SIMULATING THE MECHANICS OF MYOCARDIAL TISSUE USING STRONGLY SCALABLE PARALLEL ALGORITHMS

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Abstract: Due to preferential orientations of fibers, such as collagen or myocytes, the modeling of the mechanics of myocardial tissue leads to anisotropic and highly nonlinear material models. For micro-anatomically realistic geometries the computational effort to handle these sophisticated models is very challenging and demands the usage of strongly scalable parallel algorithms. In this context we mention two possible approaches and show numerical examples.

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Modeling

For the numerical simulation of the elastic behavior of biological tissue we consider the stationary equilibrium equations

$$- \text{Div } FS(\mathbf{u}, \mathbf{x}) = 0, \quad \text{for } \mathbf{x} \in \Omega$$

(1)

where the function \(h\) and the state variables \(\eta\) depend on the respective cell model, see CellML (www.cellml.org).

The isotropic component of the material, i.e. the underlying non-collagenous and non-muscular matrix, is modeled using the well-known Demiray model (\(\Psi_{\text{iso}}\)). Due to preferential orientations of fibers (Fig. 1) the modeling of myocardial tissue leads to an anisotropic material behavior, which is incorporated in \(\Psi_{\text{aniso}}\). From the histology of the myocardium we observe two main fiber networks, myocytes (\(f\)) and collagen fibers (\(s\)). Following Holzapfel and Ogden [1] the anisotropic contribution is then modeled using an exponential structure of the type

$$\Psi_{\text{aniso}} = \frac{a_i}{2b_i} \left\{ \exp\left[a_i(I_i(C, f_i) - 1)^2\right] - 1 \right\},$$

(5)

for each fiber group \(i = f, s\). The invariants \(I_i(C, f_i)\) represent the stretch in a fiber direction \(f_i\) and \(a_i, b_i\) are positive parameters. An additional term, structurally comparable to (5), models the interaction between the two fiber networks.

Active Stress

The active stress component is generated by the electrical activation in the myocardial tissue. In this context we make use of the bidomain equations which describe the spread of electrical activation in the heart, e.g., see [2], to compute the transmembrane voltage \(V_m\). A scalar-valued active stress term \(S_a\) is then retrieved by

$$S_a = h(V_m, \eta, \mathbf{u}),$$

(6)

Figure 1: Right and left ventricle of a rabbit heart where the black lines indicate the myocyte network. The mesh has 547 680 vertices and 3 073 529 tetrahedral elements.
Hence, the active contribution to the second Piola-Kirchhoff stress tensor is obtained by

$$S_a = S_a I_f^{-1} (f_f \otimes f_f),$$  

(7)

where $f_f$ is the main orientation of the myocyte network and $I_f$ was already introduced in (5). For more details concerning the modeling of cardiac materials see [1, 3].

**Numerical Methods and Results**

In order to obtain a numerical solution of Eqs. 1-7 we use variational and finite element techniques. To solve the non-linear system we perform a linearization to apply Newton’s method:

$$K'(u^k) \Delta u = F(u^k) - K(u^k), \quad u^{k+1} = u^k + \Delta u.$$  

(8)

In this equation $K'(u^k)$ is the tangent stiffness matrix generated by using finite element techniques, $u^k$ is the displacement vector at Newton step $k$ and $F(u^k)$ captures the loads acting on the boundary of $\Omega$. $\Delta u$ is the update to be calculated from the linearized system (8). For a fine grid, resulting from detailed geometries, this linearized system of equations involves a very large number of degrees of freedom, which can not be handled by direct solving methods or single core algorithms in a reasonable time.

Hence, we have to apply parallel solving methods such as the finite element tearing and interconnecting (FETI) method, see [4], or algebraic multigrid (AMG) methods, see [5], which both share the same principle: a decomposition of the domain $\Omega$ into subdomains (Fig. 3).

The strong scalability of the implementation up to 256 cores is illustrated by means of Fig. 2 on the example of the FETI method. We obtain similar results using the AMG approach.

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**Bibliography**


