



HOW I DO...#

## How We Perform Delayed Enhancement Imaging

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### ABSTRACT

Recently, numerous studies have demonstrated the effectiveness of a segmented inversion recovery fast gradient echo (seg IR-FGE) sequence for differentiating injured from normal myocardium. This technique for delayed enhancement imaging has been shown to be effective in identifying the presence and extent of myocardial infarction, as well as predicting improvement in contractile function after coronary revascularization. In this article we outline the procedure of delayed enhancement imaging performed at our center, describe the seg IR-FGE sequence in more detail, including our process for choosing sequence settings, review our process of image interpretation, and highlight potential pitfalls (and techniques to overcome them) that we have encountered in our experience with performing the technique in over 1500 patients.

*Key Words:* Myocardial infarction; Viability; Contrast enhancement; Inversion recovery.

### INTRODUCTION

Reports of imaging myocardial injury using T1-weighted pulse sequences after administration of intravenous gadolinium have been in the literature since the mid 1980s (Eichstaedt et al., 1986; McNamara et al., 1986; Rehr et al., 1986). A major limitation of the initial techniques was insufficient image contrast between normal and injured myocardium. Recently, a number of studies have demonstrated the effectiveness of a segmented inversion recovery fast gradient echo (seg IR-FGE) sequence for differentiating injured from normal

myocardium with signal intensity differences of nearly 500% (Kim et al., 1999; Simonetti et al., 2001). This technique for delayed enhancement imaging has been shown to be effective, in both animals and humans, in identifying the presence, location, and extent of acute and chronic myocardial infarction (Barkhausen et al., 2002; Fieno et al., 2000; Kim et al., 1999; Simonetti et al., 2001; Wu et al., 2001). Additionally, this technique provides scar size measurements that are closely correlated with positron emission tomography (PET) in patients with ischemic cardiomyopathy (Klein et al., 2002), provides results superior to single photon emission computed

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tomography (SPECT) in patients with small myocardial infarctions (Wagner et al., 2003), and can be used to predict improvement in contractile function after revascularization in patients with acute or chronic coronary artery disease (Choi et al., 2001; Kim et al., 2000). Furthermore, there is evidence that this technique may be useful in assessing myocardial injury in patients with nonischemic heart disease. For example, recent reports indicate that hyperenhanced myocardial regions can be visualized in patients with hypertrophic cardiomyopathy (Choudhury et al., 2002; Moon et al., 2003). Whereas the pattern of hyperenhancement in hypertrophic cardiomyopathy is distinct from that seen in ischemic heart disease, the clinical and prognostic significance of hyperenhancement in these patients is currently unknown.

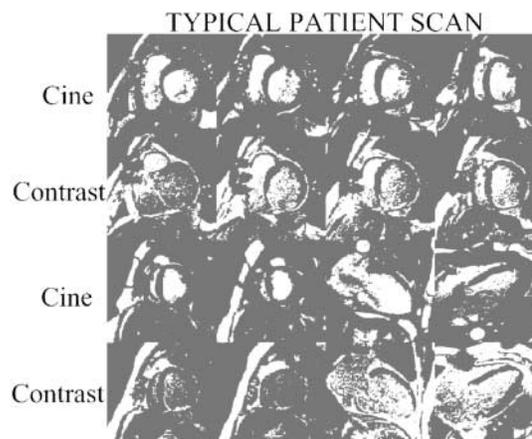
The focus of this article will be to outline the procedure of delayed enhancement imaging performed at our center. We will describe the seg IR-FGE sequence in more detail, the timing parameters that are involved, our process for selecting sequence settings, our process of image interpretation, and highlight potential pitfalls (and techniques to overcome them) that we have encountered in our experience with performing the technique in over 1500 patients.

## OVERALL PROCEDURE

The procedure for delayed enhancement imaging is relatively simple (see Table 1). It can be performed in a single brief examination, requires only a peripheral intravenous catheter which is placed before the patient enters the magnetic resonance imaging (MRI) scanner, and does not require pharmacological or physiological stress. After obtaining scout images to delineate the short- and long-axis views of the heart, we obtain cine images to provide a matched assessment of left ventricular (LV) morphology and contractile function, and to aid in the detection of small subendocardial infarcts (further described below). Short-axis views (6 mm slice thickness

**Table 1.** Procedure events.

- |                                      |
|--------------------------------------|
| 1. Insert peripheral IV              |
| 2. Place patient in scanner          |
| 3. Obtain scout images               |
| 4. Obtain cine images                |
| 5. Inject gadolinium                 |
| 6. Wait 10–15 minutes                |
| 7. Obtain delayed enhancement images |

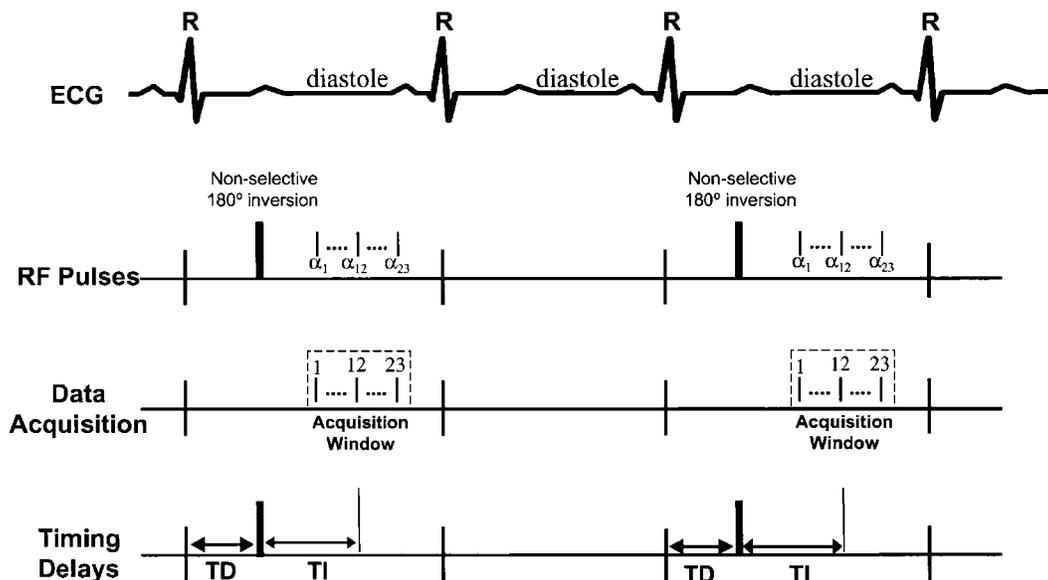


**Figure 1.** Images from a typical patient scan. Cine and contrast-enhanced images are acquired at six to eight short and two to three long-axis locations during repeated breath-holds. Images are interpreted with cine images immediately adjacent to contrast images. This particular patient had a myocardial infarction caused by occlusion of the right coronary artery. Note hyperenhancement of the inferior wall.

with 4 mm gap to match contrast-enhancement images) are taken every 10 mm from mitral valve insertion to LV apex along with two to three long-axis views in order to encompass the entire left ventricle. The patient is then given a bolus of 0.10–0.15 mmol/kg intravenous gadolinium by hand injection. After a 10–15 minute delay (timing issues further discussed below), high spatial resolution delayed-enhancement images of the heart are obtained at the same spatial locations as the cine images using the seg IR-FGE pulse sequence. Figure 1 demonstrates cine and contrast-enhanced images from a typical patient scan. Each delayed-enhancement image is acquired during an 8–10 sec breath hold, and the imaging time for the entire examination is generally 30–40 minutes.

## PULSE SEQUENCE TIMING

The timing diagram for the seg IR-FGE pulse sequence is shown in Fig. 2. Immediately after the onset of the R wave trigger, there is a delay or wait period (which we will refer to as the trigger delay or TD) before a nonselective 180° hyperbolic secant adiabatic inversion pulse is applied. Following this inversion pulse, a second variable wait period (which we will refer to as the inversion time or TI), occurs corresponding to the time between the inversion pulse and the center of the data acquisition window (for linearly ordered k-space acquisition). The data acquisition window is generally



**Figure 2.** Timing diagram of two-dimensional segmented inversion-recovery fast gradient echo pulse sequence. ECG = electrocardiogram. RF = radio-frequency, TD = trigger delay, TI = inversion time delay,  $\alpha$  = shallow flip angle excitation. Note that in this implementation 23 lines of k-space are acquired every other heartbeat. See text for further details.

140–200 msec long, depending on the patient heart rate, and is placed during mid-diastole when the heart is relatively motionless. A group of k-space lines are acquired during this acquisition window, where the flip angle used for radio-frequency excitation for each k-space line is shallow ( $20^\circ$ – $30^\circ$ ) to retain regional differences in magnetization that result from the inversion pulse and TI delay. The number of k-space lines in the group is limited by the repetition time between each k-space line ( $\sim 8$  ms) and the duration of mid-diastole. In the implementation shown on Fig. 2, 23 lines of k-space are acquired during each data acquisition window, which occurs every other heartbeat. With this implementation, typically a breath-hold duration of 12 cardiac cycles is required to obtain all the k-space lines for the image matrix.

### IMAGING PARAMETERS

The typical settings that we use for the seg IR-FGE sequence are shown in Table 2. The dose of gadolinium given is usually 0.10–0.15 mmol/kg. Previously we have given doses as high as 0.3 mmol/kg in animals (Kim et al., 1999; Simonetti et al., 2001) and 0.2 mmol/kg in patients (Kim et al., 2000; Simonetti et al., 2001). Although the original preclinical validation studies were performed at higher doses, more recently we have found that using 0.10–0.15 mmol/kg still provides excellent image

**Table 2.** Typical parameters.

Parameter	Typical values
Gadolinium dose	0.10–0.15 mmol/kg
Field of view	300–380 mm
In-plane voxel size	1.2–1.8 × 1.2–1.8 mm
Slice thickness	6 mm <sup>a</sup>
Flip angle	20–30°
Segments <sup>b</sup>	13–31
TI (inversion time)	Variable
Bandwidth	90–250 Hz/pixel
TE (echo time)	3–4 msec
TR (repetition time) <sup>c</sup>	8–9 msec
Gating factor <sup>d</sup>	2
K-space ordering	Linear
Fat saturation	No
Asymmetric echo	Yes
Gradient moment refocusing <sup>e</sup>	Yes

<sup>a</sup> Short-axis slices acquired every 10 mm, to achieve identical positions as the cine images.

<sup>b</sup> Fewer segments are used for higher heart rates. See text for details.

<sup>c</sup> TR is generally defined as the time between RF pulses; however, scanner manufacturers occasionally redefine the TR to represent the time from the ECG trigger (R wave) to the center or end of the data acquisition window (i.e.,  $\sim 100$  msec less than the ECG R–R interval).

<sup>d</sup> Image every other heartbeat. See text for details.

<sup>e</sup> Also known as gradient-moment nulling, gradient-moment rephrasing, or flow compensation. See text for details.

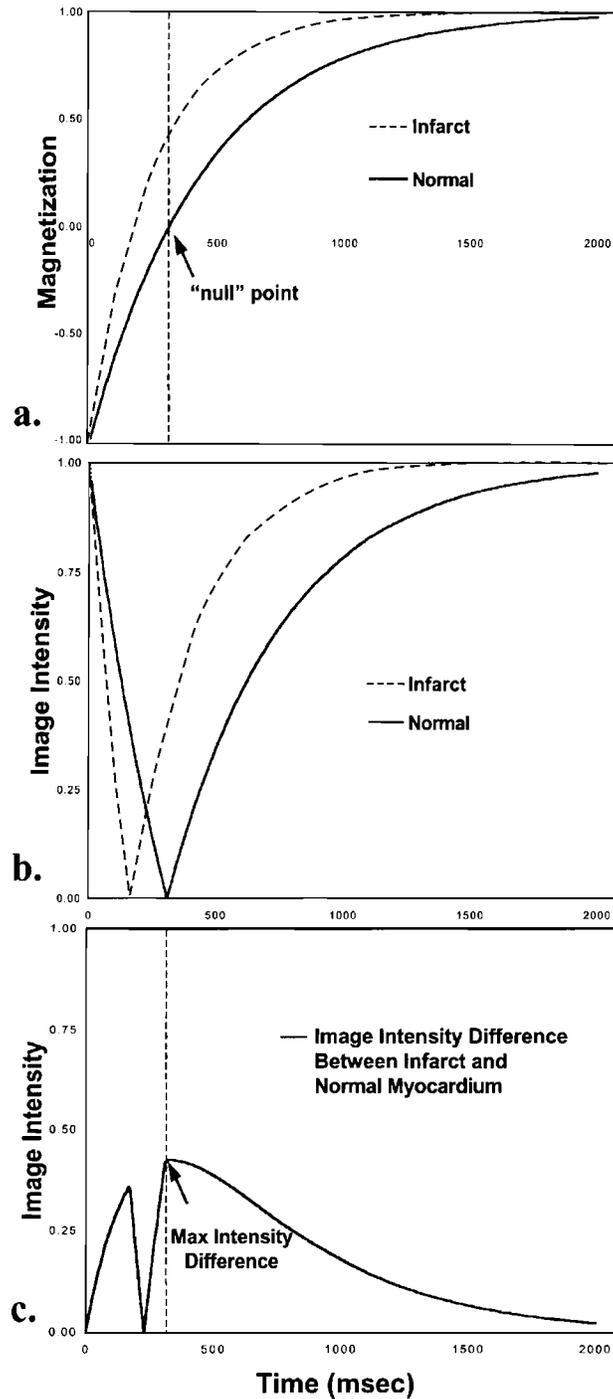
contrast between injured and normal myocardium and reduces the time required to wait between intravenous contrast administration and delayed-enhancement imaging (Choi et al., 2001; Ricciardi et al., 2001; Wagner et al., 2003; Wu et al., 2001). Sufficient time is required in order to allow the blood pool signal in the LV cavity to decline and provide discernment between LV cavity and hyperenhanced myocardium (see *Pitfalls* below). The field-of-view (FOV) in both read and phase-encode directions is minimized to improve spatial resolution without resulting in wrap-around artifact in the area of interest. For patients with heart rates less than 90 beats per minute we typically acquire 23 lines of k-space data during the mid-diastolic portion of the cardiac cycle. For a repetition time of 8 msec, the data acquisition window is 184 msec in duration ( $8 \times 23 = 184$ ). Since the mid-diastolic period of relative cardiac standstill is reduced in patients with faster heart rates, we decrease the number of segments (k-space lines) acquired per cardiac cycle in order to reduce the length of the imaging window. This eliminates blurring from cardiac motion during the k-space collection. In order to allow for adequate longitudinal relaxation between successive  $180^\circ$  inversion pulses, inversion pulses are applied every other heartbeat (Gating Factor of 2). In our experience an in-plane resolution of 1.2 to 1.8 mm by 1.2 to 1.8 mm with a slice thickness of 6 mm provides an adequate balance between signal-to-noise while avoiding significant partial volume effects. As stated previously, the flip angle is kept shallow to retain the effects of the inversion prepulse, but it can be relatively greater ( $30^\circ$ ) if larger doses of gadolinium are given (0.2 mmol/kg) and the TI of myocardium is correspondingly shorter.

### INVERSION TIME

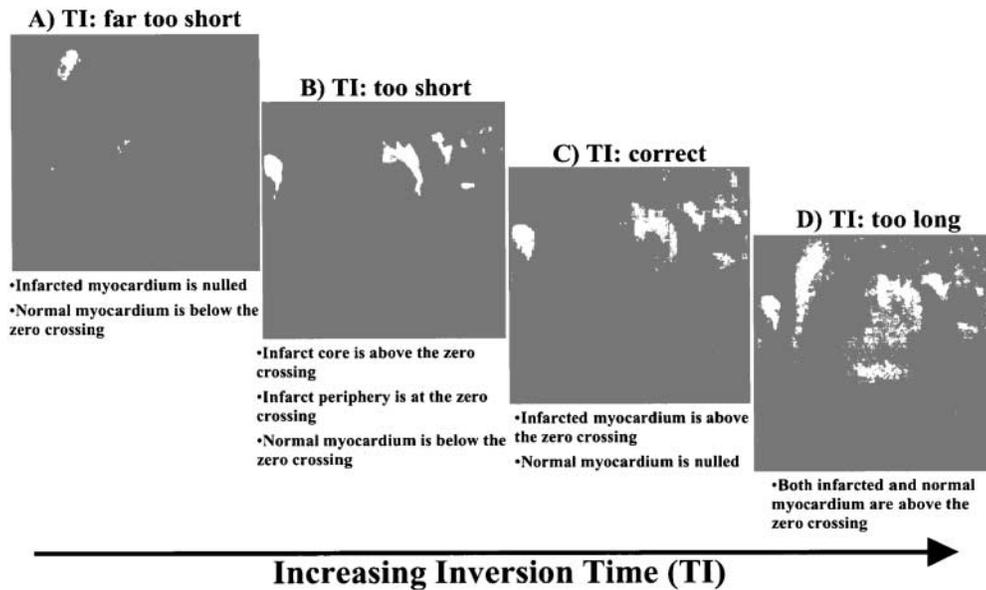
Selecting the appropriate TI is extremely important for obtaining accurate imaging results. The TI is chosen to “null” normal myocardium, the time at which the magnetization of normal myocardium reaches the zero crossing (Fig. 3a). It is at this point (or immediately just after) that the image intensity difference between infarcted and normal myocardium is maximized (Fig. 3c). If the TI is set too short, normal myocardium will be below the zero crossing and will have a negative magnetization vector at the time of k-space data acquisition. Since the image intensity corresponds to the magnitude of the magnetization vector, the image intensity of normal myocardium will increase as the TI becomes shorter and shorter, whereas the image intensity of infarcted

myocardium will decrease until it reaches its own zero crossing (Fig. 3b). At this point infarcted myocardium will be nulled and normal myocardium will be hyperenhanced. On the opposite extreme, if the TI is set too long, the magnetization of normal myocardium will be above zero and will appear gray (not nulled). Although areas of infarction will have high image intensity, the relative contrast between infarcted and normal myocardium will be reduced. In principle, the optimal TI at which normal myocardium is nulled must be determined by imaging iteratively with different inversion times. In practice, however, only one or two “test” images need to be acquired, because with experience one can estimate the optimal TI based on the amount of contrast agent that is administered and the time after contrast agent administration. Figure 4 shows images of a subject with an anterior wall myocardial infarction in which the TI has been varied from too short to too long. Note that with the TI set moderately too short (Fig. 4b), the anterior wall does have some regions that are hyperenhanced; however, the total extent of hyperenhancement is less than that seen when the TI is set correctly (Fig. 4c). This is due to the periphery of the infarcted region passing through a zero-crossing, thereby affecting its apparent size (Judd and Kim, 2002). If the TI is set too long (Fig. 4d), although the contrast is reduced as previously stated, the total extent of hyperenhancement does not change. Thus, it is better to error on the side of setting the TI too long rather than too short.

As stated previously, data is acquired every other heartbeat in order to allow for adequate longitudinal relaxation between successive inversion pulses. The time for recovery of 96% of the bulk magnetization is approximately four times the  $T_1$  [ $M(t) = M_0(1 - 2e^{-t/T_1})$  or  $M(4T_1) = M_0(1 - 2e^{-4})$ ]. Therefore, as an example, if  $T_1$  of normal myocardium with gadolinium on board is 400 msec, then in order to achieve adequate longitudinal relaxation there should be approximately 1600 msec between successive inversion pulses or every other heartbeat imaging in patients with heart rates of 75 beats-per-minute (R-R interval = 800 msec). In this example, the optimal TI to null normal myocardium would be approximately 280 msec [ $TI(\text{null}) = \ln(2) \cdot T_1 = 0.69 \cdot T_1$ ]. Occasionally imaging is performed every third heartbeat (Gating Factor of 3) if the patient is tachycardic. Conversely, due to limitations in breath-hold duration and/or bradycardia, every heartbeat imaging is occasionally performed. In this situation there may be incomplete relaxation of normal myocardium. Incomplete relaxation will result in not only an artificially shorter “effective” TI needed



**Figure 3.** a) Inversion recovery curves of normal and infarcted myocardium assuming  $T_1$  of normal myocardium is 450 msec and infarcted myocardium is 250 msec. The time at which the magnetization of normal myocardium reaches the zero crossing is defined as the inversion time to "null" normal myocardium (312 msec in this example). b) Image intensities resulting from an inversion prepulse with various inversion delay times. Note that image intensities correspond to the magnitude of the magnetization vector and cannot be negative. c) Difference in image intensities between infarcted and normal myocardium as a function of inversion time. The optimal inversion time is when the maximum intensity difference occurs.



**Figure 4.** Delayed enhancement images in a subject with an anterior wall myocardial infarction in which the TI has been varied from too short to too long. See text for details.

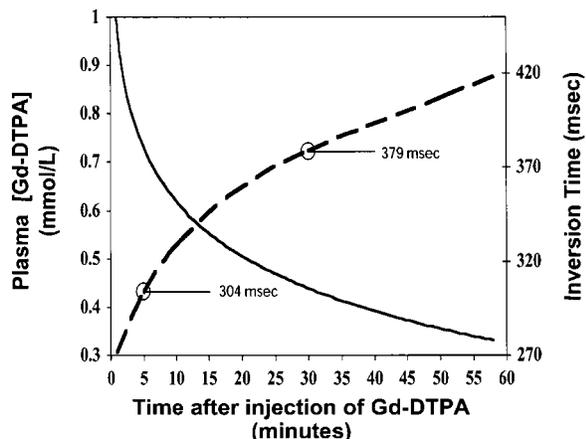
to null normal myocardium, but may also lead to a reduction in the image intensity differences between infarcted and normal myocardium.

Fortunately, for those that are unfamiliar with inversion recovery relaxation curves, newer pulse sequences that allow phase-sensitive reconstruction of inversion recovery data may allow a nominal TI to be used rather than a precise null time for normal myocardium (Kellman et al., 2002). These techniques, by restoring signal polarity, can provide consistent contrast between infarcted and normal myocardium over a wide range of inversion times and can eliminate the apparent reduction in infarct size that is seen on images acquired with inversion times that are too short (Kellman et al., 2002). TI “scout” sequences also have been developed to aid in selecting the correct inversion time (Chung et al., 2002). In our experience, these sequences only provide a reasonable “first guess” at the correct TI, since they can be off by as much as 40–50 msec. This discrepancy may be due to several reasons. For example, TI scout sequences generally do not account for the gating factor that is used in the seg IR-FGE sequence, which can lead to changes in the correct TI as described above. Additionally, recovery of longitudinal magnetization is not identical for IR-prepared steady-state free precession sequences (often used for TI scouting (Chung et al., 2002)) and IR-prepared FGE sequences (Scheffler and Hennig, 2001). In our laboratory we do not use TI scout sequences.

#### IMAGING TIME AFTER CONTRAST ADMINISTRATION

In general, once the optimal TI has been determined, the TI does not require adjustment if all delayed enhancement views can be acquired within approximately 5 minutes. However, it is important to keep in mind that the gadolinium concentration within normal myocardium gradually washes out with time, and the TI will need to be adjusted upwards if delayed-enhancement imaging is performed at multiple time points after contrast administration. For example, Fig. 5 demonstrates that the plasma concentration of gadolinium decreases exponentially with time following contrast administration (solid line). Because interstitial concentrations of Gd-DTPA in the myocardium depend primarily upon plasma concentrations, the correct inversion time needed to null normal myocardium can be estimated from basic physical principles (Mahrholdt et al., 2002). The dashed line in Fig. 5 depicts the correct inversion times that are needed to account for the pharmacokinetics of the contrast agent. In this example (gadolinium dose of 0.125 mmol/kg), the correct TI to null normal myocardium at 30 mins post contrast will be 379 msec as compared with 304 msec at 5 mins. The basic premise here, is not that one can calculate the correct TI for a given time point after contrast administration, but that the TI needs to be adjusted, sometimes significantly, if the imaging time is prolonged.

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**Figure 5.** Solid Line: Monoexponential fit to the plasma gadolinium concentration (left-hand y-axis) as a function of time post-contrast administration (0.125 mmol/kg gadolinium) extrapolated from data from Weinmann, Laniado, and Mutzel (1984) in humans. Dashed Line: MRI inversion time (right-hand y-axis) to null normal myocardium calculated from the data of the solid line. Open Circles: The calculated TI to null normal myocardium at 5 mins and 30 mins post-contrast administration.

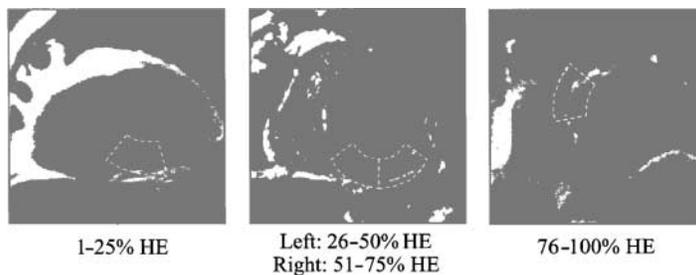
Recently, Oshinski et al. (2001) have suggested that the “accurate determination of infarct size by delayed enhancement MRI requires imaging at specific times after gadolinium-DTPA injection.” This conclusion was based on the observation that the size of the hyperenhanced regions decreased with increasing time after contrast administration. However, in this study the inversion time was not adjusted to “null” normal myocardium but held constant throughout the 40 minutes of imaging post-contrast. In this situation, one is in effect choosing a TI that is further and further from the correct TI (too short) as the time after contrast administration

increases. Mahrholdt et al. (2002) have shown in the setting of chronic infarction that when the inversion time is adjusted appropriately to “null,” normal myocardium the size of hyperenhanced regions does not change if imaging is performed between 10 to 30 minutes post-contrast. Further validation studies, however, will be required in the setting of acute myocardial injury and for imaging before 10 minutes post-contrast.

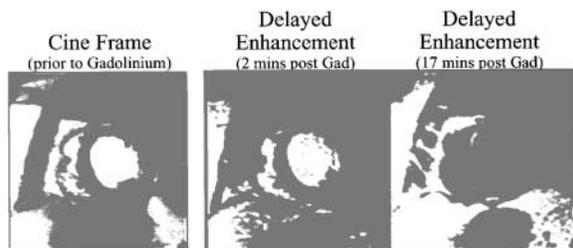
## INTERPRETATION

For general clinical reporting, we use the 17-segment model recommended by the American Heart Association (Cerqueira et al., 2002). This model divides the basal and mid-cavity levels into six segments each, an apical level into four segments, and the true apex into one segment. The extent of hyperenhanced tissue within each segment is graded visually using a 5-point scale in which a score of 0 indicates no hyperenhancement; 1, hyperenhancement of 1–25% of the segment; 2, hyperenhancement of 26–50% of the segment; 3, hyperenhancement of 51–75% of the segment; and 4, hyperenhancement of 76–100% of the segment (Kim et al., 2000). Examples of myocardial segments with various transmural extents of hyperenhancement are demonstrated in Fig. 6.

Generally the delayed-enhancement images are interpreted with the cine images immediately adjacent (see Fig. 1). The cine images can provide a reference of the diastolic wall thickness of each region. This may be helpful if delayed enhancement imaging is performed before there is significant contrast washout from the LV cavity, and there is difficulty in differentiating the bright signal from the LV cavity from hyperenhanced myocardium. As stated previously, the contrast between blood pool and hyperenhanced myocardium may be



**Figure 6.** Typical images showing myocardial segments (dashed white lines) with various transmural extents of hyperenhancement.



**Figure 7.** Short-axis view of a patient with an anterior wall myocardial infarction. Diastolic still frame taken from the cine images before gadolinium administration is compared with the delayed enhancement image taken both early and late following gadolinium injection. Note that it is difficult to differentiate the bright LV cavity from the subendocardial infarction in the early (2 mins) delayed enhancement image. The cine frame, by showing the diastolic wall thickness in the anterior wall, provides evidence that there is subendocardial hyperenhancement in the anterior wall on the early delayed enhancement image. The late (17 mins) delayed enhancement image provides confirmation that there is subendocardial hyperenhancement in the anterior wall.

improved if imaging occurs at a later time point after gadolinium administration (see Fig. 7). Additionally, hyperenhancement (i.e., infarction) of papillary muscles may be overlooked within the blood pool cavity if the delayed enhancement images are interpreted without the benefit of the cine images. One caveat should be mentioned, however. The cine frames generally have much higher temporal resolution (30–50 msec) than the delayed enhancement images (140–200 msec). Thus, a true comparison of wall thickness may require averaging 4–5 mid-diastolic still-frames from the cine image in order to represent the same time window as the corresponding delayed enhancement image.

### PITFALLS/ADVANCED ISSUES

As with any technique (MRI or otherwise) there are potential pitfalls that one should recognize. Some of these are due to patient-specific issues and others due to the MRI technique. In patients with atrial fibrillation or significant ventricular ectopy, there can be degradation in image quality, which is due to the segmented nature of the sequence. Similarly, images may be poor in patients that cannot breath-hold for the 8–10 seconds required for image acquisition. These limitations can be overcome with use of respiratory navigators or “single-shot” techniques. Although there is a reduction in spatial and temporal resolution with single-shot methods, in our

experience the IR-prepared steady-state free precession version (IR-SSFP) provides reasonable image quality for patients such as above. As compared with single-shot IR-FGE, the SSFP version typically allows high bandwidth imaging (short TE and TR for faster imaging) with preservation of signal-to-noise. One limitation, however, is that there can be a reduction in pure  $T_1$  contrast effects (such as following contrast media administration) since they are  $T_2/T_1$  weighted sequences.

Ghosting artifacts can result from regions within the field-of-view that have long  $T_1$  values (such as pericardial effusion or cerebrospinal fluid). This is due to the fact that with long  $T_1$  the train of inversion pulses in the seg IR-FGE sequence will lead to oscillation of magnetization before each group of k-space acquisitions. This artifact can be reduced by application of a single nonselective saturation pulse approximately 600 to 800 msec (~half the time interval between inversion pulses or the R to R interval for every heart beat imaging) prior to the initial  $180^\circ$  inversion pulse.

Most MR vendors have both 2-dimensional and 3-dimensional delayed enhancement sequences. Three-dimensional (3D) sequences typically require every heartbeat imaging in order to keep the breathhold time to an acceptable level. For 3D imaging the inherent improvement in signal-to-noise is balanced by the fact that there is incomplete relaxation of normal myocardium prior to sequential inversion pulses, which may reduce the contrast between infarcted and normal myocardium. Although the use of higher doses of contrast media (i.e., 0.2 mmol/kg) may alleviate this problem, this solution may impair the detection of subendocardial infarcts by producing high signal in the LV cavity for a prolonged period as described above.

Fat saturation is available on the delayed enhancement sequences by many of the vendors. We generally have not found this feature to be particularly useful, since high signal from epicardial fat may actually aid in distinguishing the epicardial border of myocardium that is nulled from the lung fields that have low signal. It is important to keep in mind, however, that if using fat saturation, the k-space lines must be acquired centrally. With linear acquisition, fat, by virtue of its short  $T_1$ , will have recovered signal by the time the center lines of k-space are acquired, thus resulting in ineffective fat saturation.

We generally have gradient moment refocusing (GMR) turned on. Although turning GMR off will allow the minimum TE and TR to be reduced by approximately 1 to 1.5 msec or allow lower bandwidth imaging (higher SNR) for the same TE and TR, we have found that motion or flow induced artifacts can occur when GMR is off, particularly when imaging the base of the heart.



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Additionally, we generally allow asymmetric or partial echo acquisition in order to reduce the TE.

### SUMMARY

When appropriately performed, delayed enhancement imaging produces reproducible, high spatial resolution information about myocardial injury and viability. Use of delayed enhancement imaging is under further investigation for patients with coronary artery disease as well as for patients with a variety of nonischemic cardiac disorders. As the utility of this technique continues to expand, delayed enhancement imaging is emerging as an integral part of the core examination in the MRI assessment of cardiovascular patients.

### ACKNOWLEDGMENTS

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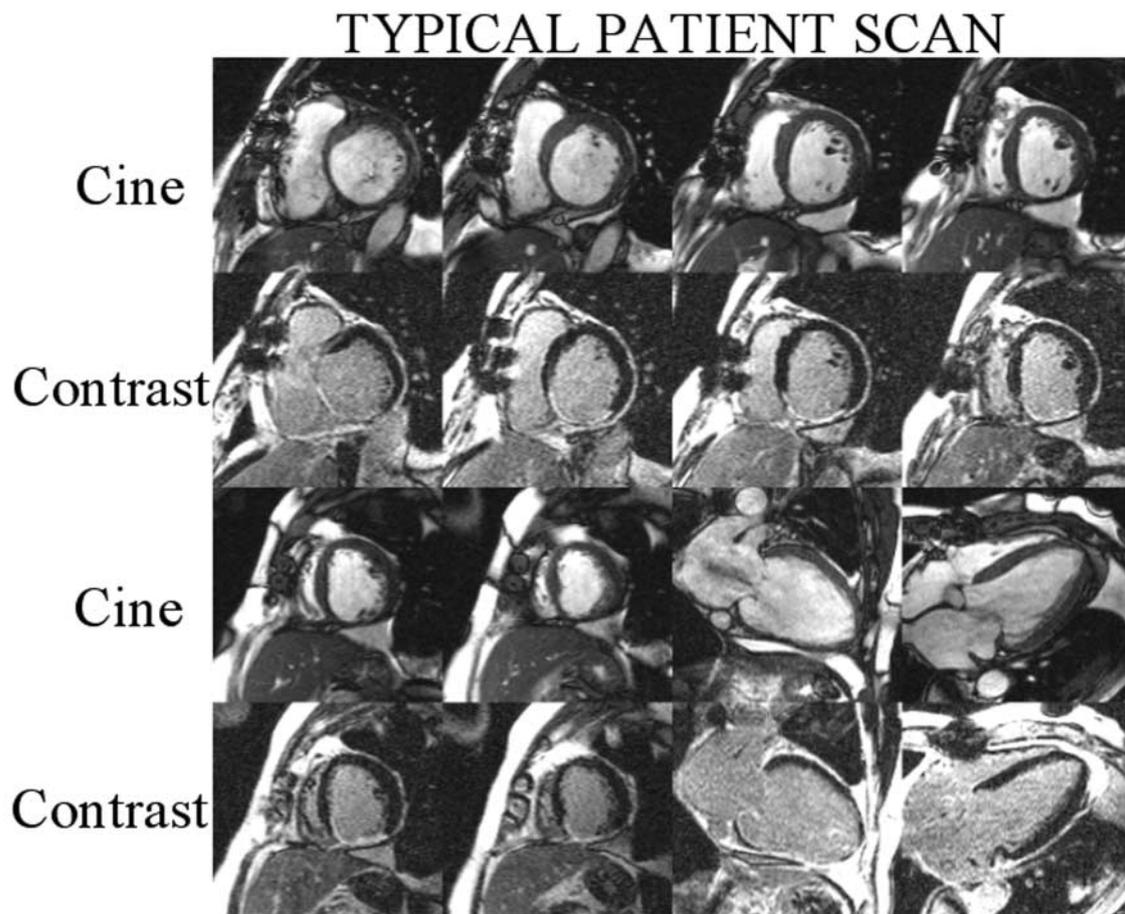
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## Erratum

“How We Perform Delayed Enhancement Imaging,”  
by R. J. Kim, D. J. Shah, and R. M. Judd, *Journal of  
Cardiovascular Magnetic Resonance*, 5(3), 505–514  
(2003).

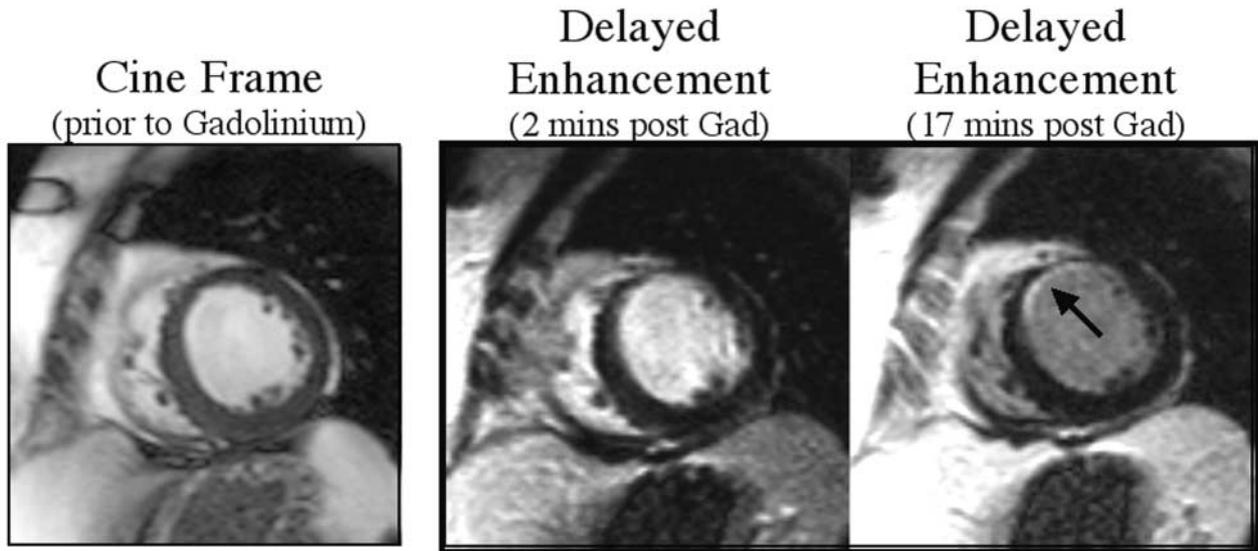
Figures 1, 4, 6 and 7 from the above article are being  
reprinted here because they were shown in poor contrast  
in the original article.



**Figure 1.** Images from a typical patient scan. Cine and contrast-enhanced images are acquired at six to eight short and two to three long-axis locations during repeated breath-holds. Images are interpreted with cine images immediately adjacent to contrast images. This particular patient had a myocardial infarction caused by occlusion of the right coronary artery. Note hyperenhancement of the inferior wall.







**Figure 7.** Short-axis view of a patient with an anterior wall myocardial infarction. Diastolic still frame taken from the cine images before gadolinium administration is compared with the delayed enhancement image taken both early and late following gadolinium injection. Note that it is difficult to differentiate the bright LV cavity from the subendocardial infarction in the early (2 mins) delayed enhancement image. The cine frame, by showing the diastolic wall thickness in the anterior wall, provides evidence that there is subendocardial hyperenhancement in the anterior wall on the early delayed enhancement image. The late (17 mins) delayed enhancement image provides confirmation that there is subendocardial hyperenhancement in the anterior wall.

