

Relation of myocardial fiber structure with cardiac wall motion using DTI and MR tagging

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Introduction

MR tagging, a noninvasive imaging technique to investigate myocardium wall motion, has been widely used to quantify regional intramyocardial strain, twist, and torsion in both normal and pathological states [1-2]. Although left ventricular (LV) structure has been proposed to be associated with cardiac function [3], the relation of myocardial fiber distribution with corresponding regional wall motion remains to be elucidated. In this study, in vivo tagging and ex vivo diffusion tensor imaging (DTI) were performed in adult rats. LV myocardium twist, circumferential strain, and myocardial fiber architecture were investigated. The underlying reasons for cardiac contraction characteristics were explored.

Method

Imaging experiments were conducted on a 7T Bruker PharmaScan (Bruker BioSpin). SD rats (female, 2 months old, ~240g, N=6) were anesthetized with 1.5% isoflurane. Two sets of SPAMM sequences with orthogonal stripe tags were employed. ECG and respiratory triggered tagging imaging were performed on three short-axis slices at apex, midventricle and base, respectively. The parameters were: TR/TE=15/2.8ms, slice thickness=1.5mm, slice gap=1.5mm, cardiac frames=10, flip angle=30°, FOV=6cm², matrix size=256x256, tagging thickness=0.2mm, tagging distance=1.0mm, tagged images were zero filled into a 512x512 data matrix. All animals were sacrificed after tagging study and the excised hearts were fixed with formalin. DTI was performed on the heart samples at the same three slices using spin echo DTI. Imaging parameters were: TR/TE=1500/29ms, diffusion b= 800s/mm², number of gradient directions=6, FOV=2.55cm², matrix size=256x256, and NEX=10. The total scan time was ~7hr per sample. Tagging images were analyzed using a tool package [4] to calculate wall motion parameters. The tagging mesh was traced and divided into non-overlapping triangular tissue elements. Regional wall motion was tracked by the displacement of the centroid of each triangle. Twist angle, defined as the rotation of the centroid of the triangles from end diastole (ED) to end systole (ES), was measured. Positive twist represented clockwise rotation viewed from base. Lagrangian strain tensor was computed by use of homogeneous stain analysis, from which circumferential strain was calculated [5]. From DTI data analysis, helix angle was computed based on primary eigenvector. Right ventricle and papillary muscles were excluded in the analysis. Three groups of fibers were categorized: left-handed helical fiber (LHF) with helix angle within -90° to -30° in epicardium, circumferential fiber (CF) within -30° to 30°, and right-handed helical fiber (RHF) within 30° to 90° in endocardium [6]. Component difference between RHF and LHF was calculated, together with the three groups of fibers, were correlated with respective twist angle and circumferential strain on 3 slices. Two-tailed unpaired student's t-test was performed with values of $p < 0.01$ was regarded as significance.

Results

Fig.1a illustrated the cardiac wall motion of the centroid of each triangular element. The LV twist angle was found from clockwise at apex (14.7±1.9°) and middle ventricle (4.7±2.0°) to counterclockwise at base (-4.5±2.3°) from ED to ES. The helix angle maps of three slices at apex, middle ventricle and base of a sample were shown in Fig.1b, from which percentages of three groups of fibers were computed and compared in Fig.2. LHF increased significantly from apex (10.0±0.5%) to base (16.8±2.0%), while RHF decreased substantially from 38.1±4.3% to 30.5±2.3%. However, no significant change of CF was found among three slices. The average magnitude of circumferential strain decreased from apex (-0.21±0.01), middle ventricle (-0.18±0.01) to base (-0.15±0.01). Correlations of fiber architecture with LV myocardium wall motion, including twist angle and circumferential strain, were summarized in Table 1. Negative correlation was found between LHF percentage and twist angle. On the contrary, positive correlations were observed between RHF and twist angle. For CF, no significant correlation was obtained, which may due to its constant values among the 3 slices.

Increasing of component difference between RHF and LHF led to LV twist more towards clockwise. These results directly indicate that RHF and LHF force LV myocardium to twist at opposite direction, and the component difference between the two types of fibers determine the twist orientation of the slice. Similar results were found for the relations between fiber percentages and average magnitudes of circumferential strain. The magnitude of circumferential strain increased with RHF percentage, but decreased with LHF percentage, suggesting that RHF bundle is a crucial factor in circumferential shortening.

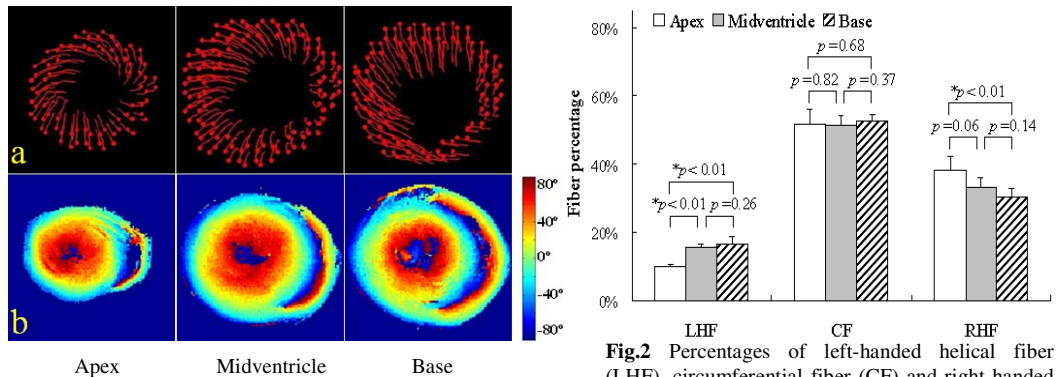


Fig.1 (a) Wall motion and (b) helix angle maps.

Fig.2 Percentages of left-handed helical fiber (LHF), circumferential fiber (CF) and right-handed helical fiber (RHF) at 3 slices.

Table 1 Correlations of fiber architecture with LV wall motion

	LHF percentage	CF percentage	RHF percentage	(RHF-LHF) percentage
Twist angle	R=-0.87, * $p < 0.01$	R=-0.11, $p=0.66$	R= 0.70, * $p < 0.01$	R= 0.83, * $p < 0.01$
Magnitude of circumferential strain	R=-0.87, * $p < 0.01$	R=-0.22, $p=0.39$	R= 0.77, * $p < 0.01$	R= 0.88, * $p < 0.01$

Discussion

In current study, twist angle and circumferential strain of LV myocardium were assessed by MR tagging, and ex vivo DTI was applied to investigate myocardial fiber structure at apex, midventricle and base. Shortening of LHF and RHF caused myocardium to twist in opposite directions due to their opposite spiraling orientation. CF typically spirals within the short-axis slice, thus its shortening causes no dramatic twist. Because fiber shortening is generally homogeneous through the ventricular wall [7], the RHF vs. LHF composition has important effect on LV myocardium twist. In this study, component difference between RHF and LHF was found to decrease from apex to base, correlating well with tagging measurement of LV myocardium twist. Circumferential strain at the endocardium is known to exceed all other shortening strains, and a very small strain is observed at the epicardium [7]. Therefore, the overall circumferential strain mainly results from the fibers at endocardium (e.g. RHF) and the CF that shows no apparent change between short-axis slice locations. Percentage of RHF was found in this study to positively correlate with magnitude of average circumferential strain, suggesting the key role of RHF in maintaining circumferential shortening. This finding may explain the RHF loss concurrent with cardiac failure in infarct patients reported in [6]. In conclusion, myocardial fiber distribution has direct relation with LV wall motion. Such integrated functional and structural analysis may provide more information for understanding the fundamental cardiac mechanics and assessment of pathological changes.

References

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