Clinical assessment of left ventricular rotation and strain: a novel approach for quantification of function in infarcted myocardium and its border zones

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Helle-Valle T, Remme EW, Lyseggen E, Pettersen E, Vartdal T, Opdahl A, Smith HJ, Osman NF, Ihlen H, Edvardsen T, Smiseth OA. Clinical assessment of left ventricular rotation and strain: a novel approach for quantification of function in infarcted myocardium and its border zones. Am J Physiol Heart Circ Physiol 297: H257–H267, 2009. First published April 24, 2009; doi:10.1152/ajpheart.01116.2008.—Left ventricular (LV) circumferential strain and rotation have been introduced as clinical markers of myocardial function. This study investigates how regional LV apical rotation and strain can be used in combination to assess function in the infarcted ventricle. In healthy subjects (n = 15) and patients with myocardial infarction (n = 23), LV apical segmental rotation and strain were measured from apical short-axis recordings by speckle tracking echocardiography (STE) and MRI tagging. Infarct extent was determined by late gadolinium enhancement MRI. To investigate mechanisms of changes in strain and rotation, we used a mathematical finite element simulation model of the LV. Mean apical rotation and strain by STE were lower in patients than in healthy subjects (9.0 ± 4.9 vs. 12.9 ± 3.5° and −13.9 ± 10.7 vs. −23.8 ± 2.3%, respectively, P < 0.05). In patients, regional strain was reduced in proportion to segmental infarct extent (r = 0.80, P < 0.0001). Regional rotation, however, was similar in the center of the infarct and in remote viable myocardium. Minimum and maximum rotations were found at the infarct borders: minimum rotation at the border zone opposite to the direction of apical rotation, and maximum rotation at the border zone in the direction of rotation. The simulation model reproduced the clinical findings and indicated that the dissociation between rotation and strain was caused by mechanical interactions between infarcted and viable myocardium. Systolic strain reflects regional myocardial function and infarct extent, whereas systolic rotation defines infarct borders in the LV apical region. Regional rotation, however, has limited ability to quantify regional myocardial dysfunction.

myocardial infarction; myocardial strain; left ventricular function; left ventricular torsion

NORMAL LEFT VENTRICULAR (LV) systolic function is the result of coordinated contraction of myocardial fibers of different orientations. The longitudinal component of fiber shortening causes the LV base to descend toward the apex, while the circumferential component causes reduction of LV short-axis diameter, with both mechanisms contributing to systolic wall thickening. In addition, due to contraction of obliquely oriented fibers, there is a torsional deformation (twist) of the LV about its long axis. In the subepicardium, the oblique fibers course toward the base in a counterclockwise spiral (viewed from the apex), and in the subendocardium they form an oppositely directed spiral. Because of larger radii and mass, the subepicardial fibers dominate and account for the normal LV apex-to-base rotation gradient, including counterclockwise rotation of the LV apex and clockwise rotation of the base.

In clinical practice, global LV function is commonly evaluated by measuring ejection fraction and regional function by visual assessment of wall thickening. More recently, myocardial velocity and strain have been introduced as supplementary quantitative indexes of LV longitudinal and radial function. Assessment of LV rotation, however, has not been incorporated into the clinical assessment of LV function. Since LV rotation reflects a fundamental property of normal LV function, it has been proposed that LV rotation may serve as a clinical marker of impaired LV function (12, 14, 19, 22, 23, 28). Because measurement of LV rotation has been possible only with tagged-magnetic resonance imaging (MRI) and other complex methodologies, there is limited insight into how this parameter can be applied clinically. The recent introduction of speckle tracking echocardiography (STE) as a method to quantify LV rotation represents an important breakthrough that has brought LV rotation into the clinical arena (1, 14, 18, 22, 28). This study investigates how this novel methodology may be applied for a detailed quantification of regional myocardial function in the infarcted left ventricle.

In patients with previous myocardial infarction, circumferential distribution of LV apical rotation and strain were measured by STE and MRI tagging, and myocardial infarct extent by late enhancement (LE) MRI. We observed a striking dissociation between regional rotation and strain within the infarct, in its border zones, and in remote segments; i.e., in contrast to systolic strain, which corresponded to regional transmural infarct extent, rotation was similar in the center of the infarct and in remote myocardium. Furthermore, maximum and minimum rotation was localized at the two border zones. We hypothesized that this dissociation between regional rotation and strain was due to mechanical interactions between infarcted and viable myocardium. To investigate the specific mechanisms that may be involved, we compared patients with myocardial infarction to healthy individuals. In addition, we utilized a mathematical finite-element model, which incorporated the essential features of LV function, including obliquely oriented fibers, to produce LV rotation. Myocardial infarction was simulated as a region with reduced active fiber force.
MATERIALS AND METHODS

Fifteen healthy subjects (7 women; mean age 34 ± 7 yr) and 23 patients with reperfused chronic myocardial infarction (9 women; mean age 60 ± 8 yr) were included. The recordings were selected from our database of patients reexamined 6 mo after primary percutaneous coronary intervention. All patients with single-artery disease and echocardiographic short-axis recordings of the LV apex obtained from the apical window were included. According to angiographic findings, 18 patients had left anterior descending (LAD) coronary artery occlusion, 3 had circumflex coronary artery occlusion, and 2 had right coronary artery occlusion. All subjects gave written, informed consent to participate in the study. The protocol was approved by the Regional Ethics Committee.

MRI and echocardiographic recordings. MRI scans were performed in healthy subjects and patients using a 1.5-T scanner (Magnetom Vision Plus, Siemens, Erlangen, Germany). Multiple LV cine and tagging images were obtained with a time resolution of 35 ms at apical, midventricular, and basal short-axis levels, as previously described (14). In patients, multiple short-axis LE images covering the entire LV with a slice thickness of 7 mm and an interslice gap of 3 mm were obtained ~10–20 min after intravenous injection of 0.1 mmol/kg gadopentetate dimeglumine (Magnevist, Schering, Berlin, Germany).

Using a Vivid 7 scanner (GE Vingmed Ultrasound, Horten, Norway), LV long-axis and apical short-axis STE recordings were obtained during breath holds with the individual in a supine, left lateral position, within 15 min of the MRI examination. To obtain optimal speckle quality at a reproducible and representative apical short-axis level (just proximal to the level with luminal closure at end systole), projection of the LV apex was recorded from a view distal to the conventional parasternal window. An effort was made to make the LV cross sections as circular as possible. Transducer frequencies (1.7–2.0 MHz), sampling rates (60–110 frames/s), focus (midventricular), sector depth (minimal), and sector width (narrow) were adjusted to optimize image quality.

MRI and echocardiographic analysis. Apical, tagged, short-axis MRI images were analyzed quantitatively using the software HARP (Harmonic Phase Imaging, version 1.0, Diagnosoft, Palo Alto, CA) (9). The endo- and epicardial borders were manually drawn, and time-derived measures of myocardial displacement and deformation were automatically calculated for 24 equiangular segments throughout the cardiac cycle. In case of poor tracking during systole, segments were excluded unless readjustment of borders improved tracking quality. Apical rotation was measured in a spatially fixed short-axis imaging plane as the angular displacement of the myocardium in the circumferential direction. Strain in the circumferential direction was measured as the change in myocardial segment length relative to its end-diastolic length. The timing of the frame of minimal LV cavity lumen of the basal tagging sequence was used to determine global end systole. End-systolic rotation and strain were calculated with reference to the first image frame of the cardiac cycle and extracted from all segments. By convention, negative strain indicates myocardial shortening, positive strain myocardial lengthening, while the directions of rotation are defined as counterclockwise (positive) or clockwise (negative), as viewed from apex. In three patients, tagged MRI images could not be analyzed due to poor image quality.

LV rotation and circumferential strain by STE were analyzed offline using dedicated software (EchoPAC, GE Vingmed Ultrasound). For each apical short-axis recording, the endocardial border was manually traced in the frame, where its complete contour was identified the best, and the automatically applied epicardial border was adjusted to cover the myocardium without including the pericardium. After automatic tracking, six regions were created by the program, and tracking quality of each region was scored. In case of inadequate tracking during systole, the endocardial delineation was manually adjusted or drawn in another frame until better tracking was achieved.

In our analyses, no regions had to be excluded. After successful tracking, rotation and strain were automatically calculated with reference to the last image of the previous cardiac cycle (1 frame before peak R of the ECG) for numerous locations evenly distributed along the circumference. The software then allowed local time-derived traces to be displayed by pointing with the cursor at any myocardial location of interest. End systole was determined from aortic valve closure, and end-systolic rotation and strain were finally extracted from the 24 locations (segments) that corresponded to the MRI segments.

The selection of apical LE MRI images, which corresponded to the echocardiographic short-axis recording, was based on LV dimensions (at matching LV intracavitary diameter and LV wall thickness) and anatomic landmarks. Using dedicated software (PACS, Sectra), the epicardial and endocardial borders of the LV were drawn. Areas with pixel intensity >2 SD above the mean pixel intensity of the normal myocardium of the same image were defined as infarcted, and the border between noninfarcted and infarcted myocardium was manually drawn (17). The LV short-axis image was further divided into 24 equiangular segments, corresponding to the tagged MRI and STE analyses, and the infarct extent of each segment was calculated as the percentage of total segment area.

To compare measures by STE and MRI tagging, the 24 segments were grouped into four apical parts of equal size (Fig. 1); an anteroseptal part (segments 19–24), an anterolateral part (segments 1–6), an inferolateral part (segments 7–12), and an inferoseptal part (segments 13–18). The mean value of the six segments constituting each apical part was used for further analyses.

Rotation and strain behavior in a mathematical simulation model of the ischemic LV. A finite-element model of the LV was constructed using the CMISS finite-element analysis program (University of Auckland, Auckland, New Zealand; http://www.cmiss.org). The LV geometry was represented as a thick-walled, truncated ellipsoid applying 48 elements and tricubic Hermite interpolation functions, as shown in Fig. 2A. The model size was similar to an adult dog’s LV with an end-diastolic cavity volume of 47 ml and a wall thickness of 1 cm. The transmurally varying fiber orientation was prescribed in the model (Fig. 2B), including the oblique subendocardial and subepicardial fibers, which are the anatomic basis for LV torsional behavior (25). The passive elastic properties of the myocardial tissue were incorporated in the model through the pole-zero energy function, which accounts for the orthotropic, nonlinear, hyperelastic characteristics of the tissue (21). In addition, the material was modeled as incompressible. Active tension ($T_a$) was added along the fiber direction during systole. Equation 1 shows the formula used to calculate $T_a$, which was a function of the myofiber stretch ratio ($\lambda$) and a time-varying activation parameter ($actn$) related to the intracellular Ca$^{2+}$ concentration (16). Further comprehensive descriptions of the models and the mathematical principles may be found in several other publications (16, 21, 25).

$$T_a(\lambda, actn) = 130\text{ kPa}[1.45(\lambda - 1)] \frac{actn^3}{actn^3 + 0.5}$$

According to a 12-segment apical short-axis model, an anteroseptal infarct was included. Infarcted myocardium is unable to generate active fiber force, whereas scarred tissue increases tissue stiffness, which limits passive stretch (15). In the mathematical model, infarct properties were simulated by abolishing active fiber force and increasing passive stiffness by a factor of 10 in the specified infarct region. To simulate a transmural infarct, infarct tissue properties were assigned to four segments (Fig. 2E, segments 11, 12, 1, and 2) from endocardium to epicardium, with a gradual reduction of the transmural infarct extent in the two adjacent segments on both sides (67 and 33% transmural extent, respectively). To simulate the setting of a nontransmural infarct, infarct properties were assigned to the inner
44% of the LV wall (Fig. 2F) of the same four segments and to the inner 22% of the adjacent segment on both sides.

The three-dimensional deformation of the LV during the cardiac cycle was simulated using physiological pressure-volume boundary conditions (Fig. 2, C and D). Rotation and circumferential strain were extracted from each of the 12 apical segments of the model and related to the location of the infarcted segments.

**Statistical analysis.** Data are presented as means ± SD, unless otherwise stated. We used the unpaired t-test or one-way repeated-measures ANOVA, followed by the Bonferroni correction for predefined comparisons (between healthy myocardial segments in controls and remote, transmural and nontransmural infarct segments in patients; SPSS version 13.01). The rotation measurements obtained by STE and the reference method were compared by a least squares linear regression method and by the Bland-Altman method (3). The relationship between rotation/strain and segmental infarct extent were assessed by the Pearson correlation coefficient. To determine interobserver variability in the measurement of end-systolic segmental rotation and strain by STE, five patients were randomly selected and independently analyzed by a blinded observer. The reproducibility was expressed by the Bland-Altman method and intraclass correlation. Statistical differences were considered significant at \( P < 0.05 \). The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agreed to the paper as written.

**RESULTS**

**Distribution of rotation and strain in healthy subjects.** Figure 1 shows representative traces of rotation and strain by MRI and STE in a healthy subject. A homogenous distribution of apical rotation and strain was demonstrated by STE (Fig. 3, A and B). The average of the individual’s mean difference of segmental rotation and strain was 1.1 ± 0.5° and 2.5 ± 1.6%, respectively. Mean apical rotation and strain for the entire cross-section were 12.9 ± 3.5° and −23.8 ± 2.3%, respectively (Table 1).

**Distribution of rotation and strain in postinfarct patients.** In the subsequent text, we will use the term clockwise infarct border when referring to the infarct border, which is in the clockwise direction of rotation, and counterclockwise infarct border for the other infarct border (Fig. 4). Mean apical rotation and strain for all segments in the apical short axis were significantly lower (\( P < 0.001 \)) in patients than in healthy subjects (Table 1). Figure 4 displays STE data from a single patient and demonstrates that strain was reduced in segments that included infarcted myocardium. Rotation, however, was similar in the center of the infarct and in remote (viable) myocardium. Furthermore, peak rotation was found at the counterclockwise infarct border, and minimum rotation at the clockwise infarct border. Figure 5 displays strain, rotation, and infarct extent from all patients and confirms that peak and minimum rotation values correspond to the anatomic localizations of the border zones (mean distance from segments of maximum and minimum rotation by STE to infarct borders by LE MRI was 2.2 ± 1.7 segments).

Since the segmental distribution of infarcts varied between patients, mean strain and rotation per segment would express a mixture of myocardium with different tissue characteristics and localizations in relation to the infarct. Therefore, we grouped...
segments with reference to the infarct (remote segment, infarct center segment, and the infarcted and noninfarcted segments on both sides of the two infarct borders). These data are displayed in Fig. 3, D–F. The strain analyses showed changes in function that corresponded to localization of infarcted and viable myocardium; i.e., approximately zero strain (no fiber shortening) in the center of the infarct, strain values similar to mean strain of the entire short axis at the two border zones, and normal strain in the remote myocardium. The rotation analyses, however, showed that rotation in the center of the infarct and in the center of remote myocardium were similar to mean rotation of the entire LV cross section. Furthermore, as demonstrated for individual patients, maximum rotation was observed at the counterclockwise infarct border, and minimum rotation at the clockwise infarct border.

The concordance between strain and segmental infarct extent was reflected in a strong correlation between these two variables ($r = 0.80$, $P < 0.0001$; Fig. 6B) and in marked differences in mean strain for segments with 0, 1–50, and >50% infarct extent (Fig. 6D). In contrast, there was no correlation between segmental rotation and infarct extent (Fig. 6, A and C). Measures of rotation and strain by STE were confirmed by MRI ($y = 0.82x + 1.27$, $r = 0.71$, $P < 0.001$, and mean difference $0.6 \pm 3.0^\circ$, and $y = 0.99x + 1.63$, $r = 0.87$, $P < 0.001$, and mean difference $1.5 \pm 4.2\%$, respectively; Fig. 7).

**Interobserver variability.** Measurements by STE of segmental rotation and strain by two independent observers showed mean differences of $0.9 \pm 2.3^\circ$ and $-2.5 \pm 2.8\%$, respectively. The intraclass correlations were $r = 0.95$ and 0.97, respectively.

**Simulation study.** Consistent with the clinical observations, the simulation study demonstrated marked dissociation between segmental strain and rotation (Fig. 8). Furthermore, the simulation study showed depressed strain (systolic lengthening) within the infarct zone, and a gradual normalization when moving from the infarct center through the border zones and toward remote segments (Fig. 8A, middle). Rotation, however, was highest at the counterclockwise infarct border and lowest at the opposite border (Fig. 8A, top). Rotation in the center of the infarct zone and in the center of the remote zone were similar and lower than in the counterclockwise border zones. In principle, similar distributions of strain and rotation were observed when a non-transmural infarct was simulated, i.e., corresponding to the reduction in circumferential infarct extent (Fig. 8A, bottom), the locations of maximum and minimum rotations were altered (Fig. 8A, top) and corresponding to the reduction in radial infarct extent, the difference between maximum and minimum rotation was reduced relative to transmural infarction.

To differentiate between the contributions to regional rotation from the circumferential and oblique components of active
myocardial fiber forces, a simulation was done with all LV fibers aligned in the circumferential direction (equivalent to a fiber angle of 0°, i.e., a horizontal line at 0° from endocardium to epicardium in Fig. 2B). Simulation with normal fiber force demonstrated no LV apical rotation. When a regional infarct was introduced, there was no rotation in the center of the infarct or in the center of remote myocardium, but oppositely directed rotations on each side of the infarct center (Fig. 8B). Maximum and minimum rotations were found at the counterclockwise and clockwise infarct borders, respectively. Because the oblique contraction vector was abolished and all oblique fibers were set with a circumferential vector only, circumferential strain was increased in viable myocardium to maintain stroke volume.

**DISCUSSION**

The present study demonstrates that regional LV apical rotation and strain reflects entirely different features of infarcted myocardium. Reductions in end-systolic strain in postinfarct patients corresponded to transmural extent of infarcted myocardium, as evidenced by a strong correlation between systolic strain and percent infarction within segments. These observations are in keeping with previous studies and confirm that systolic strain reflects regional contractile function (2, 5–7, 10). This was in contrast to regional rotation, which did not reflect regional contractility. Maximum and minimum values of regional rotation, however, were markers of the anatomic locations of the infarct borders. The simulation study reproduced the clinical findings and provided insights into the mechanism of the myocardial infarction.

Table 1. Average and segmental rotation and strain by echocardiography and infarct extent by MRI in healthy subjects and patients

<table>
<thead>
<tr>
<th></th>
<th>Rotation,°</th>
<th>Strain, %</th>
<th>Infarct, %</th>
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<tbody>
<tr>
<td>Healthy subjects (n = 15)</td>
<td>12.9 ± 3.5</td>
<td>23.8 ± 2.3</td>
<td>23.8 ± 2.0</td>
</tr>
<tr>
<td>Patients (n = 23)</td>
<td>9.0 ± 4.9*</td>
<td>23.9 ± 10.7*</td>
<td>30 ± 35</td>
</tr>
<tr>
<td>Remote myocardium</td>
<td>9.1 ± 3.9</td>
<td>23.9 ± 4.5</td>
<td>0 ± 0</td>
</tr>
<tr>
<td>Infarct center</td>
<td>9.3 ± 4.3</td>
<td>3.0 ± 9.6</td>
<td>72 ± 16</td>
</tr>
<tr>
<td>Counterclockwise border of infarcted myocardium</td>
<td>12.9 ± 4.2</td>
<td>15.0 ± 6.2</td>
<td>19 ± 14</td>
</tr>
<tr>
<td>Counterclockwise border of noninfarcted myocardium</td>
<td>12.6 ± 4.1</td>
<td>17.5 ± 6.1</td>
<td>0 ± 0</td>
</tr>
<tr>
<td>Clockwise border of infarcted myocardium</td>
<td>5.0 ± 3.5</td>
<td>13.8 ± 5.7</td>
<td>28 ± 18</td>
</tr>
<tr>
<td>Clockwise border of noninfarcted myocardium</td>
<td>5.2 ± 3.4</td>
<td>17.5 ± 6.2</td>
<td>0 ± 0</td>
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Values are means ± SD. The infarct area is in percentage of total segment area. *P < 0.001 vs. healthy subjects.
apparent inconsistency between regional apical rotation and strain.

Mechanism of dissociation between regional rotation and strain. To account for these novel observations, we propose the following mechanism: LV torsion is caused by contraction of the oblique myocardial fibers that course from base toward apex. Rotation of a given apical short-axis plane is, therefore, essentially caused by forces external to the plane. Rotation of a myocardial region within the short-axis plane reflects the circumferential displacement of the region. Therefore, the balance of contractile forces acting in the circumferential direction of the plane will determine the distribution of regional rotation. To isolate the contribution to regional rotation and strain from the distribution of circumferential contractile forces, we used a simulation model, where the oblique component of external forces was removed by aligning all LV fibers in the circumferential direction. In the setting of uniform circumferential fiber force, no rotation was observed, and the distribution of strain was homogeneous. This supports the notion that, in the healthy LV, external forces cause the overall rotation of a plane, whereas the in-plane balance of active circumferential forces causes homogeneous distribution of strain and rotation. When an infarct was introduced in this model, regional myocardial strain and rotation showed entirely different distributions (Fig. 8B): similar to our clinical findings, regional strain was markedly reduced within the infarct zone, normal in remote myocardium, whereas oppositely directed rotations were observed at the two infarct borders. There was no overall rotation of the short-axis plane, and the center of the infarct and the center of the remote myocardium did not rotate. These findings can be attributed to mechanical in-plane interaction between ischemic and nonischemic myocardium: due to the imbalance in active forces, the weaker infarct region was pulled from both sides by the stronger, nonischemic myocardium. As a result, the counterclockwise infarct border was pulled in the counterclockwise direction, whereas the clockwise infarct border was pulled in the clockwise direction, causing oppositely directed rotations (Fig. 9, A and B). Furthermore, due to the stretching forces of equal magnitude acting on opposite sides of the infarct, the infarct center and the center of the remote myocardium were not displaced in the circumferential direction. However, when external forces (ob-
liquely oriented myocardial fibers) were included in the model, the entire LV apical cross-sectional plane rotated in the counterclockwise direction (Fig. 9, C and D). Because in-plane forces and external forces both enhanced counterclockwise rotation of the counterclockwise infarct border, this region represented myocardium of hyperrotation. Conversely, because the in-plane forces enhanced rotation of the clockwise infarct border in the opposite direction of overall rotation, this region represented myocardium of hyporotation. Since the infarct center and the center of remote myocardium were set into rotation by the external forces, the magnitude of rotation of these points was similar to mean rotation of the short-axis plane, which was in accordance with the clinical results.

Although the simulation model was highly simplified compared with the real LV, it provided a controlled environment where all parameters and input conditions were known. Furthermore, it provided support for our proposed notion of how mechanical interaction contributes to the distribution of regional rotation and strain in the healthy and ischemic LV.

**Comparison with previous studies.** Several studies have confirmed that ischemia reduces LV rotation, but no studies have investigated the interaction between regional shortening and rotation in the ischemic ventricle. In an experimental MRI tagging study by Buchalter et al. (4), the relationship between distribution of LV torsion and ischemia was examined. Local torsion was measured as the rotational difference between corresponding apical and basal locations before and during LAD occlusion. Local torsion was further grouped into two LV sections, an anterior and a posterior, with only the former showing reduced torsion during ischemia.

Because LAD occlusion primarily affects the anterior LV wall, the observations made by Buchalter et al. (4) of reduced

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**Fig. 5.** Individual patient data showing segmental distribution of end-systolic rotation, end-systolic strain, and infarct extent. Maximum and minimum rotations are indicated by vertical dotted lines and segment numbers. Please note that maximum rotation occurs at the counterclockwise infarct border (to the left), and minimum rotation at the opposite infarct border. Patients 1–18 had LAD coronary artery occlusion, patients 19–21 circumflex coronary artery occlusion, and patients 22 and 23 right coronary artery occlusion.
torsion of the anterior section during ischemia can be interpreted as inconsistent with our results. However, according to the depiction of the distribution of apical ischemia in each animal in their study, the anterior and posterior sections did not represent strictly ischemic and nonischemic myocardium, respectively: in all animals, the lateral nonischemic border zone, which in our study represents myocardium of hyporotation, was located in the anterior (“ischemic”) section, while the septal ischemic border zone, which in our study represents myocardium of hyperrotation, was located in the posterior (“nonischemic”) section. Consequently, the apparent discrepancies between our observations and the findings by Buchalter et al. could be explained by their inclusion of nonischemic myocardium of hyporotation in the anterior LV section, and ischemic myocardium of hyperrotation in the posterior LV section.

Buchalter et al. (4) studied LV torsion while we studied apical rotation. Several clinical and experimental studies have demonstrated that, in the setting of acute or chronic LAD-related ischemia, minimal changes occur in LV rotation at a nonischemic basal short-axis level (11, 20, 24, 27). Since LV apical rotation represents the dominant contribution to LV

Fig. 6. Correlation between segmental infarct extent and end-systolic rotation (A) and end-systolic strain (B) by STE. While infarct extent is inversely related to a decrease in absolute strain (D), no such relationship exists for rotation (C). Infarct extent is expressed as the ratio between infarct area and total segment area by LE MRI.

Fig. 7. Comparisons between STE and MRI tagging. Shown are correlations and agreements between regional rotation (A and C) and strain (B and D) measured by MRI and STE. The mean difference between methods and ±2 SD are indicated.
torsion (24), there are strong reasons to believe that assessment of LV apical rotation essentially captures the features of LV torsional deformation.

Clinical implications. The present study demonstrates that quantitative assessment of LV apical strain and rotation by STE in patients with myocardial infarction provides different and, in principle, complementary diagnostic information. However, demonstration of potential added clinical value of combined measurement of rotation and strain compared with measuring strain only requires a study in a larger patient population.

We believe that a potential impact of the present study may be to provide better understanding of how to interpret measurement of regional apical rotation in a clinical context. The observations that rotation was maintained in the center of the infarct and was lowest just outside the infarct could easily lead to misinterpretation with regard to infarct localization. Due to mismatch between impaired rotation and infarct localization, it is unlikely that assessment of regional rotation can serve as a standalone method to quantify myocardial function. In principle, assessment of turning points of rotation may provide a means to monitor changes in extension of myocardial infarctions. As suggested by the findings in the finite element model, it is reduction in active fiber force and not infarction as such that determines locations of maximum and minimum values of rotation. Therefore, it is likely that myocardial stunning as well as infarction determine the locations of turning points of rotation. This, in turn, implies that the clinical potential of combined assessment of myocardial rotation and strain would be to define extension of dysfunction rather than extension of infarction.

In our study, most patients had LAD-related infarctions. The simulations in the finite-element model, however, made no assumptions with regard to coronary anatomy, supporting that the principle findings of this study should apply to different infarct localizations.

Fig. 9. Schematic representations of the LV viewed from the apex toward the base. Ischemic myocardium (gray area), direction of rotation (open arrows), circumferential forces acting on the ischemic region (solid arrows), and ischemic margins at onset of systole (dotted lines) are indicated. A and B: rotational deformation of an apical short-axis slice in an LV consisting of circumferential fibers only. The center of the ischemic region remains stationary. C and D: rotational deformation of an apical short-axis slice in an LV consisting of circumferential and oblique myocardial fibers. The oblique fibers cause torsion of the LV with oppositely directed apical and basal rotation. Furthermore, because both border zones are being “pulled” in opposite directions by contractile forces in the circumferential direction (as in B), the border zone in the direction of overall rotation exhibits hyperrotation, while the opposite border zone exhibits hyporotation.

Fig. 8. A: simulation study with normal LV fiber orientation (●, transmural infarction; X, nontransmural infarction). Strain traces within the infarct zone (segments 11, 12, 1, and 2) demonstrate dyskinesis with transmural infarction and akinesis with nontransmural infarction. Segmental rotation shows maximum value at the counterclockwise border zone (8-10) and minimum value at the clockwise border zone (3-5). In the centers of the infarcted (12 and 1) and remote (6 and 7) areas, there are rotations of similar and intermediate magnitudes. The distribution of strain and rotation was qualitatively similar for transmural and nontransmural infarctions. B: simulation study with circumferential fibers only. When all oblique LV myocardial fibers were replaced by circumferential fibers, there was no net rotation of the entire short-axis plane. The center of the infarct (12 and 1) and the remote myocardium (6 and 7) did not rotate. Similar to simulation with oblique fibers (A), the turning points of local rotation were found in the border zones of the infarct (8 and 9 and 4 and 5).
LIMITATIONS. The patient population in the present study is small and included mostly patients with LAD-related infarctions. The main focus, however, was on the mechanism of interactions between actively contracting and infarcted myocardium. To determine the clinical applicability of our findings, a larger study is needed that should include patients with a wider range of myocardial dysfunction and infarct localizations. The difference in age between the healthy subjects and patient groups in our study is substantial. However, with respect to LV apical strain and rotation, studies have indicated that aging is not related to significant systolic changes in their distribution (8, 26).

A fundamental problem with LV short-axis imaging is longitudinal motion of the LV through the image plane. This problem reflects normal LV long-axis motion, which includes movement of the LV base toward the apex in systole and reverse movement in diastole, while the apex shows little longitudinal motion. Therefore, with apical short-axis imaging, the problem with out-of-plane motion is minimized. In addition, the apical window used in the present study provides better acoustic condition than the conventional parasternal window. Therefore, the LV apex provides optimal conditions for short-axis imaging.

In the present study, analyses of regional myocardial rotation, strain, and infarction were limited to the LV apex. This section of the LV was chosen because it represents the dominant contribution to LV torsion and because distribution of myocardial rotation is less uniform in more basal cross sections of the normal ventricle, which complicates the interpretation (13, 24).

Given the mechanical complexity of three-dimensional myocardial deformation and interaction, a more complete approach would be to include measures of regional shear strain and torsion in our analysis. In principle, such measures can be estimated by combining analyses from multiple two-dimensional cross-sectional recordings. By current echocardiographic techniques, this method is challenging, particularly with respect to determining the distance between slices, but also to our approach of detailed strain and rotation mapping. However, given the recent advances in three-dimensional echocardiographic imaging, it is likely that simultaneous calculation of regional rotation, strain, shear strain, and torsion will be feasible in the near future.

CONCLUSION. STE allows combined assessment of LV strain and rotation, indexes that reflect fundamental characteristics of myocardial function. As demonstrated in the present study, a close association exists between regional strain and infarct transmurality and between locations of maximum and minimum rotation and infarct borders in the LV apex. Myocardial rotation, however, does not provide a site-specific measure of regional function. Combined assessment of rotation and strain by STE may have the potential to become a clinical, noninvasive tool for detection and quantification of LV ischemia.

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