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Lattice Boltzmann method for parallel simulations of cardiac electrophysiology using GPUs

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ABSTRACT

This work presents the lattice Boltzmann method (LBM) for computational simulations of the cardiac electrical activity using monodomain model. An optimized implementation of the lattice Boltzmann method is presented which uses a collision model with multiple relaxation parameters in order to consider the anisotropy of the cardiac tissue. With focus on fast simulations of cardiac dynamics, due to the high level of parallelism present in the LBM, a GPU parallelization was performed and its performance was studied under regular and irregular three-dimensional domains. The results of our optimized lattice Boltzmann parallel implementation for cardiac simulations have shown acceleration factors as high as 500× for the overall simulation and for the LBM a performance of 419 mega lattice updates per second was achieved. With near real time simulations in a single computer equipped with a modern GPU these results show that the proposed framework is a promising approach for application in a clinical workflow.

1. Introduction

Recently the fields of computational modeling and medical image processing have advanced significantly allowing the biomedical engineering community to focus on the development of patient-specific models. In the context of cardiac electrophysiology, such personalized models offer the ability to better understand the heart in pathological conditions, to develop and improve new therapies and also to optimize complicated procedures. However, before introducing these computer models for clinicians, efficient numerical and computational techniques for the fast solution of the mathematical models underlying the complex phenomena of electrical activity of the heart have to be pursued.

There exist an extensive literature about numerical methods for the solution of the reaction–diffusion partial differential equations (PDEs) that govern cardiac electrophysiology. Among them, the finite element method (FEM) [1] is the most used method for spatial discretization, although the finite difference and the finite volume methods [2,3] and also alternative schemes such as spectral methods have been proposed [4].

In the field of computational fluid dynamics (CFD), the lattice Boltzmann method (LBM) [5] has been proposed for thesimulations of fluid flows as an alternative to these conventional methods such as the FEM. The LBM has been successfully

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applied to study several fluid flows problems [5,6] as well as in the context of biomedical engineering for simulating arterial blood flow [7]. The method has also been used to simulate reaction–diffusion equations [8,9], which are similar to the ones used to describe the electrical activity in cardiac tissue.

Among the computational techniques used for accelerating simulations of cardiac electrophysiology, some studies have shown that the use of Graphics Processing Units (GPUs) for the numerical simulations is a promising alternative which can achieve a near real time performance [10–14]. In CFD some works report a real time performance for their simulations using the LBM on GPUs [15,16].

The objective of this study is to describe the lattice Boltzmann method for the simulations of cardiac electrophysiology dynamics. The LBM for the reaction–diffusion equations of electrophysiology is first described. Then it is validated and compared against a traditional FEM solution in a benchmark problem [17]. Considering that the LBM is very suitable to parallel computing due to its high locality, it turns out that a parallel GPU implementation of the LBM for solving cardiac electrophysiology models is a promising approach for performing near real time simulations. The GPU parallelization of the cardiac LBM solver is described and discussed in terms of its performance when applied to some test problems.

2. Cardiac electrophysiology equations

Cardiac tissue is made of interconnected myocytes coupled through a conductive intracellular space, which is surrounded by a conductive fluid in the extracellular space between the cells. Mathematically this can be modeled as two interpenetrating spaces (intra- and extracellular spaces) that occupy the same volume and are separated from each other by the cardiac cell membrane.

The dynamics of cardiac electrophysiology can be described by reaction–diffusion PDEs that consider the kinetics of a single myocardial cell and ionic current flow between the intra- and extracellular spaces as well as the gap junctions, which are specialized proteins that form a intercellular connection between neighboring myocytes. The most complete mathematical model of cardiac electrical activity is the bidomain model that considers the intra- and extracellular spaces in an averaged sense as conductive regions coupled through the transmembrane current. It can be formally derived by considering conservation of intra- and extracellular currents, by coupling the two domains with the transmembrane current, and making use of Ohm's law and Kirchhoff's law; see [18] for a detailed derivation.

Since the bidomain model consists of a complex PDE system, that involves computational expensive numerical solution, it is common to assume that the intra- and extracellular domains have equal anisotropy ratios to obtain a simplified model called the monodomain model. Following this assumption the monodomain model can be obtained by a reduction from the bidomain model and is entirely written in terms of the transmembrane potential $v = v(\mathbf{x})$, defined as the difference between the intra- and extracellular potentials.

The monodomain model is given by

$$\chi \left(C_m \frac{\partial v}{\partial t} + I_{ion}(v, \boldsymbol{\eta}) \right) = \nabla \cdot (\boldsymbol{\sigma} \nabla v), \tag{1}$$

$$\frac{\partial \boldsymbol{\eta}}{\partial t} = \mathbf{f}(v, \boldsymbol{\eta}), \tag{2}$$

where χ is the surface–volume ratio and C_m is the membrane capacitance. The first term on the left-hand side is the rate of change of the transmembrane potential with time, the second term I_{ion} controls the total ion current, while the term on the right-hand side describes the diffusion. The reaction term I_{ion} is a function of v and a vector of state variables $\boldsymbol{\eta}$ and is coupled to the system of ordinary differential equations (2) that controls the kinetics of the state variables. These variables represent the dynamics of the ion channels in the cell membrane, as discussed in Cell-level models section. We consider appropriate initial conditions and apply no-flux boundary conditions, which are given by $\mathbf{n} \cdot \boldsymbol{\sigma} \nabla v = 0$, where $\mathbf{n}$ is the unit vector normal to the boundary.

In the monodomain model, $\boldsymbol{\sigma}$ is the conductivity tensor that describes the electrical properties of the tissue. Anatomic studies have shown that heart muscle is a strongly anisotropic material due to the fiber structure that comprises the tissue. Therefore, the electrical propagation is faster in the fiber direction than in the cross-fiber directions. If we consider the cardiac tissue as a transversely isotropic material, whose preferential direction is given by the fiber direction $\mathbf{a}_l$, then the conductivity is given by

$$\boldsymbol{\sigma}(\mathbf{x}) = \sigma_t \mathbf{I} + (\sigma_l - \sigma_t) \mathbf{a}_l(\mathbf{x}) \mathbf{a}_l^T(\mathbf{x}), \tag{3}$$

where $\mathbf{I}$ is the identity matrix and σ_l and σ_t are the values of the conductivity in the fiber and cross-fiber direction, respectively.

2.1. Cell-level models

The dynamics of the biophysical processes that underlie the action potential (AP) in a cardiac cell is typically described by a set of ordinary differential equations (ODEs) that model the total ion current I_{ion} through ion channels (mainly Na^+, K^+ and Ca^{2+}) in the cell membrane.