

MULTI-SCALE HAEMODYNAMIC SIMULATIONS OF A1 AND ACOA FENESTRATIONS AND ANEURYSM GROWTH

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Introduction

Fenestration of the anterior cerebral artery (ACA) is rare and is frequently associated with cerebral aneurysm [Friedlander, 1996]. Saccular aneurysms associated with fenestrations have been well documented and they are often associated with cerebral hemodynamic changes [San-Galli, 1992]. The pathogenesis of A1 segment of the ACA fenestrations has not been well established so far. It is generally accepted that formation of A1 fenestrations may derive from remnant of an embryonic plexiform anastomosis between the primitive olfactory artery and the ACA, or from abnormal regression [Padget, 1948] of the vasculature. The aim of our study was to characterise the flow patterns of fenestrations and to compare haemodynamic forces acting on the vessel wall in the presence or in the absence of a cerebral aneurysm.

Methods

We used angio-CT images from two patients with fenestrations of A1 and ACoA to generate 3D surface vascular models and meshes using vmtk software (<http://www.vmtk.org>). We then performed flow simulations studies with the open-source haemodynamics solver Gnuid (<http://github.com/lorbot/gnuid>), based on a fully implicit implementation of the well known pressure correction scheme [Guermond, 1998], a discontinuous approximation for the velocity unknown and a continuous approximation for the pressure [Botti, 2011]. In order to properly assume boundary conditions in the cerebral arterial network, we used a multi-scale approach by coupling Gnuid with pyNS (<http://archtk.github.com>), a 0D/1D open-source patient-specific framework that implements wave-propagation elements based on an approximated velocity profile function [Huberts, 2012].

Results

We compared two cases of very similar A1 and ACoA fenestrations, one of them is associated with a saccular aneurysm involving

the proximal end of the fenestration. As shown in Figure 1, the results of the CFD analysis show that the vascular wall of the fenestration is subjected to very low peak wall shear stress (WSS, arrows), as compared to the initial segment of the A1 (2.4 vs. 11.5 dynes/cm² and 2.2 vs. 13.6 dynes/cm², respectively for the two patients).

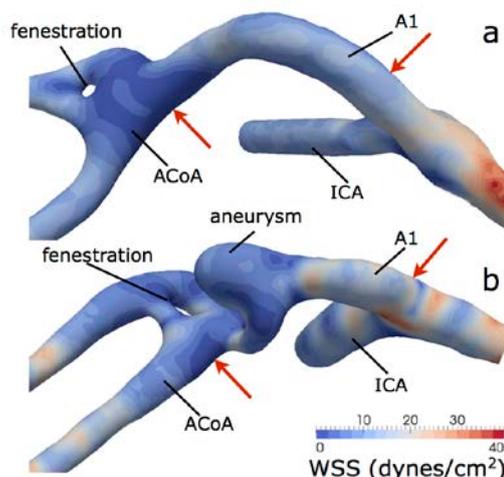


Figure 1. WSS [dynes/cm²] during systolic peak in the vascular model without (a) and with the saccular aneurysm (b).

Discussion

Our results indicate that a region of abnormally low wall shear stress is associated with A1 and ACoA fenestrations. Since in this location cerebral aneurysms are frequently observed, it is likely that aneurysm formation and growth is related to non adequate mechanical stimulation of the endothelial layer lining the vessel wall as observed in physiological condition [Boussel, 2008].

References

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