Orientation and Magnitude of the Left Ventricular Principal Strains Are Sensitive To Ischemic Injury

C. Xu[1], K. Koomalsingh[1], G. Isaac[1], J. H. Gorman[1], R. C. Gorman[1], L. Dougherty[1], and J. Pilla[1]
[1]University of Pennsylvania, Philadelphia, PA, United States

INTRODUCTION Accurate assessment of regional and global left ventricular (LV) functions is critical for ischemic heart disease. The orientation and the magnitude of the myocardial principal strains have been shown to be sensitive to ischemic development. This study presents a method to fully characterize the alterations in the magnitude and orientations of principal strains in a pig left ventricle.

METHODS To measure alterations in principals strain post-infarct a pulse sequence was developed that applied the SPAMM tag prep pulse in three distinct planes in one acquisition. Two orthogonal sets of tags were oriented through-plane, while the third tag plane was defined at optimized angle relative to the through-plane tags. Displacement of each pixel was tracked with sub-pixel resolution using an optimized 3-D Optical Flow Method (3-D OFM), and was utilized to compute Lagrangian strain tensor and ultimately principal strains (ε_1 and ε_3). A pig model at baseline and five-days after posterolateral infarction was scanned using a 2-D fast gradient echo (FGRE) sequence with the optimized 3-D tag preparatory pulse on a 3 T Siemens Trio scanner (Siemens Medical Solutions, Malvern, PA, USA). Breath hold and ECG gating were performed to minimize respiratory and cardiac motion. Anterior and posterior phased array flex coils were placed on the subject and imaging was performed with the following parameters: TR/TE/FA=3.8ms/2.53ms/15^0, Averages=3, views per segment= 6, slice thickness= 3 mm, raw data matrix 256x128, interpolated to 256x256, field of view 220mm x 220mm, 20 slices. The area of infarction was demarcated by implanting custom molded platinum wire at the infarct borderzone boundary and further confirmed by perfusion image.

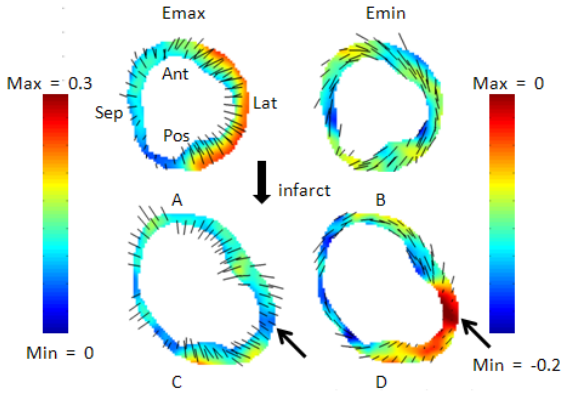

Figure 1. LV end-systolic maximum ($\boldsymbol{\varepsilon_1}$, A, C) and minimum ($\boldsymbol{\varepsilon_3}$, B, D) principal strain at one selective mid-ventricular level from baseline model (A, B) and 5 days infarct model (C, D). Strain map is color-coded by principal strain magnitude (0 to 0.3 for $\boldsymbol{\varepsilon_1}$ and -0.2 to 0 for $\boldsymbol{\varepsilon_3}$), with directions of wall thickening (A, C) and maximum shortening (B, D) superimposed. Note the significant changes in both magnitude and directions of principal strains in the infarcted region (arrows).

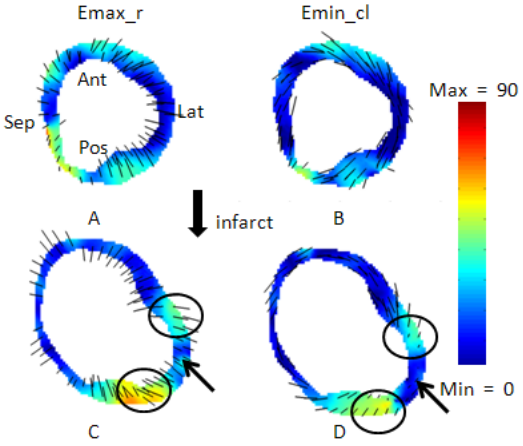

Figure 2. LV principal strain orientations in baseline (A, B) and 5 days infarct model (B, D) on the same slice. A and C demonstrate the angles between maximum wall thickening or maximum principal strain $\boldsymbol{\varepsilon_1}$ and radial direction (**r**), whereas B and D indicates the angles between maximum shortening or minimum principal strain $\boldsymbol{\varepsilon_3}$ and circumferential-longitudinal plane (**cl**). In the baseline model, $\boldsymbol{\varepsilon_1}$ and $\boldsymbol{\varepsilon_3}$ are aligned with **r** (16 +/- 7) and **cl** (9 +/- 3), respectively; while in the infarct model, border zone regions (circled) demonstrated heterogeneity, indicating deviation

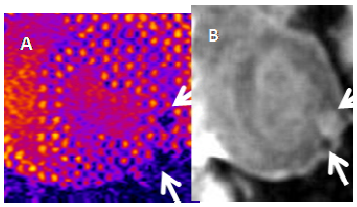

Figure 3. Identification of the area of infarct using implanted markers (A) and perfusion images (B)

RESULTS Systolic principal strains on one selective mid-ventricular slice are demonstrated in Figure 1. In the baseline model, transmural thickening occurs primarily in the mid-ventricular and basal regions as indicated by the greater $\boldsymbol{\varepsilon_1}$, while the lateral wall shows greater thickening than the septal areas (Figure 1A). Maximum shortening demonstrates a similar pattern but with more homogeneous distribution (Figure 1B). The infarct area is recognizable by a decrease of $\boldsymbol{\varepsilon_1}$ (Figure 1, A-C, 0.21 +/- 0.05 to 0.07 +/- 0.02) and greater values of $\boldsymbol{\varepsilon_3}$ (Figure 1, B-D, -0.11 +/- 0.02 to -0.03 +/- 0.01), and is confirmed by platinum markers and perfusion image (Figure 3). The results also suggest that in baseline model, $\boldsymbol{\varepsilon_1}$ or transmural thickening is oriented approximately in radial directions (16 +/- 7 degrees), while $\boldsymbol{\varepsilon_3}$ or maximum shortening is aligned in the circumferential – longitudinal wall that are tangential to the ventricular walls (9 +/- 3). As the result of infarction, direction of $\boldsymbol{\varepsilon_1}$ and $\boldsymbol{\varepsilon_3}$ in the borderzone region deviates from the radial direction (53+/- 15) and circumferential – ventricular wall (43+/- 11).

CONCLUSION This study has demonstrated significant alterations in $\boldsymbol{\varepsilon_1}$ and $\boldsymbol{\varepsilon_3}$ due to introduced infarct, indicating impaired transmural thickening and circumferential shortening. Characterization of the remote, border zone and infarct 3D strain is paramount in understanding infarct expansion and in the development of therapies to mitigate remodeling.