Cutting Second-Order Chords Does Not Prevent Acute Ischemic Mitral Regurgitation

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**Background**—Cutting anterior mitral leaflet second-order chordae has been proposed for repair in ischemic mitral regurgitation (IMR). We examined the efficacy of such chordal cutting in preventing acute IMR.

**Methods and Results**—Six sheep underwent radiopaque marker placement (left ventricle, mitral annulus, papillary muscles [PMs], and leaflets). The largest second-order chord from each PM was encircled with exteriorized wire snares. Three-dimensional marker coordinates were obtained with biplane videofluoroscopy before and during acute ischemia (80 seconds of mid-circumflex occlusion). Color Doppler transesophageal echocardiography was used to grade MR on a 0 to 4+ scale. Data were acquired immediately before and after dividing second-order chordae. Slope of the end-diastolic volume–stroke work relationship (PRSW) was calculated to assess systolic function. Chordal cutting increased anterior leaflet inflection angle (155±12 versus 162±9 degrees; P=0.03), resulting in a flatter leaflet, but did not increase effective leaflet length (1.97±0.24 versus 2.08±0.23 cm; P=0.15); PRSW decreased (63±15 versus 56±12 mm Hg; P=0.008). Both before and after chordal cutting, ischemia caused: Septal–lateral annular dilation (P=0.005), posterior PM displacement away from the mid-septal annulus (P=0.006), increased leaflet tenting area (P=0.001), and increased leaflet tenting volume (P=0.002). Before chordal cutting, MR increased significantly during ischemia (0.5±0.3 versus 1.7±0.4; P<0.001), and IMR increased similarly even after the second-order chords were cut (0.7±0.4 versus 1.9±0.9; P<0.001).

**Conclusions**—Cutting second-order chordae resulted in LV systolic dysfunction and neither prevented nor decreased the severity of acute IMR, septal–lateral annular dilation, leaflet tenting area, or leaflet tenting volume. (*Circulation*. 2004;110[suppl II]:II-91–II-97.)

**Key Words:** mitral valve ■ ischemia ■ regurgitation ■ remodeling ■ contractility

Ischemic mitral regurgitation (IMR) is an important complication after myocardial infarction that is associated with excess mortality independent of underlying left ventricular dysfunction.1 Mitral annular dilation, particularly in the septal–lateral (ie, antero-posterior) dimension, is important for the development of acute and chronic IMR.2–4 Ring annuloplasty, the standard surgical treatment for IMR, resolves this annular dilation, but can be associated with residual or recurrent MR in 9% to 29% of patients within 5 years of follow-up.5,6

Such variable results, combined with an increased understanding that IMR is as much a ventricular as a valvular problem, have prompted the advance of subvalvular adjuncts to ring annuloplasty for IMR.5–11 Kron et al have described surgical relocation of the posterior papillary muscle (PPM) using a traction suture under direct vision.7 In a series of elegant experiments using 3-dimensional echocardiography, Levine et al demonstrated the efficacy of repositioning the PPM to ameliorate IMR via surgical infarct plication8 or by placing and adjusting an external patch with an inflatable balloon over the inferior infarction.9 Such new adjunctive surgical reparative techniques for IMR are important, as failure to address the subvalvular changes may contribute to the variability of results and recurrence of MR after ring annuloplasty, which primarily addresses the annular dilation.

The subvalvular apparatus consists of the PMs and 2 major sets of chordae radiating from them to support the mitral leaflets. First-order (ie, primary or “marginal”) chordae insert on the leaflet free edges. Second-order chordae, larger and fewer in number, insert on the ventricular surface of the anterior leaflet belly at the junction of the rough and smooth zones and often include 2 thicker basal, or “strut,” chordae.12 Numerous clinical13 and experimental studies3,4,8,14 have demonstrated the role of PPM displacement, secondary to...
remodeling and distortion of the ischemic/infarcted LV, in the development of IMR. Such PPM displacement results in leaflet tethering and incomplete mitral leaflet closure via the chordal attachments.4,13,14

The importance of the mitral subvalvular apparatus for optimal LV function (ie, valvular–ventricular interaction) has been demonstrated in clinical studies showing improved patient outcomes and LV performance with chordal preservation during mitral valve replacement15,16 and experimental studies documenting global LV contractile dysfunction after transection of all the chords.17,18 Regional LV systolic dysfunction has also been observed adjacent to the PM insertion sites after transection of the anterior mitral leaflet (AML) second-order chords.19

Messas et al have recently proposed cutting these AML second-order chordae tendineae as a therapeutic option for patients with IMR to improve leaflet coaptation and decrease MR by reducing leaflet tethering at the insertion sites of the second-order chordae on the AML belly.10,11 Because mitral valvular–ventricular continuity has been shown to be important to LV systolic function, we reasoned that chordal cutting for valve repair in already impaired hearts might not be an optimal therapeutic option. To gain mechanistic understanding of cutting second-order chords for IMR, we tested the efficacy of such chordal cutting in preventing acute IMR.

Materials and Methods

All animals received humane care in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the Guide for Care and Use of Laboratory Animals prepared by the National Academy of Sciences and published by the National Institutes of Health (DHEW NIH Publication 85-23, revised 1985). This study was approved by the Stanford Medical Center Laboratory Research Animal Review committee and conducted according to Stanford University policy.

Surgical Preparation

Sixteen Dorsett hybrid sheep (73±10 kg) were premedicated with ketamine (25 mg/kg intramuscularly) and anesthesia was induced with sodium thiopental (6.8 mg/kg intramuscularly) and maintained with inhalational isoflurane (1.5% to 2.2%). Through a left thoracotomy, 12 radiopaque tantalum helical markers (outer diameter, 1.3 mm; length, 1.5 to 3.0 mm) were inserted in the subepicardium to silhouette the LV along 4 equally spaced longitudinal meridians defining anterior, lateral, posterior, and septal walls at basal, equatorial, and apical levels; one marker was placed on the LV apex (1 to 13; Figure 1a). Pneumatic occluders (In Vivo Metric Systems) were placed around the inferior vena cava (to provide a means for transient preload reduction) and the mid-circumflex artery distal to the first obtuse marginal (for inducing transient posterolateral ischemia).

On cardiopulmonary bypass (CPB) after cardioplegic arrest and working through a left atriotomy, 8 markers were sutured around the circumference of the mitral annulus ([MA] 14 to 21; Figures 1 and 2). Three markers were sutured along the central meridian of the
bretylium (50 mg) before transfer as prophylaxis against rhythm disturbances. The strut chords were then cut by passing electrocautery current through the externalized wire snares (cut). Data were then acquired before (postcut, ischemia) and after cut during (postcut, ischemia) mid-circumflex occlusion. Simultaneous transesophageal color Doppler echocardiography allowed evaluation of the degree of MR before and after cut by an experienced echocardiographer (D.L.) on a 0 to 4+ scale (none, mild, moderate, moderate–severe, and severe).

Figure 2. a, Anterior mitral leaflet inflection angle was calculated between the mid-septal annulus (14) and the leaflet edge marker (26) with the vertex defined by the centroid of the 2 central meridian mid-leaflet markers (24 and 25). This leaflet angle of inflection corresponds to the leaflet “bend” visualized on echocardiography. Arrow indicates measured inflection angle. AML indicates anterolateral mitral leaflet; PML, posterolateral mitral leaflet. b, Anterior mitral leaflet “effective” length was calculated as the distance in 3-dimensional space between the mid-septal annular and leaflet edge markers (14 and 26).

AML, including the central leaflet edge, and 2 markers were sutured along the midline of the posterior middle scallop (24 to 28; Figures 1b and 2). The largest of the second-order chordae from each PM inserting into the AML belly were identified (ie, the 2 most medial “strut” chordae), and markers were placed at their AML insertion points (29 and 30; Figure 1b). Wire snares (insulated except where they encircled the chordae) were secured around these strut chordae and exteriorized through the LV wall. Care was taken to ensure that these snares encompassed no first-order chordae. Markers were placed on each PM tip near the origin of the strut chordae (22 and 23; Figure 1a). A micromanometer-tipped catheter (Millar SPC-500, Millar Instruments, Inc) was inserted through the LV apical vent site to monitor LV pressure (LVP). Animals were weaned from CPB and received intravenous magnesium (3 g), lidocaine (100 mg), and bretylium (50 mg) before transfer as prophylaxis against rhythm disturbances. Epicardial color Doppler echocardiography confirmed normal leaflet motion and valvular competence.

Experimental Protocol
Immediately after operation, animals were transferred to the catheterization laboratory and studied with the chest open; maintenance anesthesia was continued with inhalational isoflurane (1.5% to 2.2%). Sequences of biplane videofluoroscopic and hemodynamic data were acquired over a physiological range of LV filling volumes during transient vena caval occlusion with animals in the right lateral decubitus position and ventilation briefly arrested at end-expiration (to prevent respiratory variation). With the second-order chords intact, baseline data were acquired (precut, preischemia). After a 5-minute interval, the mid-circumflex was occluded for 78 ± 32 seconds (sufficient to induce MR), at which time data were acquired during ongoing ischemia (precut, ischemia). Animals were allowed to stabilize for 3 to 5 minutes between all data acquisition sequences. Repeat baseline data (precut, postischemia) were then acquired for the purposes of another analysis.20 Before chordal cutting, animals received an additional 100 mg of lidocaine (intravenous) as prophylaxis against rhythm disturbances. The strut chords were then cut by passing electrocautery current through the externalized wire snares (cut). Data were then acquired before (postcut, preischemia) and during (postcut, ischemia) mid-circumflex occlusion. Simultaneous transesophageal color Doppler echocardiography allowed evaluation of the degree of MR before and after cut by an experienced echocardiographer (D.L.) on a 0 to 4+ scale (none, mild, moderate, moderate–severe, and severe).

Data Acquisition
A Philips Optimus 2000 biplane Lateral ARC 2/poly DIAGNOST C2 system (Philips Medical Systems) was used to record videofluoroscopic images at 60 Hz. Two-dimensional images from the 2 x-ray views were digitized using custom software.21 These data were merged to yield 3-dimensional coordinates for each radiopaque marker every 16.7 ms, the accuracy of which is 0.1 ± 0.3 mm compared with known marker-to-marker 3-dimensional lengths.22 Analog LVP and ECG signals were digitized simultaneously and recorded on videotape along with marker images during data acquisition.

Data Analysis
Of 16 animals entered into this experiment, 6 had successful transection of both second-order strut chords without damage to first-order chords or AML and without ventricular fibrillation during the cutting attempt. These 6 animals comprise the present study group. Six other animals had inadvertent damage to first-order chords, which was evident immediately on echocardiography and confirmed on necropsy, and 4 animals fibrillated during the cutting attempt. These 10 animals were excluded.

End-diastole (ED) was defined as the maximum of the second derivative of LVP, corresponding with the frame immediately before the upstroke of the LVP curve. End-systole (ES) was defined as the videofluoroscopic frame before the time of peak negative LV rate of pressure fall (−dP/dt<sub>max</sub>). Instantaneous LV volume was calculated from LV and MA markers using multiple tetrahedra constructed from the marker coordinates and corrected for LV convexity.23 Although epicardial LV volume calculated in this manner overestimates true LV chamber volume (because it incorporates an unknown amount of LV muscle mass), changes in this “epicardial” LV volume are an accurate measurement of the relative changes in LV chamber volume because LV muscle mass remains constant throughout the cardiac cycle.24 In 5 of the sheep with successful cut, adequate vena caval occlusions (preload reduction) during data acquisition allowed calculation of preload recruitable stroke work (PRSW), a load-independent index of LV systolic function, defined as the slope of the end-diastolic volume–stroke work relationship.25 Three nonocclusion beats and 2 occlusion beats were analyzed.

Measurements of mitral annular and leaflet geometry were made at LVP<sub>MAX</sub>. Mitral annular septal–lateral dimension was calculated as the distance in 3-dimensional space between the markers placed in the middle of the septal and lateral MA, respectively (14 and 18; Figures 1 and 2). Commissure–commissure dimension was calculated as the distance between the annular commissural markers (16 and 20; Figures 1 and 2). The AML angle of inflection was calculated between the mid-septal annulus and the anterior leaflet edge marker (14 and 26; Figure 2a) with the vertex defined by the centroid of the 2 mid-leaflet markers along the central meridian (24 and 25). This leaflet “angle of inflection” was calculated to approximate the bend in the anterior leaflet (ie, the “knee”) that is visualized with echocardiography.10,11 Effective leaflet length was calculated as the distance between the annular commissural markers (16 and 20) and leaflet markers (24 to 30; Figure 1b) using multiple tetrahedra constructed from the marker coordinates.

Papillary muscle displacements at LVP<sub>MAX</sub> were calculated as the distance in 3-dimensional space between PM tips (22 and 23) and MA saddle horn (14; Figure 1a).

All data are reported as mean ± SD. Hemodynamic and marker-derived data from 3 consecutive steady-state beats in sinus rhythm
TABLE 1. Hemodynamics

<table>
<thead>
<tr>
<th>Hemodynamics</th>
<th>Preischemia</th>
<th>Ischemia</th>
<th>Preischemia</th>
<th>Ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (min⁻¹)</td>
<td>107±9</td>
<td>102±10</td>
<td>106±10</td>
<td>102±11</td>
</tr>
<tr>
<td>LV dP/dtmax (mm Hg/sec)</td>
<td>2 540±647</td>
<td>1 690±503†</td>
<td>2 640±503</td>
<td>1 780±11†</td>
</tr>
<tr>
<td>LVP max (mm Hg)</td>
<td>108±18</td>
<td>90±16†</td>
<td>112±11</td>
<td>94±11†</td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>18±4</td>
<td>25±8†</td>
<td>16±4</td>
<td>22±6†</td>
</tr>
<tr>
<td>ESP (mm Hg)</td>
<td>78±11</td>
<td>60±11†</td>
<td>82±17</td>
<td>67±14†</td>
</tr>
<tr>
<td>EDV (mL)</td>
<td>194±45</td>
<td>221±44†</td>
<td>197±43</td>
<td>219±41†</td>
</tr>
<tr>
<td>PRSW (mm Hg)</td>
<td>63±15</td>
<td>37±7†</td>
<td>56±12*</td>
<td>37±10†</td>
</tr>
<tr>
<td>MR (0–4+ scale)</td>
<td>0.5±0.3</td>
<td>1.7±0.4†</td>
<td>0.7±0.4</td>
<td>1.9±0.9†</td>
</tr>
</tbody>
</table>

*P<0.05 preischemia vs ischemia, 2-way repeated measures ANOVA with Student-Newman-Keuls posttest.
†P<0.05 preischemia vs ischemia, 2-way repeated measures ANOVA with Student-Newman-Keuls posttest.

Data expressed as mean±SD.

Results

Correct marker placement and successful transection of both strut chords without damage to first-order chordae or AML in the 6 study group animals was confirmed on necropsy. CPB time was 78±10 minutes, and aortic cross-clamp time was 59±8 minutes. Duration of acute ischemia was 78±32 seconds both before and after cut. No animals had rhythm disturbances during ischemia.

Hemodynamics

Cutting the second-order chords resulted in global LV dys-function. Load-independent PRSW fell significantly after chordal cutting (63±15 mm Hg versus 56±12 mm Hg; P=0.008), although other load-dependent hemodynamic parameters were unchanged (Table 1). As expected, posterolateral ischemia caused significant deleterious hemodynamic changes.

Ischemic MR

As shown in Table 1 and Figure 3, trace MR was detected at baseline before chordal cutting and this increased significantly to moderate MR during ischemia (0.5±0.3 versus 1.7±0.4; P<0.001). After chordal cutting, there was no change in the degree of baseline MR (0.5±0.3 versus 0.7±0.4; P=0.32), and chordal cutting neither prevented nor decreased the severity of IMR (0.7±0.4 versus 1.9±0.9; P<0.001). Not surprisingly, in the 6 animals with damaged first-order chords, MR increased significantly (0.8±0.4 versus 2.5±1.2; P=0.03), demonstrating the well-known importance of primary chords to valvular competence.

Papillary Muscle and Mitral Valvular Geometry

Table 2 summarizes mitral valve and papillary muscle geometry at LVPmax. Chordal cutting increased AML inflection angle (155±12 versus 162±9 degrees; P=0.03), resulting in a flatter leaflet, but this did not increase AML effective length (1.97±0.24 versus 2.08±0.23 cm; P=0.15) nor decrease leaflet tenting area (P=0.36) or volume (P=0.13). Both before and after chord transection, posterolateral ischemia caused septal–lateral annular dilation (P=0.005), displacement of the PPM tip away from the MA “saddle horn” (P=0.06), increased leaflet tenting area (P=0.001), and increased leaflet tenting volume (P=0.002).
In this study, cutting second-order chords neither prevented nor decreased the severity of acute IMR, septal–lateral annular dilation, leaflet tenting area, or leaflet tenting volume, showing that chordal division does not benefit acute IMR. Moreover, we found that cutting the strut chords resulted in global LV systolic dysfunction, demonstrating the importance of second-order chords for mediating valvular–ventricular interaction.

Cutting the second-order chordae failed to prevent acute IMR, probably because chordal cutting does not address the septal–lateral mitral annular dilation that is an important factor in both acute and chronic IMR. For the mitral valve to remain competent, increase in this dimension requires the leaflets to cover more area, using the normal excess “reserve” of leaflet tissue. If this requirement for more mitral leaflet tissue cannot be met, the valve leaks. This is additionally compounded by less effective leaflet closure because of apical tethering or tenting of the leaflets during ischemia. In particular, geometric distortions of the mitral valvular–ventricular complex, which result in apical displacement of the posterior leaflet margin, cause relative prolapse of the AML in both acute and chronic IMR. The proposals for chordal cutting as a therapeutic intervention for both acute and chronic IMR describe improved leaflet coaptation after chordal cutting by eliminating the bend in the AML belly caused by tethering at the second-order chord insertion sites. An increase in AML effective length after such chordal cutting could help the AML reach across to the apically tethered posterior mitral leaflet. Consistent with these proposals, we did observe an increase in the AML inflection angle (which corresponds to the leaflet bend observed on echo) after chordal cutting in the present study. Increased inflection angle reflects flattening of the AML after chordal cutting; however, this was not accompanied by a significant increase in effective leaflet length (Table 2). Moreover, tenting area, which has been reported as a major determinant of functional (ie, ischemic) MR, increased similarly during ischemia. Tenting volume also increased similarly during ischemia before and after chordal cutting (Table 2), in contrast to previous reports. Increased tenting area and tenting volume during ischemia despite transection of the second-order chords may reflect septal–lateral dilation and increased mitral annular area. In addition, the leaflets may remain tethered by the first-order chords as the PPM shifts away from the mid-septal annulus during ischemia (Table 2).

We were unable to replicate the findings reported previously from a similar acute open-chest ovine preparation reported by the MGH group. In both their original acute and later chronic studies, however, Messas et al compared echo data acquired before chordal cutting on CPB with postchordal cutting data after CPB. This may account for the different results in the present study, because our experimental design allowed for sequential comparisons between precut and postcut conditions without confounding factors such as an intervening procedure on CPB or major preload shifts between study conditions. Further, in their chronic study, a separate group of 5 animals was also followed-up for 8 months after chordal cutting and inferior infarction, with no more than trace MR noted despite significant LV postinfarction remodeling (increased LVESV and LVEDV). We have recently demonstrated, however, that such inferior infarction and LV remodeling does not always result in IMR, even with the second-order chords intact; in our experiment, only 10 of 16 animals had IMR postinfarction. Duran has also reported chronic LV geometric perturbations resulting in variable degrees of IMR and heart failure after chordal cutting in sheep.

The present study also demonstrates that the second-order chordae are important for valvular–ventricular interaction and, thereby, help maintain LV systolic pump function. Despite preservation of first-order chordae, division of the

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**TABLE 2. Mitral Valve Geometry at LVPmax**

<table>
<thead>
<tr>
<th>Geometry at LVPmax</th>
<th>Precut</th>
<th>Ischemia</th>
<th>Postcut</th>
<th>Ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>AML inflection angle (°)</td>
<td>155±12</td>
<td>155±9</td>
<td>162±9*</td>
<td>160±8</td>
</tr>
<tr>
<td>AML effective length (cm)</td>
<td>1.97±0.24</td>
<td>2.08±0.23†</td>
<td>2.08±0.28</td>
<td>2.13±0.22†</td>
</tr>
<tr>
<td>Tenting area (cm²)</td>
<td>1.35±0.15</td>
<td>1.53±0.17†</td>
<td>1.41±0.22</td>
<td>1.54±0.20†</td>
</tr>
<tr>
<td>Tenting volume (cm³)</td>
<td>1.96±0.31</td>
<td>2.38±0.29†</td>
<td>1.85±0.47</td>
<td>2.20±0.33†</td>
</tr>
<tr>
<td>MA S-L dimension (cm)</td>
<td>2.64±0.18</td>
<td>2.87±0.13†</td>
<td>2.63±0.19</td>
<td>2.86±0.15†</td>
</tr>
<tr>
<td>MA C-C dimension (cm)</td>
<td>3.60±0.15</td>
<td>3.74±0.23</td>
<td>3.60±0.25</td>
<td>3.67±0.19</td>
</tr>
<tr>
<td>Saddle horn, PPM (cm)</td>
<td>5.10±0.50</td>
<td>5.18±0.54†</td>
<td>5.15±0.51</td>
<td>5.22±0.53†</td>
</tr>
<tr>
<td>Saddle horn, APM (cm)</td>
<td>4.75±0.69</td>
<td>4.79±0.71</td>
<td>5.00±1.13</td>
<td>4.83±0.77</td>
</tr>
</tbody>
</table>

*p<0.05 precut vs postcut, 2-way repeated measures ANOVA with Student-Newman-Keuls posttest.
†p<0.05 preischemia vs ischemia, 2-way repeated measures ANOVA with Student-Newman-Keuls posttest.
‡p<0.06 preischemia vs ischemia, 2-way repeated measures ANOVA, likely significant.

AML indicates anterior mitral valve leaflet; effective length, distance from mid-septal annulus to AML edge marker; MA, mitral annular; S-L, septal–lateral; C-C, commissure–commissure; saddle horn, mid-septal mitral annulus; PPM, posterior papillary muscle; APM, posterior papillary muscle.

Data expressed as mean±SD.
second-order strut chordae resulted in deterioration of global LV preload recruitable stroke work (Table 1). The mechanisms responsible may be related to the second-order chordae maintaining systolic temporal dynamics, elliptical LV geometry, and LV wall-thickening mechanics. No difference in PRSW was observed between precut preischemia and precut postischemia conditions (63±15 versus 65±13 mm Hg; P=0.43, t test for paired observations), although PRSW fell as expected during ischemia (Table 1). Hence, the fall in PRSW after chordal cutting resulted from the transection of the second-order chordae and not ischemic myocardial injury or the effects of anesthesia over time.

Ischemic MR is a complex condition resulting from mitral annular dilation (especially in the septal–lateral dimension) and displacement of the posterior PM after myocardial infarction, which combined prevent competent leaflet closure. Subvalvular adjuncts to ring annuloplasty7–11 are key to rectify restricted systolic (Carpentier type IIib) leaflet motion because of apical leaflet tethering and improve surgical outcomes. Although chordal cutting has been proposed as an adjunctive reparative technique, cutting second-order chords in the present study resulted in global LV systolic dysfunction and neither prevented nor decreased the severity of acute IMR. These findings argue against procedures that sacrifice second-order chords for mitral valve repair. Moreover, we found that cutting the strut chords in beating hearts was hazardous. Despite intraoperative isolation of the second-order chordae under direct visualization in arrested hearts, first-order chordae were inadvertently injured. Because these first-order (or marginal) chordae provide valvular competence, this experience argues against catheter-based approaches for chordal cutting in beating hearts.

Study Limitations

These data were obtained in an acute, open-chest setting in normal sheep hearts immediately after an open cardiac surgical procedure; therefore, direct extrapolation of these findings to humans must be performed with caution. Although this experiment was performed in normal animal hearts, we attempted to replicate the clinical scenario of working on injured hearts by inducing transient ischemia to create IMR before and after cutting the strut chords. Another limitation is that we studied the efficacy of chordal cutting in the setting of acute IMR; the mechanisms described, however, are probably similar in the chronic IMR context. Septal–lateral mitral annular dilation is important in both acute and chronic IMR,2–4 and similar alterations in subvalvular geometry characterized by posterior, lateral, and basal displacement of the PPM causing posterior leaflet apical displacement occur during acute ischemia,3 although not as pronounced as after chronic posterior infarction.4 These similarities support the use of an acute ischemia model to test the efficacy chordal cutting. Differences in comparative anatomy between human and sheep mitral valve and subvalvular apparatus might also be important. Sheep may have a relatively small leaflet area to annular area ratio, which makes them more susceptible to smaller increases in annular area compared with man. This raises the possibility that chordal cutting might have some efficacy in humans that we were unable to demonstrate in our laboratory experiment. However, because the strut chordae are thinner in ovine hearts, we anticipate that the role of the strut chordae in human valvular–ventricular interaction and the effect of chordal cutting on systolic function might be even more pronounced. We acknowledge, however, that the changes in LV function observed could possibly be caused by collateral damage to the endocardial LV surface from the electrocautery or perhaps mechanical “tugging” on the second-order chordae during transection. It is unlikely, however, that deterioration of ventricular function under anesthesia contributed significantly to the observed decrease in PRSW because postcut data were acquired within 5 minutes of precut data.

Acknowledgments

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References


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