Left ventricular torsional mechanics after left ventricular reconstruction surgery for ischemic cardiomyopathy

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Objectives: Surgical left ventricular reconstruction improves symptoms and potentially prognosis in patients with ischemic cardiomyopathy; however, the effects of reconstruction on myocardial mechanics are not well defined. Therefore, we have computed left ventricular rotation and torsion in patients undergoing left ventricular reconstruction to determine its effects on these quantitative measures of myocardial mechanics.

Methods: Magnetic resonance imaging with tissue grid-tagging was performed in 26 patients (19 male/7 female, 62 ± 11 years) (mean $\pm$ standard deviation) before (23 ± 29 days) and after (231 ± 106 days) left ventricular reconstruction, as well as in 7 healthy volunteers (5 male/2 female, 34 ± 7 years). Left ventricular rotation was computed at basal and apical short-axis levels; torsion was defined as the difference between apical and basal rotation.

Results: Before left ventricular reconstruction, maximal apical rotation was significantly impaired relative to that of healthy volunteers ($P = .001$), although maximal basal rotation was preserved ($P = .84$). After reconstruction, maximal torsion did not change significantly: torsion was $6^\circ \pm 3^\circ$ both before and after reconstruction ($P = .84$). However, the rate of early diastolic untwist improved significantly after reconstruction ($-18^\circ/\text{s} \pm 13^\circ/\text{s}$ vs $-23^\circ/\text{s} \pm 14^\circ/\text{s}$; $P = .04$). Furthermore, patients with relatively worse torsion before reconstruction demonstrated more improved function after reconstruction; patients with torsion of less than 6° ($n = 12$) showed greater improvement in ejection fraction (15% vs 6%; $P = .005$), torsion (1° vs -1° ; $P = .01$), and diastolic untwist ($-9^\circ/\text{s}$ vs $-25^\circ/\text{s}$; $P < .001$) than did patients with torsion of 6° or more ($n = 14$).

Conclusions: Torsional mechanics were severely impaired by ischemic cardiomyopathy. On average, left ventricular reconstruction did not affect systolic torsion generation significantly; however, patients with relatively worse torsion did show improvement. Furthermore, the rate of untwist improved after surgery, suggesting that diastolic function was improved.

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Contraction of obliquely oriented muscle fibers in the mammalian left ventricle (LV) results in twisting of the LV apex relative to the base during systole. This torsional deformation is thought to equalize sarcomere shortening across the LV wall, thus possibly reducing transmural gradients of oxygen use

Abbreviations and Acronyms

CABG	= coronary artery bypass grafting
EDV	= end-diastolic volume
EF	= ejection fraction
FOV	= field of view
ICM	= ischemic cardiomyopathy
LV	= left ventricle(ular)
LVR	= left ventricular reconstruction
MRI	= magnetic resonance imaging
MVR	= mitral valve replacement/repair
SV	= stroke volume
TE	= echo time
TR	= repetition time

and contractile work.^{1,2} In addition, rapid untwisting of the LV during isovolumic relaxation is thought to enhance early diastolic filling.³

Numerous previous studies have demonstrated that alterations in the magnitude and pattern of LV rotation occur with cardiac disease.⁴⁻⁸ In particular, apical rotation is diminished in patients with chronic anterolateral infarction and absent in patients with LV aneurysm.⁷ Similarly, LV torsion has been shown to be diminished in an animal model of chronic infarction.⁸ These studies have also demonstrated impaired early diastolic untwisting of the LV with chronic infarction.^{7,8} Furthermore, the presence of chronic ischemic mitral regurgitation has been shown to exacerbate the impairment of both systolic torsion and early diastolic untwist.⁸

Left ventricular reconstruction (LVR) surgery, often performed in conjunction with mitral valve replacement/repair (MVR) and/or coronary artery bypass grafting (CABG), has been shown to improve symptoms, and potentially prognosis, in patients with chronic ischemic cardiomyopathy (ICM), with or without LV aneurysm.⁹⁻¹² Studies have reported improved LV systolic function in these patients, typically using ejection phase indices based on LV volume, which are known to be dependent on cardiac loading conditions.¹⁰⁻¹³ Improvements in diastolic function,¹⁴ as well as in load-independent measures of LV contractility,^{15,16} after LVR have also been reported, but these studies have been limited to the acute or early postoperative phases. One recent study, however, reported no postoperative changes in myocardial strain in patients up to 1 year after LVR.¹⁷ Although correlated with torsion, myocardial strain is a local measure of deformation and represents the normalized change in distance between material points in myocardium, whereas torsion reflects global LV mechanics, defined as the difference in rotation angle between the base and apex.

Although it has been postulated that LV torsion will improve after LVR as a result of the restoration of a more elliptical LV geometry,¹⁸ no previous studies have quanti-

fied LV torsion in patients who have undergone LVR. Thus, the purpose of this study was to quantify the effects of LVR on the magnitude and pattern of LV rotation and torsion in patients with chronic ICM. Furthermore, we sought to quantify the effects on postoperative torsion of LV aneurysm and MVR.

Materials and Methods**Study Subjects**

Patient data used in this study were analyzed with an institutional review board–approved waiver of individual informed consent. The study was compliant with the Health Insurance Portability and Accountability Act. We included 26 patients (19 male/7 female, aged 62 ± 11 years) (mean $\pm$ standard deviation) with chronic ICM who had undergone clinically indicated magnetic resonance imaging (MRI) before and after LVR between 1998 and 2003. All patients underwent both a preoperative MRI study (23 ± 29 days before LVR, median 7 days) and an MRI examination at least 125 days after the operation (231 ± 106 days, median 190 days). In 21 of 26 (81%) patients, ICM was complicated by a true anteroapical LV aneurysm. It was characterized by an akinetic or dyskinetic thinned region demonstrating end-diastolic bulging of the epicardial surface, which was accentuated during systolic contraction (these patients denoted as aneurysm[+] patients). In the remaining 5 (19%) patients (denoted as aneurysm[-]), the LV demonstrated large anteroapical dyskinetic or akinetic regions, with bulging of the epicardial surface only during systolic contraction.

In addition, to define normal LV torsional mechanics, 7 healthy volunteers (5 male/2 female, mean age 34 ± 7 years) were recruited as part of an institutional review board–approved study for evaluation of normal cardiovascular function by MRI.

Surgical Technique

LVR was performed in each patient by a modification of the Dor technique.¹⁹ As described previously,⁹ this involves exclusion of the scarred anteroapical postinfarction regions of the LV using circumferential endocardial sutures, and a patch if necessary, to restore elliptical LV configuration. LVR was performed alone ($n = 2$, 8%) or combined with CABG and/or MVR as follows: LVR + CABG, $n = 15$ (58%); LVR + MVR, $n = 2$ (8%); and LVR + CABG + MVR, $n = 7$ (27%).

Imaging

Patient imaging was performed with a 1.5-tesla MRI scanner (Sonata; Siemens Medical Solutions, Erlangen, Germany). After scout imaging to locate the cardiac axes, cine images were acquired in contiguous short-axis slices from the LV base to apex with an electrocardiogram-triggered spoiled gradient echo pulse sequence (echo time [TE] = 4–6 ms, repetition time [TR] = 9–13 ms, flip angle = 30°, slice thickness = 6–10 mm, field of view [FOV] = 300–360 mm at 6/8–8/8 rectangular matrix starting at 256²), an electrocardiogram-triggered steady state free precession pulse sequence (TE = 1.6 ms, TR = 3.2 ms, flip angle = 60°, slice thickness = 8–10 mm, FOV_x = 263–360 mm, FOV_y = 300–360 mm, initial matrix = 256²), or a retrospectively electrocardiogram-triggered steady state free precession sequence (TE = 1.6 ms, TR = 3.5 ms, flip angle = 70°, slice thickness = 8–10 mm,

FOV_x = 263–360 mm, FOV_y = 300–360 mm, initial matrix = 256²). Temporal resolution of cine images ranged from 30 to 50 ms. Each image set was acquired during a single breath-hold (10–12 seconds); however, in patients unable to adequately suspend respiration, 3 signal averages were used during free breathing.

Healthy volunteers were also imaged with a 1.5-tesla MRI scanner (Avanto; Siemens Medical Solutions). Cine images were acquired in contiguous slices from the LV base to apex with a retrospectively electrocardiogram-triggered steady state free precession sequence (TE = 1.6 ms, TR = 3.5 ms, flip angle = 70°, slice thickness = 8–10 mm, FOV_x = 263–360 mm, FOV_y = 300–360 mm, initial matrix = 256², temporal resolution 30–50 ms). Each image set was acquired during a single breath-hold (10–12 seconds).

In patients and healthy volunteers, grid-tagged images were acquired with an electrocardiogram-gated, segmented k-space, gradient echo sequence (spatial modulation of magnetization with 8-mm tag spacing: TE = 4 ms, TR = 9 ms, flip angle = 15°, slice thickness = 10 mm, FOV_x = 244–350 mm, FOV_y = 300–380 mm, initial matrix = 256², temporal resolution 32–57 ms) at select short-axis locations matching the cine images.

Image Analysis

In short-axis cine images, LV endocardial contours were manually traced at end-diastole and end-systole with dedicated cardiovascular image analysis software (Argus; Siemens Medical Solutions) for determination of end-diastolic volume (EDV), stroke volume (SV), and ejection fraction (EF).

For analysis of rotation and torsion, the LV base was identified as the most basal short-axis level in which no atrial or aortic valve tissue was visible in any image frame of the cine series; the LV apex was defined as the most apical short-axis level that contained adequate myocardium for tag-lines to persist throughout systole. Then, LV rotation was computed at the LV apex and base using harmonic phase (HARP) analysis software (Diagnosoft, Inc, Palo Alto, Calif).²⁰ By manually delineating the LV endocardium and epicardium at end-systole, we specified the LV mid wall, which could then be located automatically in all other image frames. LV rotation was calculated as the average rotation (in degrees) of mid-wall points about the slice center of volume. By convention, counterclockwise rotation was considered positive as viewed from the apex toward the base. Torsion was defined as the difference in LV rotation between the apical and basal levels. Early diastolic untwist was defined as the difference between maximal torsion and torsion in the subsequent image frame, divided by the time between image frames.

Statistical Analysis

All statistical analyses were performed with SAS software (version 8.2, SAS Institute, Inc, Cary, NC). The statistical significance of comparisons between patient data before and after surgery was assessed by paired *t* tests (2-tailed). The statistical significance of comparisons between healthy volunteers and patients, at either time point (ie, pre-LVR or post-LVR), was assessed by 2-sample *t* tests (2-tailed). Comparisons between subgroups of patients were also made by 2-sample *t* tests (2-tailed). Mean ± standard deviation is presented for continuously distributed variables.

TABLE 1. LV morphology and function in healthy volunteers (n = 7) and in ICM patients (n = 26) before and after LVR

	Healthy volunteers	Patients with ICM	
		Pre-LVR	Post-LVR
End-diastolic volume (mL)	135 ± 29	277 ± 86*	212 ± 55*†
Stroke volume (mL)	81 ± 18	75 ± 20	76 ± 22
Ejection fraction (%)	61 ± 3	29 ± 8*	37 ± 10*†
Heart rate (beats/min)	61 ± 12	72 ± 12	73 ± 15

LV, Left ventricular; ICM, ischemic cardiomyopathy; LVR, left ventricular reconstruction. **P* < .05 versus normal; †*P* < .05 versus pre-LVR. See text for actual *P* values.

Results

LV Function

Compared with those of healthy volunteers, patients with chronic ICM exhibited dilated LVs (*P* < .001) and impaired EF (*P* < .001) before surgery, although SV was preserved on average (*P* = .39) (Table 1). After surgery, LV size was significantly diminished relative to preoperative values (*P* < .001), EF was significantly improved (*P* < .001), and SV was unchanged (*P* = .18). In addition, after surgery, LVs remained dilated compared with those of healthy volunteers (*P* = .002) and EF remained impaired (*P* < .001), on average.

LV Systolic Rotation and Torsion

End-systolic tagged images from the base and apex of a healthy volunteer, as well as a patient both before and after surgery, are shown in Figure 1. Myocardial trajectories, tracking the movement of sampled myocardial locations during systole, and which highlight the rotational component of systolic contraction, are superimposed on each image.

LV systolic rotation and torsion in healthy volunteers are shown in Figures 2 and 3, respectively. As expected, the apex and base rotated together during early systole, with no appreciable torsion generated over the first 20% to 25% of systole. For the remaining 75% of systole, the apex and base rotated in opposite directions, resulting in the familiar “wringing” motion during systolic ejection. Maximal systolic rotation at the LV base and apex, as well as maximal systolic LV torsion, are shown in Table 2.

LV systolic rotation and torsion in patients with ICM, before and after LVR, are shown in Figures 2 and 3, respectively. In contrast to torsion in healthy volunteers, LV torsion in patients with ICM was non-zero at all systolic time points, both before and after surgery; that is, the apex and base did not rotate together in early systole, on average. Furthermore, on average, LVR produced no noticeable effect on the pattern or magnitude of LV rotation in patients with ICM.

Figure 1. End-systolic tagged images at the base (*left*) and apex (*right*) from a healthy volunteer (*top*) and from a patient both before (*middle*) and after left ventricular reconstruction (LVR) (*bottom*). Trajectories superimposed on each image (*in yellow*) depict the motion of sampled myocardial locations from end diastole to end systole.

As shown in [Table 2](#), after surgery there was no statistically significant change in maximal systolic rotation at either the base ($P = .55$) or apex ($P = .58$) relative to preoperative values. Similarly, there was no statistically significant change in maximal systolic torsion after surgery, on average ($P = .95$). Although maximal apical rotation remained significantly diminished compared with that of healthy volunteers after surgery ($P < .001$), there was no significant difference in maximal basal rotation between ICM patients after LVR and healthy volunteers.

In general, patients with relatively worse torsion before LVR demonstrated greater improvement in function after surgery. Patients were categorized according to a maximal torsion threshold of 6° (2 standard deviations below the mean torsion in healthy volunteers, [Table 2](#)). Before sur-

gery, patients with maximal torsion of less than 6° ($n = 19$) had significantly worse EDV (321 ± 98 mL vs 242 ± 58 mL; $P = .02$), EF ($24\% \pm 7\%$ vs $32\% \pm 8\%$; $P = .01$), apical rotation ($0^\circ \pm 2^\circ$ vs $4^\circ \pm 3^\circ$; $P = .004$), and torsion ($4^\circ \pm 1^\circ$ vs $8^\circ \pm 2^\circ$; $P < .001$) than patients with torsion of 6° or more ($n = 21$). Although all patients demonstrated improved EDV and EF after surgery, on average, only patients with preoperative torsion of less than 6° showed statistically significant improvement in apical rotation ($0^\circ \pm 2^\circ$ before vs $2^\circ \pm 2^\circ$ after; $P = .03$) and torsion ($4^\circ \pm 1^\circ$ before vs $5^\circ \pm 1^\circ$ after; $P = .004$); however, basal rotation did not change significantly ($-3^\circ \pm 2^\circ$ before versus $-3^\circ \pm 1^\circ$ after; $P = .59$). Furthermore, patients with preoperative torsion of less than 6° showed significantly greater improvement in EF and torsion after LVR than did patients with

Figure 2. *Top*, Left ventricular (LV) systolic twist at the base and apex in healthy volunteers ($n = 7$). There was no statistically significant difference in basal and apical twist values through the first 25% systole. *Error bars* denote the standard deviation at each time point. *Bottom*, LV systolic twist at the base and apex in patients with ischemic cardiomyopathy before and after left ventricular reconstruction (LVR) ($n = 26$). In this plot, error bars have been omitted for clarity.

torsion of 6° or more; ΔEF was $15\% \pm 8\%$ in patients with torsion of less than 6° versus $6\% \pm 8\%$ in patients with torsion of 6° or more ($P = .005$); $\Delta\text{torsion}$ was $1^\circ \pm 1^\circ$ in patients with torsion of less than 6° versus $-1^\circ \pm 2^\circ$ in patients with torsion of 6° or more ($P = .01$).

Early Diastolic Untwist

In patients with ICM, the rate of early diastolic untwist was impaired before surgery, relative to healthy volunteers, but improved significantly after LVR (Table 2). However, after surgery, early diastolic untwist remained significantly impaired relative to healthy volunteers.

Patients with relatively worse maximal torsion (or diastolic untwist rate) before surgery demonstrated greater improvement in early diastolic untwist rate after surgery.

Figure 3. *Top*, Systolic left ventricular (LV) torsion in healthy volunteers ($n = 7$). *Bottom*, Systolic LV torsion in patients with ischemic cardiomyopathy before and after left ventricular reconstruction (LVR) ($n = 26$). In both plots, error bars denote the standard deviation at each time point.

Before surgery, patients with torsion of less than 6° exhibited a significantly worse untwist rate than did patients with torsion of 6° or more ($-9^\circ/\text{s} \pm 7^\circ/\text{s}$ vs $-25^\circ/\text{s} \pm 12^\circ/\text{s}$; $P < .001$). After surgery, patients with preoperative torsion of less than 6° demonstrated improved early diastolic untwist rate ($-9^\circ/\text{s} \pm 7^\circ/\text{s}$ to $-18^\circ/\text{s} \pm 5^\circ/\text{s}$; $P < .001$), but patients with torsion of more than 6° did not ($-25^\circ/\text{s} \pm 12^\circ/\text{s}$ to $-27^\circ/\text{s} \pm 18^\circ/\text{s}$; $P = .61$).

Similarly, patients were categorized according to a pre-LVR rate of early diastolic untwist threshold of $-18^\circ/\text{s}$ (2 standard deviations above the mean value in healthy volunteers). Both before and after LVR, there was no statistically significant difference in EF, EDV, or SV between groups. Patients with pre-LVR untwist of $-18^\circ/\text{s}$ or more showed significant improvement in untwist rate after surgery; however, patients with pre-LVR untwist of less than $-18^\circ/\text{s}$ did not demonstrate a statistically significant change after LVR.

TABLE 2. Maximal systolic LV rotation (at the apex and base) and torsion in healthy volunteers (n = 7) and in ICM patients (n = 26) before and after LVR

	Healthy volunteers	Patients with ICM	
		Pre-LVR	Post-LVR
Basal rotation (°)	-4 ± 2	-4 ± 2	-3 ± 2
Apical rotation (°)	8 ± 4	2 ± 3*	3 ± 3*
Torsion (°)	12 ± 3	6 ± 3*	6 ± 3*
Early untwist rate (°/s)	-64 ± 23	-18 ± 13*	-23 ± 14*†

LV, Left ventricular; ICM, ischemic cardiomyopathy; LVR, left ventricular reconstruction. * $P < .05$ versus normal; † $P < .05$ versus before LVR. See text for actual P values.

Effects of LV Aneurysm

Before LVR, there was no statistically significant difference in any of the measured parameters between aneurysm(+) patients and aneurysm(-) patients (Table 3). However, there was a trend toward greater maximal apical rotation in aneurysm(+) patients ($P = .08$).

Similarly, after LVR, there was no statistically significant difference in any of the measured parameters between aneurysm(+) and aneurysm(-) patients, although there was a trend toward smaller EDV in aneurysm(+) patients ($P = .05$).

The presence of LV aneurysm had an important effect on the impact of LVR in the patients included in this study. Aneurysm(-) patients did not exhibit a statistically significant improvement in EF after surgery, even though EDV decreased significantly. Furthermore, the significant improvement in apical rotation after LVR was not accompanied by a change in torsion as it was offset by a worsening of basal rotation (ie, basal rotation became less negative) (Table 3). However, the rate of early diastolic untwist improved significantly in aneurysm(-) patients after surgery. In contrast, aneurysm(+) patients had no change in basal rotation, apical rotation, torsion, or early diastolic untwist rate, despite a significant decrease in EDV and a significant increase in EF.

Effects of Mitral Annuloplasty

Patients who underwent MVR at the time of LVR (MVR[+], $n = 9$), with or without CABG, had significantly worse maximal apical rotation before surgery than had patients who did not undergo MVR (MVR[-], $n = 17$) ($0^\circ \pm 2^\circ$ vs $3^\circ \pm 4^\circ$; $P = .03$). However, there were no other statistically significant differences between these groups.

Both EF and EDV improved significantly after surgery in MVR(-) patients: EF $28\% \pm 8\%$ before LVR versus $39\% \pm 9\%$ after LVR ($P < .001$); EDV 262 ± 65 mL before LVR versus 204 ± 48 mL after LVR ($P < .001$). However, only EF improved significantly in MVR(+) patients: $29\% \pm 9\%$ versus $34\% \pm 11\%$ ($P = .04$). In neither

group did rotation, torsion, or the rate of early diastolic untwist change significantly after surgery.

Discussion

Systolic rotation of the LV apex relative to the base (torsion) is a direct consequence of the innate helical fiber architecture of the LV²¹ and is a fundamental component of wall thickening and systolic ejection.²² Furthermore, alterations in the pattern or magnitude of systolic rotation are a sensitive indicator of myocardial disease.⁴⁻⁸ Because torsion is defined as rotation of the LV apex relative to the base, changes in rotation at either short-axis level may adversely affect LV torsion.

In healthy hearts, the torsion generated during systolic contraction is released during early diastole. This diastolic untwisting has been shown to coincide with isovolumic relaxation and is thought to result from the release of energy stored in elastic elements within the extracellular matrix during the previous systole.³ Furthermore, it has been suggested that untwist promotes LV suction and aids early diastolic filling.³

Seven healthy volunteers were included in the current study to define normal LV rotation and torsion; results were consistent with those reported previously in the literature with similar methods.^{5,7,23} Similarly, diastolic untwist rate results were consistent with previous reports: according to data from Rademakers and associates,³ the early diastolic untwist rate in open-chested dogs was $-95^\circ/\text{s}$ over the first 40 ms and $-60^\circ/\text{s}$ over the first 60 ms after maximal torsion.

LV Rotation and Torsion in Patients With ICM (Before LVR)

In this investigation, we have demonstrated impaired LV torsion in patients with chronic ICM, with or without LV aneurysm, primarily resulting from significantly diminished rotation of the LV apex in these patients. Rotation of the LV base, however, remote from the site of infarction, was preserved relative to that of healthy volunteers. In addition, the rate of early diastolic untwist was significantly impaired before LVR in patients with ICM.

These results are consistent with those of previous reports. For instance, Nagel and colleagues⁷ reported a significant reduction in apical rotation in patients with chronic anterolateral infarction (but no aneurysm). In patients with LV aneurysm, apical rotation was virtually nonexistent. However, independent of the presence of aneurysm, basal rotation was not significantly different from that of healthy controls. Furthermore, diastolic untwist was found to be impaired in patients with chronic infarction.

Effects of LVR on LV Rotation and Torsion

LVR is known to improve symptoms and is associated with better outcomes in patients with chronic ICM⁹⁻¹²; however,

TABLE 3. LV volume, function, maximal rotation, and torsion in patients with ICM with and without LV aneurysm before and after LVR

	Aneurysm(−) (n = 5)		Aneurysm(+) (n = 21)		P value
	Pre-LVR	Post-LVR	Pre-LVR	Post-LVR	
Ejection fraction (%)	28 ± 10	35 ± 9	29 ± 8	38 ± 10	a. .95 b. .18 c. <.01
End-diastolic volume (mL)	300 ± 91	254 ± 87	271 ± 86	202 ± 41	a. .51 b. <.01 c. <.01
Stroke volume (mL)	81 ± 20	84 ± 17	74 ± 21	74 ± 23	a. .51 b. .76 c. .33
Basal rotation (°)	−5 ± 3	−3 ± 2	−3 ± 2	−3 ± 2	a. .13 b. .01 c. .99
Apical rotation (°)	0 ± 2	3 ± 2	3 ± 4	3 ± 3	a. .08 b. .02 c. .94
Torsion (°)	5 ± 2	6 ± 2	6 ± 3	6 ± 3	a. .40 b. .40 c. .94
Early untwist rate (°/s)	−14 ± 4	−18 ± 5	−18 ± 14	−24 ± 16	a. .50 b. .01 c. .08

LV, Left ventricular; ICM, ischemic cardiomyopathy; LVR, left ventricular reconstruction. a, Pre-LVR aneurysm(−) versus pre-LVR aneurysm(+). b, Pre-LVR aneurysm(−) versus post-LVR aneurysm(−). c, Pre-LVR aneurysm(+) versus post-LVR aneurysm(+).

its effects on LV mechanics are not well defined. Although improved systolic function has been reported after LVR,¹⁰⁻¹³ functional measures derived from cardiac volumes are probably not appropriate after surgery that so dramatically modifies LV size and shape.

It is not surprising that our results showed no improvement in apical rotation after LVR, inasmuch as the neoapex, comprised of myocardium adjacent but basal to the LVR, is usually quite dysfunctional and predominantly scar, if not transmural. In addition, an akinetic patch or scar is used to exclude the infarct, bridging the septal and anterolateral segments, and this itself is noncontractile.

The reasons that basal rotation did not improve are less clear. LV rotation depends on the strength of contraction, cardiac loading conditions, and LV fiber architecture. However, because few patients undergo LVR alone, it is difficult to isolate the relative impact of geometric changes, revascularization, and mitral valve interventions on myocardial mechanics. There is little evidence to support increased contractility after LVR, except acutely.¹⁵⁻¹⁷ Afterload has been shown to decrease after LVR,²⁴ which should increase systolic torsion.²⁵ However, preload effects after LVR are uncertain. Although torsion is preload dependent, LVR might produce a reduced LV equilibrium volume, with little

preload change, inasmuch as long-term effects on myofiber length after LVR are unknown. Last, it has been postulated that LV fiber architecture is altered, to the detriment of torsion, as the LV dilates,¹⁸ although recent empirical evidence suggests that it is not.^{26,27} The purpose of LVR is to normalize LV shape, but any mechanical benefits (to torsion) resulting from this shape change might be offset by discontinuities in the fiber continuum at the surgical site.

In the current study, we found that the rate of early diastolic untwist improved significantly after LVR; this finding is consistent with a previous study that reported improved peak diastolic filling rate in patients evaluated by x-ray angiography approximately 10 days after surgery.¹⁴ Furthermore, although LVR produced no significant change in the pattern or magnitude of systolic LV rotation or torsion in patients with chronic ICM, patients with relatively worse torsion before LVR demonstrated greater improvement after surgery, a finding that warrants further study.

The presence of LV aneurysm had a significant effect on the results of LVR. Patients with ICM without LV aneurysm demonstrated a significant worsening in basal rotation after LVR, which was offset by a significant improvement in apical rotation, resulting in no net change in maximal systolic torsion. These patients also had no significant improve-

ment in EF. We believe this result is consistent with the results of previous modeling studies. Artrip, Oz, and Burkhoff²⁸ simulated the effects of LVR in an idealized LV model, demonstrating that although removal of dyskinetic scar (aneurysm) should improve overall LV pump function, resection of akinetic scar produces little effect. Interestingly, Artrip, Oz, and Burkhoff²⁸ also showed that pump function should decrease after removal of contracting (yet functionally impaired) myocardium, as in partial left ventriculectomy. It has been shown previously that in patients with nonischemic dilated cardiomyopathy, in whom LV torsion is already diminished, partial left ventriculectomy further impairs LV rotation near the site of surgery, but does not produce much effect on rotation in remote areas of the LV.⁶

Limitations

This study is subject to several limitations. First, the apical level was changed between imaging examinations owing to the surgical procedure itself, which qualifies direct comparison of results before and after surgery, but does not obviate our primary goal of quantifying the global impact of surgery on the ability of the LV to generate torsion. Despite this fact, both basal and apical results were included to emphasize that the lack of change in torsion did not result from improvement at one level offset by worsening at the other. Second, apical results do not necessarily reflect the true LV apex; rather, analysis was performed on the most apical level that contained adequate viable myocardium for tag-lines to persist throughout systole. However, because no rotation occurs within an LV aneurysm,⁷ we believed it most appropriate to analyze myocardium contributing to torsion generation. Third, owing to the effects of chronic ICM, some portions of the LV wall were only a single tag-line width thick in some patients, which added uncertainty to the rotation measurements. Last, only 5 patients without LV aneurysm were included in this study.

Clinical Implications

LV systolic torsion is significantly impaired in patients with chronic ICM and is not improved by LVR. However, the subgroup of patients with relatively worse systolic torsion before surgery demonstrated greater improvement after LVR. Furthermore, the rate of early diastolic untwist improved significantly after surgery. Further studies are necessary to determine whether LV torsion and the rate of early diastolic untwist can be used to help select patients for LVR and predict outcomes. It is conceivable that the improvement in symptoms and prognosis that many patients with chronic ICM exhibit after LVR is perhaps related to improved diastolic function, rather than to an improvement in systolic contractile mechanics.

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