Viability Assessment With Global Left Ventricular Longitudinal Strain Predicts Recovery of Left Ventricular Function After Acute Myocardial Infarction

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**Background**—The extent of viable myocardial tissue is recognized as a major determinant of recovery of left ventricular (LV) function after myocardial infarction. In the current study, the role of global LV strain assessed with novel automated function imaging (AFI) to predict functional recovery after acute infarction was evaluated.

**Methods and Results**—A total of 147 patients (mean age, 61 ± 11 years) admitted for acute myocardial infarction were included. All patients underwent 2D echocardiography within 48 hours of admission. Significant relations were observed between baseline AFI global LV strain and peak level of troponin T ($r=0.64$), peak level of creatine phosphokinase ($r=0.62$), wall motion score index ($r=0.52$), and viability index assessed with single-photon emission computed tomography ($r=0.79$). At 1-year follow-up, LV ejection fraction was reassessed. Patients with absolute improvement in LV ejection fraction ≥5% at 1-year follow-up (n = 70; 48%) had a higher (more negative) baseline AFI global LV strain ($P<0.0001$). Baseline AFI global LV strain was a predictor for change in LV ejection fraction at 1-year follow-up. A cutoff value for baseline AFI global LV strain of −13.7% yielded a sensitivity of 86% and a specificity of 74% to predict LV functional recovery at 1-year follow-up.

**Conclusions**—AFI global LV strain early after acute myocardial infarction reflects myocardial viability and predicts recovery of LV function at 1-year follow-up. (Circ Cardiovasc Imaging. 2010;3:15-23.)

**Key Words:** echocardiography ▪ myocardial contraction ▪ myocardial infarction ▪ ventricles ▪ viability

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A acute myocardial infarction results in loss of myocardial tissue and consequently in regional and global impairment of myocardial contractile function.1,2 Whereas the process of left ventricular (LV) remodeling after infarction is associated with poor long-term clinical outcome, the presence of a preserved LV function after infarction is associated with relatively beneficial outcome.3,4 The extent of viable myocardial tissue (or stunned myocardium) is recognized as a major determinant of recovery of LV function after myocardial infarction.5–7 It has been shown that quantification of myocardial viability early after acute myocardial infarction provides a valuable tool for assessment of reversible myocardial LV dysfunction; moreover, the presence of substantial viable myocardium after infarction is associated with better outcome.7 At present, sophisticated imaging modalities, including nuclear imaging (with single-photon emission computed tomography [SPECT] and positron emission tomography), dobutamine stress echocardiography, or MRI, are used for assessment of myocardial viability.5,7

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**Clinical Perspective on p 23**

Recently, speckle tracking analysis with automated function imaging (AFI) has been introduced as a new echocardiographic technique to assess global LV longitudinal strain.8,9 Importantly, speckle tracking analysis derived strain has been associated with the extent of viable myocardial tissue in patients with chronic ischemic heart disease and was recently validated against MRI and sonomicrometry.10,11 In the current study, the role of AFI global LV strain (as marker of viable myocardium) early after acute myocardial infarction was evaluated for prediction of LV function recovery at 1-year follow-up.

**Methods**

**Patients and Study Protocol**

A total of 156 consecutive patients were evaluated. All patients were admitted for acute myocardial infarction and underwent primary percutaneous coronary intervention in our hospital. During follow-up, 9 patients died. These patients did not have follow-up assessment.
and were excluded from the study. Therefore, the final study population existed of 147 patients. Within 48 hours of admission (at baseline), all patients underwent 2D echocardiography to assess LV volumes, LV ejection fraction (LVEF), severity of mitral regurgitation, wall motion score index (WMSI), and global LV strain quantified using the AFI technique (further mentioned as “AFI global LV strain”). Within 2 months after acute myocardial infarction, patients underwent SPECT to assess a viability index, which reflects the global extent of viability in the left ventricle.

At 1-year follow-up, echocardiography was repeated with reassessment of LV volumes and LVEF. The relation between several clinical and echocardiographic variables at baseline, particularly AFI global LV strain, and recovery of LV function at 1-year follow-up was evaluated. The study was approved by the institutional ethics committee, and informed consent was obtained from all patients.

Echocardiography
Patients were imaged in the left lateral decubitus position using a commercially available system (Vivid Seven, General Electric-Vingmed, Milwaukee, Wis); imaging was performed at baseline and 1-year follow-up. Standard images were obtained using a 3.5-MHz transducer, at a depth of 16 cm in the parasternal (long- and short-axis images) and apical (2- and 4-chamber images) views. Standard 2D and color Doppler data, triggered to the QRS complex, were saved in cine-loop format. LV volumes (end-systolic [LVESV] and end-diastolic [LVEDV]) and LVEF were calculated from conventional apical 2- and 4-chamber images, using the biplane Simpson technique. For assessment of WMSI, the LV was divided into 16 segments. A semiquantitative scoring system (1, normal; 2, hypokinesia; 3, akinesia; 4, dyskinesia) was used to analyze each

AFI Global LV Strain
Global LV longitudinal strain was assessed using the AFI technique, which provides a new imaging modality based on 2D longitudinal strain imaging (Figure 1). Longitudinal strain (percentage) is defined as the physiological change in length of the region of interest from end-diastole to end-systole. During this period, strain in the longitudinal direction is a negative value as the length of the region of interest decreases. Longitudinal strain can be calculated using the following formula: longitudinal strain (%)=(L(end-systole)−L(end-diastole))/L(end-diastole)×100%; where L is the length of the region of interest.

Assessment of global LV longitudinal strain (AFI global LV strain; mentioned by the software as GLPS_Avg, Figure 1) provides
Global LV Strain Predicts Functional Recovery

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1 value representing the overall peak systolic longitudinal strain of all individual LV segments.

With the commercially available AFI technique (General Electric, Milwaukee, Wis), myocardial tissue deformation (strain) is calculated using speckle tracking from 2D gray-scale images. For this analysis, a set of 3 longitudinal 2D image planes (apical long-axis, 2- and 4-chamber views) was used. Aortic valve closure timing was marked (to determine the end of systole) in the selected views, and 3 points were anchored inside the myocardial tissue, 2 placed at the basal segments along the mitral valve annulus and 1 at the apex. These points triggered the automatic process, which analyzed myocardial motion by tracking features (natural acoustic tags). The percent of wall thinning and shortening was displayed for each plane, representing longitudinal strain. The results of all 3 planes are then combined in a single bull’s-eye map, which presents the analysis for each segment along with a global strain value for the LV.

For global LV longitudinal strain analysis, digital cine-loops were off-line processed using commercially available software (EchoPac 6.1, GE Medical Systems, Horten, Norway). Mean frame rate of the obtained images was 70 (40–100) fps. The interclass and intraclass correlation coefficients for assessment of AFI global LV strain were 0.92 and 0.95, respectively.19

Single-Photon Emission Computed Tomography

Resting myocardial SPECT imaging with technetium-99m tetrofosmin (300 MBq, injected at rest) was performed using a triple-head SPECT camera system (GCA 9300/HG, Toshiba Corp), equipped with low-energy–high resolution collimators. Around the 140-KeV energy peak of technetium-99m tetrofosmin, a 20% window was used. A total of 90 projections (step and shoot mode, 35 seconds per projection; imaging time, 23 minutes) was obtained over a 360° circular orbit. Data were stored in 64×64 matrix.

Additional reconstruction yielded standard long- and short-axis projections perpendicular to the heart axis. The short-axis slices were displayed in polar maps, adjusted for peak myocardial activity (100%). The myocardium was divided into 17 segments.20 Myocardial perfusion was analyzed quantitatively with previously validated and commercially available automated software (quantitative gated SPECT, QGS, Cedars-Sinai Medical Center, Los Angeles, Calif).21 Based on segmental tracer activity, individual segments were scored as viable (score 1, >50% of maximum tracer activity) or nonviable (score 0, ≤50% of maximum tracer activity). The viability index was then calculated as the number of viable LV segments divided by the highest possible score (which is 17) and therefore reflects the global extent of viable myocardium in the LV.22

Statistical Analysis

Univariable and multivariable linear regression analyses were performed to evaluate the relation between AFI global LV strain at baseline and changes in LVEF at 1-year follow-up (continuous outcome). Additionally, univariable and multivariable logistic regression analyses relating AFI global LV strain at baseline to recovery of LV function as a dichotomous outcome (ie, improvement in LVEF ≥5% or no improvement in LVEF ≥5%) were performed. In addition, the relations between several clinical and echocardiographic variables, and changes in LVEF at 1-year follow-up were explored. All variables that are listed in the tables presenting the univariable regression analyses were considered as potential confounding factors for the relation between AFI global LV strain at baseline and changes in LVEF during follow-up (both as a continuous outcome and as a dichotomous outcome). However, the number of covariates in the final multivariable regression models was limited via a backward selection procedure, and only variables with a probability value <0.15 were maintained.

Apart from independence of data points, linear regression analysis requires a linear relation between the dependent variable and the independent (predictor) variables, a similar variance of the residuals, and a normal distribution of the residuals. These assumptions were met for each predictor variable that was considered: The addition of squared or logarithmic terms did not result in improvement of the R² value of the regression models; predictor-residual plots confirmed homogeneity of residuals; and PP plots of residuals did not show deviation from normality. Results of linear regression analysis might be influenced by individual observations with large residual (“outliers”). The results of the present study were not affected by this phenomenon because consistent results were obtained after exclusion of the observations with a residual that belongs to the upper decile (analyzes based on the entire data set were reported).

Logistic regression analysis requires a linear relation between the (continuous) independent variables and the log odds (logit) of the dependent variable. This assumption was evaluated by fitting both linear and nonlinear (cubic) logistic models. The linearity assumption was valid for each variable that was considered because all likelihood ratio tests, with linearity as the null hypothesis, showed nonsignificant results.

Receiver operating characteristic (ROC) curve analysis for prediction of recovery of LV function was applied. Additional analysis provided the “optimal” threshold (in the current data set) of AFI global LV strain for the prediction of recovery of LV function during follow-up. This optimum was defined as the value for which the sum of sensitivity and specificity was maximized.

Continuous data are presented as mean±SD. Categorical data are summarized as frequencies and percentages. Differences between patients with and without improvement in LVEF were evaluated using unpaired Student t tests (continuous data) and χ² tests (dichotomous data) as appropriate. Paired Student t tests were applied to evaluate changes in continuous data between baseline and 1-year follow-up. All statistical tests were 2-sided. For all tests, a probability value <0.05 was considered statistically significant.

Results

Study Population

An overview of the baseline characteristics of the study population (n=147; mean age, 61±11 years) is provided in Table 1. All patients underwent primary percutaneous coronary intervention for acute myocardial infarction. Mean symptoms-to-balloon time was 199±106 minutes. According to protocol, all patients were optimally treated with aspirin, clopidogrel, β-blockers, angiotensin-converting enzyme inhibitors, and statins, if tolerated.12

Echocardiography

Echocardiographic characteristics of the study population are summarized in Table 1. At baseline, mean LVESV was 67±20 mL, mean LVEDV was 130±32 mL, and a mean LVEF of 48±7% was measured. Moderate to severe mitral regurgitation (grade ≥2) was demonstrated in 6 (4%) patients. Mean WMSI was 1.48±0.22. Importantly, mean AFI global LV strain measured −14.2±2.8% (normal values range from −20.3% to −24.1%).9,18

AFI Global LV Strain

Of note, substantial relations between AFI global LV strain and both peak level of troponin T (r=0.64, P<0.05) and peak level of creatine phosphokinase (r=0.62, P<0.05), as reflectors of enzymatic infarct size, were demonstrated. Thus, higher (more negative) AFI global LV strain was related to lesser infarct size and thus reflecting more viable myocardium. In addition, a modest relation was observed between AFI global LV strain and WMSI, as reflector of echocardiographic...
No differences in baseline clinical characteristics were demonstrated between patients with improvement in LVEF ≥5% at 1-year follow-up (further mentioned as "patients with improvement in LVEF") and those without improvement in LVEF ≥5% at 1-year follow-up (further mentioned as "patients without improvement in LVEF"), except for the infarct-related artery and the peak levels of cardiac enzymes. The left anterior descending coronary artery (LAD) was the infarct-related artery in 29 (41%) patients with improvement in LVEF and in 50 (65%) patients without improvement in LVEF (P<0.005). Mean peak troponin T and creatine phosphokinase were 4.3±3.8 μg/L and 1574±1210 U/L in patients with improvement in LVEF and 9.6±6.7 μg/L (P<0.0001) and 3447±2220 U/L (P<0.0001) in patients without improvement in LVEF, respectively.

In patients with improvement in LVEF at 1-year follow-up, mean LVEF increased from 47±6% at baseline to 58±7% at 1-year follow-up (by definition). In addition, mean LVESV decreased from 67±20 mL to 63±30 mL at 1-year follow-up (P=0.03). Mean LVEDV decreased from 130±32 mL to 126±38 mL (P=0.08). Of note, mean LVEF improved significantly from 48±7% at baseline to 51±11% at 1-year follow-up (P<0.0001). Absolute improvement in LVEF ≥5% at 1-year follow-up was observed in 70 patients (48%). The remaining 77 (52%) patients did not show improvement in LVEF ≥5%. Clinical and echocardiographic characteristics of these 2 subgroups were compared (Table 1).

Recovery of LV Function
At 1-year follow-up, improvement in LV function was observed for the overall study population. Mean LVESV decreased from 67±20 mL at baseline to 63±30 mL at 1-year follow-up (P=0.03). Mean LVEDV decreased from 130±32 mL to 126±38 mL (P=0.08). Of note, mean LVEF improved significantly from 48±7% at baseline to 51±11% at 1-year follow-up (P<0.0001). Absolute improvement in LVEF ≥5% at 1-year follow-up was observed in 70 patients (48%). The remaining 77 (52%) patients did not show improvement in LVEF ≥5%. Clinical and echocardiographic characteristics of these 2 subgroups were compared (Table 1).

### Table 1. Characteristics of the Study Population (n=147)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients With Improvement in LVEF ≥5% (abs) (n=70)</th>
<th>Patients Without Improvement in LVEF ≥5% (abs) (n=77)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>61±11</td>
<td>61±11</td>
<td>NS</td>
</tr>
<tr>
<td>Sex, male/female</td>
<td>53 (76/17 (24)</td>
<td>62 (81/15 (19)</td>
<td>NS</td>
</tr>
<tr>
<td>Risk factors for CAD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>6 (7)</td>
<td>8 (10)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>22 (31)</td>
<td>24 (31)</td>
<td>NS</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>13 (19)</td>
<td>15 (19)</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>38 (54)</td>
<td>36 (47)</td>
<td>NS</td>
</tr>
<tr>
<td>Family history of CAD</td>
<td>33 (47)</td>
<td>29 (38)</td>
<td>NS</td>
</tr>
<tr>
<td>Infarct-related artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>29 (41)</td>
<td>50 (65)</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>RCA</td>
<td>24 (34)</td>
<td>14 (18)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LCX</td>
<td>17 (24)</td>
<td>13 (17)</td>
<td>NS</td>
</tr>
<tr>
<td>Cardiac enzymes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak troponin T, μg/L</td>
<td>4.3±3.8</td>
<td>9.6±6.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Peak CKP, U/L</td>
<td>1574±1210</td>
<td>3447±2220</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Echocardiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVESV, mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>66±18</td>
<td>69±22</td>
<td>NS</td>
</tr>
<tr>
<td>1 y</td>
<td>48±15*</td>
<td>77±34†</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEDV, mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>125±29</td>
<td>133±35</td>
<td>NS</td>
</tr>
<tr>
<td>1 y</td>
<td>113±28*</td>
<td>138±41</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LVEF, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>47±6</td>
<td>49±8</td>
<td>NS</td>
</tr>
<tr>
<td>1 y</td>
<td>58±7*</td>
<td>45±11*</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>MR moderate-severe</td>
<td>4 (6)</td>
<td>2 (3)</td>
<td>NS</td>
</tr>
<tr>
<td>WMSI</td>
<td>1.40±0.23</td>
<td>1.54±0.19</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>AFI global LV strain, %</td>
<td>-16.1±2.0</td>
<td>-12.5±2.3</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or n (%). abs indicates absolute; NS, not significant; CAD, coronary artery disease; CPK, creatine phosphokinase; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; MR, mitral regurgitation; RCA, right coronary artery.

*P<0.0001 for baseline versus 1 year.
†P<0.05 for baseline versus 1 year.

infarct size (r=0.52, P<0.05). Importantly, a strong relation was observed between AFI global LV strain and viability index assessed with SPECT (r=0.79, P<0.05).

Prediction of Recovery of LV Function
In Table 2, an overview of the univariable analysis for change in LVEF during follow-up, is provided. Linear regression analysis (change in LVEF during follow-up as a continuous
demonstrated that AFI global LV strain, peak level of troponin T, peak level of creatine phosphokinase, WMSI, LAD as infarct-related artery, use of diuretics, and use of angiotensin receptor blockers are determinants for change in LVEF at 1-year follow-up after acute myocardial infarction. In Figure 3, the relation between AFI global LV strain and absolute change in LVEF at 1-year follow-up is demonstrated. Logistic regression analysis (improvement in LVEF ≥5% as a binary value) showed that AFI global LV strain, peak level of troponin T, peak level of creatine phosphokinase, WMSI, and the LAD as culprit vessel are related to change in LVEF at 1-year follow-up after acute myocardial infarction.

Table 3 provides an overview of the multivariable analysis for change in LVEF during follow-up. Linear regression analysis pointed out that AFI global LV strain is a predictor for change in LVEF during follow-up (parameter estimate, 2.32; standard error, 0.28; P<0.001). This was confirmed during logistic regression analysis, which presented AFI global LV strain as a predictor for change in LVEF during follow-up (odds ratio, 0.50; 95% confidence interval, 0.38 to 0.68, P<0.001). As a sensitivity analysis, the patients who died (and in whom data about LVEF at 1-year follow-up were therefore not available) were labeled as nonimprovers, and consistent results were obtained.

In addition, ROC curve analysis was performed (AUC, 0.87; 95% confidence interval, 0.82 to 0.93; P<0.001). A cutoff value for AFI global LV strain of −13.7% demonstrated to be predictive for recovery of LV function at 1-year follow-up after acute myocardial infarction with a sensitivity of 86% and a specificity of 74% (Figures 4 and 5).
Viability and Recovery of LV Function

The presence of substantially viable myocardium is recognized as an important determinant of recovery of LV function after acute infarction. Assessment of myocardial viability early after acute infarction provides a valuable tool for prediction of functional recovery with improved clinical outcome.\(^5\) Sciagra et al.\(^5\) evaluated 48 patients with acute infarction who were treated with primary percutaneous coronary intervention. Assessment of contractile reserve in the infarct zone with low-dose dobutamine echocardiography at 3 days after the index infarction allowed prediction of improvement in LV function at 6-month follow-up, as defined by an increase in LVEF of at least 5 U.\(^5\) The authors demonstrated that the presence of myocardial viability (contractile reserve) was associated with better survival at 9-month follow-up.\(^2\) Besides the use of low-dose dobutamine echocardiography for assessment of contractile reserve as a marker of myocardial viability, other sophisticated noninvasive imaging techniques for evaluation of viability are based on demonstration of cell membrane integrity (nuclear imaging with thallium-201), the presence of intact mitochondria (nuclear imaging with technetium-99m labeled tracers), preserved myocardial metabolism (nuclear imaging with F18-fluorodeoxyglucose), microvascular integrity (myocardial contrast echocardiography), or the absence of scar tissue (contrast-enhanced MRI in areas of dysfunctional myocardium).

Strain and Viability

Recently, echocardiographically derived LV strain assessed by speckle tracking analysis has been associated with the presence of viable myocardium in patients with chronic ischemic heart disease.\(^10\) Gjesdal et al.\(^10\) evaluated 38 patients, 9 months after first myocardial infarction, with 2D speckle tracking echocardiography and contrast-enhanced MRI (in contrast to SPECT used in the present study). The authors demonstrated that peak systolic longitudinal strain, measured by 2D speckle tracking echocardiography and contrast-enhanced MRI (in contrast to SPECT used in the present study). The authors demonstrated that peak systolic longitudinal strain, measured by 2D speckle tracking echocardiography, was able to separate infarcted from noninfarcted myocardium, as assessed with contrast-enhanced MRI. At the global level, a value of $-15\%$ for peak systolic longitudinal LV strain was demonstrated to be able to identify infarcted tissue with a sensitivity of 83% and a specificity of 93%. Peak systolic longitudinal strain correlated with infarct mass assessed with contrast-enhanced MRI in this group of patients with chronic ischemic myocardium.

### Discussion

Table 3. Multivariable Analysis for Change in LVEF During Follow-Up

<table>
<thead>
<tr>
<th>Variables</th>
<th>Parameter Estimate</th>
<th>Standard Error</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Linear regression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AFI global LV strain</td>
<td>$-2.323$</td>
<td>$0.281$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Peak level troponin T</td>
<td>$-0.202$</td>
<td>$0.124$</td>
<td>$0.11$</td>
</tr>
<tr>
<td>Culprit vessel LAD</td>
<td>$1.908$</td>
<td>$1.205$</td>
<td>$0.12$</td>
</tr>
<tr>
<td>Age</td>
<td>$0.073$</td>
<td>$0.047$</td>
<td>$0.13$</td>
</tr>
<tr>
<td>Logistic regression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AFI global LV strain</td>
<td>$0.50$</td>
<td>$0.38$–$0.68$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Peak level CPK</td>
<td>$1.00$</td>
<td>$1.00$–$1.00$</td>
<td>$0.025$</td>
</tr>
<tr>
<td>WMSI</td>
<td>$0.21$</td>
<td>$0.01$–$3.16$</td>
<td>$0.26$</td>
</tr>
</tbody>
</table>

ACE indicates angiotensin-converting enzyme; CAD, coronary artery disease; CPK, creatine phosphokinase; LAD, left anterior descending coronary artery; MI, myocardial infarction; CI, confidence interval.

Figure 4. Determination of the optimal cutoff value for AFI global LV strain to predict recovery of LV function. A cutoff value of $-13.7\%$ for AFI global LV strain was associated with the highest sum of sensitivity and specificity to predict recovery of LV function.
heart disease. Accordingly, higher (more negative) strain values were associated with the presence of viable myocardium.

Until now, data about the relation between echocardiographically derived strain and viability early after acute myocardial infarction are scarce, and the potential value of LV strain to predict recovery of LV function after infarction has not been explored. In the current study, AFI global LV strain assessed within 48 hours after acute infarction was strongly related to peak levels of troponin T, peak levels of creatine phosphokinase and echocardiographic WMSI, which have previously been described as reflectors of infarct size. Patients with the LAD as infarct-related artery had the lowest (less negative) baseline AFI global LV strain and demonstrated the smallest increase in LVEF at 1-year follow-up, probably due to the relatively larger infarct size. This suggests that infarct location might influence the relation between baseline AFI global LV strain and recovery of LV function during follow-up. This is an interesting finding and warrants further study. Some interaction between infarct location, resulting infarct size, AFI global LV strain, and change in LVEF during follow-up after infarction can be expected, as myocardial infarctions with the LAD as culprit vessel are mostly larger infarctions resulting in lesser viability (with decreased strain) and a lower probability to improve in LVEF. Further studies should be designed to more thoroughly evaluate the relation between AFI global LV strain and change in LVEF, including the contribution of interaction terms, after myocardial infarction.

Baseline AFI global LV strain was also strongly related to viability as determined by SPECT. Importantly, baseline AFI global LV strain was predictive for recovery of LV function at 1-year follow-up after acute infarction. Patients with an absolute improvement in LVEF of at least 5% also showed a significant decrease in LV volumes at 1-year follow-up. A cutoff value for baseline AFI global LV strain of $-13.7\%$ yielded a sensitivity of 86% and a specificity of 74% to predict recovery of LV function at 1-year follow-up after acute infarction.

**AFI Global LV Strain**

Speckle tracking analysis with AFI has recently been introduced as a new echocardiographic technique, based on assessment of myocardial tissue deformation, to quantify global LV longitudinal strain. The technique has been validated against MRI and sonomicrometry. Besides its angle-independence, the benefit of speckle tracking analysis is its ability to differentiate between active and passive motion by quantification of myocardial deformation. High-quality images are needed for assessment of AFI global LV strain because the technique is frame rate-dependent. Mean frame rate of the obtained images in the current study was 70 (40 to 100) fps.

Assessment of global LV longitudinal strain using the AFI technique is easily obtained and takes a few to 10 minutes, depending on the level of the operator’s experience. The software uses a step-by-step approach and provides continuous support during the process. In the current study, good intraobserver and interobserver agreement were reported for assessment of AFI global LV strain.

**Clinical Implications**

The results of the present study demonstrate that assessment of AFI global LV strain immediately after acute infarction is a useful tool to predict recovery of LV function at 1-year follow-up. Baseline AFI global LV strain is strongly related to infarct size and may serve as a marker of residual myocardial viability after infarction. An advantage of this technique is the fact that quantification of AFI global LV strain is performed at rest only. No additional analysis during exercise is needed. Furthermore, patients are not exposed to radiation. Therefore, assessment of AFI global LV strain using echocardiography may be used as an accessible imaging technique to estimate viability and to determine the probability of recovery of LV function early after acute infarction.

In the present study, a cutoff value of $-13.7\%$ for AFI global LV strain to predict recovery of LV function was derived from a large representative set of patients with acute
myocardial infarction treated with primary percutaneous coronary intervention. It is unknown whether this cutoff value is useful in different sets of patients (for example, after thrombolysis, or in the chronic phase after infarction). Further studies should therefore evaluate this issue.

In addition, future studies should study the time course of AFI global LV strain during follow-up after acute infarction. Unfortunately, in the current study, data on AFI global LV strain during follow-up were not systematically obtained. Furthermore, data about the appropriateness of assessment of longitudinal strain in inferior myocardial infarction are limited. Global LV strain is decreased (less negative) in patients with inferior myocardial infarction, suggesting that infarction of inferior segments does result in decreased longitudinal strain despite the difference in fiber orientation. This issue needs further study. In the current study, mitral regurgitation was graded on the basis of the jet area divided by the left atrial area, whereas the proximal isovelocity surface area (PISA) method may be preferred, and is a limitation of the study.

Conclusion
The results of the present study demonstrate that AFI global LV strain after acute infarction is strongly related to infarct size and may also serve as a marker of residual myocardial viability. Using a cutoff value of ~13.7% for baseline AFI global LV strain, a sensitivity of 86% and a specificity of 74% were obtained to classify recovery of LV function at 1-year follow-up.

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CLINICAL PERSPECTIVE

The extent of viable myocardial tissue is recognized as a major determinant of recovery of left ventricular (LV) function after myocardial infarction. In the current study, the role of global LV strain (as a marker of viable myocardium) assessed with novel automated function imaging (AFI) to predict functional recovery after acute infarction was evaluated. Baseline AFI global LV strain was strongly related to viability assessed with single-photon emission computed tomography. Importantly, baseline AFI global LV strain (within 48 hours after infarction) was a predictor for change in LV ejection fraction at 1-year follow-up. A cutoff value for baseline AFI global LV strain of $-13.7\%$ yielded a sensitivity of 86% and a specificity of 74% to predict LV functional recovery at 1-year follow-up. An advantage of this novel echocardiographic technique is the fact that quantification of AFI global LV strain is performed at rest only. No additional acquisitions during exercise are needed. Furthermore, patients are not exposed to radiation. Therefore, assessment of AFI global LV strain using echocardiography may be used as an accessible imaging technique to determine viability and subsequent probability of recovery of LV function after acute myocardial infarction.
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