Microvascular Obstruction and Left Ventricular Remodeling Early After Acute Myocardial Infarction

Bernhard L. Gerber, MD; Carlos E. Rochitte, MD; Jacques A. Melin, MD; Elliot R. McVeigh, PhD; David A. Bluemke, MD, PhD; Katherine C. Wu, MD; Lewis C. Becker, MD; João A.C. Lima, MD

**Background**—The presence of microvascular obstruction (MO) within infarcted regions may adversely influence left ventricular (LV) remodeling after acute myocardial infarction. This study examined whether the extent of MO directly alters the mechanical properties of the infarcted myocardium.

**Methods and Results**—Seventeen dogs underwent 90 minutes of balloon occlusion of the left anterior descending coronary artery, followed by reperfusion. Gadolinium-enhanced perfusion MRI and 3D-tagging were performed 4 to 6 and 48 hours (8 animals) and 10 days (9 animals) after reperfusion. Early increase in LV end-diastolic volume (from 42±9 to 54±14 mL, P<0.05) between 4 to 6 and 48 hours after reperfusion was predicted by both extent of MO (r=0.89, P<0.01) and infarct size (r=0.83, P<0.01), defined as MRI hypoenhanced and hyperenhanced regions, respectively. Multivariate analysis demonstrated that extent of MO had better and independent value to predict LV volume than overall infarct size. A strong inverse relationship existed between magnitude of first principal strain (r=−0.80, P<0.001) and relative extent of MO within infarcted myocardium. Also, infarcted myocardium involved by extensive areas of MO demonstrated reductions of circumferential (r=−0.61, P<0.01) and longitudinal (r=−0.53, P<0.05) stretching. Furthermore, significant reductions of radial thickening (9±6% versus 14±3%, P<0.01) occurred in noninfarcted regions adjacent to infarcts that had increased (>35%) amounts of MO.

**Conclusions**—In the early healing phase of acute myocardial infarction, the extent of MO in infarcted tissue relates to reduced local myocardial deformation and dysfunction of noninfarcted adjacent myocardium. Such strain alterations might explain the increased remodeling observed in patients with large regions of MO. (*Circulation*, 2000;101:2734-2741.)

**Key Words:** myocardial infarction ▪ tomography ▪ magnetic resonance imaging ▪ remodeling

Reperfusion therapy has been shown to have a salutary effect on postinfarction remodeling. Yet, despite restoration of epicardial blood flow, the infarct core may undergo limited reperfusion at the tissue level because of injury to the microvasculature and its subsequent obstruction by erythrocytes, neutrophils, and debris, a phenomenon also known as the “no-reflow” phenomenon. The presence of microvascular obstruction (MO) was shown to predict postinfarct remodeling. The mechanisms by which MO may influence left ventricular (LV) remodeling, however, are still poorly understood. One possible hypothesis is that MO could directly alter the mechanical properties of infarcted myocardium. Altered stiffness with consequent changes in myocardial strain in the region involved by MO could foster enhanced expansion of infarcted and/or noninfarcted myocardium.

MRI using tissue tagging offers the unique opportunity to study 3D mechanical deformation of the heart noninvasively in vivo. In addition, with gadolinium-enhanced perfusion imaging, infarct size and MO may also be quantified and followed up noninvasively over time. Using these novel techniques, we sought to determine whether MO would alter mechanical properties of the infarcted and adjacent noninfarcted tissue and what influence this might have on early myocardial infarct expansion and remodeling. In a canine model of infarction and reperfusion, we thus correlated infarct size and extent of MO with alterations of myocardial strains by MRI tissue tagging.

**Methods**

Eighteen adult mongrel dogs of either sex (20 to 25 kg) were studied. The animals in this study were handled according to the “Position of the American Heart Association on Research Animal Use,” adopted November 15, 1984. Data from these animals have been published in 2 previous studies reporting on the time course of MO after myocardial infarction (MI).

**Experimental Preparation**

The experimental preparation has been described in detail elsewhere. In summary, animals were anesthetized and underwent 90...
minutes of closed-chest occlusion of the proximal left anterior descending coronary artery (LAD) with an angioplasty balloon to produce MI. After 90 minutes of occlusion, the balloon was deflated to allow full reperfusion of the infarcted myocardium. Animals were allowed to recover and were kept alive for 2 to 10 days. Two groups of experiments were performed. Nine animals were studied early (4 to 6 hours and again at 48 hours) after reperfusion. Nine other animals were studied at 48 hours and 10 days after reperfusion. Data from 1 animal of the first group were subsequently excluded because of the absence of significant myocardial necrosis at pathology.

**MRI Protocol**

MRI was performed with a 1.5-T magnet while the animals were under general anesthesia. Tagged images were acquired with an ECG-triggered, segmented k-space spoiled gradient recalled (SPGR) pulse sequence with spatial modulation of magnetization (DANTE-SPAMM). Contiguous stacks of short-axis images were prescribed to cover the entire heart from base to apex. Six long-axis slices were then prescribed radially every 30° (Figure 1). Imaging parameters were as follows: tag separation 6 mm, 32-cm field of view, 10-mm slice thickness, matrix size 256×160, TR 6.5 ms, TE 2.3 ms, flip angle α=15°, and temporal resolution 32 ms.

After completion of the tagged imaging sequence, an intravenous bolus injection of 0.225 mmol/kg gadopentetate dimeglumine (Magnevist, Berlex) was given. Details of the MRI perfusion pulse sequence are given elsewhere. Briefly, it consisted of a fast SPGR acquisition with nonselective preparatory radiofrequency pulses used to drive magnetization to a steady state before and between image acquisition. The same short-axis slice prescription as for the tagged imaging protocol was used. Images were acquired starting 10 seconds after contrast injection and continued up until 15 minutes thereafter.

**MRI Data Analysis**

Detection of contour and taglines on tagged image sets was performed with the semiautomated detection program FINDTAGS. 3D strains were estimated by a field-fitting method and reported in the form of a strain map consisting of 12 circumferential angular sectors, 3 radial layers, and 4 to 5 longitudinal planes. The intersection of the anterior and septal walls was used as an anatomic landmark to coregister strain maps with perfusion image sets. Strains were expressed as percent change of length between the baseline and the maximally deformed state. We calculated normal strains in the 3 normal orthogonal directions (radial, circumferential, and longitudinal). In addition, we report first principal strain E₁ (maximum thickening) as vector magnitude, and angle against the circumferential (αE₁C) and longitudinal (αE₁L) axis (Figure 2).

Perfusion image sets were analyzed with NIH Image software. Two well-described patterns of myocardial contrast enhancement were used to differentiate noninfarcted regions from infarcted regions with and without MO. Briefly, infarcted tissue is characterized by persistent hyperenhancement on late images (15 minutes after contrast injection) compared with normal noninfarcted myocardium. Tissue with MO can be identified by areas of early (2 to 3 minutes after contrast injection) hypoenhancement relative to surrounding myocardium. Extent of hyperenhanced and hypoenhanced regions was expressed in % LV area, as previously described. LV end-diastolic (EDV) and end-systolic (ESV) volumes were calculated from planimetered endocardial and epicardial areas of interleaved short-axis images reported by the FINDTAGS program by use of a modified Simpson’s rule according to the following formula:

\[
V_{ol} = \left( \frac{A_1 + A_3 + A_5}{2} + \frac{A_2 + A_4}{3} \right) \times \frac{l}{5},
\]

where A₁ through A₅ are the respective areas of the LV slices numbered from apex to base and l is the total length of the LV. LV ejection fraction was computed as the ratio of (EDV−ESV)/EDV.

Figure 1. Sample tagged image data set, consisting of 2 sets of short-axis and 1 stack of long-axis views.

Figure 2. Schematic shows how infarcted, adjacent, and remote regions were defined. Infarcted regions presented late gadolinium hyperenhancement. Adjacent regions were in same plane as infarcts but not hyperenhanced. Remote regions were situated in inferior-posterior wall. Sector is magnified to illustrate direction of 3 orthogonal normal strains: E_C (circumferential), E_R (radial), and E_L (longitudinal) and of maximal first principal strain (E₁). Orientation of E₁ is expressed by 2 angles against circumferential (αE₁C) and longitudinal (αE₁L) directions.
Infarct Size and Extent of MO

Results of LV volumes and ejection fraction at different time points after reperfusion are also shown in Table 1. EDV increased significantly ($P<0.05$) between 4 to 6 hours and 48 hours after reperfusion (Figure 3). No change in EDV occurred in the animals studied between 48 hours and 10 days after reperfusion. The increase in LV volume between 4 to 6 and 48 hours was accompanied by reductions in mean diastolic wall thickness in infarcted (from 10.9±1.3 to 9.6±1.1 mm, $P<0.05$), adjacent (from 10.9±1.3 to 9.3±1.3 mm, $P<0.005$), and remote (from 10.1±1.5 to 8.3±0.9 mm, $P<0.01$) segments. No further decrease in mean wall thickness occurred beyond 48 hours of reperfusion. LV ejection fraction remained unchanged between 4 to 6 and 48 hours and between 2 and 10 days after reperfusion.

MO, Infarct Size, and Changes in LV Volumes After Reperfusion

There was no significant relationship between LV volumes and extent of MO or infarct size early (4 to 6 hours) after reperfusion. Yet, the extent of MO measured by MRI at that time strongly predicted the increase in EDV ($r=0.89$, $P<0.005$) and ESV ($r=0.69$, $P=0.05$) that occurred between 4 to 6 to 48 hours after infarction. Extent of MO also correlated highly with both absolute EDV ($r=0.77, P<0.001$) and ESV ($r=0.70, P<0.001$) 48 hours after reperfusion and at the end of the study (Figure 4). Similarly, infarct size measured by MRI 6 hours after reperfusion predicted increases of both EDV ($r=0.83, P<0.01$) and ESV ($r=0.84, P<0.01$).

The predictive values of extent of MO and infarct size on LV remodeling were compared relative to each other by multivariate analysis (Table 2). Extent of MO had higher value to predict EDVs at the end of the study than overall infarct size ($r=0.84$ versus $r=0.74$). Total infarct size did not have additional predictive value once MO was entered into a multivariate model (model 1, Table 2). Because total infarct size contains areas with MO as well as infarcted regions without MO (fully reperfused, presenting late hyperenhancement but no early hypoenhancement on MRI), we analyzed the predictive value of these 2 regions separately and combined. Extent of infarcted myocardium with MO was found to have higher predictive value for EDV than extent of infarcted myocardium without MO. Size of infarcted myocardium with MO had additional predictive value to the size of infarcted myocardium without MO (model 2, Table 2), but not vice versa (model 3, Table 2). These findings reflect that the effects of MO on LV dilatation are stronger than those of infarcted but persistently reperfused tissue. They indicate the importance of MO as a determinant of LV remodeling after MI. Findings were similar when postmortem-defined infarct size itself ($r=0.28, P=NS$). No significant changes in infarct size or in the proportion of infarcted myocardium with MO, as measured by MRI, occurred between 48 hours and 10 days. Postmortem measurements of TTC-negative area (23±14%) and thioflavine-negative area (10±10%) correlated highly with MRI estimates of infarct size and MO ($r=0.89$ and 0.91, $P<0.001$, respectively).

LV Volumes After Infarction and Reperfusion

<table>
<thead>
<tr>
<th>Infarct size, %</th>
<th>4–6 Hours</th>
<th>48 Hours</th>
<th>10 Days</th>
</tr>
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<tbody>
<tr>
<td>Group 1 (n=8)</td>
<td>25±10</td>
<td>29±12†</td>
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</tr>
<tr>
<td>Group 2 (n=9)</td>
<td>...</td>
<td>25±8</td>
<td>24±9</td>
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<tr>
<td>Size of MO, %</td>
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<td></td>
</tr>
<tr>
<td>Group 1 (n=8)</td>
<td>5.6±7.1</td>
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</tr>
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<td>Group 2 (n=9)</td>
<td>...</td>
<td>8.0±4.6</td>
<td>7.3±2.9</td>
</tr>
<tr>
<td>EDV, mL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (n=8)</td>
<td>42±6</td>
<td>54±14*</td>
<td>...</td>
</tr>
<tr>
<td>Group 2 (n=9)</td>
<td>...</td>
<td>45±5</td>
<td>42±4</td>
</tr>
<tr>
<td>ESV, mL</td>
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<td></td>
<td></td>
</tr>
<tr>
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<tr>
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<tr>
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<td></td>
<td></td>
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<tr>
<td>Group 1 (n=8)</td>
<td>36±12</td>
<td>33±14</td>
<td>...</td>
</tr>
<tr>
<td>Group 2 (n=9)</td>
<td>...</td>
<td>51±10‡</td>
<td>58±19</td>
</tr>
</tbody>
</table>

Table 1. LV Volumes, Ejection Fraction, and Size of Infarct and of MO Early and Late After Reperfusion

EF indicates ejection fraction. 
*P<0.05 and †P<0.005 vs earlier observation of the same group of animals; ‡P<0.05 vs animals from other group.

Pathology

After completion of the terminal imaging study, 20 mL of 4% thioflavine S solution was injected into the LV via a pigtail catheter. Hearts were arrested with KCl, excised, cut into 5 to 6 regularly spaced short-axis slices, and viewed under ultraviolet light to define the area of MO (thioflavine-negative myocardium). Slices were then incubated in 2% 2,3,5-triphenyltetrazolium chloride (TTC) solution for 20 minutes at 37°C to define infarct size. The extents of thioflavine- and of TTC-negative regions were planimetered and expressed as percent of total LV mass.

Statistical Analysis

Values are reported as mean±SD. Paired Student’s $t$ test was used to assess differences in continuous variables, such as LV volumes, LV mass, percent infarct size, and percent MO between 6 and 48 hours and between 48 hours and 10 days after reperfusion. Two-sample Student’s $t$ test was used to compare strains in infarcts with high and low amounts of MO. Simple linear regression analysis was used to assess the correlation between infarct size and the size of MO and cavity volumes. Stepwise regression analysis was then used to compare the individual and additional relative values of both of these parameters on LV cavity volume. All tests were 2-sided, and a value of $P<0.05$ was considered indicative of statistical significance.

Results

Infarct Size and Extent of MO

Four to 6 hours after reperfusion, infarct size measured by MRI was 25±10% of LV area (Table 1). At that time, 6±8% of the LV mass was involved by MO. Between 4 to 6 and 48 hours, both infarct size and the extent of MO increased significantly. The increase in the extent of MO was more important (by 89%, from 6±7% to 11±7% LV mass, $P<0.01$) than the increase in infarct size (by 17%, from 25±10% to 29±12% LV mass, $P<0.05$). Hence, the proportion of infarcted tissue with MO increased from 21±17% to 48 hours after reperfusion ($P<0.05$). This relative proportion of infarcted tissue occupied by MO was not significantly related to the infarct size itself ($r=0.28, P=NS$). No significant changes in infarct size or in the proportion of infarcted myocardium with MO, as measured by MRI, occurred between 48 hours and 10 days. Postmortem measurements of TTC-negative area (23±14%) and thioflavine-negative area (10±10%) correlated highly with MRI estimates of infarct size and MO ($r=0.89$ and 0.91, $P<0.001$, respectively).
size (extent of TTC-negative myocardium) and MO extent (thioflavine-negative myocardium) were used in lieu of the in vivo measures by MRI.

Myocardial Strains in Infarcted, Adjacent, and Remote Myocardium

The 3 orthogonal normal strains and the first principal strain, representing maximal thickening, were computed in infarcted and adjacent noninfarcted myocardium and compared with the relative amount of MO in infarcted tissue. To make this parameter independent of infarct size, it was expressed as a ratio of extent of MO (early gadolinium hypoenhancement) to extent of infarcted tissue (late gadolinium hyperenhancement), thus expressing the relative proportion of infarcted tissue occupied by MO.

Animals were separated into 2 groups: group 1, which had infarcts with >35% of their volume occupied by MO (n=8, mean 42±5% MO); and group 2, which had infarcts with <35% volume occupied by MO (n=9, mean 26±9% of MO). A typical strain map from a representative animal of each group is shown in Figure 5.

All 3 orthogonal normal strains and the first principal strain were similar in the subendocardium of remote myocardium in both groups (Figure 6). In the subendocardium of infarcted regions, 48 hours after reperfusion, animals with >35% MO demonstrated significantly less stretching in the longitudinal direction than animals that had infarcts with less MO (+2±5 versus +7±5%, P=0.05). In noninfarcted adjacent regions, a significant reduction of subendocardial radial thickening (9±6% versus 14±3%, P<0.01) existed when the adjacent infarct had >35% MO compared with infarcts with less MO.

First principal strain was found to be significantly reduced in the subendocardium of animals with infarcts that had >35% MO than in animals that had infarcts with less MO (13±4% versus 20±4%, P<0.001). Differences in the magnitude of first principal strain could be documented as early as 4 to 6 hours after reperfusion (12±2% versus 22±7%, P=0.06) in animals with >35% MO. The orientation of the principal strain vector was expressed by 2 angles against the longitudinal and circumferential directions (Table 3). No significant difference in orientation of principal strain existed between the 2 groups of dogs. However, the reduction of the magnitude of first principal strain correlated highly, but inversely (r=−0.80, P<0.001), with the extent of MO 48 hours after reperfusion, as shown in Figure 7. Reduction of first principal strain also correlated (r=−0.64, P<0.01) with the ratio of MO to infarct size by pathology. In addition, the magnitudes of circumferential (r=−0.61, P<0.01) and longitudinal (r=−0.53, P<0.05) stretches were found to correlate inversely with the degree of MO at pathology, indicating that animals with greater amounts of MO had reduced systolic stretching in both directions. No such differences of magnitude or orientation of normal orthogonal or principal strains could be observed in the subepicardium of animals with greater or lesser degrees of MO.

Discussion

This study examined the relationship between MO, myocardial deformation, and remodeling of the LV during the early
healing phase of reperfused MI. Our findings can be summarized as follows. (1) During the early period of reperfusion, the extent of MO predicted LV enlargement after reperfusion independently of infarct size. (2) Increased amounts of MO within infarcted myocardium correlated significantly with altered myocardial strains both in infarcted and in adjacent noninfarcted regions up to 48 hours after reperfusion of MI. The process of LV enlargement after acute MI involves architectural rearrangement both in the infarcted and in the surrounding noninfarcted myocardium over the days after the acute ischemic event. Although the exact mechanisms that govern LV remodeling are still incompletely understood, several recent studies indicate that reperfusion of infarcted myocardium may have beneficial effects on LV remodeling, even at times when no direct benefit in terms of infarct size reduction can be documented. This hypothesis is currently known as the “open-artery hypothesis.” Yet, despite restoration of epicardial blood flow, reperfusion at the tissue level may remain impaired because of MO by neutrophils, erythrocytes, and debris. It was recently shown that MO may negatively influence LV remodeling early and late after reperfusion. It is currently unknown whether the salutary effects of reperfusion on remodeling result through reduction of injury to the microvasculature. However, preliminary clinical observations using contrast-enhanced MRI suggest that nonreperfused infarcts might have more severe microvascular injury than infarcts with open arteries. This could be related to worse LV postinfarct remodeling in patients in whom reperfusion failed or was not attempted.

Methodological Considerations
By design, this study was limited to the early post-MI period (10 days after acute MI), and its findings may not apply to later periods of postinfarct remodeling. Other potential limitations of this article may relate to the use of noninvasive MRI techniques for measurements of infarct size and of extent of MO. These limitations have been discussed in detail elsewhere, and because no differences were found for measurements of infarct size and of MO made in vivo by MRI and for measurements performed in postmortem pathological studies, they most likely have not influenced our results. Our study documents a covariation of myocardial strains with the extent of MO in infarcted tissue and also a covariation of MO with LV remodeling. Although we cannot prove a direct causal relationship between these factors, we demonstrate a temporal relationship between the appearance of MO, the development of altered myocardial strains, and the onset of LV remodeling, as documented by LV cavity enlargement and wall thickening. However, other confounding factors that might have influenced this covariation cannot be excluded.

MO and LV Remodeling
In the present study, we followed increases in LV cavity size early after acute MI, from 4 to 6 hours up to 10 days after

<table>
<thead>
<tr>
<th>Parameter to Enter</th>
<th>Parameter</th>
<th>β</th>
<th>Change in r²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>Extent of MO</td>
<td>1.72</td>
<td>0.71</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Model 2</td>
<td>Extent of infarct</td>
<td>0.05</td>
<td>0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Model 3</td>
<td>Extent of infarct without MO</td>
<td>0.03</td>
<td>0.05</td>
<td>NS</td>
</tr>
<tr>
<td>step 1</td>
<td>Extent of MO</td>
<td>1.72</td>
<td>0.71</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>step 2</td>
<td>Extent of MO</td>
<td>1.68</td>
<td>0.451</td>
<td>&lt;0.001</td>
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</table>

Figure 4. Relationship between EDV and extent of MO (A) and infarct size (B) as measured by MRI at terminal study (48 hours and 10 days after reperfusion). When outlying data point (arrow) was removed, correlation coefficients were r=0.62, P=0.01 between EDV and extent of MO and r=0.55, P<0.05 between EDV and infarct size, respectively.
reperfusion. Increases in LV volume were accompanied by decreases in wall thickness in both infarcted and adjacent regions, indicating that remodeling occurred in both regions. Our data are consistent with previous observations showing that early increases in LV volumes depend on infarct size. We also demonstrated a relationship between LV enlargement and the extent of MO, confirming previous observations by Ito et al and Wu et al. We assessed the effects of infarcted myocardium with and without MO on LV enlargement separately, using multivariate analysis. Our results indicate that infarcted myocardium with MO has greater predictive power in LV enlargement than infarcted tissue with patent microvasculature. To investigate whether this might be due to a direct influence on regional myocardial mechanics, we analyzed myocardial strains in the infarcted and adjacent regions and related them to the presence and extent of MO.

As in earlier work, we documented a reduction in magnitude and redirection of myocardial strains in infarcted myocardium. These alterations in myocardial strain correlated strongly with the degree of MO present in the infarcted segment, yet were not influenced by infarct size itself. In particular, the maximum vector of the first principal strain was found to be significantly reduced when the infarcted tissue had increased amounts of MO 48 hours after reperfusion. Similarly, an inverse linear relationship existed between the magnitude of longitudinal and circumferential stretches in the infarcted myocardium and the
extent of MO as measured by the ratio of thioflavine-

negative myocardium to the total necrotic area. A possible

explanation for the observed reductions in myocardial

stretch would be enhanced stiffness of infarcted tissue with

MO. Such increased stiffness could result from intramyo-

cardial hemorrhage,20 as often observed in infarcted tissue

with microvascular injury. 21,22 It might also result from

greater intramyocardial edema, possibly contributing to

greater MO, or from other unknown mechanisms. There-

fore, our results suggest that myocardial stiffening in

regions with MO may have an adverse effect on LV

geometry and segmental function, ultimately resulting in

increased LV remodeling. Interestingly, this possibility has

been suggested previously by other authors.23

Myocardial strain alterations were also found in noninfarcted

adjacent myocardium, corroborating earlier work.11,24,25 Radial

thickening was found to be reduced in the adjacent region of

infarcts that had increased amounts of MO. Moreover, mechan-

TABLE 3. Angles of First Principal Strain in Infarcted, Adjacent, and Remote
Myocardium in Animals Having High (>35%) or Low (<35%) Amounts of
Infarcted Tissue Occupied by MO

<table>
<thead>
<tr>
<th>Angle, degrees</th>
<th>Infarcted</th>
<th>Adjacent</th>
<th>Remote</th>
</tr>
</thead>
<tbody>
<tr>
<td>αE1C &lt;35% M0</td>
<td>79±15</td>
<td>85±12*</td>
<td>116±20</td>
</tr>
<tr>
<td></td>
<td>&gt;35% M0</td>
<td>119±15</td>
<td>124±20</td>
</tr>
<tr>
<td>αE1L &lt;35% M0</td>
<td>48±13†</td>
<td>74±15</td>
<td>92±8</td>
</tr>
<tr>
<td></td>
<td>&gt;35% M0</td>
<td>89±8</td>
<td>92±7</td>
</tr>
</tbody>
</table>

Angles as in Figure 2.
*P<0.05 vs remote of the same group.
†P<0.05 vs infarcted with >35% M0.
ical alterations in adjacent regions were found to occur later (at 48 hours after reperfusion) than alterations of strains within infarcted regions, which occurred as soon as 4 to 6 hours after reperfusion, and thus, before LV enlargement. A potential mechanism would be increased local wall stress in adjacent regions secondary to the reduced elasticity of infarcted segments with widespread MO. Indeed, in this study, we demonstrated reduced radial thickening in adjacent regions when infarcts have increased degrees of MO. This might result in lengthening of noninfarcted segments, which has been documented to occur early in the LV remodeling process.

Conclusions
This study demonstrates that the extent of MO predicts LV dilatation in the early healing phase of acute MI over and above the effects of infarct size. In addition, our results demonstrate decreased systolic myocardial deformation in regions of MO compared with infarcts with patent microvascular wall injury and greater impairment of function in noninfarcted myocardium adjacent to infarcts containing large regions of microvascular damage. The latter findings on myocardial strain alteration provide important mechanistic insight into the pathophysiological link between microvascular occlusion and LV postinfarct remodeling.

Acknowledgments
This study was supported by Grant-in-Aid 92-10-26-01 from the American Heart Association, Dallas, Tex, and NHLBI grants HL-45090 and P50-HL-52315 (SCOR in Ischemic Heart Disease), NIH, Bethesda, Md.

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Circulation. 2000;101:2734-2741
doi: 10.1161/01.CIR.101.23.2734

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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