Myofiber developmental plasticity in fetal hearts delineated with diffusion tensor MRI

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Introduction: The prenatal and postnatal cardiac functions were different. Workload of right ventricle (RV) and left ventricle (LV) are dominant in prenatal and postnatal period, respectively. We hypothesis that myocardial fiber structures in fetal hearts may differ from that of the adult hearts as a response to changed cardiac function. The objective of this study was to quantify myocardial fiber structures with diffusion tensor MRI (DTI) in fetal pig hearts at 60 day gestation period, which is equivalent to 150 days in human.

Materials and Methods: Six fetal pig hearts (Nebraska Scientific, Omaha, NE) at 60 day gestation period were excised and fixed in 10% formalin solution. DTI of fetal pig hearts were performed on an 11.74 T Varian INOVA MR system using a 3cm birdcage coil and a multi-slice diffusion-weighted spin-echo pulse sequence. Imaging parameters were: TR, 2 s; TE, 33 ms; δ, 5 ms, Δ, 20 ms; b-value, 0 and 1063 s/mm^2; direction of applied diffusion-weighting gradients, 6; slice thickness, 0.5 mm; and in-plane resolution, 156×156 μm. Myofiber orientation in each voxel was estimated as the direction of the primary eigenvector. Transverse angle of myofiber was calculated as the angle between the fiber orientation and the circumferential directions [1]. Transverse angles in the region of interest (ROI) at the fusion site of LV and RV (Figs. 1A and B), transverse angles in the LV free wall (LVFW), and thickness ratios of the RV free wall (RVFW) to the LVFW were quantified. All measurements in pig fetal hearts were compared with our previous analogous results in adult rat hearts (n=6).

Results: The zoom-in view of fiber orientation map showed balanced contribution to septal myofibers from the RV and LV (Fig. 1C). In contrast, fibers in the septum of adult heart were predominantly originated from the LV (Fig. 1D). Accordingly, myofiber transverse angles at the ROI had a significantly higher standard deviation (SD) than that of LVFW in fetal hearts (Fig. 2A, $p < 0.001$), suggesting mixed contributions from RV and LV to myofibers in the fusion site of septum. In contrast, SD of myofiber transverse angles in the ROI and LVFW of adult hearts were comparable (Fig. 2B, $p > 0.05$), suggesting similarities in myofiber distribution of the two regions. Thickness ratio of RVFW to LVFW was 0.96 ± 0.11 in the fetus heart, significantly higher than the 0.47 ± 0.04 thickness ratio in the adult heart ($p < 0.001$).

Conclusions: The balanced contribution to septal myofibers from the LV and RV and the relative thicker RVFW in fetal heart were in agreement with that RV is dominant in fetal circulation with a higher workload than LV. In contrast, septal myofibers in adult hearts were predominantly contributed by the LV, agreed with that LV dominates circulation with a higher workload than RV after birth. These marked cardiac structural differences between fetal and adult hearts reflect the plasticity of myocardial fiber development in response to the programmed differential contractile functions before and after birth.

Reference:

[1] Scollan DF, et al., Histological validation of myocardial microstructure obtained from diffusion tensor magnetic resonance imaging. Am J Physiol Heart Circ Physiol, 1998

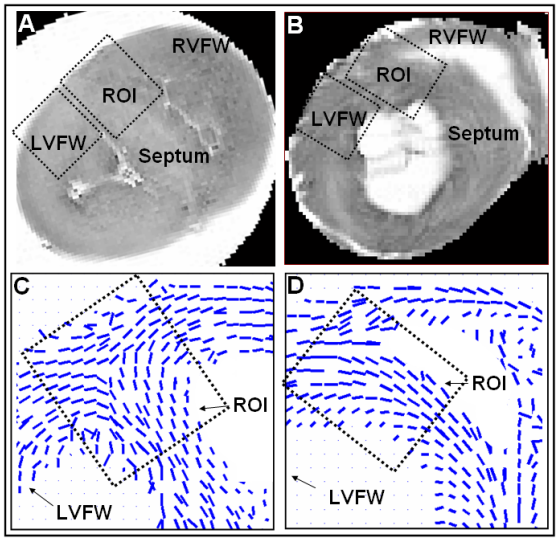

Figure1. T2-weighted images (b-value = 0 s/mm^2) of a fetal pig heart (A) and an adult rat heart (B) shows the heart anatomy. Zoom-in view of fiber orientation map at a fusion site of left ventricle (LV) and right ventricle (RV) shows the contribution to septal fibers from RV and LV in the fetus (C) and adult (D) hearts.

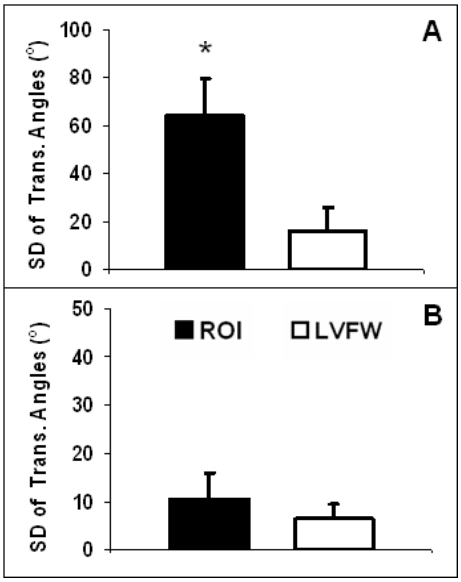

Figure 2. Standard deviation (SD) of myocardial fiber transverse angles at the LV-RV fusion site (ROI) and LV free wall (LVFW) in fetal (A) and adult hearts (B).