Imaging Time After Gd-DTPA Injection Is Critical in Using Delayed Enhancement to Determine Infarct Size Accurately With Magnetic Resonance Imaging

To the Editor:

In a recent report, Oshinski et al1 concluded that "accurate determination of infarct size by delayed enhancement MRI requires imaging at specific times after gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) injection." This conclusion was based on the observation that the size of hyperenhanced regions changed in images acquired at different times after contrast administration. This conclusion, if accurate, would have important implications for the practical use of this technique in the clinical arena.

The changes in size of the hyperenhanced regions observed by Oshinski et al,1 however, were likely caused by an incorrect implementation of the MRI technique. As we have described previously, the MRI technique requires that the correct inversion time be chosen in order to "null" normal myocardium. If the inversion time is too short, the periphery of the hyperenhanced region will pass through a zero-crossing, thereby affecting its apparent size (see Figure 2 of Simonetti et al).2 Oshinski et al acquired images during the first 40 minutes. Weinmann et al,3 who developed Gd-DTPA, showed that the blood concentration of Gd-DTPA falls from 2.06 to 0.77 mmol/L (0.25 mmol/kg dose, 40 minutes value interpolated from data at 28 and 58 minutes) from 1 to 40 minutes after injection. This 2.7-fold decrease in Gd-DTPA concentrations would require adjusting the inversion time from 195 to 330 ms (eg, Δ1/T1= [2.06 mmol/L][4.5/sec/mmol/L][0.25[extracellular space]]=2.32/sec; T1=1/[2.32+1/0.8[precontrast T1]]=280 ms; inversion time to null normal myocardium= [280][ln2]=195 ms), yet Oshinski et al held the inversion time constant (225 ms). Given that the MRI technique was applied incorrectly, it is difficult to compare the MRI results to infarct size.

We have employed the MRI technique for delayed enhancement in approximately 1000 individuals, and in many cases, images of the same location were acquired repeatedly over time. We have not observed changes in the size of hyperenhanced regions over time when the MRI technique is correctly applied. We agree with Oshinski et al,1 however, that systematic study of this issue is of interest.

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Response

We appreciate the interest of Drs Judd and Kim in our MRI study showing that soon after myocardial infarction (MI), the size of the delayed enhancement zone decreases as a function of time after the injection of a contrast agent.1 We found that 2 days after MI, there exists an area at the periphery of the infarct that has a highly transient T1 value. Within the first 5 to 10 minutes after gadolinium-diethylenetriamine pentaacetic acid (Gd-DTPA) injection, the T1 of this region was similar to infarct, but 20 minutes after contrast injection, its T1 value was similar to normal myocardium. Several studies have now shown that this peripheral area corresponds to reversibly injured myocardium.2–4

We certainly acknowledge that adjusting the inversion time can improve both image contrast and infarct detection sensitivity. The inversion time adjustments recommended by Drs Judd and Kim explain how it may be possible to underestimate infarct size if the inversion time is not set to null normal myocardium, but it does not explain how it might be possible to overestimate infarct size. Changing MRI parameters does not change the fact that this peripheral area has different washout kinetics than either normal or infarcted myocardium, and thus represents a different pathological state. Recent work in acute MI confirms that as much as half of the enhancement area washes out between 4 and 20 minutes after contrast injection. Furthermore, myocardial segments with contrast washout after 20 minutes demonstrate greater functional recovery than those with persistent enhancement.4

As noted by Drs Judd and Kim, the selection of inversion time to null normal myocardium after contrast injection is an important variable when using delayed enhancement to define the extent of MI. Currently, there is no reported standardized method to determine the "correct" inversion time for nulling normal myocardium after contrast injection. The required inversion time can vary widely between patients depending on cardiac output, renal function, and the condition of the myocardial macro- and microvasculature. Techniques that rely less on determining the precise null point show promise in making delayed enhancement MRI more robust over a wide range of clinical scenarios.5

Delayed enhancement MRI is a technique that will have a significant clinical impact, particularly for viability determination in chronic ischemic heart disease. Given its potential to differentiate between infarcted, stunned, and normal myocardium, delayed enhancement MRI will undoubtedly also prove useful in the assessment of acute myocardial infarction.

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_Circulation_. 2002;106:e6
doi: 10.1161/01.CIR.000019903.37922.9C
_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

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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