Tissue Doppler Imaging Differentiates Transmural From Nontransmural Acute Myocardial Infarction After Reperfusion Therapy

Geneviève Derumeaux, MD, PhD; Joseph Loufoua, PhD; Gérard Pontier, BS; Alain Cribier, MD; Michel Ovize, MD, PhD

Background—The evaluation of transmural extent of necrosis after acute myocardial infarction remains a major problem in clinical practice. We sought to determine whether color M-mode tissue Doppler imaging (TDI) could differentiate transmural from nontransmural myocardial infarction.

Methods and Results—Twenty-one anesthetized open-chest dogs underwent 90 or 120 minutes of left anterior descending coronary artery occlusion followed by 180 minutes of reperfusion. The transmural extension of infarct was measured by triphenyltetrazolium chloride (TTC) staining. Segment shortening in the endocardium and epicardium of the anterior and posterior walls was assessed by sonomicrometry. Regional myocardial blood flow was measured by radioactive microspheres. TDI was obtained from an epicardial short-axis view. We calculated systolic and diastolic velocities within the endocardium and epicardium of myocardial walls and the subsequent myocardial velocity gradient (MVG). TTC staining could identify 2 groups according to the transmural extent of necrosis: 15 dogs had a nontransmural (NT) necrosis (42±3% of wall thickness), and 6 dogs developed a transmural (T) infarct (81±4% of wall thickness). In both groups, ischemia resulted in a significant and similar reduction in endocardial and epicardial velocities, with a resulting low systolic MVG in the anterior wall (0.10±0.07 s⁻¹ in NT and 0.10±0.08 s⁻¹ in T). At 60 minutes of reperfusion, systolic MVG failed to change significantly in the transmural group (−0.20±0.09 s⁻¹). In contrast, it increased significantly after reflow in the NT group compared with ischemic values (−0.99±0.20 versus 0.10±0.07 s⁻¹, P<0.05).

Conclusions—TDI can differentiate transmural from nontransmural myocardial infarction early after reperfusion. (Circulation. 2001;103:589-596.)

Key Words: echocardiography □ imaging □ myocardial infarction

Early assessment of the transmural extent of necrosis after acute myocardial infarction has become essential, especially since the development of revascularization techniques, because large infarct transmurality is associated with a great number of complications, such as left ventricular (LV) thrombus, arrhythmias, and death.¹ During a prolonged coronary artery occlusion, myocardial necrosis progresses from endocardium toward epicardium as a wave-front phenomenon.² Experimental as well as clinical studies have demonstrated that early reperfusion of the ischemic myocardium can salvage jeopardized tissue.³,⁴ Anatomic-pathological studies revealed the great heterogeneity of the reperfused myocardium that contains a variable amount of necrosis surrounded by a viable but transiently stunned epicardium.⁵ This structural and functional heterogeneity complicates interpretation of wall motion abnormalities by conventional echocardiography. Lieberman et al⁶ demonstrated that the decrease in myocardial wall thickening is not proportional to the percentage of necrosis, unless the necrosis affects less than the subendocardial 20% of myocardial wall thickness. Nagueh et al⁷ recently reported that myocardial heterogeneity needs to be taken into account in analysis of functional recovery in patients with coronary artery disease. In addition, conventional methods use endocardial motion to assess regional myocardial function, which may overestimate the extent of irreversible myocardial injury because of the impossibility of distinguishing viable but severely stunned or hibernating from necrotic myocardium.⁶,⁷,⁸,⁹

New ultrasound techniques, such as integrated backscatter, myocardial contrast echocardiography, and tissue Doppler imaging (TDI) have emerged and look very promising for the quantification and characterization of transmural contractile function and perfusion.¹⁰⁻¹⁵ M-mode TDI allows quantification in real time of endocardial and epicardial velocities by detection of consecutive phase shifts of the ultrasound signal reflected from the contracting myocardium and provides new indices of myocardial function, such as the myocardial velocity gradient (MVG).¹⁰⁻¹¹,¹⁶

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The objective of the present study, performed in a canine model of irreversible ischemic injury, was to investigate whether M-mode TDI may also help to differentiate transmural from nontransmural myocardial infarction by directly comparing histological assessment of viability and M-mode TDI recordings.

Methods

All experiments performed in this study conformed to the "Guiding Principles in the Care and Use of Animals" approved by the American Physiological Society.

Surgical Preparation

Twenty-six adult dogs weighing 20 to 39 kg were anesthetized with pentobarbital (20 mg/kg IV), ventilated with room air through a tracheotomy tube, and prepared as previously described.17 A segment of the left anterior descending coronary artery (LAD) was isolated just before the first diagonal branch for further occlusion and reperfusion. Two pairs of ultrasonic crystals, used to assess regional contractile function, were inserted into the endocardium and the epicardium of both the anterior and posterior walls.

Echocardiography

Echocardiography was performed with a SEQUOIA system (Acuson) with a 7-MHz transducer. Myocardial velocities resulting from the LV circumferential contraction were measured with the beam positioned on the mid anterior wall, from an epicardial short-axis view at the level of the papillary muscles. Gray-scale receive gain was set to optimize the clarity of the endocardial and epicardial boundaries. Doppler receive gain was adjusted to maintain optimal coloring of the myocardium. Doppler velocity range was set as low as possible to avoid aliasing occurrence. The angle of interrogation of the M-mode beam was carefully aligned to be perpendicular to the LV walls. Freeze-frame images were then downloaded to a magneto-optic disk and transferred to an IBM-compatible computer for offline analysis of myocardial velocities and gradient by a custom-made software, as previously described.12

Experimental Protocol

After baseline measurements, the LAD was occluded (by means of a vascular clamp) for 90 (n=17) or 120 (n=9) minutes and reperfused for 180 minutes. Echographic and sonomicrometry recordings and regional myocardial blood flow (RMBF) measurements were performed sequentially at the following time points: at baseline, 5 minutes of occlusion, and 60 and 180 minutes after reperfusion.

At the end of each experiment, the heart was excised and cut into 5 to 7 slices 10 mm thick parallel to the atrioventricular groove for further assessment of infarct size, as previously described.18 We verified that the anterior wall at the level of the papillary muscles was unstained, ie, that the TDI interrogation was well within the ischemic area. The correct position of the 2 pairs of ultrasonic crystals (ie, endocardial or epicardial) was checked within both the area at risk and the remote nonischemic zone.

Analysis

Echocraphic Measurements

Conventional measurements (LV wall thickness, wall thickening, and LV cavity dimensions) were obtained from gray-scale M-mode tracings according to the criteria of the American Society of Echocardiography. With M-mode TDI, endocardial and epicardial velocities were calculated within both anterior and posterior walls. The anterior and posterior walls were arbitrarily divided from the endocardial to epicardial borders into 2 layers of equal thickness by manual tracing of endocardial and epicardial boundaries. Endocardial and epicardial mean velocity was defined as the average value of the velocity estimates measured along each M-mode scan line throughout the thickness of the inner and outer layers of myocardial walls. MVG was defined as the difference between endocardial and epicardial velocities divided by wall thickness. Three beats were averaged for each of these measurements.

Area at Risk and Area of Necrosis

Each transverse heart slice was incubated for 15 minutes in a 1% solution of triphenyltetrazolium chloride (TTC) at 37°C. This method has been shown to reliably identify necrotic myocardium (which appears pale) from viable myocardium (which stains brick red) (Figure 1).19 The slices were then photographed for further computerized planimetry of the necrotic area. The heart slice encompassing the papillary muscles and in which sonomicrometry crystals had been inserted (ie, the area of TDI interrogation) was set apart. Enlarged projection of this slide was traced for determination of the boundaries of the area of necrosis. From this slide, we calculated an index (called transmural extension index, TME) measuring the degree of extension of the infarct from the endocardium toward the epicardium (Figure 1). Within the 2 lateral borders of the necrotic area, 10 equidistant transmural radii were traced perpendicular to the endocardial and epicardial boundaries. On each radius, we measured the distance from the endocardial border to the external limit of the infarcted zone and expressed it as a percentage of the distance between the endocardial and epicardial borders. We then calculated the TME, defined here as the mean value of the percentages of the 10 radii. With this method, a fully transmural infarct has a TME of 100%, whereas a TME of 0% indicates the absence of necrosis. We arbitrarily opposed subendocardial infarcts, identified here as the nontransmural group, involving

![Figure 1. Infarct size determination by TTC staining and measurement of TME. Top, Example of a nontransmural infarct (NT group) caused by 90 minutes of LAD coronary artery occlusion followed by 180 minutes of reperfusion. Bottom, Calculation of TME. TME = a − b length/a − c length (TME = 35% in this example).](image-url)
only endocardial layers (ie, inner half of wall thickness and TME<50%), to the others, identified as the transmural group, involving endocardial and any degree of the epicardial layers.

**Regional Myocardial Blood Flow**

RMBF (mL·min⁻¹·g⁻¹) was assessed by injection of radioactive microspheres labeled with either ¹⁴¹Ce, ⁹⁵Nb, or ¹⁰³Ru (Dupont–New England Nuclear), as previously described.¹⁸,²⁰

**Hemodynamics, Segment Shortening, and Postsystolic Shortening**

Heart rate and arterial blood pressure were measured and averaged over 5 continuous cardiac cycles in sinus rhythm for each sample period. LV dP/dt was used to define the timing of the cardiac cycle for segment length measurements with the ultrasonic crystals: end-diastolic length (EDL) was measured at the onset of the rapid increase in LV dp/dt; and end-systolic length (ESL) was measured at peak negative LV dp/dt. Minimal segment length (mSL) was defined as the minimal separation between the 2 ultrasonic crystals, irrespective of the time point of the cardiac cycle (under baseline conditions, mSL was equal to end-systolic length). Segment shortening (SS), an index of regional contractile function, was defined as SS=[(mean EDL−mean ESL)/mean EDL]×100. Postsystolic shortening (PSS) was defined as PSS=[(ESL−mSL)/mean EDL]×100.

**Statistics**

Baseline and subsequent echographic and sonomicrometric measurements were compared by repeated-measures ANOVA. Student’s t test was used to compare nontransmural and transmural necrosis. Sensitivity and specificity of myocardial velocities and velocity gradient as indices of TME were determined by constructing receiver operating characteristic curves.²¹ Sensitivity and specificity were then plotted against the whole range of velocity and velocity gradient values for determining the best cutoff point, defined as the intersection of the sensitivity/specificity curves. The sensitivity/specificity values at these cutoff points are reported in percentage with the corresponding 95% confidence interval (CI). All values are presented as mean±SEM. A value of P<0.05 was considered statistically significant.

**Results**

**Infarct Size and TME**

Among the 26 dogs that were entered into the study, 24 developed myocardial necrosis, as demonstrated by TTC staining. Three dogs were excluded because of persistent ventricular arrhythmias. Thus, data are presented for the remaining 21 experiments.

We arbitrarily divided dogs into 2 groups. Fifteen dogs exhibited a TME of <50% and formed the nontransmural (NT) group (mean TME, 41±3%). Six dogs that displayed a TME>50% constituted the transmural (T) group: mean TME in this group averaged 81±4%.

**Hemodynamics and RMBF**

The 2 groups had comparable heart rates and blood pressures at baseline and throughout the experiment. Baseline endocardial and epicardial RMBFs were similar in the NT and T groups (Table 1). As expected, LAD occlusion resulted in a dramatic decrease in endocardial and epicardial RMBF in the ischemic zone in both groups, with mean RMBF averaging 0.05±0.03 and 0.09±0.01 mL·min⁻¹·g⁻¹ in the endocardium and 0.10±0.04 and 0.31±0.04 mL·min⁻¹·g⁻¹ in the epicardium in the T and NT groups, respectively (P<0.01 versus baseline, P=NS between groups). At 60 minutes after reflow, RMBF in the anterior wall returned to near baseline values in both groups, except for endocardial RMBF in the T group that remained significantly reduced (P<0.05 versus baseline).

**Myocardial Velocity Changes During Ischemia/Reperfusion**

**Ischemic Wall**

LAD occlusion resulted in a severe impairment of anterior wall motion in both groups, which displayed a comparable decrease in wall thickening from 49±3% to −4±2% and from 54±5% to −1±6% in the T and NT groups, respectively (P<0.05 versus baseline for both, P=NS between groups). In both groups, systolic velocities decreased dramatically to a similar extent in the endocardium and in the epicardium (Table 2 and Figure 2). Consequently, systolic MVG (sMVG) was reduced significantly from −2.6±0.3 s⁻¹ at baseline to 0.10±0.07 s⁻¹ during ischemia in the NT group, and from −2.1±0.3 s⁻¹ at baseline to 0.10±0.08 s⁻¹ during ischemia in the T group (P<0.05 versus baseline for T and NT, P=NS between groups). Early diastolic velocities decreased significantly in the NT but not in the T group, but to a lesser extent than systolic velocities. Late diastolic velocities tended to increase in both groups, although not significantly (Table 2).

Sixty minutes after reperfusion, anterior wall thickening remained severely and similarly depressed in both groups, averaging 4±3% and 2±1% in the NT and T groups, respectively (P<0.05 versus baseline for both groups, P=NS versus ischemia and between groups). At 60 minutes after

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### Table 1. RMBF in NT and in T Groups

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NT</td>
<td>T</td>
<td>NT</td>
</tr>
<tr>
<td>Ischemic zone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endocardium</td>
<td>0.94±0.08</td>
<td>0.83±0.07</td>
<td>0.09±0.01*</td>
</tr>
<tr>
<td>Epicardium</td>
<td>1.00±0.07</td>
<td>0.90±0.06</td>
<td>0.31±0.04*</td>
</tr>
<tr>
<td>Nonischemic zone</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endocardium</td>
<td>1.16±0.13</td>
<td>1.05±0.12</td>
<td>1.37±0.12</td>
</tr>
<tr>
<td>Epicardium</td>
<td>1.06±0.11</td>
<td>0.99±0.13</td>
<td>1.32±0.12</td>
</tr>
</tbody>
</table>

Values are expressed as mL·min⁻¹·g⁻¹, mean±SEM. *P<0.01 vs baseline.
reflow, systolic velocities and sMVG failed to increase significantly in the T group (Table 2 and Figure 2). In contrast, both endocardial and epicardial systolic velocities (and consequently sMVG) increased significantly with respect to the preceding occlusion values in NT hearts, with sMVG averaging $2.09\pm 0.22$ s$^{-1}$ versus 0.10$\pm 0.07$ s$^{-1}$ during ischemia ($P<0.05$) (Table 2 and Figure 2). In both groups, systolic velocities and sMVG remained stable throughout the 3 hours of reperfusion, at values similar to those at 60 minutes after reflow (Figure 2). During reperfusion, sMVG was better correlated than anterior wall thickening with the transmural extent of necrosis (Figure 3).

**TABLE 2. Comparison of Myocardial Velocities (cm $\cdot$ s$^{-1}$) and MVG (s$^{-1}$) in NT and T Groups**

<table>
<thead>
<tr>
<th></th>
<th>NT Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
<th>T Baseline</th>
<th>Occlusion</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systole</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$V_s$ epicardium</td>
<td>$-2.2\pm 0.3$</td>
<td>$-0.4\pm 0.1$</td>
<td>$-0.8\pm 0.3$</td>
<td>$-2.1\pm 0.4$</td>
<td>$-0.4\pm 0.1$</td>
<td>$-0.4\pm 0.2$</td>
</tr>
<tr>
<td>$V_s$ endocardium</td>
<td>$-4.5\pm 0.5$</td>
<td>$-0.2\pm 0.2$</td>
<td>$-1.2\pm 0.4$</td>
<td>$-4.1\pm 0.8$</td>
<td>$-0.3\pm 0.2$</td>
<td>$-0.5\pm 0.2$</td>
</tr>
<tr>
<td>MVG S</td>
<td>$-2.6\pm 0.3$</td>
<td>$0.1\pm 0.07$</td>
<td>$-0.99\pm 0.2$</td>
<td>$-2.1\pm 0.3$</td>
<td>$0.1\pm 0.08$</td>
<td>$-0.2\pm 0.09$</td>
</tr>
<tr>
<td><strong>Diastole</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$V_e$ epicardium</td>
<td>$2.5\pm 0.4$</td>
<td>$1.1\pm 0.3$</td>
<td>$1.1\pm 0.2$</td>
<td>$1.9\pm 0.3$</td>
<td>$1.4\pm 0.6$</td>
<td>$1.6\pm 1.1$</td>
</tr>
<tr>
<td>$V_e$ endocardium</td>
<td>$4.0\pm 0.4$</td>
<td>$1.6\pm 0.4$</td>
<td>$2.0\pm 0.4$</td>
<td>$3.9\pm 0.5$</td>
<td>$2.0\pm 0.6$</td>
<td>$2.1\pm 1.1$</td>
</tr>
<tr>
<td>MVG E</td>
<td>$2.3\pm 0.3$</td>
<td>$0.6\pm 0.2$</td>
<td>$0.8\pm 0.2$</td>
<td>$2.4\pm 0.4$</td>
<td>$0.9\pm 0.4$</td>
<td>$0.4\pm 0.1$</td>
</tr>
<tr>
<td>$V_s$ epicardium</td>
<td>$3.3\pm 0.9$</td>
<td>$3.5\pm 1$</td>
<td>$2.9\pm 0.8$</td>
<td>$2.6\pm 1.5$</td>
<td>$4.7\pm 1.9$</td>
<td>$2.3\pm 0.9$</td>
</tr>
<tr>
<td>$V_s$ endocardium</td>
<td>$4.0\pm 0.8$</td>
<td>$4.3\pm 1$</td>
<td>$2.8\pm 0.7$</td>
<td>$2.6\pm 0.7$</td>
<td>$6.4\pm 2.2$</td>
<td>$2.6\pm 0.7$</td>
</tr>
<tr>
<td>MVG A</td>
<td>$1.2\pm 0.2$</td>
<td>$1.5\pm 0.2$</td>
<td>$1.2\pm 0.3$</td>
<td>$1.5\pm 0.9$</td>
<td>$1.3\pm 0.3$</td>
<td>$0.6\pm 0.4$</td>
</tr>
</tbody>
</table>

$V$ indicates velocity; S, systole; E, early diastole; and A, late diastole.

* $P<0.05$ vs NT group; † $P<0.01$ vs baseline; ‡ $P<0.05$ vs occlusion.

**Figure 2.** Endocardial and epicardial systolic velocities and sMVG in T and NT groups during ischemia and reperfusion. Systolic velocities (top) and sMVG (bottom) are presented in NT (left) and T (right) groups. LAD occlusion resulted in a significant decrease of both endocardial and epicardial systolic velocities and sMVG in 2 groups. During reperfusion, systolic velocities and sMVG significantly increased only in NT group. $V_s$ indicates systolic velocities; ENDO, endocardium; EPI, epicardium. * $P<0.05$ vs baseline; † $P<0.05$ vs occlusion.
Overall, the best sensitivity for the distinction between the T and NT infarcts was provided by the analysis of the sMVG. A value of sMVG of $-0.3\ s^{-1}$ had a sensitivity and a specificity of 70% for identifying transmural necrosis (TME>50%). A value of sMVG of $-0.9\ s^{-1}$ had a sensitivity and a specificity of 81% for identifying nontransmural necrosis (TME<40%) (Table 3 and Figure 4).

Interestingly, M-mode TDI traces clearly displayed a PSS during ischemia and reperfusion (Figure 5). PSS velocities and PSS MVG were similar in the T and NT groups during ischemia, with PSS MVG averaging $-2.3\pm0.3\ s^{-1}$ in the T group versus $-2.5\pm0.4\ s^{-1}$ in the NT group ($P=NS$). In addition, during reperfusion, PSS MVG failed to change in either the NT or the T group. These TDI data were confirmed by analysis of sonomicrometry tracings that showed that during ischemia, PSS was similar in the T and NT groups, as well as in the endocardium (10±2% in T versus 13±2% in NT) and in the epicardium (8±1% in T versus 8±1% in NT) ($P=NS$). Consistent with M-mode TDI findings, PSS persisted during reperfusion and remained comparable in the T and NT groups, averaging 3±2% and 6±1% in the endocardium and 2±1% and 3±1% in the epicardium of the T and NT groups, respectively ($P=NS$ between groups) (Figure 5).

Nonischemic Wall
During ischemia, wall thickening and endocardial and epicardial systolic velocities increased slightly but not significantly in the nonischemic territory in both groups.

Discussion
The present study suggests that M-mode TDI may differentiate nontransmural from transmural myocardial infarction during reperfusion and therefore could help to assess myocardial viability.

In close agreement with previous observations using sonomicrometry, we recently demonstrated that M-mode TDI is able to assess heterogeneity of contraction across the myocardial wall after a reversible ischemic injury.12,22 Whereas conventional M-mode imaging failed to detect any significant improvement in transmural wall thickening during reflow after 15 minutes of ischemia, M-mode TDI could reveal a slight but significant increase in endocardial (but not epicardial) velocities, resulting in the resumption of the MVG.12

In the present study, we sought to determine whether M-mode TDI was able to differentiate nontransmural infarcts from transmural infarcts early after reperfusion. As expected, distinction between transmural and nontransmural infarction by M-mode TDI failed during ischemia. In contrast, it became possible as early as 1 hour after reflow. In the T group, endocardial and epicardial systolic velocities, and then sMVG, failed to recover after reflow. TTC staining revealed that this absence of functional recovery was related to the presence of extensive necrosis in both myocardial layers. In addition, measurement of myocardial blood flow suggests that there probably were areas of no reflow in the endocardium of T-group hearts at 1 hour of reperfusion. Moreover, the limited rim of viable tissue that persisted in the outer part of the epicardium was very likely stunned and tethered to the underlying necrotic endocardium, which further limited contraction.

The most important finding of the present study is that sMVG recovered significantly after reflow in the NT group. This strongly suggests that active contraction of a functionally significant amount of viable myocardial tissue resumed as a consequence of reperfusion. This increase of sMVG was due to a partial recovery of both endocardial and epicardial systolic velocities at 60 minutes after reflow (23% and 22% increase versus ischemic velocities, respectively). Absence of full recovery of sMVG at that time is not surprising and is enlightened by our histological and myocardial blood flow data. Although myocardial blood flow was restored to near baseline values, TTC staining clearly demonstrated that the endocardium was partly necrotic in most NT hearts. This suggests that part of the endocardium was salvaged by reperfusion but remained severely stunned and possibly

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**Table 3. Sensitivity/Specificity of sMVG for Various TMEs of Myocardial Infarction**

<table>
<thead>
<tr>
<th>TME, %</th>
<th>Cutoff Value, s⁻¹</th>
<th>Ss/Sp, %</th>
<th>95% CI, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>-0.9</td>
<td>81</td>
<td>17</td>
</tr>
<tr>
<td>50</td>
<td>-0.3</td>
<td>70</td>
<td>20</td>
</tr>
<tr>
<td>80</td>
<td>-0.3</td>
<td>76</td>
<td>18</td>
</tr>
</tbody>
</table>

Ss indicates sensitivity; Sp, specificity.
Figure 4. Receiver operating characteristic plots of sMVG in relation to TME >40%, >50%, and >80%. A value of sMVG of $0.3 \text{ s}^{-1}$ had a sensitivity and a specificity of 70% for distinguishing nontransmural from transmural infarcts. A value of sMVG of $0.3 \text{ s}^{-1}$ had a sensitivity and a specificity of 76% for identifying transmural necrosis (TME >80%), and a value of sMVG of $0.9 \text{ s}^{-1}$ had a sensitivity and a specificity of 81% for identifying nontransmural necrosis (TME <40%).
tethered to the adjacent necrotic part of the inner layer. The epicardium was fully viable in the NT group, and epicardial blood flow was restored to near baseline values at 1 hour of reperfusion. Limited recovery of epicardial systolic velocities most likely results from the combined effects of several factors. First, as demonstrated by Myers et al., a gradient of thickening exists across the myocardial wall, with the inner half contributing to 70% of the total systolic thickening. Second, in agreement with previous reports, the epicardium was probably tethered to the underlying dysfunctional endocardium. Third, the outer wall, which had been severely ischemic during coronary artery occlusion, as demonstrated by myocardial blood flow measurements, was probably stunned at 60 and 180 minutes after reflow. Because the duration of reperfusion was limited to only a few hours in our experimental preparation, we cannot know to what extent contractile function would finally have recovered in the NT group. In any case, as early as 60 minutes after reperfusion, M-mode TDI was able to accurately quantify the regional inner and outer myocardial layer dysfunction and identify viable myocardium within the ischemic/reperfused territory.

In the present study, both sonomicrometry and M-mode TDI identified postsystolic shortening during ischemia and reperfusion. Several studies have reported that PSS is consistently observed during severe ischemia. The meaning of its persistence after reperfusion and the underlying mechanism, however, are a matter of debate. Mainly, it is not clear whether PSS represents an active delayed contraction (ie, identifying viable myocardium) or a passive elastic recoil. In the present study, during reperfusion, fully necrotic myocardium of T hearts exhibited PSS similar to that of NT hearts, on both sonomicrometry and TDI traces, as depicted in Figure 5. This strongly suggests that PSS is a passive phenomenon due to elastic recoil of dysfunctional myocardium.

The present study has potential important clinical implications, ie, identification and quantification of the regional contractile dysfunction that may arise during or as a consequence of acute coronary syndromes. This is particularly important because asynergic myocardium in the clinical setting usually combines an admixture of areas in different states (scar, ischemia, stunning, or hibernation). Further studies are needed, however, to determine whether these data can apply to human patients.

Acknowledgment

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