Detection and characterization of acute myocardial infarction in man with use of gated magnetic resonance

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ABSTRACT To evaluate the capability of magnetic resonance imaging (MRI) in the detection and characterization of alterations in signal intensity and T2 relaxation time in acutely infarcted relative to normal myocardium 16 adult patients and normal volunteers were studied by electrocardiographically gated proton MRI. The seven volunteers were entirely asymptomatic and had no history of cardiovascular abnormality. The nine patients had each suffered an acute myocardial infarction within 5 to 12 days before the MRI studies. The diagnosis in each patient was confirmed by electrocardiographic (ECG) criteria and elevated levels of fractionated creatine kinase (CK) isoenzymes. Electrocardiographically gated MRI was performed with a superconducting system operating at 0.35 tesla. MRI demonstrated infarcted myocardium as a region of high signal intensity relative to that of adjacent normal myocardium; regions of high intensity corresponded anatomically to the site of infarction as defined by the ECG changes. The mean percent difference between normal and infarcted myocardium was substantially greater on 56 msec images (70.2 ± 21.3%) compared with 28 msec images (27.1 ± 13.6%). Region of interest analysis revealed that infarcted myocardium had a significantly (p < .01) prolonged T2 relaxation time (mean T2 = 80.9 msec) relative to that in normal myocardium (mean T2 = 42.3 msec) and relative to the mean T2 of left ventricular myocardium in the volunteers (mean T2 = 42.4 msec). An additional finding for each patient with myocardial infarction was a high intraluminal flow signal on 56 msec images, but this was also observed in normal subjects and is therefore a nonspecific finding. In humans MRI detects myocardial infarction as a region of high signal intensity relative to that of adjacent normal myocardium and identifies significant prolongation of infarct T2 relaxation time in the damaged region. Quantification of T2 relaxation times provides differential characterization of tissue in normal and infarcted myocardium.


MAGNETIC RESONANCE IMAGING (MRI) has shown significant potential for imaging of the cardiovascular system.¹ A high degree of natural contrast between blood and surrounding vascular and cardiac walls results from the fact that rapidly flowing blood generates little or no magnetic resonance signal and thus no administration of contrast media is required. Additional advantages of proton MRI are the potential for tissue characterization by use of the magnetic relaxation times T1 and T2 and by mobile proton spin density. By gating the image acquisition sequences to the events of the electrocardiographic (ECG) cycle the problems with cardiac motion can be largely overcome and images with excellent resolution of cardiac morphology can be obtained.² MRI has demonstrated utility for the detection and characterization of ischemically damaged myocardium.³⁻⁵ MRI of the heart ex situ has depicted acutely infarcted canine myocardium as a discrete region of high signal intensity relative to the signal from normal myocardium; this was the result of significantly prolonged T2 relaxation time of the infarct.⁶ Subsequently, gated MRI of the intact beating dog heart has characterized acutely infarcted myocardium as a region of high signal intensity within 2 to 7 days after ligation of the left anterior descending coronary artery.⁷ This was again associated with significantly prolonged T2 relaxation time of infarcted myocardium relative to normal myocardium.

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The purposes of this study were to determine whether electrocardiographically gated spin-echo MRI could detect acute myocardial infarction in humans and to characterize the alterations in MRI signal intensity and T2 relaxation time of the infarcted tissue.

Methods

Study population. Sixteen adult patients and normal volunteers were studied by electrocardiographically gated proton MRI. The seven normal volunteers had no history of cardiovascular abnormality and were entirely asymptomatic. The age of the volunteers was 26 to 34 years; five of these subjects were men. The nine study patients had acute myocardial infarctions that had been documented within 5 to 12 days before the MRI studies. The nine patients were 58 to 73 years of age; six were men. The diagnosis in each patient was confirmed by observation of acute ECG abnormalities and by abnormal serum levels of creatine kinase (CK) isoenzymes. The ECG abnormalities and peak levels of CK (total and MB fraction) are listed in table 1. The sites of the infarctions defined by electrocardiography were located in the anterior, anteroseptal, or anterior lateral wall in five patients. Two patients had myocardial infarctions involving the inferior wall according to ECG changes. One patient had a myocardial infarction that involved both the anteroseptal and lateral walls of the left ventricle. ECG localization of acute myocardial infarction was estimated according the standard criteria.8 One additional patient had poorly resolved images because of inadequate gating of magnetic resonance data acquisition as a consequence of atrial fibrillation. Patients with anterior infarctions were intentionally selected since these infarcts can be better evaluated with transaxial images than posterior and inferior infarctions. The major intention of the current study was to define the appearance and magnetic relaxation characteristics of infarcted compared with normal myocardium. There was no attempt in this initial group of patients to define the sensitivity and specificity of MRI for diagnosing acute myocardial infarction or for detecting anterior as opposed to posterior infarctions.

Imaging techniques. Imaging was performed with a superconducting magnet operating at 0.35 tesla with a corresponding hydrogen resonance frequency of 15 MHz. This system has been previously described.8,9 Nonferromagnetic ECG leads were placed on the right and left subclavian and right abdominal regions. A special gating device designed for safe use in high electromagnetic fields and rapidly changing switched-field gradients was used to obtain the gating signal.2 The gating signal was initiated by the R wave and a suppression circuit was used to prevent activation of the radiofrequency and gradient pulse sequence for a period of 500 msec after inscription of the R wave.

Spin-echo MRI was performed with an echo delay (TE) of either 28 or 56 msec between the application of the initial radiofrequency pulse and the receipt of the corresponding signal (spin echo). The pulse sequence repetition time (TR) was determined by each subject’s heart rate and thus varied from about 0.6 to 1.0 sec (60 to 100 beats/min). Images were acquired with a multisection technique whereby five adjacent 7 mm thick sections were obtained with a delay of 100 msec between each adjacent section. Thus, each subsequent section was 100 msec out of phase with the previous section. The radiofrequency and gradient magnetic pulse sequences were applied to 512 cardiac cycles to obtain images. The time required to obtain five transaxial images that encompassed most of the left ventricle was determined by each individual’s heart rate and varied from approximately 5.5 to 8.5 min.

The MRI spin-echo signal was determined from the following equation:

$$I = N(H)(f(v) \exp (-TE/T2) [1 - \exp - (TR/T1)])$$

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>ECG abnormalities</th>
<th>Peak CK</th>
<th>Clinical diagnosis</th>
<th>Diagnosis by MRI</th>
<th>Interval between infarction and MRI (days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>$V_{1,-2}$, $V_{1,-4}$</td>
<td>70°C 10%</td>
<td>Anteroseptal AMI</td>
<td>Anteroseptal AMI</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>$V_{1,-4}$, $V_{2,-6}$</td>
<td>2055 25%</td>
<td>Anterolateral AMI</td>
<td>Anteroseptal AMI</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>$V_{1,-6}$, $V_{1,-4}$</td>
<td>1810 23%</td>
<td>Anteroseptal AMI</td>
<td>Anteroseptal AMI</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>II: III; aVF (small and non-diagnostic)</td>
<td>1140 27%</td>
<td>Inferior AMI infarct of uncertain age; acute ischemic changes in anterior lateral wall</td>
<td>Inferior AMI</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>II: III; aVF</td>
<td>1545 31%</td>
<td>Inferior AMI</td>
<td>Inferolateral AMI</td>
<td>7</td>
</tr>
<tr>
<td>6</td>
<td>$V_{2,-4}$, $V_{2,-5}$</td>
<td>445 6%</td>
<td>Anterolateral AMI</td>
<td>Anterolateral AMI</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>II: III; aVF, $V_{1,-4}$</td>
<td>352 26%</td>
<td>Inferior AMI</td>
<td>Inferolateral AMI</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>II: III; aVF</td>
<td>3145 28%</td>
<td>Anterolateral AMI (remote inferior MI)</td>
<td>Anterolateral AMI</td>
<td>10</td>
</tr>
</tbody>
</table>

aNormal $= 2$ to 83 units.
bNormal $= less than 4%$. cCK measurements obtained 3 days after onset of symptoms.
where \( I = \) MRI signal intensity; \( N(H) = \) mobile hydrogen spin density; \( f(v) = \) function of both the speed with which hydrogen nuclei move through the imaged region and of the fraction of the moving nuclei; \( TE = \) time to reception of spin-echo signal (28 or 56 msec after 90 degree radiofrequency pulse); \( T2 = \) spin-spin or transverse relaxation time; \( TR = \) radiofrequency pulse sequence repetition time; \( TI = \) spin-lattice or longitudinal relaxation time. 3 From this equation it is apparent that magnetic resonance signal intensity increases with shortening of the T1 relaxation time, with lengthening of the T2 relaxation time, or with an increase in hydrogen spin density.

Data analysis. For each series of magnetic resonance images in each patient with acute infarction the spin-echo signal intensity values (in arbitrary units of magnitude) were calculated by region of interest after visual inspection of the images. The regions of interest contained at least 50 voxels for infarcted myocardium and for normal myocardium. Technically satisfactory first spin echoes (28 msec) and second spin echoes (56 msec) were obtained for 15 subjects. One patient with myocardial infarction had technically poor images that were not analyzed for this study. Regions of interest for the normal volunteers were in the anterior and posterolateral portions of the left ventricle for all subjects. A region of interest was constructed in the anterior, anterolateral, anteroseptal, or inferior portion of the left ventricle, depending on the site of increased signal intensity, for the eight patients with myocardial infarction. These measurements were obtained on one occasion by two observers and at one additional time by one observer after an 8 week interval to determine interobserver and intraobserver variability. A region of interest for normal myocardium was constructed in the wall opposite the infarct in each patient. A T2 relaxation time value was calculated from the two Fourier-transformed spin-echo intensities (I₁ and I₂) at echo delay times of 28 msec (TE₁) and 56 msec (TE₂) with the use of the following formula:

\[
T2 = \frac{TE_2 - TE_1}{\ln I_1/I_2}
\]

Since T2 measurements were not obtained from calculated T2 images there were no pixel standard deviations. The T1 relaxation time could not be calculated because imaging at two different TRs is required. In this study images were gated to every heartbeat only.

The percent difference in signal intensity (I) of normal and infarcted myocardium was calculated as follows:

\[
\% \text{ Difference} = \frac{I \text{ (infarct)} - I \text{ (normal)} \times 100\%}{I \text{ (normal)}}
\]

The percent difference in signal intensity (I) of anterior and posterior regions of interest in the normal volunteers was calculated as follows:

\[
\% \text{ Difference} = \frac{I \text{ (anterior)} - I \text{ (posterior)} \times 100\%}{I \text{ (posterior)}}
\]

Statistical analysis. All data (MRI signal intensity and T2) are expressed as mean ± SD. The significance of differences in signal intensity and T2 in normal and infarcted myocardium was calculated with Student's two-tailed t test. A p value less than .05 was considered indicative of statistical significance and a p value greater than or equal to .05 was considered to indicate a nonsignificant finding.

Results

Magnetic resonance images. The gated magnetic resonance images through the left ventricle of the normal volunteers demonstrated good resolution of the cardiac structures, including the myocardium. In each volunteer the myocardium was characterized by homogenous signal intensity throughout the myocardial tissue of the septum and free wall of the left ventricle. No focal regions of high signal intensity in the myocardium were observed in these subjects on either the first echo (TE = 28 msec) or second echo (TE = 56 msec) images (figure 1). The myocardium was uniformly characterized by visibly lower signal intensity on the second echo images relative to the first for all of the volunteers.

The gated images at the level of the left ventricle from the eight patients with acute myocardial infarction displayed focal regions of high signal intensity in the anteroseptal and lateral walls (one patient) anteroseptal wall (three patients), anterolateral wall (two patients), or inferolateral wall (two patients). In each case the region of high signal intensity corresponded to the location of myocardial infarction as determined by observation of ECG abnormalities.

The region of high signal intensity was visible on both first echo and second echo images, but in general was more intense relative to that of adjacent myocardium on the second echo images (figures 2 and 3). In one

FIGURE 1. Cross-sectional electrocardiographically gated magnetic resonance images of the heart of a normal human volunteer. The images shown are 28 msec (top) and 56 msec (bottom) spin-echo images. Natural contrast from rapidly flowing blood results in excellent delineation of the myocardial walls, with absence of flow signal on both first and second spin-echo images. Note the homogeneity of myocardial signal intensity.
patient the high intensity region occupied the subendocardial half of the myocardial wall and was in the shape of a rim that circumscribed nearly the entire apical region of the left ventricle (figure 2).

**Myocardial intensity and T2 relaxation times.** The signal intensity values of the first and second spin-echo images for the normal volunteers (anterior and posterolateral myocardium of the left ventricle) and for the eight patients with infarction (infarcted myocardium and normal myocardium) are shown in tables 2 and 3. There were no statistically significant differences ($p > .05$) in the mean intensity values for myocardial regions in the anterior and posterolateral wall of the left ventricle for either the first or second spin-echo images from the normal volunteers. The percent differences in signal intensity of the two regions was greater on the first echo image ($16.3 \pm 13.4\%$ vs $9.1 \pm 15.6\%$), although this difference did not attain statistical significance. The intensity values for all regions of interest were lower on 56 msec than on 28 msec images.

The intensity values for the region of myocardial infarction were significantly greater ($p < .01$) than the values measured in regions of normal myocardium on both the 28 msec and 56 msec images. The mean percent difference in intensity between the two regions of interest was greater on second echo images (mean = $70.2 \pm 21.3\%$) compared with first echo images (mean = $27.1 \pm 13.6\%$).

The interobserver and intraobserver variabilities for the measurements of the signal intensity of normal myocardium were, respectively, 3% and 7% for both the 28 and 56 msec images. The variabilities for the

**FIGURE 2.** Electrocardiographically gated magnetic resonance images through the mid-left ventricle (top) and through a more apical section (bottom) in patient 1 (table 1). The 28 msec images are on the left, the 56 msec images are on the right. Note the subendocardial region of high signal intensity that displays greater contrast from normal myocardium on the 56 msec images. Region of interest analysis revealed that the inner layer of this signal represented stasis of blood while the remainder represented infarcted myocardium.
measurements in infarcted myocardium were 3% and 4% for 28 msec images and 4% and 5% for 56 msec images.

The T2 relaxation times for the regions of interest within the myocardium of the left ventricle of the normal volunteers and the patients with infarction are listed in tables 2 and 3. There was no significant difference in T2 relaxation times for the two regions of interest in the normal volunteers. The T2 of the infarcted myocardium (mean = 78.7 ± 37.3 msec) was significantly prolonged (p < .01) compared with the T2 of normal myocardium (mean = 38.3 ± 9.8 msec) in the patients and T2s of the anterior (mean = 39.1 ± 2.3 msec) and posterolateral (mean = 43.1 ± 4.3 msec) walls of the normal volunteers.

The interobserver and intraobserver variabilities for the measurements of T2 of normal myocardium were 5% and 10%, respectively, and the variabilities for the measurements of the T2 of infarcted myocardium were 15% and 14%, respectively.

Intracavitary flow signal was observed in five of the seven normal volunteers, occupying the central portion of the left ventricle at the apical level in each case. Flow signal within the lumen of the left ventricle was noted in each patient with infarction as a region of high signal intensity adjacent to the myocardial walls. The site of flow signal was adjacent to the site of infarct in six of the patients; in three the site of flow signal was restricted to the site of the infarct only and in the other three the flow signal involved a majority of the lumen. Two of the patients had a flow signal adjacent to a portion of the wall of the left ventricle that was distant

**FIGURE 3.** Electrocardiographically gated magnetic resonance images through two adjacent sections of the left ventricle of patient 6 (table 1). The 28 msec images are on the left. Note the high signal intensity of the anterolateral wall of the left ventricle (arrowheads), which represents infarcted myocardium. The relative intensity of the infarct is greater on the 56 msec images (right). Note also the abnormal flow signal (curved arrows), which shows relatively greater intensity on 56 msec images and has a negative T2 value compared with the positive value for infarcted myocardium.
TABLE 2
MRI signal intensity values\(^a\) and T2 relaxation times in myocardium of normal volunteers

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Anterior LV</th>
<th>Posterior LV</th>
<th>% Difference(^b)</th>
<th>Anterior LV</th>
<th>Posterior LV</th>
<th>% Difference</th>
<th>T2 (msec)</th>
<th>Postero-lateral LV</th>
<th>Intraluminal flow signal (56 msec image)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intensity (28 msec)</td>
<td>Intensity (56 msec)</td>
<td>T2 (msec)</td>
<td>Intensity (56 msec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3012 ± 295</td>
<td>2546 ± 357</td>
<td>18.3</td>
<td>1437 ± 286</td>
<td>1402 ± 370</td>
<td>2.4</td>
<td>38</td>
<td>47</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>4657 ± 541</td>
<td>4690 ± 451</td>
<td>-0.7</td>
<td>1266 ± 547</td>
<td>2617 ± 478</td>
<td>-9.6</td>
<td>41</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2486 ± 348</td>
<td>2051 ± 320</td>
<td>21.2</td>
<td>1217 ± 347</td>
<td>1134 ± 344</td>
<td>7.3</td>
<td>39</td>
<td>47</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>2911 ± 222</td>
<td>2304 ± 360</td>
<td>26.3</td>
<td>1487 ± 221</td>
<td>1166 ± 221</td>
<td>27.5</td>
<td>42</td>
<td>41</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>2178 ± 439</td>
<td>1803 ± 299</td>
<td>20.8</td>
<td>106 ± 295</td>
<td>940 ± 293</td>
<td>12.8</td>
<td>39</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>2428 ± 346</td>
<td>2522 ± 474</td>
<td>-3.7</td>
<td>1196 ± 314</td>
<td>1283 ± 280</td>
<td>-6.8</td>
<td>40</td>
<td>41</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>4895 ± 436</td>
<td>3718 ± 371</td>
<td>31.7</td>
<td>2184 ± 358</td>
<td>1675 ± 332</td>
<td>30.4</td>
<td>35</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>3223.9</td>
<td>2804.9</td>
<td>16.3</td>
<td>1563.9</td>
<td>1459.6</td>
<td>9.1</td>
<td>39.1</td>
<td>43.1</td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>1100.0</td>
<td>1029.4</td>
<td>13.4</td>
<td>509.8</td>
<td>560.6</td>
<td>15.6</td>
<td>2.3</td>
<td>4.3</td>
<td></td>
</tr>
</tbody>
</table>

LV = left ventricle.

\(^a\)All intensity values are expressed as intensity ± SD and represent arbitrary units.

\(^b\)Defined as posterior intensity minus anterior intensity divided by posterior intensity × 100%.

from the site of myocardial infarction. Measurement of the T2 value for the intracavitary signal provided a negative value in each instance. This negative T2 value enabled clear distinction of the intracavitary flow signal from the high intensity region in the myocardium.

**Discussion**

Previous investigations in the heart ex situ\(^c\) and studies in which ECG gating was used in the beating heart\(^d\) have shown that acutely infarcted myocardium is displayed as a region of high intensity relative to normal tissue on magnetic resonance images and that ischemically damaged myocardium has a significantly prolonged T2 relaxation time relative to normal myocardium.\(^6\)\(^7\) It has also been demonstrated, both in excised dog hearts\(^4\)\(^6\) and in the beating heart (with gated magnetic resonance),\(^1\) that the T1 relaxation time of acutely infarcted canine myocardium is prolonged. The findings of prolongation of T1 and T2 in acutely infarcted myocardium are probably related to local changes in tissue water resulting from myocardial edema, since investigators have shown a significant increase in the percent water content of infarcted compared with normal myocardium.\(^6\)\(^7\) Also, a close linear relationship between T2 relaxation time and percent water content of the infarcted myocardium has been demonstrated.\(^6\)

This study shows that spin-echo electrocardiographically gated MRI can detect acute myocardial infarction in humans as regions of high signal intensity compared with that of the adjacent normal myocardium. This phenomenon was noted in all eight of the patients.

**TABLE 3**

MRI signal intensity values\(^a\) and T2 relaxation times from normal and infarcted myocardium of patients

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Normal MI Intensity (28 msec)</th>
<th>Normal MI Intensity (56 msec)</th>
<th>T2 (msec)</th>
<th>FS (56 msec image)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% Difference(^a)</td>
<td>Intensity (28 msec)</td>
<td>% Difference(^a)</td>
<td>Intensity (56 msec)</td>
</tr>
<tr>
<td>1</td>
<td>2440 ± 340</td>
<td>2773 ± 535</td>
<td>13.6</td>
<td>1191 ± 357</td>
</tr>
<tr>
<td>2</td>
<td>2047 ± 501</td>
<td>3057 ± 239</td>
<td>49.3</td>
<td>1240 ± 368</td>
</tr>
<tr>
<td>3</td>
<td>2889 ± 262</td>
<td>3781 ± 445</td>
<td>30.9</td>
<td>1327 ± 510</td>
</tr>
<tr>
<td>4</td>
<td>2835 ± 334</td>
<td>3873 ± 376</td>
<td>36.6</td>
<td>1153 ± 320</td>
</tr>
<tr>
<td>5</td>
<td>1967 ± 298</td>
<td>2330 ± 468</td>
<td>18.5</td>
<td>709 ± 271</td>
</tr>
<tr>
<td>6</td>
<td>2176 ± 186</td>
<td>2332 ± 347</td>
<td>7.2</td>
<td>1187 ± 126</td>
</tr>
<tr>
<td>7</td>
<td>2165 ± 329</td>
<td>2908 ± 299</td>
<td>34.3</td>
<td>1172 ± 184</td>
</tr>
<tr>
<td>8</td>
<td>2474 ± 456</td>
<td>3183 ± 432</td>
<td>26.7</td>
<td>1241 ± 243</td>
</tr>
<tr>
<td>Mean</td>
<td>2347.1</td>
<td>3023.5</td>
<td>27.1</td>
<td>1202.5</td>
</tr>
<tr>
<td>SD</td>
<td>348.0</td>
<td>578.7</td>
<td>13.6</td>
<td>234.9</td>
</tr>
</tbody>
</table>

\(^a\)MI = region of myocardial infarct; normal = opposite wall of left ventricle relative to region of MI; FS = intraluminal flow signal.

\(^b\)Defined as MI intensity minus normal myocardial intensity divided by normal intensity × 100%.
studied. In each of the eight patients there was also significant prolongation of T2 in infarcted myocardium relative to normal myocardium as determined by region of interest analysis. Since water content increases in areas of myocardial infarct, it is probable that the findings of T2 prolongation result from this increase. Such a change in water content does not specifically alter T2, however. As shown in previous studies there is an accompanying increase in T1 that results from the increase in water content. Considering that the increased signal intensity on spin-echo magnetic resonance images is caused by a decrease in T1 and prolongation of T2, it is apparent that prolongation of T1 and T2 make opposing contributions to signal intensity. It is most likely that the contribution of T2 prolongation predominates over the T1 effect when the spin-echo technique is used and thus it is the former that produces the overall relative increase in signal intensity. An additional factor to be considered in the spin-echo equation is N(H), hydrogen spin density. An increase in water content resulting from edema should also increase the local spin density, which would also contribute to a local increase in MRI signal intensity within the region of infarct.

Although the mean age of the patients with infarction in the study was significantly greater than that of the volunteers, the mean T2 times for normal myocardium in the two groups were quite similar (42.1 ± 8.4 vs 41.1 ± 4.1 msec). The purpose of this investigation, however, was not to compare the age-related range of myocardial T2 times but instead to compare the relative alterations of T2 and MRI signal intensity due to acute myocardial infarction.

Although the absolute values for the infarcted regions of interest were greater on first spin-echo images, there was a marked increase in contrast (greater percent difference in signal intensity between infarcted and normal myocardium) on second echo images. This was seen in all of the patients. The signal intensity of the normal myocardium decreased markedly on second echo images but remained high in the infarcted myocardium because of the prolonged T2 relaxation time in the area of infarct.

There is a potential pitfall in the diagnosis of infarcted subendocardial regions of the myocardial wall by MRI. A prior report and our continuing experience has shown that high signal intensity can be caused by stasis of blood adjacent to akinetic and dyskinetic regions of myocardium on the second spin-echo images. This phenomenon can be definitely discriminated from that which occurs in acutely infarcted myocardium since measurement of T2 time in a high intensity region of adjacent blood results in a negative value of T2 when the spin-echo formula described above is used. Only even echo rephasing of slowly moving blood results in a negative T2 value; measurement of tissue will not produce a negative value. Thus, although stasis of blood flow was noted on the 56 msec images from all eight patients with infarction, region of interest analysis enabled the differentiation between flow signal and adjacent infarcted myocardium by recognition of the prolonged positive T2 value for flow signal (figures 2 and 3). Although this signal suggests stasis as a consequence of regional contraction abnormalities in patients with infarction, this finding is not specific for these patients. Its presence in normal subjects is likely indicative of stasis of blood in the left ventricle during some portion of the cardiac cycle. Although a flow signal adjacent to the myocardium was not observed in normal volunteers but was seen in some of the patients, it nonetheless appears that it is quite nonspecific for the assessment of myocardial function.

We conclude that acute myocardial infarction can be detected in humans as a focal region of high signal intensity on electrocardiographically gated spin-echo magnetic resonance images. Discrimination between normal and infarcted myocardium is sufficient to estimate location of infarct. Analysis of regional relaxation times reveals significant prolongation of T2 in infarcted myocardium relative to that in normal myocardium.

References

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Detection and characterization of acute myocardial infarction in man with use of gated magnetic resonance.
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