Toward Quantification and Visualization of Active Stress Waves for Myocardial Biomechanical Function Assessment

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Introduction: Estimating and visualizing myocardial active stress wave patterns is crucial to understanding the mechanical activity deep within myocardial tissue and provides a potential non-invasive method to assess myocardial function. These patterns can be reconstructed by analyzing 2D and/or 3D tissue displacement data acquired via magnetic resonance imaging (MRI) or ultrasound imaging.

Methods: Here we describe an application that utilizes a 3D finite element formulation to reconstruct the active stresses from displacement data in response to the action potential wave propagation. As a proof of concept, a simple cubic mesh (Fig. 1) was used to represent a "sample" of myocardial tissue consisting of a 10 x 10 x 10 lattice of nodes featuring different fiber directions that rotate with depth, mimicking cardiac transverse isotropy.

Fig. 1: Tissue lattice mesh and resulting deformation field in response to the active stress wave propagation.

In the forward model, tissue deformation was generated using a test wave with active stresses that mimic the myocardial contractile forces. Two of the three components of the deformation field were used as input to an inverse model designed to reproduce the original active stress distribution. In a subsequent iteration, simulated "dead" tissue regions (experiencing limited contractility and hence active stresses) were numerically simulated within the healthy tissue experiencing normal active stress patterns. Lastly, model sensitivity was assessed by adding random 1% standard deviation displacement noise to the forward model deformation field.

Results: In absence of noise, the reconstructed active stresses look nearly identical to the original wave (Fig. 2). When the deformation field is subjected to noise, the reconstructed active stress distribution features a high frequency checkerboard error pattern, but the overall wave and the "abnormal" tissue regions experiencing low active stress were still discernible (Fig. 3).

Fig. 2: Original and reconstructed active stresses at three time points during wave propagation in absence of noise. Dashed line encloses the original test wave; dotted lines indicated the locations of the "dead" tissue. The difference image between the original and reconstructed active stresses showed that even in absence of noise, the checkerboard error pattern was still present, albeit on the order of 10^-12, suggesting this error is inherent in the model.

Fig. 3: Reconstructed active stress from a noisy (1% Std. dev.) deformation field.

Conclusions: The model accurately estimates active stresses from tissue deformation data with a high signal-to-noise ratio. The error pattern is expected to follow the Nyquist criterion, suggesting that model would provide reasonable results at depicting details that are at least 2 computation units in size. Ongoing work is focusing on improving model performance.

Future Work: A more realistic geometry can be obtained using an image-derived left ventricle mesh. We will implement our model formulation using the deformable left ventricle mesh. Moreover, noise will be minimized by either increasing the mesh resolution and/or applying a low-pass filter as long as the signal contains no high-frequency information.