Reduced Systolic Torsion in Chronic “Pure” Mitral Regurgitation

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Background—Global left ventricular (LV) torsion declines with chronic ischemic mitral regurgitation (MR), which may accelerate the LV remodeling spiral toward global cardiomyopathy; however, it has not been definitively established whether this torsional decline is attributable to the infarct, the MR, or their combined effect. We tested the hypothesis that chronic “pure” MR alone reduces global LV torsion.

Methods and Results—Chronic “pure” MR was created in 13 sheep by surgically punching a 3.5- to 4.8-mm hole (HOLE) in the mitral valve posterior leaflet. Nine control (CNTL) sheep were operated on concurrently. At 1 (WK-01) and 12 weeks (WK-12) postoperatively, the 4D motion of implanted radiopaque markers was used to calculate global LV torsion. MR-grade in HOLE was greater than CNTL at WK-01 and WK-12 (2.5±1.1 versus 0.6±0.5, P<0.001 at WK-12). HOLE LV mass index was larger at WK-12 compared with CNTL (195±14 versus 170±17 g/m², P<0.01), indicating LV remodeling. Global LV systolic torsion decreased in HOLE from WK-01 to WK-12 (4.1±2.8° versus 1.7±1.7°, P<0.001), but did not change in CNTL (5.5±1.8° versus 4.2±2.4°, P=NS). Global LV torsion was lower in HOLE relative to CNTL at WK-12 (P<0.05) but not at WK-01 (P=NS).

Conclusions—Twelve weeks of chronic “pure” MR resulting in mild global LV remodeling is associated with significantly increased LV mass index and reduced global LV systolic torsion, but no other significant changes in hemodynamics. MR alone is a major component of torsional deterioration in “pure” MR and may be an important factor in chronic ischemic mitral regurgitation. (Circ Cardiovasc Imaging. 2009;2:85-92.)

Key Words: mechanics ■ mitral valve ■ myocardium ■ ventricles ■ torsion

Chronic ischemic mitral regurgitation (CIMR) remains one of the most challenging life-threatening clinical problems in cardiac surgery, affects a large number of patients, leads to congestive heart failure which limits life expectancy and functional capacity, and has major adverse implications on U.S. health care costs.¹

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In CIMR, the mitral valve leaks, yet the leaflets appear normal. The mitral regurgitation (MR) associated with CIMR is the result of left ventricular (LV) geometric and micro-structural remodeling that leads to alterations in the geometry of the mitral valve, annulus, and subvalvular apparatus. These alterations include annular dilation, which reduces leaflet coaptation, and papillary muscle displacement, which leads to leaflet tethering.

Uncorrected CIMR is associated with decreased LV torsion.² LV torsion in the normal heart reflects a balance between epicardial and endocardial function and helps to produce a nearly homogeneous transmural distribution of myocardial stress and fiber strain during ejection.³ An impairment of LV torsion may increase transmural differences in stress and foment deleterious LV remodeling or reduce the elastic energy stored in the myocardium, thereby impairing filling.³ In this way decreases in LV torsion as seen in CIMR may contribute to a vicious cycle wherein “MR begets MR.”⁶ It has not been definitively established, however, whether the reduced LV torsion in CIMR is a direct result of the infarct, the MR, or a combination of the 2.

With respect to the treatment options for patients with CIMR, sorting out the impact of MR from that of ischemia on ventricular function may have important clinical consequences. For example, the surgical treatment options for patients with CIMR include coronary artery bypass grafting (CABG) or mitral valve annuloplasty or replacement. Currently, the most common technique to restore valve competence is the placement of an undersized annuloplasty ring. There exists, however, an ongoing debate about whether or not mitral valve annuloplasty at the time of CABG improves outcomes over and above CABG alone.⁷-⁹

Hypothetically, if the reduction in LV torsion could be attributed to ischemia alone, then CABG might be sufficient...
to either restore or stop the deterioration of LV torsion. If, however, MR alone contributes to impaired LV torsion, mitral valve repair may be important for minimizing the deterioration in LV torsion.

The goal of this study was to sort out the impact of MR alone on LV torsion in an ovine model of chronic “pure” MR. We tested the hypothesis that chronic “pure” MR alone reduces global LV torsion.

Methods

Animal Welfare
All animal procedures were approved by our Institutional Animal Care and Use Committee and followed guidelines set forth by the National Institutes of Health. Results from some of the animals used in this report were described previously. The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Adult male sheep (Dorsett-hybrid) were premedicated with an injection of ketamine, anesthetized, intubated, mechanically ventilated, and sedation was maintained with inhalational isoflurane. Blood pressure, body temperature, blood gases (CO₂ and O₂), and cardiac output were monitored throughout the study.

Surgery
A left lateral thoracotomy was performed under surgically sterile conditions to expose the heart for the implantation of miniature radiopaque markers. Thirteen LV markers were placed subepicardially to silhouette the LV chamber. One marker was placed at the apex and 4 each were placed in the septal, anterior, lateral, and posterior walls at apical, midequatorial, and basal levels (Figure 1). These markers were used to generate the results reported herein. To investigate changes in regional function additional beads were implanted transmurally in the antero-basal and lateral-equatorial LV wall. The animal was then placed on cardiopulmonary bypass, the heart arrested, and the left atrium opened to provide access to the mitral valve annulus, leaflets, and subvalvular apparatus. To investigate changes in annular and leaflet function, markers were sewn to the mitral valve annulus, leaflets, and on the papillary muscle tips.

Animals were randomized into a control group (CNTL, n=8) and a chronic “pure” mitral regurgitation (PMR) group (HOLE, n=12) while on bypass. In HOLE animals, a 3.5- to 4.8-mm hole was created in the posterior leaflet using an aortic hole punch to generate “pure” mitral regurgitation. The animals were then weaned off bypass. A Konigsberg micromanometer pressure transducer was placed in the LV chamber through the apex and exteriorized. The chest was closed in layers, and the animal was recovered and returned to the holding facility. Throughout the whole study period the animals were followed for clinical signs of heart failure (tachypnea, lethargy, and anorexia). Body weight and transthoracic echocardiographic MR-grade were also monitored.

Data Collection—Catheterization Laboratory
At 1 (WK-01) and 12 (WK-12) weeks all animals underwent examination in the catheterization laboratory. As before, the animals were premedicated with ketamine, anesthetized, intubated, mechanically ventilated, and sedation was maintained with inhalational isoflurane. LV pressure (LVP) was recorded using the implanted Konigsberg pressure transducer and aortic pressure (AoP) was recorded using a micromanometer catheter (Millar Instruments) introduced via the left carotid artery. Pressure and ECG data were recorded at 240 Hz and synchronized with the recording of the x-ray images. MR was graded qualitatively (0 to 4+) by an expert echocardiographer (D.L.) on the basis of transesophageal echocardiography color doppler regurgitant jet extent and width.

Marker motion was recorded during normal sinus rhythm with controlled apnea using biplane cine fluoroscopy at 60 frames/s. After WK-01 data collection the animals were recovered and returned to animal care facility. After the WK-12 study the animals were euthanized with a bolus infusion of potassium chloride (80 mEq).

Data Analysis
The animals selected for this study were required to have all LV markers in place, regardless of the successful placement of the other markers. During 3 consecutive heartbeats, each markers’ image coordinates were digitized using custom computer aided detection software and merged to reconstruct 3D marker motion. The 3D LV marker coordinates were used to calculate the volume of space-filling tetrahedra that approximate the total LV volume during the cardiac cycle. The change in LV volume calculated from subepicardial markers is an accurate measurement of the change in LV chamber volume, despite the inclusion of the LV wall volume. Sphericity index (SI) was calculated as the ratio of the length of the LV long axis and the LV diameter provided by Fischer et al. End diastolic (ED), end isovolumic contraction (IVC), end systolic (ES), and end isovolumic relaxation (IVR) cardiac cycle time points were determined semiautomatically from plots of LV pressure-volume loops. The body surface area was then used to calculate indexed values of the LV end-diastolic volume index (LVEDVI), LV end-systolic volume index (LVESVI), and LV mass index (LVMI). Peak systolic
torsion-volume curve during ejection for each group was computed. Significant regional differences (eg, anterior versus posterior wall) were not evident (data not shown). Note, that we did not observe a significant alteration in global LV torsion during any phase of the cardiac cycle. WK-01 versus WK-12, indicating that 1 week of PMR did not result in a significant decrease in the magnitude of IVR recoil (1.5° versus 1.8°, P<0.01). There were no significant differences within CNTL or within HOLE between WK-01 and WK-12. Therefore, MR grade was elevated throughout the experiment in the HOLE group and was typically moderate-severe to severe, whereas the CNTL group had trace MR on average throughout the experiment. Aortic cross-clamp times for the HOLE and CNTL groups were significantly different (34±3 and 29±3 minutes, P<0.01). Creation MR in the HOLE group accounts for the time difference. LVMI at WK-12 (the only time point available) was significantly increased in HOLE compared with CNTL (195±14 versus 170±17 g, P<0.01). There were, however, no significant differences between groups or over time for heart rate, body weight, LV end-diastolic volume index, LV end-diastolic pressure, LV end-systolic volume index, peak systolic LV pressure (LVPmax), or sphericity index. LV end-diastolic pressure, however, was decreased from WK-01 to WK-12 in both CNTL and HOLE.

Global LV Torsion—No Week-01 Differences
A summary of the phases of global LV torsion is found in Table 2 and graphically depicted in Figure 2. There were no significant differences between CNTL and HOLE groups at WK-01, indicating that 1 week of PMR did not result in a significant alteration in global LV torsion during any phase of the cardiac cycle.

Global LV Torsion—Similar Week-12 Changes
Twelve weeks after surgery there were significant differences within the CNTL group (WK-01 versus WK-12) for torsion during IVC and IVR. LV cocking during IVC was restored from WK-01 to WK-12 in the CNTL group (0.6±0.9° versus −1.3±1.8°, P<0.01) and torsion recoil during IVR nearly abolished (−1.7±1.5° versus 0.4±1.6°, P<0.01). Similarly, at WK-12 the HOLE group demonstrated a restoration from WK-01 of LV cocking during IVC (0.4±1.4° versus −1.8±1.5°, P<0.01) and a significant decrease in the magnitude of IVR recoil (−1.8±1.2° versus 0.0±1.8°, P<0.01). These changes in the HOLE group paralleled those of the CNTL group, thus are likely associated with recovery from surgery.
Figure 2. LV torsion for CNTL and chronic “pure” MR (HOLE) group at 1 (WK-01) and 12 (WK-12) weeks postoperatively during 5 phases of the cardiac cycle. Notably, global LV torsion during ejection, filling, and systole (ED to ES) all demonstrated significant changes between HOLE and CNTL groups at WK-12. Furthermore, LV systolic torsion was reduced at WK-12 in HOLE compared with HOLE at WK-01. Significant differences both within CNTL and HOLE during IVC and IVR were also observed. ED-ES, systolic torsion from end-diastole to end-systole. All measures are degrees. *P < 0.01 within group; †P < 0.05 between groups; ‡P < 0.01 between groups.

Global LV Torsion—Differential Week-12 Changes
Global LV torsion during ejection, filling, and systole (ED to ES) all demonstrated significant changes between HOLE and CNTL groups at WK-12. Global ventricular torsion during ejection was decreased for HOLE at WK-12 compared with CNTL at WK-12 (3.4°±1.4° versus 5.5°±2.7°, P < 0.05). Similarly, global ventricular torsion during filling was decreased in HOLE at WK-12 relative to CNTL at WK-12 (−1.7°±2.1° versus −4.5°±2.3°, P < 0.01). These results indicated impaired torsion during ejection and decreased untwisting during filling for HOLE relative to CNTL at WK-12.

LV systolic (ED to ES) torsion was reduced at WK-12 in HOLE compared with both HOLE at WK-01 (1.7°±1.7° versus 4.1°±2.8°, P < 0.01) and CNTL at WK-12 (1.7°±1.7° versus 4.2°±2.7°, P < 0.05). Thus, LV systolic torsion was impaired after 12 weeks of chronic “pure” MR.

Rates of Global LV Torsion—No Week-01 Changes
A summary of the rates of global LV torsion during IVC, ejection, IVR, and filling is shown in Table 3 and in Figure 3. There were no significant differences in any cardiac phase component of the rate of global ventricular torsion between the CNTL and HOLE groups at WK-01, indicating that 1 week of PMR did not have a deleterious impact.

Rates of Global LV Torsion—Similar Week-12 Changes
The rate of ventricular torsion during IVC, however, significantly decreases and changes sign from WK-01 to WK-12 in both CNTL (9.8°±12.4°/s versus −14.5°±17.5°/s, P < 0.01) and HOLE (5.4°±17.0°/s versus −17.2°±17.6°/s, P < 0.01) groups. Furthermore, the rate of ventricular torsion during IVR significantly increased from WK-01 to WK-12 in both the CNTL (−16.3°±11.3°/s versus 3.5°±13.7°/s, P < 0.01) and HOLE (−17.9°±11.1°/s versus −2.1°±18.4°/s, P < 0.01) groups. These similar changes in the rates of global LV torsion may reflect ventricular changes associated with postoperative recovery.

Rates of Global LV Torsion—Differential Week-12 Changes
The least squares estimate of the slope of the torsion-volume curve during ejection for each group is compared in Table 4. The path of the torsion-volume curve during ejection was very linear. At WK-01 there was no difference between the CNTL and HOLE groups (−0.35°±0.12°/mL versus −0.29°±0.18°/mL). At WK-12, however, the HOLE group slope was significantly different from both CNTL at WK-12 (−0.18°±0.06°/mL versus −0.32°±0.10°/mL, P < 0.05) and HOLE at WK-01 (−0.18°±0.06°/mL versus −0.29±0.18°/mL, P < 0.05).
during IVR was nearly zero. The magnitude of torsion at the end of IVR was higher in the CNTL group than the HOLE group and the CNTL group exhibited greater negative torsion during the subsequent filling phase.

**Discussion**

**Depressed Ventricular Torsion**

Twelve weeks of chronic PMR resulted in significant reductions in torsion during systole (ED to ES), ejection (end-IVC to ES), and during filling (end-IVC to ED) relative to CNTL. Only decreases in systolic torsion, however, were significantly different in HOLE at WK-12 compared with both HOLE at WK-01 and CNTL at WK-12. The small difference in aortic cross-clamp time, though statistically significant, is not likely to induce differences in ventricular torsion between the 2 groups.

Changes in systolic torsion must be understood in the context of the restoration of ventricular cocking during IVC. Cocking, an expected mode of torsion during IVC, is restored in both CNTL and HOLE at WK-12. The notable change in systolic torsion in HOLE at WK-12 is primarily attributable to a persistent reduction in torsion during ejection, combined with increased cocking during IVC.

To account for changes associated with the recovery from the operation we focused on measures of torsion during ejection—a phase of the cardiac cycle during which the CNTL group undergoes no significant change from WK-01 to WK-12. Ejection torsion in the HOLE group is significantly reduced at WK-12 compared with CNTL but is not statistically different between WK-01 and WK-12 within HOLE, indicating that it is not further depressed after 12 weeks of PMR.

Furthermore, during ejection the slope of the torsion-volume curve is not different within the CNTL group, or between CNTL and HOLE at WK-01. All slopes are approximately $-0.32/\text{mL}$, indicating that this relationship is preserved during acute PMR and in the postoperative recovery period. This measure, however, is significantly decreased in the HOLE group at WK-12 ($-0.18/\text{mL}$) compared with both CNTL at WK-12 and HOLE at WK-01. This significant difference within the HOLE group may indicate that the torsion-volume relationship continues to decline with persistent PMR.

**Physiologic Adaptation to Pure MR**

The physiological adaptation to chronic “pure” MR primarily included a significant increase in LVMI. LVEDVI and LVESVI were both increased 15% in HOLE compared with CNTL at WK-12, but the results were not statistically significant. The LV changes after 12 weeks of pure MR in HOLE substantially differ from the larger LVMI, LVEDVI, and LVESVI changes that are known to occur in patients with longstanding chronic MR attributable, at least in part, to the duration of the disease process. Our measures of LVEDVI and LVESVI are derived from subepicardial markers, which results in larger measures when compared with endocardial-derived values as they include changes in myocardial mass. Furthermore, though sphericity index in HOLE tended to be lower (more spherical), no significant differences between CNTL and HOLE were observed. The results derived from endocardial measures may demonstrate significant differences if there are significant differences in wall thickness between CNTL and HOLE. One strength of this study and previous work is that it reflects a very early stage of the

Table 4. Slope of the Torsion vs LV Volume Curve During Ejection for Control and Chronic “Pure” Mitral Regurgitation Animals at 1 and 12 Weeks Postoperatively

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<th>WK-01</th>
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<td></td>
<td>CNTL, n=9</td>
<td>HOLE, n=13</td>
<td>CNTL, n=9</td>
<td>HOLE, n=13</td>
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<tr>
<td>Torsion-volume ejection slope, degrees/mL</td>
<td>$-0.35\pm0.12$</td>
<td>$-0.29\pm0.18$</td>
<td>$-0.32\pm0.10$</td>
<td>$-0.18\pm0.06^*$</td>
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*P<0.05 within group; and $^*P<0.05$ between groups.
disease process, shown by only mild signs of LV remodeling (LVMI), before overt changes in traditionally measured clinical indices.

The decreased global systolic torsion may be attributed to the increase in LV end-diastolic volume index and the significant increase in LV myocardial mass index. Because our results are definitive with regards to changes in systolic torsion and equivocal in regards to changes in LVEDVI, it may be the case that ventricular torsion or the torsion-volume slope during ejection is a more sensitive measure of LV dysfunction and remodeling using the experimental conditions and quantitative methods reported herein.

Although parameters like LVEDVI and LVESVI trend upward toward significant increases in the HOLE group, power calculations demonstrate that a group size of 44 and 38, respectively, would be needed to attain a power of 0.9 for an alpha of 0.05. In comparison, the systolic torsion results have a power of 0.96.

**Postoperative Recovery**

Changes in global LV torsion and rates of torsion reflect a combination of the deleterious effects of chronic PMR and the recovery from the operation. A primary advantage of this study, compared with previous work,21 is the inclusion of a sham operated control group. Changes in torsion during IVC and IVR are very similar between the CNTL and HOLE groups, and such changes may be attributed to recovery from the surgical procedure between WK-01 and WK-12.

Tibayan et al,22 in a model of dilated cardiomyopathy that also required the use of bypass, observed an increase in the magnitude of early systolic cocking from −0.7° in animals 1-week postsurgery to −1.5° after 15-days of rapid pacing (−0.5° versus −1.2°). Our results are similar, suggesting that this change in torsion may be largely attributable to postoperative recovery time and not dilated cardiomyopathy or chronic mitral regurgitation. The surgical intervention, which includes cardiopulmonary bypass and cardiac arrest, may, however, alter the measured torsion as compared with animals that forego this procedure, but still have epicardial makers implanted. Additional work is needed to compare 2 such groups.

**Alternate Mechanism of Reduced Torsion Dynamics**

Global LV torsion can be decreased by alterations in electric conduction patterns. In normal myocardium the endocardium is electrically and mechanically activated before the epicardium.23 It is understood that early activation of the endocardium may cause LV cocking during IVC.24,25 If the pattern of electric activation was altered and endocardial activation was made more coincident with epicardial activation then early LV cocking would be reduced. It is possible that this is the mechanism underlying the loss of LV cocking in both CNTL and HOLE at WK-01 during the early postoperative period, but further study is needed to confirm this. If this were the case then it would seem that normal electric conduction patterns were restored at WK-12 in both CNTL and HOLE, and it would be difficult to ascribe such alterations to the MR alone.

Another possible cause of reduced ventricular torsion in chronic “pure” MR is heterogeneous transmural remodeling. It is possible that at WK-12 the HOLE animals have undergone eccentric hypertrophy and maintained the radius to wall-thickness ratio, but nonuniformly redistributed myocardial mass. If the epicardial fibers atrophy, while endocardial fibers hypertrophy, wall thickness could be maintained or increased, but ventricular torsion would decrease as the balance between epicardial and endocardial force generation remodeled. Investigations into the heterogeneity of transmural remodeling are ongoing. Also, it should be noted that reduced torsion during ejection may also arise from paradoxical septal wall motion as a result of cardiopulmonary bypass.26 Lastly, changes in torsion and torsion rates during IVR and filling may not be attributed to the MR per se, but rather to the effects of ventricular remodeling that deleteriously impacts torsion during diastole.

Each of these alternative mechanisms of reduced global ventricular torsion warrants further investigation.

**Comparison With Previous Results**

Caution must be used when comparing observations of ventricular rotation and ventricular torsion. Furthermore, it is imperative to account for the definitions used by each author to avoid confusion in interpreting and comparing results.

Tibayan et al21 demonstrated in a different model of nonischemic chronic mitral regurgitation (Cope biopsy needle disruption of the leading edge of the posterior leaflet) that maximum torsional deformation decreased (6.3° to 4.7°), and torsion during early diastolic recoil (first 5% of filling) decreased in magnitude (−3.8° to −1.5°) from 1-week to
7-weeks. Though the methods of chronic MR creation differ, the salient aspects of the experiments are very similar. Most importantly the MR-grade (“moderate to severe”) and duration were similar. The presence of a sham operated control group in our study, however, allowed us to control for the impact of MR alone.

Another study by Tibayan et al., in a sheep model of inferior myocardial infarction (MI), describes the differences in ventricular torsion for animals that developed chronic ischemic MR (CIMR) compared with those that did not show significant MR. In the CIMR animals ventricular torsion was significantly decreased in the posterior wall 7 weeks after infarction, but the decrease in torsion in the non-MR infarct group was not large enough to be statistically significant. Hence, the decrease in LV torsion is greater in CIMR than in MI alone. This indicates that MR is an important determinant of decreased ventricular torsion. Our study specifically isolates the effects of MR alone and shows that ventricular torsion is significantly decreased. Therefore, decreases in ventricular torsion in CIMR or in “pure” MR are largely attributable to the MR alone.

Limitations

This model provides a unique way to study the isolated effects of “pure” MR without confounding factors such as ischemia, infarction, disruption of subvalvular apparatus (chordae tendinae or papillary muscles), or disruption of the leaflets along the line of coaptation.

The invasiveness of the surgical procedure may acutely and chronically alter ventricular function. Although the implanted myocardial marker technique affords certain advantages (high spatial and temporal resolution and the ability to track precise tissue locations over long experimental durations) it also requires a thoracotomy and disruption of the pericardium. The presence of a sham operated control groups provides a way to account for the impact of surgery and bypass. Although the only experimental difference between the CNTL and HOLE groups is the surgical creation of the mitral valve leaflet hole in the HOLE group, the study is not without other confounding factors. In fact, parallel changes in the CNTL and HOLE group over the course of 12 weeks indicate that the effects of the operation on the ventricle are still present at WK-01. Furthermore, the measures made with subepicardially implanted markers in this study are precise but do not afford the ability to quantify transmural differences in the myocardial kinematics. Therefore, with the current technique we are also unable to report on regional septal dysfunction that may result from the bypass procedure. Interestingly, MRI may not be able to capture the motion of early systolic events attributable to ECG gating and tagging pulse delays, and its lower temporal resolution compared with echocardiography unless specialized pulse sequences are used.

Conclusions

Twelve weeks of chronic “pure” MR resulted in significantly reduced global LV torsion during systole, ejection, and filling. The decreases in global torsion may be attributed to subtle increases in LVEDVI and LVMi but may also be attributable to ventricular microstructural remodeling. These data suggest that in patients with CIMR the MR itself may promote deterioration of LV torsion. MV repair in patients with CIMR concomitant with CABG may therefore help to slow down, stop, or even restore physiological LV torsion.

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Disclosures

None.

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