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Efficient simulation of cardiac electrical propagation using high-order finite elements II: Adaptive *p*-version

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ABSTRACT

We present a computationally efficient method of simulating cardiac electrical propagation using an adaptive high-order finite element method to automatically concentrate computational effort where it is most needed in space on each time-step. We drive the adaptivity using a residual-based error indicator, and demonstrate using norms of the error that the indicator allows us to control it successfully.

Our results using two-dimensional domains of varying complexity demonstrate that significant improvements in efficiency are possible over the standard linear FEM in our single-thread studies, and our preliminary three-dimensional results suggest that improvements are also possible in 3D. We do not work in parallel or investigate the challenges for adaptivity such as dynamic load-balancing which are associated with parallelisation. However, based upon recent work demonstrating that in some circumstances and with moderate processor counts parallel *h*-adaptive methods are efficient, and upon the claim that *p*-adaptivity will outperform *h*-adaptivity, we argue that *p*-adaptivity should be investigated for efficiency in parallel for simulation on moderate numbers of processors.

1. Introduction

Due to the implication of cardiac failure in a large proportion of deaths, over one-sixth of all those in the UK [1], investigating the dynamics of electrical activation in the heart is of much interest to medical science and to computational physiology. In order to gain understanding of the phenomena involved, a variety of different simulations using the monodomain or bidomain equations of cardiac electrical propagation [2,3] with varying parameters are often required. The problem is multi-scale; simulations can be required to run for hundreds of seconds in order to simulate large numbers of heart beats, but must be discretised on the sub-millisecond scale in order to capture the fast dynamics of cellular ionic currents. This necessitates 10^4–10^5 time steps, and thus solves of the spatial component of the problem, to be performed during each heart beat. Solving the spatial component can be extremely computationally demanding as it must capture highly localised spatial dynamics on a very small scale compared to the length-scales of cardiac tissue.

This computational demand means that some studies that we would like to perform are impractical or unnecessarily difficult, such as those which involve long periods of pacing to prepare for the actual investigation. In previous work [4], we demonstrated that it is possible to reduce the computational demand of highly-accurate monodomain simulations by employing high-order finite element methods in space; in this approach, the simulation domain Ω is divided into a mesh of elements of diameter h and solutions are approximated on each element by polynomials of degree p. Because solutions

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to the monodomain equation are travelling waves of electrical activation with a steep interface at the wave-front, the values of h and p are dictated by the accuracy requirements in the interface region. This means that away from the wave-front, which occupies a very small proportion of the domain at any given time, we are wasting computational effort when not using spatial adaptivity.

In this work, we address this waste, presenting an application to the monodomain system of the finite element adaptive p-version [5–9]. We designed the non-adaptive form of our method using hierarchical finite elements with an eye to adaptivity [4], meaning that in adaptive mode, to change the polynomial degree of our finite element space local to an element we can simply add or remove rows and columns in the system matrices corresponding to the introduced or removed basis functions. This avoids the time-consuming matrix reconstruction required when performing h-adaptivity, which is the only form of spatial adaptivity in the literature [10–12], and is a major reason why adaptivity is not widely used. Our adaptivity is informed by a residual error indicator which we compute on every time-step, so we can have confidence in the quality of our simulation results; this is something that we confirm via analysis of the error.

The p-adaptive finite element approach has been applied to other problems such as stress analysis [5] and thermoelasticity of anisotropic solids [7], and there is a body of theoretical results available [13–16]. To the best of our knowledge, it has never been successfully applied to the equations of cardiac electrical propagation.

The remainder of this paper is organised as follows. In Section 2 we present the error indicator that we use and the strategy that we use for adaptively setting element degrees. In Section 3 we demonstrate the scheme in practice, showing error norms in 1D and working with activation times in 2D, including simulation of domains with blood vessels passing through them and of re-entrant behaviour. We present a preliminary demonstration of the behaviour of adaptivity in 3D. Our results are analysed in Section 4, and some error bound analysis is presented in Appendix A. A video of our scheme at work on a re-entrant wave is available in the online supplementary material.

2. Methods

2.1. Governing equations

We work here with the monodomain system of equations, more details about which can be found in [2]. The system is written as

$$C_m \frac{\partial u}{\partial t} - \frac{1}{\beta}\nabla\cdot(\sigma\nabla u) - I_{ionic}(u,w) = I_{stim}(x,t) \quad \text{in } \Omega, \tag{1a}$$

$$\frac{\partial w}{\partial t} - g(u,w) = 0 \quad \text{in } \Omega, \tag{1b}$$

$$u(x,0) = u_0(x) \quad \forall x \in \Omega, \tag{1c}$$

$$\hat{n}\cdot(\sigma\nabla u) = 0 \quad \text{on } \partial\Omega, \tag{1d}$$

$$w(x,0) = w_0(x) \quad \forall x \in \Omega, \tag{1e}$$

which describes the propagating waves in the transmembrane potential u which coordinates contraction of cardiac myocytes. Here, x is a point in the l-dimensional myocardial domain Ω, Ω has boundary $\partial\Omega$ which is often polygonal due to the methods used to generate it from cardiac MRI [17], outward-pointing unit surface normal $\hat{n}$ to $\partial\Omega$, transmembrane potential $u(x,t)$, initial conditions u_0 and w_0, (possibly anisotropic) conductivity tensor $\sigma(x)$ time t, cell membrane capacitance C_m, cell surface area to volume ratio β and current $I_{total}(u,w,x,t) = I_{ionic}(u,w) + I_{stim}(x,t)$, consisting of the transmembrane ionic current $I_{ionic}(u,w)$ as described by the cell model and the stimulus current $I_{stim}(x,t)$ as determined by the experimental protocol. g describes how the m non-diffusing cell model state variables $w(x,t) = (w_1(x,t),\ldots,w_m(x,t))^T$ vary in time. In this work, we use the Luo–Rudy Phase I (LR91) cell model [18], modified according to [19], for g and I_{ionic}; these quantities describe how the cardiac cells locally affect the transmembrane potential u.

2.2. Discretisation

The finite element approach for the space discretisation of Eq. (1a), is outlined in what follows. We work with the Hilbert space $\mathcal{H}^1(\Omega)$ and the norms

$$\|\chi\|_{m,q} = \left(\sum_{0\leqslant|\alpha|\leqslant m}\int_\Omega |D^\alpha\chi|^q\,dx\right)^{\frac{1}{q}},$$

with $\alpha = (\alpha_1,\ldots,\alpha_n)$, $|\alpha| = \sum_{i=1}^n \alpha_i$ and $D^\alpha := D_1^{\alpha_1}\ldots D_n^{\alpha_n}$, where $D_i := \frac{\partial}{\partial x_i}$ [20]. In what follows, for convenience we shall often write $(a,b) = \int_\Omega a\cdot b\,dx$.

We cast the transmembrane potential PDE from System (1) into its weak form: find $u \in \mathcal{H}^1(\Omega)$ such that for all $\chi \in \mathcal{H}^1(\Omega)$,