Journal of Biomechanics 43 (2010) 2189–2195

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech
www.JBiomech.com

Cross-flow at the anterior communicating artery and its implication in cerebral aneurysm formation

Liang-Der Jou [a,*], Deok Hee Lee [b], Michel E. Mawad [a]

[a] Department of Radiology, Baylor College of Medicine, 1 Baylor Plaza, MS-360, Houston, TX 77030, USA
[b] Department of Radiology, Asan Medical Center, Seoul, Republic of Korea

ARTICLE INFO

Article history:
Accepted 23 March 2010

Keywords:
Hemodynamics
Cerebral aneurysms
Circle of Willis

ABSTRACT

The anterior communicating artery (ACoA) is an important element of the circle of Willis. While the artery itself is short and small, a large number of intracranial aneurysms can be found at the ACoA. Four subject-specific ACoA models are constructed from 3D rotational angiographic images. The ACoA of these models ranged from 1.7 to 2.7 mm in diameter and 1.5 to 5.7 mm in length. Pulsatile flows through these four ACoA models are studied numerically. Blood is found to move in two opposite directions simultaneously within the ACoA, giving a much higher wall shear at the ACoA. These two opposite flow streams produce a cross-flow that is dependent on the flow rates at the anterior cerebral arteries and internal carotid arteries (ICAs). A larger and shorter ACoA allows flow through the ACoA easily, leading to a greater cross-flow and higher hemodynamic forces on the artery. This cross-flow may disappear when there is a sufficient net flow for a smaller and longer ACoA. Wall shear stress can be as high as 185 Pa at smaller ACoAs, but it can be lowered by asymmetric waveforms at the ICAs. A functional circle of Willis also promotes cross-flow at both the ACoA and posterior communicating arteries.

1. Introduction

The circle of Willis is a network of cerebral arteries that are essential to the function of brain, and the anterior communicating artery (ACoA) is an important element of the circle of Willis. It connects the right and left anterior cerebral artery (ACA) and establishes co-lateral flow, permitting perfusion of the brain in case of carotid occlusion (Kluytmans et al., 1999). An experimental study on phantoms shows that the wall shear stress is almost zero at the ACoA when the ACAs are perfectly symmetric both anatomically and hemodynamically (Ujiie et al., 1996), indicating that very little flow goes through the ACoA. Numerical studies have also shown that flow rate at the ACoA is small with a fully functional circle of Willis, but it may increase as one of the internal carotid arteries (ICA) becomes stenotic (Alastruey et al., 2007; Long et al., 2008).

While these studies provide useful information on cerebral hemodynamics in case of carotid occlusion (Cieslicki and Ciesla, 2005; Moorehead et al., 2006; Alastruey et al., 2007; Devault et al., 2008; Long et al., 2008), little emphasis is given to the role of the ACoA in the formation of cerebral aneurysms (Alnaes et al., 2007). The ACoA itself is very short in length and small in size, but cerebral aneurysms are frequently observed at the ACoA. In total 35% of ruptured aneurysms are found to form at the ACoA (Weir et al., 2002), and these aneurysms also rupture when they are small (Forget et al., 2001). If the ACoA carries very little blood flow, why is it a frequent site of cerebral aneurysms? What role does the hemodynamics play in aneurysm formation at the ACoA? We study four cases of the ACoA based on subject-specific models and show that there is a non-zero cross-flow at the artery despite of zero net flow. This cross-flow raises the wall shear at the ACoA and may play a critical role in development of cerebral aneurysms.

2. Methods

Four patient-specific models of the ACoA were reconstructed from digital subtraction angiographic images using bilateral carotid injection. Three cases did not have an ACoA aneurysm, but they did have intracranial aneurysms elsewhere (Cases 1–3). Only one case had an ACoA aneurysm (Case 4). 3D rotational

Abbreviations: ACoA, anterior communicating artery; PCoA, posterior communicating artery; MCA, middle cerebral artery; ACA, anterior cerebral artery; ICA, internal carotid artery; Q, flow rate; Q_{ICA}, flow rate at the internal carotid artery; Q_{MCA}, flow rate at the middle cerebral artery; Q_{ACA}, flow rate at the anterior cerebral artery; Q_L, flow rate at the left internal carotid artery; Q_R, flow rate at the right internal carotid artery; Q_{lr}, flow rate from the left anterior cerebral artery to the right anterior cerebral artery; Q_{rl}, flow rate from the right anterior cerebral artery to the left anterior cerebral artery; Q_{cf}, flow rate of the cross-flow; Q_{net}, net flow rate at the anterior communicating artery

* Corresponding author. Tel.: +1 832 355 3419; fax: +1 713 798 8050.
E-mail address: jou@bcm.edu (L.-D. Jou).

0021-9290/$ - see front matter © 2010 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jbiomech.2010.03.039

angiographic images at 0.2 mm isotropic resolution were obtained from Siemens Axiom Artis dBA scanners (Erlangen, Germany) during clinical examinations. Diameter of the ACoA ranged from 1.7 to 2.8 mm, and the average diameter of the internal carotid artery for these cases was 4.2 mm. Selection of these patients was based on whether the ACoA could be clearly visualized from bilateral carotid injections since a unilateral injection could not provide a clear 3D anatomy of the ACoA. Models from unilateral carotid injections then were merged to form the anterior cerebral circulation network (Castro et al., 2006a, 2006b), consisting of both ICAs, middle cerebral arteries (MCAs), ACAs, and the ACoA. Posterior cerebral circulation was considered only in Case 4.

Images were first segmented and processed by Volview (Kitware; Clifton Park, New York) to produce smooth arterial surfaces. Numerical meshes were generated from these arterial surfaces by GAMBIT (ANSYS; Canonsburg, PA) at various grid sizes. Solutions for different grid sizes were compared to assure that solutions were converged and independent of grid size. FLUENT (ANSYS; Canonsburg, PA) was used for flow simulations. Blood was assumed to have a constant viscosity of 3.5 cp. Pulsatile flow profiles were prescribed at both ICAs. These profiles were from an earlier study of Ford and his colleagues in which they measured the flow rate at the ICA and vertebral artery (Ford et al., 2005). Only the six lowest harmonics of their waveform were used and modes from higher frequencies (>6 Hz) were ignored because these modes might be subject to individual differences. The carotid waveform was adjusted to achieve a mean flow rate of 4.1 ml/s at the ICA (Marshall et al., 2004). Flow division at the outlets (Q_{ACA}, Q_{MCA}) were varied to achieve different flow rates at the ACAs and MCAs. However, Q_{ACA}:Q_{MCA}=1:3 was used as a reference (Tanaka et al., 2006). At a symmetric hemodynamic condition, flow rates for the right and left ACAs are the same, and so are the right and left MCAs. With these flow rate ratios, $Q_{ACA}=Q_{ICA}/4$. A typical flow waveform at the ACA can be found later in Fig. 2. Heart rate for all simulations was 68 beats/min, and four pulsatile cycles were simulated at 200 time points per cardiac cycle. The mean Reynolds number at the ICA was 345, and Womersley number was 1.2 at the ACA.

In Case 4, the ACoA aneurysm was removed from the model by Rapidform (Rapidform; Seoul, South Korea). The original ACoA aneurysm was adjacent to the right ACA, and there was a section of the ACoA unaffected by the aneurysm. The ACoA aneurysm was first removed from the right ACA. The new right ACA was then reconstructed from the A1 to A2 segment without the aneurysm. The new section transitioned smoothly both in diameter and cross-section. The A1 and A2 segments refer to the sections of the ACA proximal and distal to the ACoA, respectively. Last, a straight ACoA was extended from the unaffected ACoA section at the left ACA to the right ACA with a constant diameter. Sharp edges at the junctions were also eliminated to avoid unrealistic high wall shear stresses.

Mesh-independent convergence was first confirmed before analyses. Four meshes with different grid sizes, ranging from 1 to 6.4 million cells, were used. The error of mean cross-flow velocity was 16% for the coarsest mesh and 2.5% for the mesh with 3.6 million. Results from the finest mesh are reported. Mesh requirement for these simulations was higher than other previous studies on the ICA aneurysms, because cross-flow converged slower than pressure and velocity, and resolving cross-flow required a finer mesh.

In Case 1 where the artery of Heubner was clearly visible, flow rate at the artery of Heubner was set to be 20% of the flow rate at the right ACA. However, a detailed analysis later showed that the flow at the artery of Heubner did not affect the qualitative flow behavior at the ACoA.

3. Results

The anatomy and cross-flow at the ACoA for the first three cases without an ACoA aneurysm are presented in Fig. 1. Velocity vector plot, streamlines, and wall shear are also shown in the figure. Both Cases 2 and 3 have bilateral mirrored carotid aneurysms, and Case 1 has an MCA aneurysm. When boundary conditions for flow rates are symmetric laterally, there is a zero net flow at the ACoA.

Streamline trafficking is evident in Cases 1 and 2; each A2 segment receives blood from both A1 segments. In these cases, two flow streams exist at the ACoA in each case, one from the left to the right (Q_{lr}) and one from the right to the left (Q_{rl}). These two flow streams, referred to as cross-flow, have the same flow rate, but they result in a zero net flow rate at the ACoA. No streamlines crossing the ACoA in Case 3 is merely a result of streamline sampling.

The flow rate of cross-flow (Q_{cf}) is defined as the amount of flow that does not contribute to the net flow rate at the ACoA. For example, $Q_{lr}=0.5$ ml/s and $Q_{rl}=0.6$ ml/s, then $Q_{cf}=0.5$ ml/s and $Q_{net}=0.1$ ml/s to the left. Mathematically, $Q_{cf}=\min(Q_{rl}, Q_{lr})$ and $Q_{net}=|Q_{rl}-Q_{lr}|$. The maximum of Q_{lr} and Q_{rl} is the sum of the net flow rate and cross-flow. The net flow rate is determined by the hemodynamic conditions specified at the boundary, but not by the anatomy. Cross-flow, on the other hand, is affected by the anatomy. Therefore, the effect of anatomy is observed only in cross-flow.

Fig. 2 presents the cross-flow and wall shear at the ACoA for Cases 1–3, and a waveform at the ACA is shown as a reference in Fig. 2A. The maximum wall shear is evaluated at the mid-section of the ACoA. Both cross-flow and maximum wall shear appear to follow the waveform at the ICA. The amount of cross-flow is 0.8 ml/s for Case 1, 0.17 ml/s for Case 2, and 0.005 ml/s for Case 3. The maximum wall shear for Case 3 is 1.5 Pa, but the maximum wall shear for Case 1 and Case 2 are 65 and 185 Pa, respectively. Fig. 2C shows the relationship between the cross-flow and maximum wall shear. For these individuals, maximum wall shear increases with the cross-flow.

Table 1 lists the dimensions, cross-flow, and maximum wall shear for these cases. Case 2 has an extremely high wall shear, even though it has a lower cross-flow than Case 1. Therefore, the higher maximum wall shear for Case 2 is a combination of higher cross-flow and smaller ACoA. Nearly 88% of the flow rate to the A2 segment of the ACA on average comes from the contra-lateral side in Case 1, and both ACAs receive blood mostly from the contra-lateral ICA. This is confirmed by angiographic images of this patient. Flow division between the MCA and ACA is also altered to achieve a different flow rate at the ACAs, and cross-flow is found to vary linearly with the total flow rate at the ACAs.

Fig. 3 demonstrates the effect of carotid asymmetry on cross-flow for Cases 1–2. Case 3 is not studied because of its low cross-flow. Q_L and Q_R are the flow rate at the left and right ICA, respectively. Asymmetric flow rates at the ICAs ($Q_R \neq Q_L$) can lead to a non-zero net flow rate at the ACoA, but the cross-flow remains. In Case 1, one of the flow streams (Q_{rl}) is varying with $Q_L - Q_R$, the flow rate difference between two ICAs, while Q_{lr} remains relatively a constant. Therefore, we observe a decrease of Q_{rl} when Q_L increases, but no increase of Q_{lr}. In Case 2, Q_{rl} remains the same when $Q_L > Q_R$. Q_{cf} decreases only when $Q_L < Q_R$. Qualitatively, Q_{cf} for both Cases 1 and 2 behaves similarly. Q_{cf} decreases when only one of the flow rates at the ICA increases: Q_L for Case 1 and Q_R for Case 2. This domination of cross-flow by one artery is probably due to the anatomy of the ACAs. For both cases, cross-flow is at its greatest for symmetric conditions. Note that the net flow rate at the ACoA is determined by the boundary conditions, and $Q_{net}=|Q_L-Q_R|/2=|Q_{rl}-Q_{lr}|$ in these cases.

Fig. 4 shows how cross-flow and wall shear are affected by the carotid flow conditions for Case 1. If blood reaches the entrances at two ICAs at different time, there will be a delay in the waveform. The phase lag between the carotid waveforms at the ICAs is set at 30° (or 73 ms), and this time delay represents an extreme condition during which the pulse wave can travel from one ICA to the other ICA through the CCAs (Salvi et al., 2008). This phase delay gives approximately 40% decrease in cross-flow and 17% reduction in maximum wall shear. The waveforms with no delay at the ICAs lead to a greater cross-flow and higher wall shear.

The entire circle of Willis is modeled in Case 4. Fig. 5A is for the model with an ACoA aneurysm. The model without the ACoA aneurysm is shown in Fig. 5B. The pressure shown in the figure is much smaller than the blood pressure variation during a cardiac cycle. Fig. 6 presents the cross-flows at the ACoA and posterior communicating arteries (PCoA). Because the vertebral waveform is different from the carotid waveform (Ford et al., 2005), the circle of Willis for Case 4 is not symmetric hemodynamically.