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Whole-heart T2-weighted (T2w) sequence for imaging post-infarct edematous area at risk (AR) in mice

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Introduction

T2w cardiac magnetic resonance (CMR) defines the area at risk (AR) in large mammals by imaging post myocardial infarct (MI) edema. Here, we expand the T2w CMR to cover the entire left ventricle (LV) in mice and jointly applied late gadolinium enhanced (LGE) CMR to non-invasively define MI size as percent AR. Fast murine heart rate, flow and motion present challenges to T2w CMR success. The typical 60 ms echo time for edema contrast occupies a large portion of the murine cardiac cycle which prohibits traditional T2w methods.

Purpose

Develop a whole-heart, multi-slice, T2w CMR sequence for mice with high immunity to flow and tissue motion artifacts, has dark-blood suppression and maintains sufficient signal-to-noise (SNR) and contrast-to-noise (CNR) performance.

Methods

Our non-selective T2prep sequence employed a Malcolm Levitt-weighted (MLEV-14) composite $C180_x$ (90_x:180_y:90_x) refocus pulse train followed by a multi-slice, gradient-echo readout sequence. Flow-sensitization gradients were placed optimally during the T2prep to provide dark-blood capability. We imaged three mice on Days 1 thru 4 (D1-4) after reperfused MI induced by 60-minute coronary occlusion. Parameters included TR = 3 sec, TE = 60 ms, FOV = 25 × 25 mm, slice thickness = 1 mm, matrix = 128 × 128, BW = 520 Hz/pixel. LGE CMR was also performed on D1 to define MI size after T2w CMR was completed. Scans were performed on a Bruker 7 T ClinScan that covered the entire LV in 6 or 7 contiguous short axis slices.

Results

See figure 1. Panels A-D compare same-slice long and short axis sets of LGE MI and T2w AR with good concurrence. Panels B and D show the effective dark-blood results of the flow sensitization gradients and low occurrence of flow and motion artifacts. Panel E compares D1 MI size to D1-4 AR size as percent LV mass, with all days AR size significantly larger than D1 MI size ($p < 0.01$, ANOVA) and no significant difference in AR size from D1 through D4. Mean MI size was 34 ± 0.3 percent LV mass and 75 ± 1.2 percent AR on D1 (mean ± SEM). T2w SNR was 68 ± 4 and CNR measured between remote and AR regions was 34 ± 2.

Conclusion

T2w CMR in mice yields results consistent with those in larger mammals. T2w CMR can now be applied in knock-out mice to study the influence of individual genes and treatments on MI size measured as percent area at risk.

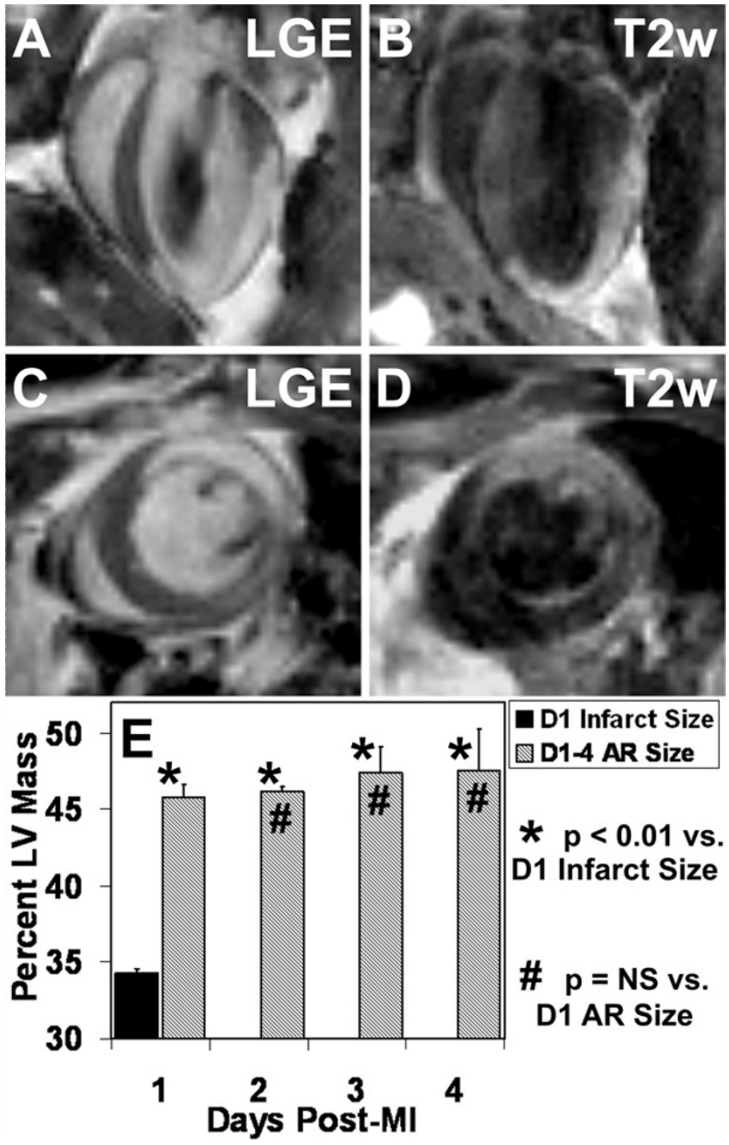

Figure 1