Mapping regional right ventricular myocardial strain using 3D cine DENSE MRI

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Introduction: Displacement encoding with stimulated echoes (DENSE) is a quantitative MRI technique used for measuring myocardial displacement and strain. DENSE encodes tissue displacement directly into the image phase (typically with reference to end diastole), thus allowing for the extraction of motion data at a pixel resolution [1, 2]. Studies of the right ventricle (RV) have previously been limited due to its complex geometry and motion. A free-breathing navigator-gated 3D spiral cine DENSE sequence, well suited for quantifying the complex behavior of the RV, has recently been developed [3]. This work introduces tailored processing techniques for assessing detailed regional RV motion and surface strain using 3D cine DENSE, and presents results for the healthy human heart.

Methods: In accordance with protocols approved by the University of Virginia Institutional Review Board, whole heart 3D cine DENSE data were acquired from 5 healthy volunteers on a 1.5T Siemens Magnetom Avanto MRI scanner using a 3D spiral cine DENSE sequence [3]. The entire heart was imaged with the imaging volume aligned along the long axis at a $2.8 \times 2.8 \times 5$ mm^3 spatial resolution and 32 ms temporal resolution. Imaging parameters included: ramped flip angle up to 20°, TR = 16 ms, TE = 1.3 ms, number of spiral interleaves = 6, and cardiac phases = 22. Fourteen 3D partitions were acquired, and zero-padding to 28 partitions was performed during reconstruction. After the Fourier Transform in the partition direction, 3 partitions at each end of the volume were discarded to avoid aliasing. The resulting 3D image matrix size was $128 \times 128 \times 22$. The RV myocardium was demarcated from surrounding structures by manually drawing each epicardial and endocardial contour on reconstructed short-axis DENSE magnitude images. Three dimensional spatio-temporal phase unwrapping was performed within the contoured area to remove phase aliasing [4]. Due to the limited number of transmural pixels spanning the RV wall, a full 3D strain tensor could not be calculated for all voxels in the RV. Consequently, strain was only assessed along a surface contour traversing the RV midwall. This was achieved by defining contours mid-way between the epicardial and endocardial contours, and spatially smoothing these within and across partitions using 4th order polynomials to ensure a continuous RV midline surface. Pixel-spaced midline points were tracked through the majority of the cardiac cycle using 3D distance weighted linear interpolation (using methods adapted from [4]). The resulting 3D motion trajectories were slightly smoothed in the temporal dimension using 10th order polynomials. Lagrangian strain oriented tangentially to the RV midline surface was then calculated directly from the 3D motion trajectories. The two resulting surface strain eigenvalues were typically of similar magnitude, and we were unable to reliably discern between the two principal strains. Furthermore, eigenvector orientation was not a suitable classifier due to the variation in myofiber orientation across the RV surface. The two principal strains were thus averaged for evaluation. One dimensional circumferential and longitudinal strain values were also calculated to provide directional strain indicators. Unlike for the LV, there is currently no standard method for dividing the RV into anatomical sub-regions. The supraventricular crest (SVC) is a muscular attenuation found at the base of the RV demarcating the inflow and outflow tracts. The moderator band (MB) is a muscular band connecting the interventricular septum to the anterior papillary muscle, found towards the apex of the RV. These two anatomical landmarks are used to divide the RV into four distinct anatomical regions: inflow, outflow, body and apex, as shown in Figure 1. The strain was calculated in each region, and averaged over the five volunteers.

Results: Figure 2 shows the mean 2D plots of strain versus time, and Table 1 shows the corresponding peak strain and time to peak strain for the four RV subdivisions. All strain values are consistently negative indicating muscle shortening. The time sequence of peak regional myocardial shortening, as confirmed by all strain estimates is: inflow, outflow, body and then apex. The inflow region consistently demonstrated the lowest strains. The regional variation in the time to peak strain compares well with previous SENC studies [5], and the peak strain values in the apical, body and outflow regions are consistent with previous myocardial tagging studies [6, 7]. The 1D strains followed a similar trend, but with longitudinal strains being larger than the corresponding circumferential strain values for all RV anatomical regions.

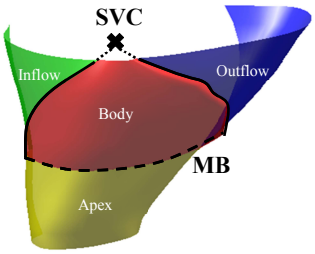

Figure 1: RV surface illustrating anatomical regions, the level of the moderator band (MB) and the supraventricular crest (SVC)

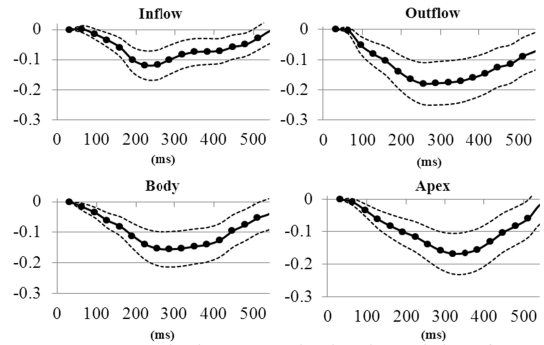

Figure 2: Mean strain time curves showing the evolution of mean principal strain for the RV anatomical regions. Data are shown as mean ± one standard

Table 1: Mean 2D Lagrangian peak strain and time to peak strain for the four RV regions.

Anatomical region	Peak strain	Time to peak strain (ms)
Inflow	-0.12 ± 0.05	224
Outflow	-0.18 ± 0.07	256
Body	-0.16 ± 0.05	288
Apex	-0.17 ± 0.06	320

Conclusion: The work presented here enables the motion and deformation of the entire RV to be captured at a pixel resolution. The results compare favorably with previous myocardial tagging and SENC studies, while the measured temporal strain evolution in the proposed RV anatomical sub-divisions indicate that these regions may also be functionally distinct. Future work will involve applying these techniques to studying regional RV function in various types of cardiac disease.

References: 1. Aletras et al. Journal of Magnetic Resonance 137:247–252, 1999. **2.** Kim et al. Radiology, 230(3):862-871, 2004b. **3.** Zhong et al. MRM, 64:1089-1097, 2010. **4.** Spottiswoode et al. IEEE Transactions on Medical Imaging, 26(1):15-30, 2007. **5.** Hamdan et al. JMRI, 28:1379-1385, 2008. **6.** Fayad et al. MRM, 39:116-123, 1998. **7.** Haber et al. *AJP - Heart* 289:1826-1833, 2005.

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