Preliminary Application of in vivo Cardiac Diffusion Weighted MRI at 3T in Chronic Myocardial Infarction Porcine Model

Christopher Nguyen[1,2], Yibin Xie[1,2], Zhaoyang Fan[1], Behzad Sharif[1], James Dawkins[3], Eleni Tseliou[3], Xiaoming Bi[4], Rohan Dharmakumar[1], Eduardo Marban[3], and Debiao Li[1,2]

[1]Biomedical Imaging Research Institute, Cedars-Sinai Medical Center, Los Angeles, CA, United States, [2]Bioengineering, University of California Los Angeles, Los Angeles, CA, United States, [3]Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, United States, [4]MR R&D, Siemens Healthcare, Los Angeles, CA, United States

Target Audience: MR engineers, physicists, and clinicians (cardiologists and radiologists) interested in cardiac diffusion weighted MRI.

Introduction: Cardiac diffusion-weighted MRI is a non-contrast technique that has the potential to identify changes in tissue microstructure in acute myocardial infarction (MI) in humans and rats[1, 2]. The trace apparent diffusion coefficient (trADC) was significantly increased in the infarcted region relative to remote regions. This increase in trADC is attributed to an increase in extracellular space following cell death, where restriction in water diffusion is less. The aim of this study was to perform in vivo cardiac diffusion-weighted MRI in a chronic MI porcine model to see if this increased trADC chronically persists and correlate it with scar tissue delineated by late gadolinium enhancement (LGE) imaging.

Methods: Four chronic MI (8 weeks post MI induced by completely occluded proximal LAD) mini pigs were scanned on a 3T MR system (Siemens Verio). Before contrast was administered, M1M2 motion compensated[3] diffusion-prepared 2D segmented TSE was performed to derive trADC maps (TR/TE=3RR/8.4ms, α=180°, 2.1x2.1x6mm^3, 6 shots, 3 slices, diffusion TE_{prep}=105ms, b=400s/mm^2, G_{diff}=43mT/m, three orthogonal DW directions). Diffusion preparation was applied in the most quiescent phase identified by CINE imaging (TR/TE=3.4/1.6, α=50°, 35 cardiac phases). Contrast LGE GRE imaging was performed shortly after at the same slice locations roughly 15 min post contrast injection (TR/TE/TI=326/1.47/300ms, α=20°, 1.3x1.3x6mm^3). The lateral wall of the most basal slice (furthest from the LAD occlusion) was designated as the unaffected (remote) region. The region of infarction was determined by elevated trADC and signal intensity in LGE (both required $\mu_{infarct} > \mu_{remote} + 5\sigma_{remote}$). For each pig, the mean trADC's calculated for the infarct and remote regions. The infarct area determined by elevated trADC was compared to the LGE infarct area. Differences in means were tested for significance with a two tailed paired t-test. Cardiac phase mismatch between trADC maps and LGE images was corrected using a nonrigid registration, whose transform was derived from two phases of CINE closest to the trADC map and DE.

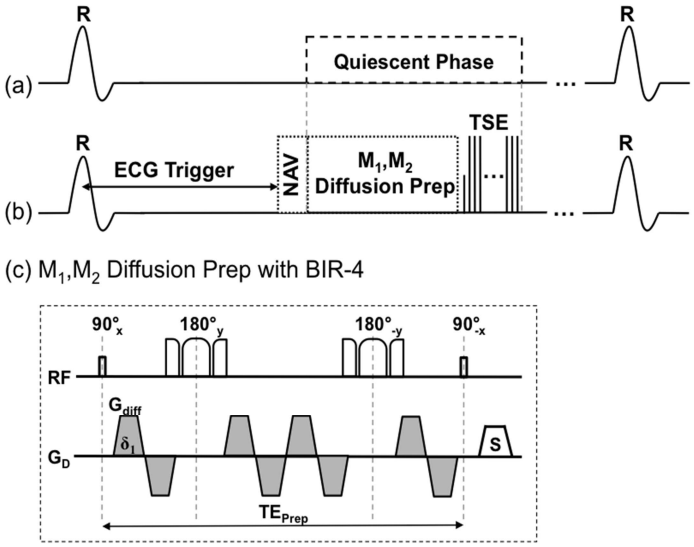

Figure 1 – Pulse sequence diagram. (a) The longest quiescent period is found using CINE imaging and (b) the motion compensated diffusion prep and segmented TSE readout is placed during this period. In the case the quiescent period is too short to fit both the prep and the readout, the diffusion prep was prioritized and the number of shots were increased. The bulk motion compensated diffusion prep (c) is m1 and m2 compensated and a non-selective adiabatic BIR-4 was used. The magnetization was allowed to recover for 2 RR after the TSE acquisition.

Results: M1M2 diffusion-prepared TSE scans resulted in mean infarct trADC values of 2.4±0.2x10^{-3} mm^2/s and mean remote trADC values of 1.4±0.4x10^{-3} mm^2/s ($p<0.05$). The location of elevated trADC infarct region agreed well with the LGE infarct area. The mean trADC and mean LGE infarct area was 0.9±0.1cm^2 and 0.8±0.1cm^2, respectively ($p = 1$). In the infarct regions, wall motion was akinetic.

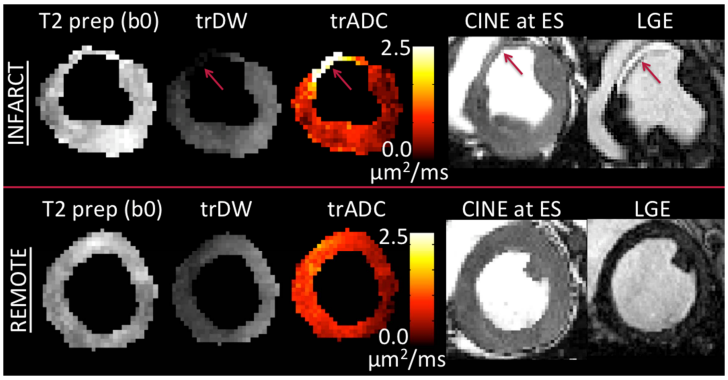

Figure 2 – T2 prep (b0), trDW, trADC, CINE at end systole, and LGE of two slices that contained the infarct and remote regions. TrDW and trADC of the remote and the T2 prep of the infarct illustrated no statistically significant changes. In the infarct slice, the trace DW showed a sharp decrease while the trADC depicted an increase (red arrows). The LGE image is hyperintense and the CINE image shows a thin wall that is akinetic. Note the sharp decrease in trace DWI demonstrates that bulk motion corruption is minimal since the myocardium is akinectic in the infarcted region.

Discussion: The chronic MI pig model is a great environment to test the resilience of bulk motion effects of an *in vivo* cardiac diffusion MRI technique. Because the infarcted region is akinetic relative to remote regions, bulk motion corruption of a diffusion technique would result in a decrease in trADC, which is the exact opposite of the expected increase in trADC for infarcted tissue. This lends confidence in our proposed technique in delineating infarct regions with elevated trADC, which was found to correlate well with LGE-defined infarct regions matching both area size and location. Furthermore, the roughly 50% increase in trADC values found in the infarct region relative to the remote region was statistically significant. However, further histological studies and larger sample size are needed to confirm the origin of the increase in trADC and its relationship with infarcted tissue.

Conclusion: In this preliminary study, we demonstrated that M1M2 compensated cardiac diffusion-prepared TSE was able to reveal statistically significant increases (~50%) in mean trADC in chronic MI regions. The agreement in spatial correlation and lesion area with hyperintense regions of LGE images suggests that the proposed technique was able to characterize infarct tissue. This may potentially allow for non-contrast tissue characterization of chronic MI.

References: [1] Wu, et al. Circ (2006). [2] Chen, et al. Am J Physio Heart Circ Physio (2003) [3] Nguyen, et al. MRM (2013)