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Diagnostic Capability and Reproducibility of Strain by Doppler and by Speckle Tracking in Patients With Acute Myocardial Infarction

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OBJECTIVES The objective of the present study was to investigate the ability of strain by Doppler and by speckle tracking echocardiography in the acute phase in patients with ST-segment elevation myocardial infarction (STEMI) to diagnose left ventricular (LV) infarct size. Furthermore, we wanted to study at which time during the cardiac cycle strain should be measured.

BACKGROUND The assessment of regional myocardial dysfunction may be an important diagnostic tool in the evaluation of acute myocardial injury.

METHODS Strain by Doppler and speckle tracking were assessed in the acute phase and after 10 days in 36 patients (61 ± 11 years) with STEMI treated with thrombolysis. In a 16-segment model of the LV, peak systolic, end systolic, and peak negative strain were validated against the corresponding myocardial segments measured by contrast-enhanced cardiac magnetic resonance. The 16 segments were averaged to assess LV global longitudinal strain. In addition, 6 segments were analyzed from parasternal short-axis recordings at the papillary muscle level to assess circumferential strain. Reproducibility was tested in 20 patients.

RESULTS The different segmental strain assessments separated significantly ($p < 0.0001$) between the different levels of infarct transmurality regardless of method, with better reproducibility for speckle strain. Circumferential strain separated better than longitudinal strain. With a cutoff value of −13.3% for segmental circumferential strain, sensitivity was 80% and specificity was 74% for prediction of transmural infarction. The LV global strain showed a good correlation with LV infarct size, with the best correlation for LV global peak systolic speckle strain ($\beta = 0.76$, $p < 0.0001$).

CONCLUSIONS On a segmental level, circumferential strain separated transmural from subendocardial necrosis better than longitudinal strain in the acute phase in patients with STEMI. Our findings suggest that in the acute phase in patients treated with thrombolysis, LV global peak systolic speckle strain should be the preferred method for predicting final LV infarct size. (J Am Coll Cardiol Img 2009; 2:24–33) © 2009 by the American College of Cardiology Foundation

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Echocardiographic techniques are easily accessible and may be used as bedside tools to study regional function in acute myocardial infarction (AMI). Direct visualization of wall motion in the ischemic myocardium is subject to significant variability (1). Myocardial strain by Doppler is superior to wall motion score index and post-systolic shortening in the diagnosis of myocardial ischemia and the detection of viable myocardium (2–4). Strain determines regional myocardial function and can be measured by Doppler or speckle tracking. Both methods have been validated against sonomicrometry in experimental studies of acute myocardial ischemia (5,6), and by different cardiac magnetic resonance techniques in patients with ischemic heart disease (4,7–9), and are promising tools in the evaluation of myocardial injury (10,11). Strain corresponds well with left ventricular (LV) infarct size in patients with acute anterior ST-segment elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention (PCI) (12). Thrombolysis is still a widely used method of reperfusion in patients with acute STEMI, despite increasing use of primary PCI. Evaluation of the degree of myocardial injury in the acute phase of STEMI treated with thrombolysis may be of clinical importance to guide further revascularization and add important diagnostic and prognostic information in these patients.

The 2 strain methods are based on different principles and can potentially give different results. Strain by Doppler is limited to the measurement of movement parallel to the ultrasound beam. Speckle strain may be measured independently of angle and measures regional deformation in circumferential and longitudinal directions of the LV. Such measurements may add important information in the separation of subendocardial from transmural necrosis. Longitudinal deformation mainly represents subendocardial contraction, whereas circumferential deformation mainly represents contraction of the midmyocardial and subepicardial layers (10,13). However, it is not clear whether Doppler or speckle strain should be preferred in acute STEMI to estimate final infarct size. In addition, it is unclear whether strain should be measured as peak systolic, end systolic, or peak negative strain.

The aim of the present study was to determine the diagnostic capability and reproducibility of the 2 ultrasonic strain techniques in the acute phase of anterior and inferior STEMI treated with thrombolysis and to validate them against LV infarct size measured by contrast-enhanced cardiac magnetic resonance (ceCMR). Furthermore, we wanted to study at which time during the cardiac cycle strain should be measured.

METHODS

Patient population. The study group consisted of 36 randomly selected patients with first-time acute STEMI (Table 1). All patients underwent conventional and color Doppler echocardiography within 3 h after treatment with thrombolysis and at discharge. Coronary angiography was performed 16 ± 21 h after thrombolysis. Twenty-eight patients (78%) had achieved reperfusion of the infarct related artery at the time of angiography. Eight patients had an occluded artery. In all patients the infarct-related artery was revascularized, either with PCI or coronary bypass surgery within a few days after thrombolysis. All patients received medical treatment according to guidelines.

Twenty-nine patients were examined by ceCMR more than 6 months after the acute event. Seven patients were not examined by ceCMR because of contraindications or technical failure during examination. None of the patients experienced re-infarction between the first event and ceCMR. Patients with significant valve disease, arrhythmia, or a history of previous myocardial infarction were excluded.

The study was approved by the Regional Committee for Medical Research Ethics. Written informed consent was obtained from all individuals.

ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

ceCMR = contrast-enhanced cardiac magnetic resonance

LV = left ventricle/ventricular

PCI = percutaneous coronary intervention

STEMI = ST-segment elevation myocardial infarction

TDI = tissue Doppler (velocity) imaging

Echocardiography. Examinations were performed with a digital ultrasonic device system (Vivid 7, GE Vingmed Ultrasound, Horten, Norway). The patients were examined in the left supine position using the parasternal short axis at the papillary

Table 1. Clinical Characteristics of the Patients During Acute STEMI (n = 36)

Parameters	Results
Age (yrs)	61 ± 11
Sex (male/female)	30/6
Anterior infarction/inferior infarction	18/18
Time of ischemia (min)	163 ± 128
LVEF by echocardiography (%)	50 ± 11

Continuous variables are presented as mean ± SD.
LVEF = left ventricular ejection fraction; STEMI = ST-segment elevation myocardial infarction; time of ischemia = time from symptom onset to start thrombolysis.

muscle level and apical 4-chamber, 2-chamber, and long-axis views of the LV. Great care was taken to obtain high-quality recordings of all LV walls (Fig. 1). Three cardiac cycles were stored during breath-hold. Two-dimensional grayscale images were obtained at a frame rate of 76 ± 2 frames/s, and color tissue Doppler (velocity) images (TDI) were obtained at 154 ± 2 frames/s. No patients were excluded because of technical limitations or poor image quality. The recordings were stored digitally for off-line analysis with a dedicated image processing and analyzing program (Echopac, GE Vingmed Ultrasound). Left ventricular ejection fraction was assessed by the Simpson method from grayscale recordings.

Image analysis. A 16-segment LV model was obtained from the 4-chamber, 2-chamber, and long-axis recordings. In addition, 6 segments were analyzed from parasternal short-axis recordings (14). Three different strain parameters were measured during 1 heart cycle by both methods. Peak systolic strain was defined as the peak positive or peak negative strain value during systole. End systolic strain was defined as the magnitude of deformation at the time of aortic valve closure, and peak negative strain was the maximum negative strain value during systole or early diastole (Figs. 1A, 1B, and 1C). Post-systolic shortening was calculated as the difference between deformation after aortic valve closure and end systolic strain. Strain measurement from 16 segments were averaged to assess a LV global longitudinal parameter based on peak systolic, end systolic, and peak negative strain.

Measurement of strain by Doppler. Three myocardial longitudinal strain curves were obtained in the basal part of each segment from the TDI recordings, using a region of interest of 6 × 6 mm, which was set as a default. The velocity signal was optimized, including avoidance of reverberation artifacts, and the region of interest was tracked frame by frame. Strain values from 1 of the 3 representative curves were used in further analysis. Segments that were poorly visualized, with aliasing on tissue velocity, or with insonation angles >30°, were excluded.

Measurement of strain by speckle tracking. Two-dimensional strain software identified the endocardial border, and after tracing myocardial motion was automatically tracked in each imaging view. Longitudinal and circumferential strain curves reflected the average value of all of the acoustic markers in each segment. In segments with poor tracking, the observer readjusted the endocardial trace line until a better tracking score was achieved. If this was impossible, the segment was excluded.

ceCMR. Patients were scanned in a supine position by a 1.5-T whole-body scanner (Intera R 10.3 Philips Medical Systems, Best, the Netherlands) using a dedicated cardiac coil. The images were electrocardiogram-gated and obtained during breath-hold. Myocardial mass was obtained by a steady-state free precession technique (balanced fast field echo) covering LV with 10 to 14 contiguous slices (8-mm thickness, 2-mm gap). Late-enhancement images were acquired 10 to 15 min after administration of 0.25 mmol/kg of a gadolinium-based contrast agent, using an inversion-recovery-prepared T1-weighted gradient-echo sequence. Similar density (1.05 g/cm^3) was assumed for both hyperenhanced and nonhyperenhanced myocardium.

Post-processing was performed with the View Forum Software (Philips Medical Systems). For the segmental assessment of the LV, a 16-segment model was used (14). The infarct size was expressed as percent necrosis of segmental volume for each of the LV segments, and the segments were divided into 3 groups based on the extent of myocardial infarction (no infarction, 1% to 50% infarct size, and 51% to 100% infarct size). The total infarct size was reported as percent of total LV mass. Patients were divided into groups depending on the LV global infarct size (small infarcts <20% LV infarct size, large infarcts >20% LV infarct size).

Statistical methods. The data were analyzed using standard statistical software (SPSS version 14.0, SPSS, Inc., Chicago, Illinois). Continuous variables are expressed as mean ± SD. The paired nominal data were evaluated by McNemar tests calculated by exact methods. Segment-wise analyses are uncorrected.

Paired Student *t* tests were used to compare strain in the acute phase of the AMI and after 10 days. The segmental infarct size by ceCMR was compared with the corresponding strain values for Doppler and speckle strain using analysis of variance with the post hoc Scheffe test. We performed a multivariate regression analysis (backward method) to compare LV segmental or global strain and to find the best time during the cardiac cycle for estimating final infarct size, using segmental or total infarct size by ceCMR as the dependent variable. The correlation between each LV global strain parameter and total infarct size was analyzed by linear regression.

Receiver-operator characteristic curves were constructed, and areas under the curves were measured. Reproducibility was calculated by intraclass corre-

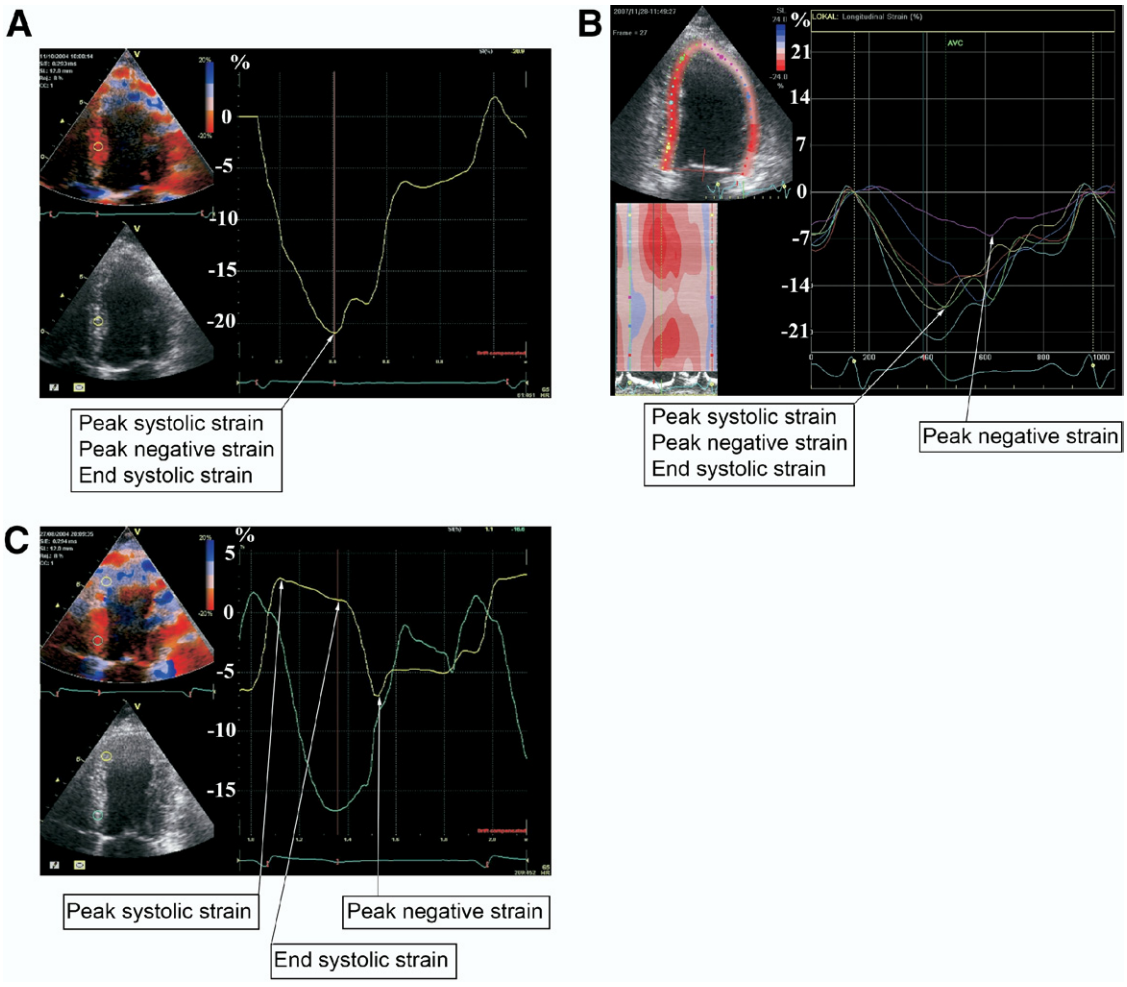

Figure 1. Representative Strain Curves

Representative strain curve from apical 4-chamber view with electrocardiogram from **(A)** a segment without myocardial infarction illustrated by Doppler, **(B)** a segment with subendocardial infarction illustrated by speckle tracking, and **(C)** a segment with transmural infarction illustrated by Doppler, showing measurements of peak systolic, end systolic, and peak negative strain. **(A)** There is no post-systolic shortening and peak negative strain coincides with systolic strain. **(B)** Segments with reduced systolic shortening and post-systolic shortening as seen in segments with small infarcts compared with normal segments. **(C)** The differences between peak systolic strain, end systolic strain, and peak negative strain as seen in segments with large infarcts where there is systolic bulging and post-systolic shortening. This pathologic strain curve is compared with a normal strain curve.

lation coefficient in 20 random selected patients. A value of $p < 0.05$ was considered statistically significant.

RESULTS

Echocardiography in the acute phase was performed 139 ± 59 min after start of thrombolytic therapy. Echocardiography at discharge was performed after 10 ± 4 days. From apical views, a total of 558 of 576 segments (97%) had sufficient image quality for Doppler and a total of 538 of 576 segments (93%) for speckle strain analysis. After 10 days, 93% had sufficient image quality for Doppler and 91% for speckle strain analysis. In the short axis, a total of 194 of 216 segments (90%) were analyzable in the acute phase and 84% were analyzable after 10 days. Medical treatment is presented in Table 2.

Strain. Paired Student *t* tests showed a significantly impaired longitudinal strain in the acute phase compared with strain after 10 days for Doppler strain, but no difference for speckle strain. Details are presented in Table 3.

Strain and infarct size. The LV infarct size by ceCMR was 25 ± 25 g, which represents 15 ± 11% of the LV mass. The different segmental longitudinal strain assessments (peak systolic, end systolic, and peak negative Doppler and speckle strain)

Table 2. Comparison of Medical Treatments During Follow-Up

	Day 1, n = 36 Patients (%)	6 Months, n = 35 Patients (%)	p Value
Beta-blockers	35/36 (97)	35/35 (100)	—
Statins	35/36 (97)	33/35 (94)	1.00
Acetylsalicylic acid	36/36 (100)	34/35 (97)	—
ACE inhibitors	13/36 (36)	17/35 (49)	0.227
A2-antagonists	3/36 (8)	10/35 (29)	0.016
Aldosterone antagonists	0/36 (0)	3/35 (9)	—

We lost medical information from 1 patient at 6 months.
— = not analyzable by McNemar test; ACE = angiotensin-converting enzyme; A2-antagonists = angiotensin II antagonists.

separated significantly ($p < 0.0001$) between the different levels of infarct transmurality in the whole patient group. Mean values for segmental circumferential and longitudinal speckle strain based on the extent of myocardial infarction are presented in Table 4. However, segmental circumferential strain separated subendocardial from transmural necrosis better than longitudinal strain, as shown in the receiver-operator characteristic analyses in Table 5. The diagnostic capabilities of longitudinal strain in anterior and inferior myocardial infarctions are presented in Figures 2A and 2B. When using a multivariate regression analysis, segmental peak systolic speckle strain correlated significantly with segmental infarct size measured by ceCMR in the acute phase ($p < 0.0001$).

Table 6 shows the correlation between each LV global strain parameter and LV infarct size by ceCMR. When using multivariate regression analysis for Doppler and speckle strain in the acute phase, LV global peak negative Doppler ($\beta = 0.67$) and peak systolic speckle strain ($\beta = 0.76$) were statistically the best parameters for predicting total infarct size ($p < 0.0001$). Average strain measurements in both longitudinal and circumferential direction did not add diagnostic precision of LV global strain beyond the use of longitudinal strain alone in the separation of small and large LV infarcts. Post-systolic shortening was not associated with LV infarct size (global post-systolic shortening for Doppler: $\beta = 0.012$, $p = 0.951$, and speckle strain: $\beta = -0.165$, $p = 0.258$).

Table 3. Comparison of Segmental Strain and LV Global Strain by Doppler and by Speckle Tracking in the Acute Phase and After 10 Days

	Mean ± SD		
	Acute Phase	At Discharge	p Value
Segmental strain			
Segmental longitudinal peak systolic Doppler strain	−14.1 ± 7.8	−14.8 ± 7.2	0.029
Segmental longitudinal end systolic Doppler strain	−14.1 ± 7.5	−15.0 ± 6.7	0.008
Segmental longitudinal peak negative Doppler strain	−16.4 ± 6.7	−17.1 ± 6.1	0.012
Segmental longitudinal peak systolic speckle strain	−14.8 ± 9.6	−15.5 ± 7.0	0.087
Segmental longitudinal end systolic speckle strain	−14.5 ± 8.5	−15.0 ± 6.7	0.093
Segmental longitudinal peak negative speckle strain	−16.6 ± 7.7	−16.5 ± 6.5	0.732
Segmental circumferential peak systolic speckle strain	−15.6 ± 11.2	−16.4 ± 9.2	0.272
Segmental circumferential end systolic speckle strain	−15.1 ± 10.9	−15.6 ± 8.5	0.560
Segmental circumferential peak negative speckle strain	−17.0 ± 10.0	−18.1 ± 7.8	0.128
LV global strain			
LV global longitudinal peak systolic Doppler strain	−14.0 ± 3.0	−14.8 ± 3.2	0.033
LV global longitudinal end systolic Doppler strain	−14.0 ± 2.9	−15.0 ± 3.3	0.018
LV global longitudinal peak negative Doppler strain	−16.3 ± 2.8	−17.1 ± 2.8	0.019
LV global longitudinal peak systolic speckle strain	−15.1 ± 4.4	−15.6 ± 4.0	0.353
LV global longitudinal end systolic speckle strain	−14.7 ± 4.1	−15.2 ± 3.9	0.369
LV global longitudinal peak negative speckle strain	−16.8 ± 4.1	−16.6 ± 4.0	0.624

Comparisons are made by paired-samples Student *t* test.
LV = left ventricular.

Table 4. Segmental Circumferential and Longitudinal Speckle Strain Related to Myocardial Infarct Size by ceCMR

	None (Mean ± SD)	n	1% to 50% (Mean ± SD)	n	51% to 100% (Mean ± SD)	n
Peak systolic circumferential strain	−20.5 ± 9.6	79	−15.6 ± 9.9*	50	−4.7 ± 8.7*†	25
End systolic circumferential strain	−19.8 ± 10.0	79	−15.0 ± 9.5*	50	−5.4 ± 7.3*†	25
Peak negative circumferential strain	−21.2 ± 9.5	79	−16.8 ± 9.1*	50	−8.6 ± 5.5*†	25
Peak systolic longitudinal strain	−18.5 ± 8.0	251	−13.1 ± 9.4*	111	−8.0 ± 10.3*†	68
End systolic longitudinal strain	−17.6 ± 7.4	251	−13.0 ± 8.3*	111	−9.1 ± 8.8*†	68
Peak negative longitudinal strain	−19.6 ± 6.7	251	−15.2 ± 7.2*	111	−11.6 ± 7.4*†	68

Relationship between different groups based on the extent of myocardial infarction. Analyses are made by analysis of variance with post-hoc Scheffe tests. *$p < 0.05$ vs. none. †$p < 0.05$ vs. 1% to 50%.
n = number of segments; 1% to 50% = subendocardial infarction, 51% to 100% = transmural infarction.

Reproducibility. The reproducibility tests are presented in Table 7. The intraclass correlation coefficient was ranked as good or excellent in tests with segmental strain and excellent for all tests with global strain.

DISCUSSION

The present study shows that LV global peak systolic strain by speckle tracking should be preferred to estimate LV infarct size in patients with AMI treated with thrombolysis. Circumferential strain separates infarct size on a segmental level better than longitudinal strain.

Strain in AMI. The systolic phase of strain in normal myocardium is characterized by shortening, whereas the transmurally ischemic myocardium is characterized by systolic lengthening and post-systolic shortening. Longitudinal deformation mainly represents subendocardial contraction, whereas circumferential deformation mainly represents contraction of the midmyocardial and subepicardial layers. Therefore, longitudinal contraction is more sensitive to subendocardial ischemia and necrosis than circumferential contraction (10,15). Several studies have shown characteristic features of strain in ischemic myocardium. Peak systolic strain has been shown to be superior to TDI and wall motion analyses in detection of acute ischemic myocardium (2). The measurement of post-systolic shortening has not been able to detect viable myocardium nor describe necrosis with precision in acute and chronic myocardial infarction (3,4). In our study, we confirmed that post-systolic shortening had a poor correlation with LV infarct size. Vartdal et al. (12) showed that LV global peak negative strain correlated well with final infarct size in patients with AMI. Sachdev et al. (16) showed that decreasing peak systolic strain in chronic myocardial infarction correlated well with increasing transmurality of infarction. These studies have used different strain parameters and show the lack of consensus on whether strain should be measured as peak systolic, end systolic, or peak negative strain as a diagnostic and prognostic parameter in ischemic myocardium. Furthermore, these studies have only investigated longitudinal strain and not circumferential strain. Chan et al. (10) showed that circumferential strain was better than longitudinal strain in the differentiation of segments with subendocardial from transmural necrosis. We found that peak systolic, end systolic, and peak negative segmental longitudinal strain were able to discriminate normal from necrotic myocardium and differentiate subendocardial and transmural infarctions on a group

Table 5. ROC Analyses for Different Peak Systolic Strain Parameters

	AUC	Sensitivity (%)	Specificity (%)	Cutoff Value (Strain %)
Segmental circumferential strain, IS >50%	85	80	74	−13.3
Segmental longitudinal Doppler strain, IS >50%	75	81	53	−15.1
Segmental longitudinal speckle strain, IS >50%	75	82	53	−15.1
LV global longitudinal Doppler strain, IS >20%	85	75	73	−14.3
LV global longitudinal speckle strain, IS >20%	88	88	91	−12.2

Sensitivities and specificities to assess the ability of strain to identify transmural necrosis (IS >50%) in myocardial segments and to identify large LV global infarcts (IS >20%).
AUC = area under the curve; IS = infarct size; LV = left ventricular; ROC = receiver-operator characteristic.

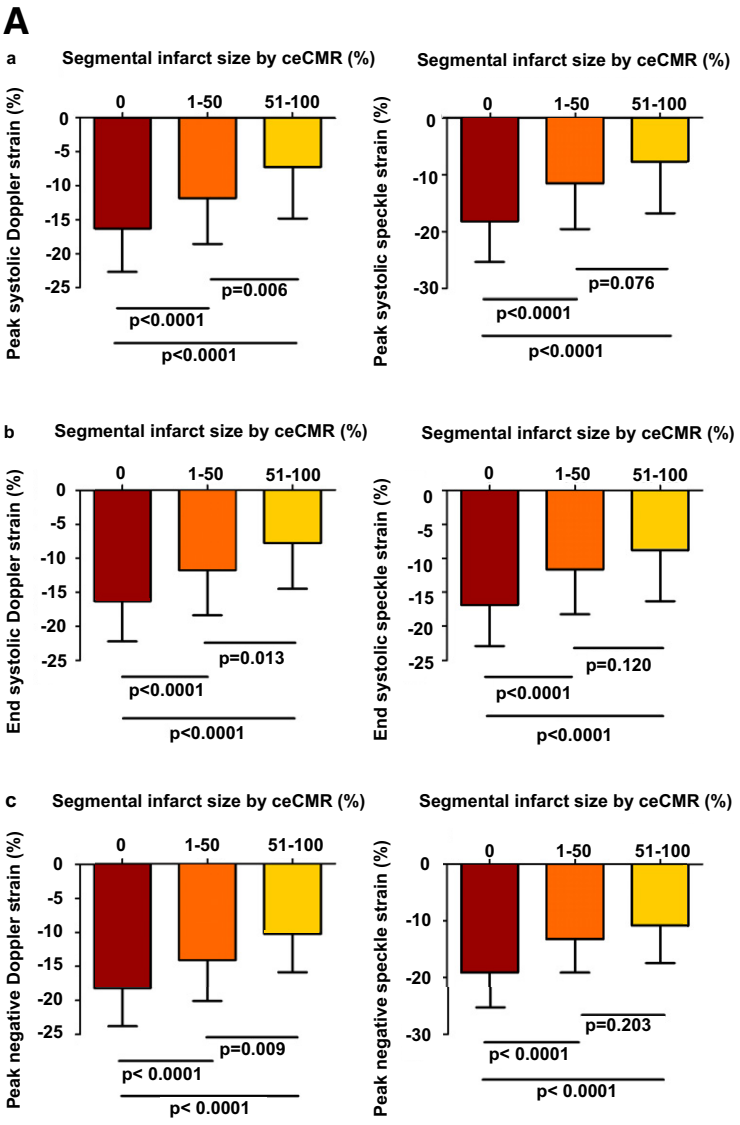

Figure 2. Relationship Between Segmental Infarct Size and Longitudinal Strain by Doppler and Speckle Tracking in Anterior and Inferior Myocardial Infarction

(A) These figures show that infarct size in each segment is inversely related to a decrease in corresponding **(a)** peak systolic, **(b)** end systolic, and **(c)** peak negative strain values regardless of strain method in patients with anterior myocardial infarction. **(B)** These figures show that infarct size in each segment is inversely related to a decrease in corresponding **(a)** peak systolic, **(b)** end systolic, and **(c)** peak negative strain values regardless of strain method in patients with inferior myocardial infarction. 0 = no infarction; 1-50 = 1% to 50% late enhancement by ceCMR (subendocardial infarction); 51-100 = 51% to 100% late enhancement by ceCMR (transmural infarction); ceCMR = contrast-enhanced cardiac magnetic resonance. *Continued on next page.*

level. These findings were regardless of whether strain was measured by Doppler or speckle tracking. However, because longitudinal strain in segments with subendocardial necrosis is more affected than circumferential strain, the deterioration of longitudinal strain is not as pronounced as for circumferential strain in segments with transmural necrosis. As a consequence, the measurement of circumferential strain in the present study separated segments with transmural necrosis from segments with subendocardial necrosis better than longitudinal strain. This is important because patients with subendocardial necrosis in the region of the infarct-related artery may benefit from early revascularization after thrombolytic treatment.

Some of the differences between LV global strain by Doppler and speckle tracking in the present study may be caused by the effect of myocardial

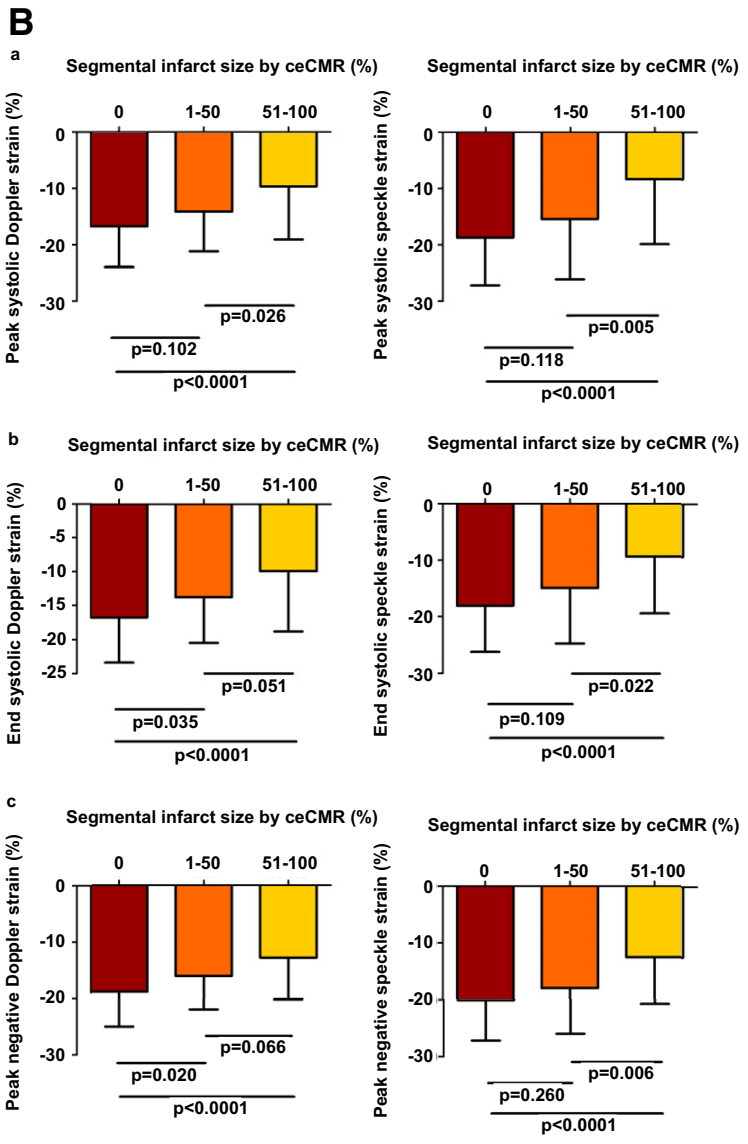

Figure 2. Continued

stunning and ischemia, which was detected by Doppler strain but not by longitudinal speckle strain. The latter may be caused by methodological differences. Doppler strain may identify the most pathologic strain within 1 segment, whereas speckle strain is based on the sum of strain values within the whole segment. Thus, Doppler strain values in the acute phase of AMI may be slightly lower as a consequence of myocardial stunning and ischemia, which affects the correlation between strain and final infarct size.

We found that injury is separated slightly better by speckle strain in anterior than in inferior myocardial infarction. These findings may be caused by reduced lateral resolution in echocardiograms of the LV. Speckle strain may be less sensitive for diagnosing injury in the posterior myocardial circulation as shown by Hanekom et al. (17). By assessing LV global strain, some of these difficulties may be overcome. This parameter correlates well with LV infarct size in both inferior and anterior infarcts and may separate large from small LV necrosis as shown in the present study and by Vartdal et al. (12). Because strain on the segmental level has a relatively high standard deviation, it seems necessary to assess LV global strain for the most accurate assessment of myocardial injury in AMI. Although circumferential strain was able to separate subendocardial from

Table 6. Correlations Between Infarct Size by ceCMR and Different LV Global Strain Parameters

	Correlation Coefficient (β), Acute Phase	p Value
LV global longitudinal peak systolic Doppler strain	0.64	<0.0001
LV global longitudinal end systolic Doppler strain	0.64	<0.0001
LV global longitudinal peak negative Doppler strain	0.67	<0.0001
LV global longitudinal peak systolic speckle strain	0.76	<0.0001
LV global longitudinal end systolic speckle strain	0.72	<0.0001
LV global longitudinal peak negative speckle strain	0.71	<0.0001

Standardized beta-coefficients are made by linear regression analysis.
ceCMR = contrast-enhanced cardiac magnetic resonance; LV = left ventricular.

transmural necrosis better than longitudinal strain on a segmental level, the addition of circumferential strain in the assessment of LV global strain did not increase the diagnostic precision. This finding is most probably caused by improved circumferential strain values in segments without necrosis or with subendocardial necrosis compared with longitudinal strain. Circumferential strain may compensate for the reduced longitudinal strain in these segments. All 3 strain parameters regardless of method could assess LV global strain with a significant correlation to LV infarct size, but the correlation was slightly higher for speckle strain. Thus, LV global speckle strain separated small and large LV infarcts with better precision than strain by Doppler.

Reproducibility. Our results show that strain on a segmental level by Doppler and speckle tracking have good or excellent reproducibility in patients with AMI. Speckle strain showed better reproducibility for the same observer and between different observers. This may be explained by several differences between the methods. The measurement of Doppler strain is angle dependent. Speckle strain may measure strain independent of angle and quantify contraction in a longitudinal, circumferential, and radial direction. Furthermore, speckle strain measures strain in a whole segment by generating an average of strains. Doppler strain is measured from a representative region of interest within the segment and does not reflect strain from the whole segment. This may lead to greater variability in AMI. Doppler strain is susceptible to signal noise artifacts and drifting, which may increase variability. By assessing LV global strain, reproducibility is strengthened for both Doppler and speckle strain with no differences between the 2 methods. These findings support that LV global strain should be assessed to describe LV injury in AMI. Furthermore, by combining analysis of segmental infarct size by circumferential strain and LV infarct size by peak systolic speckle strain, we found that speckle strain has several diagnostic advantages over Doppler strain and should be the preferred method in acute STEMI.

Study limitations. It has been shown that LV infarct size is greater if measured in the acute phase

Table 7. Reproducibility of Strain for All Segments and LV Global Strain

	Doppler Strain		Speckle Strain	
	Intraobserver	Interobserver	Intraobserver	Interobserver
Reproducibility of strain for all segments				
Peak systolic longitudinal strain	0.80 (0.76–0.84)	0.74 (0.68–0.79)	0.88 (0.85–0.90)	0.79 (0.74–0.83)
End systolic longitudinal strain	0.80 (0.75–0.84)	0.70 (0.64–0.75)	0.89 (0.86–0.91)	0.77 (0.72–0.81)
Peak negative longitudinal strain	0.77 (0.72–0.81)	0.71 (0.65–0.76)	0.91 (0.89–0.93)	0.82 (0.78–0.85)
Peak systolic circumferential strain			0.84 (0.78–0.89)	0.82 (0.74–0.87)
End systolic circumferential strain			0.83 (0.77–0.88)	0.82 (0.74–0.87)
Peak negative circumferential strain			0.83 (0.76–0.88)	0.82 (0.75–0.87)
Reproducibility of LV global strain				
LV global peak systolic strain	0.94 (0.87–0.98)	0.89 (0.75–0.95)	0.92 (0.80–0.97)	0.85 (0.67–0.94)
LV global end systolic strain	0.95 (0.88–0.98)	0.87 (0.71–0.95)	0.93 (0.84–0.97)	0.85 (0.66–0.94)
LV global peak negative strain	0.93 (0.84–0.97)	0.88 (0.72–0.95)	0.96 (0.90–0.98)	0.91 (0.78–0.96)

Values are intraclass correlation coefficient (ICC). The ICC was considered poor when ICC <0.4, fair when ICC = 0.40 to 0.59, good when ICC = 0.60 to 0.74, and excellent when ICC ≥0.75. Numbers in parentheses are 95% confidence intervals.
LV = left ventricular.

compared with months later (18). This phenomenon may be explained by infarct-associated edema in the acute phase, which comprises a larger volume than collagenous scar tissue in the chronic phase. In our study, all 3 strain parameters measured by both ultrasonic techniques showed similar good relations to infarct size. Furthermore, our findings are in accordance with previous findings of a good relation between strain in AMI and chronic infarct size (12).

Drug therapy could affect remodeling and potentially affect long-term infarct size. However, we found that there were nearly no differences in drug treatment between the acute phase and long-term follow-up, so the correlation between strain values in the acute phase and infarct size should not be significantly affected by drug treatment.

CONCLUSIONS

On a segmental level, circumferential strain separated transmural from subendocardial necrosis better than longitudinal strain in the acute phase in patients with STEMI. Our findings suggest that in the acute phase in patients treated with thrombolysis, LV global peak systolic speckle strain should be the preferred method for diagnosing the degree of LV injury.

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