Transmural Changes in Fibre Helix Angle in Normal and Failing Canine Ventricles

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Abstract

Fibre orientation relative to the base-apex axis (the helix angle) varies across the ventricular wall. The aim of this study was to examine the consistency of transmural helix angle in 6 normal canine hearts, and 3 canines with heart failure from the Johns Hopkins DT-MRI dataset.

In the normal hearts, the helix angle across the LV free wall and septum changed smoothly from between 50° and 60° in the epicardial layer (RV endocardium in the septum) to between -50° and -60° in the LV endocardial layer. However, we observed abrupt changes in helix angle abrupt and within an endocardial layer around 3 mm thick. Close to the insertion of the RV and the septum, fibre orientation was much more variable. In failing hearts, changes in helix angle across the LV and RV free walls as well as the septum was much less well defined.

Our findings indicate that the helix angle is more variable close to the ventricular endocardium, at the insertion of the RV, and in failing hearts.

1. Introduction

Computational models of electrophysiology and mechanics in the heart have become a valuable research tool. Increasingly, simulations involve detailed anatomical models of atria and ventricles, and the importance of tissue structure is beginning to be appreciated. Several anatomical models are now available to the research community [1].

The anisotropic structure of cardiac tissue is important not only for mechanics [2], but also for action potential propagation [3].

The first anatomically detailed ventricle models were developed by painstaking dissection and measurement of the orientation of cardiac muscle fibres [4], which varies within ventricular tissue [5].

Idealised geometries representing a small section of the ventricular wall often assume axial anisotropy, with a change in fibre rotation of 120° from endocardium to epicardium [6, 7]. These ideas are based on the pioneering work of Streeter and others [5].

Diffusion tensor MRI (DT-MRI) measures the diffusion of protons within the imaged volume. In cardiac tissue, diffusion of protons is constrained by the tissue fibre structure, and so the principal component of the DT-MRI signal provides an indication of fibre orientation. has been These include an MRI dataset obtained by Johns Hopkins University from normal and failing canine hearts [8, 9]. The aim of this study was to examine the consistency of transmural changes in fibre helix angle in the Johns Hopkins dataset of normal and failing cardiac ventricles.

2. Methods

This study used imaging data from canine hearts forming part of the Johns Hopkins DT-MRI data sets. These hearts were originally imaged by Drs. Patrick A. Helm and Raimond L. Winslow at the Center for Cardiovascular Bioinformatics and Modeling, Johns Hopkins University, and Dr. Elliot McVeigh at the National Institute of Health. The DT-MRI data are archived and available to the research community at http://gforge.icm.jhu.edu/gf/project/dtmri_data_sets/wiki/

We downloaded the data for six normal canine hearts (data sets 042604, 080803, 101003, 101703, 103103, and 123103) and four failing canine hearts (data sets 040204, 041804, and 082303). Figure 1 illustrates an example canine anatomy from these data

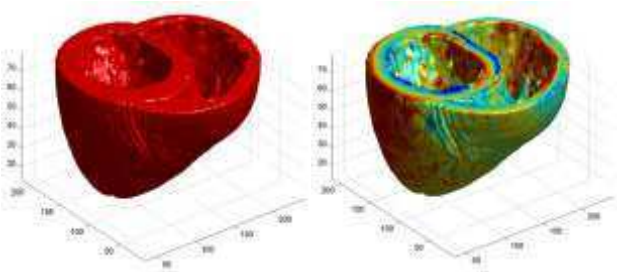

Figure 1. Example ventricular anatomy. Left panel shows tissue surface. Right panel encodes helix angle using colourmap.

We examined transverse slices, midway between apex to base, and close to the apex. In each slice we measured the helix angle at locations in the left ventricular (LV) free wall, the septum, and the right ventricular (RV) free wall. This process is illustrated in Figure 2. Helix angle was evaluated along two line segments; one running from the LV free wall to the RV free wall, and the other running from the anterior RV insertion to the posterior RV insertion. The location of each pair of lines was determined manually. Helix angles were calculated along each line, and along a line displaced by 2 and 4 pixels either side of the line. From the five values of helix angle obtained at each transmural location, a mean and standard deviation was calculated.

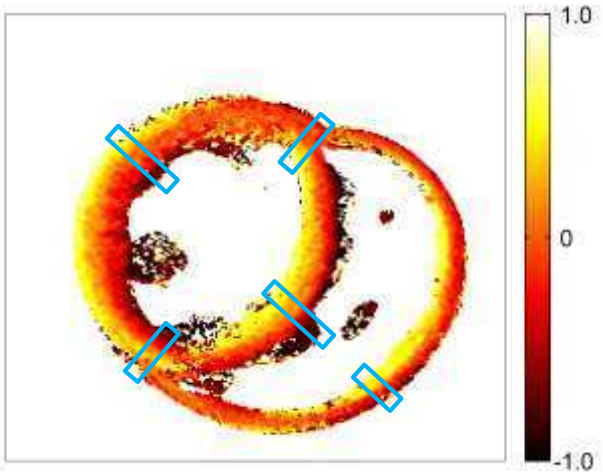

Figure 2. Transverse slice 65 from data set DT080803. Colours indicate cosine of fibre helix angle. Blue regions indicate regions from which fibre data were extracted.

3. Results

Our results for the LV free wall, septum, and RV free wall are shown in Figure 3. Each graph includes a single line from each individual heart, and the points plotted are the mean helix angle. Normalised transmural distance runs from LV endocardium to LV epicardium, LV septum to RV septum, and RV endocardium to LV epicardium.

These findings indicate that in normal hearts, the helix angle across the LV free wall changed smoothly from between 0 and 40° in the endocardial layer to between -20

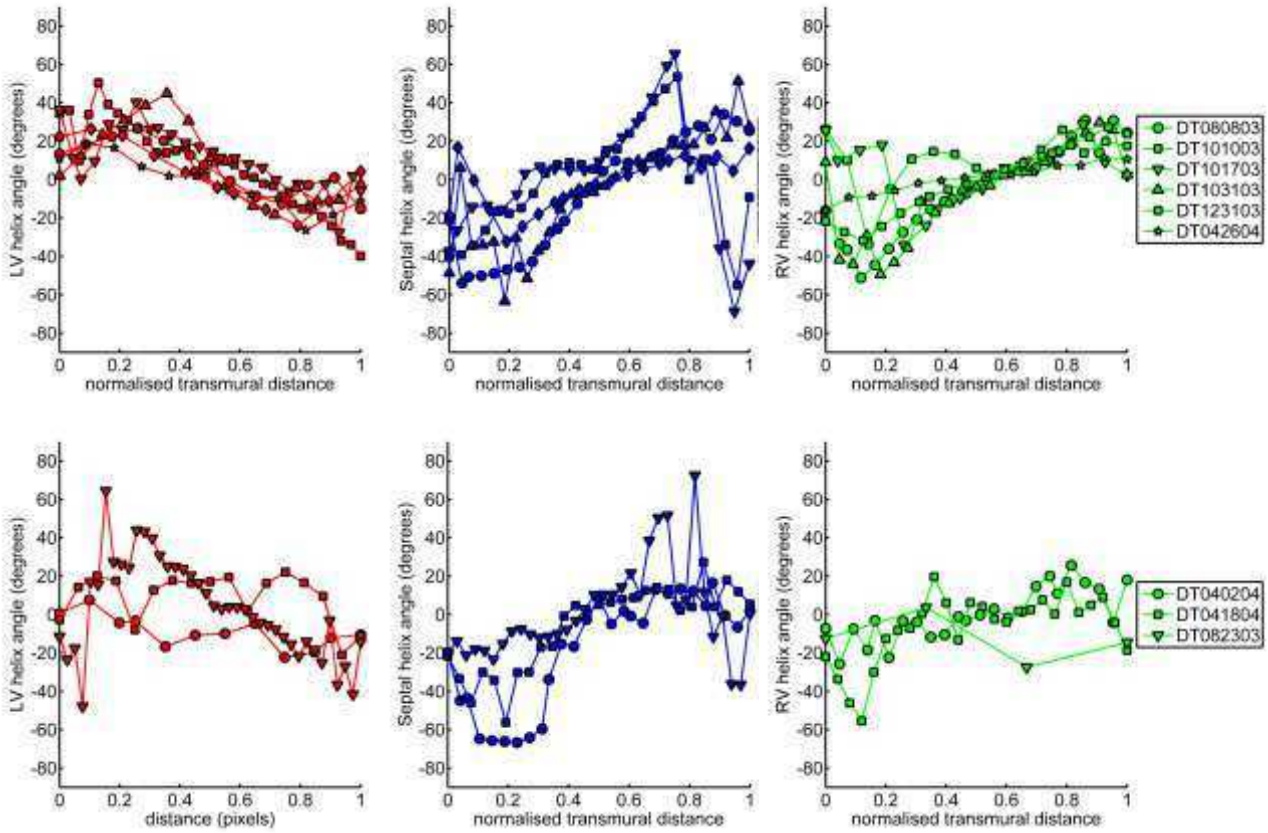

Figure 3. Helix angle for each heart in the LV free wall (left), septum (middle) and RV free wall (right. Top row shows data for normal hearts, bottom row data for failing hearts

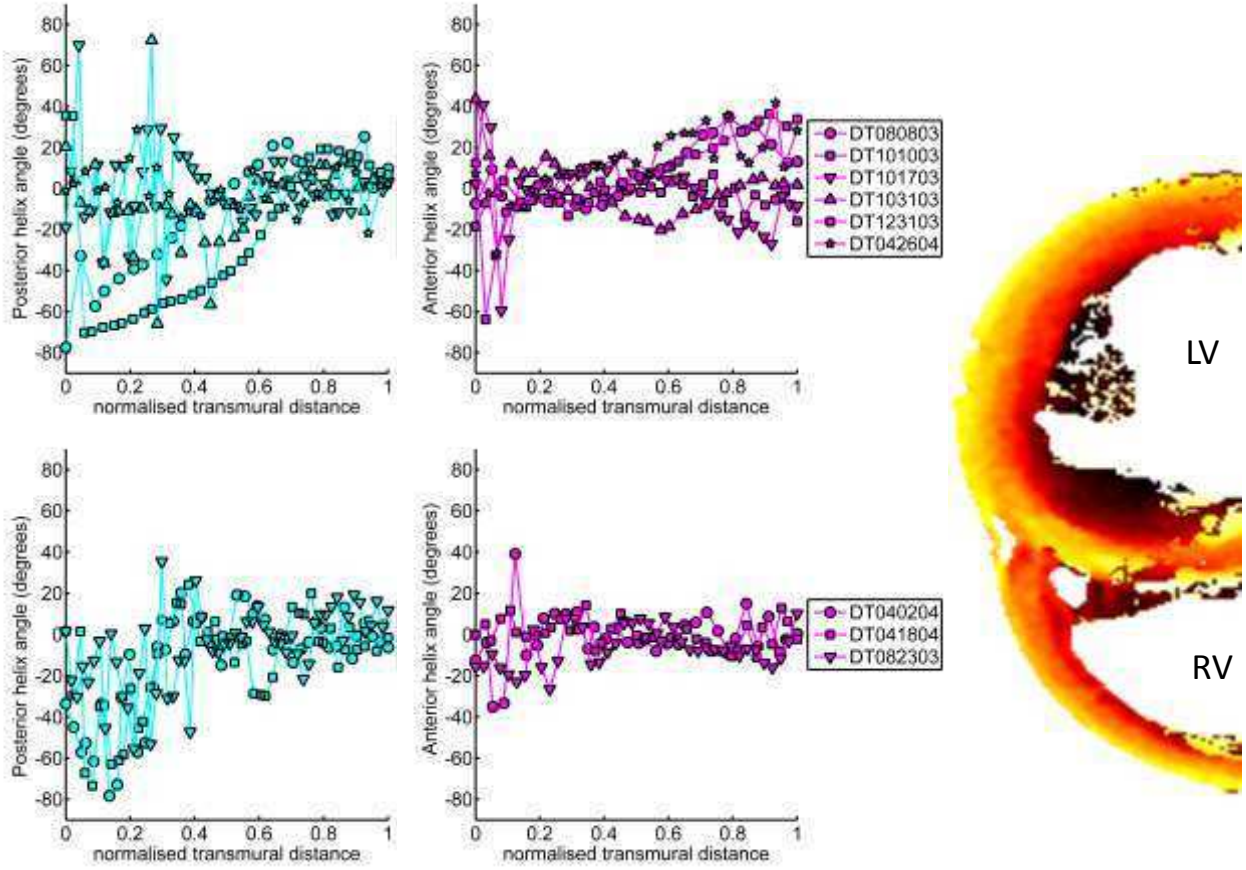

Figure 4. Helix angle for each heart in the RV insertion on the posterior (left), and anterior (right) walls. Top row shows data for normal hearts, bottom row data for failing hearts. Right hand panel illustrates complexity of helix angle changes near the RV insertion in data set DT123103

and -40 degrees in the LV epicardial layer, which is consistent with other studies in the rabbit [8, 10] and canine [4] heart. However, the helix angle in the septum and RV free wall showed an opposite change from negative to positive angles. Close to the epicardium and within an endocardial layer around 3 mm thick we observed abrupt changes in helix angle.

Although the pattern of changes in the normal and failing hearts was similar, the transmural change in helix angle was less smooth.

Close to the insertion of the RV, helix angle was much more variable. Our data for the RV insertion points are shown in Figure 4. In some hearts (for example DT080803 and DT123103) there was a relatively smooth

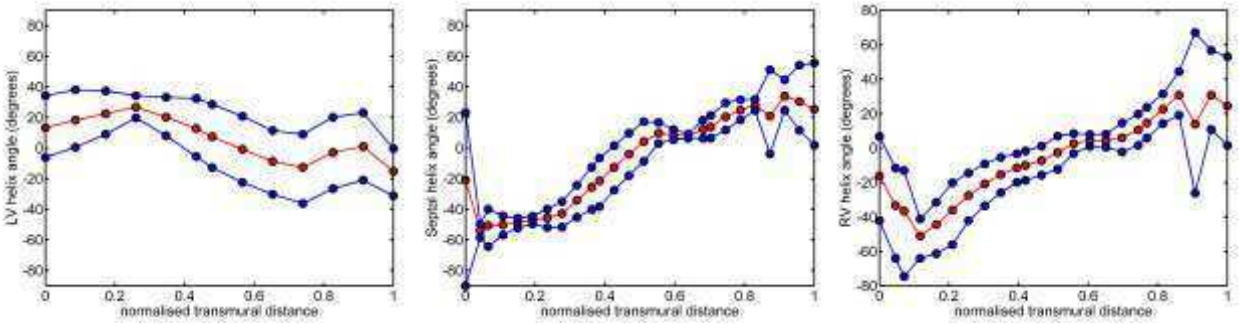

Figure 5. Mean (red) and mean ± standard deviation (blue) helix angle for heart DT080803, LV (left), septum (middle), and RV (right).

change in helix angle, however in others the changes were much more erratic. Part of the variability between hearts could be accounted for by the region that was included in the analysis. The right hand panel of Figure 4 shows the helix angle close to the RV insertion in heart DT123103. At the insertion point there are abrupt changes in helix angle , whereas the continuation of the LV into the tissue preserves a smoother transition.

Within an individual heart, the variability of transmural helix angle in a region of interest was typically between 10 and 20°. Figure 5 gives an example of the transmural changes in helix angle in heart DT080803, and shows the mean and standard deviation helix angle at each transmural location.

4. Discussion and conclusions

The transmural change in helix angle is often characterised as smooth, with a total rotation of 120°. Our findings are consistent with this idea, but show that the helix angle is more variable close to the ventricular endocardium, at the insertion of the RV, and in failing hearts. We have also shown that for the data sets included in this study, the total change in helix angle across the LV free wall is rather less than the value of 120° that is often quoted.

Our study found only small differences in helix angle between normal and failing hearts.

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