Presented at SCMR 2005

MR-based cardiac electromechanical mapping in ischemic heart

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Introduction: Sudden cardiac death accounts for approximately 50% of cardiovascular mortality, resulting in more than 400,000 deaths annually in the US. Most cases are associated with prior myocardial infarction (MI), and the majority of sudden cardiac death results from reentry ventricular tachycardia. MI confers significant heterogeneity in material property of the myocardium, and regions of greater compliance that experience greater relative stretch during dynamic ventricular loading may provide foci of stretch-activated arrhythmias that serve as a trigger to initiate fatal ventricular tachyarrhythmia.

Purpose: We developed a cardiac electromechanical mapping technique using high-resolution DENSE and a 247-lead epicardial electrical sock, and qualitatively assessed anatomical correlation of abnormal electromechanics with reference to the MI boundary in chronic post-MI hearts in vivo.

Methods: Six dogs underwent MR studies 3 weeks after creating anterior MI by occluding proximal LAD for 2 hours with a balloon angioplasty catheter. All MR studies were conducted in Siemens Sonata 1.5T scanner. MI location and geometry were evaluated with a high-resolution delayed hyperenhancement inversion recovery sequence following intravenous injection of a contrast agent (Gd-DTPA, Berlex Magnevist) at 0.2 mmol/kg (BW \pm 140Hz/Px, TE/TR 4.7/32ms, 30° readout flip angle, FOV 119x200mm², 122x256 image matrix, slice 3.0mm). Epicardial circumferential strain (Ecc) was calculated from 3D displacement fields in five short axis slices using a high-resolution DENSE sequence (FOV 175x350mm², 96x250image matrix, slice 8.0mm). Isochrone map of the epicardial electrical activation time was determined using 247-lead sock electrodes.

Results: Ecc strain map showed abnormal myocardial stretch in the anterior MI regions and akinetic areas in the septal and lateral wall. The region of abnormal strain extended far beyond the MI boundary. In contrast, the region of delayed electrical activation, defined as the region with a greater than 50% delay, was over the MI region, but was smaller than the MI boundary, likely due to a complex transmural 3-D geometry of the infarct structure.

Conclusion: The abnormal mechanics region is larger than the MI region, whereas delayed electrical activation region is smaller than the MI region. The resultant large area with abnormal mechanics and normal electrical activation in the MI border zone may provide a potential electroanatomical substrate for ventricular tachyarrhythmia via stretch activated ectopy.

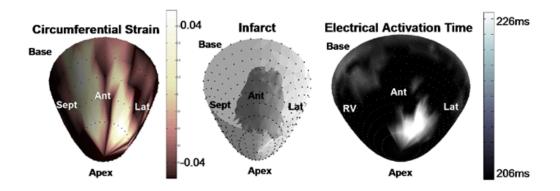


Figure: Viewing anterior wall from the apex.