

Quantitative analysis of cardiac motion effects on *in vivo* diffusion tensor parameters

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Background

Cardiac motion is a crucial problem in *in vivo* diffusion tensor imaging (DTI) of the human heart. Despite its importance, the effects of cardiac motion on diffusion tensor parameters, which contain the richest information about the fiber architecture of the human heart, have not been well established, mainly because of large signal loss. Recently, an efficient method was proposed that acquires cardiac diffusion weighted (DW) images at different time points of the cardiac cycle and removes motion-induced signal loss using principal component analysis (PCA) filtering and temporal maximum intensity projection (MIP) techniques (PCATMIP) [1]. Meanwhile, polarized light imaging (PLI) presently appears as the only technique allowing physically measuring the three-dimension (3D) fiber architecture of an entire human heart with a high spatial resolution ($100 \times 100 \times 500 \mu\text{m}^3$) and provides us the ground-truth of the heart fiber architecture. On the other hand, displacement encoding with stimulated echoes sequence (DENSE) can provide 3D high spatial resolution displacement fields [2] of *in vivo* human heart. These different imaging possibilities led us to investigate a multimodal approach to quantitatively analyzing the effects of cardiac motion on diffusion tensor parameters such as fractional anisotropy (FA) and mean diffusivity (MD).

Method

The method (Fig.1) consists of (i) using physical measurements from PLI to generate realistic DW images at different diffusion gradient directions [3], (ii) obtaining motion information from DENSE acquisition and DW images on the same subject (TD), (iii) constructing an empirical model to describe the relation between cardiac motion and diffusion signal intensity (Fig. 2(a)), (iv) applying such model to the original simulated DW images in order to obtain the motion-induced datasets, (v) applying the PCATMIP technique to the simulated and *in vivo* DW images for obtaining the motion-corrected images, and (vi) computing diffusion tensor parameters and the result was compared to the reference derived from static PLI images.

The PLI data used here were acquired on histological sections of the human heart embedded in methyl methacrylate (MMA).

All tissues were obtained in compliance with French legal and ethical guidelines. The acquisition was performed on an *ex vivo* heart using the imaging technique described in [4]. Then, the diffusion behavior of water molecules in the cardiac fiber structure is simulated based on a series of cardiac PLI data and using the Monte Carlo method [3]. In the simulation, we applied six diffusion gradient directions ($[1\ 0\ 0; 0\ 1\ 0; 0\ 0\ 1; 0.707\ 0.707\ 0; 0\ 0.707\ 0.707; 0.707\ 0\ 0.707]$) and the diffusion coefficient for the water molecule in the cardiac tissues was set as $1 \times 10^{-3} \text{ mm}^2/\text{s}$. From the resulting DW images, the MD was calculated that has a mean value of $0.78 \times 10^{-3} \text{ mm}^2/\text{s}$. The *in vivo* Cardiovascular Magnetic Resonance experiments were performed on a 1.5T clinical scanner (MAGNETOM Avanto, Siemens, Erlangen, Germany). Six volunteers were involved in the study. The motion in healthy volunteers was measured using a DENSE sequence. The acquisition parameters are: TE/TR=2/50ms, spatial resolution=3.5×3.5×8mm³, matrix=48×128, FOV=168×448. In total, 20 cardiac phases were acquired throughout the entire cardiac cycle. Raw data of DENSE acquisitions were processed with IDL (Research systems, Inc., Boulder, CO, USA). Then phase map differences were constructed from the reference scan and the encoded images [2]. Following manual myocardial border segmentation, the maps were phase-unwrapped and scaled to the position-encoding gradient strength, yielding separate displacement maps for the X, Y, and Z directions. The *in vivo* diffusion weighted images measurements were achieved using a diffusion EPI sequence. The acquisition parameters are: TE/TR=51/100ms, spatial resolution=2.6×2.6×6mm³, acceleration rate=2 (GRAPPA), partial Fourier=6/8, base resolution matrix=90×160, and b=200s/mm².

Results

Cardiac motion induced an overestimation of FA and MD and a reduced range of fiber angles (Fig. 2(b)). After processing by PCATMIP, both FA (0.59 ± 0.02) and MD ($1.13 \pm 0.4 \times 10^{-3} \text{ mm}^2/\text{s}$) are smaller than those derived from without motion-corrected acquisition (0.61 ± 0.05 and $1.99 \pm 0.3 \times 10^{-3} \text{ mm}^2/\text{s}$, respectively). Regular angle variation patterns were nearly recovered despite a relative higher noise level. The elevation angle range is narrower ($41^\circ \pm 13^\circ$ on the endocardium, $35^\circ \pm 12^\circ$ on the epicardium) than the reference ($57^\circ \pm 9^\circ$ on the endocardium, $51^\circ \pm 13^\circ$ on the epicardium). After motion correction by PCATMIP, measurement accuracy of fiber architecture properties such as FA, MD and fiber angles was significantly improved.

Discussion and Conclusion

We have proposed a multimodal approach to assessing the effects of cardiac motion on *in vivo* fiber architecture of the human heart. With the aid of ground-truth provided by the combined use of polarized light imaging data and simulated DW images and based on the motion information derived from DENSE imaging, the proposed cardiac motion model has been shown to allow effectively investigating the relation between cardiac motion and *in vivo* fiber architecture properties of the human heart. Cardiac motion resulted in great signal loss in DW images, an overestimation of both FA and MD, and a reduced range of fiber angles between endocardium and epicardium. PCATMIP method improves effectively the quality of *in vivo* DW images as well as subsequent measurement accuracy of fiber architecture properties, which suggests new solutions to the problem of obtaining *in vivo* fiber architecture of the human heart in clinical conditions.

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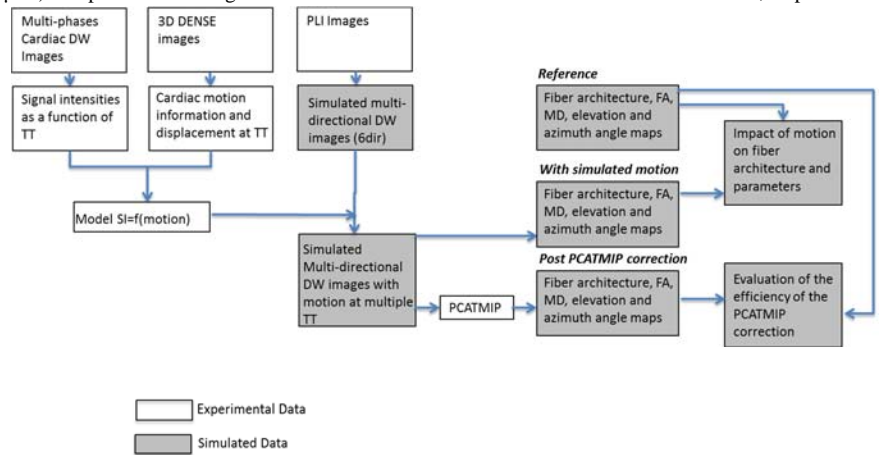


Fig. 1: Flowchart of simulated data generation and post-processing. Blocks with gray background represent simulated data and blocks with white background represent experimental data. "TT" in the flowchart means trigger delay time.

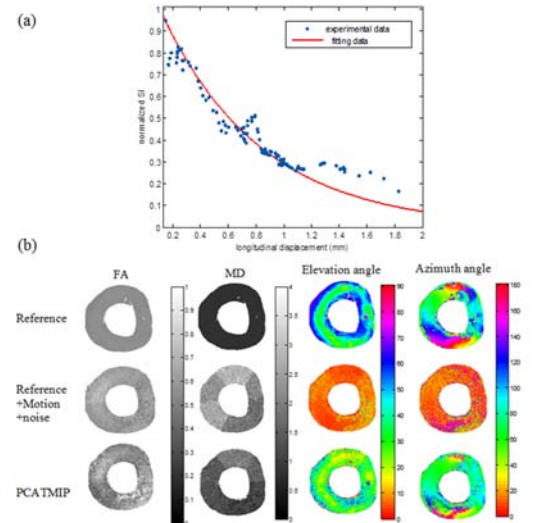


Fig. 2: (a) Normalized myocardium DW signal intensity as a function of cardiac motion. The cardiac motion is represented as the relative longitudinal displacement amplitude between two consecutive time points of DENSE acquisition. (b) Impacts of cardiac motion on fiber