Parallel FEM Simulation of Electromechanics in the Heart

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Abstract. Cardiovascular disease is the leading cause of death in America. Computer simulation of complicated dynamics of the heart could provide valuable quantitative guidance for diagnosis and treatment of heart problems. In this paper, we present an integrated numerical model which encompasses the interaction of cardiac electrophysiology, electromechanics, and mechanoelectrical feedback. The model is solved by finite element method on a Linux cluster and the Cray XT5 supercomputer, kraken. Dynamical influences between the effects of electromechanics coupling and mechanic-electric feedback are shown.

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INTRODUCTION

Cardiovascular disease is the leading cause of death in America. There have been intensive research efforts on developing accurate computer models to advance the understanding on the mechanisms of cardiovascular dynamics [1], yet many challenges remain in computational cardiovascular dynamics.

In this work, we present a parallel numerical platform which integrates properties of cardiac electrophysiology, electromechanics, and mechanoelectrical feedback to investigate the multi-scale multi-physics problems of cardiovascular dynamics. The coupled electrical and mechanical problem is solved using finite element method based on updated Lagrangian formulation. Computations are carried out on a Linux base cluster and the CRAY XT5 supercomputer, Kraken [2].

MODELING CARDIAC ELECTROMECHANICS

Cardiac electromechanics is governed by the following equations:

\[
\begin{align*}
\frac{\partial v}{\partial t} - \nabla \cdot (D \cdot \nabla v) + I_{ion} &= 0 \\
\nabla \cdot \sigma + b &= 0
\end{align*}
\]

(1)

where the two unknowns are \( v \) and \( x \). Here, \( v \) represent the transmembrane potential in a cell, \( x \) represents spatial coordinate, \( t \) represents time, \( D \) is the diffusion tensor, \( I_{ion} \) represents total current through the cell membrane, \( C_m \) represents membrane conductance, \( \sigma \) is the Kirchhoff stress, and \( b \) represents body force in the heart. Since deformation changes the coordinate of a cell, mechanical contraction directly affects the voltage dynamics through the diffusion term. The influence of voltage to stress is through active contraction of the cell.

Denote the surface domain of the heart by \( \partial \Omega \). The reaction-diffusion equation, which is the first part of (1), satisfies no-flux boundary condition,

\[
\frac{\partial v}{\partial n} = \nabla_v \cdot n = 0 \quad \text{on} \ \partial \Omega
\]

(2)

where \( n \) is the outward surface normal on \( \partial \Omega \).

For the equilibrium equation, the second part of (1), either the natural boundary condition or the essential boundary condition is imposed. The natural boundary condition is defined as

\[
\sigma \cdot n = 0 \quad \text{on} \ \partial \Omega
\]

(3)
Meanwhile, essential boundaries conditions are imposed at points which are fixed to ensure that the mechanical problem is well defined:

\[ \mathbf{x} = \bar{\mathbf{x}} \text{ on } \partial \Lambda \]  

(4)

where \( \partial \) indicates the domain where the essential boundary condition is imposed on.

The propagation of electrical excitation of cardiac cells regulates the excitation induced contraction, which is crucial for the heart’s pumping function.

The model follows the pioneering work of Hodgkin and Huxley [3]:

\[ \frac{\partial \mathbf{v}}{\partial t} + \frac{I_{\text{ion}}}{C_m} = 0 \]  

(5)

The current platform has incorporated three different ionic models, including Beeler-Reuter (BR) model [4], Fox-McHarg-Gilmour (FMG) model [5] and Shiferaw et al. model [6]. In this paper, Fox-McHarg-Gilmour model is used. The \( I_{\text{ion}} \) function is given as follows:

\[ I_{\text{ion}} = -(I_{\text{stim}} + I_{\text{Na}} + I_{\text{K}} + I_{\text{Cl}} + I_{\text{K}}^r + I_{\text{Na}} + I_{\text{K}} + I_{\text{Ca}} + I_{\text{Ca}}^r + I_{\text{Na}}^r + I_{\text{Ca}}^r) \]  

(6)

where we have introduced an addition current, \( I_{\text{sac}} \), to account for stretch-activated channels.

Equation (6) is augmented by a number of ordinary differential equations (ODEs) describing conductance of different channels.

Then, the displacement of the heart is represented by

\[ \mathbf{x} = \Phi(X, t) \]  

(7)

which is a map between the initial configuration and the configuration at time \( t \).

In this work, the updated Lagrangian formulations are used and more accurate for nonlinear, large deformations. The updated Lagrangian description is characterized by making the material points remain coincident with mesh points. Therefore, the Lagrangian description simplifies the imposition of boundary conditions since the element boundaries of the mesh remain coincident with material boundaries. However, since Lagrangian meshes deform with material, the mesh may become distorted if the deformation of the heart is too large.

For each time step \( \Delta t \), the displacement of a material point is defined by the difference between its current position and its previous position:

\[ \mathbf{u}(X, t) = \Phi(X, t) - \Phi(X, t - \Delta t) \]  

(8)

The displacement \( \mathbf{u}(X, t) \) is governed by the linear momentum equilibrium equation

\[ \nabla \cdot \sigma(X, t) + \mathbf{b}(X, t) = 0 \]  

(9)

**COMPUTATIONAL APPROACH**

The operator splitting method [7], is used to reformulate the reaction-diffusion equation of cardiac electrophysiology. The ordinary differential equations are solved by the explicit Euler method.

Using classical Galerkin procedure yields the following equations,

\[ G_x = \int_{\Omega} \nabla \cdot (\delta \mathbf{x}) : \sigma dV - \int_{\partial \Omega} \delta \mathbf{x} \cdot \mathbf{n} da - \int_{\Omega} \delta \mathbf{x} \cdot \mathbf{b} dV = 0 \]  

(10)

\[ G_v = \int_{\Omega} [\delta \mathbf{v} \frac{\partial}{\partial t} + \nabla \cdot (\delta \mathbf{v}) \cdot (\mathbf{D} \cdot \nabla \mathbf{v})] dV - \int_{\partial \Omega} \delta \mathbf{v} \cdot \mathbf{n} da = 0 \]  

(11)

Applying the natural boundary conditions to Equations (10) and (11) leads to

\[ G_x = \int_{\Omega} \nabla \cdot (\delta \mathbf{x}) : \sigma dV - \int_{\Omega} \delta \mathbf{x} \cdot \mathbf{b} dV = 0 \]  

(12)

\[ G_v = \int_{\Omega} [\delta \mathbf{v} \frac{\partial}{\partial t} + \nabla \cdot (\delta \mathbf{v}) \cdot (\mathbf{D} \cdot \nabla \mathbf{v})] dV = 0 \]  

(13)

The conventional FEM isoparametric Galerkin procedure is followed to discretize the continuous weak form equations. The \( \theta \)-implicit Euler method is utilized in the discretization of the equation.
SOFTWARE IMPLEMENTATION

A parallel software package is implemented in C++. The resulted linear system equations are solved using the Trilinos package [8]. Simulations are performed on the supercomputer, Kraken, located at the National Institute for Computational Sciences at the University of Tennessee.

The package consists of four major modules: input, initialization, equations solving and output. Figure 1 shows the performance of the overall model with a mesh of 1.56 million elements and 1.64 million nodes.

![Performance of the overall model with a mesh of 1.56 million elements. The overall PDE solving time decreases quickly for the first 480 CPUs, and then much slower, and it takes the least time when using 960 CPU cores.](image1)

RESULTS AND DISCUSSION

Simulations were executed to study the influences of the electromechanics coupling and the mechanico-electro feedback. Figure 2 shows the results when a 2D heart tissue was stimulated at the center. The simulation lasted for 250ms, and snapshots of the appearance and membrane potentials of the tissue at 0.45ms, 6.45ms, 30.45ms, and 198.45ms are shown here. In top panels of Figure 2, electrical excitations spread from the center to all directions symmetrically, and in the last snapshot, which corresponds to 198.45ms after the beginning of the simulation, the central area of the tissue repolarized first. In bottom panels of Figure 2, because the fiber orientation was along vertical direction, the tissue contracted vertically. At 6.45ms after the beginning, there was an indentation in the middle of the tissue. That is because the tissue was stimulated at the center and thus more force was generated there, which stretched the tissue vertically toward the center.

![Block with stimulation at center; in top panels, only electrical diffusion is considered; in bottom panels, the block can deform. The snapshots are at 0.45ms, 6.45ms, 30.45ms, and 198.45ms respectively. The gray scale indicates the values of the membrane potential, which have units of mini voltage. In bottom panels, the deformation is shown relative to the original shape.](image2)
The mechanical deformation and electrical propagation of the membrane potential in a normal heart is shown in Figure 3. In top panels, the deformation is not considered, and in bottom panels, the electromechanics coupling is taken into account.

FIGURE 3. Mechanical deformation and electrical propagation of the membrane potential in a normal heart without contraction (top) and with contraction (bottom). The snapshots are at 1ms, 5ms, 10ms and 195ms. The gray scale indicates the values of the membrane potential, which have units of mini voltage. In bottom panels, the deformation is shown relative to the original shape.

CONCLUSION

A coupled electromechanical model for cardiovascular dynamics has been developed. Electro-mechanical simulations have been successfully carried out on both 2D structured and 3D unstructured meshes. Models of cardiovascular dynamics have potentials to facilitate diagnosis and treatment of heart diseases. To ensure the ability of such models, it is crucial to validate model development against experimental findings at each scale and from each discipline involved. High-performance computational platform will help to advance the understanding of cardiovascular dynamics, particularly for their capability in revealing detailed spatial and temporal dynamics at various scales.

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