

# Sequential Changes of Myocardial Function During Acute Myocardial Infarction, in the Early and Chronic Phase After Coronary Intervention Described by Ultrasonic Strain Rate Imaging

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**Objective:** The aim of this prospective clinical study was to follow up patients with acute myocardial infarction from the ischemic event, over the primary coronary intervention (PCI), up to the chronic phase after survived myocardial infarction by non-invasive strain rate (SR) imaging and to determine its role in the assessment of transmural of infarction.

**Methods:** In all, 41 patients with acute S-T elevation infarction were examined immediately before, 3 days after, and 5 months after PCI. Regional myocardial function was assessed by the use of ultrasonic SR imaging and peak systolic SR and systolic strain were extracted. In addition, late-enhancement (LE) imaging with magnetic resonance imaging was done after 5 months to assess the transmural of residual scar distribution.

**Results:** Magnetic resonance imaging showed that 8 patients had no LE (complete recovery = no-scar group), 16 patients had subendocardial LE (non-

transmural infarction = NT group), and 17 patients had a transmural LE (transmural infarction = T group) in the region of interest. Before PCI both SR and strain were markedly reduced in the ischemic segments compared with the nonischemic remote region in all 3 groups (SR: ischemia =  $-0.6 \pm 0.3 \text{ s}^{-1}$ ; remote =  $-1.3 \pm 0.4 \text{ s}^{-1}$ ,  $P < .001$ ). Three days after PCI, systolic SR only increased significantly in the regions that were not transmurally infarcted. After 5 months the measurement of systolic strain could accurately distinguish the different groups. (no-scar group =  $-24 \pm 5\%$ , NT group =  $-13 \pm 4\%$ , T group =  $-1 \pm 3\%$ ).

**Conclusions:** This clinical study shows that with SR imaging: (1) the ischemic segment can be precisely detected; (2) the absence of transmural early after coronary intervention can be predicted; and (3) in the chronic phase the transmural of scar distribution can be assessed. (J Am Soc Echocardiogr 2006; 19:839-847.)

Noninvasive monitoring of regional myocardial function from the acute event up to the chronic phase after survived myocardial infarction is preferable in clinical practice. In addition, the distinction between nontransmural and transmural necrosis after chronic myocardial infarction is clinically important as an increase in the degree of infarct transmu-

rality is associated with a great number of infarct-related complications, such as left ventricular (LV) dysfunction, arrhythmias, and an increased incidence of sudden death.<sup>1-3</sup>

Ultrasonic strain rate (SR) imaging has been introduced as a new noninvasive method to quantify regional myocardial deformation.<sup>4</sup> It has been validated in a correlative experimental sonomicrometric study<sup>5</sup> and used in a series of experimental and clinical studies.<sup>4,6-10</sup> Recently we have shown that in the experimental setting SR imaging can accurately differentiate chronic nontransmurally and transmurally infarcted myocardium.<sup>11</sup>

The aim of this prospective clinical study was to follow up patients with acute myocardial infarction from the ischemic event, over the primary coronary intervention (PCI), up to the chronic phase after survived myocardial infarction by noninvasive SR

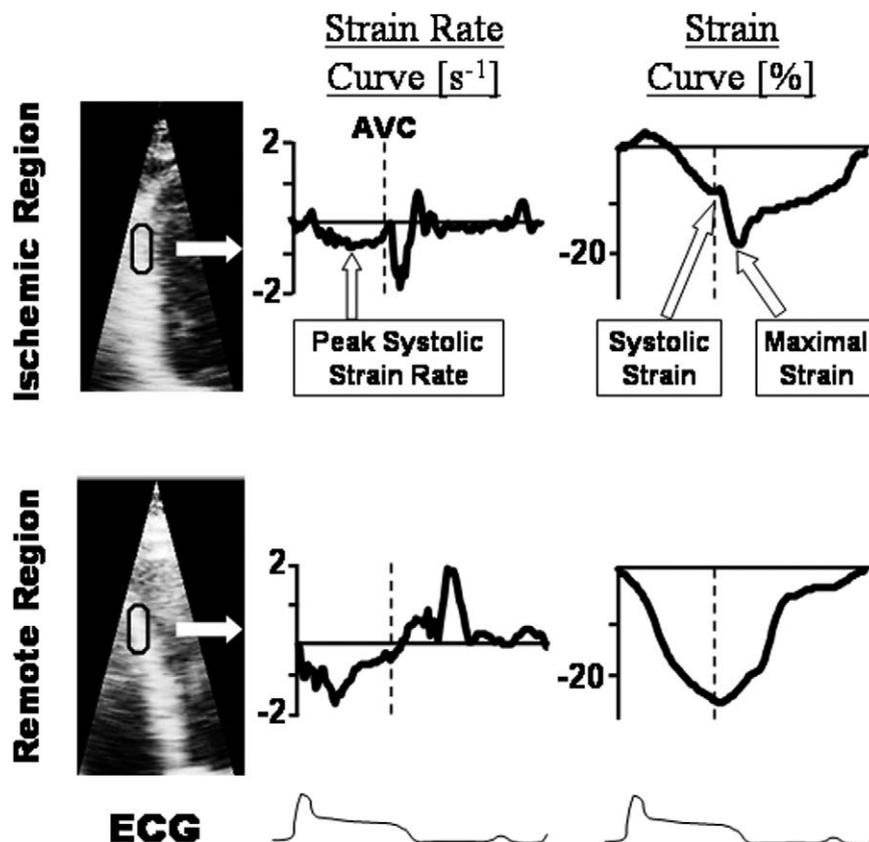
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**Figure 1** Typical example of ischemic deformation with extracted strain rate and strain curves of ischemic and remote region pre-primary coronary intervention. Note that ischemic myocardium continues to shorten after aortic valve closure (AVC), resulting in delayed shortening peak (postsystolic shortening). ECG, Electrocardiogram.

imaging. We hypothesized that in each of these stages SR imaging by echocardiography is feasible in clinical routine and can discriminate between transmural and nontransmural infarction, and reversible and irreversible damage.

## METHODS

### Protocol

All patients with acute first S-T elevation myocardial infarction (maximal creatine kinase > 190 U/L with a significant creatine kinase subfraction BM) who were sent to the intensive care department were included in the study. Patients with a history of chronic myocardial infarction or more than mild valvular heart disease were excluded. Immediately before PCI, echocardiographic gray scale and tissue Doppler images from apical views were acquired with the patient on his or her back. Three days after PCI a complete standard echocardiography and Doppler tissue study was done in all patients who had no indication for bypass operation. In the follow-up period all patients received standard therapy after myocardial infar-

tion including angiotension-converting enzyme inhibitors and  $\beta$ -blockers. Five months after the acute myocardial infarction the patients were scheduled for a follow-up echocardiographic and tissue Doppler study. In addition, late-enhancement (LE) imaging with magnetic resonance imaging (MRI) was done to assess qualitatively the transmural distribution of residual scar. Only the patients who completed the 3 echocardiographic studies and the MRI scanning at the end of the protocol were included in the final data set. MRI and echocardiographic data were analyzed by two independent investigators blinded to the results of the different techniques and to patient data. The investigation conformed to the principles outlined in the Declaration of Helsinki and informed consent was obtained from patients.

### Echocardiographic Image Acquisition

Transthoracic echocardiography was performed using a Vingmed System 5, (GE Ultrasound, Horten, Norway). Three heart cycles of apical 4- and 2-chamber views were captured by conventional 2-dimensional B-mode. In addition, a complete standard echocardiographic study with assessment of LV and left atrial dimension, LV ejection

fraction, and deceleration time of the mitral inflow pattern was assessed 3 days after PCI and after 5 months. All data were stored digitally for subsequent offline analysis. Conventional 2-dimensional loops were read by an experienced reader blinded to all patient data and the ischemic region was assessed by wall motion scoring. Ischemia was defined as regional reduction of myocardial motion in at least one segment.

### SR Imaging

Real-time 2-dimensional tissue Doppler data were recorded from the interventricular septum and the LV lateral and inferior walls using 4- and 2-chamber views with the wall in the middle of the sector. Data were analyzed using dedicated software as previously described (TVI, GE Ultrasound).<sup>11</sup> Longitudinal SR in the basal, mid, and apical segments of each wall were estimated by measuring the spatial velocity gradient (area of computation = 12 mm in the axial direction). SR profiles were averaged over 3 consecutive cardiac cycles and integrated over time to derive natural strain profiles using end diastole as the reference point (Speqle, K.U., Leuven, Belgium). From the averaged SR and strain data, peak systolic SR, systolic strain ( $\epsilon_{\text{SYS}}$ ), and maximal strain were calculated (Figure 1).  $\epsilon_{\text{SYS}}$  was defined to be the magnitude of deformation measured from end diastole to end systole. Aortic valve closure as a marker for end systole was derived from echocardiographic recordings. Maximal strain was defined to be the magnitude of deformation from end diastole to maximal thickening. The amount of shortening after aortic valve closure (postsystolic shortening [ $\epsilon_{\text{PSS}}$ ]) was calculated by the following equation:  $\epsilon_{\text{PSS}} = \text{maximal strain} - \epsilon_{\text{SYS}}$ .

### MRI

All images were acquired on a 1.5-T machine (Vision, Siemens Medical, Erlangen, Germany) equipped with a 4-channel cardiac phased array. The contrast agent (0.1 mmol/kg) (body weight) (Omniscan, Amersham Health, Braunschweig, Germany) was injected by an antecubital vein and after a 15-minute delay, 3 short-axis sections (acquired from the basal, mid, and apical level of the LV) were imaged in a single breath hold with an electrocardiographically gated inversion prepared fast low-angle shot single-shot sequence (repetition 2.1 milliseconds, echotime 1.1 milliseconds, matrix  $80 \times 128$ , flip angle  $8^\circ$ , field of view 320 mm [7/8], slice thickness 10 mm, inversion time 240-280 milliseconds). Each slice was separated by a 2-second delay, resulting in total breath hold time of approximately 7 seconds.

### MRI Analysis

Delayed short-axis images were analyzed for LE. Contours of enhanced regions and myocardial borders were drawn manually in each image. Transmurality of infarcts were graded in no scar (LE = 0%), nontransmural scar (0%-75%), and transmural scar (LE > 75%).

### Definition of Region of Interest

The ischemic wall of interest was defined by the target vessel of the baseline intervention. According to this, the wall for the left descending artery was the septum (in patients with anteroseptal infarction only the septum was analyzed because of technical considerations of SR imaging), for circumflex artery the lateral wall, and for the right coronary artery the inferior wall. In every wall the basal, mid, and apical segment were analyzed. Within each ischemic wall the region of interest (ROI) was retrospectively defined by localization of LE detected by MRI in the chronic phase of infarction after 5 months. In case of absence of LE only the target vessel distribution combined with the information of wall-motion abnormalities was used for the definition of the ROI. The neighboring segments of the ROI within the same wall were also analyzed. In addition, the 3 segments of the opposite wall were defined as the remote nonischemic region. Thus, in the final data set results are presented for the ischemic, neighboring, and remote segments. In average,  $5.9 \pm 0.3$  segments were analyzed in every patient.

### Statistical Methods

Data are presented as mean  $\pm$  1SD. Differences between the regions were tested using the Mann-Whitney *U* test. Multiple comparisons among different groups were performed using analysis of variance with post hoc Duncan test. Within each of the 3 groups, the comparison among the different stages was performed by analysis of variance for repeated measurements. Receiver operating characteristic (ROC) curves were used to assess the ability of the different parameters to identify the ischemic segment during the infarction and to identify the absence of transmural over time. A *P* value less than .05 was considered to indicate significance.

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## RESULTS

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Of 51 patients who were initially screened, 41 (29 male and 12 female) completed the 3 echocardiographic and MRI studies and were included in the final data set. These 41 patients were subdivided into 3 groups (no-scar, nontransmural scar, and transmural scar) according to the LE distribution assessed by MRI. The baseline characteristics of the 3 patient groups are presented in Table 1. The patients in the transmural group had a significantly longer time period from onset of pain to intervention ( $24.3 \pm 18$  hours) than patients in the nontransmural ( $10.2 \pm 11.2$  hours) and no-scar ( $4.9 \pm 5.3$  hours) groups. In the follow-up examination after 5 months the patients with transmural infarction had a significantly higher New York Heart Association (NYHA) classification compared with the no-scar and nontransmural groups (Table 2).

**Table 1** Baseline characteristics of the 3 patient groups

	No scar (n = 8)	Nontransmural scar (n = 16)	Transmural scar (n = 17)
Age, y	57 ± 8	57 ± 11	62 ± 10
Heart rate, beats/min	66 ± 10	68 ± 13	68 ± 12
SBP, mm Hg	136 ± 12	135 ± 19	139 ± 26
DBP, mm Hg	76 ± 7	80 ± 14	82 ± 12
t (onset of pain to intervention), h	4.9 ± 5.3	10.2 ± 11.2*	24.3 ± 18*†
Maximal creatine kinase	664 ± 518	935 ± 558	1199 ± 1295
BMI	24.8 ± 1.8	26.6 ± 3.2	29.6 ± 3.3*†
Hypertension	2 (25)	9 (56)	13 (76)*
Hypercholesterinemic	7 (88)	10 (63)	12 (71)
Diabetes mellitus	4 (50)	5 (31)	6 (35)
Nicotine <sup>a</sup>	4 (50)	10 (63)	7 (41)
Thrombolysis	1 (13)	5 (31)	6 (35)
IIb/IIIa receptor antagonist	7 (88)	14 (88)	15 (88)
TIMI preintervention:	1.0 ± 0.8	0.5 ± 0.6	0.7 ± 0.9
TIMI postintervention:	2.8 ± 0.5	2.5 ± 0.6	2.3 ± 0.9

BMI, Body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure; t, time.

\*P < .05 vs no scar.

†P < .05 vs nontransmural scar.

**Table 2** Five month follow-up data of the 3 patient groups

	No scar (n = 8)	Nontransmural scar (n = 16)	Transmural scar (n = 17)
General			
NYHA	1.5 ± 0.5	1.6 ± 0.5	2.3 ± 0.7*†
SBP, mm Hg	128 ± 15	142 ± 18	143 ± 27
DBP, mm Hg	79 ± 6	82 ± 10	87 ± 12
ECG			
Sinus rhythm	8 (100)	15 (94)	16 (94)
HR, beats/min	63 ± 6	66 ± 10	64 ± 10
Q waves	0 (0)	2 (13)	8 (47)*†
R-wave loss	0 (0)	7 (44)*	9 (53)*
Echocardiography			
LVEDD, mm	50.5 ± 1.7	54.1 ± 4.9*	54.4 ± 5.5*
LVESD, mm	31.5 ± 4.7	35.2 ± 4.2*	38.9 ± 8.3*
LA, mm	34.7 ± 7.4	37.4 ± 4.4	39.8 ± 4.4
EF, %	63.1 ± 4.8	58.4 ± 7.7	50.1 ± 12.8*
DT, ms	248 ± 44	268 ± 89	220 ± 72

DBP, Diastolic blood pressure; DT, deceleration time; ECG, electrocardiogram; EF, ejection fraction; HR, heart rate; LA, left atrial diameter; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; NYHA, New York Heart Association; SBP, systolic blood pressure.

\*P < .05 vs no scar.

†P < .05 vs nontransmural scar.

## MRI

All patients completed the MRI study without complications. According to the American Heart Association guidelines, the target coronary artery was assigned to the appropriate LV segments and, thus, the ROI was defined in every patient.<sup>12</sup> The MRI study showed 8 patients with no LE. These patients were termed as the “no-scar group.” In this group 4 patients had septal and 4 patients inferior myocardial infarctions at baseline. Of 41 patients, 16 had a

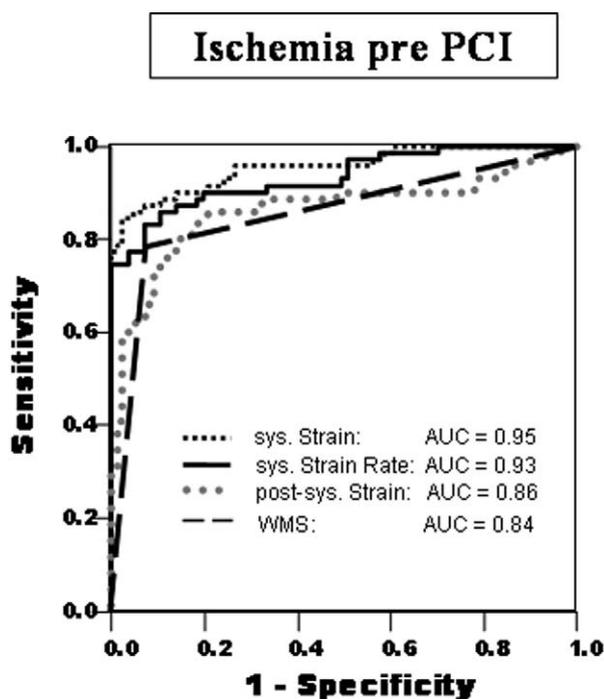
nontransmural LE in the ROI and were termed as “nontransmural group” (6 with septal, 3 with lateral, 7 with inferior myocardial infarctions). The third group consisted of 17 patients with transmural LE (transmural group). In this group 9 showed septal, 1 lateral, and 7 inferior myocardial infarctions.

## Standard Echocardiography

Post-PCI LV end-diastolic and end-systolic diameters were not significantly different among the 3 groups and averaged 52 ± 4 mm for the LV end-diastolic diameter and 35 ± 6 mm for the LV end-systolic diameter. In addition, the 3 groups showed no significant difference in left atrial diameter (37 ± 4 mm) and deceleration time (214 ± 35 milliseconds). The results of the echocardiographic measurements in the 5-month follow-up study are presented in Table 2.

## Wall Motion Scoring

During the acute infarction (pre-PCI) 85% of the ischemic segments (based on the target vessel) were correctly detected by wall motion scoring. In 12% of the patients gray scale image quality was not good enough for wall motion scoring. In the ROC analysis the area under the curve for detection of the ischemic wall by wall motion scoring was 0.84 (Figure 2). In the no-scar group 3 patients showed normal wall motion scoring 3 days after successful PCI. In contrast, 5 patients (63%) remained hypokinetic in the ROI like pre-PCI. After 5 months 7 patients (87%) in this no-scar group displayed normal wall motion scoring. In the nontransmural group wall motion scoring was unchanged 3 days post-PCI compared with pre-PCI (69% hypokinesia and 19% akinesia). In this group 38% showed normokinesia and 63%

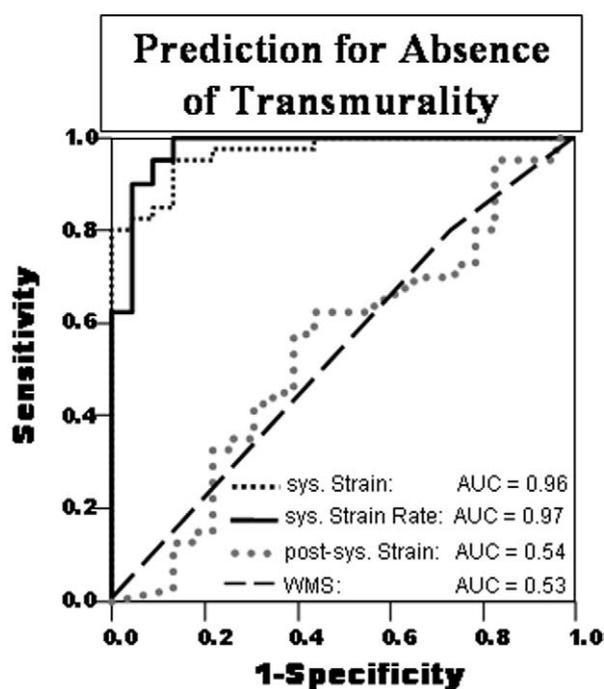


**Figure 2** Receiver operating characteristic curves for identification of ischemic segments pre-primary coronary intervention (PCI). Area under curve (AUC) is given for different parameters. *Sys*, Systolic; *WMS*, wall motion scoring.

hypokinesia in the chronic phase after 5 months. In the transmural group 77% had either hypokinesia or akinesia in the initially ischemic segments at day 3 (12% were not readable). After 5 months 83% of these patients with transmural scar had hypokinesia or akinesia in the infarcted segments. Two patients in this group showed normal wall motion in the transmural infarcted area. These two patients had very small, localized infarctions with a  $CK_{MAX}$  of 462 and 481 U/L. The ROC analysis for the detection of transmural 3 days after PCI demonstrated to be poor for wall motion scoring (Figure 3).

### SR Imaging

Doppler tissue images could be acquired in all patients and analyzed in 95%. Figure 4 shows the typical deformation properties (illustrated by SR maps) in the 3 different groups pre- and post-PCI and after 5 months. Pre-PCI systolic SR and  $\epsilon_{SYS}$  were significantly reduced in the ischemic (ROI) and neighboring segments compared with the remote segments (ischemic:  $SR = -0.6 \pm 0.3 \text{ s}^{-1}$ ,  $\epsilon_{SYS} = -6 \pm 6\%$ ; neighboring:  $SR = -0.9 \pm 0.3 \text{ s}^{-1}$ ,  $\epsilon_{SYS} = -11 \pm 6\%$ ; remote:  $SR = -1.3 \pm 0.4 \text{ s}^{-1}$ ,  $\epsilon_{SYS} = -21 \pm 6\%$ ,  $P < .001$ ). In addition,  $\epsilon_{PSS}$  was significantly higher in the ischemic ( $-4.6 \pm 3.5\%$ ) and neighboring ( $-3.1 \pm 2.7\%$ ) segments compared with the remote segments ( $-0.7 \pm 1\%$ ). The ROC curves demonstrated SR,  $\epsilon_{SYS}$ , and  $\epsilon_{PSS}$

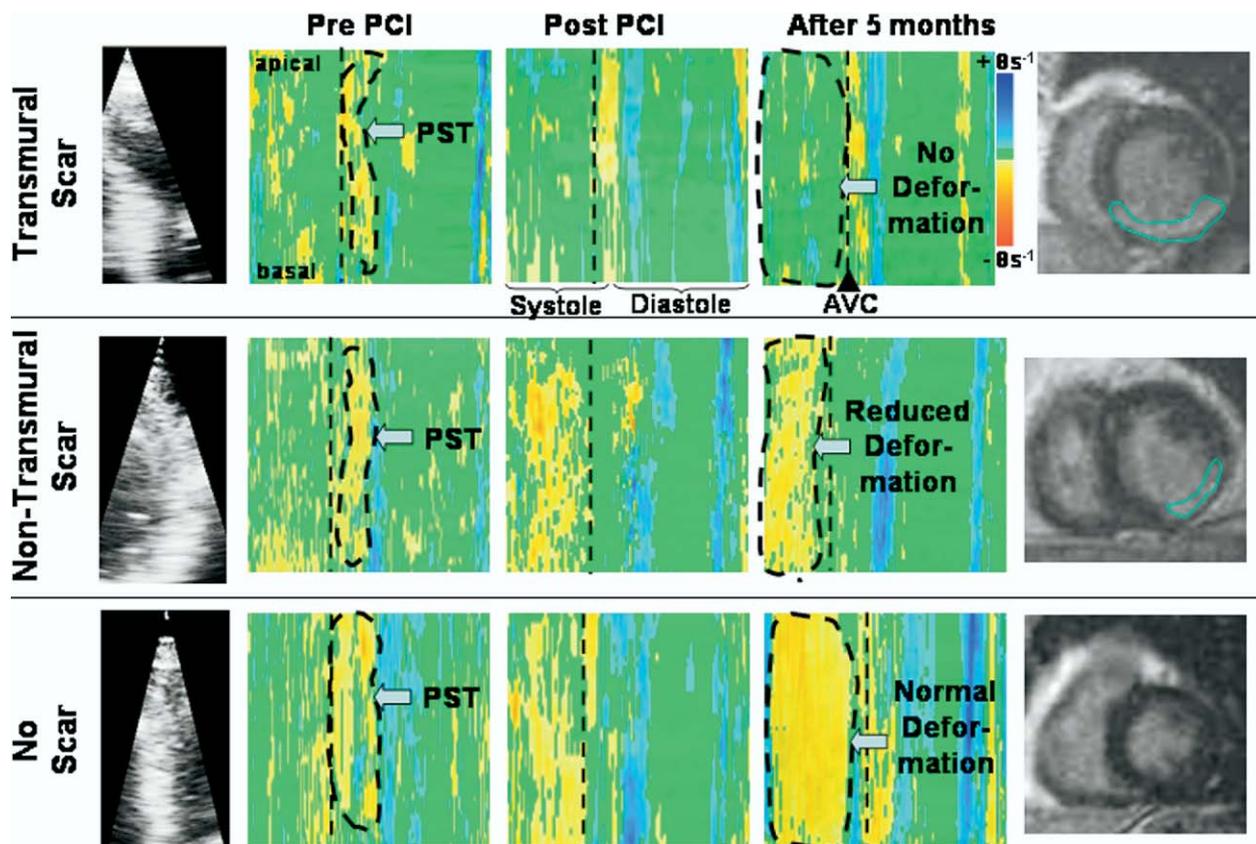


**Figure 3** Receiver operating characteristic curves for prediction of absence of transmural (defined as nontransmural infarct or no scar after 5 months in primary ischemic region) in region of interest 3 days post-primary coronary intervention. Area under curve (AUC) is given for different parameters. *Sys*, Systolic; *WMS*, wall-motion scoring.

to be good parameters for the detection of the ischemic region (Figure 2). Figure 1 shows a typical longitudinal SR and strain profile in the ischemic and remote region.

Post-PCI systolic SR in the ischemic segments increased in the no-scar ( $SR = -1.1 \pm 0.4 \text{ s}^{-1}$ ) and nontransmural ( $SR = -1.0 \pm 0.3 \text{ s}^{-1}$ ) groups significantly. In contrast, systolic SR remained reduced in the transmural group ( $SR = -0.4 \pm 0.3 \text{ s}^{-1}$ ). In parallel,  $\epsilon_{SYS}$  increased only in the no-scar and nontransmural groups, but not in the transmural group (Figure 5). The ROC curves demonstrated systolic SR and  $\epsilon_{SYS}$  to be the best parameters for the detection of absence of transmural 3 days post-PCI with a sensitivity of 95% and a specificity of 91% for SR using a cut-off point of  $-0.6 \text{ s}^{-1}$ . In contrast,  $\epsilon_{PSS}$  was similar in the 3 groups and not very sensitive to detect absence of transmural (Figure 3).

In the chronic phase 5 months after the acute myocardial infarction the typical deformation patterns for each group are illustrated in Figure 6. In the no-scar group systolic SR and  $\epsilon_{SYS}$  were not significantly different compared with the remote region (ROI:  $SR = -1.5 \pm 0.3 \text{ s}^{-1}$ ,  $\epsilon_{SYS} = -24 \pm 5\%$ ; remote:  $SR = -1.4 \pm 0.3 \text{ s}^{-1}$ ,  $\epsilon_{SYS} = -23 \pm 4\%$ ). In the patients with nontransmural myocardial infarction systolic SR and  $\epsilon_{SYS}$  were significantly lower than



**Figure 4** Strain rate (SR) maps pre- and post-primary coronary intervention (PCI) and after 5 months together with echocardiographic and magnetic resonance image showing regions with late enhancement. SR maps are displaying longitudinal deformation properties from apical to basal segment over one heart cycle. No deformation (*green*), shortening (*yellow*), and lengthening (*blue*) of segment. *Top*, Inferior, transmural myocardial infarction: pre-PCI systolic shortening is markedly reduced and some shortening occurs after aortic valve closure (AVC). Three days post-PCI and after 5 months almost no shortening occurs in systole. *Middle*, Lateral, nontransmural myocardial infarction. Compared with pre- and post-PCI some shortening is restored in systole. However, after 5 months systolic shortening is not completely normal. *Bottom*, Septum, no scar. Initially after 3 days post-PCI systolic shortening improved and after 5 months complete recovery of deformation could be documented. *PST*, Postsystolic shortening.

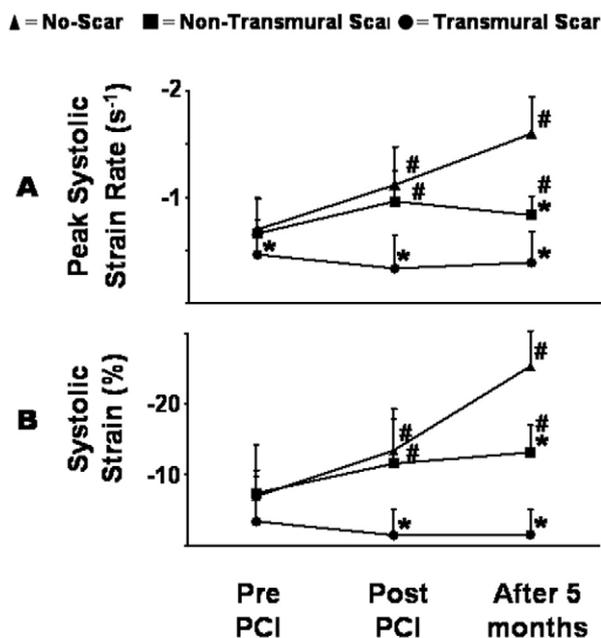
in the no-scar group ( $SR = -0.8 \pm 0.2 \text{ s}^{-1}$ ,  $\epsilon_{\text{SYS}} = -13 \pm 4\%$ ,  $P < .001$  vs no-scar group) but higher than in the patients with transmural scar ( $SR = -0.4 \pm 0.3 \text{ s}^{-1}$ ,  $\epsilon_{\text{SYS}} = -1 \pm 3\%$ ,  $P < .001$  vs nontransmural scar) (Figure 5). Compared with the complete deformation during the heart cycle only the patients with transmural myocardial infarction showed still some shortening after systole in the ROI ( $\epsilon_{\text{PSS}} = -4 \pm 5\%$ ). Thus, in the no-scar group both parameters increased up to normal values, in the nontransmural group the deformation parameters recovered partially and in the transmural group both deformation parameters stayed during the follow-up studies on the very low baseline level (Figure 5).

Analog to the ROI deformation in the neighboring segments deformation increased in the no-scar group ( $\epsilon_{\text{SYS}}$  post-PCI =  $-15 \pm 6\%$ ,  $\epsilon_{\text{SYS}}$  after 5 months =  $-23 \pm 3\%$ ) and in the nontransmural

group ( $\epsilon_{\text{SYS}}$  post-PCI =  $-14 \pm 6\%$ ,  $\epsilon_{\text{SYS}}$  after 5 months =  $-22 \pm 6\%$ ) but only slightly in the transmural group ( $\epsilon_{\text{SYS}}$  post-PCI =  $-9 \pm 6\%$ ,  $\epsilon_{\text{SYS}}$  after 5 months =  $-12 \pm 7\%$ ). SR in this neighboring region changed in the same direction as  $\epsilon_{\text{SYS}}$ .  $\epsilon_{\text{PSS}}$  was only detectable in the transmural group (post-PCI =  $-4 \pm 3\%$ , after 5 months =  $-4 \pm 4\%$ ). Deformation parameters in the remote region were not significantly different among the 3 stages (pre-PCI, post-PCI, and after 5 months).

## DISCUSSION

In this clinical study patients with acute myocardial infarction were followed up from the ischemic event, over the coronary intervention, up to the chronic phase after survived myocardial infarction.



**Figure 5** Changes in regional peak systolic strain rate (unit  $s^{-1}$ ) (A) and systolic strain (unit %) (B) in no-scar, nontransmural, and transmural myocardial infarction groups. \* $P < .05$  vs no scar; # $P < .05$  vs pre-primary coronary intervention (PCI).

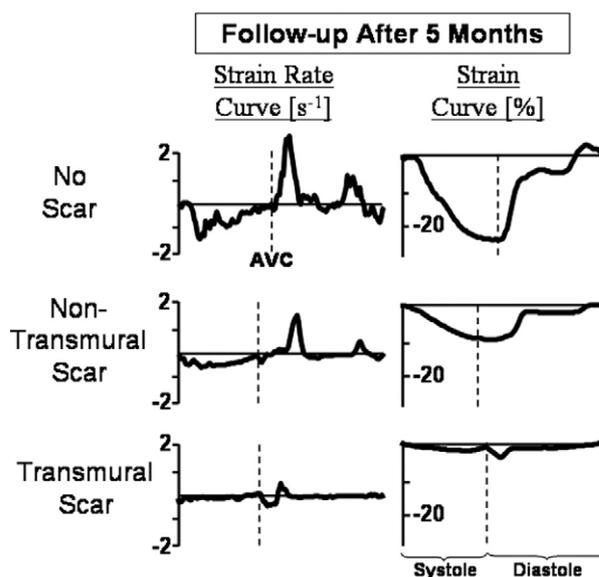
It shows by the use of SR imaging that: (1) the ischemic segment can be precisely detected; (2) the absence of transmural infarction immediately after coronary intervention can be predicted; and (3) in the chronic phase the transmural distribution of scar can be assessed.

### Deformation in Acute Myocardial Infarction

From experimental work it is known that acute coronary occlusion induces a typical ischemic deformation response with a decrease of systolic shortening ( $\epsilon_{SYS}$ ) and an ongoing shortening after aortic valve closure ( $\epsilon_{PSS}$ ).<sup>13-15</sup> Our clinical study confirms these experimental findings and proves that the reduction in  $\epsilon_{SYS}$  and systolic SR are clinical feasible parameters to detect acute ischemic myocardium. However, also chronic nontransmural infarcted myocardium shows reduced  $\epsilon_{SYS}$  but almost no  $\epsilon_{PSS}$ . Thus, in the context of clinical symptoms the combination of reduced  $\epsilon_{SYS}$  and increased  $\epsilon_{PSS}$  can help to discriminate acute ischemia from chronic nontransmural infarction.

### Deformation after PCI

Already 3 days after the coronary intervention deformation properties increased in the two groups with no scar or nontransmural scar in the ROI indicating a potential contractile reserve. Experimental data document that peak systolic SR is the appropriate parameter for quantification of the contractile re-



**Figure 6** Typical examples of strain rate and strain curves in nontransmural and transmural infarcted tissue and in tissue that had no scar. Curves are extracted from region of interest in chronic phase (5 months) after survived myocardial infarction. *AVC*, Aortic valve closure.

serve.<sup>7,8,11</sup> Also in this study peak systolic SR turned out to be a good parameter to predict the potential recovery in groups with potential viable myocardium (no-scar + nontransmural infarction) as there was a partial improvement of SR. However, 3 days after PCI it was not possible to predict whether the tissue will recover completely using regional deformation parameters at rest. This might be because of the fact that those segments were still stunned. As expected for stunned myocardium there was no detectable improvement of wall motion scoring compared with baseline. This indicates that SR might be more sensitive to detect subtle increase in regional myocardial function. From experimental data it is known that these stunned segments can be nicely detected with SR imaging using a dobutamine challenge,<sup>7</sup> which was not done in this study because of ethical considerations. In contrast, already at this very early time point after PCI transmurally infarcted myocardium can be detected as both systolic SR and  $\epsilon_{SYS}$  were still unchanged compared to pre-PCI values.

### Deformation in the Chronic Phase After Survived Myocardial Infarction

In the chronic phase after 5 months LE imaging studies were done to visualize the transmural distribution of scar caused by the previous myocardial infarction. Recent studies have demonstrated the use of this technique to distinguish noninvasively between scar and viable myocardium in patients with chronic myocardial infarction.<sup>16,17</sup> In experimental studies

on acute and chronic myocardial infarction it could be demonstrated that measuring the deformation properties by the use of tissue Doppler can accurately differentiate nontransmural from transmural infarction.<sup>11,15</sup> In the current study, the patients with no scar in the ROI displayed normal SR and  $\epsilon_{\text{SYS}}$  values and almost no  $\epsilon_{\text{PSS}}$  comparable with the remote region. Thus, they survived the previous infarction with no morphologic abnormalities (no scar), normal myocardial function, and the best NYHA class. The patients with nontransmural infarction had reduced SR and  $\epsilon_{\text{SYS}}$  values in the ROI. This reduction of deformation was most probably related to the combination of changes induced by the presence of nondeforming scar tissue in the endocardium together with deforming tissue in the epicardium. In the experimental setting a good correlation between the extension of scar (assessed by histology studies) and systolic strain could be demonstrated.<sup>11</sup> However, in this clinical study only a qualitative but not a quantitative assessment of transmural strain was done. This was done because: (1) the correct determination of transmural strain is much more precise with histologic studies compared with MRI studies; and (2) the exact anatomic matching between echocardiography and MRI is not possible. The patients with transmural myocardial infarction had almost no systolic shortening ( $\epsilon_{\text{SYS}}$ ) and severely reduced systolic SR in the region with transmural scar. MRI studies with LE showed that a transmural strain of more than 75% is very unlikely to recover after revascularization.<sup>18</sup> In addition, they developed some  $\epsilon_{\text{PSS}}$ , which is suggested to be the typically passive recoil after the end of systole as a result of pressure decrease in the LV.<sup>11,19</sup> Thus, these patients appear to have major structural abnormalities (transmural scar), almost no systolic function, and the highest NYHA class.

### Clinical Implications

In an attempt to overcome the limitations inherent in the visual scoring of wall motion in hearts with regional ischemia, several noninvasive quantitative imaging methods have been developed.<sup>4,20,21</sup> In contrast to motion-based techniques SR imaging is independent of tethering effects and can assess the regional contractile status of all myocardial segments.<sup>5,8,22</sup> In the current study this noninvasive technique proved to be suitable even under emergency conditions during an acute myocardial infarction on the intensive care department. The image acquisition (on the back) took only 2 minutes and, thus, did not prolong the time period between onset of pain and PCI.

As transmural myocardial infarction is associated with a great number of infarct-related complications<sup>1-3,23</sup> it is clinically important to assess the transmural strain of necrosis or scar. By the use of SR

imaging it was possible to detect the transmural necrosis early after the acute event and the transmural scar in the chronic phase after survived myocardial infarction. In contrast, the detection of complete recovery after myocardial infarction has an impact on patient treatment as these patients require less medication and show a better exercise capacity and quality of life.<sup>1</sup>

### Limitations

The methodology used does not allow quantification of deformation in differing myocardial layers but measures averaged deformation across the whole wall. As the ROI was based on the target vessel localization and the LE distribution after 5 months, segments that were initially stunned but recovered in the follow-up period were included in the data set of the neighboring segments. Thus, these neighboring segments might include ischemic and nonischemic segments. Therefore, the current data for the ischemic segments display a condensed data set focusing on the center of the infarcted area.

Another limitation is the fact that in patients with anteroseptal infarction only the septum was analyzed because of technical considerations of SR imaging. In most patients, especially when on the back, the angle and the image quality of the anterior wall was not appropriate for SR imaging.

### Conclusions

This study shows that SR imaging can accurately assess different kinds of infarcted myocardium during all stages after acute myocardial infarction in clinical settings.

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