Strain Rate Imaging Differentiates Transmural From Non-Transmural Myocardial Infarction
A Validation Study Using Delayed-Enhancement Magnetic Resonance Imaging

Yan Zhang, MB, PHD,* Anna K. Y. Chan, MRCP,* Cheuk-Man Yu, MD, FRCP,* Gabriel W. K. Yip, MD,* Jeffrey W. H. Fung, FRCP,* Wynnie W. M. Lam, FRCP,† Nina M. C. So, FRCR,‡ Mei Wang, MB, PHD,* Eugene B. Wu, MD, MRCP,* John T. Wong, MRCP,* John E. Sanderson, MD, FACC*

Hong Kong, SAR

OBJECTIVES
The aim of this study was to determine if strain rate imaging (SRI) correlates with the transmural extent of myocardial infarction (MI) measured by contrast-enhanced magnetic resonance imaging (Ce-MRI).

BACKGROUND
Identification of the transmural extent of myocardial necrosis and degree of non-viability after acute MI is clinically important.

METHODS
Tissue Doppler echocardiography with SRI and Ce-MRI were performed in 47 consecutive patients with a first acute MI between days 2 and 6 and compared to 60 age-matched healthy volunteers. Peak myocardial velocities and peak myocardial deformation strain rates were measured. Location and size of the infarct zone was confirmed by Ce-MRI using the delayed enhancement technique with a 16-segment model.

RESULTS
Contrast-enhanced MRI identified transmural infarction in 21 patients, non-transmural infarction in 15 (mean transmurality of infarct 72.3 ± 10.6%), and another 11 patients with subendocardial infarction (50% transmural extent of the left ventricular wall). Peak systolic strain rate (SRs) of the transmural infarction segments was significantly lower compared to normal myocardium or with non-transmural infarction segments (both p < 0.0005). A cutoff value of SRs > −0.59 s⁻¹ detected a transmural infarction with high sensitivity (90.9%) and high specificity (96.4%), and −0.98 s⁻¹ >SRs > −1.26 s⁻¹ distinguished subendocardial infarction from normal myocardium with a sensitivity of 81.3% and a specificity of 83.3%.

CONCLUSIONS
Peak myocardial deformation by SRI can differentiate transmural from non-transmural MI, and it allows noninvasive determination of transmurality of the scar after MI and thereby the extent of non-viable myocardium. (J Am Coll Cardiol 2005;46:864–71) © 2005 by the American College of Cardiology Foundation

Previous experimental and clinical evidences suggest that the presence or absence of Q waves on surface electrocardiography does not distinguish between transmural and subendocardial myocardial infarction (MI) (1,2), and the degree of reduction in myocardial wall thickening by standard echocardiography is not proportional to the percentage of necrosis (3). The differentiation of transmural from non-transmural MI is clinically important because transmural infarcts are associated with a higher likelihood of adverse cardiac events, poorer prognosis, and lack of recovery after revascularization (4,5), and the identification of non-transmural infarction implies that viable myocardium is present, which may benefit from revascularization. Magnetic resonance imaging with gadolinium-based contrast (Ce-MRI) offers high spatial resolution and can identify infarcted non-viable tissue using the delayed enhancement technique. Its utility for the detection and sizing of MI is well validated (6,7). Non-viable tissue appears “hyperenhanced” or bright due to the accumulation of contrast in non-viable fibrotic tissues. Many studies have confirmed the concept that hyperenhanced regions have sustained irreversible ischemic injury (8). Tissue Doppler imaging (TDI) has been introduced as a new noninvasive method to quantitatively assess regional myocardial function, but it is limited by translational motion and tethering effects (9,10). Tissue Doppler-derived strain rate imaging (SRI) quantifies local rate of myocardial deformation (11–15) and has the potential to differentiate viable from infarcted myocardium (13,14,16,17) and thus may be used for the selection of patients with salvageable left ventricular (LV) dysfunctional myocardium who will benefit most from coronary revascularization. Weidemann et al. (17) applied this approach to determine the transmurality of chronic infarction in a correlative functional/histopathologic animal study, and showed that peak systolic strain rate and systolic strain could...
Abbreviations and Acronyms

Am = atrial contraction velocity
Ce-MRI = contrast-enhanced magnetic resonance imaging
Em = early diastolic velocity
FDG-PET = [18F]fluorodeoxyglucose positron emission tomography
LV = left ventricle/ventricular
LVEF = left ventricular ejection fraction
MI = myocardial infarction
ROC = receiver-operating characteristic curve
Sm = peak systolic velocity
Sra = atrial strain rate
Sre = early diastolic strain rate
SRI = strain rate imaging
SRs = peak systolic strain rate
TDI = tissue Doppler imaging
WMSI = wall motion score index

clearly differentiate non-transmural from transmural infarction. However, the validity and the reliability of these novel parameters in defining the transmurality in clinical practice has not been established. Therefore, in this study, we sought to determine the correlation between SRI and the transmural extent of MI measured by Ce-MRI in acute MI patients and establish practical cutoff values for differentiating transmural scar tissue from non-transmural or subendocardial infarction with viable myocardium.

METHODS

Patients. Patients admitted to the coronary care unit of a university teaching hospital with a first acute MI who had an echocardiography and Ce-MRI performed on the same day within two to six days of the acute event were recruited. Diagnosis of acute MI was made according to the criteria based on the Joint European Society of Cardiology/ American College of Cardiology Committee for the Redefinition of MI (18). Patients were excluded from the study if they had a history of MI or uncontrolled hypertension; an echocardiogram showing significant valvular disease, hypertrophic obstructive cardiomyopathy; significant chronic obstructive airway disease or who were unable to hold their breath in expiration during image acquisition; and devices or implants that contraindicated Ce-MRI examination. Sixty healthy volunteers who had no history of cardiovascular disease or systemic illness, with normal physical examination, electrocardiogram, and echocardiographic examinations served as controls. Informed consent was obtained from all subjects.

Coronary angiographic data were available in 35 patients and were analyzed visually by two experts blinded to the clinical data. Coronary stenosis was defined as a luminal narrowing more than 70% in two orthogonal views.

Echocardiography. Echocardiograms were performed using a standard commercial ultrasound machine (Vivid 5, GE Vingmed, Horten, Norway) with a 2.5- or 3.5-MHz multiphase-array probe. All echocardiographic data were acquired in expiration to minimize translation movement of the heart. Techniques and calculations of the various cardiac dimensions were performed according to the recommendations of the American Society of Echocardiography. A 12-segment model (4 basal, 4 mid, and 4 apical) was used to subdivide the LV for subsequent analysis. The myocardial motion of each segment was evaluated according to the standard American Society of Echocardiography wall motion scoring system, which assigns a wall motion score ranging from 1 to 4 to describe normokinesia, hypokinesia, akinesia, and dyskinesia, respectively, and wall motion score index (WMSI) was calculated as the ratio of the sum of wall motion score over total segments.

Tissue Doppler imaging and SRI were acquired at the apical four- and two-chamber views with a single wall imaged using a narrow sector angle (30° to 60°) with the ventricular wall parallel to the ultrasound beam and high frame rates (80 to 180 frames/s) (13,14,17). Pulse repetition frequency was chosen between 1 and 2 kHz to avoid any aliasing within the image. Three consecutive cardiac cycles were stored, and the images were digitized and analyzed offline (EchoPac 6.3.6, GE Vingmed). Myocardial longitudinal velocity and deformation curves were obtained, and peak systolic signal during ejection phase (peak systolic velocity [Sm] and peak systolic strain rate [SRs]), early diastolic velocity (Em) and deformation (early diastolic strain rate [SRe]), and atrial contraction velocity (Am) and deformation (atrial strain rate [Sra]) were measured. The sample volume for strain rate was set to 9.2 mm, which proved to be the best compromise between good spatial resolution and optimal signal-to-noise ratio.

Ce-MRI. Short-axis Ce-MRI was performed on the same day of echocardiography using a 1.5-T whole-body scanner (Sonata, Siemens-Medical, Erlangen, Germany). Short-axis images were acquired every 4 to 8 mm from base to apex, 15 min after intravenous injection of a gadolinium-based contrast agent (Gd-DTPA for dose of 0.1 mmol/kg) as described previously (6,19). A 16-segment model was used dividing the LV into six basal, six mid-ventricular, and four apical segments. Regional contrast enhancement was scored with a scheme based on the spatial extent of hyperenhanced tissue within each segment (transmural = 100% hyperenhanced extent of the LV wall; non-transmural = 50% to 90% hyperenhanced extent of the LV wall; and subendocardial = <50% hyperenhanced extent of the LV wall) (20). End-diastolic wall thickness was measured at the center of each myocardial segment from the leading endocardial edge to leading epicardial edge. Infarct size was determined as a percentage of LV volume, as the sum of hyperenhanced areas from each of all the short-axis images divided by the total area of pixels within the LV myocardium multiplied by 100 (7). For comparison with echocardiography, 12 segments were used: basal septum, lateral, inferior, and anterior segments; mid-septum, lateral, inferior, and anterior segments; and apical septum, lateral, inferior, and anterior segments.
The analyses of echocardiography and Ce-MRI examinations were performed by investigators in a blinded manner.

### Statistical analysis
Data are presented as mean ± SD. For the comparison of parametric variables between patient group and normal controls, an independent-sample *t* test was employed. Differences among peak myocardial velocities in the same wall from basal to apical segments and differences among the four different sites at the same ventricular level were analyzed by repeated-measures analysis of variance. The same analysis was also used for assessing strain rates. Correlations between TDI or SRI and infarct size were performed by linear regression analysis. Receiver-operating characteristic (ROC) curves were constructed, and areas under curves were measured to determine cutoff values with maximum sensitivity and specificity. Inter- and intraobserver reproducibility was obtained for the analysis of the TDI and SRI parameters from 10 randomly chosen patients by the Bland-Altman methods (21). A *p* value <0.05 was considered statistically significant.

### RESULTS
Forty-seven consecutive patients (mean age of 59.9 ± 11.6 years, 75.8% men) with acute MI were studied at two to six days after admission. The baseline characteristics are shown in Table 1. Thirty-four patients were given thrombolytic therapy on presentation, and 35 had undergone either primary coronary intervention or early revascularization (i.e., 44 patients had received reperfusion therapy by either thrombolytic or coronary revascularization). Among those with coronary angiography performed, 19 patients with anterior MIs, 17 had stenosis of the left anterior descending artery, 1 left circumflex artery, and 1 triple-vessel disease. Of the remaining 16 with inferior MIs, 8 had a significant stenosis in the right coronary artery, 1 left circumflex artery, 6 both right coronary artery and left circumflex artery, and 1 triple-vessel disease. All the patients had a normal QRS complex duration (87.2 ± 10.1 ms). Pathologic Q waves were present on the electrocardiogram in 37 (78.7%) patients, and were absent in the other 10 (21.3%) patients. Sixty healthy volunteers of similar age and gender served as normal controls (Table 2).

### Ce-MRI and standard echocardiography parameters
The Ce-MRI identified transmural MI in 21 (44.7%) patients with 80 transmural and 19 non-transmural infarcted segments, non-transmural MI in 15 (31.9%) patients with 70 non-transmural and 12 subendocardial infarcted segments, and 11 (23.4%) patients with subendocardial MI with 37 subendocardial infarcted segments (Table 2). Infarct size measured by Ce-MRI ranged from 4.5% to 46.1%. The degree of transmurality was 72.3 ± 10.6% in the non-transmural group and 40.3 ± 5.8% in the subendocardial group. End-diastolic wall thickness of infarct segments was 7.0 ± 1.1 mm in the transmural group, 8.0 ± 1.5 mm in the non-transmural group, and 8.2 ± 1.7 mm in the subendocardial group. End-diastolic wall thickness showed signifi-
significant differences: p = 0.01 transmural versus non-transmural; p = 0.04 transmural versus subendocardial groups.

Patients with either transmural or non-transmural MI had reduced left ventricular ejection fraction (LVEF), increased LV end-systolic and end-diastolic dimensions, and greater LV mass when compared to controls (all p < 0.05) (Table 2). Left ventricular transmitral inflow analysis showed no significant difference between patient and control group in early diastolic filling velocity, atrial contracting velocity, deceleration time of early filling, or isovolumic relaxation time (Table 2); WMSI was not related to transmurality of the infarction (Table 3).

**TDI and SRI measurements.** In normal subjects, a systolic myocardial velocity gradient was present from base to apex in each wall. All myocardial velocities decreased as the Doppler sample was moved from the LV base to the apex along four walls. In contrast, the myocardial deformation in both systole and diastole were uniform, and there was no significant difference in Srs, SRe, or SRa between segments (Table 4, Fig. 1). However, in MI patients, Srs of the transmural infarcted segments were significantly decreased when compared with non-transmural, subendocardial MI, and normal segments; Srs and SRe were also significantly reduced in subendocardial infarction compared with normal subjects (Table 3, Fig. 2). However, by TDI both Sm and Em were reduced in the transmural myocardium compared with subendocardial infarction, but there was no significant difference between transmural and non-transmural MI or between non-transmural and subendocardial MI (Table 3).

Linear regression analysis showed that both Srs and Sm of the infarct area had a significant correlation with the infarct size (r = 0.63, p < 0.0005 and r = −0.38, p = 0.01, respectively) (Fig. 3) and with LVEF (r = 0.338, p = 0.033, and r = 0.555, p < 0.0005, respectively), and infarct Sm also correlated with baseline end-systolic volume (r = 0.298, p = 0.045). However, neither SRe nor Em had any correlation with infarct size or LVEF (all p > 0.05).

**ROC analysis.** Receiver-operating characteristic analysis was applied to determine the cutoff values for differentiating transmural, non-transmural, and subendocardial MI (Fig. 4). A cutoff value of Srs > −0.59 s⁻¹ identified transmural from non-transmural and subendocardial MI with a sensitivity of 90.9% and 90.9%, specificity of 96.4% and 100%, respectively. A cutoff value of −0.98 s⁻¹ > Srs > −1.26 s⁻¹ was able to distinguish a subendocardial infarction from normal subjects with sensitivity of 81.3% and specificity of 83.3% (p = 0.001). Similarly, a cutoff value of SRe > −1.45 s⁻¹ detected subendocardial infarction from normal myocardium with an area under the curve of 0.777 (p < 0.0005); however, both the sensitivity and the specificity were lower than Srs (72% and 70%, respectively). On the other hand, occurrence of pathologic Q waves, akinesia, and end-diastolic wall thickness <5.5 mm identified transmural from

**Table 3.** Comparisons of TDI, SRI, and WMS Among Trans-MI, Nontrans-MI, Subendo-MI, and Control Segments

<table>
<thead>
<tr>
<th></th>
<th>Sm (cm/s)</th>
<th>Em (cm/s)</th>
<th>Am (cm/s)</th>
<th>SRI (s⁻¹)</th>
<th>SRe (s⁻¹)</th>
<th>SRa (s⁻¹)</th>
<th>WMSI (s⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trans-MI (80 segments)</td>
<td>3.01 ± 1.53†</td>
<td>−4.17 ± 2.4†</td>
<td>−4.13 ± 2.3</td>
<td>−0.51 ± 0.17†</td>
<td>1.04 ± 0.4†</td>
<td>1.14 ± 0.8</td>
<td>2.35 ± 0.55</td>
</tr>
<tr>
<td>Nontrans-MI (89 segments)</td>
<td>3.28 ± 1.55*</td>
<td>−4.42 ± 2.2*</td>
<td>−4.44 ± 2.3</td>
<td>−1.06 ± 0.29§</td>
<td>1.29 ± 0.7*</td>
<td>1.19 ± 0.6</td>
<td>2.17 ± 0.71</td>
</tr>
<tr>
<td>Subendo-MI (49 segments)</td>
<td>4.26 ± 1.84*</td>
<td>−5.52 ± 1.9*</td>
<td>−4.99 ± 2.3</td>
<td>−1.21 ± 0.41‡</td>
<td>1.23 ± 0.5*</td>
<td>1.15 ± 0.6</td>
<td>2.12 ± 0.78</td>
</tr>
<tr>
<td>Controls (720 segments)</td>
<td>5.02 ± 1.44</td>
<td>−6.78 ± 1.7</td>
<td>−6.64 ± 1.5</td>
<td>−1.58 ± 0.38</td>
<td>1.68 ± 0.4</td>
<td>1.60 ± 0.8</td>
<td>1.0</td>
</tr>
</tbody>
</table>

* p < 0.05 vs. controls; †p < 0.01 vs. controls; §p < 0.05 vs. Nontrans-MI; ‡p < 0.0005 vs. Trans-MI or control.

Am = atrial contraction velocity; Em = early diastolic velocity; MI = myocardial infarction; Nontrans-MI = non-transmural myocardial infarction; Sm = peak systolic velocity; Srs = atrial strain rate; SRe = early diastolic strain rate; SRI = strain rate imaging; Srs = peak systolic strain rate; Subendo-MI = subendocardial myocardial infarction; TDI = tissue Doppler imaging; Trans-MI = transmural myocardial infarction; WMSI = wall motion score index.

**Table 4.** TDI and SRI Measurements in Normal Subjects

<table>
<thead>
<tr>
<th></th>
<th>Sm (cm/s)</th>
<th>Em (cm/s)</th>
<th>Am (cm/s)</th>
<th>SRI (s⁻¹)</th>
<th>SRe (s⁻¹)</th>
<th>SRa (s⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal sep.</td>
<td>5.80 ± 1.22*</td>
<td>−6.75 ± 2.01*</td>
<td>−7.53 ± 1.55*</td>
<td>−1.48 ± 0.33</td>
<td>1.56 ± 0.88</td>
<td>1.88 ± 1.12</td>
</tr>
<tr>
<td>Mid-sep.</td>
<td>4.82 ± 1.31</td>
<td>−5.27 ± 2.54</td>
<td>−6.22 ± 1.86</td>
<td>−1.39 ± 0.46</td>
<td>1.48 ± 0.53</td>
<td>1.73 ± 0.60</td>
</tr>
<tr>
<td>Apical sep.</td>
<td>2.91 ± 1.30</td>
<td>−3.16 ± 1.98</td>
<td>−3.15 ± 2.13</td>
<td>−1.53 ± 0.28</td>
<td>1.62 ± 0.66</td>
<td>1.82 ± 0.66</td>
</tr>
<tr>
<td>Basal lat.</td>
<td>6.28 ± 1.26*</td>
<td>−7.88 ± 2.22*</td>
<td>−7.76 ± 1.47</td>
<td>−1.74 ± 0.27</td>
<td>1.95 ± 0.90</td>
<td>1.78 ± 0.91</td>
</tr>
<tr>
<td>Mid-lat.</td>
<td>5.43 ± 1.56</td>
<td>−6.95 ± 2.33</td>
<td>−5.80 ± 1.74</td>
<td>−1.49 ± 0.34</td>
<td>1.78 ± 0.76</td>
<td>1.25 ± 0.93</td>
</tr>
<tr>
<td>Apical lat.</td>
<td>3.20 ± 1.33</td>
<td>−3.96 ± 1.95</td>
<td>−3.09 ± 1.87</td>
<td>−1.62 ± 0.38</td>
<td>2.12 ± 0.71</td>
<td>1.49 ± 0.78</td>
</tr>
<tr>
<td>Basal inf.</td>
<td>5.50 ± 1.42*</td>
<td>−6.33 ± 2.32*</td>
<td>−6.12 ± 1.36†</td>
<td>−1.57 ± 0.48</td>
<td>1.12 ± 0.45</td>
<td>1.40 ± 0.54</td>
</tr>
<tr>
<td>Mid-inf.</td>
<td>4.35 ± 1.74</td>
<td>−5.51 ± 2.13</td>
<td>−4.46 ± 1.97</td>
<td>−1.40 ± 0.49</td>
<td>1.57 ± 0.81</td>
<td>1.38 ± 0.74</td>
</tr>
<tr>
<td>Apical inf.</td>
<td>2.52 ± 1.89</td>
<td>−2.91 ± 1.95</td>
<td>−3.13 ± 2.01</td>
<td>−1.49 ± 0.32</td>
<td>1.66 ± 0.57</td>
<td>1.56 ± 0.79</td>
</tr>
<tr>
<td>Basal ant.</td>
<td>5.67 ± 1.37†</td>
<td>−6.21 ± 1.96†</td>
<td>−6.06 ± 1.68†</td>
<td>−1.88 ± 0.30</td>
<td>1.80 ± 0.79</td>
<td>2.35 ± 1.77</td>
</tr>
<tr>
<td>Mid-ant.</td>
<td>3.37 ± 1.55</td>
<td>−4.43 ± 1.86</td>
<td>−3.38 ± 1.62</td>
<td>−1.62 ± 0.30</td>
<td>2.19 ± 1.43</td>
<td>1.76 ± 0.97</td>
</tr>
<tr>
<td>Apical ant.</td>
<td>1.98 ± 1.21</td>
<td>−2.66 ± 1.85</td>
<td>−2.53 ± 1.46</td>
<td>−1.64 ± 0.37</td>
<td>1.98 ± 0.43</td>
<td>1.79 ± 0.88</td>
</tr>
</tbody>
</table>

* p < 0.001 basal vs. apical; †p < 0.05 basal vs. mid vs. apical. ant. = anterior; inf. = inferior; lat. = lateral; sep. = septal; SRI = strain rate imaging; TDI = tissue Doppler imaging. Other abbreviations as in Table 3.
non-transmural MI with sensitivity of 76.2%, 65.0%, 80.2%, and specificity of 30.8%, 40.0%, 67.9%, respectively, and these sensitivities and specificities were much lower than that of SRs.

**Reproducibility.** The intraobserver and interobserver variability for TDI parameters were similar at 3.9%, 4.2%, and 4.7% for Sm, Em, and Am, respectively. However, the intraobserver variability for SRs, SRe, and SRa were slightly higher than TDI, at 4.6%, 5.0%, and 6.1%, respectively. The interobserver variability of SRI parameters were 5.7%, 7.6%, and 8.2%, respectively.

The intraobserver and interobserver variability were compatible with previous studies, (13,17) and were considered to be reasonable in the context of the limitations of ultrasound technology.

**DISCUSSION**

The accurate identification of infarcted non-viable myocardium from viable but hypokinetic segments has important clinical implications. Revascularization only benefits patients with a sufficient amount of viable myocardium and is unlikely to benefit those with transmurally infarcted myocardium. Contrast-enhanced MRI has excellent spatial resolution and tissue characterization abilities, which make it ideal for defining areas of fibrotic nonviable myocardium and is now considered to be one of the “gold standard” tests of viability (8). However, it is not readily available, and it is expensive. Our present study has demonstrated the value of regional deformation by SRI when compared to Ce-MRI. It is an accurate, noninvasive, repeatable tool for defining the transmural extent of a myocardial scar and is superior to TDI; SRI reflects local segmental contractile function and is less affected by adjacent tissue tethering or overall parallel motion of the heart (12–15,22) and therefore is superior to TDI for assessing regional myocardial function. Our SRI data from normal subjects are consistent with previous studies that longitudinal segmental strain rate is uniform throughout all segments. In contrast, TDI has a significant velocity gradient across basal to apical LV segments (10,13,14). In this study, both SRs and SRe of the transmural infarcted segments were markedly reduced compared with normal myocardium, non-transmural, and subendocardial infarction segments. Moreover, SRs and SRe were also significantly lower in subendocardial infarction compared
with normal myocardium. In contrast, Sm and Em measured by TDI were unable to differentiate transmural from non-transmural infarcted segments. These results are compatible with previous studies by Voigt et al. (13) and Jamal et al. (14), respectively, who found that the longitudinal myocardial deformation by SRI correlated better with regional wall motion score than longitudinal myocardial velocities by TDI. However, the degree of infarction was not directly assessed in these studies, and the decrease in myocardial wall thickening is not always proportional to the percentage of necrosis. Our present study overcomes these limitations, as infarct size and transmural extent could be measured accurately by Ce-MRI, and we established the direct relationship between SRI, transmurality, and infarct size.

Our results are compatible with previous experimental studies. Weidemann et al. (17), in a correlative functional/histopathologic closed-chest animal study, showed that SRs and strain could also clearly differentiate non-transmural from transmural infarction. Our results, therefore, confirm their findings but in a clinical setting. In addition, we have demonstrated the corresponding cutoff values for SRs and SRe, which can differentiate transmural from non-transmural infarction, transmural from subendocardial infarction, and subendocardial MI from normal viable myocardium. These cutoff values provide an easy and rapid estimation of the transmurality of infarction, which may be of important clinical value.

In pathological studies, the chronically scarred transmural infarction is associated with a wall thickness of <0.6 cm (23,24). In our study, the mean wall thickness in the acute transmurally infarcted segments was slightly greater at 7.0 ± 1.1 mm, as completion of wall thinning may take up to three months after MI. However, the mean wall thickness was still significantly lower than the non-transmural group. Although wall thickness can be accurately measured both by echocardiography and MRI, the presence of akinesia and reduced end-diastolic wall thickness are unreliable as single criteria for the identification of completely scarred myocardium. Studies using [18F]fluorodeoxyglucose positron emission tomography (FDG-PET) have found residual metabolic activity, in the form of glycolysis, in myocardial regions with reduced end-diastolic wall thickness and absent systolic wall thickening (25). Baer et al. (26) found in a study comparing MRI with FDG-PET that using a definition of an end-diastolic wall thickness of ≤5.5 mm (the mean minus 2.5 standard deviations of a healthy control group) as an indicator of myocardial viability gave a sensitivity of 72% and specificity of 89% with a positive predictive accuracy of 91%. However, in our study using a cutoff wall thickness value of <5.5 mm only as an indicator of non-viability would have misclassified many transmurally infarcted non-viable myocardial segments, although the two studies are...
not directly comparable, as our patients were studied early after the infarction when thinning and remodeling may not have been fully completed. Therefore, the early assessment of infarct transmurality by wall thickness in the acute phase offered limited value for clinical decision making.

Our study also confirmed that the Q-wave in the electrocardiogram has limited value in differentiating transmural from non-transmural MI, which is consistent with previous studies (1,2). Importantly, SRs provides incremental value in identifying transmural MI over conventional electrocardiography and echocardiography.

**Study limitations.** Strain rate imaging signals are highly dependent on angle of insonation. In this study, every effort was made to ensure the tissue direction was less than 30° and as parallel as possible to the ultrasound beam. A narrow sector angle approach on an individual wall and breath-holding effort at the end of expiration obviates some of the above problems. Also, the signal-to-noise ratio of strain rate measurements can be improved with increasing sample volume but in exchange for lower spatial resolution. In this study, a sample volume of 9.2 mm proved to be the best compromise.

A major limitation to this study is the small number of patients with moderately or severely reduced ejection fraction, even those with transmural infarctions. In addition, patients' apical aneurysms are not commented on. However,
it reflects a consecutive series of patients with a first infarction undergoing contemporary management.

Conclusions. Strain rate imaging could differentiate a transmural infarction both from a non-transmural and from a subendocardial MI in the acute phase of MI and could differentiate a subendocardial MI from normal myocardium in patients with no conduction system abnormalities. Our derived cutoff values provide a rapid and easy assessment of the degree of transmurality of necrotic myocardium, which should be clinically useful.

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Reprint requests and correspondence: Dr. John E. Sanderson, Keele University Medical School, University Hospital of North Staffordshire NHS Trust, Department of Cardiology, City General Hospital, Stoke-on-Trent ST4 6QG, United Kingdom. E-mail: John.Sanderson@uhns.nhs.uk.

REFERENCES