Diffusion Spectrum MRI Tractography Reveals the Presence of a Complex Network of Residual Myofibers Within Infarcted Myocardium.

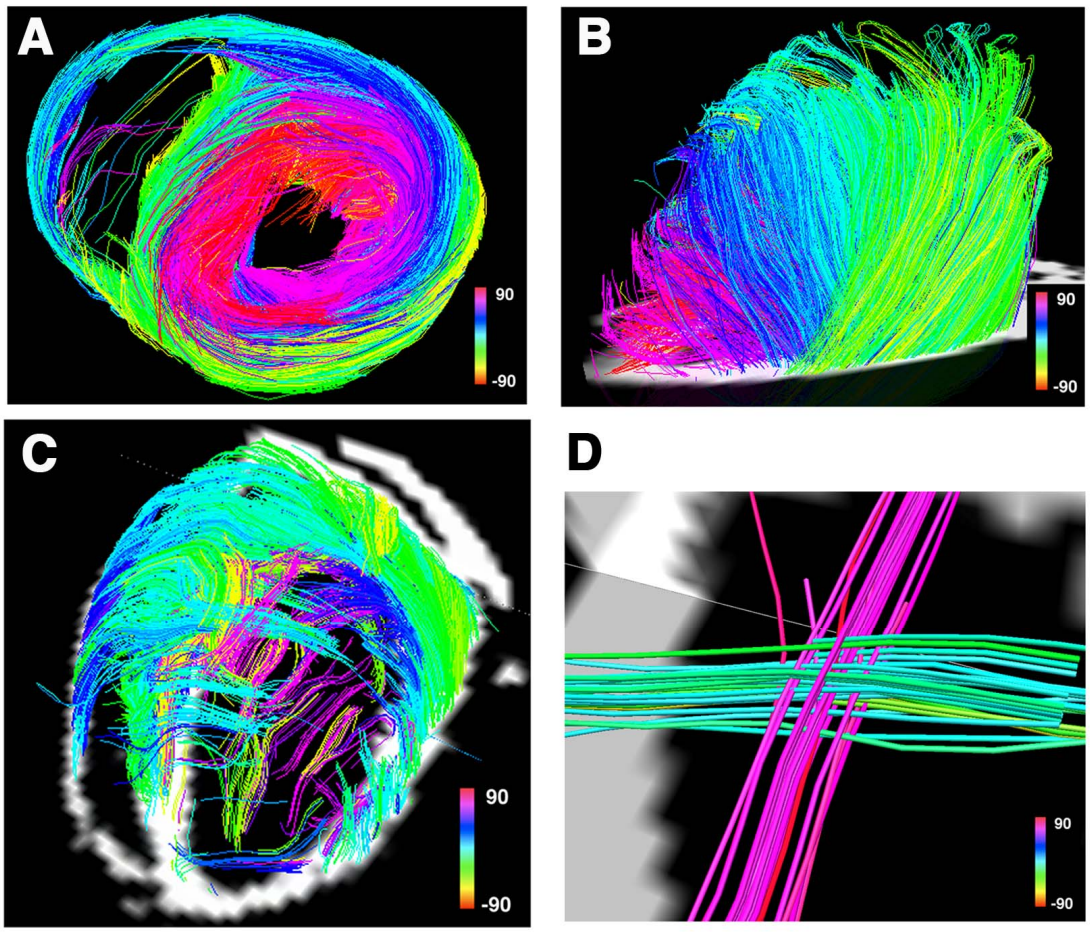
D. Sosnovik[1], R. Wang[2], G. Dai[2], T. Wang[3], E. Aikawa[4], M. Novikov[5], A. Rosenzweig[5], R. Gilbert[3], and V. Wedeen[2]

[1]Martinos Center for Biomedical Imaging, Massachusetts General Hospital,Harvard Medical School, Boston, MA, United States, [2]Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School, [3]Massachusetts Institute of Technology, [4]Massachusetts General Hospital, Harvard Medical School, [5]Beth Israel Deaconess Medical Center, Harvard Medical School

Background and Aims: Changes in myocardial microstructure play an important role in the mechanical and electrical response of the myocardium to infarction. Diffusion tensor imaging (DTI) has been previously used to sample average fiber orientation at discrete points in infarcted myocardium, but is not able to fully resolve complex and crossing fiber geometries. A novel technique, diffusion spectrum MRI tractography (DSI-tractography), has been used to image complex fiber geometry in the brain and tongue, and has been shown to resolve fiber architecure in these organs with better accuracy than DTI. Unlike DTI, DSI-tractography resolves multiple myofiber populations per voxel, thus generating accurate 3D tractograms of crossing and converging fiber geometries.

The aim of the present study was to use DSI-tractography to characterize myocardial fiber architecture in infarcted rat hearts. We hypothesized that in normal myocardium a smooth evolution in myofiber helix angle would be seen, and that myofibers with orthogonal helix angles would be separated by intervening layers of myofibers. We further hypothesized that in the border zone of infarcted myocardium that this smooth pattern of myofiber helix angle would be lost, and that crossing myofibers with orthogonal helix angles would lie in direct contact with each other.

Methods: DSI-tractography was performed at 4.7 Tesla in excised rat hearts 3 weeks following left coronary artery ligation (n=4), and in 4 age-matched controls. The DSI acquisition involved the collection of data during the application of 515 diffusion-encoding vectors, used to fill q-space. Generation of the fiber tractograms was performed using custom software developed in our center. Myofiber orientation was encoded in terms of the helix or spiral angle with the long axis of the left ventricle.



Results: Fiber architecture in the control hearts varied smoothly from endocardium to epicardium, producing a symmetric array of crossing helical structures in which orthogonal myofibers were separated by fibers with intermediate helix angles (Figure A, B). In all the infarcted hearts, however, a complex network of residual myofibers was present in the infarct and border zones (Figure C). The orientation of these residual myofibers was highly perturbed (Figure C, D). Longitudinal myofibers connecting the infarct zone to the basal-anterior wall and transversely oriented myofibers connecting to the septum lay in direct contact with each other, forming nodes of orthogonal myofiber intersection or contact (NOMIC). A mean of 7 +/- 0.7 NOMICs were seen in each of the infarcted hearts.

Figure: Subendocardial myofibers have positive (pink) helix angles and subepicardial fibers have negative (yellow green) helix angles. (A, B) Normal heart viewed (A) in its short axis and (B) from its lateral wall. (C) Rat heart with large anterior infarct. Residual fibers with positive helix angles lie in direct contact with orthogonally-oriented residual myofibers with negative helix angles. (D) Magnified view of a node of orthogonal myofiber intersection or contact (NOMIC) in an infarcted heart.

Conclusion: DSI-tractography resolves complex 3D myofiber architecture, and is able to reveal the presence of crossing and contacting myofibers in infacted myocardium. An orthogonal network of residual myofibers was seen within the infarct and border zones in all the infarcted hearts in this study. Mesh-like networks of orthogonal myofibers in infarcted myocardium may resist mechanical remodeling, but likely also increase the risk for lethal re-entrant arrhythmias. DSI-tractography thus provides a new and important readout of tissue injury following myocardial infarction.