A COMPUTATIONAL ELECTROMECHANICAL MODEL FOR THE MYOCARDIUM INCLUDING FIBER AND SHEET DISARRAY

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Summary Fiber orientation is a key factor in the processes governing the cardiac cycle. However, the disarray of the fiber alignment found both in healthy and diseased myocardial tissues is largely ignored in many modeling studies. A fully electromechanical coupled ventricular model is, therefore, presented where a novel dispersion model for both the active and passive responses is used. The results show that the myocardial disarray plays an important role when modeling cardiac behavior, especially for diseased hearts.

INTRODUCTION

Under healthy conditions the heart pumps blood around the circulatory system with a remarkable efficiency. A heartbeat is initiated with an electrical activation in which sarcomeres generate the active stress which makes the heart contract. This process is regulated via both the active and the passive electrical and mechanical material properties of the myocardium. The structure of the myocytes in the left ventricle (LV) is often considered to be aligned in a right-handed helical pathway from the endocardium towards the mid-wall, and a left-handed helical pathway from the mid-wall towards the epicardium. Additionally, four to six myocyte cells are bundled together in a sheet structure that vary in orientation both transmurally and in the apicobasal direction, thus, constituting an orthotropic material structure [5].

There is a strong alignment of the myocytes in a healthy heart with an angular dispersion (AD) of the fiber orientations of only about 12–15°. In a diseased state, however, the AD is often increased as seen, e.g., in hypertrophic cardiomyopathy (HCM) where the AD is about 25° [6]. The AD in the sheet structure has not been studied in great detail, but experiments show that the orientations are fairly dispersed in healthy hearts [1]. Therefore, a structurally based orthotropic myocardial model has been developed, incorporating the dispersion of both myocytes and sheet orientations [3]. The model is based on a circular von Mises distribution function developed for the dispersed collagen structure evident in arteries [4].

The dispersion affects both the passive structural response of the myocardium as well as the active electrically driven response through a dispersion parameter which may be fitted to histological data of fiber/sheet dispersion, as seen in Fig. 1 (top panel). The nearly incompressible, hyperelastic passive material response is governed by a strain-energy function while the active response originates from an evolution equation. Both these responses are coupled via the Cauchy stress tensor in an additive decomposition [2].

METHOD

Four modified isochoric invariants defined as $\bar{I}_1 = \mathrm{tr}\overline{\mathbf{C}}$, $\bar{I}_{4\,\mathrm{f}} = \mathbf{f}_0 \cdot \overline{\mathbf{C}}\mathbf{f}_0$, $\bar{I}_{4\,\mathrm{s}} = \mathbf{s}_0 \cdot \overline{\mathbf{C}}\mathbf{s}_0$ and $\bar{I}_{8\,\mathrm{fs}} = \mathbf{f}_0 \cdot \overline{\mathbf{C}}\mathbf{s}_0$, are used where $\overline{\mathbf{C}} = J^{-2/3}\mathbf{C}$ is the modified isochoric right Cauchy Green tensor, $J = \det\mathbf{F}$ is the volume ratio, $\mathbf{F}$ is the deformation gradient, and $\mathbf{f}_0$ and $\mathbf{s}_0$ are the undeformed fiber and sheet orientations, respectively. The passive behavior of the myocardium is described by the strain-energy function

$$\overline{\Psi}_{\mathrm{p}} = \frac{a}{2b}\{\exp[b(\bar{I}_1 - 3)] - 1\} + \sum_{i=\mathrm{f,s}} \frac{a_i}{2b_i}\{\exp[b_i(\bar{I}^*_{4\,i} - 1)^2] - 1\} + \frac{a_{\mathrm{fs}}}{2b_{\mathrm{fs}}}\{\exp[b_{\mathrm{fs}}\bar{I}^2_{8\,\mathrm{fs}}] - 1\},$$

where $\bar{I}^*_{4\,i} = \kappa_i\bar{I}_1 + (1 - 3\kappa_i)\bar{I}_{4\,i}$, $i \in \{\mathrm{f}, \mathrm{s}\}$ is a modified invariant. Using a von Mises probability density function, the dispersion parameter κ_i is fitted to the AD-data in the fiber f and sheet s directions, respectively, as shown in Fig. 1 a)-c). The passive stress tensor is given by $\boldsymbol{\sigma}_{\mathrm{p}} = 2J^{-1}\mathbf{F}(\partial\overline{\Psi}_{\mathrm{p}}/\partial\mathbf{C})\mathbf{F}^{\mathrm{T}} - p\mathbf{I}$, and an active stress tensor is defined as $\boldsymbol{\sigma}_{\mathrm{a}} = J^{-1}S_{\mathrm{a}}\hat{\mathbf{h}}_{\mathrm{a}}$, where S_{a} is a scalar-valued active stress given by an evolution equation, and $\hat{\mathbf{h}}_{\mathrm{a}}$ is a normalized dispersed structure tensor given by

$$\hat{\mathbf{h}}_{\mathrm{a}} = \frac{\kappa_{\mathrm{f}}}{1 - 2\kappa_{\mathrm{f}}}\mathbf{I} + \frac{1 - 3\kappa_{\mathrm{f}}}{1 - 2\kappa_{\mathrm{f}}}\hat{\mathbf{f}} \otimes \hat{\mathbf{f}},$$

where $\hat{\mathbf{f}}$ is a normalized deformed fiber direction vector. The active stress $\boldsymbol{\sigma}_{\mathrm{a}}$ is incorporated in the model using an additive decomposition of the Cauchy stress tensor $\boldsymbol{\sigma}$, according to $\boldsymbol{\sigma} = \boldsymbol{\sigma}_{\mathrm{p}} + \boldsymbol{\sigma}_{\mathrm{a}}$ and a LV model is created incorporating fiber and sheet orientations where the influence of the dispersion parameters are examined. The two-component Windkessel equation is coupled to the LV model and governs the applied pressure on the endocardial wall.

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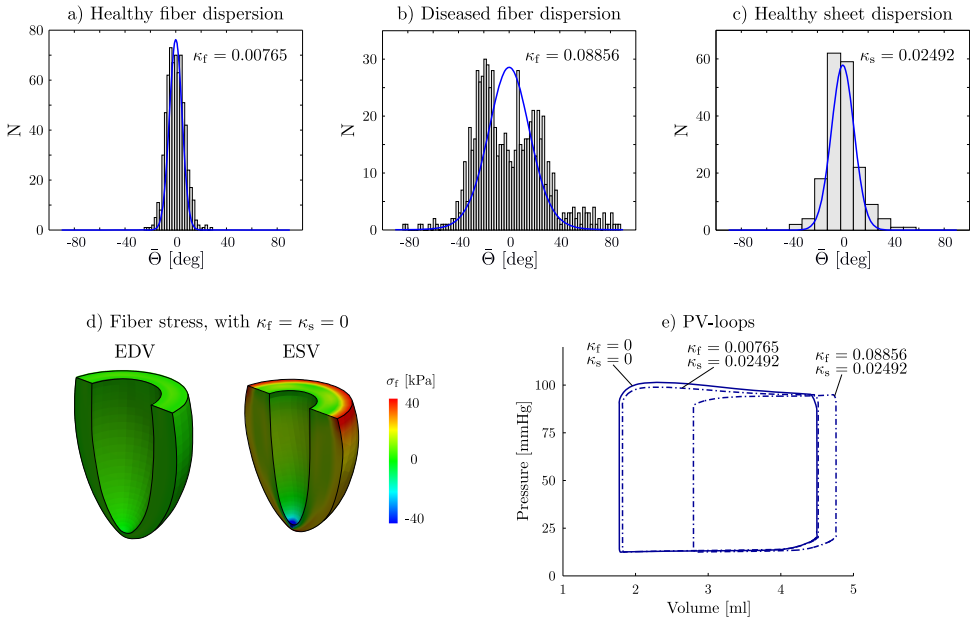

Figure 1. Fit of the κ_i parameter to histogram data for fiber and sheet arrays, adapted from [6, 1] (top panel). a) Healthy fiber dispersion with $\kappa_f = 0.00765$; b) diseased fiber dispersion with $\kappa_f = 0.08856$; c) healthy sheet dispersion with $\kappa_s = 0.02492$. Results from simulation of a LV model (bottom panel). d) Cauchy stress in the fiber direction, i.e. σ_f, at EDV and ESV; e) PV-loops using different dispersion parameters.

The electrical and mechanical models are weakly coupled, meaning that first the solution of the electrical quantities is calculated on a static mesh of the ventricular model, using The Cardiac Arrhythmia Research Package (CARP) [8], which is built on top of the MPI-based library PETSc. The relevant electrophysical parameters are then used in a separate subsequent simulation of deformation and stress analysis performed in FEAP [7].

RESULTS AND CONCLUSION

The model is implemented in a FE framework and simulations show that the level of dispersion has a great effect on deformations and stresses that develop in myocardial tissues. For example, as shown in the pressure volume (PV) loops in Fig. 1 e), when using a fiber dispersion parameter consistent with HCM diseased myocardium, there is a significant increase of end diastolic volume (EDV) and end systolic volume (ESV) (configurations shown in Fig. 1 d)) as when compared to a case of healthy (or no) dispersion. Therefore, an accurate description of dispersion must be considered carefully, especially for the diseased myocardium why the model presented here may advantageously be considered in, e.g., growth and remodeling of HCM.

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