Journal of Cardiovascular Magnetic Resonance

BioMed Central

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Why do we see myocardial edema in acute myocardial infarcts with balanced SSFP imaging?

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from 13th Annual SCMR Scientific Sessions
Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, **12**(Suppl 1):M12 doi:10.1186/1532-429X-12-S1-M12

This abstract is available from: http://jcmr-online.com/content/12/S1/M12

Introduction

Recent studies have demonstrated that conventional balanced steady-state free precession (b-SSFP) cine imaging can identify regions of myocardial edema in-and-around acute myocardial infarctions (AMI). However, the underlying mechanisms of b-SSFP edema contrast are not well understood. A more detailed understanding of the contrast mechanisms at play may enable opportunities for optimization of SSFP-based edema contrast.

Purpose

To investigate the mechanisms contributing to the b-SSFP-based edema contrast surrounding AMI in conventional cine b-SSFP images

Methods

Dogs (n = 3) subjected to ischemia-reperfusion injury (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 short-axis cine b-SSFP images, and T2- and T1-maps were acquired at late diastole using a Siemens Espree (1.5 T) system. All studies were terminated with PSIR late-gadolinium-enhancement (LGE) acquisitions to confirm LAD infarction. Cine SSFP imaging was performed with TR/TE = 3.5/1.75 ms; flip angle = 70°; 20-25 phases. The cardiac phase corresponding to the T1 and T2 maps were identified from the cine b-SSFP images. On these SSFP images, and the relaxation maps, a semi-automated approach was used to identify the edematous territory. Using the Freeman-Hill equation for b-SSFP and the measured signal, T1 and T2 values for edematous and healthy territories, the relative contributions from relaxation and thermal magnetization (M_0) effects were estimated.

Results

Semi-automated and visual analysis did not identify regions of hyperenhancement in T1 and T2 maps, or SSFP or LGE images acquired under baseline conditions in any of the animals. Hyperintense LAD territories were readily identified on T1, T2, and cine SSFP images on days 0, 2, 5, and 7 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. For all studies, signal contrast between edematous and healthy territories was 56.4 ± 14.7%. From the measured T1 and T2 maps, the theoretically expected edema contrast due to relaxation changes was 18.7 ± 9.5%.

Conclusion

Relaxation effects alone cannot explain the observed myocardial edema contrast in b-SSFP images. Results show

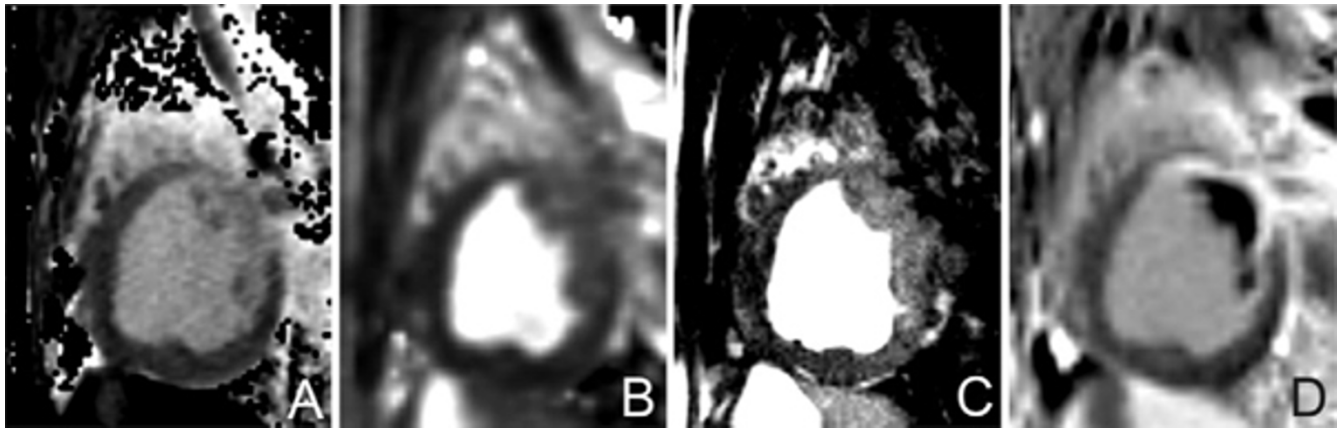

Figure 1
Representative short-axis images obtained from a dog subject to ischemia-reperfusion injury in the LAD territory (day 0, 2-hours post reperfusion). **A:** T1 map; **B**: T2 map; **C**: b-SSFP; **D**: PSIR LGE phase image. Note that the LAD territory appears hyperintense in T1 and T2 maps, as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

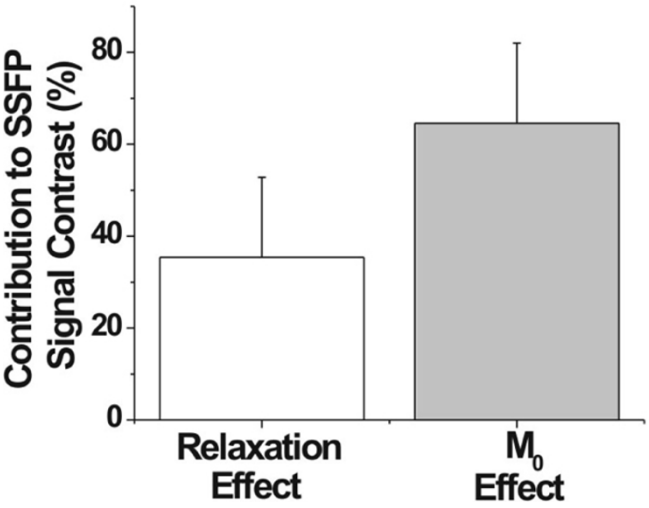

Figure 2
Mean, estimated contributions from relaxation and M_0 effects to myocardial edema contrast in b-SSFP images. Note that in addition to the relaxation effects, a substantial fraction of the edema contrast originates from M_0 effects, likely meditated through proton density and/or magnetization transfer mechanisms.

that M_0 effects, likely from proton density and/or magnetization transfer changes between healthy and edematous territories, have a substantial contribution to the b-SSFP image contrast. Optimization strategies that wish to maximize myocardial edema contrast in b-SSFP imaging should take relaxation and M_0 effects into account.