Fluid and Structure Mechanical Modeling and **Simulation of Carotid Arterial Plaques** 10

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Introduction

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Stroke is one of the most common reasons of death and permanent disability worldwide. 25% of strokes can be traced back to the rupture of an atherosclerotic plaque in the carotid bifurcation [1]. Therefore critical carotid plaques are treated surgically in recent medicine. The treatment implies perioperative risk of stroke and that is why surgical indication for patients has to be improved. Thereby, modeling and simulation of local haemodynamics and mechanical stresses in carotid plaques is a promising approach. In this work, modern imaging techniques and application of Finite Element Methods (FEM) are used to investigate carotid plaques while different mechanical quantities are evaluated.



Validation of the segmentation of the carotid plaque: (left) MR data; (middle) HE-staining; Figure 4: (right) EvG-staining. Good overall correlation is found between segmented components and histological slices. Letters (a) - (e) refer to components introduced in Figure 1.

Results

Methods



Processing of a carotid plaque from excision (left) to the virtual 3D model (right): Figure 1: (a) calcifications, (b) lipid pool, (c) fibrous cap, (d) intimal tissue (e) lumen.

The plaque is excised and then scanned with a Small-Animal-Scanner to gain 3D magnetic resonance (MR) data with high resolutions of 0.1x0.1x0.1mm³ voxel size. The image data is then segmented into three components namely intimal tissue, calcifications and lipid pools (Figure 1). Additionally the geometry of the inside lumen is extracted. The segmentation process has been validated by a correlation of histological slices with the MR data (Figure 4).



Windkessel parameters at the outlets can be adjusted to obtain patient-specific flow rates and velocities in the ACE and ACI during FSI-simulations (Figure 5).



Figure 5: FSI-simulation at systolic flow conditions (left); profiles of maximum flow velocities for the ACC, ACE and ACI approximating patient specific measurements (right).

In the fluid field of FSI-simulations, *in vivo* pressure has been achieved through the windkessel BCs on both outlets. Compared to plain structural simulations, a significant pressure drop occurs along the stenosis of the plaque (Figure 6).







The Geometry is meshed hex dominantly (Figure 2). The adventitial layer is extruded at the abluminal surface of the plaque mesh. The complete mesh consists of 130638 nodes on structural and 22339 nodes on fluid side. Blood is modeled as a Newtonian incompressible fluid. Material models for atherosclerotic tissue are taken from literature (Figure 3). FE fluid-structure-interaction (FSI) calculations are performed using the multi-physics solver BACI [2]. Thereby, patient-specific measurements (luminal pressure and flow) are used to adjust the Boundary Conditions (BCs).



FSI-simulation with pressure distribution during the systolic phase in the fluid (left); Figure 6: slices through the structure visualizing von Mises stresses distribution (right).

The luminal surface of the intima is focused in terms of causing rupture and stroke. Maximal von Mises stresses in the intimal tissue occur in the fibrous cap between lumen and lipid pool.



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Cross section of the FSI-model displaying distribution of structural von Mises stresses. Figure 7: Highest intimal stress is found next to the next to the lipid pool.

Conclusion

Precise FE models of atherosclerotic carotid plaques can be created using the presented process. BCs can be adjusted to patient-specific measurements. In order to improve clinical decision for surgery, some more models have to be simulated to specify the mechanical quantities capable of quantifying the individual patient's risk of stroke.

