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Author: Katarzyna Jesionek, Dominik Szczerba, Jarosław Wasilewski, Marcin Kostur

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Modeling perfusion by fractal tree and stochastic dynamics

Katarzyna Jesionek[1,2], Dominik Szczerba[1,2,4], Jarosław Wasilewski[3], and Marcin Kostur[1,2]

[1] Institute of Physics, University of Silesia, 40-007 Katowice,
[2] Silesian Center for Education and Interdisciplinary Research, University of Silesia, 41-500 Chorzów, Poland
[3] Silesian Center for Heart Diseases, 41-800 Zabrze, Poland
[4] Future Processing, 44-100 Gliwice, Poland

Abstract

We model the perfusion process based on the generation of an artificial arterial tree using a fractal approach. The model can be incorporated into a multiscale framework by providing the initial points from a specific geometry of a real patient. Additionally, we supplement the small scale by diffusion processes which are captured by the GPU-accelerated stochastic dynamics. The preliminary results of such modeling and its possible application to real case scenarios are discussed.

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Keywords: fractal tree, perfusion, modeling

1 Introduction

The perfusion is a transport process in which blood delivers oxygen to the tissue. It originates in large arteries which form a fractal-like tree structure and develops into a network of capillaries. This is a complex multi-scale problem which starts at vessels of millimeter size and ends up at the micrometer scale. Additionally, there exists a mechanical coupling of the flow and muscle structure which is most clearly evidenced when flow in epicardial coronary vessels is impeded during a heart contraction. Modeling such a complex problem has drawn attention of many research groups for over four decades now.

The most common approach to perfusion is to replace the flow in complex geometry by a volume averaged quantity. It is assumed that its transport is governed by the Darcy law. The multi-scale nature of the coronary vessel geometry used to be tackled by splitting the process into so called compartments [10, 2, 5]. Inside each compartment corresponding to certain length-scale of the vessel tree the transport process is an effective Darcy flow through a porous medium. Those models usually included also the coupling to the mechanical state of the heart muscle. The reason for applying such an approach is mostly the need to obtain manageable computer model of the system which captures all essential phenomena. The multi-compartment

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formulation has been compared with another approach based on explicit network flow solution with reasonable qualitative [10] and even qualitative results [2].

On the other hand, a single-compartment approach on rigid geometry has also been used in modeling [1]. The argument for even further simplification is that perfusion takes place only around 0.5s during the diastole phase, when the heart can be considered steady.

Here we propose to model the perfusion process by a one-dimensional flow in rigid vessels. This process takes place in a artery tree which is truncated before the capillary level. The output of this model is coupled to stochastic dynamics which is constructed in such a way that it captures both the transport through capillaries and the processes in the extravascular region. The main advantage of such an approach is relative simplicity of adding additional factors like reaction terms for contrast dynamics, boundary conditions as well as anisotropy of the medium. We will use an ensemble of stochastic "tracer" particles to obtain the final state of the perfusion process. The simulation of particles will be accelerated using GPU processors.

2 The model

The main component of the model is the generation of an atrificial fractal tree resembling a real network of coronary vessels. Then we compute the blood flow in that tree and finally we use stochastic particles which reflect the last two stages of perfusion.

In the computed tomography (CT) only vessels down to about 0.5mm can be resolved. This means that the CT of a patient only provides information about highest hierarchy of the vessel network.

In the order to compensate for the missing smaller vessels we employ fractal approaches as described by Szczerba at al. [9, 8]. Using the Murray's law and statistical distributions of angles and radii available in the literature we take the real geometry extracted from the tomography and enhance it with the invisible lower grade vessels. We solve for the flow conditions in such a structure using techniques from the computational fluid dynamics (CFD). The details of the approach can be found in [9, 8].

Graphical Processing Units (GPUs) have proven to be a very effective tool for different kinds of simulation, both particle-based like Molecular Dynamics (MD) and stochastic dynamics as well as lattice-based as Lattice Boltzmann Method [3], or based on partial differential equations. Remarkably large speedups have been demonstrated for solving stochastic differential equations (SDE) [4, 7]. Here, we propose to apply this technique to modeling transport processes during perfusion.

It is known that stochastic dynamics can be used to simulate diffusion problems. Assuming the isotropic and homogeneous in space diffusion constant D one can write a system of stochastic differential equations:

$$\dot{\vec{x}} = \sqrt{2D}\vec{\xi}(t) \tag{1}$$

where $\vec{x} = \vec{x}(t)$ is a particle position, $\vec{\xi}$ is a delta correlated white Gaussian noise. Thus instead of solving a partial differential equation one can simulate large enough number of realizations of SDE (eq. 1) and estimate required quantities. It remains the question whether it is computationally better to simulate particles or distributions. Clearly, the numerical solution od the PDE gives noise free quantities. However, it is a boundary problem, which requires to use a mesh which resolves all details of the underlying geometry. On the other hand, mesh-free particle simulations are straightforward to implement but have the inherent disadvantage of providing only statistical information on quantities. Particle simulation directly benefits from

GPU architectures, as the realizations of (eq. 1) are independent on each computing core and can be performed in parallel. Therefore, we have implemented this part in our perfusion model.

In this paper we limit ourselves only to a toy-model and proceed as follows. The last branch of the vessel tree is assumed to be arterioles. Closing points of arterioles are points where the blood enters the capillaries where the oxygen is ultimately released by red blood cells and diffused into the surrounding muscular tissue of the heart. The transport in capillaries can already be modeled by the stochastic dynamics. In capillaries the particles are advected to the nearest venule. Since we do now known their position we further simplify our model by assuming that this process can be replaced by setting initial conditions for particles according to some Gaussian distribution which represents the distribution of points where oxygen or contrast marker enters the extravascular region. Then, we proceed with stochastic dynamics which models the diffusion of oxygen or contrast marker outside of capillaries. In this work we perform simulation for some time and estimate the spatial density of particles. This case is closest to a scenario of marker dynamics during the myocardial perfusion imaging[6].

3 Results

We have used a model including the generation of an artificial arterial structure. Having the whole geometry of a vessel structure, we can investigate the influence of stenosis of selected arteries on oxygen delivery to the tissue.

As a result of the fractal generation we have obtained a vessel tree, which is presented in the figure 1. In this picture the radii of all the branches of the tree are indicated. The form of the resulting vessel structure can also be noticed in this figure. Two branches on the top of the figure are the initial branches for the fractional simulation. In this simulation each vessel is divided into at most two further arteries with smaller radii, as long as the radius of the vessel reaches a specified minimal value. This algorithm is reflected in the arrangement of the generated vessels.

First we calculated the flow in the network of vessesel (1) assuming free outflow and equal pressure at the terminal vessels. Then we simulated stenosis conditions by restricting completely the flow in one of main branches - see the arrow in figure 1.

Vessels at the lowest level of the tree represent the arterioles. The next hierarchy level is capillaries which deliver the oxygen to the tissue. In this approach we assume that the capillaries are on average isotropically distributed around each arteriole's exit. Thus we include the effective transport of the red blood cell in capillaries in the initial condition of the particle model. The number of particles which started from vicinity of a given arteriole's end was proportional to its outflow. Eventually we have performed the diffusion simulation which reflected the oxygen distribution in the tissue.

We present the estimated spatial density of particles in non-stenosed and stenosed arterial tree in figure 2. It can be seen that certain regions of the muscle are depleted of oxygen.

4 Discussion and outlook

Our approach can be further used for patient specific perfusion simulation. In the first step the computed tomography provides images of largest coronary arteries. They are segmented and used as initial condition for the fractal generation of the complete coronary arterial tree down to the capillary level. Then the smallest diameter representing arterioles becomes the initial condition for stochastic dynamics which completes the model.

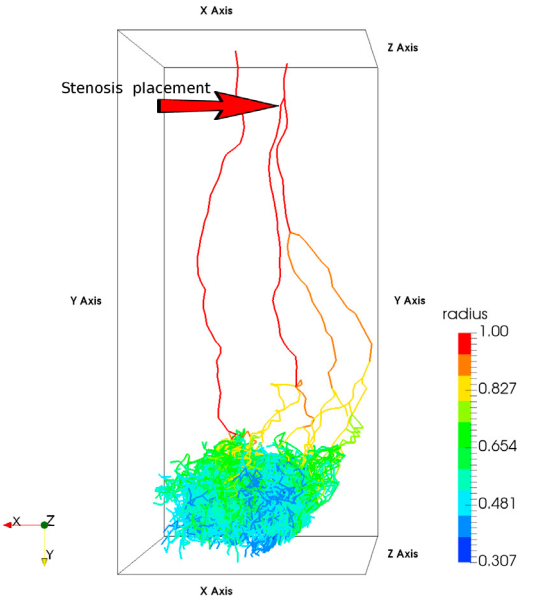

Figure 1: The fractional tree representing the structure of the vessel network. The two most top branches are the initial ones. The colors represents the radii of the different levels of the tree. The radii are normalized to the radius of the two root branches. It can be noticed that subsequent levels have smaller vessel diameters. The red arrow denotes the place where the stenosis takes place.

There are many places where the model presented in this paper can be improved. The fractal tree has to be generated in the confined geometry of a studied organ e.g. here, myocardium. To this end we have to relate the model parameters and distributions of radii and angles as well as bifurcation probabilities etc. to the real cases obtained e.g. from high precision imaging like electron microscopy. The ultimate goal is to approximate the patient's specific oxygenation map of the heart muscle. The network model must also incorporate the Fahraeus-Lindqvist effect, straightforward to implement in our framework, since it can include effective viscosities of the smallest vessels.

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References

[1] J.R. Alves, R.A.B. de Queiroz, and R.W. dos Santos. Simulation of cardiac perfusion by contrast in the myocardium using a formulation of flow in porous media. *Journal of Computational and Applied Mathematics*, 295:13–24, mar 2016.

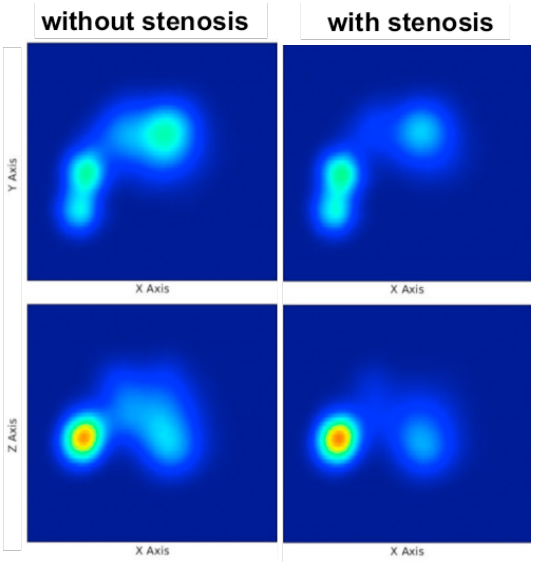

Figure 2: The estimated spatial density of oxygen for the case with and without stenosis. The upper row presents a projection of the 3d distribution on the (x,y) plane, while the bottom row a projection on the (x,z) plane. The concentration range is the same for all figures, so the relative impact of the stenosis on perfusion process is clearly visible.

[2] A.N. Cookson, J. Lee, C. Michler, R. Chabiniok, E. Hyde, D.A. Nordsletten, M. Sinclair, M. Siebes, and N.P. Smith. A novel porous mechanical framework for modelling the interaction between coronary perfusion and myocardial mechanics. *Journal of Biomechanics*, 45(5):850–855, mar 2012.

[3] M. Januszewski and M. Kostur. Sailfish: A flexible multi-gpu implementation of the lattice boltzmann method. *Computer Physics Communications*, 185(9):2350 – 2368, 2014.

[4] Michal Januszewski and Marcin Kostur. Accelerating numerical solution of stochastic differential equations with cuda. *Computer Physics Communications*, 181(1):183–188, 2010.

[5] C Michler, AN Cookson, Radomir Chabiniok, E Hyde, J Lee, M Sinclair, T Sochi, A Goyal, G Vigueras, DA Nordsletten, et al. A computationally efficient framework for the simulation of cardiac perfusion using a multi-compartment darcy porous-media flow model. *International journal for numerical methods in biomedical engineering*, 29(2):217–232, 2013.

[6] Piotr J. Slomka, James A. Patton, Daniel S. Berman, and Guido Germano. Advances in technical aspects of myocardial perfusion SPECT imaging. *Journal of Nuclear Cardiology*, 16(2):255–276, feb 2009.

[7] J Spiechowicz, Marcin Kostur, and Lukasz Machura. Gpu accelerated monte carlo simulation of brownian motors dynamics with cuda. *Computer Physics Communications*, 191:140–149, 2015.

[8] Dominik Szczerba and Gábor Székely. Macroscopic modeling of vascular systems. In *International Conference on Medical Image Computing and Computer-Assisted Intervention*, pages 284–292. Springer Berlin Heidelberg, 2002.

[9] Dominik Szczerba and Gábor Székely. Simulating vascular systems in arbitrary anatomies. In *International Conference on Medical Image Computing and Computer-Assisted Intervention*, pages 641–648. Springer Berlin Heidelberg, 2005.

[10] WJ Vankan, JM Huyghe, JD Janssen, A Huson, WJG Hacking, and W Schreiner. Finite element analysis of blood flow through biological tissue. *International Journal of Engineering Science*, 35(4):375–385, 1997.