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Primal-dual active set strategy for large scale optimization of cardiac defibrillation

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ABSTRACT

A feasibility study of optimal control techniques for cardiac defibrillation on anatomical three spatial dimensional rabbit ventricle geometry in the presence of bilateral control constraints is presented. The work addresses the numerical treatment of multi-scale and multi-domain simulations of the bidomain equations and is based on the primal-dual active set method to solve the optimality system for this large scale optimization problem. Numerical results are presented for a successful defibrillation study. Robustness of the optimization algorithm w.r.t to variations in the model parameters is demonstrated. A feasibility study for multiple small boundary control support is included as well. Finally, the numerical convergence of the optimization algorithm and the parallel efficiency is demonstrated.

1. Introduction

In this paper we present computational techniques to solve large scale optimal control problems modeling the termination of cardiac arrhythmia in electrophysiology. Cardiac defibrillation is a standard procedure to restore the regular heart rhythm. Electric shocks are delivered through specified electrodes. Computational tools have been progressively developed to study cardiac defibrillation and some of them are available for anatomical realistic geometries [2,13,19,34,35].

Here we briefly mention a widely accepted bidomain model [31,36] which describes excitation propagation and external stimulation of heart tissue. The cardiac tissue domain is denoted by Ω_H, surrounded by a bath domain Ω_B. We also set the complete domain $\Omega = \Omega_H \cup \Omega_B$. The space-time cylinder of the whole domain is denoted by $Q = \Omega \times (0, T]$ and the cardiac tissue and bath volume are denoted by $Q_B = \Omega_B \times (0, T]$ and $Q_H = \Omega_H \times (0, T]$ respectively. Designing a proper cost functional for the optimization for such complex phenomena is not trivial. In our computational modeling, a natural optimal control approach to cardiac defibrillation is to determine the control variable in such a way that the undesired values of the transmembrane voltage v are minimized based on the given desired trajectory. Abstractly the optimal control approach to cardiac defibrillation is given as follows:

$$(\mathrm{P}) \qquad \begin{cases} \min J(v, I_e), \\ e(u_b, u_e, v, w, I_e) = 0 \quad \text{in} \quad Q \quad \text{and} \quad I_e \in [-R, R], \end{cases} \tag{1}$$

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where u_b, u_e, v and w are the state variables and I_e denotes the control variable representing the external current. Moreover, R is a given maximum threshold value for the control in order to avoid damage of the tissue. Here the control input acts on the boundary of the bath domain.

The coupled system of partial differential equation constraints is expressed by $e(u_b, u_e, v, w, I_e) = 0$ which consists of the following equations

$$0 = \nabla \cdot (\bar{\sigma}_i + \bar{\sigma}_e)\nabla u_e + \nabla \cdot \bar{\sigma}_i \nabla v \quad \text{in } Q_H \tag{2}$$

$$\frac{\partial v}{\partial t} = \nabla \cdot \bar{\sigma}_i \nabla v + \nabla \cdot \bar{\sigma}_i \nabla u_e - I_{ion}(v, w) + I_{tr}(x, t) \quad \text{in } Q_H \tag{3}$$

$$\frac{\partial w}{\partial t} = G(v, w) \quad \text{in } Q_H, \tag{4}$$

together with initial and boundary conditions including the control I_e which are specified below. Here $u_e : Q_H \to \mathbb{R}$ is the extracellular potential, $v : Q_H \to \mathbb{R}$ is the transmembrane voltage, $w : Q_H \to \mathbb{R}^n$ represents the cell model state variables, $\bar{\sigma}_i : \Omega_H \to \mathbb{R}^{d\times d}$ and $\bar{\sigma}_e : \Omega_H \to \mathbb{R}^{d\times d}$ are respectively the intracellular and extracellular conductivity tensors. The term I_{tr} is the transmembrane current density stimulus as delivered by the intracellular electrode which is active during the S1-S2 stimulation protocol. Moreover $I_{ion}(v, w)$ is the current density flowing through the ionic channels and the function $G(v, w)$ determines the evolution of the gating variables, which are determined by an electrophysiological cell model, see [1] for more description on these models. We remark that the ODE holds on each mesh point of the computational geometry.

In general, compared to (2)–(4), an additional Poisson problem has to be solved when the heart is immersed in a conductive fluid, e.g. a tissue bath in an experimental context or a surrounding torso, to model in vivo scenarios.

$$0 = \nabla \cdot \bar{\sigma}_b \nabla u_b \quad \text{in } Q_B, \tag{5}$$

where $u_b : Q_B \to \mathbb{R}$ is the bath potential, and $\bar{\sigma}_b : \Omega_B \to \mathbb{R}^{d\times d}$ is the bath conductivity tensors.

As a consequence of the myocardium geometry the intra and extra cellular conductivity tensors of the tissue are anisotropic. Let $\mathbf{a}_l(\mathbf{x})$, $\mathbf{a}_t(\mathbf{x})$ and $\mathbf{a}_n(\mathbf{x})$ denote the fiber, sheet and normal to the sheet directions respectively in the orthonormal basis [19] which depends on the position in the heart. In our computational study, we assumed rotational isotropy at the tissue structure, i.e. $\sigma_n^{i,e} = \sigma_t^{i,e}$. Then the local intracellular conductivity tensor $\bar{\sigma}_i$ is expressed as

$$\bar{\sigma}_i = (\sigma_i^l - \sigma_t^i)\ \mathbf{a}_l(\mathbf{x})\mathbf{a}_l^T(\mathbf{x}) + \sigma_t^i\ I. \tag{6}$$

Here σ_l^i, σ_t^i denote the measured conductivity coefficients along the corresponding directions and I is the identity matrix. The fiber directions of the cardiac tissue have been modeled based on anatomical observations, see Fig. 2 for the distributions of the fiber orientation in our computations. A rule-based method was used to impose fiber orientations within the cardiac tissue geometry [5] using fiber angles of -60° and $+60^\circ$ at the endocardial and epicardial surfaces, respectively, and a smooth linear variation of fiber angles as a function of depth in between.

It is assumed that there is no current flow between the intracellular and extra-myocardial (bath) domains. Hence homogeneous Neumann boundary conditions are applied to the boundaries on the intracellular space:

$$\eta \cdot (\bar{\sigma}_i \nabla v + \bar{\sigma}_i \nabla u_e) = 0 \quad \text{on} \quad \Sigma_H = \partial\Omega_H \times [0, T]. \tag{7}$$

The boundary conditions for the extracellular potential on the extracellular domain were set up as a current balance between the extracellular domain and the surrounding bath domain [36]. This means, at the boundary of the tissue-bath interface the continuity of the normal component of the extracellular currents u_b and u_e is enforced:

$$\eta \cdot \bar{\sigma}_e \nabla u_e = \eta \cdot \bar{\sigma}_b \nabla u_b \text{ on } \Sigma_H. \tag{8}$$

Here η is the outward normal vector on $\partial\Omega_H$. Moreover, the extracellular potentials must also match the values at the common boundary:

$$u_e = u_b \text{ on } \Sigma_H. \tag{9}$$

We assume that no current flows out of the bath domain and therefore we use homogeneous Neumann boundary conditions except for those parts of the boundary where external stimuli are applied.

$$\eta \cdot \bar{\sigma}_b \nabla u_b = \hat{I}_e(t) \quad \text{on} \quad \Gamma_{12} \times (0, T] \tag{10}$$

$$\eta \cdot \bar{\sigma}_b \nabla u_b = 0 \quad \text{on} \quad \partial\Omega_B \backslash \Gamma_{12} \times (0, T]. \tag{11}$$

The current $\hat{I}_e$ acts as control along the boundary $\Gamma_{12} = \Gamma_1 \cup \Gamma_2$, where Γ_i, $i = 1, 2, 3$ are mutually disjoint and satisfy $\Gamma_1 \cup \Gamma_2 \cup \Gamma_3 = \partial\Omega_B$, see Fig. 1 for a pictorial representation. For compatibility reasons associated to Eq. (5) it is assumed throughout that

$$\int_{\partial\Omega_B} \hat{I}_e(t, \cdot)\, ds = 0 \quad \forall\ t \in (0, T). \tag{12}$$