Diffusion Tensor Imaging of Left Ventricular Hypertrophy

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Introduction

The left ventricle (LV) responds to chronic pressure overload by increasing its wall thickness. Structural changes include muscle fiber hypertrophy, increased collagen content, and decreased capillary density [1]. Increased diastolic stiffness has also been reported [2]. However, there has been disagreement as to whether muscle fiber orientation changes as a result of hypertrophy [2]. Previous studies have shown that diffusion tensor imaging (DTI) correlates well with muscle fiber orientation [3].

Methods

We studied 3 controls (mean age = 6 wks) and 3 rabbits with LV hypertrophy resulting from aortic banding at 10 days of age. After the animals were euthanized at 5.5 weeks of age (a time point corresponding with maximal wall thickening [1]), the hearts were excised and fixed in 4% paraformaldehyde. Each fixed heart was encased in a cylindrical holder filled with fomblin (Ausimont, NJ), a signal-less susceptibility matching fluid, and place inside a 4.0 cm-diameter birdcage RF coil. Three-dimensional DTI acquisition (250 μm isotropic resolution; diffusion encoded with 748 s/mm2 b value in each of a set of 12 directions; acquisition time of 9.1 hr) was performed as described in [4] on a 7.1 T MR microscopy system. In post-processing, diffusion tensors were estimated on a pixel-by-pixel basis via nonlinear least-squares fitting. The eigenvalues of the diffusion tensor ($\lambda_1>\lambda_2\geq\lambda_3$) were used to compute the tissue fractional anisotropy (FA) [5] and the apparent diffusion coefficient (ADC = $\sum \lambda_i /3$). The eigenvector corresponding to λ_1, a proxy for myocardial fiber orientation, was converted to a helix angle [6]. All scalar diffusion parameters were averaged over a mid-ventricular, short-axis section. Once selected radial trajectories from endocardium to epicardium were defined on the section, linear regression was performed on the helix angle as a function of normalized transmural distance.

Results and Discussion

A significant ($p<0.02$) reduction in λ_1 was accompanied by a notable decrease in fractional anisotropy and apparent diffusion coefficient in the hypertrophied hearts. The slope (degrees/unit thickness) of the transmural helix angle profile in these hearts increased significantly ($p<.005$) with no detected difference in the regression coefficient (R^2) between the two groups.

Group	*FA*	*ADC*	λ_1	λ_2	λ_3	*Slope (^{o}/unit dist.) (n=167)*	R^2
Control	0.28±0.05	0.83±0.05	1.1±0.03*	0.78±0.05	0.62±0.06	-94±2.6*	0.9±0.009
Hypertrophy	0.18±0.03	0.73±0.02	0.88±0.05	0.72±0.03	0.61±0.02	-106± 3.4	0.89±0.008

(diffusion units: mm^2/s, *$p<0.02$ vs. hypertrophy)

The transmural variation in muscle fiber orientation enables equalization of stress across the left ventricular wall [7]. An increased transmural variation in muscle fiber orientation may be a result of increased stress and pressure at endocardium. Furthermore, a decrease in the anisotropy index is consistent with muscle fiber hypertrophy. However, the source of greater isotropy was a decrease in the largest eigenvector, rather than an increase in the two smallest eigenvectors. Changes in extracellular matrix contents may be responsible for this observation, as well as the average decrease in the apparent diffusion coefficient (ADC).

REFERENCES:
1. Friehs, I, et al., *Ann Thorac Surg*, 77:2004.
2. Fung, YC, Biomechanics, Springer, 1991.
3. Hsu, EW, et al., *Am J Phys Heart*, 274:1998.
4. Jiang, Y, et al., *Magn Red Med*, 52:2004.
5. Pierpaolu, C, et al., *Magn Red Med*, 36:1996.
6. Streeter, DDJ, et al., *Circ Res*, 24:1969.
7. Arts, T, et al., *J. Biomechanics*, 25(10):1992.

Note:
-All animal procedures were approved by the ARCH of our institution.

-The last two authors contributed equally to this work.