On the Mechanism of Myocardial Edema Contrast in SSFP Imaging

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Introduction: Recent studies have demonstrated that conventional balanced steady-state free precession (bSSFP) cine imaging can identify regions of myocardial edema in-and-around acute myocardial infarctions (AMI) [1,2]. However, the underlying mechanisms of bSSFP edema contrast are not well understood. A more detailed understanding of the contrast mechanisms at play may enable opportunities for optimization of bSSFP-based edema contrast. The purpose of this study is to investigate the mechanisms contributing to the b-SSFP-based edema contrast surrounding AMI in conventional cine bSSFP images.

Methods: Animal Preparation & Imaging: Dogs (n=4) subjected to ischemia-reperfusion injury (left anterior descending artery (LAD) occlusion for 3 hours followed by reperfusion) were studied at baseline (pre-injury), 2-hours post-reperfusion (day 0), and on days 2, 5, and 7. Multiple breath-held whole heart short-axis cine bSSFP images, and T1- and T2-maps were acquired at late diastole using a Siemens Espree (1.5T) system. All studies were terminated with PSIR late-gadolinium-enhancement (LGE) acquisitions to confirm LAD infarction. T1 and T2 maps were generated from images scanned by IR-SSFP with different TI (11 TIs raging from 80ms to 4000ms) and T2-prepared-SSFP with different T2-prep time (0, 24 and 55ms). Cine SSFP imaging was performed with TR/TE =3.5/1.75 ms; flip angle=70^o; 20-25 phases; resolution=1.25x1.25x8.0mm^3; BW = 930 Hz/pixel. Data Analysis: The cardiac phase from the cine bSSFP images corresponding to the T1 and T2 maps were identified on the basis of trigger times. On the bSSFP images and the relaxation maps, the edematous territories were identified as regions with pixel values that are 2 standard deviation greater than the mean value of the remote (healthy) territories. Using the Freeman-Hill equation for bSSFP signal and the measured signal intensity, T1 and T2 values for edematous and healthy territories, the relative contributions from relaxation and thermal magnetization (M_0) effects were estimated. Relaxation effect was calculated as R, where $R=(1-E_1)\sin\alpha/[1-(E_1-E_2)\cos\alpha-E_1E_2]$, and $E_{1,2}=\exp(-TR/T_{1,2})$. Thus bSSFP signal intensity was estimated as $S=M_0 \cdot R$. Edema contrast from signal intensity differences between edematous and healthy contrast was computes as $\Delta S = (E - H)/H$, where E and H are the mean signal intensities of the edematous and healthy territories, respectively. The relaxation and M_0 contributions were independently averaged across all imaging slices positive for edema and over all studies. A one-tailed t-test was used to test whether the mean independent contributions from M_0 and R are greater than zero. A paired t-test was used to test whether the contributions from relaxation and M_0 effects were different. Statistical significance was set at $p<0.05$.

Results: Semi-automated and visual analysis did not identify edematous territories in relaxation maps, bSSFP or LGE images acquired under baseline conditions in any of the animals. LAD territories were readily identified on relaxation maps, and cine bSSFP images as hyperintense regions following reperfusion in all dogs and the presence of AMI within the same territories was confirmed by LGE images (Figure 1). Mean relative contributions from relaxation and M_0 effects, assessed over each imaging slice (across all studies), are shown in Figure 2. One-tailed t-test confirmed that relaxation effects and M_0 were significantly greater than zero. Paired t-test showed that the contribution from M_0 was greater than the contributions from the relaxation effects ($p<0.05$). Across all studies, the mean signal contrast between edematous and healthy territories was 56.4±14.7%, and the theoretically estimated relaxation- and M_0-based contrast were 18.7±9.5% and 32.7±19.0%.

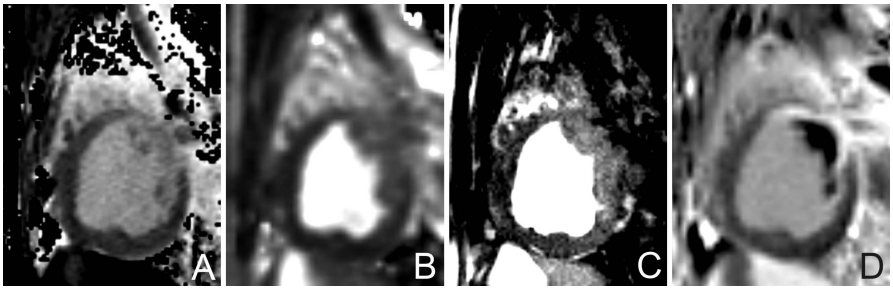

Figure 1. Representative short-axis images obtained from a dog subject to ischemia-reperfusion injury in the LAD territory (day 0, 2-hours post reperfusion). **A**: T1 map; **B**: T2 map; **C**: bSSFP; **D**: PSIR LGE phase image. Note that the LAD territory appears hyperintense in T1 and T2 maps, as well as in the bSSFP image. **D** confirms the presence of a LAD infarct.

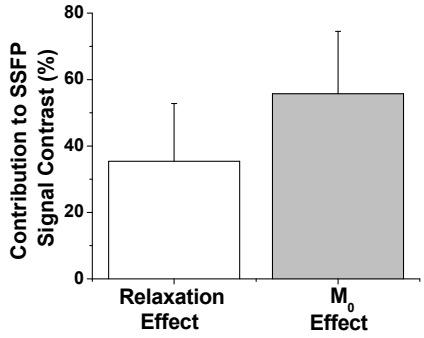

Figure 2. Mean, estimated contributions from relaxation and M_0 effects to myocardial edema contrast in bSSFP images. Note that in addition to the relaxation effects, a substantial fraction of the edema contrast originates from M_0 effects, likely mediated through proton density change and/or magnetization transfer [3] mechanisms.

Conclusions: Relaxation effects alone cannot explain the observed myocardial edema contrast in bSSFP images. Results show that M_0 effects, likely from proton density difference between healthy and edematous territories, and/or magnetization transfer changes within edematous and healthy myocardial territories [3] have a substantial contribution to the edema against myocardium contrast in bSSFP images. Acquisition strategies that wish to maximize myocardial edema contrast in bSSFP imaging should take both relaxation and M_0 mediated effects into account.

References: [1] Dharmakumar R *et al* ISMRM 2008; [2] Kumar A *et al* ISMRM 2008; [3] Weber OM *et al* MRM 2009.