Global longitudinal strain measured by two-dimensional speckle tracking echocardiography is closely related to myocardial infarct size in chronic ischaemic heart disease

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ABSTRACT

2D-STE (two-dimensional speckle tracking echocardiography) is a novel echocardiographic modality that enables angle-independent assessment of myocardial deformation indices. In the present study, we tested whether peak systolic $\varepsilon_{||}$ (longitudinal strain) values measured by 2D-STE could identify areas of MI (myocardial infarction) as determined by CE MRI (contrast-enhanced magnetic resonance imaging). Conventional echocardiographic apical long-axis recordings were performed in 38 patients, 9 months after a first MI. Peak systolic $\varepsilon_{||}$ measured by 2D-STE in 16 left ventricle segments was compared with segmental infarct mass and transmurality assessed by CE MRI. Segmental values were averaged to global and territorial values for assessment of global function and myocardial function in the coronary distribution areas. CE MRI identified transmural infarction in 27 patients, and a mean infarct size of 36 ± 25 g. Peak systolic $\varepsilon_{||}$ correlated with the infarct mass at the global level ($r = 0.84$, $P < 0.001$). A strain value of -15% identified infarction with 83% sensitivity and 93% specificity at the global level and 76% and 95% at the territorial level, and a strain value of -13% identified transmural infarction with 80% sensitivity and 83% specificity at the segmental level. Global infarct mass correlates with the wall motion score index ($r = 0.70$, $P < 0.001$), and left ventricular ejection fraction measured by MRI or echocardiography ($r = -0.71$ and -0.58, both $P < 0.001$). In chronic infarction, peak systolic $\varepsilon_{||}$ measured by 2D-STE correlates with the infarct mass assessed by CE MRI at a global level, and separates infarcted from non-infarcted tissue. Global strain is an excellent predictor of myocardial infarct size in chronic ischaemic heart disease.

INTRODUCTION

Accurate assessment of myocardial function is an important clinical issue in patients with acute MI (myocardial infarction) or chronic ischaemic LV (left ventricular) dysfunction [1]. Evaluation of regional function adds important diagnostic and prognostic information to the global assessment of LVEF (LV

Key words: ischaemic heart disease, longitudinal strain, myocardial infarction, two-dimensional speckle tracking echocardiography.
Abbreviations: AUC, area under the curve; $\varepsilon_{||}$, longitudinal strain; CE MRI, contrast-enhanced magnetic resonance imaging; CK MB, creatine kinase MB; 2D-STE, two-dimensional speckle tracking echocardiography; LAD, left anterior descending artery; LCX, left circumflex artery; LV, left ventricular; LVEF, LV ejection fraction; MI, myocardial infarction; MRI, magnetic resonance imaging; PCI, percutaneous coronary intervention; PSSI, post-systolic shortening index; RCA, right coronary artery; ROC, receiver operating characteristic; ROI, region of interest; TDI, tissue Doppler imaging; WMSI, wall motion score index.
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ejection fraction) and volume measurements [2]. Analyses of regional endocardial motion and local wall thickening and thinning characteristics using echocardiography or MRI (magnetic resonance imaging) have been used to evaluate regional myocardial function. Although crucial to clinical decision-making, echocardiographic assessment of regional LV function based on qualitative visual analysis is observer-dependent [3]. Despite this, echocardiography is the standard technique for assessment of regional myocardial dysfunction and LVEF. The global LV function, as calculated by LVEF, can be regarded as the sum of regional LV functions. Strain has been introduced as a clinical index of regional [4–7] and global [8] myocardial function. Assessment of regional systolic function after MI by strain quantification using tagged MRI or TDI (tissue Doppler imaging) has been validated and assessed in clinical and experimental studies [9–11]. Assessment of peak systolic strain from each LV segment provides detailed knowledge about myocardial function. This method is, however, time consuming and has so far had limited use in clinical practice. To eliminate the problem of angle-dependency in TDI, the measurement of strain based on 2D-STE (two-dimensional speckle tracking echocardiography) has been developed [12]. This new method enables assessment of regional myocardial deformation from conventional B-mode echocardiographic images [13]. 2D-STE has held promise as an accurate method for assessing LV function in patients with ischaemic LV failure [14] and hypertrophic cardiomyopathy [15], and has recently been validated against tagged MRI and sonomicrometry [16–18].

A recent study by Vartdal et al. [8] reports that global strain assessed by TDI in the acute phase of a MI can predict the final infarct size as measured by MRI [8]. We hypothesized that peak systolic ε_{ll} (longitudinal strain) measured by 2D-STE provides additional information regarding the extent of infarct mass and myocardial function when compared with the LVEF and WMSI (wall motion score index) in patients with chronic MI. Peak systolic ε_{ll} from all LV segments was averaged into a global strain index and a territorial strain index was calculated in order to assess myocardial function in each coronary perfusion area. CE MRI (contrast-enhanced MRI) was used as a reference method for determination of the infarct extent.

MATERIALS AND METHODS

Patients

Thirty-eight patients (55 ± 10 years; ten women) and 15 healthy controls (46 ± 12 years, $P < 0.01$; six women) were included in the present study. The patients suffered from acute ST elevation MI and were treated with PCI (percutaneous coronary intervention). The coronary angiograms were interpreted by an experienced operator.

Table 1 Patient characteristics

Values are means $\pm$ S.D or numbers. ACE, angiotensin-converting enzyme. A2, angiotensin II type 1 receptor.

Characteristics	Value
n	38
Age (years)	55 ± 10
Gender (*n*) (male:female)	28:10
History of hypertension (*n*)	10
History of diabetes mellitus (*n*)	1
History of hypercholesterolaemia (*n*)	3
History of transient ischaemic attack (*n*)	1
Medication (*n*)	
β-Blocker	38
Antiplatelet therapy	38
Statin	38
ACE inhibitor/A2-blocker	30
Heart rate (beats/min)	57 ± 10
Systolic blood pressure (mmHg)	126 ± 25
Diastolic blood pressure (mmHg)	79 ± 13
LVEF by MRI (%)	58 ± 11
LVEF by echocardiography (%)	49 ± 10
LV end-diastolic volume by MRI (ml)	157 ± 53
LV mass by CE MRI (g)	159 ± 40
Infarct mass by CE MRI (g)	36 ± 25
Infarcted segments (*n*)	7.9 ± 3.5
Angiographic LAD stenosis (*n*)	37
Angiographic LCX stenosis (*n*)	11
Angiographic RCA stenosis (*n*)	10
Peak CK MB (μg/l)	301 ± 188
Peak troponin I (μg/l)	61 ± 73

At inclusion, none of the patients was in cardiogenic shock or had previous MI; however, chronic occlusion of the RCA (right coronary artery) was found in one patient. The clinical data are presented in Table 1. All patients were examined by echocardiography and cardiac MRI at follow-up, 9.2 ± 5.7 months after PCI. The echocardiographic study was typically performed within 4 h of the MRI. Patients were haemodynamically stable during the studies, and there were no signs or history of new coronary events after the initial PCI. Serum was collected before PCI and 6, 12 and 24 h after the PCI for measurement of troponin I and CK MB (creatine kinase MB). The peak value for each marker was used in the statistical analysis.

As a reference group for normal values, 15 healthy volunteers were examined by echocardiography. All study subjects were in sinus rhythm and had a QRS-width < 120 ms. No one had significant valvular dysfunction as defined by echocardiography.

MRI

MRI was performed using either of two 1.5 Tesla units (Magnetom Vision Plus or Magnetom Sonata; Siemens) and a phased array body coil. Breath-hold cine images

with a time resolution of 50 ms or less were acquired in short-axis and long-axis views, and LV volumes and LVEF were calculated. Late-enhancement images were obtained 10–20 min after intravenous injection of 0.1 mmol/kg of body weight gadopentetate dimeglumine (Magnevist; Schering) in long-axis views, as well as multiple short-axis slices covering the left ventricle (slice thickness 7 mm, inter-slice gap 3 mm). A breath-hold segmented magnetization-prepared turbo gradient echo sequence was used with an inversion time of 210–260 ms.

The LV myocardium was manually divided into 17 segments [19] and the infarcted as well as the total myocardial area of each segment was drawn (PACS; Sectra) based on the short-axis views. Areas with pixel intensities more than 2 S.D. above the mean pixel intensity of normal myocardium of the same slice were considered infarcted [20,21]. The total myocardial volume, and the absolute and relative infarct volumes, were calculated for each segment. The 17th segment (apex) was assigned to the LAD (left anterior descending artery) perfusion area for comparison with echocardiographic data. The myocardial and infarct masses were obtained from the volume measurements by multiplying by 1.05 g/ml [22], and the myocardial mass was used for subsequent analyses [23].

The transmural infarct extent of each segment was assessed. Subendocardial infarcts were defined as less than 50 % transmurality, and segments with 50 % or more of the wall thickness involved were considered transmurally injured [20].

Echocardiography

The study examinations were performed with either a Vivid 5 scanner (seven examinations) or a Vivid 7 scanner (46 examinations) (GE Vingmed Ultrasound), using a phased-array transducer. Three consecutive heart cycles from the three standard apical planes (four-chamber, two-chamber and long-axis) were obtained by conventional two-dimensional greyscale echocardiography, and the average frame rate was 58 ± 20 frames/s. The digital loops were stored and analysed by EchoPac software (GE Vingmed Ultrasound). LVEF was assessed by the modified Simpson's rule.

To measure strain, a new algorithm calculating the myocardial deformation from ultrasound speckles (2D-STE) [13,14,16] was used. This method assesses myocardial deformation based on greyscale images, and is semi-automatic and angle-independent. The principles of strain have been described previously [24,25]. The peak systolic ε_{ll} values, peak positive systolic strain (termed early stretch) and PSSI (post-systolic shortening index) in a 16 segment LV model [19] were used in the present study. End systole was defined as aortic valve closure in the apical long-axis view. PSSI was calculated as the ratio of post-systolic strain divided by the maximal strain [26]. The regions of interest were manually outlined by marking the endocardial borders at the mitral annulus level and at the apex on each digital loop, and adjusted when the automatic tracking was considered suboptimal by visual or automated assessment. Conversion from 18 segments into a 16 segment model was performed by averaging the strain values in the corresponding apical segments in the apical long-axis and four-chamber views. The total procedure time was typically less than 5 min for all segments.

Wall motion

Wall motion was assessed with a 16 segment model according to the American Society of Echocardiography [27]. WMSI was calculated as the sum of scores over the number of analysed segments. A territorial WMSI was calculated for each coronary perfusion area.

Analyses

In the present study, we compared peak systolic ε_{ll} values and the WMSI of each segment with the infarct mass and level of transmurality as assessed by CE MRI of the corresponding segments (Figure 1). Analyses (speckle tracking, WMSI and MRI) were performed independently by three different observers, blinded to the other results. Territorial indices were calculated by averaging values from segments assigned to each of the three major coronary arteries. All anterior and anteroseptal segments (including the apical septal segment) were assigned to the LAD and the basal and mid inferoseptal, and all inferior segments to the RCA. All inferolateral and anterolateral segments (including the apical lateral segment) were assigned to the LCX (left circumflex artery) perfusion area according to the recommendations from American Heart Association [19]. Global strain values were obtained by averaging all segmental peak systolic strain values in a 16 segment model [8]. Correlations between the LVEF measured by echocardiography or MRI and the global infarct mass were assessed. Echocardiographic studies were performed repeatedly by two independent observers on ten randomly selected patients. Reliability analyses of strain values revealed a Cronbach's α of 0.90 for inter-observer, and 0.95 for intra-observer variation at the global level, 0.92 and 0.97 at the territorial level and 0.81 and 0.92 at the segmental level.

Statistical analysis

Values are presented as means $\pm$ S.D. Differences between groups were analysed with one-way ANOVA. Bonferroni correction was applied for post-hoc tests and for multiple regression analyses. Values were compared for correlation by regression analysis using a least-squares method. We performed a multivariate regression analysis to find the best determinant of the global strain.

ROC (receiver-operating characteristic) curves were constructed, and AUCs (areas under the curves) were measured to determine cut-off values for optimal

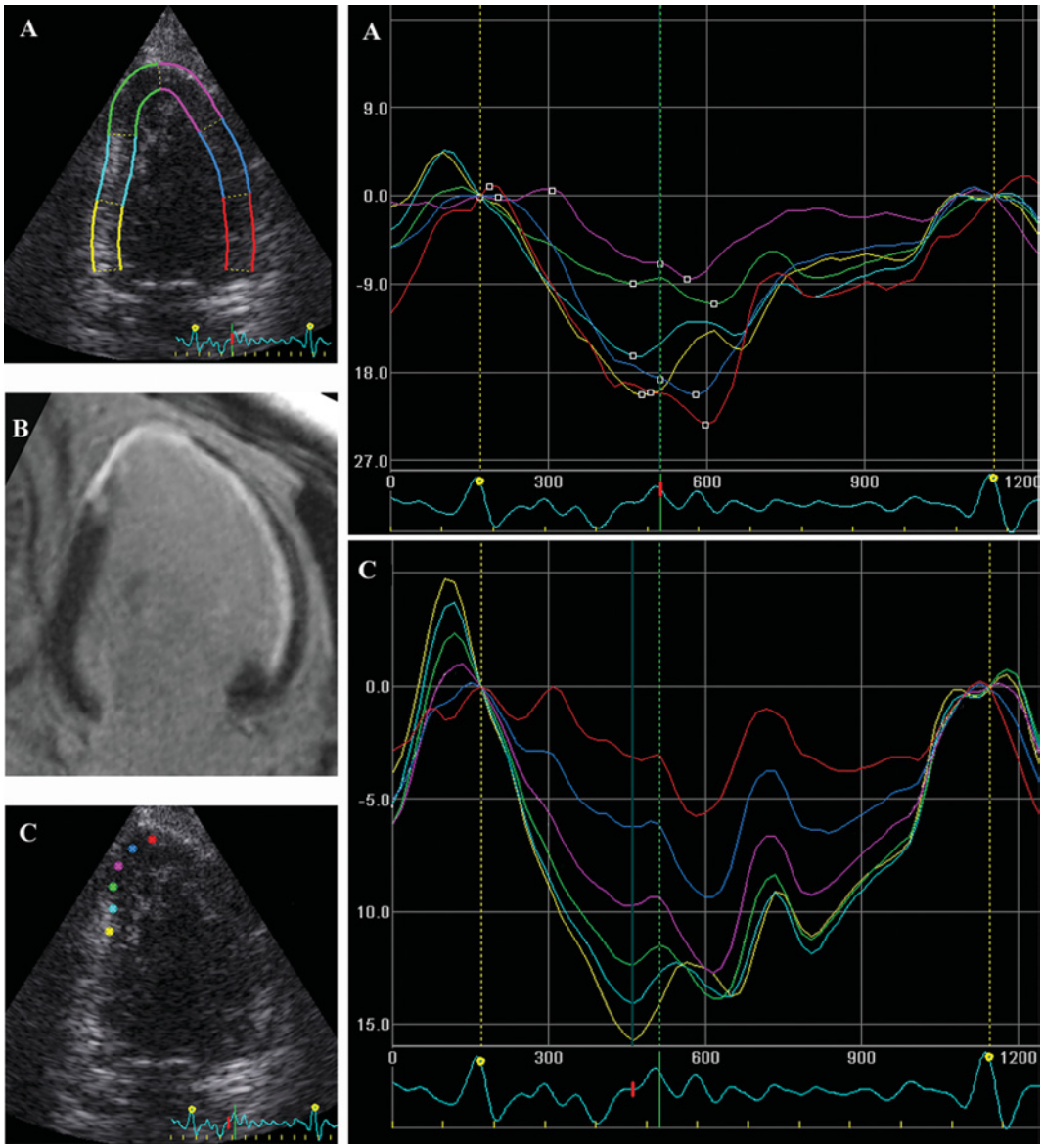

Figure 1 Strain curves

Segmental ε_{II} curves measured by 2D-STE from an apical two-chamber view (A and C). A corresponding CE MRI image from the same patient is shown for comparison (B). In the present study, MRI data were obtained from short-axis views. (A) displays segmental strain curves. The green and purple strain curves are derived from transmural-infarcted segments, the red and blue curves represents subendocardial-infarcted segments and the yellow and cyan curves are from non-infarcted segments. In (C), the coloured asterisks correspond to the strain curves (right) within one segment of transmural infarct (apical inferior segment). Peak systolic strain indicates increasing shortening as the infarct transmurality decreases.

sensitivity and specificity. The ROC analyses were set to identify infarcts of $\geqslant 30$ g at the global level, an infarct size that has been shown to predict poor prognosis [2]. Limits were set to identify $\geqslant 5$ g by territorial strain and transmural infarct at the segmental level. For all statistical comparisons, $P < 0.05$ was considered significant. Statistical analyses were performed using SPSS version 14.

The study was approved by the Regional Committee for Medical Research Ethics (REK Sør, Oslo, Norway), and all subjects gave written informed consent.

RESULTS

The LVEF assessed by echocardiography was 49 ± 10 % and the LVEF assessed by MRI was 58 ± 11 % (Table 1). The average LV mass was 159 ± 40 g, of which 36 ± 25 g was infarcted, 7.9 ± 3.5 segments in each patient were affected by ischaemic injury and 2.6 ± 2.3 segments had transmural infarction. Subendocardial infarct was identified in 206 segments, transmural infarct in 94 segments and no infarct in 308 segments, as measured by

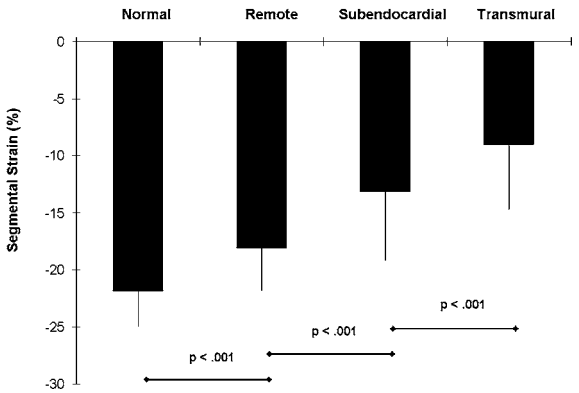

Figure 2 Segmental strain differs with the level of transmurality

Normal segments (healthy volunteers, $n = 215$), non-infarcted segments (remote, $n = 263$), segments with subendocardially infarcted segments ($n = 191$) and transmurally infarcted segments ($n = 95$) are shown. Values are means $\pm$ S.D.

CE MRI. The average transmural extent was 23 ± 15 % in subendocardial-infarcted segments and 70 ± 16 % in transmural-infarcted segments.

Peak systolic $\varepsilon_{||}$ could be measured in 764 segments (90 %). The remaining segments were excluded due to reverberations ($n = 18$), valvular interference ($n = 2$), tracking difficulties ($n = 22$) and poor image quality ($n = 42$). These segments were 10 % of the LAD segments, 6 % of the RCA segments and 14 % of the LCX segments and represented 13 % of the basal segments, 8 % of the mid-ventricular segments and 7 % of the apical segments. Territorial and global strain values were analysed in all subjects. The averaged strains in normal hearts were similar in all of the distribution areas (LAD, -21.5 ± 1.7 %; RCA, -21.5 ± 2.8 %; and LCX, -21.7 ± 2.2 %), as well as for the whole left ventricle (-21.7 ± 1.6 %; P = not significant). The average end-diastolic volume was 157 ± 53 ml in patients and 106 ± 33 ml in normal hearts ($P < 0.01$).

Segmental analyses

Peak systolic $\varepsilon_{||}$ measured by 2D-STE differentiated significantly ($P < 0.001$) between non-infarcted segments (-18.1 ± 3.9 %), segments with subendocardial infarcts (-13.1 ± 6.1 %) and transmurally infarcted segments (-9.0 ± 5.8 %) (Figure 2). A segmental strain value of -13 % identified transmural infarction with a sensitivity of 80 % and a specificity of 83 % (Table 2), and a value of -16 % predicted a non-transmural infarction with a sensitivity of 77 % and specificity of 80 %, the AUC was 0.86.

The extent of myocardial scar varies within the LV segments. Figure 1(C) exemplifies the heterogeneity of the transmural extent within one LV segment. The MRI image (Figure 1B) showed one segment (the apical inferior segment) that may consist of a mixture of transmural and non-transmural areas. The Figure further reveals an abrupt transition from 100 % transmural to non-infarcted regions within the same segment and demonstrated the spectra of myocardial function within one infarcted LV segment. The cyan to red traces are all within the same segment and demonstrated peak systolic strain from -13 % to -3 %. As the ROI (region of interest)/sample volume by the 2D-STE method consisted of the whole segment, the resultant segmental strain value represented an average of strain values from more or less scarred regions within the LV segments. This demonstrated the potential and accuracy of the 2D-STE method to analyse even smaller areas of the left ventricle rather than a whole segment. We analysed strain within a ROI in an area with 100 % infarct from each patient by CE MRI (Figure 1C). In these areas the average peak systolic $\varepsilon_{||}$ was -5.4 ± 6.7 %, with a marked initial systolic stretching of 3.7 ± 3.5 %.

There were significant differences in early systolic stretching, PSSI and WMSI between transmural-infarcted segments, subendocardial-infarcted segments and normal segments. The average peak systolic strain value in segments with a WMSI of 3–4 was -7.2 %. The average transmurality of a- and dys-kinetic segments was 58 ± 26 %.

Territorial analyses

Territorial strain reflects the averaged peak systolic strain value of segments assigned to the different theoretical coronary distribution areas [19]. The average strain in non-infarcted and in territories with any sign of infarct (including subendocardial and transmural segments) for the different territories was -18.0 ± 1.8 % compared with -11.1 ± 4.1 % for LAD, -18.2 ± 2.0 % compared with -13.7 ± 2.7 % for RCA and -17.6 ± 2.7 %

Table 2 Segmental analyses

Segmental values (means $\pm$ S.D.) for non-infarcted, subendocardial-infarcted (sub-endo) and transmural-infarcted (transmural) segments (all $P < 0.01$ compared with normal).

	Non-infarcted	Sub-endo	Transmural	Sensitivity	Specificity	AUC
$\varepsilon_{||}$	-18.1 ± 3.9%	-13.1 ± 6.1%	-9.0 ± 5.8%	0.80	0.83	0.87
WMSI	1.1 ± 0.3	1.6 ± 0.7	2.4 ± 0.9	0.80	0.82	0.85
PSSI	1 ± 5	13 ± 22	24 ± 30	0.55	0.85	0.74
Early stretch	0.3 ± 0.9%	0.7 ± 1.6%	1.4 ± 1.9%	0.56	0.78	0.68

Table 3 Territorial analyses

Territorial values (means ± S.D.) for non-infarcted territories and territories with any infarct (all $P < 0.01$).

	Non-infarcted	Infarcted	Sensitivity	Specificity	AUC	Accuracy
ε_{ll}	− 18.2 ± 2.0%	− 12.4 ± 3.6%	0.76	0.95	0.93	0.85
WMSI	1.2 ± 0.2	1.7 ± 0.4	0.75	0.89	0.89	0.84
PSSI	1 ± 2	11 ± 14	0.75	0.85	0.87	0.81
Early stretch	0.1 ± 0.2	0.8 ± 0.8	0.75	0.74	0.80	0.75

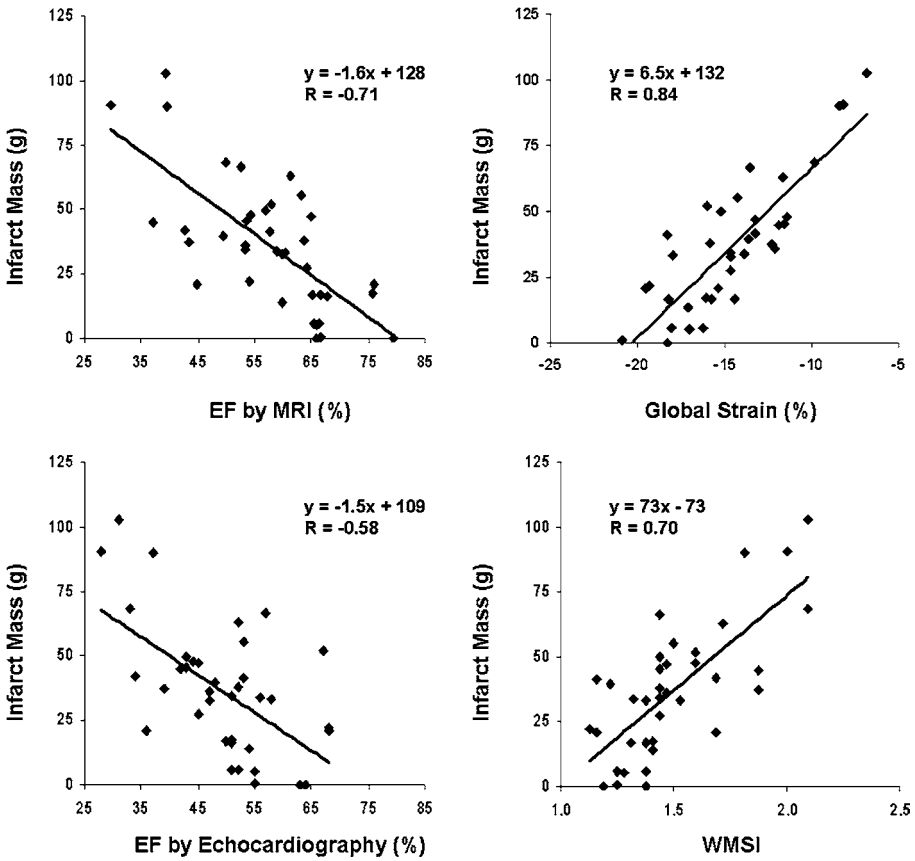

Figure 3 Correlations between the global infarct mass and global indices of LV function

LVEF measured by MRI (upper left-hand panel), LVEF measured by echocardiography (lower left-hand panel), global strain (upper right-hand panel) and WMSI (lower right-hand panel).

compared with − 14.5 ± 3.1 % for LCX ($P < 0.01$). Correlations between territorial strain and infarct mass were significant for all three distribution areas, $r = 0.73$ (LAD), $r = 0.63$ (LCX) and $r = 0.66$ (RCA) (all $P \leqslant 0.001$).

Average values for territorial WMSI, early stretch and PSSI for non-infarcted and infarcted territories are displayed with the results from ROC analyses (Table 3).

Global analyses

Peak systolic segmental strains were averaged into a global strain value for each study object as a parameter for global LV performance. The average global strain was − 21.7 ± 1.6 % in normal individuals and − 14.3 ± 3.1 % in patients with MI ($P = 0.01$). Global strain showed a good correlation with global infarct mass ($r = 0.84$, $P < 0.001$) (Figure 3). By ROC analysis, a cut-off value of − 15 % identified clinically significant infarction with 83 % sensitivity and 93 % specificity (Figure 4).

The average global early stretch and PSSI was 0.1 ± 0.2 % and 0.2 ± 0.6 in normal, and 0.7 ± 0.6 % and 9.1 ± 7.7 in infarcted left ventricles ($P < 0.001$). Results from global ROC analyses and correlations with global infarct mass are shown in Table 4. Correlations between global strain and peak troponin I and CK

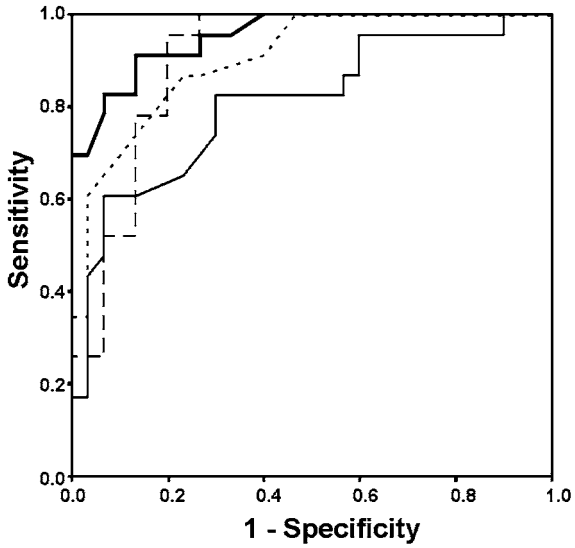

Figure 4 ROC curves of global indices to identify infarcts

ROC curves estimating the sensitivity and specificity to identify an infarct mass of $\geqslant 30$ g. Global strain (thick solid line), WMSI (dotted line) and LVEF by MRI (dashed line) or echocardiography (thin solid line) are shown. Sensitivity, specificity and AUC are shown in Table 4.

Table 4 Global analyses

Global sensitivities, specificities and AUC from ROC analyses and accuracy (all $P < 0.01$).

	Correlation	Sensitivity	Specificity	AUC	Accuracy
ε_{II}	0.84	0.83	0.93	0.95	0.87
WMSI	0.70	0.69	0.87	0.90	0.81
LVEF by MRI	−0.71	0.78	0.80	0.90	0.77
LVEF by echo	−0.58	0.70	0.74	0.81	0.75
CK MB	0.68	0.70	0.67	0.77	0.77
Troponin I	0.73	0.70	0.67	0.77	0.81
PSSI	0.85	0.83	0.87	0.94	0.85
Early stretch	0.67	0.72	0.80	0.82	0.77

MB were $r = 0.70$ ($P < 0.01$) and $r = 0.62$ ($P < 0.01$) respectively.

A multivariate regression analysis was performed to identify the determinants of global strain. Global infarct mass was the only significant predictor of global strain, whereas LVEF, volume, blood pressure, heart rate and age were not significant in a multivariate analysis.

DISCUSSION

The present study demonstrates that the LV longitudinal function assessed by peak systolic ε_{II} measured by 2D-STE correlates well with the infarct mass as seen on CE MRI over a wide range of infarct sizes and anatomical locations. This relationship was found at three levels: the global, the territorial and the segmental levels. The best echocardiographic parameter to predict the global infarct mass was global strain. This novel parameter was found to be superior to the more traditional echocardiographic methods such as LVEF and WMSI. Strain values could be obtained in approx. 90% of all LV segments, demonstrating that the 2D-STE technique is feasible for most patients. Strain values measured by the novel 2D-STE technique revealed reduced contractility in LV segments with greater transmural scar extent after MI. A strain value of −15% revealed optimal sensitivity and specificity for identification of $\geqslant 5$ g infarct mass at the territorial and $\geqslant 30$ g infarct mass at the global level, and the optimal cut-off value for identifying transmural infarction was −13% at the segmental level.

Global indices

2D-STE allows fast evaluation of regional ε_{II} values from all LV segments, and these can be averaged to global strain. Global strain is a relatively new parameter for assessment of LV function [14,28] and tends to predict the infarct mass better than established indices of global function such as LVEF and WMSI. By multiple regression analysis, infarct mass was the only significant predictor of global strain in the present study.

LVEF can be regarded as the sum of all LV systolic deformation. Impairment of LVEF, however, requires a decrease in several LV segments and might not be present in patients with relatively limited myocardial scars. The findings of our present study are in accordance with previous studies. In a recent study by Vartdal et al. [8], global strain measured by TDI immediately after PCI was found to be superior to LVEF for predicting final infarct mass in patients with acute MI. These findings support the idea that global strain can serve as an important new marker of LV function both in the acute and chronic phase of MI. As expected, the relationship between infarct mass and strain was better in the present study. This is probably due to areas of stunned myocardium that affects strain measurements in the acute MI patients studied by Vartdal et al. [8]. We do not attribute the better relationship to the recent 2D-STE technique used in the present study. However, a direct comparison of strain assessment by TDI and 2D-STE is needed in future studies. Ingkanisorn et al. [23] found a similar correlation between LVEF and infarct mass as reported in our present study. Although their study related infarct mass to the clinical measurements, our present study compares the anatomical and functional consequences of MI.

Global PSSI is reported for the first time in the present study. This index was demonstrated to be an excellent marker for the identification of myocardial infarcts. Early systolic stretching during isovolumic contraction had less sensibility and sensitivity compared with global systolic strain and PSSI to identify myocardial infarcts.

The latter might be due to the differences in scar tissue properties in chronic revascularized and acute ischaemic myocardium.

Territorial indices

Territorial strain is introduced as a new parameter in the present study. Assignment of segments to coronary distribution territories (LAD, LCX and RCA) was performed in accordance with established recommendations [19]. An averaged strain value from each of the three major vascular beds exhibited a significant correlation with the infarcted mass in the same regions. This correlation was strongest within the LAD area, possibly due to the large number of LAD infarcts in our present study, but also because there is less interindividual variation in the LAD distribution area [29].

Territorial strain is a parameter for regional functional analysis, and provides the advantage of assessing the regional consequences of infarcts at the level of the coronary vasculature. Furthermore, it has the potential of predicting the arterial branches responsible for ischaemic injury. The distribution of infarcted tissue within the coronary territories is heterogeneous, and the average value reflects a mixture of infarcted and normal myocardium. In the present study, the schematic and theoretical coronary distribution as recommended by the American Heart Association was used [19]; we did not correct for individual variation in artery dominance. In the clinical setting, the coronary distribution of the individual patient is unknown, and a theoretical scheme is therefore a useful tool for estimating the artery at risk. Assessment of territorial myocardial function might be a useful clinical tool in a pre-revascularized population. A more comprehensive study will be needed to address this question, including more LCX- and RCA-related myocardial infarcts.

Segmental strain and transmurality

As expected, peak systolic ε_{ll} was significantly reduced in infarcted segments compared with remote myocardial segments. More than 50 % transmurality reduces the likelihood of increased contractility after revascularization and is, therefore, considered to be an important threshold [20]. Strain values in remote segments were found to be significantly reduced compared with segmental strain in normal subjects. This has also been shown in a MRI-tagging study [7], and is most probably due to differences in LVEF and LV diameter [10]. We found that segmental WMSI and strain had a similar sensitivity and specificity in identifying transmural infarcts.

In previous echocardiographic studies, segmental strain measurements have been compared with indirect signs of MI, such as reduced wall thickening indices or angiography and/or ECG signs of myocardial damage [7,14,30]. Due to the angle dependency of TDI, most studies did not include all LV segments. In our present study, echocardiographic ε_{ll} was directly compared with the infarct mass assessed by CE MRI in a 16 segment model. A recent study [31] compared circumferential and radial strain with infarct mass assessed by CE MRI at the segmental level. Our present indicate a slightly better ability to identify transmural infarct distribution by ε_{ll}. A head-to-head comparison is needed to address this question.

Early systolic stretching was a marked finding when strain was measured in smaller regions from within the area of 100 % infarction. Early systolic stretching and post-systolic shortening have previously proved to be markers for viability in acute myocardial infarcts [32] and in a stress echocardiographic study [26]. In the present study of chronic MI, there were significant differences between segments of different infarct transmurality. Both markers had good specificity, but less good sensitivity to identify transmural infarction.

Clinical perspectives

STE is based on conventional greyscale echocardiographic images, and provides segmental analyses of 16 LV segments. Strain measurements with 2D-STE provide regional and global information about the myocardial deformation, and correlates well with the infarct mass. Sensitivity and specificity for detecting infarcts tended to be superior for strain measurements compared with LVEF by echocardiography in the present study, but these findings need to be confirmed in future studies. A close relationship between different degrees of ischaemic injury and mortality and morbidity has been demonstrated previously [2]. There is reason to believe that reduced contractility secondary to ischaemic injury will affect prognosis in a similar way, and this should be a topic for future investigations.

Limitations

Myocardial systolic strain is load-dependent and should be interpreted with care when there are changes in loading conditions [24]. However, we examined our patients in a stable condition, verifying the clinical usefulness of the method in this circumstance.

Moreover, myocardial deformation is a complex three-dimensional process that is a composite of regional elastic properties as well as intrinsic and extrinsic forces. The ε_{ll} measurements reflect only one aspect of this process, and there are reasons to believe that the correlation may improve further with the availability of three-dimensional strain measurements.

2D-STE measurements have the advantage of being angle-independent as opposed to TDI. It is, however, like all echocardiographic techniques, dependent on image quality, and 10 % of all segment analyses were discarded in our present study. The average frame rate in the present study was relatively low, which might have an influence on the reported strain values. The average strain

of transmurally infarcted segments was −9 %, indicating conserved function. Myocardial areas that consist of 100 % scar after a myocardial infarct are not supposed to have preserved function, and the myocardial deformation in these segments should theoretically be close to zero. The average scar extent of transmural-infarcted segments in the present study was 70 %, and segmental strain values thus reflect a mixture of infarcted and normal myocardium as demonstrated in Figure 1(C). Our findings are consistent with other speckle tracking and MRI-tagging-based infarct studies which demonstrate strain values of approx. −8 % in transmural-infarcted segments [7,31]. The 2D-STE method is also appropriate for an even more detailed study of the left ventricle as shown in Figure 1, but the 'segmented' approach as we have used in the present study is well-suited as a clinical tool. Furthermore, it is easy to use and less time-consuming compared with older methods for assessment of myocardial strain.

Cross-registering identical segmental locations between the echocardiographic and MRI modalities [9] might be a problem when comparing segments from two different imaging modalities. Strain values of the segments within the infarct border zone are more susceptible to this possible inaccuracy, due to the fact that myocardial deformation is more heterogeneous in these regions. Care was taken to minimize the problem.

Territorial strain, like other non-invasive assessments of coronary stenosis, is based on a schematic distribution of territories and thus ignores the individual variation in coronary topography [29]. When interpreted in the clinical context, segments are more likely to be assigned to the true coronary artery than to the schematic territory.

Conclusions and perspectives

Global ε_{ll} measured by 2D-STE is an excellent predictor of myocardial infarct size in chronic ischaemic heart disease. Territorial strain is a specific index of the infarcted coronary artery. Peak systolic ε_{ll} measured by 2D-STE discriminates between non-infarcted, transmural-infarcted and subendocardial-infarcted segments. Our findings indicate that this method provides feasible non-invasive assessment of regional and global myocardial function.

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