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MODELING ERROR ESTIMATES IN THE SIMULATION OF THE ELECTRICAL ACTIVITY OF THE HEART

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ABSTRACT

The periodic contraction of the heart, which pumps blood in the arterial system, is caused by the electrical excitation of the myocardial cells which respond to an electrical potential difference (action potential) across the cell membrane. The electrical activation process is initiated by the heart's pacemaker cells and propagates all over the myocardial tissue in each cardiac beat. In normal conditions, the propagation occurs in a coordinated way in a relatively short fraction of the cardiac beat, preceding the contraction of the fibers.

Irregularities in the propagation of the action potential, might have severe (even catastrophic) consequences on the functionality of the heart. Mathematical modeling and numerical simulation are precious tools which may help to understand the development of such irregularities and could be very useful in the design of proper therapies or in surgical planning.

The propagation of the action potential in the myocardial tissue can be described by a system of parabolic/elliptic equations with anisotropic conductivity tensors, describing the intra- and extracellular potentials coupled with a system of ordinary differential equations describing the ionic current through the cellular membrane (see e.g. [1]). The resulting coupled system is of reaction/diffusion type.

A widely accepted model to describe the diffusion process is the so called *Bidomain* model. It describes the intra- and extra-cellular potentials by a system of degenerate parabolic equations, with orthotropic conductivity tensors, aligned to the myocytes fibers orientation. Due to the degenerate parabolic nature of the equations, the Bidomain model leads to extremely ill-conditioned linear systems of equations at the discrete level. Although advanced preconditioning techniques based on Domain Decomposition and multigrid algorithms have been recently proposed to reduce its computational cost, the systematic use of the Bidomain model in practical applications and realistic geometrical models of the whole heart, remains extremely challenging.

For this reason, a simplified model, called *Monodomain*, has been proposed, which describes only the propagation of the transmembrane potential by a non-degenerate parabolic equation. It leads to a

numerical system of half the size of the corresponding Bidomain system and much better conditioned, making it affordable the simulation of the action potential in the whole heart. The Monodomain model can be derived from the Bidomain one, making some simplifying assumptions. Unfortunately, this model, while capturing some important features of the physics of the problem, is not able to reproduce certain patterns of propagation of the action potential that have been found experimentally and that can be predicted, instead, by the Bidomain model.

In this talk we propose a mixed Monodomain/Bidomain model, where the more accurate Bidomain description is employed only in some regions of interest, where the solution might present steep gradients, while in the remaining part of the heart the simpler Monodomain model is used.

We will describe an a posteriori modeling error estimator, which estimates in a proper norm the error committed when using the Monodomain model instead of the Bidomain one. Such estimator is then used in a model adaptation strategy, which automatically switches to the more accurate Bidomain model in the regions where the error is large. An optimal implementation of this strategy should be combined with a domain decomposition approach, to couple the two models. We will present some preliminary numerical results in this direction.

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