Early Passive Ventricular Restraint Post-MI Normalizes Borderzone Wall Thickening Dynamics as Assessed by Tissue Tagged MRI

J. J. Pilla[1], A. S. Blom[1], J. Affuso[1], J. H. Gorman III[1], M. A. Acker[1], R. C. Gorman[1]

[1]University of Pennsylvania, Philadelphia, PA, United States

Introduction: Borderzone myocardium has been classified as a unique form of dysfunctional myocardium adjacent to an infarct that is normally perfused but hypocontractile. The post-infarction remodeling process causes this borderzone region of hypocontractility to extend to involve progressively more normally functioning myocardium, resulting in ventricular dilatation and CHF (1,2). This study hypothesizes that borderzone wall thickening and wall thickening dynamics would be impaired as a result of infarction induced remodeling and that early post-MI ventricular restraint would normalize these parameters of regional function.

Methods: We used an ovine anterobasal infarct model (24% LV mass) and high temporal resolution tissue tagged MRI to characterize wall thickening and wall thickening dynamics in normal myocardium and borderzone myocardium 8 weeks after infarction in an untreated control group and in a group that had complete heart wrapping with the Acorn CSD® 1 week post-infarction. High-temporal resolution tissue-tagged short axis images were obtained using a 1.5T whole body scanner (Signa, GE Medical Systems) with the following parameters. FOV 24cm x 24cm, TR/TE 8ms/3ms, Slice thickness 6mm skip 0mm, 256 x128 matrix, 5mm tag spacing, ±64kHz bandwidth, 1 VPS, flip angle 15°, NEX 2. Borderzone region was defined as the area adjacent to the infarct within a 20° arc (figure 1) and dynamic wall thickening was calculated from the λ_1 strain within this region. Systole was divided into three periods: isovolumic contraction (IVC), early ejection (EE) and late ejection (LE). BZ wall thickening was assessed for each period (and normalized to end diastole) in uninfarcted animals, untreated infarct animals and animals having complete heart wrapping one week after MI.

Results: End-systolic BZ wall thickening was impaired after 8 weeks of remodeling (0.075) and was improved by ventricular restraint (0.11) when compared to normal (0.125) (figure 2). Wall thickening increased linearly during systole in uninfarcted animals (IVC=0.05, EE=0.09, LE=0.125). In control animals wall thickening only occurred during IVC (0.075) with the wall thinning through EE (0.065) and LE (0.05). In wrapped animals wall thickening normalized during IVC (1.1), remained constant during EE and dramatically improved during LE (0.11).

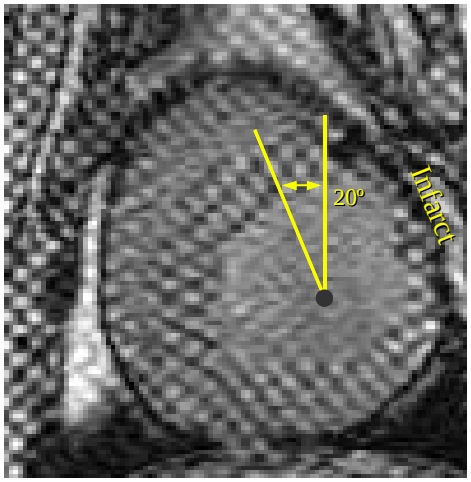

Figure 1. Tissue –tagged LV short axis image depicting borderzone as an arc of 20° adjacent to the infarct region

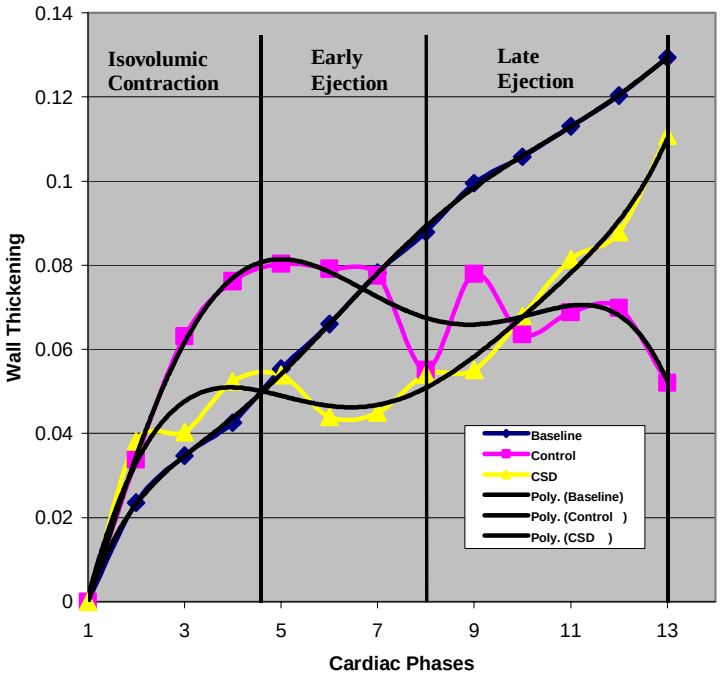

Figure 2. Dynamic Myocardial wall thickening through the cardiac cycle for baseline, control and CSD. CSD normalizes wall thickening compared to baseline while the control experienced thinning.

Conclusion: Early post myocardial infarct ventricular restraint improves borderzone wall thickening and wall thickening dynamics during remodeling as assessed by high temporal resolution tissue-tagged MRI. This may be attributed an "offloading" of borderzone stress by the CSD resulting in an improvement in function of this region.

References

1. Jackson et al. *J Am Coll Cardiol* 40: 1160–1167, 2002.
2. Narula J et al. *J Am Coll Cardiol* 36: 1913–1919, 2000.