

A Comparison of Delayed Contrast Enhanced and T1rho MRI for Assessment of LV Remodeling

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Introduction

Left ventricular remodeling (LV) results in long-term changes to the size, thickness and geometry of the heart, which adversely affects cardiac output and patient prognosis (1,2). Two conventional MRI methods for detection of long-term infarct changes are T2-weighted imaging and delayed, contrast enhanced (DCE-MRI) perfusion imaging, however, the latter is associated with additional costs and can potentially compromise patient safety and comfort (3,4). It was recently observed *ex vivo* that a spin locking pulse, during which spins undergo T1ρ relaxation, can overcome low frequency relaxation mechanisms to improve endogenous contrast between the infarct and perfused, healthy myocardium compared to T2 relaxation-based contrast (5). We hypothesize that high, endogenous contrast can be achieved by spin locking *in vivo* at 3 T and that the contrast-to-noise (CNR) ratio is comparable to DCE. To test this hypothesis, we measured T1ρ relaxation and DCE CNR in a swine model of myocardial infarction (MI).

Methods

Animal Model. 5 Yorkshire swine weighing approximately 50 kg were used in a study approved by the Institutional Animal Care and Use Committee at the University of Pennsylvania. The animal was sedated with IM ketamine (25 mg/kg) and prepared for surgery performed similarly to a previously described ovine model (6). A left thoracotomy was performed, the pericardial sac was opened and the left anterior descending artery was ligated, two-thirds the distal distance, to produce apical infarcts. Four weeks following surgically-induced MI, a pressure transducer (Millar Instruments, Houston TX) was guided into the left ventricle for cardiac gating and the animal was transported to an MRI scanner and underwent cardiac imaging.

In Vivo MRI. MRI was performed on a 3 T imaging system (Tim Trio Model; Siemens Healthcare; Erlangen, Germany), equipped with a nominal 40 mT/m gradient system and a combined spine and body RF receive array with 8 channels. A T1ρ-prepared, centrically segmented, multiecho, gradient echo sequence with Cartesian readout was used to acquire T1ρ-weighted images during systole. The parameters used for acquisition were as follows: bandwidth/pixel = 400 Hz, TE/TR = 3.04/6.54 ms, slice thickness = 8 mm, resolution = 1.56 mm², matrix = 256², FOV = 300 mm², flip angle = 12 degrees, 12 shots, TSL = 6-48 ms in 6 ms increments, scan time = 4.9 min, ν_1 = 450 Hz, the delay between the QRS complex and the heart was approximately 190 ms. DCE MRI was performed with the following imaging parameters: TI=500-540 ms (estimated using a TI scout), 0.14 ml/kg contrast agent (Multihance, Bracco, Inc.), FOV = 300 mm², matrix = 256², TE/TR = 3.04/1000 ms, BW = 399 Hz/pixel, flip angle = 25 degrees.

Relaxation Mapping and Statistical Analysis. T1ρ relaxation times were calculated by pixelwise L2 norm minimization of linearized signal decay equation $S = S_0 e^{-TSL/T1\rho}$. Relaxation times were estimated by region-of-interest analysis of the infarct, myocardium immediately adjacent to the infarct (borderzone), and functional, remote myocardium. Two-way ANOVA was performed in PASW Statistics 18 (SPSS, Inc., 2009, Chicago) at a significance level of $p = 0.05$.

Results

T1ρ relaxation times in the infarct region (T1ρ = 67.5 ms) were significantly different from the remote myocardium (T1ρ = 44.8 ms, $p < 0.05$). Mean signal decay across all six animals is shown in Fig. 1 and all three regions fit a single exponential model ($R^2 > 0.97$). T1-weighted DCE images are shown adjacent to T1ρ-weighted images and relaxation maps for a single swine in Fig. 2. Artifacts associated with significant field heterogeneity during the spin locking pulse were observed in the left ventricular free wall, but not in the septum, where quantification of the infarct relaxation times was performed. T1ρ CNR between infarct and remote regions was 30 ± 2.2 (TSL > 24 ms) and in DCE MRI was 31 ± 2.1 .

Discussion

Contrast between infarct and healthy myocardium in T1ρ-weighted images (TSL > 24 ms) had comparable or superior contrast to images obtained using T1-weighted DCE imaging. These results suggest that T1ρ may be a viable alternative to DCE MRI in healthy subject studies or for patients in whom contrast is not a possibility. T1ρ relaxation times were previously reported in a swine model of myocardial infarction *in vivo* at 3T and *ex vivo* at 7 T (5). At 7 T, relaxation times were measured over a wide range of spin lock amplitudes (ν_1 = 500-2500 Hz) and relaxation time differences between the remote and infarct regions varied from 74.3 ms (ν_1 = 500 Hz) to 137.1 ms (ν_1 = 2500 Hz). Differences in relaxation times between the two studies are attributed to several factors: (1) differential effects of chemical exchange broadening of relaxation times at higher magnetic field strength, (2) changes in the spin-spin relaxation rate, among other mechanisms of low-frequency relaxation dispersion. *In vivo* relaxation times were previously reported for the infarct region (T1ρ = 93.3 ms) and remote myocardium (T1ρ = 49.9 ms), which are comparable to those here, but these measurements were only performed in 3 swine with infarctions in the LV free wall, which is highly sensitive to heterogeneity of the B0 field. Visual inspection of T1ρ-weighted and DCE images revealed small differences in infarct size associated with origin of contrast in the two methods. DCE MRI is suspected to originate from late uptake and slow release kinetics of the infarct region to contrast agent, whereas T1ρ MRI is most likely sensitive to differences between water ¹H nuclear relaxation associated with collagen in the infarct or, conversely, cellular material in the remote region. The predominant differences between the two techniques may occur in the borderzone. Studies are ongoing to measure relaxation times across a range of periods following MI to monitor changes to the biochemistry of the infarct zone.

References: (1) Gheorghiade, et al. Circulation (1998); 97:282-89 (2) AHA Statistical Update. Circulation (2008);117. (3) Grobner, et al. Nephrol Dial Transplant (2006). (4) Burns M. The U.S. Market for Medical Imaging Contrast Media (2006). (5) Witschey, et al. Magn Reson Med. (2010).

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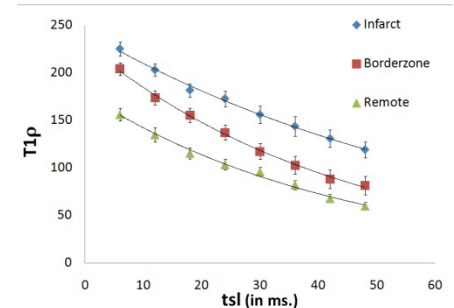


Figure 1: Decay of the magnetization during the spin locking pulse (TSL) measured from the infarct, borderzone and remote regions in swine.

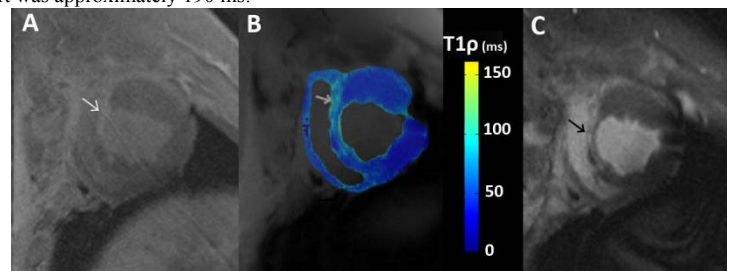


Figure 2: LV DCE MRI (A), T1ρ relaxation map (B) and T1ρ-weighted image (TSL = 48 ms) in the septal wall. There are spin lock stop band artifacts associated with field heterogeneity on the free wall.