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# Regional Myocardial Strain Before and After Mitral Valve Repair for Severe Mitral Regurgitation

Rekha Mankad,[1] Charles J. McCreery,[1]
Walter J. Rogers Jr.,[1] Robert J. Weichmann,[2]
Edward B. Savage,[2] Nathaniel Reichek,[1]
and Christopher M. Kramer[3]

[1]*Department of Medicine and* [2]*Department of Surgery, Allegheny General Hospital, Pittsburgh, Pennsylvania*
[3]*Departments of Radiology and Medicine, University of Virginia Health System, Charlottesville, Virginia*

## ABSTRACT

*Magnetic resonance tagging (MRI) can be used to study intramyocardial trains in human in vivo. We wished to determine whether patients with severe mitral regurgitation demonstrate subtle myocardial contractile dysfunction despite normal left ventricular (LV) ejection fraction (EF) and how mitral valve repair (MVR) may preserve EF in such patients. MRI was performed on seven patients with severe mitral regurgitation (mean age ± SD, 65 ± 13 years) and normal EF day 1 (range, 0–8 days) before (Pre) and week 8 ± 3 after (Post) MVR and on nine normal volunteers (mean age, 32 ± 4). LV mass index (LVMI), end-diastolic and end systolic volume, mass/volume ratio, EF, and sphericity index were measured Pre and Post. Two-dimensional strain analysis of MR tagged images was performed and expressed as L1 (greatest systolic lengthening, radial in normal subjects), L2 (greatest systolic shortening, circumferential in normals), and β (angular deviation of L1 from the radial direction). LVMI fell from 142 ± 38 g/m² Pre to 117 ± 44 g/m²* Post ($p \leq 0.008$) *as did LV end-diastolic volume (117 ± 26 to 69 ± 12 ml,* $p \leq$

Address correspondence and reprint requests to Christopher M. Kramer.

*0.003), whereas EF remained unchanged (59 ± 7% at both time points). LV mass/volume ratio increased from 2.2 ± 0.3 g/ml Pre to 3.1 ± 0.4 g/ml Post* ($p \leq 0.02$) *and sphericity index fell from 0.86 ± 0.10 to 0.71 ± 0.13* ($p = 0.02$). *In the short axis, L1 was greater in patients with mitral regurgitation than normal subjects (19 ± 9% vs 16 ± 6%,* $p \leq 0.003$) *and tended to increase further after MVR (21 ± 8%,* $p \leq 0.06$ *vs. Pre).* $\beta$ *was abnormal in mitral regurgitation (19 ± 8 vs. 12 ± 8 degrees in control subjects,* $p < 0.0001$) *and remained abnormal after MVR (19 ± 9 degrees). L2 in the short axis was depressed in mitral regurgitation compared with control subjects (12 ± 6% vs. 21 ± 6%,* $p \leq 0.001$) *and was further depressed after MVR (9 ± 7%,* $p < 0.001$ *vs. Pre). As detected by MRI, regional myocardial strains are abnormal in severe mitral regurgitation despite normal EF, characterized by increased short-axis systolic lengthening that is abnormally directed and by reduced shortening. After MVR, the further increase in short-axis lengthening may preserve EF despite its abnormal direction and a fall in shortening. The increase in short-axis lengthening may be due in part to the reduction in LV sphericity after MVR.*

***Key Words:*** *Magnetic resonance imaging; Mechanics; Mitral valve; Regurgitation; Surgery*

## INTRODUCTION

Patients with chronic mitral regurgitation may demonstrate myocardial contractile dysfunction, which may be irreversible, despite normal ejection fraction (EF) (1,2). Patients with more manifest evidence of impaired left ventricular (LV) performance at the time of mitral valve surgery do substantially worse postoperatively, with greater declines in EF (3–5). Conventional mitral valve replacement can result in a significant fall in LV EF, whereas mitral valve repair (MVR) does not (3,6–9). Investigators have suggested that MVR preserves LV systolic function by maintaining more ellipsoid chamber geometry and allowing a greater reduction in end-systolic wall stress (10).

Global myocardial mechanics of mitral regurgitation and the effects of MVR have been studied in animal models (11–13), but the study of regional mechanics has been limited to implanted sonomicrometry crystals in the region of the papillary muscle insertions (14,15). Regional intramyocardial strains in patients with mitral regurgitation have not been studied to date, in part due to limitations in imaging techniques. Regional myocardial deformation can be assessed noninvasively by magnetic resonance myocardial tagging (MRI) (16–18). We wished to use MRI to examine regional LV strains in patients with severe chronic mitral regurgitation and to compare them with those of normal volunteers and with the postrepair state.

## MATERIALS AND METHODS

### Patient Selection

Seven symptomatic patients (four women and three men aged 65 ± 13 years) undergoing MVR for isolated chronic severe mitral regurgitation at Allegheny General Hospital were studied (Table 1). All had symptoms of dyspnea and decreased exercise tolerance with mean symptom duration of 6 ± 4 months. The study was approved by the Institutional Review Committee and was in accordance with institutional guidelines. All gave informed consent. All patients underwent coronary angiography, left ventriculography, and intraoperative transesophageal echocardiography. Severe mitral regurgitation and normal EF were documented by the latter two methods. The etiology of the mitral regurgitation was myxomatous degeneration with prolapse or partially flail leaflets. Patients were excluded from the study if they had significant obstructive coronary artery disease (>50% stenosis), other significant valvular disease, mitral stenosis, or atrial or ventricular arrhythmias. All patients were in normal sinus rhythm.

All patients underwent MVR with annuloplasty rings (either Duran or Cosgrove-Edwards rings) (Table 1). The type of repair performed was based on the surgeon's assessment of the mitral valve apparatus. Five of the seven had a portion of the posterior leaflet resected, one had a partial anterior leaflet resection, and one received a ring

***Table 1***

*Patient Characteristics*

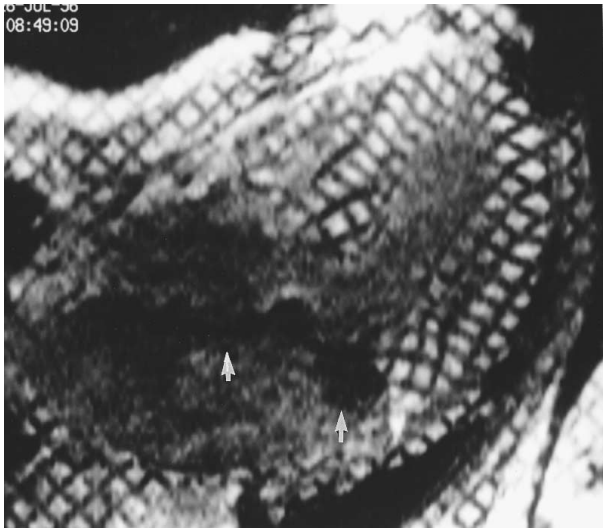
| Patient | Age | Sex | Symptom Duration (mo) | Ring Type | HR Pre | BP Pre | HR Post | BP Post |
|---|---|---|---|---|---|---|---|---|
| 1 | 68 | F | 1 | Duran | 88 | 150/75 | 80 | 138/80 |
| 2 | 75 | F | 3 | C.E. | 80 | 145/80 | 73 | 134/82 |
| 3 | 75 | F | 10 | C.E. | 78 | 104/60 | 70 | 150/70 |
| 4 | 38 | F | 6 | C.E. | 3 | 117/60 | 89 | 118/80 |
| 5 | 72 | M | 12 | C.E. | 80 | 140/80 | 80 | 152/80 |
| 6 | 56 | M | 8 | Duran | 60 | 145/70 | 70 | 124/82 |
| 7 | 68 | M | 1 | C.E. | 80 | 135/75 | 76 | 138/90 |

C.E., Cosgrove-Edwards ring; HR; heart rate; BP, blood pressure.

alone. One patient underwent surgery using the Heart-Port technique (© Heart-Port Inc., Redwood City, CA). Nine normal human volunteers with no historical or echocardiographic evidence of heart disease, aged 32 ± 4, were also studied.

## MRI

All patients underwent MRI at a mean of 1 day (range, 0–8 days) before (Pre) and week 8 ± 3 after (Post) surgery. All normal volunteers were imaged once with the same protocol. MRI was performed on a Siemens (Erlangen, Germany) 1.5-T scanner. Two patients and all control subjects were imaged prone on an elliptical spine surface coil. Due to the availability of a phased-array body coil, the last five patients were imaged in the supine position. Each patient was imaged in the same position at Pre and Post. Continuous electrocardiographic monitoring and gating were performed. Localizing scout images were followed by a single short-axis cine series in a plane near the mitral valve,

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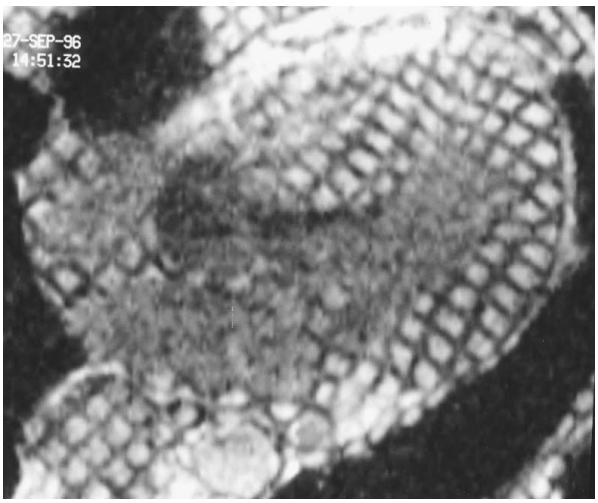


B

**Figure 1.** (A) End-systolic, long-axis, breathhold, gradient echo MR tagged image in a patient before MVR. Myocardial deformation within the septum, apex, and lateral walls is demonstrated in this view. The signal void (black region) beginning behind the posterior mitral annulus (right arrow) is the turbulent jet of severe mitral regurgitation that heads anteriorly, behind the anterior leaflet and along the atrial septum (left arrow). (B) End-systolic image in the same patient 8 weeks after MVR in the equivalent location and orientation as in A. The mitral regurgitation jet is no longer seen.

which provided images to identify end-systole as the point of minimal left ventricle cavity area.

A series of short-axis, single-slice, multiple cardiac phase, tagged images were then obtained using a breathhold gradient echo method with a segmented k-space acquisition (19,20). Tag stripe separation was 7 mm, field of view 280 mm, matrix size 128 × 256, with a pixel size of 1.09 × 2.19 mm. Interpolation was performed to 256 × 256 for display. Repetition time, which determined the temporal resolution, was adjusted between 35 and 60 msec so as to time one image of the image series at end-systole. Each breathhold spanned 18 heartbeats. Seven-mm-thick contiguous image planes or slices were generated to span the entire left ventricle from base to apex. Long-axis images in the four-chamber and two-chamber planes were also obtained (Fig. 1). The entire imaging session lasted about 45 minutes. The identical imaging protocol was performed 8 weeks after MVR.

## Data Analysis

Images were analyzed using a software package (SPAMMVU, © University of Pennsylvania) (18,21,22) loaded on a Silicon Graphics Indigo workstation. LV mass (LVM) was calculated from manually planimetered epicardial and endocardial areas of interleaved short-axis end-diastolic images using previously published techniques (19,20) and then was indexed to body surface area (LVM index). LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), and EF were also calculated using planimetered end-diastolic and end-systolic areas as previously described (20). A sphericity index

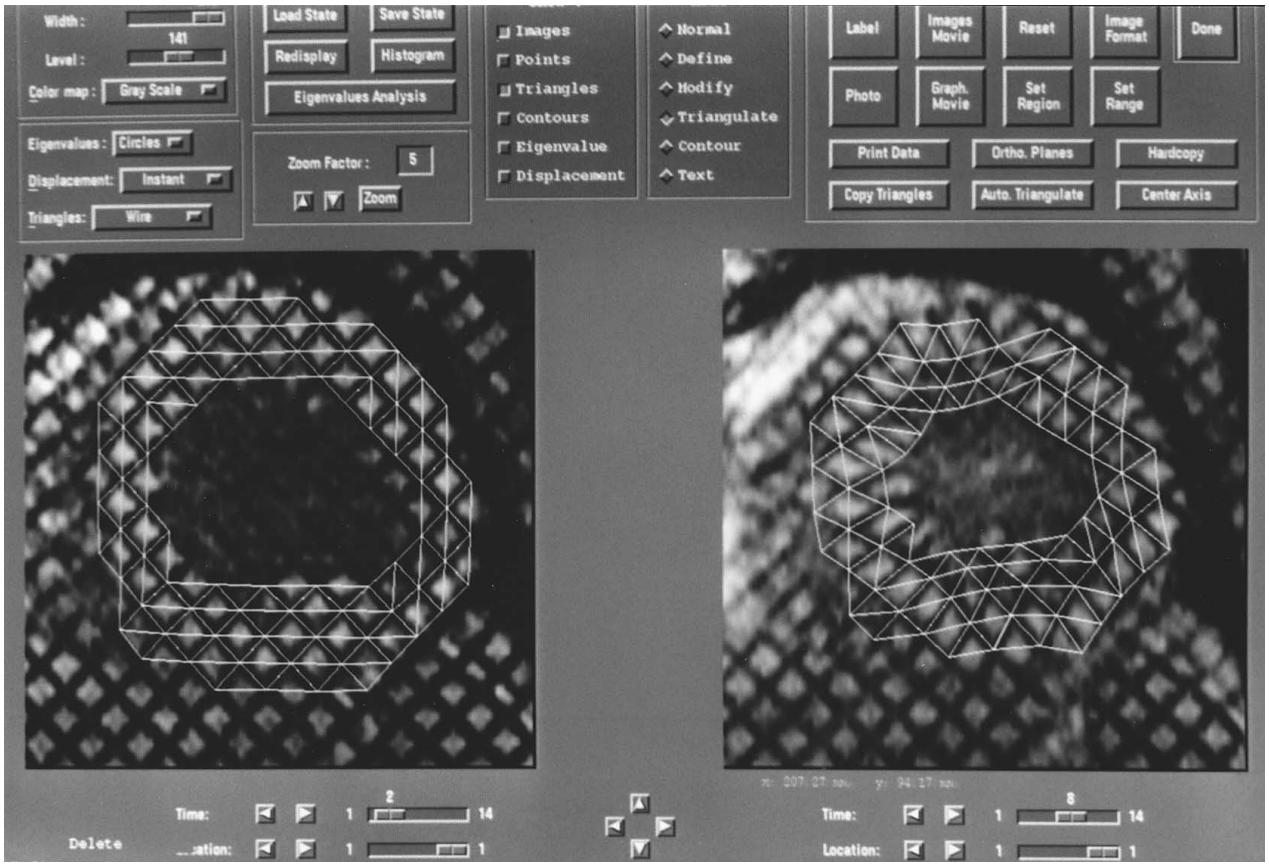


**Figure 2.** End-diastolic (left) and end-systolic (right) short-axis MR tagged images loaded on SPAMMVU software on a Silicon Graphics workstation (© Univ of PA). The anterior wall is from 11 o'clock to 2 o'clock, the lateral wall from 2 o'clock to 5 o'clock, the inferior wall from 5 o'clock to 8 o'clock, and the septum from 8 o'clock to 11 o'clock. The stripe intersections have been identified and triangular elements have been created, as defined by those intersections, and tracked through the cardiac cycle. Note the marked deformation of the tag stripes and then the triangles at end-systole.

was calculated from the four-chamber long-axis end-diastolic image as the ratio of short-axis to long-axis dimension. These were measured as the distance across the short-axis of the LV cavity below the mitral valve apparatus and the distance from the apex of the LV cavity to the center of mitral valve plane, respectively.

Intramyocardial strain analysis was performed on all short-axis image sets for each patient before and after MVR and compared with normal volunteers. One two-chamber and one four-chamber long-axis image set were also analyzed in five patients. In two patients one of the long-axis image sets was technically inadequate for analysis. The short-axis slices were divided into basal, mid, and apical thirds of the left ventricle and into septum, anterior, lateral, and inferior quadrants. The four-chamber long-axis slices were divided into septal and lateral regions, whereas the two-chamber slices were divided into anterior and inferior regions.

The tag stripe intersections were automatically tracked through the cardiac cycle with triplets of stripe intersections defining triangular elements within the myocardium (18,21–23) (Fig. 2). Two-dimensional strain analysis was performed between the initial state of end-diastole and the deformed state of end-systole (Fig. 2). Results are given as L1, the greatest systolic stretch or lengthening; L2, the greatest systolic shortening; and β, the angular deviation of L1 from the radial direction. If β is equal to zero on the short-axis images, then L1 can be regarded as a measure of radial wall thickening and L2 as a measure of circumferential shortening. In the long-axis, L2 is equivalent to longitudinal shortening when β is equal to 0.

### Statistical Methods

LVM index, LVEDV, LVESV, LVM/LVEDV ratio, EF, and sphericity index were compared between the Pre and Post studies by the Student paired *t*-test. L1, L2, and β were compared by region in patients between Pre and Post using a paired *t*-test and were compared with normal volunteers using an unpaired *t*-test. Regions were averaged by short-axis and long-axis location and for the entire left ventricle and then similarly compared between Pre and Post. All results are expressed as means ± SD. A $p < 0.05$ was considered statistically significant.

## RESULTS

The patients' clinical data are presented in Table 1. Heart rate was not different at the time of the two studies (78 ± 9 beats/min Pre and 77 ± 8 beats/min Post, not significant) nor was systolic blood pressure (134 ± 1 7 vs. 136 ± 12 mm Hg, not significant). Diastolic blood pressure rose between studies (72 ± 9 to 81 ± 6 mm Hg, $p \leq 0.02$).

LVM index declined after MVR from 142 ± 38 to 117 ± 44 g/m$^2$ ($p < 0.008$). For reference, LVM index in control subjects was 85 ± 10 g/m$^2$. LVEDV decreased from 117 ± 26 to 69 ± 12 ml ($p < 0.003$). The mass/EDV ratio increased between the prevalve repair study and the postvalve repair study from 2.2 ± 0.3 to 3.1 ± 0.4 g/ml ($p < 0.02$). LVESV also fell from 47 ± 10 to 28 ± 7 ml ($p < 0.002$). The study cohort demonstrated a normal mean LV EF before surgery that did not change after MVR (59 ± 7% vs. 59 ± 7%, not significant). At Pre, the sphericity index was 0.86 ± 0.10 (Fig. 3). However, at Post the ratio fell to 0.71 ± 0.13 ($p = 0.02$), indicating a more ellipsoid chamber.

The results for greatest systolic lengthening (L1) for all the short-axis slices by region are shown in Table 2. When values for the whole heart were averaged, L1 was 16 ± 6% in normal volunteers. In patients with severe mitral regurgitation, L1 was greater at 19 ± 9% ($p < 0.003$). Most of the increase was found at the mid-ventricular level (21 ± 10% vs. 15 ± 5%, $p < 0.002$) and in the inferior wall (21 ± 10% vs. 15 ± 5%, $p < 0.009$) (Table 2). After MVR, L1 increased further to 21 ± 8% ($p < 0.0001$ vs. control subjects, $p < 0.06$ vs.

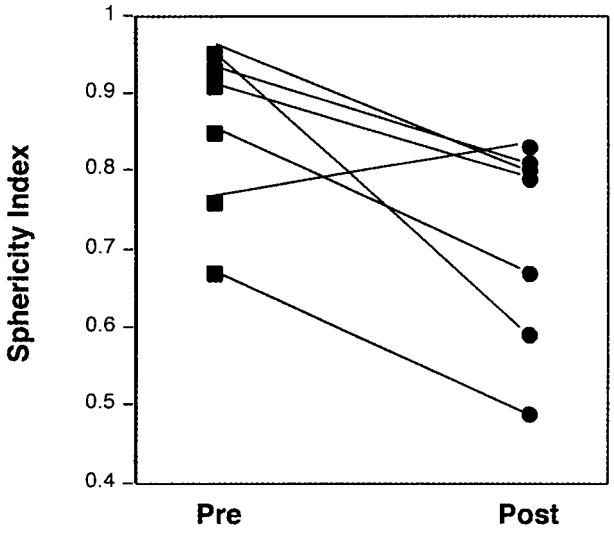


**Figure 3.** Graph of sphericity (short-axis dimension/long-axis dimension) before MVR (Pre, closed squares) compared with after MVR (Post, closed circles). Note the fall in sphericity between Pre and Post in six of seven subjects.

***Table 2***

*Results for L1 (%) for All Short-Axis Slices*

| | Apex | Mid | Base | Average |
|---|---|---|---|---|
| Septum | | | | |
| Controls | 19 ± 6 | 16 ± 4 | 22 ± 8 | 19 ± 7 |
| Pre | 15 ± 9 | 24 ± 12* | 22 ± 9 | 20 ± 10 |
| Post | 16 ± 9 | 24 ± 8* | 21 ± 7 | 21 ± 9 |
| Anterior | | | | |
| Controls | 12 ± 5 | 11 ± 5 | 15 ± 6 | 13 ± 5 |
| Pre | 14 ± 9 | 18 ± 9 | 17 ± 7 | 16 ± 8 |
| Post | 22 ± 9*† | 19 ± 5* | 21 ± 6 | 21 ± 7*† |
| Lateral | | | | |
| Controls | 14 ± 4 | 17 ± 6 | 21 ± 6 | 18 ± 6 |
| Pre | 17 ± 7 | 22 ± 9 | 21 ± 7 | 20 ± 8 |
| Post | 24 ± 7*† | 25 ± 8* | 23 ± 7 | 24 ± 7*† |
| Inferior | | | | |
| Controls | 14 ± 5 | 14 ± 4 | 16 ± 6 | 15 ± 5 |
| Pre | 23 ± 13 | 19 ± 8 | 19 ± 8 | 21 ± 10* |
| Post | 18 ± 8 | 21 ± 7* | 18 ± 8 | 19 ± 8* |
| Average | | | | |
| Controls | 15 ± 5 | 15 ± 5 | 19 ± 7 | 16 ± 6 |
| Pre | 17 ± 10 | 21 ± 10* | 20 ± 7 | 19 ± 9* |
| Post | 20 ± 8*† | 22 ± 7* | 21 ± 7 | 21 ± 8*‡ |

*$p < 0.04$ vs. controls.
†$p < 0.04$ vs. Pre.
‡$p < 0.06$ vs. Pre.

Pre), particularly at the apex (20 ± 8% Post vs. 17 ± 10% Pre, $p < 0.04$) (Table 2). Lengthening in the inferior wall, which was increased in patients with mitral regurgitation, did not change after MVR (19 ± 8% Post vs. 21 ± 10% Pre). The anterior and lateral walls demonstrated increased lengthening only after MVR (21 ± 7% Post vs. 16 ± 8% Pre, $p < 0.004$, and 24 ± 7% Post vs. 20 ± 8% Pre, $p < 0.007$, respectively).

The direction of lengthening or β is 12 ± 8 degrees in normal subjects (Table 3). This angle was increased to 19 ± 8 degrees ($p < 0.0001$) (directed further away from the cavity central) in patients with severe mitral regurgitation and remained unchanged Post at 19 ± 9 degrees ($p < 0.0001$ vs. control subjects). The only regional difference after MVR was an increase in β in the mid-inferior wall (17 ± 4 Post vs. 13 ± 5 degrees Pre, $p < 0.04$) (Table 3).

Shortening, or L2, was significantly decreased in patients with severe mitral regurgitation (12 ± 6% vs. 21 ± 6% in control subjects, $p < 0.001$) when averaged for the whole heart. This decrease was seen throughout every region of the LV (Table 4). After MVR, L2 decreased even further (9 ± 7% Post vs. 12 ± 6% Pre, $p < 0.001$). This decrease was more pronounced in the mid-left ventricle (9 ± 8% Post vs. 14 ± 5% Pre, $p < 0.0005$) and the anterior wall (9 ± 7% Post vs. 14 ± 5% Pre, $p < 0.01$).

In the long axis, there was no difference in L1 between control subjects and patients with severe MR (13 ± 8% in control subjects vs. 16 ± 8% Pre, not significant). β was likewise no different between the study population at Pre and control subjects (30 ± 13 degrees in control subjects vs. 29 ± 11 degrees Pre). After MVR, L1 was unchanged (13 ± 8% in control subjects vs. 15 ± 6% Post, $p$ = NS) and showed no directional change (29 ± 11 degrees Post vs. 29 ± 11 degrees Pre). No significant regional variation was found.

Long-axis shortening (L2) was 18 ± 5% in normal volunteers and was not different in patients with severe mitral regurgitation at 16 ± 5% (not significant). However, after MVR, long-axis shortening overall decreased to 11 ± 4% ($p < 0.0001$ vs. control subjects, $p < 0.002$ vs. Pre). The anterior and lateral walls showed the greatest decline from Pre to Post (16 ± 3% Pre to 8 ± 2% Post, $p < 0.02$ and 17 ± 3% Pre to 12 ±2 % Post, $p < 0.02$, respectively).



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***Table 3***

*Results of β (Degrees) for All Short-Axis Slices*

| | Apex | Mid | Base | Average |
|---|---|---|---|---|
| Septum | | | | |
| Controls | 11 ± 7 | 9 ± 2 | 8 ± 3 | 9 ± 4 |
| Pre | 22 ± 6* | 18 ± 9* | 21 ± 8* | 20 ± 8* |
| Post | 22 ± 9* | 17 ± 7* | 24 ± 16* | 21 ± 11* |
| Anterior | | | | |
| Controls | 18 ± 6 | 10 ± 3 | 17 ± 7 | 15 ± 6 |
| Pre | 22 ± 7 | 15 ± 7 | 26 ± 12 | 21 ± 10* |
| Post | 22 ± 10 | 15 ± 4* | 23 ± 9 | 20 ± 8* |
| Lateral | | | | |
| Controls | 6 ± 4 | 6 ± 2 | 9 ± 6 | 7 ± 4 |
| Pre | 15 ± 9* | 14 ± 4* | 22 ± 6* | 17 ± 7* |
| Post | 15 ± 9* | 12 ± 5* | 15 ± 4* | 14 ± 6* |
| Inferior | | | | |
| Controls | 11 ± 6 | 15 ± 10 | 21 ± 15 | 16 ± 11 |
| Pre | 18 ± 8 | 13 ± 5 | 24 ± 5 | 19 ± 8 |
| Post | 19 ± 2* | 17 ± 4† | 28 ± 12 | 21 ± 8 |
| Average | | | | |
| Controls | 12 ± 7 | 10 ± 6 | 14 ± 10 | 12 ± 8 |
| Pre | 19 ± 8* | 15 ± 6* | 24 ± 8* | 19 ± 8* |
| Post | 19 ± 8* | 15 ± 5* | 23 ± 11* | 19 ± 9* |

*$p < 0.05$ vs. controls.
†$p < 0.04$ vs. Pre.

***Table 4***

*Results for L2 (%) for All Short-Axis Slices*

| | Apex | Mid | Base | Average |
|---|---|---|---|---|
| Septum | | | | |
| Controls | 19 ± 3 | 16 ±2 | 16 ± 4 | 17 ± 3 |
| Pre | 12 ± 4* | 12 ± 5* | 3 ± 5* | 9 ± 6* |
| Post | 11 ± 7* | 6 ± 8* | 2 ± 6* | 6 ± 8* |
| Anterior | | | | |
| Controls | 25 ± 4 | 22 ± 4 | 20 ± 5 | 22 ± 5 |
| Pre | 14 ± 4* | 17 ± 3* | 10 ± 4* | 14 ± 5* |
| Post | 12 ± 7* | 9 ± 8*† | 6 ± 7* | 9 ± 7*† |
| Lateral | | | | |
| Controls | 25 ± 4 | 27 ± 3 | 21 ± 5 | 24 ± 5 |
| Pre | 15 ± 6* | 14 ± 6* | 8 ± 6* | 12 ± 6* |
| Post | 14 ± 6* | 13 ± 7* | 12 ± 6* | 13 ± 6* |
| Inferior | | | | |
| Controls | 24 ± 6 | 19 ± 4 | 14 ± 7 | 19 ± 7 |
| Pre | 16 ± 4* | 13 ± 4* | 3 ± 5* | 11 ± 7* |
| Post | 12 ± 8* | 8 ± 7* | 4 ± 5* | 8 ± 7* |
| Average | | | | |
| Controls | 23 ± 5 | 21 ± 6 | 17 ± 6 | 21 ± 6 |
| Pre | 14 ± 5* | 14 ± 5* | 6 ± 6* | 12 ± 6* |
| Post | 12 ± 7* | 9 ± 8*† | 6 ± 7* | 9 ± 7*† |

*$p < 0.03$ vs. controls.
†$p < 0.02$ vs. Pre.



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## DISCUSSION

In this study, patients with severe mitral regurgitation and normal global LV EF were studied with MRI before and after MVR. After MVR, LVM index, LVEDV, and LVESV all fell, whereas EF remained unchanged. The fall in LVEDV outstripped the fall in LVM, leading to an increase in LV mass-to-volume ratio. The LV chamber also became less spherical after MVR.

MRI noninvasively demonstrated abnormal intramyocardial strains in patients with severe mitral regurgitation, despite normal LV EF. Systolic short-axis lengthening (L1) was increased in patients with severe mitral regurgitation compared with normal volunteers. However, the direction of lengthening (β) was more oblique than normal in patients with severe mitral regurgitation. Short-axis shortening (L2) was markedly depressed for the whole heart compared with normal volunteers. Long-axis strains were no different from control subjects. After MVR, there was a strong trend toward an increase in short-axis L1 compared with the prerepair state. The direction of L1 was essentially unchanged after MVR. Short-axis L2 was depressed even further after MVR. Long-axis L2 decreased after MVR, particularly in the anterior and lateral wall.

EF in patients with severe mitral regurgitation was maintained by the increase in L1, although it occurred less efficiently because β was significantly increased. The increase in β may be due to the increase in sphericity in chronic mitral regurgitation. The depressed short-axis L2 in severe mitral regurgitation may be secondary to the compensatory LV hypertrophy demonstrated in these patients. This relationship has also been described in patients with concentric LV hypertrophy due to hypertension but normal EF using MRI (24). Structurally in chronic mitral regurgitation, a decrease in myofibril content and an increase in myocyte length have been described (25) and may contribute to decreased shortening.

After MVR, the further increase in short-axis lengthening may be facilitated by the reduction in sphericity, although no further directional change in L1 occurred. The fall in long-axis shortening after MVR, seen particularly in the anterior and lateral walls, is likely secondary to the tethering effect imposed by the annuloplasty ring on longitudinal shortening.

### Comparison to Prior Studies

Severe chronic mitral regurgitation produces significant increases in LVEDV, LVESV, total stroke volume, and LVM (26,27). Kleaveland et al. (26) found in dogs that although there was an average 32% increase in LVM, both LV mass-to-volume ratio and relative wall thickness fell, implying eccentric hypertrophy. They also found a fall in length-to-diameter ratio at end-diastole, indicating an increase in sphericity in response to chronic volume overload. Our results concur with these findings and demonstrate the regression of the volume overload and eccentric hypertrophy after repair.

Subtle changes in the global contractile state of the left ventricle in severe mitral regurgitation have been demonstrated. Starling et al. (1) found subgroups of patients with chronic severe mitral regurgitation and normal EF that have contractile dysfunction as evidenced by depressed LV chamber elastance. They concluded that this patient population benefits by earlier surgery, before irreversible contractile dysfunction occurs. Latent ventricular dysfunction may be indicated by a limited contractile reserve, manifest as an inadequate increase in EF with exercise (28).

Conventional mitral valve replacement with complete excision of the native valve apparatus has been associated with postoperative low cardiac output syndrome and higher mortality rates (3). The importance of the integrity of the mitral valve apparatus has been underscored in both animal (29) and clinical studies (6,7). After valve repair, overall operative mortality is lower and overall survival at 10 years is significantly higher than with valve replacement (7). The LV sphericity in severe mitral regurgitation declines after MVR compared with replacement (10), and this may be a predictor of postoperative survival (30).

Regional mechanics have been studied in animal models in the setting of mitral valve replacement rather than repair. After replacement, a decline in segmental LV function has been found in areas subtended by papillary muscle insertion via piezoelectric crystal implantation within canine hearts (14). In another canine model, investigators found that chordal transsection resulted in unloading of the myocardium at the site of papillary muscle insertion, which accounted for heterogeneity in regional function, thus compromising global systolic function (15).

### Limitations

One limitation of the present study is the small number of patients enrolled. Nonetheless, the patient group is homogeneous (all had severe mitral regurgitation secondary to intrinsic valve disease). Only patients with normal baseline EF were studied. The results cannot be extrapolated to patients with severe mitral regurgitation in the

setting of LV dysfunction secondary to coronary disease or dilated cardiomyopathy.

Only one time point after MVR was studied. More information on the time course of changes in regional strains and global LV size and function would add to the understanding of the natural history of MVR for severe mitral regurgitation. A two-dimensional analytic approach was used rather than a three-dimensional approach (31). The principal shortening strains may be higher when using a three-dimensional method. However, the two-dimensional approach may be more practical in the clinical setting due to the time necessary and degree of difficulty of the three-dimensional analysis.

Age-matched control subjects were not used. However, other investigators demonstrated with MRI that circumferential fiber shortening decreases only slightly with age (from the second to the seventh decades) from 26% to 24% (32). In the present study, the absolute reduction in L2 relative to normal subjects was 9% and hence is unlikely to be due to differences in age alone. The postsurgical state may confer abnormalities of septal strains.

### Future Directions

Although clinical characteristics of patients who undergo mitral valve replacement rather than MVR may be different, it is important to compare regional strains between these two types of patients. In addition, the effect of MVR on regional strains in patients with dilated cardiomyopathy and severe mitral regurgitation is of considerable interest (33).

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