Right ventricular strain imaging with single-acquisition simultaneous 3-D SPAMM

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Background: Right heart strain imaging is relatively preload independent. Changes in strain pattern may precede changes in systolic function (Simon 2009,Hankiewicz 2008). Different strain patterns characterize different right ventricular pressure overload states (Jurcut 2011). We are developing a right heart strain assessment that can predict increased likelihood of right heart failure before frank failure has occurred and may be irreversible. 3-D SPAMM tagging acquires strain imaging in all 3 dimensions in a single acquisition (Xu 2010).

Methods: Animal model: 4 Yorkshire swine, 2 healthy and 2 with posterobasal myocardial infarctions induced by circumflex artery ligation at open thoracotomy, weighing approximately 90 kg, were used in an IACUC-approved study. One infarct animal was imaged at 4 weeks post, the other at 12 weeks post, infarction. MRI was performed on a 3 T clinical imaging system (Tim Trio Model, Siemens Healthcare, Erlangen, Germany). **Imaging:** The MRI sequence used for this study is based on SPAMM tagging, with an extra tag direction which permits assessment of strain in all 3 dimensions in a single acquisition. Pulse sequence parameters for 3D SPAMM were: spoiled gradient echo acquisition with SPAMM magnetization preparation spatial tagging, TR = 34ms, TE = 2 – 3ms, flip angle = 15 degrees, bandwidth/pixel = 330 Hz/pixel;12-16 phases over the cardiac cycle for a temporal resolution of approximately 32 ms. Typical parameters for the 3-D tagging sequence in these scans: slice thickness 2mmx1mmx2mm interpolated to 1mm x1mm x2mm ; whole heart coverage; 6mm tag spacing; 4 averages; 45 minute acquisition time. Imaging was obtained using respiratory triggering, with animals ventilated under general anesthesia, and triggering from invasive monitoring of left-sided pressures. Short axis images were acquired. **Postprocessing:** Tag tracking was performed using an optical flow method (Dougherty 2006, Xu 2010) and principal systolic strain was calculated from the displacement using a custom program based on Matlab. Maximal 3D strain over systole was assessed on multiple regions of interest in the right and left ventricles. Analyses were performed on short-axis slices from the apical third, mid-third, and basal third of the heart.

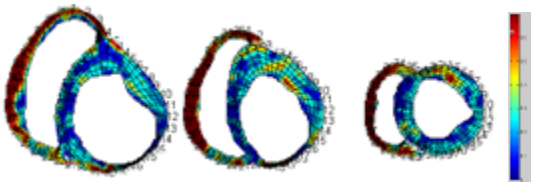
Note-due to software constraints, all figures are composites. The free walls (RV and LV, septum cut out) are assessed, then the LV(septum and free wall) assessed separately, and the figures aligned. The color scale is identical on all images and is chosen to better show differences in the RV free wall.

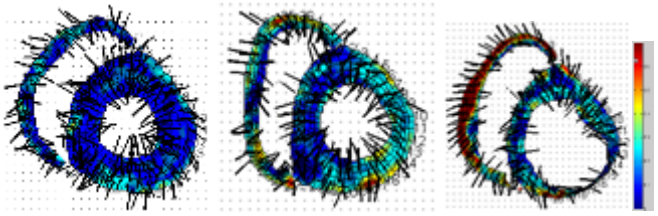
Animal A – baseline: basal, mid, apical

Animal B -- Four weeks after posterobasal infarct: basal, mid, apical

Animal A -- 12 weeks after posterobasal infarction

Vectors show direction of contraction – vectors are 3d, projected onto the 2d image. A shorter vector reflects a greater proportion of strain in the longitudinal direction. Shown are Animal C –control, animal A – control, animal A – 12 weeks after posterobasal infarction.





Results: Control animals had the lowest septal strains and there was a trend for increasing septal strain from the control, to the 4-week post-infarct, to the 12-week post-infarct state. Similar findings pertain to the right ventricular free wall. There were obvious morphologic changes to the left ventricle in the post-infarct animals. Strain in the anterior left ventricle free wall was similar in Animal A before and after infarct. However, a marked increase in right ventricular strain was seen in Animal A at 12 weeks post-infarct. The 4-weeks post-infarct animal had right ventricular strains that were intermediate between the control state and 12-week post-infarct cases.

Discussion: Attempts were made to match slice levels. However, localizers were not available for these studies and given morphologic changes related to infarction, exact matches were not possible. The post-infarct animals had extensive pericardial adhesions, which may have contributed to alterations in the strain pattern. The small sample size limits the ability to draw conclusions regarding right ventricular strain changes after posterobasal infarction.

Conclusions: It was possible by this method to obtain right ventricular strains, with 1-2mm spatial resolution and approximately 32 msec temporal resolution, adequate to permit conclusions about changes in maximal 3-D strain over systole in the vicinity of the RVOT, the mid-RV free wall, and the posterior RV free wall near the septum, at different levels from base to apex, as well as throughout the LV. Prospective studies permitting better localization of slices for comparison purposes are planned. Further work will optimize sequence parameters to permit imaging of patients. This sequence is currently experimental in humans.

Refs:1.Simon MA, et al. Tissue Doppler imaging of right ventricular decompensation in pulmonary hypertension. Congest Heart Fail 2009;15:271-6 **2.** Xu C, et al. Deformation analysis of 3D tagged cardiac images using an optical flow method. JCMR 2010, 12:19. **3**. Jurcut R, et al. Different Patterns of Adaptation of the Right Ventricle to Pressure Overload: A Comparison between Pulmonary Hypertension and Pulmonary Stenosis. JASE 2011;24:1109-17. **4.** Hankiewicz JH, et al. Principal strain changes precede ventricular wall thinning during transition to heart failure in a mouse model of dilated cardiomyopathy. Am J Physiol Heart Circ Physiol 2008, 294(1):H330-336 **5.**Dougherty L,et al.Use of an optical flow method for the analysis of serial CT lung images. Acad Radiol 2006, 13(1):14-23.