Conclusions—Basal repositioning of the PPM with STRING-1 reduced acute IMR without concomitant annular reduction.

Methods and Results—Ten sheep had radiopaque markers placed on the left ventricle (LV) and mitral apparatus. A suture was anchored at the right fibrous trigone, passed through the PPM tip and LV wall, and exteriorized through a tourniquet (STRING-1). A second suture was anchored transmurally in the high septum (anterobasal LV wall) and passed through the PPM and LV wall (STRING-2). Reversible posterolateral ischemia was induced by temporarily occluding the proximal circumflex artery. Under open chest conditions, 3D marker coordinates were obtained with biplane videofluoroscopy at baseline and during acute ischemia before and after tightening of each STRING using transesophageal echocardiography to grade IMR. IMR decreased (mean±SEM, 2.0±0.1 to 1.2±0.1; P<0.05) when STRING-1 was tightened, did not change after tightening STRING-2 (2.3±0.1 to 2.3±0.1), and decreased after tightening both sutures (STRING-1+2, 2.3±0.2 to 1.3±0.2; P<0.05). STRING-1 and STRING-1+2 (STRING-1, 1.7±0.4 mm; STRING-2, 0.7±0.5 mm; STRING-1+2, 1.5±0.3 mm; P<0.05) resulted in significant PPM basal repositioning. Tightening of any STRING sutures did not affect anterior mitral leaflet excursion.

Conclusions—Basal repositioning of the PPM with STRING-1 reduced acute IMR without concomitant annular reduction. This technique may be a useful adjunct if residual IMR is likely after undersized ring annuloplasty. (Circulation. 2005; 112[suppl I]:I-383-I-389.)

Key Words: ischemic mitral regurgitation ■ mitral valve ■ mitral valve repair

Subvalvular Repair

The Key to Repairing Ischemic Mitral Regurgitation?

Frank Langer, MD; Filiberto Rodriguez, MD; Saskia Ortiz, MD; Allen Cheng, MD; Tom C. Nguyen, MD; Mary K. Zasio, BA; David Liang, MD, PhD; George T. Daughters, MS; Neil B. Ingels, PhD; D. Craig Miller, MD

Background—Residual or recurrent mitral regurgitation frequently occurs after mitral ring annuloplasty repair for ischemic mitral regurgitation (IMR), because annuloplasty primarily addresses annular dilatation. We describe a subvalvular repair technique addressing posterior papillary muscle (PPM) displacement.

Methods and Results—Ten sheep had radiopaque markers placed on the left ventricle (LV) and mitral apparatus. A suture was anchored at the right fibrous trigone, passed through the PPM tip and LV wall, and exteriorized through a tourniquet (STRING-1). A second suture was anchored transmurally in the high septum (anterobasal LV wall) and passed through the PPM and LV wall (STRING-2). Reversible posterolateral ischemia was induced by temporarily occluding the proximal circumflex artery. Under open chest conditions, 3D marker coordinates were obtained with biplane videofluoroscopy at baseline and during acute ischemia before and after tightening of each STRING using transesophageal echocardiography to grade IMR. IMR decreased (mean±SEM, 2.0±0.1 to 1.2±0.1; P<0.05) when STRING-1 was tightened, did not change after tightening STRING-2 (2.3±0.1 to 2.3±0.1), and decreased after tightening both sutures (STRING-1+2, 2.3±0.2 to 1.3±0.2; P<0.05). STRING-1 and STRING-1+2 (STRING-1, 1.7±0.4 mm; STRING-2, 0.7±0.5 mm; STRING-1+2, 1.5±0.3 mm; P<0.05) resulted in significant PPM basal repositioning. Tightening of any STRING sutures did not affect anterior mitral leaflet excursion.

Conclusions—Basal repositioning of the PPM with STRING-1 reduced acute IMR without concomitant annular reduction. This technique may be a useful adjunct if residual IMR is likely after undersized ring annuloplasty. (Circulation. 2005; 112[suppl I]:I-383-I-389.)

Key Words: ischemic mitral regurgitation ■ mitral valve ■ mitral valve repair

Ring annuloplasty is the preferred repair technique for ischemic mitral regurgitation (IMR), but residual or recurrent mitral regurgitation (MR) is seen in up to 30% of patients. Although undersized annular reduction can correct both annular and subvalvular geometry in IMR, annular reduction with ring annuloplasty primarily addresses the annular dilatation in IMR, and leaflet tethering because of posterior papillary muscle (PPM) displacement often persists, which can result in early or midterm repair failure.

Experimental studies have suggested repositioning of the PPM to ameliorate IMR via surgical infarct plication or by placing and adjusting an external patch with an inflatable balloon over the infarction territory. Infarct restraint has also been shown to attenuate remodeling and reduce chronic IMR. Cutting second-order chordae has also been proposed to reduce leaflet tethering, but chordal cutting depresses global left ventricular (LV) systolic function. Clinically, only the “edge-to-edge” technique of Alfieri et al has been used as an adjunctive repair technique in combination with ring annuloplasty; however, midterm results of this approach have been discouraging.

Recently, Kron et al reported successful internal direct repositioning of the displaced PPM as an adjunct to ring annuloplasty using a subvalvular transventricular suture to anchor the PPM to the mitral annulus just posterior to the right fibrous trigone. Building on this report and based on our observation from a previous experiment that chronic IMR was associated with significant PPM posterolateral displacement, we tested the hypothesis that direct internal repositioning of the PPM reduces IMR. We evaluated 2 different repositioning directions to identify a preferred method for this repair approach without concomitant annular size reduction.

Methods

Surgical Preparation.

Ten sheep (68±5 kg) were premedicated with ketamine (25 mg/kg IM), and anesthesia was induced with sodium thiopental (6.8 mg/kg IV).
IV) and maintained with inhalational isoflurane (1% to 2.5%). Through a left thoracotomy, miniature tantalum myocardial markers (#2 to 14, Figure 1A) were inserted in the LV epicardial layer along 4 equally spaced longitudinal meridians, with 1 marker at the LV apex (#1, Figure 1A). A snare was placed around the left circumflex artery proximal to the first obtuse marginal branch for induction of reversible posterolateral myocardial ischemia sufficient in severity and duration to result in acute IMR. After establishment of cardiopulmonary bypass, the ascending aorta was cross-clamped. Cardioplegia was induced by retrograde infusion of crystalloid cardioplegic solution. A left atriotomy was performed, and tantalum markers were placed at the tips and bases of both anterior papillary muscle (#28 and #29) and PPM (#30 and #31). Eight markers were sutured around the circumference of the mitral annulus [1 near each commissure (#16 and #20) and 3 along the septal (#17, #18, and #19) and lateral (#15, #21, and #22) annulus (Figure 1A and 1B). Leaflet edge markers were sutured to the ventricular side of the anterior mitral leaflet (AML) and posterior mitral leaflet (PML) edges at the leaflet center (#25 and #26, Figure 1B). Markers were also sutured to the atrial side along the midlines of the AML (#23 and #24, Figure 1B) and PML (#27, Figure 1B).

A suture (2-0 polypropylene) was anchored at the right fibrous trigone (marker #21, Figure 1B and 1C) using a Teflon felt pledget, passed through the PPM tip and the posterior LV wall, and was exteriorized through a tourniquet (STRING-1). A second suture was anchored transmurally at the anterobasal LV wall (right side of the LAD, adjacent to marker #4, Figure 1A and 1C) using a Teflon felt pledget and also passed through the PPM tip and LV wall (STRING-2).

The atriotomy was closed, the heart deaired, the cross-clamp removed, and the heart defibrillated (mean cardiopulmonary bypass time 87±5 minutes, aortic cross-clamp time 52±3 minutes, dopamine 4.6±0.7 μg/kg/min). A pressure transducer (Millar SPC-500, Millar Instruments, Inc.) was placed in the LV chamber through the apex.

**Experimental Protocol**

The animals were transferred to the catheterization laboratory and studied intubated with the chest open. Anesthesia was maintained with inhalational isoflurane (1% to 2.5%). Animals received 100 mg of lidocaine (IV) as prophylaxis against rhythm disturbances. A micromanometer-tipped catheter (Millar SPC-500, Millar Instruments, Inc.) zeroed in a 37°C water bath was introduced through a left carotid sheath and advanced to the aortic arch for aortic blood pressure measurement. Simultaneous biplane videofluoroscopic marker data and hemodynamic data were acquired at baseline, during circumflex ischemia, and during ischemia after tightening of the tourniquets. Each sequence (baseline, ischemia, and ischemia+tightening) was repeated for each STRING intervention, and was designated as “preischemia” (preischemia-1, preischemia-2, or preischemia-3), “ischemia” (ischemia-1, ischemia-2, or ischemia-3), and “STRING” (STRING-1, STRING-2, or STRING-1+2), respectively. The severity of IMR was graded by an experienced cardiologist (D.L.) according to the extent and width of the color Doppler regurgitant jet visualized with transesophageal echocardiography and categorized as none (0), trace (0.5+), mild (1+), moderate (2+), moderate to severe (3+), or severe (4+). The data sequence ischemia was started at the onset of at least moderate IMR (time range, 45 to 120 s). The tension on the STRING suture was adjusted according to the observed effect on echocardiography to avoid overcorrection of papillary muscle position causing leaflet prolapse.

All of the 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. This study was approved by the Stanford University Medical School Laboratory Animal Review committee and conducted according to Stanford University policy.
LV muscle mass remains constant throughout the cardiac cycle.\textsuperscript{20} This approach is based on the premise that changes in "epicardial" LV volume are accurate measures of LV mass,\textsuperscript{9} and that LV mass can be used as a measure of LV volume.\textsuperscript{20} Therefore, changes in epicardial LV volume are directly related to changes in LV mass.\textsuperscript{20}

Opening and closing of the AML were assessed by calculating the AML excursion angle ($\theta$) defined by lines through the middle of the lateral, apical, and posterior components.\textsuperscript{16} The total excursion of the mitral annulus was quantified as the sum of the component excursions.\textsuperscript{16}

### Data Analysis

#### Cardiac Cycle Timing and Hemodynamics

For each cardiac cycle, end systole (ES) was defined as the time at which the aortic valve closed.\textsuperscript{17} End diastole (ED) was defined as the time at which the aortic valve opened.\textsuperscript{17} Cardiac cycle timing was determined from LV pressure (LVP), aortic pressure, and ECG voltage.\textsuperscript{17} An analog LV pressure (LVP), aortic pressure, and ECG voltage were recorded every 16.7 ms using custom software.\textsuperscript{17} The accuracy of 3D reconstructions from planar videoangiograms of length measurements, expressed as mean percentage error of a known marker-to-marker distance, has been shown to be 0.2% with a reproducibility of 1%.\textsuperscript{18}

#### Mitral Annular Geometry

Mitril annular area was calculated as the sum of the areas of 8 leaflet base markers defining leaflet angle. AML indicates anterior mitral leaflet; PML, posterior mitral leaflet.

#### Statistical Analysis

All of the data are reported as mean ± SEM. Hemodynamic and marker-derived data from 2 consecutive steady-state beats were time-aligned at end systole, and data from these beats were averaged for each animal and data acquisition run. Comparisons between the different conditions were made using repeated-measures ANOVA followed by Dunnett's post-hoc test.

### Results

Table 1 summarizes group mean hemodynamic data for preischemia, ischemia, and STRING for each STRING sequence (ie, 1, 2, and 1+2). During ischemia, IMR occurred, LV end-diastolic and end-systolic volumes increased significantly, whereas LV dP/dt decreased. None of the 3 different STRING-interventions affected heart rate, LV volumes, or pressures. MR, however, was reduced with STRING-1.
Chronic IMR is a common and important complication after myocardial infarction and is associated with poor prognosis.15,23–30 Annular dilatation causes ventricular remodeling and is associated with poor outcome.1,21 Despite surgical advances, IMR remains a vexing problem in cardiac surgery.22 Ring annuloplasty, the standard surgical treatment1,21 in most centers, has frustratingly variable results and is frequently associated with residual and recurrent MR in up to 30% of patients.2–6 Though undersized annular reduction can correct both annular and subvalvular geometry in IMR7, annuloplasty primarily addresses the annular dilatation, and even undersized ring

Table 2 summarizes PPM tip displacements during ischemia. As expected, septal-lateral diameter, as well as mitral annular area, increased during ischemia. Tightening STRING-1 prevented additional septal-lateral dilation and annular area increase, although progressive annular dilatation occurred with both STRING-2 and STRING-1+2.

### Discussion

The results of this study support the following conclusions: (1) basal repositioning of the displaced PPM reduces acute IMR in this ovine model; and (2) AML excursion remains unchanged with subvalvular sutures in situ.

Chronic IMR is a common and important complication after myocardial infarction and is associated with poor prognosis.1,21 Despite surgical advances, IMR remains a vexing problem in cardiac surgery.22 Ring annuloplasty, the standard surgical treatment,1,21 in most centers, has frustratingly variable results and is frequently associated with residual and recurrent MR in up to 30% of patients.2–6 Clinical and experimental studies have elucidated the pathogenesis of chronic IMR.15,23–30 Annular dilatation causes Carpentier type-I leaflet dysfunction, and in a large subset of patients, restricted leaflet motion (Carpentier type-IIIb) results from PPM displacement with associated leaflet tethering. Al-

**Figure 3.** (A) Schematic representation of the end-systolic posterior papillary muscle tip displacement with ischemia (significant shifts shown in red) and repositioning with STRING-1 (significant shifts shown in green). Data shown as mean±SEM. *P<0.05, repeated-measures ANOVA with Dunnett’s post-test vs ischemia. (B) Schematic representation of the end-systolic posterior papillary muscle tip displacement with ischemia (significant shifts shown in red) and repositioning with STRING-2. Data shown as mean±SEM. *P<0.05, repeated-measures ANOVA with Dunnett’s post-test versus ischemia. (C) Schematic representation of the end-systolic posterior papillary muscle tip displacement with ischemia (significant shifts shown in red) and repositioning with STRING-1+2 (significant shifts shown in green). Data shown as mean±SEM. *P<0.05, repeated-measures ANOVA with Dunnett’s post-test vs ischemia.

**Table 2.** End-Systolic PPM Tip Displacement and Repositioning

<table>
<thead>
<tr>
<th>Variables</th>
<th>PPM Displacement (Ischemia vs Preischemia)</th>
<th>PPM Repositioning (STRING vs Ischemia)</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral (mm)</td>
<td>5.3±0.8*</td>
<td>−0.7±0.5</td>
<td>P=0.001</td>
</tr>
<tr>
<td>Apical (mm)</td>
<td>−0.3±0.3</td>
<td>−1.7±0.4*</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Posterior (mm)</td>
<td>7.1±0.7*</td>
<td>1.9±0.2*</td>
<td>P&lt;0.001</td>
</tr>
</tbody>
</table>

Mean±SEM, repeated-measures ANOVA with Dunnett’s post-hoc test. *P<0.05 vs ischemia.

(2.0±0.1 to 1.2±0.1; P<0.05) and STRING-1+2 (2.3±0.2 to 1.3±0.2; P<0.05) but remained unchanged with STRING-2 (2.3±0.1 to 2.3±0.1).

Table 2 summarizes PPM tip displacements during ischemia, as well as the repositioning with the STRING interventions. Only STRING-1 and STRING-1+2 were associated with basal repositioning of the displaced PPM (Figure 3a–3c).

Table 3 summarizes AML leaflet motion. Ischemia reduced maximal and minimal AML excursion angles, although total leaflet excursion remained unchanged. Importantly, none of the STRING interventions affected AML motion, and both maximal and minimal AML excursion angles were preserved.

Table 4 summarizes mitral annular dynamics. As expected, septal-lateral diameter, as well as mitral annular area, increased during ischemia. Tightening STRING-1 prevented additional septal-lateral dilation and annular area increase, although progressive annular dilatation occurred with both STRING-2 and STRING-1+2.
annuloplasty fails to eliminate the component of leaflet tethering. Based on this mechanistic insight, several adjunctive techniques have been proposed. Liel-Cohen et al reported improvement of IMR with infarct plication using plicating sutures, whereas Moamie et al suggested an infarct restraint using a Marlex mesh patch. Hung et al proposed external repositioning of the displaced PM using an epicardial Dacron patch containing an inflatable balloon, which is adjusted under echo guidance. Internal PM repositioning has been evaluated in this laboratory by Timek et al using interpapillary sutures, although repositioning of the displaced PM toward the anterobasal LV resulted in reduced MR. Only this latter technique resulted in the mitral annulus aiming posterior to the right fibrous trigone. Based on this report and on the geometric distortions associated with chronic IMR reported by Tibayan et al that the distance between the midseptal fibrous annulus and the PPM tip plays a key role, we tested the hypothesis that internal direct repositioning of the PPM toward the fibrous annulus improves IMR. In the current investigation, we evaluated 2 different repositioning directions (right fibrous trigone and anterobasal LV) to identify the preferred approach for this innovative adjunct.

**Papillary Muscle Tip Position**

Consistent with previous publications for acute and chronic IMR, we observed posterolateral displacement of the PPM tip at end systole during acute posterolateral ischemia. Although repositioning of the PPM toward the anterobasal LV (STRING-2) failed to reduce IMR in this experiment, repositioning toward the right fibrous trigone with STRING-1 resulted in reduced MR. Only this latter technique resulted in basal repositioning of the displaced PPM, and combining

### TABLE 3. Anterior Mitral Leaflet Motion

<table>
<thead>
<tr>
<th>Variables</th>
<th>Preischemia</th>
<th>Ischemia</th>
<th>STRING</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Data sequence 1 (STRING-1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O AML max. ()</td>
<td>85±2*</td>
<td>79±2</td>
<td>78±2</td>
<td>P&lt;0.003</td>
</tr>
<tr>
<td>O AML min. ()</td>
<td>33±1*</td>
<td>30±1</td>
<td>31±1</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>AML excursion ()</td>
<td>51±2</td>
<td>49±1</td>
<td>46±3</td>
<td>P&lt;0.07</td>
</tr>
<tr>
<td>Data sequence 2 (STRING-2)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O AML max. ()</td>
<td>86±2*</td>
<td>80±3</td>
<td>81±3</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>O AML min. ()</td>
<td>34±2*</td>
<td>31±2</td>
<td>30±2</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>AML excursion ()</td>
<td>52±1</td>
<td>49±2</td>
<td>50±2</td>
<td>P&lt;0.2</td>
</tr>
<tr>
<td>Data sequence 3 (STRING-1+2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O AML max. ()</td>
<td>85±2*</td>
<td>81±2</td>
<td>79±2</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>O AML min. ()</td>
<td>34±2*</td>
<td>30±1</td>
<td>30±2</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>AML excursion ()</td>
<td>52±2</td>
<td>50±2</td>
<td>49±2</td>
<td>P&lt;0.1</td>
</tr>
</tbody>
</table>

Mean±SEM, repeated-measures ANOVA with Dunnett’s post-hoc test. *P<0.05 vs ischemia.

### TABLE 4. Mitral Annular Dynamic Motion

<table>
<thead>
<tr>
<th>Variables</th>
<th>Preischemia</th>
<th>Ischemia</th>
<th>STRING</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Data sequence 1 (STRING-1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Max. septal-lateral diameter (cm)</td>
<td>3.17±0.08*</td>
<td>3.56±0.07</td>
<td>3.51±0.09</td>
<td>P&lt;0.02</td>
</tr>
<tr>
<td>Min. septal-lateral diameter (cm)</td>
<td>2.48±0.08*</td>
<td>2.94±0.07</td>
<td>2.92±0.09</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Max. annular area (cm²)</td>
<td>9.82±0.40</td>
<td>11.13±0.53</td>
<td>11.21±0.54</td>
<td>P&lt;0.02</td>
</tr>
<tr>
<td>Min. annular area (cm²)</td>
<td>7.15±0.39*</td>
<td>8.90±0.46</td>
<td>9.09±0.48</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Data sequence 2 (STRING-2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Max. septal-lateral diameter (cm)</td>
<td>3.27±0.07</td>
<td>3.58±0.09</td>
<td>3.75±0.12</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Min. septal-lateral diameter (cm)</td>
<td>2.62±0.08</td>
<td>2.92±0.11</td>
<td>3.25±0.15</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Max. annular area (cm²)</td>
<td>10.43±0.50</td>
<td>11.34±0.57</td>
<td>11.90±0.75</td>
<td>P&lt;0.03</td>
</tr>
<tr>
<td>Min. annular area (cm²)</td>
<td>7.74±0.56*</td>
<td>8.89±0.62</td>
<td>10.04±0.89*</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Data sequence 3 (STRING-1+2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Max. septal-lateral diameter (cm)</td>
<td>3.32±0.08*</td>
<td>3.66±0.07</td>
<td>3.71±0.07</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Min. septal-lateral diameter (cm)</td>
<td>2.63±0.09*</td>
<td>3.03±0.08</td>
<td>3.18±0.08*</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>Max. annular area (cm²)</td>
<td>10.66±0.62*</td>
<td>11.78±0.64</td>
<td>12.06±0.59</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>min. annular area (cm²)</td>
<td>7.80±0.62*</td>
<td>9.47±0.60</td>
<td>10.15±0.60*</td>
<td>P&lt;0.001</td>
</tr>
</tbody>
</table>

Mean±SEM, repeated-measures ANOVA with Dunnett’s post-hoc test. *P<0.05 vs ischemia.
both techniques (STRING 1+2) did not result in additional improvement. Since our group reported that undersized annuloplasty may result in improved subvalvular geometry,7 the efficacy of this subvalvular technique was tested without concomitant annuloplasty in the present experiment. In a previous experiment involving sheep with posterolateral infarcts and resulting chronic IMR, we demonstrated that the Kron technique does not reduce IMR in the absence of concomitant annular reduction.39 We hypothesize, however, that a different repositioning direction with a suture between right fibrous trigone and PPM tip, which reduced acute IMR in the present experiment without annular reduction, could be even more effective in the clinical setting when combined adjunctively with ring annuloplasty, as proposed by Kron et al.14 Also, the right fibrous trigone is a safe location for the repair suture, because a suture placed at the midseptal annular saddle horn could result in severe aortic regurgitation if the adjacent noncoronary aortic cusp is inadvertently involved.

**Leaflet Excursion**

A major concern with the use of subvalvular repair sutures is impaired leaflet excursion. With the repair sutures tightened, we did not observe decreased excursion of the AML. Furthermore, we postulate that the presence of a subvalvular repair suture could help prevent LV outflow tract obstruction by a systolic anterior movement of the AML. Although the incidence of systolic anterior movement after mitral valve repair for IMR is rare in general, it might occur with profound downsizing of the mitral annulus in the presence of a hypovolemic and hypercontractile hemodynamic condition.

**Mitral Annular Dynamics**

Annular dilatation plays a key role in the pathogenesis of IMR.27,30,39 As expected, increase of septal-lateral diameter, as well as mitral annular area during acute posterolateral ischemia, were observed in this experiment. Interestingly, septal-lateral diameter and annular area did not increase additionally with STRING-1 tightened, although progressive annular dilatation was observed with STRING-2 and STRING-1+2. Because the concept of a STRING repair suture is intended as an adjunctive repair technique in conjunction with ring annuloplasty, the clinical relevance of the observed annular dynamics is limited.

**Summary**

In this study of the 3D distortions of the mitral valve apparatus associated with acute ischemia, direct internal PPM repositioning toward the right fibrous trigone resulted in improvement of IMR. Of interest, excursion of the AML remained unchanged with the repair sutures tightened. As originally proposed by Kron et al.,14 this adjunctive surgical reparative technique might help prevent residual or recurrent MR after mitral valve annuloplasty repair. The annuloplasty addresses the annular component of IMR, whereas the adjunctive technique corrects the subvalvular changes. In patients with confirmed Carpentier type IIib leaflet motion on preoperative and intraoperative echocardiographic assessment of the mechanism of MR, our experimental findings may well be translated into clinical practice with the repair suture connecting the right fibrous trigone and the PPM tip placed at the time of the annuloplasty and tied loosely. After weaning from cardiopulmonary bypass, the repair result will be evaluated by intraoperative transesophageal echocardiographic guidance. If residual MR is observed after annuloplasty, the tension on the repair suture is adjusted under echocardiographic guidance with the final knots tied to eliminate or reduce the residual MR. If no residual MR occurs after annuloplasty, the suture is tied with minimal tension, and this suture will be equivalent to a tertiary chord that might prevent recurrent MR postrepair, because it limits additional PPM displacement in the continued LV remodeling process.3

**Study Limitations**

The investigation of acute IMR in open-chest sheep who previously had normal hearts is far different from the clinical scenario of chronic IMR. Also, distinct differences in cardiac anatomy, including annular, leaflet, PM, and coronary anatomy between sheep and humans, are well known.24,25 Thus, direct extrapolation of the present experimental findings is inappropriate. Proximal circumflex artery occlusion resulted in both annular dilatation and PPM displacement, which mimics 2 pathophysiologic causal components of chronic IMR. Nevertheless, additional studies in chronic IMR animal models or human subjects are needed to investigate this new adjunctive suture repair method when used with ring annuloplasty.

The tension on the STRING sutures was neither predetermined in terms of desired repositioning distance nor measured by a force transducer. The tension on the tourniquet was adjusted according to the echocardiographic effect. Although we cannot speculate on the durability of this type of repair in the clinical setting, placement of artificial chordae using polytetrafluoroethylene sutures in mitral valve repair is associated with encouraging midterm results.40 In fact, with the use of polytetrafluoroethylene sutures, a repair suture connecting the right fibrous trigone and PPM tip (like our STRING-1) can be considered as an artificial tertiary chord, which may help prevent recurrent IMR as result of continued LV remodeling.3

**Acknowledgments**

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