Fluid-structure interaction in an intracranial aneurysm: The effect of patient-specific wall thickness on the hemodynamic flow

S. Voß^{1,*}, P. Berg², S. Glaßer³, T. Hoffmann⁴, G. Janiga⁵

¹ Department of Fluid Dynamics and Technical Flows, University of Magdeburg, Universitätsplatz 2, Magdeburg 39106, Germany, Samuel.Voss@st.ovgu.de

² Department of Fluid Dynamics and Technical Flows, University of Magdeburg, Universitätsplatz 2, Magdeburg 39106, Germany, Philipp.Berg@ovgu.de

³ Department of Simulation and Graphics, University of Magdeburg, Universitätsplatz 2, Magdeburg 39106, Germany, Glasser@isg.cs.uni-magdeburg.de

⁴ Department of Neuroradiology, University Hospital Magdeburg, Leipzigerstr. 44, Magdeburg 39120, Germany, T.Hoffmann@ovgu.de

⁵ Department of Fluid Dynamics and Technical Flows, University of Magdeburg, Universitätsplatz 2, Magdeburg 39106, Germany, Gabor.Janiga@ovgu.de

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Cerebral aneurysms are pathologic dilations of a cerebral artery, a phenomenon that affects approximately 2-6 % of the adult population [1], [2]. Statistically, in 10 cases per year out of 100 000 a cerebral aneurysm ruptures and causes a subarachnoid hemorrhage [3], [4]. Treatment options exist in the clinical routine such as coil embolization or flow diverter implantation. Since the treatment involves interventional risks, clinicians have the objective to treat only critical aneurysms. Therefore, parameters are needed to reliably describe the risk of rupture.

Based on computational fluid dynamics (CFD) simulations in patient-specific aneurysm models, wall shear stress was associated with vessel wall remodeling. In particular, its effect on the endothelial vessel layer, which regulates the wall growth, was already described [5]. Interestingly, both low as well as high wall shear stresses have been related to aneurysm rupture [6].

Some investigations also consider non-rigid vessel walls by applying fluid-structure interactions. Because of wall deformation the hemodynamics change and therefore the wall shear stress distribution vary compared to rigid wall computations [7]. Due to the lack of clinical data, the vessel geometry is often simply generated by extrusion of the lumen surface. However, differences to simulations with the assumption of rigid vessel walls can be

noticed [8]. The objective of the present study is the evaluation of hemodynamic changes connected to the wall behavior. For this purpose, blood flow simulations with 1) rigid or flexible walls and 2) walls of constant or patient-specific thickness are considered.



Figure 1: Exemplary illustration of the inner and outer vessel wall surface in a pig kidney acquired with intravascular ultrasound (left). Velocity field in a patient-specific intracranial aneurysm using fluid-structure interaction (right).

This study is based on patient-specific vessel data of a cerebral artery including geometry information of the inner and outer vessel surface and, therefore, of the corresponding wall thickness. Different blood flow simulations were performed taking into account rigid walls as well as flexible walls with constant and variable thickness. The comparison addresses the differences in wall shear stress in the fluid and stresses inside the wall.

Computations accounting for fluid-structure interactions of the patient-specific artery walls show significant changes in the stresses and particularly in the locations of highest values compared to simulation based on rigid walls. Hence, the consideration of patient-specific and therefore locally variable wall thickness appears to be essential to understand and predict aneurysm rupture. However, the collection of required vessel wall data remains difficult and further research is needed in order to include additional material behavior of the vessel wall into a simulation environment.

REFERENCES

- [1] G. J. Rinkel, M. Djibuti, A. Algra, J. van Gijn Stroke 29 (1998) 251.
- [2] J. M. Wardlaw, P. M. White Brain 123 (2000) 205.
- [3] O. G. Nilsson J. Neurol. Neurosurg. Psychiatry 69 (2000) 601.
- [4] L. H. Pobereskin J. Neurol. Neurosurg. Psychiatry 70 (2001) 340.
- [5] M. Shojima, M. Oshima, K. Takagi, R. Torii, M. Hayakawa, K. Katada et al. Stroke 35 (2004) 2500.
- [6] H. Meng, V. M. Tutino, J. Xiang, A. Siddiqui Am. J. Neuroradiol. 35 (2013) 1254.
- [7] Y. Bazilevs, M.-C. Hsu, Y. Zhang, W. Wang, X. Liang, T. Kvamsdal et al. Comput. Mech. 46 (2010) 3.
- [8] A. Valencia, P. Burdiles, M. Ignat, J. Mura, E. Bravo, R. Rivera, J. Sordo Comput. Math. Methods Med. 2013 (2013) 1