

AbstractID: 7501 Title: Monitoring Myocardial Infarction Induced Calcium Homeostasis Alteration by MRI in a Small Murine Model

Purpose:

Alterations in myocyte calcium regulation for both the mechanical dysfunction and the arrhythmogenesis associated with congestive heart failure. In spite of the established importance calcium regulation in the heart both prior to, and following, myocardial injury, monitoring strategies to assess calcium homeostasis in affected cardiac tissues are extremely limited. We propose to characterize the dynamic and temporal features of calcium responses due to myocardial injury in a small murine model using Mn^{2+} as a contrast agent.

Method and Materials:

There are 3 groups of mice (6-10 weeks) namely control, sham-operated, and myocardial infarction (MI). In the MI studies, permanent myocardial infarcts were produced by ligating the left anterior descending coronary artery. Images were acquired on a horizontal 7.0 T Bruker BioSpec MRI spectrometer equipped with a micro imaging gradient. A series of short-axis T_1 -weighted cardiac images were acquired as well as pre- Mn^{2+} and post- Mn^{2+} infusion T_1 maps using an ECG-gated, flow-compensated Lock-Locker MRI pulse sequence.

Results:

ECG gated cardiac MRI provided high quality images for left-ventricle, and the infusion of Mn^{2+} clearly showed a large change in T_1 values. The left-ventricular post- Mn^{2+} relaxivity, ΔR_1 ($=1/\Delta T_1$), thus far for control, sham-operated, and MI groups are 3.54 ± 0.94 , 2.63 ± 1.37 , and 1.91 sec^{-1} , respectively. The post-MI group showed potentially lower ΔR_1 values. Increased sample size for each animal group is warranted. Further investigation is necessary to determine if Mn^{2+} could provide insights into the temporal myocardial remodeling process where Ca^{2+} influx might be altered.

Conclusion:

One motivation for this study is that myocardial injury causes physiological remodeling leading to potential Ca^{2+} handling alteration. This process can be potentially monitored with a cardiac manganese-enhanced T_1 mapping technique. Furthermore, changes in ΔR_1 could potentially be calibrated to the absolute manganese content for left-ventricular myocardium.