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CARDIAC CONTRACTION MODELING USING A VARIATIONAL MULTISCALE APPROACH

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INTRODUCTION

Computational modeling of the heart dynamics and function can help further our understanding of different heart diseases, which are still the dominant cause of death in the world. The pumping action of the heart is dictated by the active contraction of the myocardium, which is commonly modeled as an incompressible material [1]. Numerically satisfying the incompressible constraint is quite challenging as it leads to a saddle point problem and the well-known *inf-sup* condition [2]. Common methods to enforce incompressibility include Lagrange multiplier methods and penalty methods [3]. They usually require high-order elements for vectors in order to satisfy the *inf-sup* condition and can be computational expensive.

Inspired by the application of stabilized finite element methods in fluid dynamics, researchers have been developing the counterpart in solid mechanics [2,4]. In such approach, the momentum balance is stated in a rate form (velocity), and the incompressibility is enforced through divergence of velocity equaling zero. The introduction of stabilization terms enables application of linear elements in modeling of problems involving large deformations.

In this study, we couple the new stabilized finite element method for mechanics with an active strain model to simulate the cardiac contraction. A classic block contraction case is used to demonstrate the capability of the solver.

METHODS

Computational Biomechanics: The governing equations for the passive responsive of the myocardium expressed in the current configuration are:

$$\dot{\boldsymbol{u}} = \boldsymbol{v} \tag{1}$$

$$\rho\dot{\boldsymbol{v}} = -\nabla_x p + \nabla_x \cdot \boldsymbol{\sigma}_{dev} \tag{2}$$

$$\nabla_x \cdot \boldsymbol{v} = 0 \tag{3}$$

Here, $\boldsymbol{u}$, $\boldsymbol{v}$ and p are the displacement vector, velocity vector and pressure. $\boldsymbol{\sigma}_{dev}$ is the deviatoric stress tensor, and its expression is determined by the constitutive relationship of the material. It is noted that the above governing equations are very similar to the Navier-Stokes equations, where the momentum equation is expressed in the rate form and the incompressibility is enforced through the divergence of velocity. Moreover, the volumetric component of the stress is grouped into the pressure, while $\boldsymbol{\sigma}_{dev}$ strictly represents isochoric parts. Equations (1-3) are solved by the variational multiscale method developed by Ju & Marsden [4] using linear elements.

Active Strain Model: In the active strain model, the deformation gradient follows a multiplicative decomposition [5]

$$\mathbf{F} = \mathbf{F}_\mathrm{E}\mathbf{F}_\mathrm{A} \tag{4}$$

Here, $\mathbf{F}_\mathrm{E}$ is the passive elastic deformation and $\mathbf{F}_\mathrm{A}$ is the active deformation of the heart muscles initiated by electric signals. The assumption here is that the energy is only stored during the passive elastic process and not in the active deformation process. Hence, the first PK stress in the reference configuration is

$$\mathbf{P}(\mathbf{F}) = \det(\mathbf{F}_\mathrm{A})\frac{\partial W(\mathbf{F}_\mathrm{E})}{\partial \mathbf{F}_\mathrm{E}}\mathbf{F}_\mathrm{A}^{-T} \tag{5}$$

where $W(\mathbf{F}_\mathrm{E})$ is the strain energy.

The active deformation gradient is usually expressed as

$$\mathbf{F}_\mathrm{A} = \left(1+\gamma_f\right)\mathbf{f_0}\otimes\mathbf{f_0} + \left(1+\gamma_s\right)\mathbf{s_0}\otimes\mathbf{s_0} + \left(1+\gamma_n\right)\mathbf{n_0}\otimes\mathbf{n_0} \tag{6}$$

Here, $\mathbf{f_0}$, $\mathbf{s_0}$ and $\mathbf{n_0}$ are the fiber, sheetlet and norm directions, respectively. γ_f is the active shortening in the fiber direction and $\det(\mathbf{F}_\mathrm{A}) = 1$ due to the incompressibility. Different choices of γ_s and γ_n will lead to different activation models. The transversely isotropic model can be recovered by setting $\gamma_s = \gamma_n = 1/\sqrt{1+\gamma_f} - 1$. The orthotropic activation model developed by Rossi et al. [6] requires $\gamma_n = k\gamma_f$. The parameter k, which is set to 4 in the current study, links the

active deformation in the microscopic scale and macroscopic scale and helps to capture the wall thickening during contraction. The transmurally heterogeneous orthotropic model, which takes into account the transmural variation of contractibility of muscle fibers, can be expressed as a linear combination of the previous two models [7], i.e. $\gamma_n(\lambda) = (1-\lambda)k\gamma_f + \lambda\left(1/\sqrt{1+\gamma_f} - 1\right)$, where λ is the transmural coordinate.

RESULTS

A classic block contraction case is used to demonstrate the accuracy of the proposed method. The computational domain is a cube with unit length, i.e., $\Omega_0 = [0, 1] \times [0, 1] \times [0, 1]$. The fiber and sheetlet directions align with Y and X axes respectively (see the wireframe in Fig. 1a). The domain is discretized by tetrahedral elements. The widely-used fiber reinforced hyperelastic material model is used to describe the passive response of the myocardium slab [1]. The parameters can be found in Rossi et al. [6].

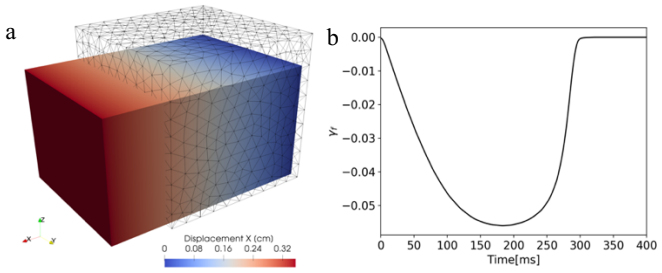

Figure 1: (a) The wireframe represents the reference configuration and the color map shows the displacement under orthotropic activation. (b) Temporal evolution of fiber shortening.

Since the propagation of electrical signal is usually much faster than the mechanical activation, the fiber shortening can be considered homogeneous within the computational domain. Instead of solving the electrophysiology for the active fiber shortening, it is prescribed during the simulations and its evolution is plotted in Fig. 1b. Symmetric boundary conditions are applied at planes X = 0, Y = 0 and Z = 0, while a stress-free boundary condition is enforced on the other faces.

The deformation at maximum fiber shortening is plotted in Fig. 1a. The fiber shortening triggers a more aggressive shrinkage along the norm direction (Z direction). Due to the incompressible constraint, this leads to a substantial wall thickening along X axis at this phase.

Fig. 2 tracks the temporal history of the displacement at the center point of face X=1. The orthotropic model leads to nearly 40% maximum wall thickening, which is in good agreement with the physiological value [8]. On the other hand, the transversely isotropic model only produces 3% maximum wall thickening. Moreover, results from the current study match closely with those extracted from Rossi et al. [6]. It is worth emphasizing that linear tetrahedral element is used in the current study while high order elements were used in the other study.

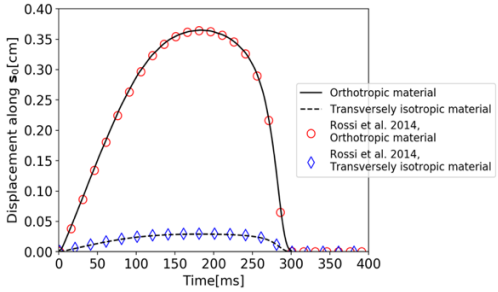

Figure 2: Displacement along the sheetlet direction at [1.0, 0.5, 0.5]. The lines are from the current study.

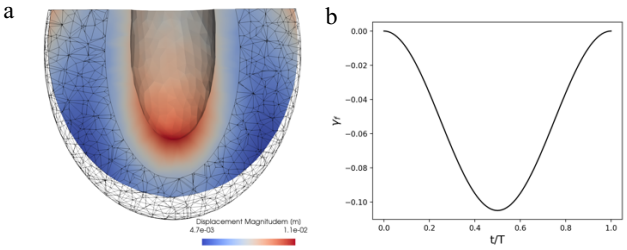

Figure 3: (a) Idealized LV model; the color map shows the deformation under transmurally heterogeneous orthotropic activation. (b) Temporal evolution of fiber shortening.

The capability of the proposed method is further demonstrated by simulating the active contraction of a left ventricle (LV) model shown in Fig. 3a. The fiber direction $\boldsymbol{f}_0$ varies linearly in the transmural direction from −60° at the epicardium to +60° at the endocardium. Details about the geometry can be found in Ref. [7]. The transmurally heterogeneous orthotropic model is used to simulate the LV contraction under prescribed fiber shortening in Fig. 3b. It is observed that the endocardium generally shows larger deformation than the epicardium and the apical region undergoes largest displacement, which are in agreement with clinical observations and findings in [7].

DISCUSSION

The current study successfully demonstrates the capability of the new stabilized finite element method in modeling muscle that undergoes large deformation. Translating the incompressible condition from Jacobian determinant equal one to zero velocity divergence enables application of tools developed for fluid flow in solid dynamics, such as the variational multiscale approach. This leads to efficient modeling of myocardium contraction with linear finite elements at accuracy comparable to higher-order elements. Application of this method in modeling idealized LV contraction shows qualitative agreement with previous literature and clinical observations. More in depth validation is ongoing and for a broader range of examples.

Individually, computational fluid dynamics and solid mechanics have each made significant inroads into understanding cardiac function and heart diseases. The current method provides a unique perspective to couple these physics using a unified theoretical and numerical framework to study cardiac fluid-structure-interaction.

There are two common models for active contraction: active stress and active strain models. We employed the latter. While it is more difficulty to implement, a main advantage of this model is that the ellipticity of the stress is guaranteed even under substantial deformation [5]. However, the active stress model is more widely used due to its intuitiveness and ease of implementation. A comparison of these two models under the same numerical framework is ongoing.

ACKNOWLEDGEMENTS

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