Different impacts of acute myocardial infarction on left ventricular apical and basal rotation

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Aims
The impacts of acute myocardial infarction (AMI) with different regional wall motion abnormalities on left ventricular (LV) rotation have not been well investigated. We assessed the impacts of AMI on LV rotational mechanics and to compare the alterations in basal and apical rotation between patients with anterior and inferior AMI.

Methods and results
Thirty-five patients with anterior AMI and 31 patients with inferior AMI who had a single culprit lesion were analysed. Thirty age-matched subjects were included for controls. The apical and basal rotations were obtained and LV twist and torsion were measured by two-dimensional speckle tracking imaging. Compared with normal, LV twist was reduced in all AMI patients. The basal rotation was larger in anterior AMI than in inferior AMI and normal (29.0 ± 2.6° vs. 23.4 ± 2.1° and −6.0 ± 1.9°, P < 0.001), although the apical rotation was lower in anterior AMI. As a result, LV twist and torsion were not different between anterior AMI and inferior AMI (17.0 ± 4.6 vs. 16.7 ± 3.3° and 2.08 ± 0.59 vs. 2.07 ± 0.44°/cm, P = NS, respectively), although LV ejection fraction was lower in anterior AMI. By multivariate analysis, LV torsion [odds ratio (OR) = 0.13, 95% confidential interval (CI) = 0.02–0.75, P = 0.02] and basal rotation (OR = 0.67, 95% CI = 0.45–1.00, P = 0.05) were independently related to LV recovery in patients with anterior AMI and in patients with inferior AMI, respectively.

Conclusion
Although LV twist and torsion were decreased either by reduced apical and basal rotation in AMI patients; the basal rotation was rather increased in anterior AMI. LV functional recovery can be predicted by LV torsion in anterior AMI and by basal rotation in inferior AMI. The basal rotation has often been ignored; however, our findings suggest that the basal rotation has an important role in LV function.

Keywords
Myocardial infarction • Left ventricle • Rotation • Twist

Introduction
Cardiac rotation during systole is an important component of left ventricular (LV) function.

The obliquely oriented LV muscle fibres from a right-hand helix in the subendocardium to a left-hand helix in the subepicardium generate a wringing motion.1,2 Normally, the apex rotates counterclockwise, whereas the base rotates clockwise when viewed from the apex during systole. LV twist is this net rotational difference between the LV apex and base and induces a uniform distribution of LV fibre stress and fibre shortening across the wall.

It has been reported that acute myocardial ischaemia and infarction are responsible for a decrease in LV twist that is related to global ventricular function in animals and humans.3,4 The myocardium is composed of multiple oriented myocardial fibres and layers, and may cause different myocardial rotational changes with different regional mechanical disturbances. An anterior myocardial infarction (MI) is accompanied by regional wall motion abnormalities, predominantly in the LV apex, and hypercontractile LV basal walls are frequently observed. In contrast, an inferior MI is accompanied by regional wall motion abnormalities, predominantly in the LV base.

However, the impacts of acute myocardial infarction (AMI) with different regional wall motion abnormalities on LV twisting have not been well investigated. Moreover, basal rotation was often ignored in earlier studies because the absolute value is

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small and simple measurement of apical rotation only can be used for LV twist. However, the basal rotation may be essential to generate LV twist and may contribute significantly to net twist motion.

Therefore, we sought to evaluate the impacts of AMI on LV rotation and twisting and to compare the alterations in apical and basal rotational motion between patients with anterior and inferior AMI.

Methods

Study subjects

Consecutive patients were prospectively enrolled who were admitted with a first AMI. The inclusion criteria were as follows: (i) ST-elevation MI on electrocardiography (diagnosis based on standard criteria); (ii) successful primary percutaneous coronary intervention on a single infarct-related coronary artery, defined as residual stenosis <30% and thrombolysis in MI (TIMI) flow grade ≥2, performed within 12 h from the onset of symptoms or between 12 and 24 h if chest pain persisted; (iii) no significant stenosis of other coronary arteries (<50% stenosis); and (iv) presence of akinetic walls with more than two contiguous segments in one wall corresponding to infarct-related coronary artery on conventional two-dimensional echocardiography (2DE). Patients with significant arrhythmia, valvular heart disease, or cardiogenic shock requiring mechanical ventilation or receiving inotropic agents or haemodynamic support with an infarct-related coronary artery on conventional two-dimensional echocardiography (2DE). Patients with significant arrhythmia, valvular heart disease, or cardiogenic shock requiring mechanical ventilation or receiving inotropic agents or haemodynamic support with an infarct-related coronary artery on conventional two-dimensional echocardiography (2DE).

As a control group, we included 30 healthy age- and gender-matched subjects who had no history of cardiovascular disease and normal resting electrocardiographic and echocardiographic results. All patients gave informed consent, and study approval was obtained from the Institutional Review Board of the Korea University College of Medicine.

Echocardiographic study

All echocardiography for conventional and speckle tracking imaging was performed using a standard commercial system (Vivid 7; GE Medical Systems, Horten, Norway) with an M3S probe done within 24 h after primary revascularization.

LV volume and ejection fraction were calculated by biplane Simpson’s method from the apical four- and two-chamber views. Early diastolic mitral inflow velocity (E) was obtained by pulsed wave Doppler echocardiography with the sample volume between mitral leaflet tips during diastole, and early diastolic mitral annular velocity (e’) was obtained from the septal annulus by tissue Doppler imaging. LV regional wall motion analysis was performed with the calculation of the wall motion score (WMS) index according to the American Society of Echocardiography 16-segment model on a 1–4 scale, as follows: 1 = normal; 2 = hypokinesia; 3 = akinesia; 4 = dyskinesia; and x = assessment not possible.

Functional recovery was considered to be functional on the 6-month follow-up 2DE if the patient’s wall motion improved in at least two contiguous dysfunctional segments by >1 grade.

Two-dimensional speckle tracking imaging analysis

Parasternal basal and apical short-axis views, as well as three standard apical views, were acquired. All greyscale images were obtained at a frame rate of $77 \pm 6$ frames/s (range: 63–99 frames/s) using second harmonic imaging. While acquiring images, the LV basal short-axis view was identified by the presence of mitral leaflets, while excluding the mitral annulus and the apical view was identified by the presence of a LV cavity in the absence of papillary muscles. We made every effort to obtain the LV cross-section as circular as possible. For each view, three consecutive cardiac cycles were acquired during a breath hold and digitally stored on a magneto-optical disc for offline analysis. Measurement of strain and rotational parameters was performed offline using dedicated software (Echopac PC, version 10.0; GE Medical Systems).

LV global longitudinal strain (GLS, %) was calculated as the mean longitudinal peak negative strain from 18 apical segments during a cardiac cycle. Basal and apical LV radial and circumferential peak strain (RS and CS, %, respectively) were defined as the mean strain from the six basal and four apical parasternal short-axis segments, respectively.

Rotation (degrees) was obtained at the basal level and the LV apex. A region of interest was manually adjusted to include the entire myocardial thickness; care was taken to avoid including the pericardium in the region of interest. The software then selected stable speckles within the myocardium and tracked these speckles frame-by-frame throughout the cardiac cycle. Counterclockwise rotation was marked as a negative value and clockwise rotation as a positive value when viewed from the LV apex. LV twist was defined as the net difference (in degrees) of apical and basal rotations at isochronal time points and was autocomputed by the software from the values of the basal and apical rotation (Figure 1). LV torsion was then calculated as the ratio between LV twist (in degrees) and the LV diastolic longitudinal length (in cm) between the LV apex and the mitral plane. A pulse wave Doppler tracing obtained from the LV outflow tract was used to identify the timing of aortic valve opening and closure.

Statistical analysis

Values are expressed as the mean ± SD, or as percentages. Comparisons between groups were made using the χ² test for categorical variables and Student’s t-test for continuous variables, using standard statistical software (SPSS, version 12.0; SPSS Inc., Chicago, IL, USA). Differences in continuous variables between two groups were assessed by one-way analysis of variance test. A P-value of <0.05 was considered statistically significant.

To analyse independent predictors of LV recovery, univariate factors with a P < 0.1 were analysed using forward stepwise logistic regression (multivariate analysis). Based on receiver-operating characteristic (ROC) curves, the best cut-off value was obtained as the optimal point with the highest sum of sensitivity and specificity for predicting LV recovery. Ten patients were randomly selected for assessment of intra- and inter-observer variability, which were tested by the Bland–Altman method and expressed as a coefficient of variation and the mean ± SD of the absolute differences between the two measurements divided by the mean value (%).

Results

Clinical and echocardiographic characteristics

Thirty-eight consecutive patients with anterior AMI and 34 patients with inferior AMI were enrolled. Three patients in each group were excluded because their images were not suitable for measurement. There were no differences in age, heart rate, the prevalence of diabetes mellitus, hypertension, and dyslipidaemia, and medication between patients with anterior and inferior AMI (Table 1).
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The LV volume and WMS index were larger in patients with anterior AMI than in patients with inferior AMI. The LV ejection fraction and LV GLS were lower in patients with anterior AMI than in patients with inferior AMI (Table 2). In patients with anterior AMI, the LV ejection fraction was lower ($P = 0.05$) and the WMS index was higher ($P = 0.01$) than in patients with inferior AMI. The $e''$ velocity tended to be smaller in patients with anterior AMI than in patients with inferior AMI ($P = 0.078$).

Table 1 Clinical characteristics of study subjects

<table>
<thead>
<tr>
<th></th>
<th>Normal controls (n = 30)</th>
<th>Anterior AMI (n = 35)</th>
<th>Inferior AMI (n = 31)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>55 ± 8</td>
<td>58 ± 15</td>
<td>58 ± 9</td>
<td>0.96</td>
</tr>
<tr>
<td>Men/women</td>
<td>21/9</td>
<td>28/7</td>
<td>26/5</td>
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<td>Heart rate (/min)</td>
<td>66 ± 14</td>
<td>72 ± 11</td>
<td>68 ± 12</td>
<td>0.24</td>
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<tr>
<td>Diabetes (n, %)</td>
<td>0</td>
<td>8 (22.9%)</td>
<td>8 (25.8%)</td>
<td>0.79</td>
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<tr>
<td>Hypertension (n, %)</td>
<td>0</td>
<td>13 (37.1%)</td>
<td>8 (25.8%)</td>
<td>0.33</td>
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<tr>
<td>Lipidaemia (n, %)</td>
<td>0</td>
<td>6 (17.1%)</td>
<td>8 (25.8%)</td>
<td>0.14</td>
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<tr>
<td>Peak CK-MB (ng/mL)</td>
<td>–</td>
<td>234 ± 211</td>
<td>136 ± 88</td>
<td>0.03</td>
</tr>
<tr>
<td>Medications</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium antagonists (n, %)</td>
<td>0 1 (2.9%)</td>
<td>0</td>
<td>0.32</td>
<td></td>
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<tr>
<td>Beta blockers (n, %)</td>
<td>0</td>
<td>31 (88.6%)</td>
<td>24 (77.4%)</td>
<td>0.23</td>
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<tr>
<td>ACEI/ARB (n, %)</td>
<td>0</td>
<td>29 (82.9%)</td>
<td>25 (80.6%)</td>
<td>0.82</td>
</tr>
<tr>
<td>Diuretics (n, %)</td>
<td>0</td>
<td>5 (14.3%)</td>
<td>1 (3.2%)</td>
<td>0.12</td>
</tr>
<tr>
<td>Nitrates (n, %)</td>
<td>0</td>
<td>12 (34.3%)</td>
<td>6 (19.4%)</td>
<td>0.18</td>
</tr>
</tbody>
</table>

AMI, acute myocardial infarction; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin II receptor blocker.

*P, patients with anterior AMI vs. patients with inferior AMI.

In patients with anterior AMI, LV GLS, apical RS and CS were significantly decreased, but basal RS and CS were not decreased or rather increased. In patients with inferior AMI, all parameters except the apical CS were decreased (Table 2).

Impact of acute myocardial infarction on left ventricular Rotation and LV twist

The apical rotation and LV twist were decreased in all AMI patients compared with controls (Table 2). LV twist was related to apical rotation ($r = 0.75, P < 0.001$), basal rotation ($r = 0.20, P = 0.05$), LV end-diastolic volume index ($r = -0.22, P = 0.03$), LV end-systolic volume index ($r = 0.33, P = 0.001$), LV ejection fraction ($r = 0.47, P < 0.001$), WMS index ($r = -0.52, P < 0.001$), and LV GLS ($r = 0.47, P < 0.001$). Multiple regression analysis showed that the apical rotation was the strongest determinant of LV twist ($r^2 = 0.56, P < 0.001$). There was a significant negative correlation between the apical rotation and basal rotation ($r = -0.589, P < 0.001$) in all AMI patients.

The apical rotation was lower in patients with anterior AMI than in patients with inferior AMI ($8.8 \pm 5.7$ vs. $12.9 \pm 3.6$, $P < 0.001$). In contrast, basal rotation was higher in patients with anterior AMI than in normal controls ($-9.0 \pm 2.6$ vs. $-6.0 \pm 1.9$, $P < 0.001$) and in patients with inferior AMI ($-9.0 \pm 2.6$ vs. $-3.4 \pm 2.1$, $P < 0.001$). As a result, LV twist was not significantly different between anterior and inferior AMI (15.8 ± 5.0 vs. 15.3 ± 5.0, $P = 0.81$, Figure 2), and LV torsion was not significantly different between anterior and inferior AMI (2.08 ± 0.59 vs. 2.07 ± 0.44/°/cm, $P = 0.94$), neither.

Follow-up in 6 months and prediction of functional recovery

Twenty of 35 patients with anterior AMI and 19 of 32 patients with inferior AMI showed a functional recovery in 6 months (Table 3). AMI patients with recovery showed increased LV twist and...
normal and the basal rotation was also decreased to the normal function was recovered, the basal function was returned to the torsion to that of normal. In anterior AMI, as the apical wall function was recovered, the basal function was returned to the normal and the basal rotation was also decreased to the normal ($-6.5 \pm 2.4^\circ$). However, in patients with no recovery, the apical rotation and LV twist were more decreased, but the basal rotation was still high ($-7.8 \pm 1.9^\circ$, Table 3).

In patients with anterior AMI, by univariate analysis, several variables were significantly related to LV recovery: LV ejection fraction, DT, LV twist, torsion, and apical rotation (Table 4). However, by multivariate analysis, only LV torsion ($\beta = 2.06$, odds ratio (OR) = 0.13, 95% confidential interval (CI) = 0.02–0.75, $P = 0.02$) and DT ($\beta = 0.02$, OR = 0.98, 95% CI = 0.96–1.00, $P = 0.05$) were independently associated with LV recovery. Based on ROC curve analysis, the sensitivity and specificity for prediction of LV recovery were 100.0 and 50.0% [area under the ROC curve (AUC) = 0.778, 95% CI = 0.606–0.900], using a cut-off value of LV torsion >1.54°/cm.

On the other hand, in patients with inferior AMI, only basal rotation was related to LV recovery. Based on ROC curve analysis, the sensitivity and specificity for prediction of LV recovery were 76.5 and 71.4% (AUC = 0.723, 95% CI = 0.533–0.867) using a cut-off value of LV torsion >3.09°.

**Measurement variability**

Since the LV twist was automatically derived from measured basal and apical rotation by Echopac PC software, we conducted a variability test with basal and apical rotation. The inter- and intra-observer correlations for basal rotation were 0.91 and 0.95 ($P < 0.001$), respectively, and the variability was 9.0 ± 6.1 and

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Baseline echocardiographic parameters</th>
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<tbody>
<tr>
<td></td>
<td>Normal controls ($n = 30$)</td>
</tr>
<tr>
<td>LVEDV index (mL/m²)</td>
<td>48.0 ± 7.2</td>
</tr>
<tr>
<td>LVESV index (mL/m²)</td>
<td>18.4 ± 3.0</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>63.7 ± 4.6</td>
</tr>
<tr>
<td>LA V index (mL/m²)</td>
<td>23.4 ± 6.5</td>
</tr>
<tr>
<td>E velocity (cm/s)</td>
<td>63.9 ± 16.9</td>
</tr>
<tr>
<td>DT (ms)</td>
<td>203.0 ± 38.8</td>
</tr>
<tr>
<td>e’ velocity</td>
<td>7.5 ± 1.6</td>
</tr>
<tr>
<td>E/e’</td>
<td>9.2 ± 4.0</td>
</tr>
<tr>
<td>PA systolic pressure (mmHg)</td>
<td>30.4 ± 3.8</td>
</tr>
<tr>
<td>LV twist (degree)</td>
<td>21.4 ± 4.0</td>
</tr>
<tr>
<td>LV apical rotation (degree)</td>
<td>15.6 ± 3.5</td>
</tr>
</tbody>
</table>

AMI, acute myocardial infarction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LAV, left atrial volume; DT, deceleration time of E velocity; E/e’, the ratio of early diastolic mitral inflow velocity (E) to early diastolic mitral annular velocity (e’); PA, pulmonary artery; WMS, wall motion score; LV GLS, left ventricular global longitudinal strain; LV RS, left ventricular radial strain; LV CS, left ventricular circumferential strain.

*0.05 vs. normal controls.
† P < 0.05 vs. control subjects.
‡ P < 0.05 vs. patients with anterior AMI.
* P = 0.078 compared with patients with anterior AMI.

Figure 2 Different impacts of acute myocardial infarction on left ventricular apical and basal rotation. The apical rotation is significantly lower and basal rotation is significantly higher in anterior acute myocardial infarction than in normal and in inferior acute myocardial infarction. As a result, left ventricular twist is not different between anterior and inferior acute myocardial infarction.

A variability test with basal and apical rotation. The inter- and intra-observer correlations for basal rotation were 0.91 and 0.95 ($P < 0.001$), respectively, and the variability was 9.0 ± 6.1 and
6.7 ± 6.6%, respectively. The inter- and intra-observer correlations for apical rotation were 0.94 and 0.98 (P < 0.001), respectively, and the variability was 7.0 ± 3.8 and 5.8 ± 2.7%, respectively.

**Discussion**

There have been little data about the impacts of AMI on LV rotation and twist. The purpose of this study was to assess the alteration in LV rotational characteristics by regional myocardial wall motion abnormalities after ST elevation AMI.

Cardiac rotation around the long-axis is an important component of LV systolic function. LV twist is the net rotational difference between the LV apex and base and the wringing motion of the heart imparted by contraction of its oblique spiral fibres. Cardiac rotation around the long-axis is an important component of LV systolic function. LV twist is the net rotational difference between the LV apex and base and the wringing motion of the heart imparted by contraction of its oblique spiral fibres. Cardiac rotation around the long-axis is an important component of LV systolic function. LV twist is the net rotational difference between the LV apex and base and the wringing motion of the heart imparted by contraction of its oblique spiral fibres.

LV twist is related to myocardial contractility and structure and has recently been recognized as a sensitive indicator of cardiac performance. It has been shown that myocardial ischaemia and MI affect the transmural balance of LV torsional movements, thereby disturbing the LV systolic performance.
In our study, LVt was related to WMS index, LV ejection fraction, and LV volume and the apical rotation was more related to LV twist than the basal rotation. These findings were similar with other previous studies. The apical rotation is large, and in previous studies the simple measurement of apical rotation only can be used for LV twist. In contrast, the basal rotation has often been ignored because the absolute value is small and is less related to the LV ejection fraction. Ignoring basal motion may result in a more straightforward analysis, but may be an oversimplification of complex cardiac dynamics. The basal rotation may be essential to generate LV twist and may contribute significantly to net twist motion.

In this study, the LVt was significantly reduced in patients with AMI, but was not different between patients with anterior AMI and inferior AMI even though the LV ejection fraction and WMS index were worse in patients with anterior AMI. The LV basal rotation was significantly increased in patients with anterior AMI.

There are several possible mechanisms for this finding. First, insofar as apical and basal rotations are in opposite directions, there has to be an equatorial level at which the rotation changes from one direction to the other. Because the LV basal radius is larger than the LV apical radius, the base may rotate less than the apex and may have a reserve to rotate more for compensation. Moreover, the opposite power was eliminated by akinetic apical wall and the basal rotation can be exaggerated.

Second, it has been known that the hypercontraction of LV basal walls is frequently observed in anterior AMI. LV basal hypercontractility is presumably compensatory hyperkinesia of the normally perfused residual myocardium proximal to the coronary occlusion in anterior MI. Therefore, our result can be explained by this mechanism. However, this compensatory hyperkinesia is usually found in AMI with single-vessel coronary artery disease, since multivessel disease could prevent the development of baseline hyperkinesia.

In addition, the fibres in the anterior wall endocardium may be more numerous or oriented in a way that more strongly opposes the rotational direction imposed by the epicardium, resulting in less rotation and the LVt in anterior MI can be compensated by the rotation of other posteroinferior walls. In contrast, in inferior MI, there was no increase of the apical wall function and LV twist is similar with that of anterior MI although LV ejection fraction and apical rotation is better.

Bansal et al. studied the effects of prior MI with normal LV systolic function on LV twist and they also found LV twist was equally compromised in 13 patients with anteroseptal MI and in 16 patients with inferoposterior MI, but there were no differences in the LV ejection fraction and WMS index between the two groups and they did n’t confirmed MI or ischaemia on coronary angiography.

In contrast, we studied patients with AMI who had primary percutaneous coronary intervention on a single infarct-related coronary artery and LV ejection fraction was significantly lower in patients with anterior AMI than in patients with inferior AMI. Moreover, our subgroup analysis with AMI patients with preserved LV ejection fraction showed a tendency of larger LV twist in patients with anterior AMI than in patients with inferior AMI, which was due to significantly increased basal rotation even with reduced apical rotation in anterior AMI.

All AMI patients with recovery showed an increased LV twist to that of normal. As the apical wall function was recovered, as expected, the basal function was returned to the normal and the basal rotation was also decreased to the normal in 6 months. However, in patients with no recovery, the apical rotation and LV twist were more decreased, but the basal rotation was still high. LV twist and torsion is emerging as a sensitive parameter of LV systolic myocardial performance. A significant impairment of LV torsion after AMI will therefore result in increased myocardial stress and oxygen demand of the remaining non-infarcted myocardium. There were several studies about the incremental value of LV torsion for prediction of LV remodelling after AMI. However, these studies included patients who had both ST-elevation MI and non-ST-elevation MI and the majority of patients who showed remodelling had a culprit on anterior descending coronary artery.

In our study, LV torsion was a good predictor for LV recovery in patients with anterior AMI, which was consistent with previous studies. Interestingly, in patients with inferior AMI, only basal rotation was related to the recovery. Because the regional wall motion abnormalities are relatively focal and small and global LV dysfunction is not significant in inferior AMI, the impairment of basal rotation itself may be more important than global LV rotational performance. Since we studied the patients who had ST elevation MI and akinetic walls with more than two contiguous segments in one wall corresponding to single infarct-related coronary artery, the results might be different in patients with multivessel coronary artery disease or severe LV dysfunction.

**Clinical implications**

Regional wall motion abnormalities by AMI affect LV rotation and twisting. This study may help us understand the compensatory mechanism of cardiac function, especially of the basal wall, and may suggest that LV rotation and twisting mechanics are a coordinated motion from the base-to-apex as a global motion.

In addition, early assessment of LV rotational mechanics after AMI could identify patients who are not recovered from AMI and may benefit from aggressive therapy to prevent LV remodelling, heart failure, and poor outcome.

**Limitations**

We did not assess the myocardium with other imaging modalities, such as cardiac magnetic resonance imaging. The use of beta blocker may affect the LV twist; however, there was no significant difference of medication and heart rate was similar between patients with anterior and inferior AMI. All study subjects averaged six decades in age, and the results may be different from younger or older subjects because it is known that LV rotational characteristics differ with age.

Although two-dimensional speckle tracking imaging has been evolving as a robust variable for routine clinical application, this technology is under scrutiny due to the lack of standardization. In addition, speckle-derived strain can be measured using only dedicated software and the accuracy of speckle tracking is dependent on two-dimensional image quality and frame rates. Lastly, two-dimensional speckle tracking analysis cannot track the real three-dimensional motion of the heart and, thus, cannot eliminate
the errors introduced by through-plane motion. However, we attempted to recognize correct basal and apical short-axis levels as much as possible, and we also verified the tracking accuracy visually, retracing and repositioning the region of interest as needed.

Conclusion

The regional wall motion abnormalities in AMI altered LV rotationalal mechanics either by reduced apical and basal rotation. LV twist and torsion were similarly reduced between anterior AMI and inferior AMI even though the LV ejection fraction and WMS index were worse in anterior AMI, which might be due to increased basal rotation as a compensatory mechanism. LV functional recovery can be predicted by LV torsion in anterior AMI and by basal rotation in inferior AMI. The basal rotation has often been ignored; however, our findings suggest that the basal rotation has an important role in LV function.

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