was higher postoperatively (71 ± 18 vs

ACD