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Application of optimal control to the cardiac defibrillation problem using a physiological model of cellular dynamics

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ABSTRACT

Optimal control techniques are investigated with the goal of terminating reentry waves in cardiac tissue models. In this computational study the Luo–Rudy phase-I ventricular action potential model is adopted which accounts for more biophysical details of cellular dynamics as compared to previously used phenomenological models. The parabolic and ordinary differential equations are solved as a coupled system and an AMG preconditioner is used to solve the discretized elliptic equation. The numerical results demonstrate that defibrillation is possible by delivering a single strong shock. The optimal control approach also leads to successful defibrillation and demands less total current. The present study motivates us to further investigate optimal control techniques on realistic geometries by incorporating the structural heterogeneity in the cardiac tissue.

1. Introduction

Cardiac rhythm disorders are among the leading causes of death in the industrialized world. The heart is an electrically controlled mechanical pump whose vital function is to drive blood through the circulatory system to supply organs with oxygen and metabolites. Under healthy conditions the heart fulfills this duty with remarkable efficiency by synchronizing mechanical contraction via fast, highly organized electrical activation of the ventricles, i.e. the main pumping chambers of the heart. Under pathological conditions electrical activation patterns become less regular and may, ultimately, transition to highly disorganized reentrant activation patterns, referred to as ventricular fibrillation. Under such conditions mechanical pumping function is severely impaired and sudden cardiac death ensues within minutes without appropriate interventions. The only reliable therapy to restore a normal heart rhythm is the timely application of a strong electrical shock, a procedure referred to as electrical defibrillation therapy, which terminates reentrant activations.

In this study, we explore the use of electrical defibrillation shocks to terminate reentrant activation and the implications of employing optimal control techniques to obtain optimal shock waveforms. In particular, we focus on the termination of reentrant spiral waves, a scenario which would be classified as monomorphic ventricular tachycardia in a clinical context. While non-lethal, spiral waves are often a precursor of more severe rhythm disturbances such as ventricular fibrillation.

In literature, one of the most accurate models of cardiac bioelectricity is the bidomain model, which describes both the extracellular and the intracellular potentials. Mathematically, the bidomain model consists of partial differential equations coupled with ordinary differential equations which model the associated ionic currents traversing the membranes of cardiac

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cells [14,22,29]. The bidomain model can be cast into an elliptic partial differential equation (PDE) that links the distribution of the transmembrane voltage, within the tissue to the extracellular potential, and a parabolic PDE that describes the cellular activation and recovery processes and the diffusive effect onto the adjacent tissue. Ordinary differential equations (ODEs) are employed to model the dynamical behavior of electricity in the myocardial cells. The dimension of the system of ODEs depends on the chosen membrane model. The present work focuses on the Luo–Rudy phase-I [17] ionic model which is a widely used computational model for the guinea pig ventricular action potentials. This model extends the Beeler–Reuter model [2] to enhance the representation of depolarization and repolarization phases and their interaction, its state space of the model consists of 6 gating variables and calcium concentration.

The optimal control approach to defibrillation seeks to determine an applied electrical field in such a way that a given design objective, which is, in our case, the restoration of a tissue state where the propagation of spiral waves is terminated, is minimized. For this purpose most of the tissue is driven to an excited state, such that the excitable gap, i.e. the portion of the tissue sufficiently close to the resting state to be excitable, is small, thus minimizing the space for the movement of reentrant waves. Achieving these objectives is challenging since, on biophysical grounds, defibrillation shocks always induced changes in polarization of both polarities [25,21]. That is, the application of shocks always induces hyperpolarization in different regions of the heart, even regions which were refractory prior to shock administration may recover excitability, a phenomenon referred to as shock-induced deexcitation [11]. The optimal extracellular current density, injected through a set of electrodes, establishes an extracellular potential distribution which can dampen the voltage gradients in the tissue during the post shock simulations.

The optimal control approach is based on minimizing a properly chosen cost functional

$$J(v, I_e) \tag{1}$$

depending on the extracellular current I_e as input and on the transmembrane potential v as one of the state variables. In previous work [8,7,6] the controller action representing the current delivered by electrodes was modeled as distributed force. Recently, we modeled the injected current via Neumann boundary conditions in the bidomain equations [9] using the simplified FitzHugh–Nagumo ionic model [23].

Turning to numerical aspects, it is well known that solving the bidomain equations is an inherently expensive procedure. The fast upstroke of the cardiac action potential translates into steep wave fronts in space, thus necessitating very fine spatio-temporal discretizations [32,18]. In our study, we have chosen the piecewise linear finite element method for the spatial discretization and higher order linearly implicit Runge–Kutta time stepping methods for the temporal discretization. There are numerous efforts to solve the linear algebraic system of the bidomain models efficiently [20]. Here we have chosen the algebraic multigrid method [27,15,3] as preconditioner to solve the elliptic system, since it has been demonstrated that AMG is a highly efficient preconditioner for this particular problem [20]. It is well know that the building the matrix hierarchy on coarser level demands a lot of computational time for AMG. In this regard, we built the matrix hierarchy at the first iteration of the temporal loop and reused it for the subsequent time steps in the AMG solver. To solve the coupled parabolic and ODEs we used the ROS3PL [16] method which has 4 internal stages and is third order accurate. We stress that the system matrix is same during all internal stages at each time step. Therefore, we use the standard BiCGSTAB method with ILU preconditioner at each stage. Here as well, we compute the LU decomposition only once at each time step and reuse it while solving each internal stage of the ROS3PL method. In this way we could save the computational time for the matrix decomposition at each internal stage of the Rosenbrock method.

The organization of the remaining article is as follows: The bidomain model equations along with the Luo–Rudy ionic model is explained in the next section. The optimal control formulation and the derivation of the optimality system is given in Section 3. In Section 4, the numerical discretization of the primal and dual equations to solve the optimality conditions is explained. Furthermore, the solution procedure to solve the decoupled elliptic and parabolic part, as well the optimization algorithm is explained. Numerical results showing optimal defibrillation are given in Section 5. A short Conclusion section ends the paper.

2. Bidomain model equations

We denote by $\Omega \subset \mathbb{R}^2$ a bounded connected domain with Lipschitz continuous boundary $\partial\Omega$. For brevity, the space–time domain and its lateral boundary are denoted by $Q = \Omega \times (0, T]$ and $\Sigma = \partial\Omega \times (0, T]$, respectively. The dynamical behavior of the cardiac tissue is described as a two-phase medium, one phase represents the intracellular space, the other one the extracellular space. The two phases are connected by a network of resistors, representing the ion channels, and capacitors, representing a capacitative current driven across the membranes by the potential difference, see e.g. [14,22,29]. Mathematically, the complete description of cardiac electricity is given by the following well known bidomain equations which consist of a linear elliptic partial differential equation and a non-linear parabolic partial differential equation of reaction-diffusion type, where the reaction term is described by a set of ordinary differential equations:

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

$$\frac{\partial v}{\partial t} = \nabla \cdot \bar{\sigma}_i \nabla v + \nabla \cdot \bar{\sigma}_i \nabla u - I_{ion}(v, \bar{\mathbf{w}}) + I_{stim} \quad \text{in } Q \tag{3}$$