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Reversed Robin Hood Syndrome in the Light of Nonlinear Model of Cerebral Circulation

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ABSTRACT: The brain is supplied by the internal carotid and vertebro-basilar systems of vessels interconnected by arterial anastomoses and forming at the base of the brain a structure called the Circle of Willis (CoW). An active intrinsic ability of cerebral vascular bed maintains constant Cerebral Blood Flow (CBF) in a certain range of systemic pressure changes. This ability is called autoregulation and together with the redundant structure of the CoW guarantee maintaining CBF even in partial occlusion of supplying arteries. However, there are some situations when combination of those two mechanisms cause an opposite effect, called the Reversed Robin Hood Syndrome (RRHS). In this work we proposed a model of the CoW with autoregulation mechanism and investigated a RRHS, which may occur in case of Internal Carotid Artery (ICA) stenosis combined with hypercapnia. We showed and analyzed mechanism of stealing the blood by the contralateral side of the brain. Our results were qualitatively compared with the clinical reports available in the literature.

KEYWORDS: Cerebral blood flow, autoregulation, steal syndrome, Circle of Willis, Computational Fluid Dynamics

1. Introduction

The brain is supplied by the internal carotid and vertebro-basilar systems of vessels. Both systems begin in the aortic arch and they are interconnected at the base of the brain forming the redundant structure called the Circle of Willis (CoW). Blood is then redistributed by six major branches of the CoW to different parts of the brain. Despite the brain is only of 2% of the total human body weight, it is supplied by about 13-15% of whole blood volume [1]. Maintaining constant Cerebral Blood Flow (CBF) is a priority due to continuous oxygen and glucose demand of neurons. This is the reason why a number of protective mechanisms were created by nature.

Investigations how the local occlusion of supplying arteries or acute forms of hypotension disturb CBF are of large importance. A great number of mathematical and physical models [2- 9] of cerebral circulation were used for simulations of different pathological situations. Numerical models were based on different approaches: mechano-electric analogy, method of characterictics [6-11] or full Navier-Stokes solution using Finite Volume Method [11-13]. However, in majority of them, authors neglected autoregulation mechanisms. In this paper we presented a numerical model of blood flow in the CoW with an autoregulation mechanism, which importance will be highlighted by simulating ~~a~~ Reversed Robin Hood Syndrome (RRHS).

Existing arterial anastomoses, both on the level of the CoW and distal parts of cerebral vascular bed, can in certain situations lead to an undesirable effect of blood stealing. We are faced with this problem, when flow of blood in the ischaemic area of tissue (due to obstruction of supplying artery) is compensated through the existing vascular anastomoses from the surrounding area and, in turn, discriminate their circulation. As far as the brain circulation is concerned, the RRHS appears, when blood from the ischemic region of the brain is stolen by the non-affected, contralateral part. Such situation can occur for example, when patient with one-sided stenosis of ICA that induced decrease of ipsilateral peripheral vessels resistance (due to autoregulation)~~,~~ simultaneously suffers from sleepy apneas. In the brain tissue, the concentration of carbon dioxide during stopped respiration grows up, what induces vasodilation at the arteriolar level. On the affected side, the vessels have already been expanded (vasomotor reactivity is exhausted), so the flow resistance decreases only on the contralateral side. As a result of anastomotic connections of the CoW (mainly ACoA), the cerebral flow in the non-affected side increases, not only due to autoregulation but additionally at the expense of the affected side.

2. Material and Method

Numerical model of the CoW was prepared by using commercial software ANSYS Fluent. The geometry of the CoW was created in CAD program based on a wax model used in previous experimental studies [14] (Fig. 1).

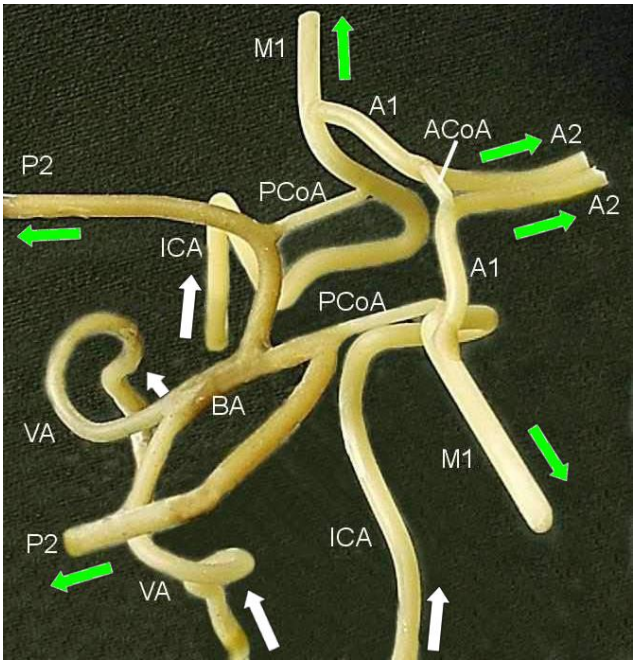

Fig. 1. Modelled geometry. Shortcuts denotes cerebral arteries: VA – Vertebral Artery, BA – Basilar Artery, P1 – Posterior Artery, M1 – Media Artery, A1 – Anterior Artery, ICA – Internal Carotid Artery, ACoA – Anterior Communicating Artery, PCoA – Posterior Communicating Artery. Arrows denotes direction of blood flow.

Created geometry was discretised using ANSYS Meshing software. Mesh dependency test was performed. Finally a mesh with about 1 600 000 elements with 5 boundary layer elements was used. Simulation was performed using finite volume method with SIMPLE algorithm to solve Navier-Stokes equations of flow. The pressure inlet boundary conditions was applied. Autoregulation model was based on the flow resistance characteristic presented in Fig. 1. As there is no such boundary condition model provided in the used software, it was implemented using c code as the user defined function.

3. Results and Discussion

The following simulations sequence were performed:

1. Reference CoW with patency of all supplying vessels
2. CoW with hypercapnia
3. CoW with ICAL stenosis
4. CoW with ICAL stenosis with hypercapnia

In the reference CoW total blood flow is on the level of 770 ml/min. Pressure and flow values in all of the distributing arteries are within the range of autoregulation. In condition of hypercapnia the total blood flow is increased by about 31 % what agree with clinical findings [15].

In the next step we simulat occlusion of left ICA. In this case, pressures in all branches of the CoW (A2, M1, P2) are lowered, particularly in that lying on the side of occlusion. Blood flow in the left anterior part of the brain is lowered by approximately 22%. Nevertheless, due to autoregulation the global CBF is maintained almost on the same level. Hypercapnia condition made CBF only slighty increased (appr. 6%) and a stealing syndrome appears. To analyze this mechanism we contrasted all considered cases in Fig. 2 on the pressure-flow autoregulation characteristic.

The orange dots denote the reference CoW case without and with hypercapnia. In the latter situation the dot lies on the constant resistance line (dashed-line). Due to symmetry of the CoW dots concerning the left and right side of the brain overlap. Dark blue dots describe the CoW with occlusion. In this case the left middle cerebral artery pressure lies below the bottom limit of autoregulation. So after desaturation of oxygen only arteries in the right side of the brain are changing their resistance. Accordingly, flow in the non-affected side increases partially at the expense of the affected side. In the presented model the steal factor calculated for the middle cerebral arteries is about 5%. According to Alexandrev [15] report of six patient with ICA/MCA oclusions, steal factor can vary beetween patients, from none existing up to 43.2 %. It depends on a specific CoW anatomy and vasomotor reactivity. In computational model we can also observe a stealing from the anterior part of the brain.

4. Conclusions

A numerical model of the CoW with implemented autoregulation mechanism was proposed. Four cases of cerebral blood flow was simulated and contrasted. It was shown that autoregulation mechanism combined with the ICA occlusion and hypercapnia could cause a Reversed Robin Hood Syndrome. It cannot be modeled without taking into account vasomotor reactivity.

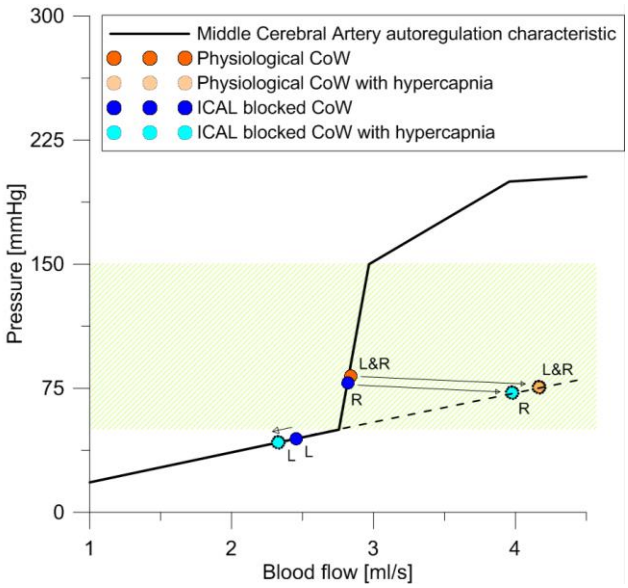

Fig. 2. Reversed Robin Hood Syndrom mechanism presented on the autoregulation curve of middle cerebral artery. Green area denotes autoregulation range.

Intracerebral blood flow modeling remains within the scope of many studies, however in a majority of proposed models the autoregulation mechanism is not taken into account. As was shown on an example of the RRHS, implementation of autoregulation mechanism is essential to capture clinically observed CBF phenomena.

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