An Improved MR Imaging Technique for the Visualization of Myocardial Infarction[1]

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PURPOSE: To design a segmented inversion-recovery turbo fast low-angle shot (turboFLASH) magnetic resonance (MR) imaging pulse sequence for the visualization of myocardial infarction, compare this technique with other MR imaging approaches in a canine model of ischemic injury, and evaluate its utility in patients with coronary artery disease.

MATERIALS AND METHODS: Six dogs and 18 patients were examined. In dogs, infarction was produced and images were acquired by using 10 different pulse sequences. In patients, the segmented turboFLASH technique was used to acquire contrast material–enhanced images 19 days ± 7 (SD) after myocardial infarction.

RESULTS: Myocardial regions of increased signal intensity were observed in all animals and patients at imaging. With the postcontrast segmented turboFLASH sequence, the signal intensity of the infarcted myocardium was 1,080% ± 214 higher than that of the normal myocardium in dogs—nearly twice that of the next best sequence tested and approximately 10-fold greater than that in previous reports. All 18 patients with myocardial infarction demonstrated high signal intensity at imaging. On average, the signal intensity of the high-signal-intensity regions in patients was 485% ± 43 higher than that of the normal myocardium.

CONCLUSION: The segmented inversion-recovery turboFLASH sequence produced the greatest differences in regional myocardial signal intensity in animals. Application of this technique in patients with infarction substantially improved differentiation between injured and normal regions.

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It has been known for several years that regions of myocardial injury exhibit higher signal intensity than does the normal myocardium on T2-weighted magnetic resonance (MR) images without contrast agent enhancement (1–4) and T1-weighted MR images following administration of contrast agents such as gadopentetate dimeglumine (5–7). Since these initial observations, numerous studies of myocardial injury have been performed by using a variety of pulse sequences with and without contrast agent administration to differentiate injured from normal myocardium.

Ultimately, tissue characteristics such as MR relaxation parameters and physiologic motion limit the differentiation of injured and normal regions. Therefore, in a clinical setting, selecting the "best" MR imaging technique for the examination of injury becomes an important goal. Trade-offs between image contrast, signal-to-noise ratio, imaging time, and motion sensitivity are required. A variety of techniques for imaging of injured myocardium have been described in the literature.

We previously described T2-weighted breath-hold techniques for imaging of myocardial infarction (8) and more recently have implemented a breath-hold inversion-recovery segmented turbo fast low-angle (turboFLASH) sequence for T1-weighted postcontrast imaging of infarction. This sequence produces strongly T1-weighted images owing to the use of an inversion pulse before image acquisition. Several features of the sequence are similar to those of the original segmented turboFLASH technique for breath-hold abdominal imaging without a contrast agent described by Edelman et al (9). Edelman et al (9) demonstrated that segmentation of the acquisition can generate images that are more strongly dependent on the inversion recovery of magnetization than are single-shot

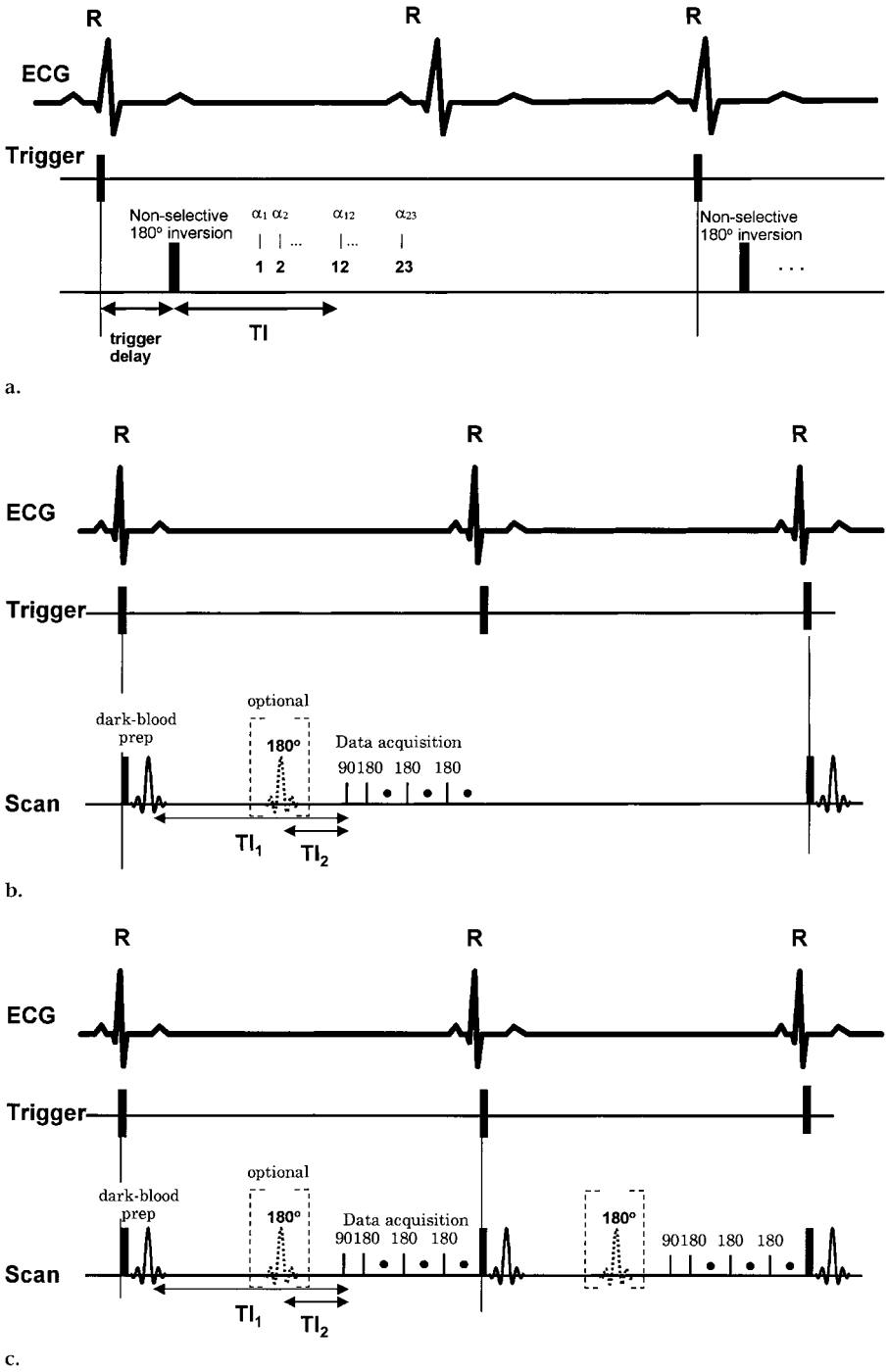


Figure 1. Diagrams of different pulse sequences. **(a)** Segmented inversion-recovery turboFLASH sequence with TI set to null normal myocardium after contrast agent administration. **(b)** T2-weighted turbo spin-echo sequence with (short TI inversion recovery) and without (conventional turbo spin echo) an inversion pulse. The first TI (TI_1) is set to null blood signal intensity, and the second TI (TI_2) is set to null the signal intensity of fat and other short-T1 tissue. *prep* = preparation. **(c)** T1-weighted turbo spin-echo sequence with (inversion-recovery turbo spin echo) and without (conventional turbo spin echo) an inversion pulse. The first TI (TI_1) is set to null blood signal intensity, and the second TI (TI_2) is set to null normal myocardial signal intensity after contrast agent administration. *prep* = preparation. In **a–c**, *ECG* = electrocardiogram, *R* = R wave (*Fig 1 continues*).

turboFLASH images. Because the more recent sequence is intended for cardiac rather than abdominal imaging and specifically designed for use with a contrast agent, several aspects of it are different from those of the technique described by Edelman et al. Specifically, the acquisition is synchronized with diastole, and gradient-moment refocusing is used to reduce motion artifact. More important, however, the inversion time (TI) of this sequence is specifically chosen to null the signal intensity of normal myocardium after contrast agent administration.

This segmented turboFLASH sequence has already been commercially distributed, and several groups have demonstrated its use for imaging of myocardial infarction in animal models (10,11). In this study, we compared this sequence with nine others in an animal model of myocardial infarction and evaluated its clinical utility in patients with coronary artery disease.

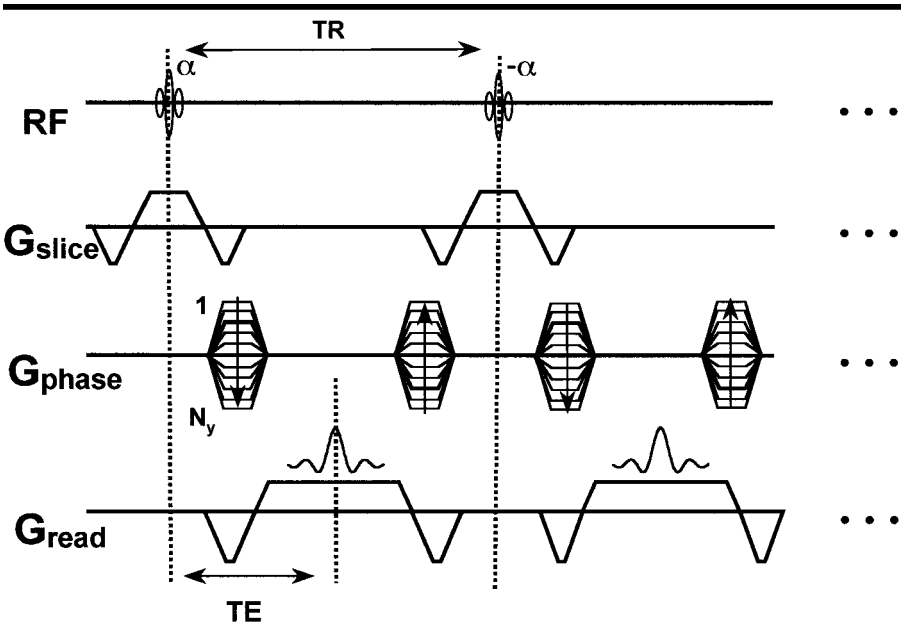
MATERIALS AND METHODS

All animal and human studies were approved by Northwestern University's Animal Care and Use Committee and Institutional Review Board, respectively. Written informed consent was obtained from each patient after the nature of the procedure(s) had been fully explained.

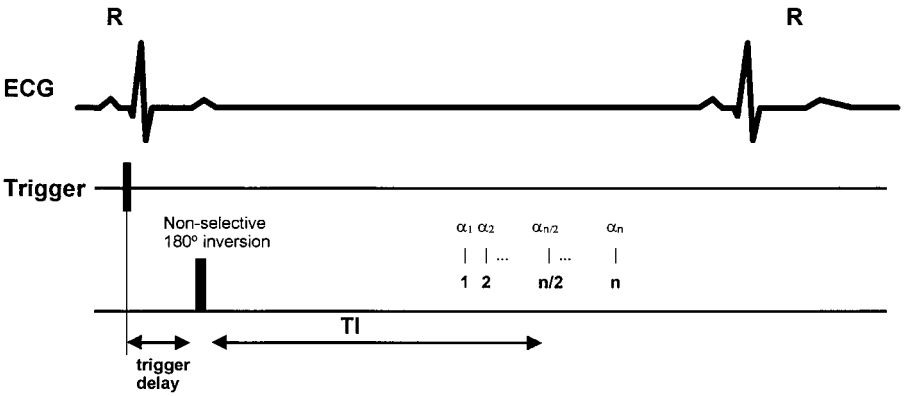
Pulse Sequences

All the sequences tested are summarized in Table 1. The details of specific sequences are illustrated in Figure 1. To ensure fair comparison among the images acquired by using the different sequences, spatial resolution was maintained nearly constant (1.2 × 1.2 × 5 mm) by adjusting the number of phase-encoding steps and the degree of the rectangular field of view and using the same section thickness. Timing values within each individual sequence were chosen to be similar to those commonly used for cardiac imaging.

Segmented turboFLASH.—A diagram of the segmented turboFLASH pulse sequence is shown in Figure 1a. After a variable delay to allow acquisition of the image data at end diastole, a nonselective 180° hyperbolic secant adiabatic inversion pulse was applied. After the 180° pulse, a variable TI delay was used to allow T1 relaxation. Regional myocardial differences in image signal intensity are primarily determined by this TI delay. After the TI delay, a group of k-space lines were acquired. The flip angle used for radio-frequency excitation for each



d.



e.

Figure 1. (*continued*) **(d)** Timing diagram of two-dimensional trueFISP pulse sequence. *TE* = echo time, G_{phase} = phase-encoding gradient, G_{read} = readout gradient, G_{slice} = section-selection gradient, N_y = number of phase encode lines, *RF* = radio frequency, *TR* = repetition time. **(e)** Single-shot inversion-recovery turboFLASH sequence with TI set to null normal myocardial signal intensity either before or after contrast agent administration. The entire image is acquired during one cardiac cycle. *ECG* = electrocardiogram, *R* = R wave.

k-space line is chosen to be relatively shallow (eg, 20°–30°) to retain regional differences in magnetization that result from the 180° pulse and TI delay. The number of k-space lines in the group is limited by the repetition time between each k-space line and the duration of diastole.

In our implementation, 23 k-space lines were acquired per cardiac cycle (8.0/4.0 [repetition time msec/echo time msec], 230 Hz/pixel bandwidth, gradient-moment refocusing). At a repetition time of 8 msec, the 23 lines are acquired in 184 msec, which is fast enough to be insensitive to cardiac motion at middle diastole. To allow for more complete T1 relaxation between image acquisitions, the entire wait-invert-wait-collect cycle was triggered every other cardiac cycle. With this implementation, image acquisition typically necessitated a breath-hold duration of 12 cardiac cycles.

Other sequences.—The first two sequences listed in Table 1 are T2 weighted and were performed before contrast agent administration. Two variations of the same 23-echo, black-blood–prepared, breath-hold turbo spin-echo sequence were tested with a preparatory inversion pulse and without one: T2-weighted short TI inversion recovery and T2-weighted turbo spin echo, respectively (Fig 1b). The use of these sequences in the imaging of myocardial infarction has been previously described (8). An effective echo time of 96 msec and a repetition time of two to three cardiac cycles were used to provide T2 weighting. In the T2-weighted short TI inversion-recovery sequence variant, this readout was combined with a nonselective short TI pulse designed to suppress the signal intensity of short-T1 tissues. A single 138 × 256-pixel image could be obtained in 12–18 heartbeats during breath holding by using either of these techniques.

The last eight sequences listed in Table 1 are T1 weighted and were performed after contrast agent administration. These sequences can be divided into spin-echo and gradient-echo techniques. The spin-echo sequences tested were T1-weighted spin echo, turbo spin echo, and inversion-recovery turbo spin echo (Fig 1c). T1-weighted spin-echo imaging is not performed with breath holding, and, thus, image quality is often poor owing to respiratory motion artifacts. For this reason, this sequence has been largely discarded for postcontrast imaging of the heart. However, because much of the existing research on MR imaging of myocardial infarction has been done by using the T1-weighted spin-echo sequence, it is included here for comparative purposes. The TI of the T1-weighted inversion-recovery spin-echo sequence was set to null postcontrast normal myocardial signal intensity.

Of the five gradient-echo sequences tested, two were steady-state imaging techniques and three involved inversion recovery to improve T1 weighting. The magnetization-driven FLASH technique (12) involves constant radio-frequency pulsing with a spoiled gradient-echo readout. The electrocardiographically gated trueFISP sequence (Fig 1d) has not, to our knowledge, been described for this application before now. The TrueFISP sequence (13,14) involves constant radio-frequency pulsing and gradient refocusing of transverse magnetization to produce images sensitive to the T2-to-T1 ratio. Two types of inversion-recovery turboFLASH pulse sequences were evaluated: a single-shot technique designed to enable acquisition of an entire image within one heartbeat and the segmented sequence designed to enable acquisition of a single image during breath holding over several cardiac cycles (described in the previous section). Single-shot inversion-recovery turboFLASH (Fig 1e) with a TI set to null precontrast normal myocardial signal intensity has been reported for first-pass and postcontrast imaging of infarction in the literature (15,16). The same pulse sequence with a TI set to

TABLE 1
Description of Pulse Sequences Tested

Sequence	Magnetization Preparation*	Weighting	Parameters†
T2-weighted turbo spin echo	Double IR to null blood	T2	2·RR/96, 23 lines per beat
T2-weighted turbo short TI inversion recovery	Double IR to null blood, IR for short-T1 tissue suppression	T2 and T1	2·RR/96, 160-msec TI, 23 lines per beat
T1-weighted spin echo	None	T1	RR/14, one line per beat
T1-weighted turbo spin echo	Double IR to null blood	T1	RR/24, nine lines per beat
T1-weighted inversion-recovery turbo spin echo	Double IR to null blood, IR for suppression of postcontrast normal myocardium	T1	RR/24, 200-msec TI, nine lines per beat
Steady-state spoiled gradient echo‡	Dummy RF pulses applied to maintain steady state	T1	8.0/4.0, nine lines per beat, 45° flip angle
Steady-state true fast imaging with steady precession (TrueFISP)	Dummy RF pulses applied to maintain steady state	T1/T2	3.0/1.5, 20 lines per beat, 70° flip angle
Single-shot inversion-recovery turboFLASH, precontrast null	IR pulse, with TI set to null precontrast myocardium	T1	5.0/2.1, 650-msec TI, 128 lines per beat, 10° flip angle
Single-shot inversion-recovery turboFLASH, postcontrast null	IR pulse, with TI set to null postcontrast myocardium	T1	5.0/2.1, 300-msec, TI, 128 lines per beat, 10° flip angle
Segmented inversion-recovery turboFLASH, postcontrast null	IR pulse, with TI set to null postcontrast myocardium	T1	8.0/4.0, 200-msec TI, 23 lines per beat, 30° flip angle

Note.—Imaging parameters were chosen to optimize image quality or to match the reported use of these sequences.
* IR = inversion recovery, RF = radio frequency.
† In each listing, the first parameter is the repetition time in either RR interval of the electrocardiogram (RR) or msec/echo time in msec.
‡ Same as magnetization-driven FLASH.

TABLE 2
Percent Elevations in MR Signal Intensity of Infarcted versus Normal Myocardium and Voxel Sizes: Previously Published and Current Data

				Canine		Human	
Year	Reference	Technique*	Breath Hold	ΔI/R (%)†	Voxel Size (mm^3)‡	ΔI/R (%)†	Voxel Size (mm^3)
1986	5	Spin echo	No	80	NS		
1986	6	Spin echo	No	70	NS		
1986	7	Spin echo	No			42§	29.3‖
1988	18	Spin echo	No			60	29.3‖
1989	19	Spin echo	No			36	29.3‖
1990	20	Spin echo	No			32§	31.3‖
1991	21	Spin echo	No			31§	27.5
1991	22	Spin echo	No			42	29.3‖
1994	23	Spin echo	No			41§	29.3‖
1995	24	MD-SPGR	Yes			103§	33.3
1995	25	MD-SPGR	Yes	123#	39.6		
1998	26	MD-SPGR	Yes			58#	30.8
1999	27	MD-SPGR	Yes	79#	14.7		
1999	15	Single-shot inversion-recovery FLASH	Yes			39	54.9**
Mean of previous studies				86	27.2	48	32.4
Current study				1,080	6.2	485	16.8

* All images were acquired in vivo at least 5 minutes after the administration of a United States Food and Drug Administration–approved MR imaging contrast agent. MD-SPGR = magnetization-driven spoiled gradient echo.
† ΔI/R = percent elevation in MR signal intensity of infarcted myocardium compared with signal intensity of normal mycocardium.
‡ NS = not stated.
§ Published data were reported as pre- versus postcontrast values; values in Table 2 were calculated as follows: (postcontrast value − precontrast value)/precontrast value.
‖ Assuming a field of view of 320 mm.
Estimated from data reported in graphical format.
** Assuming a rectangular (6/8) field of view.

null postcontrast myocardial signal intensity also was tested.

Animal Preparation and Imaging

Mongrel dogs (25–30 kg) were premedicated intramuscularly with 0.4 mg/mL of fentanyl and 20 mg/mL of droperidol followed by 11 mg/kg of methohexital sodium (Brevital; Eli Lilly, Indianapolis, Ind) intravenously. The animals were then intubated and ventilated with gas anesthesia (halothane). Their chests were opened under sterile conditions, and a single coronary artery—either the left anterior descending or left coronary artery—was ligated to produce infarction. The chest was then closed, and the animals were allowed to recover.

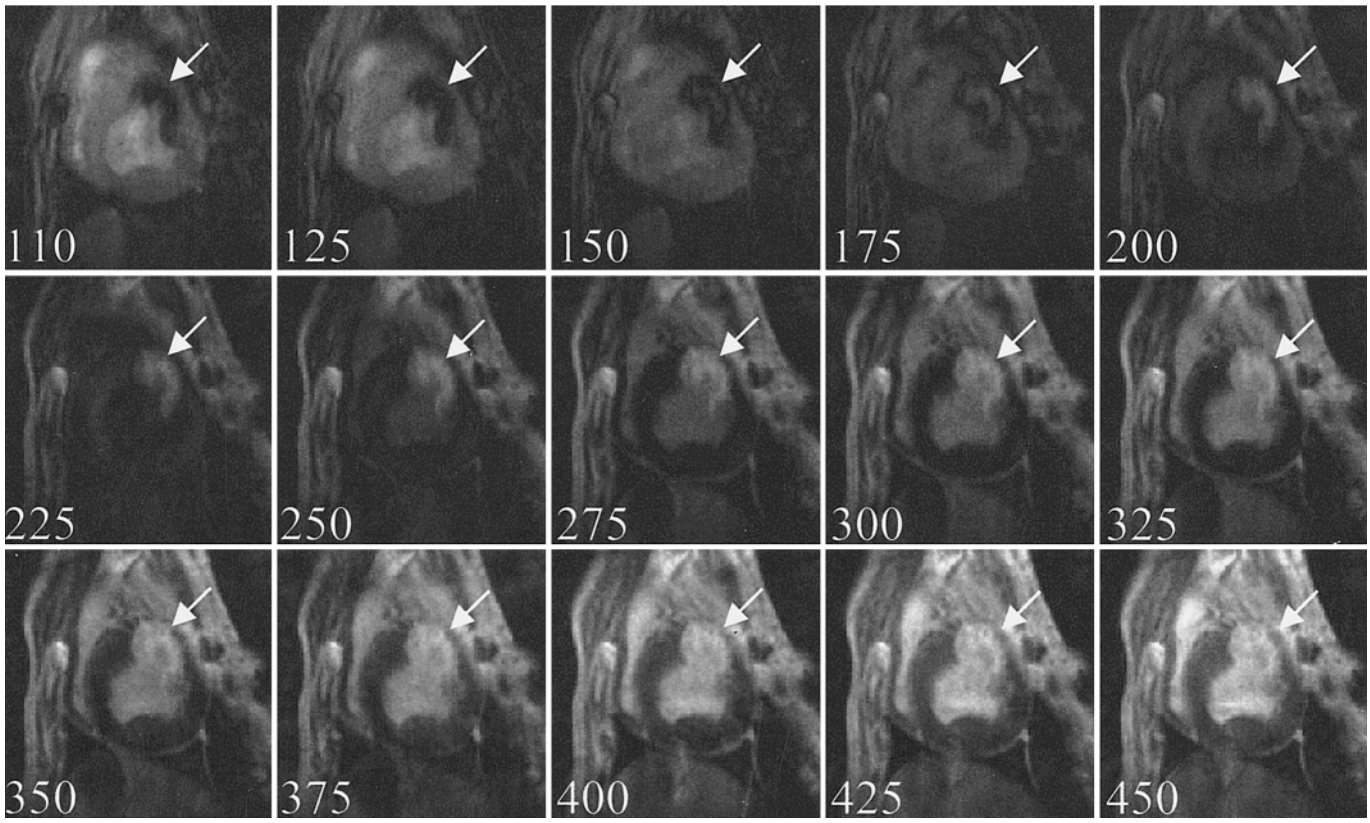


Figure 2. Effect of TI on regional myocardial signal intensities on 15 short-axis segmented turboFLASH images (8/4) obtained in one dog. On each image, the TI (in milliseconds) is labeled in the bottom left corner, and the infarcted region is indicated by the arrow. Note the transition from low to high signal intensity in the anterior infarcted region as the TI increases. In this case, optimal contrast enhancement is achieved at a TI of 275 msec, the point at which the signal intensity of normal myocardium is nulled.

The animals were imaged at 3 days ($n = 2$), 4 weeks ($n = 2$), or 8 weeks ($n = 2$) after infarction with a 1.5-T MR imaging system (Sonata; Siemens, Erlangen, Germany). All animals were anesthetized with 25 mg/kg of sodium pentobarbital intravenously, intubated, and imaged in the right lateral decubitus position by using a 14 × 28-cm flexible surface coil. After scout images were acquired, a single cardiac view encompassing the region of myocardial injury was chosen for further study on the basis of cine MR imaging findings.

The first two pulse sequences listed in Table 1 were performed before administration of the contrast agent (gadoteridol 0.3 mmol/kg, ProHance; Bracco Diagnostics, Princeton, NJ), which was then administered intravenously. Fifteen minutes later, the last eight sequences listed in Table 1 were performed in random order to acquire postcontrast images. All eight sequences were performed within 10 minutes. With all the sequences except T1-weighted spin echo, images were acquired during a breath hold of approximately 8 seconds and were electrocardiographically gated to end diastole. In one animal, the effect of varying the TI of the segmented turboFLASH sequence was investigated by imaging with 10 different TIs.

Patient Selection and Imaging

The utility of the segmented turboFLASH sequence was evaluated by testing the hypothesis that patients with prior myocardial infarction will demonstrate high signal intensity at imaging. Elevated serum levels of creatine kinase were used as the reference standard for the presence of myocardial infarction. The patient population consisted of eighteen consecutive patients (10 men, eight women; mean age [± SD], 55 years ± 10; age range, 35–72 years) who were referred for cardiac MR imaging with a myocardium-specific creatine kinase isoenzyme elevation of more than twice the upper limits of normal. Peak serum levels of creatine kinase were 1,516 U/L ± 418 (range, 126–5,912). The myocardium-specific creatine kinase isoenzyme level, or CK-MB, and percentage were 131 U/L ± 46 and 8.5% ± 0.8 (SI unit, .085 ± .008), respectively. Patients underwent imaging 19 days ± 7 (range, 1–90 days) after documented enzyme elevations. No patients were excluded for insufficient image quality or other reasons.

Images were acquired with a 1.5-T Sonata MR imaging system (Siemens). After scout images were acquired to obtain standard short- and long-axis views, patients were then injected intravenously by hand with a 0.2 mmol per kilogram of body weight dose of gadoteridol. At least 5 minutes was allowed for the contrast agent to circulate, then contrast-enhanced images were acquired at multiple short- and long-axis locations in the heart by using the segmented turboFLASH technique. The TI (200–300 msec) was adjusted with each patient and set to null the normal postcontrast myocardial signal intensity by acquiring one or two test images. Each image was acquired in a single breath hold (<10 seconds). The total time for the MR imaging procedure was approximately 20 minutes.

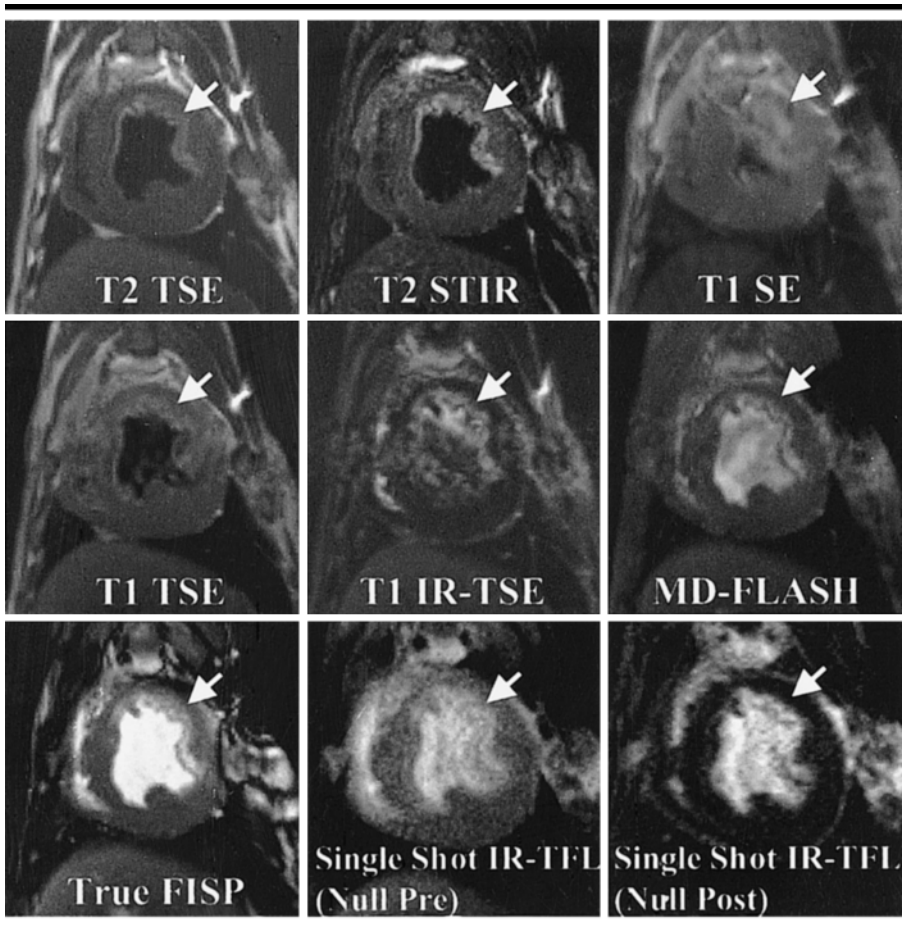
Data Analysis

For each of the six sets of images obtained in the animal studies, the 10 images were randomized and presented to two independent observers (R.M.J., O.P.S.) who were blinded to the pulse sequence used. Each observer measured the mean signal intensity in the myocardial regions of elevated signal intensity and in a remote myocardial region. They were instructed to outline the region of elevated signal intensity by hand on a single image of their choice and then use the same region of anatomy for all 10 images. Each observer also measured the SD of the noise measured in a rectangular region outside the body. The percent signal intensity elevation in the myocardium was calculated by using the following equation: percent elevation $\equiv 100 \times$ (mean signal intensity of high-signal-intensity region − mean signal intensity of normal region)/(mean signal intensity of normal region. Image contrast-to-noise ratios were calculated by using the following equation: (mean signal intensity of the region of elevated signal intensity − mean signal intensity of the remote region)/1.5 × SD of noise. On images for which the TI was varied, a single observer outlined a region of interest by hand and used the same region of interest for all images.

From each of the 18 patient studies, a single short-axis image showing the largest high-signal-intensity region and the normal regions of myocardium was selected. A single observer (R.M.J.) measured the mean signal intensity of the myocardial regions with elevated signal intensity and that of a remote myocardial region. The percent elevation in signal intensity was calculated in the same manner as that used to obtain animal data.

Statistical Analyses

All results were expressed as the mean plus or minus SEM. Percent signal intensity elevation and contrast-to-noise values were compared by using one-way repeated measures analysis of variance. Two dogs each were missing a measurement for a pulse sequence (T1-weighted spin-echo in one case and trueFISP in the other), so the regression approach to repeated measures analysis of variance was used (17). Pairwise differences between pulse sequences were assessed by using the Tukey method of adjustment for multiple comparisons. All statistical tests were two tailed; a P value less than .05 was considered to be significant.



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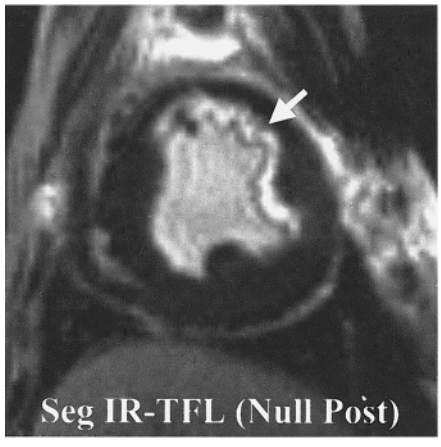


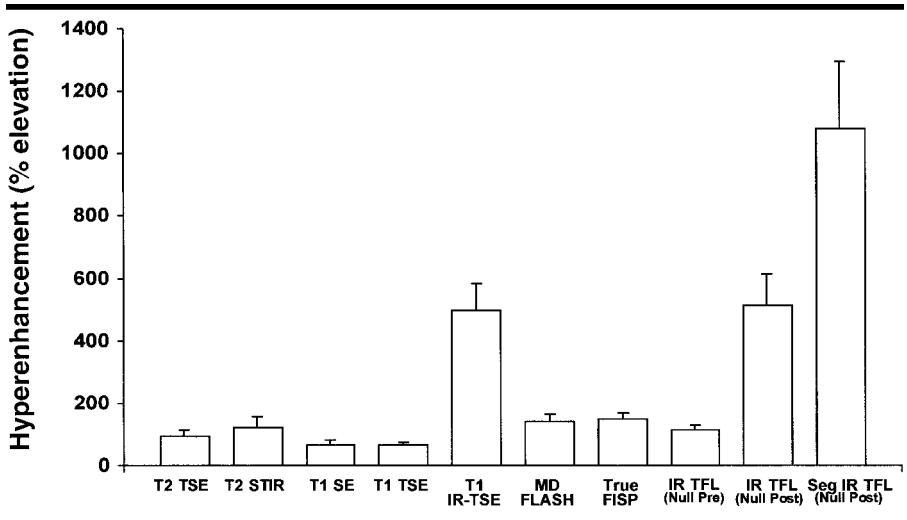
b.

Figure 3. Short-axis images obtained with all the MR techniques applied in one dog. The repetition times, echo times, and TIs used to perform all the sequences are listed in Table 1. **(a)** The T2-weighted turbo spin-echo (*TSE*) and T2-weighted short TI inversion-recovery (*STIR*) images were acquired before contrast agent administration. All techniques except T1-weighted spin-echo (*SE*) imaging were performed during breath holding. *IR-TFL* = inversion-recovery turboFLASH, *IR-TSE* = inversion-recovery turbo spin echo, *MD-FLASH* = magnetization-driven FLASH. **(b)** The segmented inversion-recovery turboFLASH (*Seg IR-TFL*) image demonstrates the highest contrast-to-noise ratio between the infarcted and normal myocardium; this was in agreement with the overall results. In **a** and **b**, the region of subendocardial infarction is indicated by the arrow on each image.

RESULTS

Animal Study

Figure 2 shows the effect of varying the TI delay on regional myocardial signal intensities at segmented turboFLASH imaging. When the TI delay was short (<150 msec), magnetization of blood in the left ventricular cavity and in the normal myocardial regions was below zero, and these regions had high signal intensity (magnitude images). The infarcted region had magnetization that approached zero and therefore low signal intensity. As the TI was increased (175–225 msec), the left ventricular cavity had magnetization of blood that approached zero and low signal intensity; the normal myocardium had magnetization of blood that remained below zero and high signal intensity; and the infarcted



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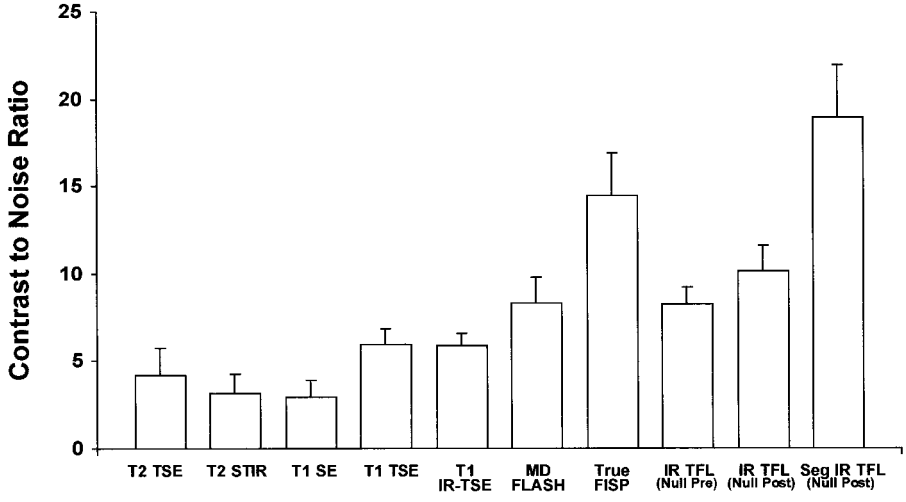

b.

Figure 4. Graph summarizes myocardial MR image signal intensities and contrast-to-noise ratios in all animals. In **a** and **b**, *IR-TSE* = inversion-recovery turbo spin echo, *MD-FLASH* = magnetization-driven FLASH, *SE* = spin echo, *Seg IR TFL* = segmented inversion-recovery turboFLASH, *STIR* = short-TI inversion recovery, *TSE* = turbo spin echo. **(a)** Percent signal intensity elevation in infarcted myocardial regions relative to that in normal regions. A value of 100% means the signal intensity of the infarcted region is twice that of the normal myocardium. **(b)** Contrast-to-noise ratios in high-signal-intensity versus normal regions. Note that the high signal intensity and contrast-to-noise ratio are substantially greater with segmented turboFLASH imaging than with any of the other techniques tested. *IR TFL* = inversion-recovery turboFLASH.

region had high signal intensity with a border region of low signal intensity. With the TI set to null the signal intensity of normal myocardium (275 msec), blood had high signal intensity; the normal myocardium, low signal intensity; and the infarcted region, high signal intensity. With longer TIs (>275 msec), the magnetization in all regions was above zero and differences between regions became less evident. With all TIs longer than the null point of normal myocardium, the size and shape of the high-signal-intensity region remained the same.

Figure 3 shows typical images obtained with all 10 pulse sequences performed in the same dog. Although the infarcted region could be detected on T2-weighted images before contrast material administration, the infarction was generally more evident on the postcontrast images. The standard spin-echo technique, in which images are acquired without breath holding, resulted in motion artifacts in all animals. The infarcted region was detected but not obvious at turbo spin-echo imaging with a preparatory inversion pulse. The turbo spin-echo sequence without an inversion pulse, magnetization-driven FLASH sequence, and trueFISP sequence all produced images on which the infarcted region could be detected and were of reasonable quality.

The single-shot inversion-recovery turboFLASH sequence was of particular interest (Fig 3). The last two of the nine small panels in Figure 3a (bottom, middle and right) underscore the importance of TI in the visualization of myocardial injury. The sequences used to acquire the images in the last two panels were identical except that the TI in the eighth panel (bottom middle) was set to null the signal intensity of normal myocardium before contrast agent administration, whereas that in the ninth panel (bottom right) was set to null the signal intensity of normal myocardium after contrast material administration. As seen in the ninth small panel (bottom right), this seemingly minor change dramatically increased regional differences in myocardial signal intensity. Selection of the TI that nulled the signal intensity of normal myocardium combined with segmentation of the k-space data across multiple cardiac cycles resulted in the segmented inversion-recovery turboFLASH image shown in the large panel (Fig 3b).

The graph in Figure 4a summarizes the signal intensity ratios for all images and sequences. The eighth and ninth bars again underscore the importance of TI selection. Overall, the sequences with preparatory inversion pulses produced greater regional differences in myocardial signal intensity. The highest regional differences were produced by using the segmented turboFLASH sequence (1,080% ± 214, $P < .05$ compared with values for all other sequences). The graph in Figure 4b summarizes the contrast-to-noise ratios. The segmented turboFLASH sequence produced the highest contrast-to-noise values ($P < .05$). In the six dogs studied, no correlation between infarct age and signal intensity was observed.

Patient Study

Figure 5 shows representative images obtained in six of the 18 patients with myocardial infarction. All 18 patients with myocardial infarction demonstrated high signal intensity at imaging. On average, the high-signal-intensity regions on the patient images had signal intensities that were 485% ± 43 higher than those of the normal myocardial regions.

DISCUSSION

The main finding of this study was that the segmented turboFLASH pulse sequence with a TI set to null normal myocardial signal intensity after contrast material administration produced the greatest differences in regional myocardial MR image signal intensity compared with the nine other MR imaging techniques tested. Because in vivo image quality ultimately limits the ability of MR images to depict physiologic information, the use of this sequence is expected to improve the capability of MR imaging to depict and delineate myocardial injury.

Several signal intensity ratios at in vivo imaging in dogs and humans that are reported in the literature are summarized in Table 2. In previous canine studies performed by using a variety of MR pulse sequences (Table 2), the reported signal intensities of infarcted myocardium were 70%–123% (mean, 86%) higher than those of normal myocardium. In our canine study, at segmented turboFLASH imaging with the TI set to null normal myocardial signal intensity after contrast material administration, the infarcted myocardium showed, on average, a 1,080% higher signal intensity than did the normal myocardium, which was a 10-fold improvement in contrast.

In previous human studies involving a variety of MR imaging methods (Table 2), the range of enhancement was 31%–103% (mean, 48%), whereas in the current study involving segmented turboFLASH imaging performed in 18 patients, we observed a mean enhancement of 485%, which again is a 10-fold improvement. The large difference in enhancement that we observed in dogs versus humans (1,080% vs 485%) was probably due to the difference in the contrast agent dose used (0.3 mmol/kg in dogs vs 0.2 mmol/kg in humans). Imaging in animal models under well-controlled circumstances often results in better image quality than does imaging in patients; this may also explain some of the differences in results between the animals and humans. In some cases, regions of infarction are intertwined with viable tissue and a signal of mixed intensity will result.

It should be pointed out that many of the previous studies involved T1-weighted spin-echo sequences at postcontrast imaging, which often produce severe respiratory motion artifacts. However, a number of recent publications in which breath-hold and single-shot imaging techniques were described also were included in our comparison (Table 2).

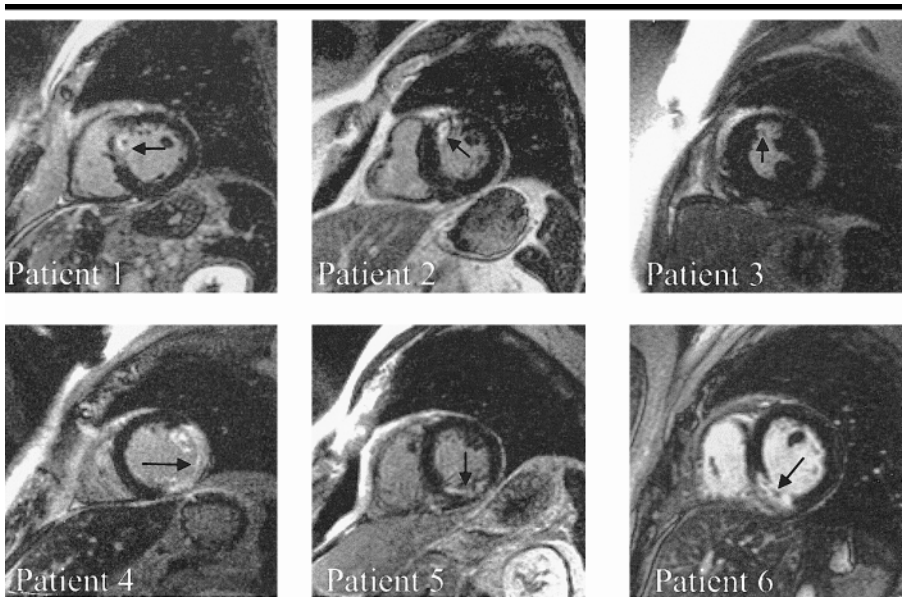

Figure 5. Short-axis images obtained in six patients by using the segmented turboFLASH sequence. The arrows point to the region of infarction. Note the high contrast between the infarcted (ie, high-signal-intensity) versus normal (ie, low-signal-intensity) myocardium.

The dramatic difference in image contrast between the contrast-enhanced segmented turboFLASH technique and the others tested in this study and used in previous studies may be due to a variety of factors. It has previously been reported that postcontrast T1-weighted imaging techniques are more sensitive for the detection and delineation of myocardial injury than T2-weighted noncontrast techniques (5,22). Our study results were in agreement with this finding. Many of the earlier studies of contrast-enhanced imaging of the myocardium were performed by using gated spin-echo imaging during free breathing. This technique often produces severe respiratory motion artifacts, which can mask the presence of high-signal-intensity areas. Later studies involved breath-hold techniques that substantially reduced respiratory motion artifacts.

In previously published descriptions of postcontrast inversion-recovery imaging of myocardial infarction, a TI to null normal myocardial signal intensity before the injection of the contrast agent was selected. This approach was tested in our study, and the resultant contrast was found to be inferior to that in which the TI was set to null normal myocardium after contrast agent administration. When the TI is set to null normal myocardium before contrast agent administration, both the normal and abnormal myocardium show signal intensity enhancement on postcontrast images, and the percent difference is reduced fivefold (510% vs 112%, respectively; eighth and ninth bars in Fig 4a).

The segmented breath-hold approach can provide images with higher resolution than can the single-shot approach. Breath holding results in insensitivity to respiratory motion. Only a fraction of the data lines are acquired within one cardiac cycle, so more lines can be acquired in total without corruption from cardiac motion. Single-shot acquisitions tend to drive the magnetization into steady state during the acquisition and thus nullify the effects of the inversion pulse. Segmentation of the acquisition can help generate image contrast that is highly dependent on T1 inversion recovery.

One potential pitfall of postcontrast inversion-recovery imaging is that, depending on the contrast agent dose and elapsed time since injection, the blood may have the same signal intensity as the infarcted myocardium. This may cause difficulty in differentiating subendocardial lesions from the intracavity blood pool. One practical solution to this problem is to view the postcontrast and cine images side by side to aid in defining the true wall thickness. It can also be helpful to wait longer for the contrast agent to wash out of the blood pool.

As demonstrated in Figure 2, the signal intensity ratio between normal and high-signal-intensity tissue is maximized when the signal intensity of normal myocardium is nulled. In principle, determining the ex-

act TI delay at which this occurs must be done iteratively for each patient. In practice, however, only one or two images typically need to be acquired, because with experience one can estimate the TI reasonably well on the basis of the patient's size, the amount of contrast agent administered, and the time after contrast agent administration. In our experience, a TI of 200–300 msec is typical for images acquired in patients 5–20 minutes after the administration of 0.1–0.2 mmol of gadoteridol per kilogram of body weight. It should be noted that subregions of low signal intensity may be observed within high-signal-intensity regions. These low-signal-intensity regions are believed to correspond to regions of microvascular obstruction (24,25,28).

The finding that the regional differences in signal intensity with segmented turboFLASH imaging are substantially greater than those with the other techniques raises the question of whether further improvements can be made. Further T1 shortening in infarcted regions might be achieved by developing a new MR imaging contrast agent or by using the highest practical contrast agent dose. The technique might also be improved by using faster gradient technology. Currently, single-section acquisitions are performed in six to 10 breath holds to cover the heart. Faster gradient hardware permits the acquisition of a three-dimensional volume of data within a single breath hold.

Our group has already implemented and begun evaluating a three-dimensional pulse sequence that is similar to the described two-dimensional segmented turboFLASH sequence (Fig 1a) except that the receiver bandwidth is higher, the repetition time is shorter, and three-dimensional image encoding is used. Our initial observations of similar contrast enhancement patterns with the three-dimensional technique suggest that further reductions in image acquisition times and/or improvements in image quality can be expected as gradient performance improves.

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