

Comparison of Delayed Contrast-Enhanced Viability Imaging at 1.5T and 3T: Initial Experience

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Introduction: The improved signal-to-noise ratio (SNR) at 3T compared to 1.5T has been observed in cardiac MRI applications [1]. This benefit can be used to improve image resolution and enhance the visualization of small pathologies. For the assessment of myocardial viability, increased visualization can be a particular advantage, since quantification of infarct size has a direct correlation with patient prognosis [2]. However, delayed enhancement is not only reliant on SNR and resolution, but also contrast-to-noise ratio (CNR) of the enhanced infarct tissue (MI) and remote normal myocardium and/or the left-ventricular (LV) blood pool. Since the T1 relaxation time and T2* effects increase at 3T, question remains whether these trade-offs will compromise the image signal and contrast of delayed enhancement imaging at 3T.

Purpose: The purpose of this preliminary investigation was to measure the SNR and CNR in the delayed enhancement images of MI at 1.5T and 3T. Our goal was to quantify the mean SNR of infarct tissue, the CNR between infarct and normal myocardium, and the relative signal contrast between infarct and blood. We hypothesize that despite potential trade-offs at 3T, the SNR and CNR will be greater at 3T than 1.5T.

Methods: Six individuals with known myocardial infarction were recruited for this study. The protocol was approved by the university's internal review board and all subjects gave written consent prior to the study. Each subject underwent two MRI exams: the first exam was performed at 1.5T (Philips Intera, Best, The Netherlands) to confirm the presence and location of the MI, and the second exam was performed at 3T (Philips Intera or Siemens Trio, Erlangen, Germany). Exams were separated by at least three days. Following localization of the short-axis plane, 0.2mmol/kg Gd-DTPA-BMA (Omniscan, Amersham, Oslo, Norway) was administered intravenously. Acquisition of delayed enhancement images began 10 minutes post-contrast and continued for approximately 25 minutes. The protocol was a segmented inversion recovery (IR) spoiled gradient echo (2D-FLASH) sequence with a 350mm field of view (FOV), 256 matrix (75-100% phase encode acquisitions), TR/TE/ $\alpha = 5\text{ms}/2\text{ms}/20^\circ$, 10mm slice thickness, 16 lines/segment, 2 heartbeats/segment, inversion time (TI) = 250-300ms, and 225 Hz/pixel bandwidth. The pulse sequence parameters were kept as close as possible at 3T in light of SAR constraints and differences in gradient performance. The only significant 3T changes were TR/TE = 4.6ms/2.3ms, 448 Hz/pixel bandwidth, and TI = 250-310ms. Region-of-interest (ROI) measurements of the mean signal intensity were made in the LV blood pool, enhanced infarcted tissue, normal myocardium (adjacent and remote to MI), and background noise. SNR was quantified as the mean signal value divided by the background standard deviation (SD), which can be approximated in phased array receiver systems from the mean value of the background (air) using previous methods [3]. CNR was measured as the SNR difference between two tissues, while the relative signal contrast (RSC) was the ratio of infarct-to-blood signal strengths.

Results: Two subjects did not complete the 3T examination, leaving 4 subjects with SNR and CNR measurements. An example of delayed enhancement imaging at 1.5T and 3T is shown in Figure 1. There is improved visualization of the inferior MI at 3T and distinction with the blood pool. However, this trend was not evident in all subjects. In one particular subject, MI enhancement was much greater at 1.5T, possibly due to a very high contrast agent concentration, which may have induced significant T2* dephasing at 3T. However, on average, the mean SNR of infarct tissue was higher at 3T (39.0 ± 14.6 vs. 28.4 ± 13.3), along with CNR between infarct and normal myocardium (33.3 ± 13.9 vs. 24.3 ± 11.4) (Figure 2). The mean infarct-to-blood RSC also favored 3T (1.63 vs. 1.38), possibly enabling distinction of small subendocardial infarcts adjacent to the blood pool. These results were significant ($p < 0.05$) in all but one subject, as mentioned above.

Conclusions: This preliminary investigation of delayed enhancement imaging at 3T reveals an average increase in infarct SNR and CNR relative to 1.5T in the same subjects and dose. This study suggests that 3T may provide additional benefits of improved resolution and infarct-to-blood contrast for the visualization of small subendocardial infarcts.

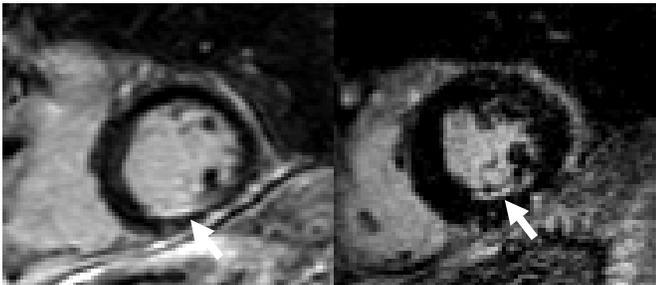


Figure 1. Comparison of delayed enhancement imaging at 1.5T (left) and 3T (right) in one subject (20 min post-contrast). The MI is indicated by the arrow.

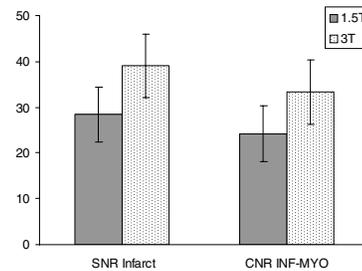


Figure 2. Signal and image contrast of infarct tissue measured at 1.5T and 3T

References:

1. Noeske R, et al. MRM 2000;44:978-982
2. Kim RJ, et al NEJM 2000;343:1445-53
3. Constantinides CD, et al. MRM 1997;38:852-57.