

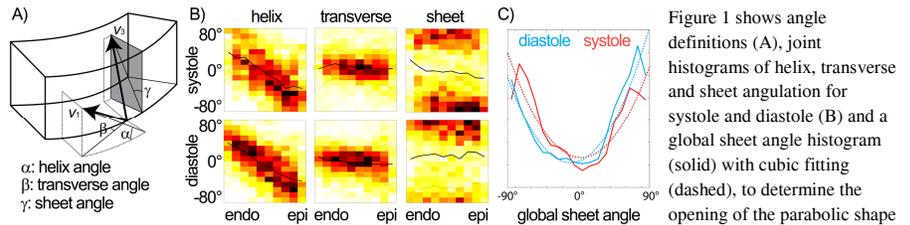
In-vivo dual-phase cardiac DTI with 3D strain correction

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Introduction: Cardiac diffusion imaging by Stimulated Echo Acquisition Mode (STEAM) encodes diffusion over two consecutive heart beats. As cardiac tissue undergoes material stretch during contraction of the heart an intrinsic strain encoding is introduced by the applied sequence¹. To avoid strain encoding, bipolar diffusion gradient waveforms² and the concept of sweet spot imaging³ were introduced. While bipolar diffusion encoding is limited to MR systems equipped with strong gradient systems, sweet spot imaging limits the range of possible imaging windows during the cardiac cycle to early systole and mid diastole. However, if the myocardial strain history is known, as it was already demonstrated by Reese et al.¹, DTI measurements can be corrected. In this work we combine 3D myocardial tagging with dual heart phase cardiac DTI to obtain geometrical information of the myocardial microstructure of the in-vivo heart in systole and diastole.

Materials and Methods: Five healthy subjects (3 male, age 27±7), were imaged on a 1.5T Philips Achieva system (Philips Healthcare, Best, The Netherlands) equipped with a five channel cardiac receiver array. In each volunteer a 3D tagging sequence using Complementary Spatial Modulation of Magnetization was performed in three consecutive navigator gated breath holds⁴. Data were acquired using multi-shot echo planar imaging (EPI) with following



parameters: FOV 108×108×108mm³, spatial resolution 3.8×7.7×7.7mm³, TE/TR 2.8/5.9ms, temporal resolution 17.6ms, and orthogonal tag line orientations with a tag line distance of 7mm. Three-dimensional displacement fields were derived from tagging data using the 3D SinMod algorithm⁵. Cardiac DTI was acquired in six slices during end-systole and end-diastole in each volunteer. The DTI acquisition consisted of an ECG triggered slice interleaved STEAM sequence acquiring one slice at end-systole and a second slice at end-diastole⁶. A local-look technique was applied in conjunction with non-coplanar excitation to limit the FOV in phase encoding direction. Ten diffusion encoding directions with a b-value of 500s/mm², were acquired in eleven respiratory navigator gated breath holds per slice (gating window 5mm). Diffusion imaging parameters were as follows: FOV 224×104mm², spatial resolution 2×2mm², slice thickness 8mm, partial Fourier factor 0.63, TE/TR 19ms/2R-R, 8 signal averages. The left ventricle (LV) was manually segmented. The diffusion tensors were estimated at each position within the segmentation mask by solving the modified Stejskal Tanner equation, taking into account the b-value of the FID crushing gradients in the b=0 image. The spatial positions of the tensors were then registered onto the masked displacement fields using the coherent point drift algorithm⁷. A local neighborhood was defined around the mapped coordinates of each tensor using a cube with edge dimension of 6mm. The virtual tissue voxels were tracked over time. In each heart phase the

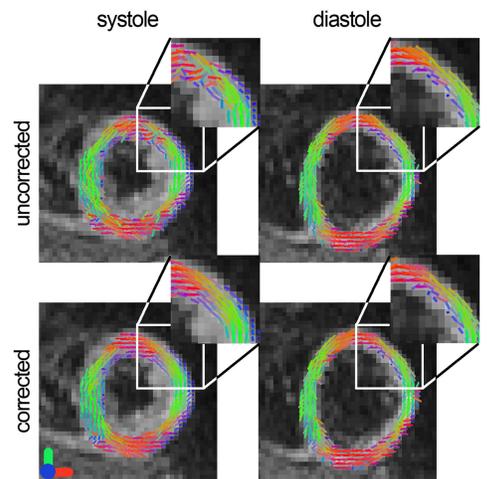


Figure 2: shows tensor maps for the systolic and diastolic heart phase, with and without strain correction.

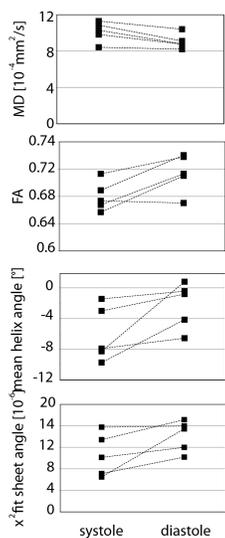


Figure 3: MD, FA, mean helix angle and cubic fit parameter in systole and diastole.

deformation gradient field $f = [\vec{e}_x; \vec{e}_y; \vec{e}_z]_{t \neq t_0}^T / [\vec{e}_x; \vec{e}_y; \vec{e}_z]_{t=t_0}$ was calculated relative to the heart phase at which diffusion data was acquired (t_0) with \vec{e}_i being the vector along the i^{th} edge of the voxel⁸. The right hand stretch tensor U was computed by $U = (f^T \cdot f)^{\frac{1}{2}}$. From the full history of the myocardial stretch tensor and the observed diffusion tensor, the true diffusion tensor D_0 was estimated by computing $D_{obs} = \frac{1}{\Delta t} \int_{t_0}^{t_0+\Delta} U(t)^{-1} D_0 U(t)^{-1} dt$. From D_0 the mean diffusivity MD, the fractional anisotropy FA and the eigenvectors were calculated. A local cylindrical coordinate system was defined in which the helix, transverse and sheet angles are given as shown in Figure 1(A). Data was pooled across the acquired slices and joint histograms were calculated for visualization of the transmural distribution of the characteristic angles. Global histograms were produced and the mean helix and transmural angles across the LV were calculated for systole and diastole. In order to estimate the change in sheet angulation, the histogram of the sheet angle distribution was fitted by second order polynomials (Fig. 1C) and the coefficients of the cubic component are reported.

Results: Figure 2 shows color coded diffusion tensors in systole and diastole prior and after strain correction. The reversal of tensor misalignment by correcting for materials stretch is more pronounced in systole. After strain correction the standard deviation of the transverse angle is reduced by 25%. Figure 1(B) shows joint histograms for the transmural helix, transverse and sheet angle distribution for both heart phases after strain correction as well as a global histogram of the sheet angle. Figure 3 shows the change of MD, FA, mean helix angle and the cubic fit parameter of the sheet angle distribution. No significant change in transverse angle was found between both heart phases. MD was found to be 10% higher in systole than diastole and FA was 4% lower. The fit parameter of the sheet histogram increased by approximately 30% in diastole versus systole.

Discussion and Conclusion: In this study 3D tagging data was integrated to correct cardiac DTI acquisitions for material stretch. The effect of material stretch was found to be more pronounced in systole making strain correction necessary for systolic cardiac DTI using STEAM. Sheet angle histograms are widened in systole compared to diastole, indicating a tilting of laminar fiber sheets, similar to the findings reported in isolated rat hearts⁹.

References: 1. Reese et al. MRM 1995 ; 2. Dou et al. MRM 2002; 3. Tseng et al. MRM 1999; 4. Rutz et al. MRM 2008 ; 5. Wang et al. IEEE 2013 ; 6. Stoeck et al. ISMRM 2012 ; 7. Myronenko et al. IEEE 2010 ; 8. Hess et al. MRM 2009 ; 9. Hales et al. Prog. in Biophys. and Mol. Biol. 2012