# A Framework for the Numerical Simulation of Early Stage Aneurysm Development with the Lattice Boltzmann Method

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**Abstract** In this paper, we describe a new approach towards numerical simulation of flow induced early stage development of cerebral aneurysm. The wall shear stress gradient, computed by a CFD simulation inside a bifurcating flow channel, triggers a physiological process leading to the remodelling, and in the worst case, degeneration of the vessel walls. The lattice Boltzmann method, extended by a generic vessel wall model to allow an efficient modification of the flow geometry during run-time, is employed for simulating the modification of the vessel wall, which is considered as initial step for aneurysm formation. First results presented here show a thinning of the vessel wall at locations left and right of the apex of the bifurcation, in good agreement with experimental studies.

## 1 Introduction

Aneurysms are extreme widenings of vessels which can be, if they rupture, life threatening. For fully developed cerebral aneurysms (CA), the estimation of the rupture risk has been studied within various research projects [1], and simulation supported treatment planning is subject to ongoing research [2, 3].

In contrast to this, the process of early stage development of CA is not yet well understood. The locations of typical CAs at proximal arterial bifurcations and along the outer curvatures of intracranial vessels implicate a significant contribution of haemodynamic parameters for the formation process [4]. Experimental studies indicate that remodelling of the artery is a physiological reaction on a combination of high wall shear stress (WSS) and high WSS gradient (WSSG) near the apex of a carotid bifurcation [5].

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In this paper, we suggest a novel approach towards the lattice Boltzmann based simulation of flow induced early stage formation of cerebral aneurysm. We show that the combination of a lattice Boltzmann flow solver and a simple generic vessel wall model, reacting only to flow properties, leads to modification of the vessel walls at experimentally predicted locations. We further demonstrate the ability and performance of the underlying simulation package, which forms the basis for a later extension towards improved models coupling the output of the flow solver with a more detailed and accurate biological model for the vessel wall.

The next sections are organised in the following way: first, we introduce the medical problem and biological aspects of flow-induced remodelling of cerebral arteries. Then, our approach of extending a lattice Boltzmann flow solver for simulating this process is described and we discuss performance considerations. Preliminary results are presented in Sect. 5, followed by an outlook describing our strategy for further extending the method.

## 2 Medical Problem and Biological Process

Simply speaking, the initial process of aneurysm development can be summarised as follows: the innermost cellular layer of an artery (the endothelial cells) is able to react to the shear rate of the flow. Changed flow conditions trigger a remodelling of the vessel wall (as described in more detail below), which is an adaptive process to restore required flow rates for keeping up the functionality of the arterial network. Aneurysm development can in that way be understood as an adoption to changed flow conditions.

Modifications in the arterial network, caused either by unhealthy life-style (leading to arteriosclerosis) or surgery (e.g., by-pass or stenting), can dramatically change the distal flow properties (in the network "upstream" of the affected area). When this results in higher flow rates (e.g., through re-opened blockages or by compensating occlusion of a parallel flow channel), the affected arteries remodel in an attempt to adapt to the new flow properties. Thus, the flow provides conditions for a remodelling of the vessel wall and thinning of the internal lamina. Eventually, this degenerative biological process can initiate the formation of an aneurysm.

Since the key players for the remodelling, namely a high wall shear stress gradient, are known from animal experiments, numerical parameter studies to identify typical configurations of geometry and flow properties leading to aneurysm formation become a possibility. Although biological details are not yet fully clear, we can already figure out the general chain of some critical factors in this remodelling process, leading to aneurysm initiation.

High wall shear stress (WSS) and WSS gradient, which occur through changes in the arterial network, cause dysfunction of endothelial cells by migrating along the accelerating flow, detaching from each other [6], or even worse, getting damaged [7]. The smooth muscle cells, which lay next to the endothelium, are then exposed to these frictional forces and degraded as the endothelial cells. The

dysfunction and the decrease in the density of both endothelial cells and smooth muscle cells further lead to remodelling of the extracellular matrix, which is essential for structural maintenance of the vessel wall [8]. Due to that process, the blood vessel loses its mechanical support, and might eventually suffer local expansion through hydrostatic pressure. Therefore, this local de-stabilisation of the vessel wall can be considered as the first step in the development of an aneurysm.

A better understanding of why and where cerebral aneurysm (CA) develop can significantly contribute to fundamental research, as well as patient-specific treatment planning. As a long-term perspective, we can imagine the patient-specific estimation of the risk to develop CA, ideally prior to a planned treatment such as stenting or by-pass operation.

## 3 Simulation Approach

A complete model for the numerical simulation of flow-induced thinning of the vessel wall, as described in the previous paragraph, must fulfil the following requirements:

- Flow properties (as the wall shear stress and its gradient) must be efficiently and locally determined in complex changing geometries.
- A significant modification of the flow domain, based on computed flow properties, must be possible during run-time of the simulation.
- A numerical model simulating the previously described biological reaction of endothelial and smooth muscle cells to the flow must be developed.
- The various time-scales for the process (below seconds for the flow and above days for the aneurysm formation) have to be considered within a multi-scale simulation approach [9].

The lattice Boltzmann (LB) method allows an efficient local computation of the shear stress from the non-equilibrium part of the density distributions in non-Newtonian flow through complex aneurysm geometries [10]. Also, changing solid boundary conditions during run-time have been successfully applied in the context of medical-physics simulations [11].

Our suggested extension of a standard LB flow solver consists of iterating the following steps within the framework of a coupled simulation:

- Compute the relevant flow parameters for the current geometry, namely WSS and WSS gradient.
- Communicate these data to the biological model.
- Compute the flow-induced reformation of the cellular topology (in this paper achieved by a first simplified approach).
- Communicate the increment of the geometric boundaries to the flow solver.

For the preliminary studies presented in this paper, we reduced the complexity of the simulation approach by collapsing all biological processes into a WSSG

threshold model: above a certain WSSG value, the loss of internal elastic lamina is modelled by turning a solid lattice node into a fluid node. This first simple approach can already indicate if a remodelling of the arterial wall occurs in the expected area.

## 4 Performance Considerations

As explained in the previous sections, the modelling of the overall process is fairly complex and requires the interaction of flow and physiology. Further the flow simulation requires a relatively high resolution to accurately describe the small scale boundary layer properties. Therefore, it is essential to use a highly efficient implementation for the flow simulation, while maintaining the flexibility for interactions with the additional simulation influencing factors.

The lattice Boltzmann method in itself is very well suited for efficient computations, due to the very compact kernel, which can be highly optimised. In our solver we use an octree data structure to represent the mesh, which allows fast access and change to geometrical properties (see e.g., [12]). At the same time the computational kernel is kept in a uniform mesh, where all data is explicitly accessible as in unstructured meshes. None of the logic for the various tasks in the embedding framework has to be put into the kernel. This enables a high degree of optimisations for the kernel itself, while maintaining the flexibility to change the mesh and represent arbitrary complex geometries. Furthermore the octree data structure describing the mesh also allows for an efficient description of the partitions in a parallel computation. With the inherent knowledge about the topology of the tree it is possible to locally compute neighbour relations on each partitions and decide neighbourhood relations without larger amounts of communications. Information can be kept local to a large degree, which is very important for transient simulations as described in this work with geometric changes.

The approach was successfully shown to scale to hundred thousand processes and thus allow full usage of todays supercomputing facilities. Most of the running time is spent in the kernel, which does not notice the outer complexity of the embedding program. For this reason a high sustained performance of around 10 % of the theoretical peak performance is achieved by the overall application in serial. As the mesh organisation is completely unstructured, this performance is not influenced by the complexity of the geometry. In fact in parallel runs, the simulation might even be faster for complex geometries, due to the reduced communication surfaces by the introduced walls, which decouple the partitions in the fragmented computational domain. This is shown in the speed-up comparison between a generic cube with 134 million elements and no boundaries at all and a rather complex geometry resembling a porous media with 66 million elements in Fig. 1. The graph shows the performance in million lattice updates per second over the number of processes on the Cray XE 6 system Hermit at the HLRS in Stuttgart. As can be seen, the absolute performance is not affected by the complex geometry on small process counts and the strong scaling is even improved by the walls subdividing the overall domain.

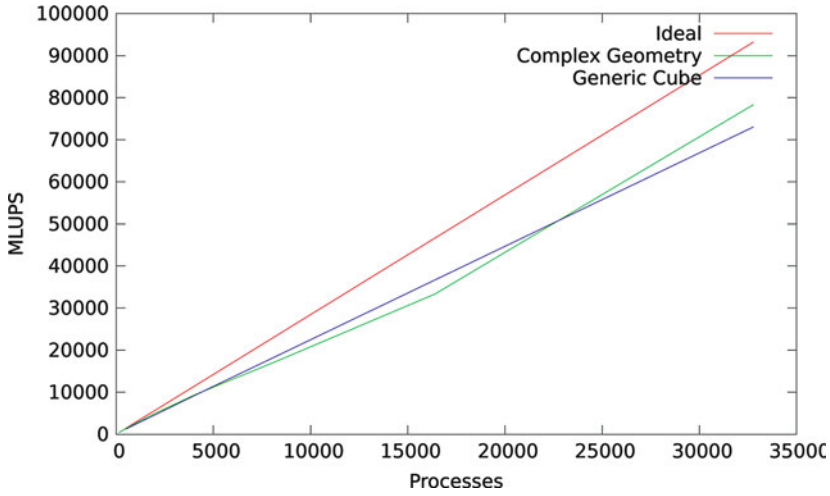


**Fig. 1** Speed up comparison on Hermit between domains with geometry and without

# 5 Results

## *5.1 Simulation Setup*

A lattice BGK implementation was extended to compute the wall shear stress from the non-equilibrium part of the density distribution function, and from that to compute a local wall shear stress gradient (WSSG). Further, the conversion of a solid node into a fluid node was enabled, if at least one fluid node adjacent to the solid node under consideration a certain threshold of the WSSG was reached. This simple approach mimics the thinning of the vessel wall, it will be replaced by a more complex model taking into account details of the biological process in later steps.

New fluid nodes were primed by an equilibrium distribution for zero flow velocity. To ensure that no artefacts from initialisation of the fluid nodes will disturb the WSS computation and further progress of the simulation, the flow field was allowed to adopt to the new configuration with a sufficient number (order of several hundred in our case) of iterations, before the WSS measurement was re-activated.

A simple initial 2-D flow geometry modelling a symmetric bifurcation was constructed (Fig. 2), with velocity inlet and pressure outlet boundaries. At the walls, half way bounce back boundary condition was employed.

The region of interest (shown in Fig. 3) had a resolution of $lx \times ly = 100 \times 100$ lattice nodes. After reaching a steady state flow profile after approximately 4,000 iterations, the functionality for measuring the wall shear stress gradient and for

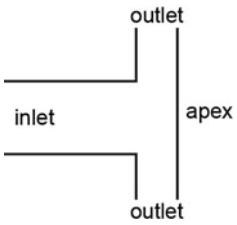


**Fig. 2** Initial flow geometry, inlet, outlets and the apex are indicated

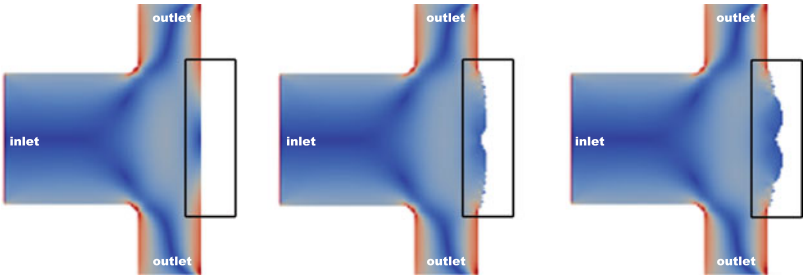


**Fig. 3** Thinning of the vessel wall, time increasing from *left* (initial geometry) to *right* (steady state). The *colour* indicates the wall shear stress (WSS, *red* = high)

turning solid into fluid cells was activated. In this preliminary study, the WSSG threshold was set to a sufficient value to produce an effect, and not yet directly correlated to data from experimental data.

## *5.2 Observations*

Near the apex of the bifurcation, a sharp increase of the WSS can be observed (see Fig. 3). As expected (and in good agreement with numerical simulations reported in [4]) the maximum of the WSSG can be observed left and right upstream of the apex.

After turning on the subroutine modelling the biological process, a reduction of solid cells (representing the vessel wall) can be observed at the regions of maximum WSSG (see Fig. 3).

This reduction of solid cells is interpreted in our model as loss of internal elastic lamina, or thinning of the vessel wall, which later would lead to the formation of an aneurysm.

After a certain time, the adaptation process comes to an end, and a steady state is reached.

### *5.3 Discussion*

The resulting reduction of solid cells left and right downstream the apex of the bifurcation correlates with the experimentally observed thinning of the vessel walls, and the location is qualitatively in good agreement with data reported in the literature (see e.g., [4]). Our much simplified initial approach of simulating the first step in a flow induced aneurysm growth can therefore be considered as qualitatively successful, in spite of the simplicity of the model employed.

## 6 Conclusion and Outlook

We presented a new model for the numerical simulation of flow induced early stage cerebral aneurysm development. First simulation results, produced with a simplified approach based on an extended 3-D lattice Boltzmann solver, show results in qualitatively good agreement with experimental data.

In the future, this approach will be extended in various ways to produce quantitatively correct results in patient specific geometries:

- The biological routines, currently only acting on one threshold parameter to turn solid cells into fluids, will significantly be refined and extended, to take into account relevant biological processes for a correct simulation of the cell migration.
- With this extended biological model, it might become necessary to integrate the lattice Boltzmann (LB) flow solver into a multi-scale simulation environment, to take into account the different time-scales of biological and flow processes.
- A three-dimensional flow geometry will be employed, and the half way bounce-back wall boundary condition will be replaced by a more sophisticated model, to allow an accurate computation of the wall shear stress.
- Transient inlet- and outlet boundary conditions will be employed for a physiological flow velocity and pressure of the cardiac cycle. This might require coupling the flow solver to a systemic model.
- A structure model has to be coupled, in order to simulate the actual development of the aneurysm shape, caused by a mechanical reaction of the thinning vessel wall to the flow.

This complete model will be a coupled simulation of transient fluid flow in changing geometries, a biological model for flow induced degeneration of arterial cells, and a structure mechanics model to compute the aneurysm shape from flow properties and mechanical properties of the degenerating vessel wall. Performance and efficiency of the solver are necessary prerequisites for simulating the complex coupled biology and flow processes. In this paper we demonstrated the applicability of the underlying approach and the suitability of the flow solver and its extensions.

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