Mitral Valve Replacement With and Without Chordal Preservation in Patients With Chronic Mitral Regurgitation
Mechanisms for Differences in Postoperative Ejection Performance

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Background. Standard mitral valve replacement (MVR) in patients with chronic mitral regurgitation consistently results in a decrease in postoperative left ventricular (LV) ejection performance. This fall in ejection performance has been attributed, at least in part, to unfavorable loading conditions imposed by the elimination of the low-impedance pathway for LV emptying into the left atrium. In contrast to standard MVR in which the chordae tendineae are severed, however, MVR with chordal preservation (MVR-CP) does not usually decrease LV ejection performance despite similar removal of the low-impedance pathway. The purpose of the present study was to define the mechanisms responsible for this discordance in postoperative ejection performance between MVR with and without chordal preservation.

Methods and Results. Echocardiography and sphygmomanometer blood pressures were obtained in 15 patients with pure chronic mitral regurgitation before and 7–10 days after mitral valve surgery. These measurements were used to calculate ventricular volume, wall stress, and ejection fraction. Seven patients underwent MVR with chordal transection (MVR-CT), and eight patients underwent MVR-CP. MVR-CT resulted in no postoperative change in LV end-diastolic volume, a significant increase in LV end-systolic volume, a significant increase in end-systolic stress, from 89±9 to 111±12 g/cm² (p<0.05), and a significant decrease in ejection fraction, from 0.60±0.02 to 36±0.02 (p<0.05). In contrast, patients who underwent MVR-CP had a significant decrease in LV end-diastolic and end-systolic volumes. End-systolic wall stress actually fell from 95±6 to 66±6 g/cm² (p<0.05), and ejection fraction was unchanged (0.63±0.01 before and 0.61±0.02 after mitral valve surgery) instead of reduced.

Conclusions. MVR-CT resulted in a decrease in ejection performance caused in part by an increase in end-systolic stress, which in turn increased end-systolic volume. Conversely, MVR-CP resulted in a smaller LV size, allowing a reduced end-systolic stress and preservation of ejection performance despite closure of the low-impedance left atrial ejection pathway. (Circulation 1992;86:1718-1726)

Key Words • regurgitation • ejection fraction • valves

In patients with chronic mitral regurgitation, standard mitral valve replacement in which the chordae tendineae are transected (MVR-CT) usually results in a decrease in left ventricular ejection performance.1–7 Reduced postoperative ejection performance has been attributed to the immediate increase in afterload (systolic wall stress) that accompanies the abolition of the low-impedance pathway for ejection into the left atrium8,9 and to ventricular dysfunction caused by disruption of the mitral valve apparatus.5,10,11 In contrast to MVR-CT, valve repair or valve replacement during which the mitral valve apparatus is preserved (MVR-CP) does not result in this fall in postoperative ejection performance.5,12–14 Because these latter procedures also remove the low-impedance left atrial ejection pathway, they might also be expected to cause changes in loading conditions similar to MVR-CT. In both situations, afterload should increase, causing a decrease in ejection performance. Thus, it is unclear how ejection performance is maintained instead of reduced after procedures that conserve the mitral apparatus. To date, although many studies have documented that mitral apparatus preservation results in normal postoperative ejection performance, none have examined changes in postoperative loading conditions that affect ejection performance, and none have explained the mechanisms causing the differences in postoperative ejection performance between chord-conservative and chord-ablative procedures. The major determinants of ejection perfor-
mance are preload, afterload, and contractility. Thus, differences in loading or contractility should explain the observed differences in ejection performance between procedures that preserve versus those that ablate the mitral valve apparatus. In this study, we tested the specific hypothesis that differences in postoperative loading produced by these two different types of mitral valve operations helped explain the observed differences in ejection performance. A further increase in preload after apparatus-conserving procedures could have maintained ejection performance, but this seemed an unlikely occurrence in ventricles that were already volume-overloaded. Therefore, we suspected that it was afterload that failed to increase despite closure of the low-impedance pathway that helped explain maintenance of ejection performance after MVR-CP. To test this hypothesis, we examined performance and loading before and after surgery in two groups of patients: those undergoing MVR-CT and those undergoing MVR-CP. Although this study ignores a variety of other surgical techniques used to correct chronic mitral regurgitation while preserving the mitral apparatus (e.g., primary mitral valve reconstruction), the experimental design of the present study has the advantage that both groups of patients received a mitral valve prosthesis; the only difference between the groups was the preservation or transection of the chordal attachments.

Methods

Patient Selection

Fifteen consecutive, symptomatic patients undergoing mitral valve surgery for pure chronic mitral regurgitation at the Medical University of South Carolina were studied. All gave informed consent. Patients were excluded from the study if they had significant coronary disease; aortic, pulmonic, or tricuspid valve disease; mitral stenosis; or poor-quality echocardiograms. The decision to perform mitral valve surgery was made by physicians not involved in this study and was based on clinical, echocardiographic, hemodynamic, and angiographic criteria. The decision regarding the type of corrective surgery that was performed was made by the cardiovascular surgeon on the sole basis of intraoperative anatomic status of the mitral valve. The mitral apparatus was preserved when possible but ablated when necessary. Thus, this was not a randomized trial.

Each patient was evaluated for symptoms and assigned a functional classification in accordance with the New York Heart Association criteria. All medications being administered before and after mitral valve surgery were recorded. Severe mitral regurgitation was demonstrated in all patients at left ventriculography. The pathogenesis of mitral regurgitation was determined from pathological examination of the excised valve, cardiac catheterization, echocardiography, and by intraoperative inspection.

Surgery

Seven patients underwent standard MVR-CT in which the leaflets were excised. Eight patients underwent MVR-CP.

Mitral valve replacement was performed during cold hyperkalemic cardioplegia. Seven porcine valves (three in MVR-CT, four in MVR-CP) and eight St. Judes prosthetic valves (four in MVR-CT, four in MVR-CP) were inserted. In the MVR-CP patients, one underwent preservation of the anterior leaflet chords only, four underwent preservation of the posterior leaflet chords only, and three underwent preservation of both anterior and posterior leaflet chords. After surgery, serial ECGs and cardiac enzyme determinations detected no evidence of intraoperative myocardial injury. Aortic cross-clamp time and total cardiopulmonary bypass time were similar in the two groups. Doppler echocardiography demonstrated that no patient had postoperative mitral regurgitation.

Measurements

All patients underwent two-dimensional, M-mode, and color flow Doppler echocardiography studies using a Hewlett Packard 77020A ultrasonoscope with either a 2.25- or 3.5-MHz phased-array medium-focus transducer. Preoperative studies were performed within 7 days before surgery; postoperative studies were performed between 7 and 10 days after mitral valve surgery. Echocardiograms were recorded from standard acoustic windows (parasternal long- and short-axis, apical two- and four-chamber, and subcostal views). Echocardiographic data were measured according to American Society of Echocardiography criteria. End diastole was defined as the onset of the Q wave of the ECG, and end systole was defined as the time of peak downward motion of the interventricular septum. We recognize that this definition of end systole does not account for separation of end systole from end ejection in mitral regurgitation.15 We chose this definition because, although considerable left ventricular ejection occurs in mitral regurgitation before aortic valve opening, little occurs after aortic valve closure.16 Thus, minimum volume and volume at aortic valve closure are similar. Further, the above definition uses a definable end point that is reproducible from patient to patient. Blood pressure was obtained by sphygmomanometry.

Calculations

Fractional shortening (FS) was calculated as

\[
FS = \frac{[(EDD - ESD) / EDD] \times 100}{(1)}
\]

where EDD is the end-diastolic dimension and ESD is the end-systolic dimension.

Muscle cross-sectional area (CSA) was used as an index of left ventricular mass and was calculated as

\[
CSA = \pi (EDD/2 + h)^2 - \pi (EDD/2)^2
\]

where h is the left ventricular end-diastolic wall thickness.

Left ventricular volumes (V) were calculated from echocardiographic data as

\[
V = \pi /6D^2L
\]

where D is the left ventricular minor axis dimension acquired from the short-axis view and L is the long-axis dimension acquired from the apical four-chamber view.

End systolic stress was calculated as

\[
P \cdot h / [(1 - h/2b - (b^2/2a^2)] \times 1,336 \text{ dynes/mm Hg}
\]
FIGURE 1. Scatterplot showing correlation between aortic end-systolic (dicrotic notch) pressure and simultaneously obtained sphygmomanometrically determined mean systolic blood pressure for normal subjects. Dashed lines represent 95% confidence limits.

where P is 0.98 times the mean arterial pressure (cuff) plus 11 mm Hg, h is the end-systolic wall thickness, b is the end-systolic semi minor axis [(D+h)/2], and a is the end-systolic semi major axis [(L+h)/2].

End-systolic stress was calculated from the calculated mean arterial blood pressure (cuff), the end-systolic dimension, and the end-systolic wall thickness. Mean arterial pressure (MAP) was calculated as

\[
MAP = \frac{\text{[systolic BP}+2(\text{diastolic BP})]}{3} \tag{5}
\]

MAP is the mean arterial pressure calculated by sphygmomanometer in this study, aortic dicrotic notch pressures were not available. Preliminary observations made from invasive and noninvasive data suggested that mean arterial pressure calculated from cuff pressures closely approximated the aortic dicrotic notch pressure. To confirm these observations, the correlation between aortic dicrotic notch pressure and mean aortic pressure was examined in 25 normal patients. These patients had angiographically confirmed normal coronary arteries, did not have chronic hypertension or valvular heart disease, had angiographically determined ejection fractions ≥50%, and had no segmental wall motion abnormalities. Aortic dicrotic notch pressure was measured with a fluid-filled catheter placed in the ascending aorta during routine diagnostic catheterization in patients being evaluated for chest pain. Mean arterial pressure was calculated by use of Equation 5 from cuff blood pressure measurements made simultaneously with invasively determined aortic pressures. Figure 1 demonstrates an excellent correlation (least-squares linear fit) between aortic dicrotic notch and mean arterial pressures \((y=0.98x+11 \text{ mm Hg}; r=0.99)\). However, chronic mitral regurgitation, which allows left ventricular emptying into the left atrium, producing an early systolic unloading effect, might alter the relation between aortic dicrotic notch and mean arterial pressure. Therefore, we also examined this correlation before mitral valve replacement in all 15 patients with chronic mitral regurgitation included in this study. Figure 2 demonstrates an excellent correlation between aortic dicrotic notch and mean arterial pressures in mitral regurgitation patients; the slope and intercept were similar to those of the normal patients \((y=0.98x+11 \text{ mm Hg}; r=0.99)\). On the basis of these data, the pressure term in the equation for end-systolic stress was derived as

\[
0.98 \text{ (mean arterial cuff pressure)} + 11 \text{ mm Hg}
\]

Statistics

Data are presented as mean±SEM. Differences between preoperative and postoperative values within a group were determined by a Student's paired \(t\) test. Differences between groups were determined by an unpaired \(t\) test. A value of \(p<0.05\) was considered significant.

Results

Preoperative Clinical and Hemodynamic Data

Table 1 demonstrates that there were no differences in the clinical and catheterization data between patients undergoing MVR-CT and MVR-CP. The extent of symptoms, type and number of medications given before surgery, and hemodynamics were comparable in the two groups.

Preoperative Echocardiographic Data

Table 2 provides the raw data from which the volume, wall stress, and ejection fraction data presented in Figures 3–6 were calculated. Preoperative left ventric-
Age (years) 57±5 57±6
Sex (M/F) 3/4 3/5
NYHA functional class, n (%) I 0 0
II 2 (29) 3 (38)
III 3 (42) 3 (38)
IV 2 (29) 2 (25)
Catheterization
Heart rate (bpm) 75±4 76±4
Aortic pressure (systolic, mm Hg) 110±4 111±4
Aortic pressure (diastolic, mm Hg) 66±3 69±4
LV pressure (systolic, mm Hg) 110±4 111±4
LV pressure (diastolic, mm Hg) 18±3 20±3
Pulmonary wedge pressure (mm Hg) 18±3 20±3
Ejection fraction (%) 59±2 63±3
Extent of MR (angiographic grade) 3.6±0.2 3.6±0.2
Pathogenesis of MR (n)
Rheumatic fever 2 1
Mitral prolapse 1 2
Degenerative 4 5
Infective endocarditis 1 1
Rhythm (n)
Sinus 5 7
Atrial fibrillation 2 1
Medications (n)
ACE inhibitor 3 2
Nitrites 2 2
Digoxin 6 5
Calcium channel blocker 0 2
Diuretics 6 5

MVR-CT, mitral valve replacement with chordal transsection; MVR-CP, mitral valve replacement with chordal preservation; NYHA, New York Heart Association; bpm, beats per minute; LV, left ventricular; MR, mitral regurgitation; ACE inhibitor, angiotensin converting enzyme inhibitor. Values are mean±SEM.

Figure 4 demonstrates that circumferential end-systolic stress in the two groups was also similar before surgery. After surgery, end-systolic stress increased significantly from 89±9 to 111±12 g/cm² in the MVR-CT group. In the MVR-CP group, however, there was actually a significant decrease in end-systolic stress from 95±6 to 66±6 g/cm². These opposite and significant changes in end-systolic stress produced a significant difference in end-systolic stress between the two groups after surgery. Figure 5 demonstrates that changes in end-systolic volume mirrored changes in end-systolic stress. In the MVR-CT group, end-systolic volume increased significantly, whereas in the MVR-CP group, end-systolic volume fell significantly. Figure 6 demonstrates that ejection fraction, a reflection of the interaction of the changes noted above, was significantly reduced after surgery in the MVR-CT group but unchanged in the MVR-CP group. Ejection fraction was also significantly worse in the MVR-CT group compared with the MVR-CP group.

Figure 7 demonstrates the ratio of systolic long to short axis, an index of ventricular shape. This ratio fell after surgery in the MVR-CT group, indicating a shape change toward spherical in that group.

We further analyzed the results of those MVR-CP patients who had one versus both sets of chordae tendineae preserved. No differences were found. However, there were only a small number of patients in each subset, reducing the chance of finding a statistical difference.

Discussion

The purpose of this study was to help define the mechanisms by which ejection performance is preserved after MVR-CP but declines after MVR-CT. MVR-CT resulted in a decrease in ejection performance. This decline resulted from an increase in postoperative end-systolic volume that probably was caused by the observed increase in postoperative end-systolic stress. The shape change toward spherical that we observed in the MVR-CT group probably also was responsible for the impaired ejection performance. Unexpectedly, despite removal of the volume overload, end-diastolic volume did not fall significantly in this group. A likely explanation for this finding is that the increased afterload (afterload mismatch) led to the use of preload reserve, which prevented the end-diastolic volume from falling after surgery. It is also possible that with time, end-diastolic volume may decrease as ventricular remodeling occurs in this group.

By contrast, MVR-CP resulted in no significant change in ejection performance. Preservation of ejection performance resulted from a complex interaction of contractile function, preload, and afterload. Reduced preload, indicated by the reduction in end-diastolic volume that we measured in the MVR-CP group, would have actually tended to reduce ejection performance. However, reduced preload also allowed ventricular chamber size to be reduced. Reduced chamber size reduced the radius (or dimension) term in the wall stress equation, allowing systolic wall stress (afterload) to also be reduced. Reduced afterload, probably in conjunction with better contractile function as a result of chordal preservation, allowed a smaller end-systolic volume, offsetting the fall in end-diastolic vol-
Table 2. Preoperative vs. Postoperative Echocardiographic and Hemodynamic Data

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<th>End systolic dimension (cm)</th>
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Group 2: MVR-CP

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*bpm, Beats per minute; BP, blood pressure; Pre, preoperative; Post, postoperative; MVR-CT, mitral valve replacement with chordal transection; MVR-CP, mitral valve replacement with chordal preservation. Data are mean±SEM.

Comparison With Previous Studies

Several clinical studies have examined the effects of mitral valve replacement (with and without chordal preservation) and the effects of primary mitral valve repair on left ventricular volume, mass, and function. Some studies have examined the effects of corrective volume to allow ejection performance to remain unchanged. Thus, although both surgical procedures restored mitral competence and removed the left atrial low-impedance pathway, they had markedly different effects on left ventricular loading and ejection performance.

Figure 3. Graph showing preoperative (pre) and postoperative (post) left ventricular (LV) end-diastolic volume for patients undergoing mitral valve replacement with chordae tendineae severed (open squares) and mitral valve replacement with chordae tendineae preserved (closed circles). Data for individual patients are represented by smaller symbols; mean±SEM is represented by larger symbols. *p<0.05 vs. between two patient groups; †p<0.05 pre vs. post.
mitral surgery in the immediate intraoperative period; others examined late postoperative results, at time periods 6 months to 1 year after corrective surgery. The present study focused specifically on the early postoperative results 1–2 weeks after surgery. At this time, patients were ambulatory, ready for hospital discharge, were relatively pain free, and had normal heart rates, suggesting absence of excessive adrenergic tone. Comparisons between the present and previous studies will concentrate on those studies examining postoperative results at a comparable time period, 1–3 weeks after surgery. At least three studies have examined left ventricular end-diastolic volume, end-systolic volume, and ejection fraction 1–3 weeks after surgery in patients with chronic mitral regurgitation.\textsuperscript{1,4,5} David et al,\textsuperscript{5} using angiographic data, found that MVR-CT resulted in no change in end-diastolic volume and an increase in end-systolic volume after surgery. Schuler et al,\textsuperscript{4} using echocardiographic techniques, found similar results in a group of patients with preoperative left ventricular dysfunction. These studies are concordant with our results for the MVR-CT group. In patients with relatively preserved ventricular function, however, Schuler found a decrease in end-diastolic volume and a decrease in end-systolic volume, unlike our MVR-CT patients. Boucher et al,\textsuperscript{1} using radionuclide angiography, found a postoperative decrease in end-diastolic volume but no change in end-systolic volume. Differences in preoperative left ventricular function among the patients studied may help to account for the differences that MVR-CT produced in ventricular volumes and function in the studies cited.

All three of the above studies performed 1–3 weeks after surgery and many studies performed more than 3 weeks after surgery are concordant with our MVR-CT data, which demonstrated that MVR-CT caused a

**Figure 4.** Graph showing preoperative (pre) and postoperative (post) left ventricular (LV) end-systolic stress for patients undergoing mitral valve replacement (MVR) with chordae tendineae severed (open squares) and MVR with chordae tendineae preserved (closed circles). Data for individual patients are represented by smaller symbols; mean±SEM is represented by larger symbols. *p<0.05 vs. between two patient groups; \(tp<0.05 \) pre vs. post. MVR with chords severed resulted in increased LV afterload, whereas MVR with chords preserved resulted in decreased afterload.

**Figure 5.** Graph showing preoperative (pre) and postoperative (post) left ventricular (LV) end-systolic volume for patients undergoing mitral valve replacement (MVR) with chordae tendineae severed (open squares) and MVR with chordae tendineae preserved (closed circles). Data for individual patients are represented by smaller symbols; mean±SEM is represented by larger symbols. *p<0.05 vs. between two patient groups; \(tp<0.05 \) pre vs. post. MVR with chords preserved resulted in a significant reduction in end-systolic volume.
significant decline in ejection fraction.\textsuperscript{1-7,13} Reduced postoperative ejection fraction after mitral valve replacement is thought to result in part from increased afterload caused by elimination of the left atrial low-impedance ejection pathway. However, only one intraoperative study and two late studies have generated the actual stress data to support this concept.\textsuperscript{8,22,23} The present study lends further support to this hypothesis and confirms that the fall in ejection performance after surgery is caused at least in part by such a postoperative increase in afterload after MVR-CT.

Just as most studies of MVR-CT show a fall in postoperative left ventricular performance, most studies of procedures that conserve the chordal attachments have found maintenance of ejection performance at its preoperative level. A study of MVR-CP and a study of primary mitral valve repair have examined postoperative left ventricular end-diastolic volume, end-systolic volume, and ejection fraction in patients with chronic mitral regurgitation 1–3 weeks after surgery.\textsuperscript{5,24} These studies, using angiographic data, demonstrated that MVR-CP results in a decline in left ventricular end-diastolic volume, a decline in end-systolic volume, and no significant change in left ventricular ejection fraction and are entirely in agreement with our data. Several other studies of procedures that use chordal preservation confirm continued maintenance of ejection performance at late follow-up.\textsuperscript{12,13,25-27} However, none of these studies examining early postoperative results or the studies examining late postoperative results using chordal preservation techniques have examined the effect of corrective surgery on systolic wall stress or have attempted to determine how chordal sparing preserves postoperative ejection performance. Data from the present study are the first to demonstrate that preservation of the chordae tendineae results in a reduced performance.
end-systolic wall stress despite removal of the low-impedance left atrial pathway and suggest this mechanism as one means by which postoperative ejection performance is preserved. Although we did not examine the effects of pure mitral valve repair on ventricular mechanics, our results probably extend to this procedure, because it is probably the preservation of chordal integrity in both replacement and repair procedures that is responsible for improved ventricular mechanics in comparison to procedures that transect the chordae tendineae.

Although our study found that chordal preservation maintained ventricular performance, at least in part as a result of reduced afterload compared with chordal transection, our study did not specifically examine the effects of chordal preservation on chamber contractile function. In the normal heart with normal papillary-annular continuity, Rushmer\textsuperscript{20} found that during contraction in the early isovolumic portion of systole, shortening of the major axis produced lengthening of the minor axis, causing the left ventricle to become more spherical. Early movement of the atrioventricular ring toward the apex produced by papillary muscle contraction may increase preload in the circumferential midwall fibers and enhance ejection performance via the Frank-Starling mechanism. This early systolic shape change toward spherical, which is beneficial, should not be confused with late sphericity, which may be detrimental.\textsuperscript{18} The importance of preserving the mitral valve apparatus in maintaining normal left ventricular mechanics and contractile function has been emphasized in several experimental and clinical studies. These studies demonstrated that disrupting the chordae tendineae caused a reduction in contraction in left ventricular segments adjacent to the papillary muscles, a less spherical geometry during isovolumic systole, a more spherical geometry at end systole, and a reduction in contractile state.\textsuperscript{10,28,29} In isovolumic canine heart preparations, chordal disruption resulted in a decline in the slope of the end-systolic pressure–volume relation, a relatively load–insensitive index of chamber contractile function.\textsuperscript{23} Reattachment of the mitral chordae caused the slope to return to normal. Chordal preservation was also important in preserving systolic function after mitral valve replacement in vivo in two recent studies in animals with chronic mitral regurgitation.\textsuperscript{30,31} Experimental studies have also demonstrated that the anterior and posterior leaflet chordae had similar but additive contributions to contractile function.\textsuperscript{32} In this regard, although we did not find a significant difference in function between patients with one versus both sets of chordae tendineae preserved, the numbers of patients in both groups were too small for optimum statistical comparison.

Taken together, these data suggest that reduced chamber size, reduced systolic load, and preservation of chamber contractile function act in concert to maintain ejection performance after chordal-conservative procedures for correction of mitral regurgitation. Conversely, an increased chamber size, increased systolic load, and probable reduction in chamber contractile function act in concert to reduce ejection performance after MVR-CT.

Limitations

A limitation of this study is that it was not a prospective, randomized trial. The decision regarding the choice of corrective procedures was left entirely to the operating surgeon and was not based on preoperative hemodynamic or echocardiographic data. The purpose of randomization, however, is to eliminate differences other than the experimental variable being tested as causes for the observed results. In this regard, all of the preoperative clinical hemodynamic and echocardiographic variables were comparable in the two groups. Thus, the patients in the two groups were similar to one another before surgery. We believe that the significant postoperative differences we observed are based primarily on differences accrued from chordal transection versus preservation. We cannot rule out, however, that subtle differences between the groups caused by selection bias impinged on our results.

It is well recognized that in the postoperative period, abnormal motion of the interventricular septum may occur in some patients after mitral valve replacement and may alter the indexes of ejection performance. This is most important when M-mode echocardiographic measurements are used. In the present study, dimensions and thickness were determined by both twodimensional and M-mode measurements. More importantly, mitral valve replacement was performed in both experimental groups, which should control for this potential problem.

Finally, differences in ventricular performance have been noted when midwall fiber shortening versus endocardial shortening is examined.\textsuperscript{33} We believe that examination of muscle performance is best served by midwall fiber examination, whereas examination of pump performance is best examined by endocardial shortening, because the latter is responsible for expulsion of volume from the heart. In this study, our purpose was to examine pump performance, not contractile function; therefore, we examined endocardial volumes and shortening.

Summary

Data from the present study help reconcile the observed differences in ejection performance after MVR-CP versus MVR-CT. Both surgical procedures restore mitral competence and remove the low-impedance left atrial ejection pathway. Although these events might be expected to have similar effects on left ventricular afterload, our data demonstrate that severing the chordae tendineae causes an increase in afterload and a decrease in ejection performance. In contrast, chordal preservation allows a smaller chamber size, prevents the postoperative increase in systolic stress, and maintains normal ejection performance. This study implies that in patients with marginal or diminished preoperative left ventricular function, every effort should be made to preserve the chordae tendineae with the aim of minimizing or eliminating a postoperative decline in left ventricular systolic function.

Acknowledgment

The authors wish to thank Bev Ksenzak for her assistance in the preparation of this manuscript.
References

J D Rozich, B A Carabello, B W Usher, J M Kratz, A E Bell and M R Zile

_Circulation_. 1992;86:1718-1726
doi: 10.1161/01.CIR.86.6.1718

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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