Computational analysis of anterior communicating artery aneurysm shear stress before and after aneurysm formation

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Abstract. It is widely accepted that complexity in the flow pattern at the anterior communicating artery (AComA) is associated with the high rate of aneurysm formation at that location observed in large studies. The purpose of this work is to study associations between hemodynamic patterns, and AComA aneurysm initiation by comparing hemodynamics in the aneurysm and the normal model where the aneurysm was computationally removed. Vascular models of both right and left circulation were independently reconstructed from three-dimensional rotational angiography images using deformable models after image registration of both images, and fused using a surface merging algorithm. The geometric models were then used to generate high-quality volumetric finite element grids of tetrahedra with an advancing front technique. For each patient, the second anatomical model was created by digitally removing the aneurysm. It was iteratively achieved by applying a Laplacian smoothing filter and remeshing the surface. Finite element blood flow numerical simulations were performed for both the pathological and normal models under the same personalized pulsatile flow conditions imposed at the inlets of both models. The Navier-Stokes equations were numerically integrated by using a finite-element formulation. It was observed that aneurysms initiated in regions of high and moderate WSS in the counterpart normal models. Adjacent or close to those regions, low WSS portions of the arterial wall were not affected by the disease. These results are in line with previous observations at other vascular locations.

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1. Introduction
The anterior communicating artery (AComA) is a unique vascular location that ideally receives blood from two sources of inflow and redistributes it toward the anterior part of the brain through two efferent arteries [1]. However, aneurysms of the AcomA complex are more likely to have hypoplastic A1 segments [2] or to have exclusive filling angiographically from one A1 segment in up to 78% cases [3]. Those AComA aneurysms receiving blood from both A1 segments have flow patterns dependent of individual flow rates and wave forms, which may result in regions of elevated wall shear stress (WSS) that change their locations during the cardiac cycle [4]. It is accepted that complexity in the flow pattern is associated with the high rate of aneurysm formation observed in large studies [5-7].

Wall shear stress (WSS) play an important role in the formation, growth and ultimately rupture of cerebral aneurysms [8,9]. A previous computational hemodynamic study showed a possible association between high maximum WSS at the systolic peak with rupture in a cohort of AComA aneurysms [10]. In that study ruptured aneurysms had in average as much as twice maximum WSS as in the unruptured group. This result is in line with previous experimental studies [11] and computational studies at other locations [12]. Additionally, in a large study including 210 cerebral aneurysms at different locations it was found a statistical significant association between rupture and WSS distributions with elevated maximum WSS, high flow concentration and small impingement size [13]. Cebral et al. also showed a connection between location of aneurysm blebs and regions of high WSS in models where blebs were virtually removed [14]. In another study, Kulcsar et al. presented a relationship between initiation, and coexistence of high WSS and high positive spatial WSS gradient, observed in three-patient scanned before and after aneurysm formation [15]. The normal vasculature had been incidentally imaged when investigating other pathologies. Other investigators reported possible associations between low shear stress and either rupture [16,17] or blister formation [18].

The purpose of this work is to study associations between hemodynamic patterns and aneurysm initiation by comparing AComA hemodynamics between the original aneurysm model and the vascular model where the aneurysm was computationally removed. Patients included in this study were not scanned before aneurysm formation. Therefore, there is no evidence to determine whether or not the original vascular configurations were affected by the disease. In this first pilot study, in order to minimize the effects of the reconstruction method, only a selected group of patients whose aneurysms affected a limited region of the AComA and the parent artery was considered.

2. Materials and methods

2.1 Patients, images and vascular reconstruction
Three patients with cerebral aneurysms in the AComA were selected from our data base. In order to minimize the dependency of the results on the reconstruction of the normal vascular models, all images presenting an extension of the disease towards further regions beyond the aneurysm neck were rejected for this study. Bilateral rotational scans were obtained to visualize both avenues of flow into the anterior communicating artery (AcomA) aneurysm using a Philips Integris System (Philips Medical Systems, Best, The Netherlands). These images were obtained during a 180° rotation and imaging at 15 frames per second for a total of 8 seconds. The corresponding 120 projection images were reconstructed into a 3D dataset of 128 128 128 voxels covering a field of view of 54.02 mm on a dedicated Philips Integris workstation. The voxel resolution was therefore 0.422 mm. These data were exported into a PC for mathematic vascular modelling using a previously presented methodology [19,20,10]. Each pair of images was aligned using landmark-based rigid registration, and each model was reconstructed using isosurface extraction and deformable models. Models were merged and edited, and aneurysms were virtually removed. In order to achieve that, neck points were identified. Aneurysm was smoothed using a Gaussian filter starting from the smaller structures, without moving points beyond the neck. Remeshing was required in order to preserve element aspect ratio after point relocation. The procedure continued until the aneurysm was completely removed.
2.2 Hemodynamic modeling
Finite element blood flow numerical simulations were performed for both the original and the pre-aneurysm models under the same flow conditions. Blood was modelled as an incompressible Newtonian fluid with attenuation 1.0 g/cm$^3$ and viscosity 0.04 Poise. The governing equations were the unsteady Navier-Stokes equations in 3D [26]. Vessel walls were assumed rigid, and no slip boundary conditions were applied at the walls. Pulsatile flow conditions derived from phase-contrast magnetic resonance measurement in a healthy subject were imposed at the inlet of the models. Flow waveforms were scaled with the inlet area to achieve a mean WSS of 15 dyne/cm$^2$ at the inflow boundary of each model. This choice is consistent with studies relating vessel area and flow rates in internal carotid and vertebral arteries [27], as well as with the principle of minimal work expressed by Murray’s law [28]. Fully developed pulsatile velocity profiles were prescribed with use of the Womersley solution [29,30]. Assuming that all distal vascular beds have similar total resistance to flow, traction-free boundary conditions with the same pressure level were applied at outlet boundaries. The Navier-Stokes equations were numerically integrated by using a fully implicit finite element formulation [19]. We computed 2 cardiac cycles using 100 time-steps per cycle, and all of the results reported correspond to the second cardiac cycle. All elements in the normal model whose distance to the aneurysm started to form. WSS distributions were computed from the blood velocity field and studied over those regions. Average WSS values were compared to those typical values in the surrounding regions.

The following list summarizes the steps presented in Sections 1.1 and 1.2 [10,14]:

- Image acquisition for both left and right circulation
- Image rigid registration using landmarks
- Image segmentation
- Isosurface extraction
- Isosurface deformable models to adjust surface point location to vascular boundary in image
- Surface merging to generate a single model with containing the whole anterior vasculature
- Identification of neck points
- Aneurysm smoothing and remeshing iteratively within neck until aneurysm is removed
- 3D tetrahedral grid generation using an advancing front technic for both pre and post models
- Flow rate scaling to impose at both internal carotid arteries (ICA)
- Computation of Womersley profile assumed as the solution at the model inflows
- Model assumptions: traction free boundary conditions at outlets, no slip boundary condition at arterial walls, Newtonian rheology and rigid walls
- Numerical integration over two cardiac cycles using a fully implicit finite element formulation
- Computation of wall shear stress from velocity field
- Wall shear stress visualization

3. Results
For all three cases, model A (without aneurysm) and model B (with aneurysm) were created. The geometric information is shown in Table 1. All the patients considered in this study had a unilateral configuration, having a dominant ICA that provides blood to the whole anterior arterial tree. Consequently, there is almost no flow through the A1 segment of the anterior cerebral artery contralateral to the dominant internal carotid artery (ICA), resulting in a reduced arterial diameter (see Figure 1). Original models for all patients are shown in Figure 1 (first and third columns), while models after digital intervention to remove the aneurysm from the original models are shown in the second and fourth columns of the same figure.
Table 1. Geometric information of vascular models

| (a) Model # | (b) # elem (A) | (c) # elem(B) | (d) Area ICA left (cm$^2$) | (e) Area ICA right (cm$^2$) | (f) Max dist $\perp$ (cm) | (g) Max dist $\parallel$ (cm) | (h) AR | (i) Loc. | (j) Unilateral |
|-------------|----------------|---------------|--------------------------|--------------------------|-------------------------|-------------------------|-------|--------|-------------|
| 1           | 1,431,826      | 1,680,751     | 0.116                    | 0.140                    | 0.90                    | 0.50                    | 1.80  | right  | yes         |
| 2           | 2,427,619      | 2,532,393     | 0.251                    | 0.177                    | 0.57                    | 0.44                    | 1.29  | left   | yes         |
| 3           | 2,056,090      | 2,151,738     | 0.238                    | 0.185                    | 0.48                    | 0.38                    | 1.26  | left   | yes         |

a) number of model; b) number of tetrahedra elements in the model A (without aneurysm); c) number of tetrahedra elements in the model B (with aneurysm); d) cross-sectional area of the left ICA at the inflow; e) cross-sectional area of the right ICA at the inflow; f) maximum length of the aneurysm perpendicular to the vessel; g) maximum length of the aneurysm parallel to the vessel; h) aspect ratio; i) location of the aneurysm at the juncture of the A1 and A2 segments of the anterior cerebral artery, and the anterior communicating artery based on the acquired images; j) the system is considered unilateral if there is basically no flow through the A1 segment contralateral to the dominant ICA. In all the three cases, both A2 segments are filled by the dominant ICA.

Figure 1. Final finite element models after surface merging of individually reconstructed right and left models: original (first and third columns) and after virtual intervention (second and fourth columns) for patients 1, 2 and 3 (first, second and third row, respectively).

WSS distributions at the systolic peak are displayed in Figure 2 after for model A (first column) and model B (second column) for all patients.

For Patient #1 a high WSS region with a peak of 120 dyne/cm$^2$ is observed in the AcomA, surrounded by a larger region with an average WSS of 50 dyne/cm$^2$. Comparison between both models
shows that the aneurysm started to form in the high WSS region. A low WSS region of 25 dyne/cm$^2$ in the feeding artery opposite to the aneurysm is also observed.

For patient #2, two disconnected high WSS regions with peak values of 75 dyne/cm$^2$ and 50 dyne/cm$^2$ were observed. The extended area affected by the aneurysm includes both regions. Adjacent to them there is a low WSS portion of the feeding vessel with values less than 10 dyne/cm$^2$. For patient #3, a extended region with WSS values ranging between 200 dyn/cm$^2$ and 340 dyn/cm$^2$ was observed, which surrounds a smaller area of moderate WSS values (120 dyne/cm$^2$). The aneurysm formed over a region with both high a moderate WSS values, which also had high WSS gradient. A low WSS region in arterial wall opposite to the aneurysm has WSS values less than 50 dynm/cm$^2$. 

Figure 2: WSS distributions at the systolic peak after virtual intervention (first column) and in the original aneurysm model (second column), for patients 1, 2 and 3 (first, second and third rows, respectively), using the same colour scale for each patient.
4. Discussion

The methodology has some potential limitations. The computational removal of AComA aneurysms may not be applied to large aneurysms with wide necks where the original vasculature may not be easily reconstructed. Ford et al. presented an approach to digital removal of saccular aneurysms [31]. Although the methodology presented there is automatic and may be helpful to process large amount of data, it may generate simplified bifurcation geometries due to the centreline based reconstruction. That may affect WSS computation in models where aneurysms where removed from those locations. Images before aneurysm formation are usually acquired by chance when the patients are screened because of another pathology or aneurysms at other locations [15], therefore they can not be used to carry out a large study. Another potential limitation is the use of non-patient-specific flow rate waveforms imposed at the inlet of our models. However, this limitation is suffered by most computational studies since flow rate measurements are not routinely acquired in clinical evaluations. This limitation is partially overcome by WSS normalization at inflow segments. Other model assumptions had limited impact on aneurismal hemodynamics, as shown in previous works [19].

5. Conclusion

The present work shows a possible association between regions of elevated WSS before aneurysm formation, and aneurysm location. This observation is in line with previously reported results from experimental studies and patient-specific blood flow finite element simulations at other vascular locations. This trend will have to be corroborated by larger studies, which also include other hemodynamic quantities and differences between unilateral and bilateral aneurysms.

References

[1] Perlmutter D, Rhoton ALJ. Microsurgical anatomy of the anterior-anterior communicating-recurrent artery complex. J Neurosurg 1976;45:259–72
[2] Kasuya H, Shimizu T, Nakaya K, et al. Angles between A1 and A2 segments of the anterior cerebral artery visualized by three-dimensional computed tomographic angiography and association of anterior communicating artery aneurysms. Neurosurgery 1999;45:89–93
[3] Charbel FT, Seyfried D, Metha B, et al. Dominant A1: angiographic and clinical correlations with anterior communicating artery aneurysms. Neuror Res 1991;13:253–56
[4] Castro MA, Putman CM, Cebral JR. Patient-Specific Computational Fluid Dynamics Modeling of Anterior Communicating Artery Aneurysms: A Study of the Sensitivity of Intra-Aneurysmal Flow Patterns to Flow Conditions in the Carotid Arteries. Am J Neurorad 2006;27:2061-2068
[5] Horiuchi T, Tanaka T, Hongo K. Surgical treatment for aneurysmal subarachnoid hemorrhage in the 8th and 9th decade of life. Neurosurgery 2005;56:469–75
[6] Leipzig TJ, Morgan J, Horner TG. Analysis of intraoperative rupture in the surgical treatment of 1674 saccular aneurysms. Neurosurgery 2005;56:455–68
[7] Brilstra EH, Rinkel GJ, van der Graaff Y. Treatment of intracranial aneurysms by embolization with coils: a systematic review. Stroke 1999;30:470–76
[8] Crompton M. Mechanisms of growth and rupture in cerebral berry aneurysms. Br J Med 1966;1:1138-42
[9] Nakatani H, Hashimoto N, Kang H, Yamazoe N, Kikuchi H et al. Cerebral blood flow patterns at major vessel bifurcations and aneurysms in rats. J Neurosurg 1991;74:258-262
[10] Castro MA, Putman CM, Cebral JR. Hemodynamic Patterns of Anterior Communicating Artery Aneurysms: A Possible Association with Rupture. Am J Neurorad 2009;30(2): 297-302
[11] Hashimoto N, Handa H, Nagata I, et al. Experimentally induced cerebral aneurysms in rats. Part V. Relation of hemodynamics in the circle of Willis to formation of aneurysms. Surg Neurol 1980;13:41–45
[12] Castro MA, Putman CM, Radaelli A, Frangi A, Cebral JR. Hemodynamics and rupture of terminal cerebral aneurysms. Acad Radiol 2009;16 (19):1201-1207

[13] Cebral JR, Mut F, Weir J, Putman CM. Quantitative characterization of the hemodynamic environment in ruptured and unruptured brain aneurysms. Am J Neuroradiol 2011;32:145-151

[14] Cebral JR, Sheridan M, Putman CM. Hemodynamics and Bleb Formation in Intracranial Aneurysms. Am J Neuroradiol 2010;31:304-310

[15] Kulcsar Z, Ugron A, Marosfo M, Berentei Z, Paal G, Szikora I. Hemodynamics of Cerebral Aneurysm Initiation: The Role of Wall Shear Stress and Spatial Wall Shear Stress Gradient. Am J Neuroradiol 2011;32(3):587-594

[16] Jou LD, Lee DH, Morsi H, Mawad ME. Wall Shear Stress on Ruptured and Unruptured Intracranial Aneurysms at the Internal Carotid Artery. Am J Neuroradiol 2008;29:1761-1767

[17] Shojima M et al. Magnitude and role of wall shear stress on cerebral aneurysm. Computational fluid dynamic study of 20 middle cerebral aneurysms. Stroke 2004;35:2500-2505

[18] Shojima M, Nemoto S, Morita A, Oshima M, Watanabe E, Saito N. Role of Shear Stress in the Blister Formation of Cerebral Aneurysms. Neurosurgery 2010; 67(5):1268-1275

[19] Cebral JR, Castro MA, Appanaboyina S, Putman C, Millán D, Frangi A. Efficient Pipeline for Image-Based Patient-Specific Analysis of Cerebral Aneurysms Hemodynamics: Technique and Sensitivity. IEEE - Transactions on Medical Imaging - Special Issue on Vascular Imaging 2005; 24(4):457-467

[20] Yim PJ, Vasbinder B, Ho VH, et al. A deformable isosurface and vascular applications. In: Sonka M and Fitzpatrick JM, ed. Medical Imaging 2002: Image Processing, SPIE Vol. 4684. San Diego, Calif: SPIE; 2002:1390 –97

[21] Castro MA, Putman CM, Cebral JR. Patient-specific computational modeling of cerebral aneurysms with multiple avenues of flow from 3D rotational angiography images. Acad Radiol 2006;13:811–21.

[22] Cebral JR, Löhner R, Choyke PL, et al. Merging of intersecting triangulations for finite element modeling. J Biomech 2001;34:815–19

[23] Löhner R. Automatic unstructured grid generators. Finite Elements Analysis Design 1997;25:111–34

[24] Löhner R. Extensions and improvements of the advancing front grid generation technique. Comp Methods Appl Mech Eng 1996;5:119 –32

[25] Löhner R. Regridding surface triangulations. J Comp Phys 1996;126:1–10

[26] Mazumdar JN. Biofluid Mechanics. World Scientific, Singapore 1992

[27] Cebral JR, Castro MA, Putman CM, Alperin N. Flow-area relationship in internal carotid and vertebral arteries. Physiol Meas 2008;29(10):585-594

[28] Sherman T F 1981 On connecting large vessels to small. The meaning of Murray’s law. J Gen Physiol 1981:78:431–53

[29] Womersley JR. Method for the calculation of velocity, rate of flow and viscous drag in arteries when the pressure gradient is known. J Physiol 1955;127:553–563

[30] Taylor CA, Hughes TJR, Zarins CK. Finite element modeling of blood flow in arteries. Comp Methods Appl Mech Engin 1998;158:155–196

[31] Ford MD, Hoï Y, Piccinelli M, Antiga L, Steinman AD, An objective approach to digital removal of saccular aneurysms: Technique and applications. The British Journal of Radiology 2009;82:56-61