

Progressive Alteration of Regional Principal Strain Magnitude and Orientation during Post-Infarct Left Ventricular Remodeling

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Introduction:

Cardiac function after infarction is impaired due not only to deceased contracting myocardium but also to the acute and chronic mechanical effects of the infarct region on the remaining normal myocardium [1]. Although infarct material properties have been theoretically predicted to have a significant effect on cardiac performance surprisingly [2], little is known about the change in material properties that occur as the infarct matures. Following myocardial infarction, (MI) we hypothesize that changes in infarct material properties alter regional wall strain acutely and chronically during left ventricular (LV) remodeling and could contribute to global dysfunction and the development of heart failure.

Methods:

Alterations in principal strain with remodeling were studied using a swine postero-lateral infarct model (N=5). The LV infarct was created by ligating the circumflex and marginal arteries. LV volumes and regional strain were measured at baseline, 1-week, and 4-weeks post infarct using a 3T Siemens Trio scanner (Siemens Medical Solutions, Malvern, PA, USA). LV volume was acquired using a 2D bSSFP sequence with the following parameters: TR/TE/FA=26ms/1.64ms/40°, BW=1000, FOV=300mm x 243mm, Matrix=192x159, slice thickness=6, ave.=1. Systolic regional LV maximum principal strain was determined using a 3D FGRE with an optimized 3D SPAMM tag preparatory pulse and the following parameters: TR/TE/FA=3.8ms/2.53ms/15°, BW=400, FOV=220mm x 220mm, Matrix=256x256, slice thickness=2, ave.=4. All scans were cardiac and respiratory gated to minimize motion artifact. An optical flow method was used to track the 3D systolic LV displacement from which the maximum principal strain vector was calculated in the remote, border zone (BZ), and infarct regions during infarct development [3].

Results:

Post-MI LV remodeling was evident by an increase in end-diastolic volume (66.2±1.5ml (baseline), 90.3±5.7ml (1-week) p<0.05, 119.9±6.8ml (4-weeks) p<0.05) and a decrease in ejection fraction (40.5±0.5% (baseline), 35.4±0.3% (1-week p<0.05), 28.7±1.8% (4-weeks p<0.05). Representative strain maps from a single animal demonstrates a progressive alteration in the strain magnitude and angle with infarct healing (**Figure 1**). Composite results show a significant decrease in strain magnitude (Emax) for all regions at the 1-week time point with strain increasing in the BZ and infarct at 4-weeks (**Figure 2**). Strain angles (Emax_r) for all regions at baseline are aligned with the radial direction signifying wall thickening while as the infarct heals the infarct and BZ angles become less radial indicating infarct and BZ stretch.

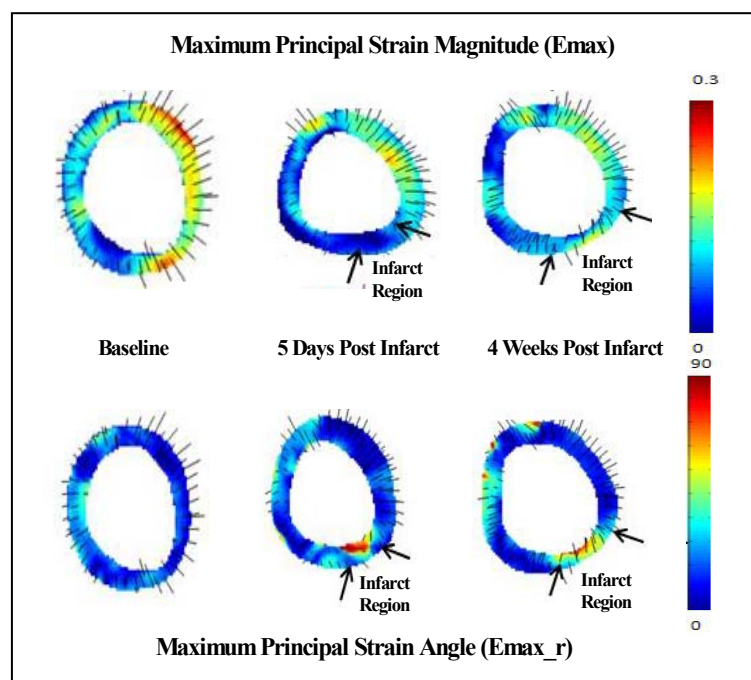


Figure 1. Representative maximum principal strain maps for a single animal during LV remodeling. Border zone and infarct magnitudes and angles are altered during remodeling indicating regional stretch.

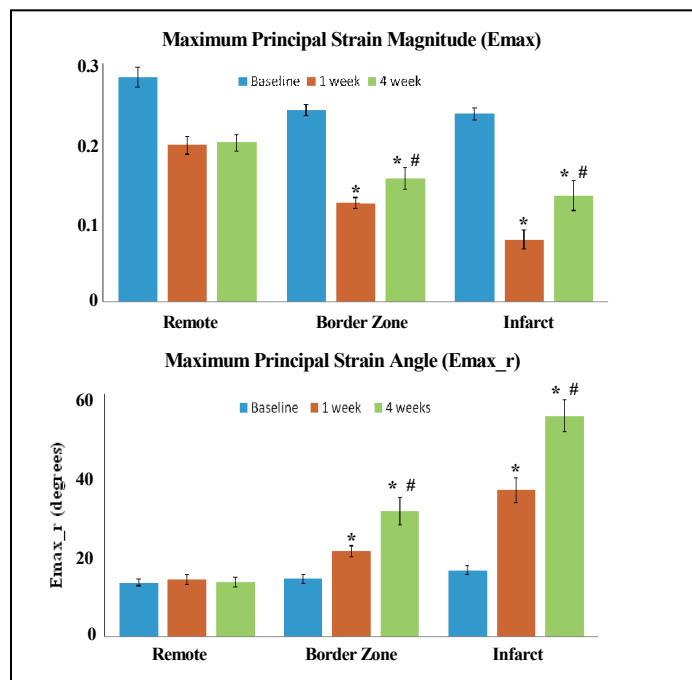


Figure 2. Principal strain composite results illustrating significant changes in magnitude and angle as the infarct heals. (* p<0.05 compared to baseline, # p<0.05 compared to 1-week).

Discussion:

Border zone and infarct regions demonstrate a progressive increase in systolic stretch as the LV remodels. This regional systolic stretch is likely the stimulus for continued remodeling that potentially could result in heart failure. Therapies that target border zone and infarct material properties may attenuate remodeling. Assessing the effect of therapies such as ventricular restraint and stem cell injection on infarct and border zone stretch using MRI derived principal strain could be a useful measure of their efficiency.

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References: 1) Jackson BM, et al. Extension of borderzone myocardium in postinfarction dilated cardiomyopathy. J Am Coll Cardiol 2002;40:1160–7. 2) Pilla JJ, et al. Theoretic Impact of Infarct Compliance on Left Ventricular Function. Ann Thorac Surg 2009;87:803–11. 3) C. Xu, J. J. Pilla, et al., Deformation analysis of 3D tagged cardiac images using an optical flow method, JCMR, 2010 Mar 30; 12:19