HELIX ANGLE MOBILITY DURING SYSTOLE USING HIGH RESOLUTION CARDIAC DIFFUSION TENSOR IMAGING

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Background

Cardiac Diffusion Tensor Imaging (cDTI) is an emerging method for probing the organization of the heart's microstructure *in vivo* [1,2]. The complex and highly organized cardiomyocyte network can be characterized by measuring the helix angle (HA) at each voxel. Cardiomyocytes form the functional basis of systolic contraction, but *in vivo* microstructural mobility (i.e., changes in HA) during systole remains poorly described. Our objective was to use dual-phase, high-resolution cDTI to measure transmural changes in HA mobility during systolic contraction.

Methods

cDTI at beginning systole (BS) and end systole (ES) were acquired at 3T (Prisma, Siemens) in healthy human volunteers (N=7) after obtaining IRB approved informed consent. Images were acquired using a high resolution, first and second order motion compensated, single-shot spin-echo EPI cardiac sequence (TE=61ms, TR=4000ms, 1.6x1.6x8mm interpolated to 0.8x0.8x8mm, end-respiratory, and ECG triggering). For each cardiac phase, two b-values (0 and 350s/mm²) with 12 diffusion encoding directions and 5 averages were obtained in a single mid-ventricular short-axis slice (~5 minutes per cardiac phase). A patient-specific trigger delay (TD) scout was used before every cDTI scan to determine the earliest and latest possible TDs during systole (Table 1).

Transmural Wall Depth (WD) and HA were computed at each image voxel after manual segmentation of the LV (Fig. 1) [3]. Across each mid-ventricular slice the HA distribution as a function of WD was estimated with linear regression of the HA medians (Fig. 2). HA was computed at the endocardium, mid-wall, and epicardium for each volunteer and for both cardiac phases.

Results

Transmural HA results are reported in Table 1. At BS, mean transmural HA was 43±6.3°, 10±3.8° and -23±9.4° at the endocardium, mid-wall, and epicardium. At ES, transmural HA was 70±12.3°, 12±8.8° and -46±15.8° at the endocardium, mid-wall, and epicardium. Transmural HA range increased from 66° at BS to 116° at ES ($p < 0.001$, two-tailed student's t-test).

Conclusions

Microstructural mobility during contraction has been shown to differentiate purported mechanisms of wall thickening in diseased hearts [5]. In this work, we characterize HA mobility in healthy human subjects, thus laying the groundwork for future analysis of HA mobility in patients with cardiovascular disease.

A significant steepening of the HA was observed between BS and ES phases in healthy volunteers. The measured increase in HA range from BS to ES agrees well with previous DTI studies performed on *ex vivo* [4] and *in vivo* [3] hearts in multiple contractile states.

Midwall circumferential strain (Ecc) has been reported as a surrogate for cardiomyocyte shortening. However, we show that midwall HA is greater than 0° at both BS and ES, suggesting that the mid-wall circumferential vector does not align with the tissue's microstructure. This work highlights the importance of using patient-specific cDTI in the assessment of cardiomyocyte performance.

References

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[4] Chen et al. Am J Physiol Heart Circ Physiol 2005
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Figures:

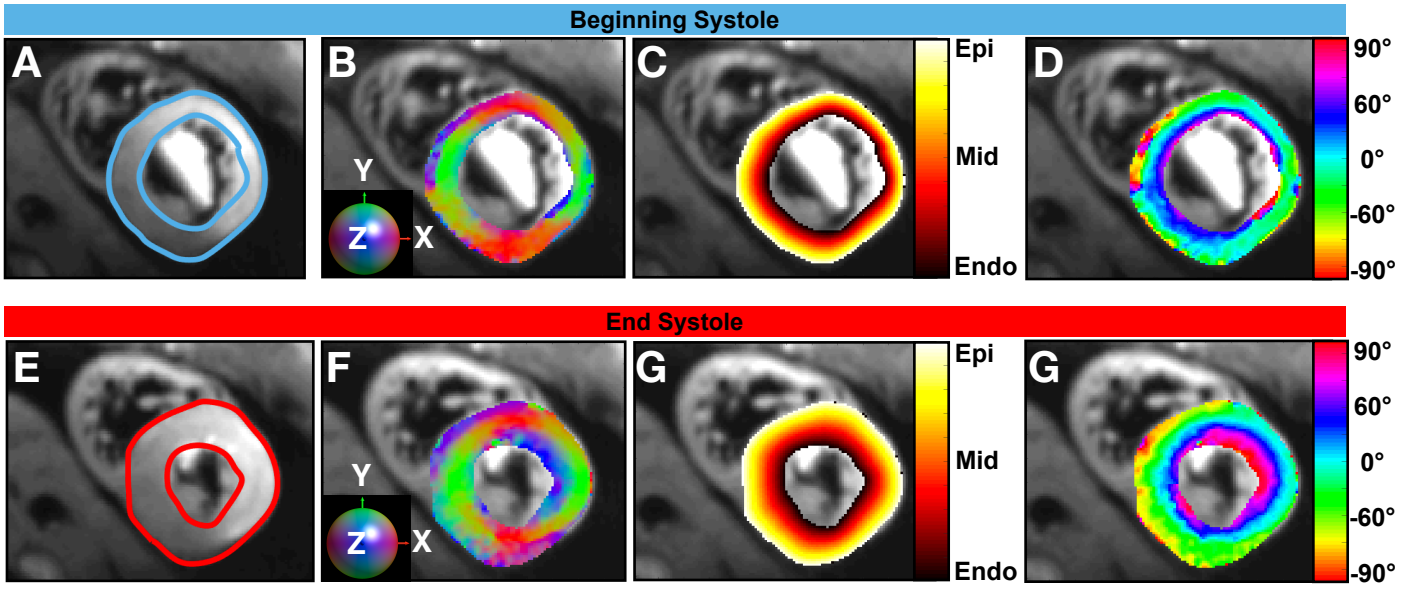

Figure 1 – Post-processing pipeline for an example volunteer. (A,E) Segmentation, (B,F) tensor reconstruction, (C,G) WD calculation using harmonic lifting [6], and (D,G) HA calculation at beginning and end systole.

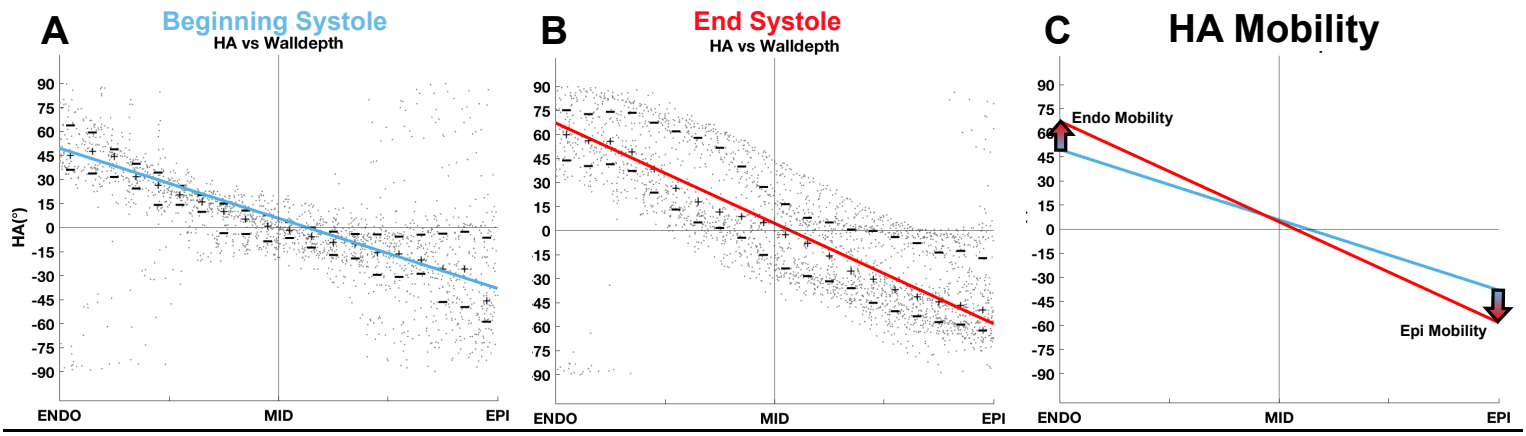

Figure 2 – Transmural HA results at (A) beginning systole and (B) end systole for the example volunteer in Fig 1. To visualize the trends in HA distribution, medians (crosses) and IQR (horizontal lines) are shown for 20 transmural bins of the HA data. Linear fits (blue and red lines) to the 20 HA medians are also shown. Notice the steepening of the fit and the broadening of the IQR from BS to ES. (C) Linear fits from BS and ES overlaid to illustrate epicardial and endocardial HA mobility.

Volunteer	HA - Beg. Systole (°)			HA - End Systole (°)			TD_{BS} (ms)	TD_{ES} (ms)	HA Range		HA Mobility	
No.	Endo	Mid	Epi	Endo	Mid	Epi			Beg. Systole	End Systole	Endo	Epi
1	51	11	-30	88	14	-60	150	335	81	148	37	30
2	38	15	-10	74	6	-62	140	262.5	48	136	36	52
3	41	6	-28	83	28	-28	137.5	300	69	111	42	0
4	48	15	-18	59	19	-22	147.5	270	66	81	11	4
5	35	8	-18	59	7	-44	150	325	53	103	24	26
6	40	10	-20	58	5	-48	150	327.5	60	106	18	28
7	50	6	-38	67	5	-58	112.5	312.5	88	125	17	20
Mean	**43**	**10**	**-23**	**70**	**12**	**-46**	**141**	**305**	**66**	**116**	**26**	**23**
SD	**6.3**	**3.8**	**9.4**	**12.3**	**8.8**	**15.8**	**13.6**	**28.6**	**14.4**	**22.4**	**11.9**	**17.4**

Table 1 – HA results across all volunteers. Using the computed linear fit, HA is reported at the endocardium (endo), midwall (mid), and epicardium (epi) for BS and ES. The difference in trigger delay between ES (T_{ES}) and BS (T_{BS}) is also reported. Finally, the HA range is reported at BS and ES, as well as the mobility at the endo- and epicardial surfaces.