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***Brief note***

# REVERSED ROBIN HOOD SYNDROME IN THE LIGHT OF NONLINEAR MODEL OF CEREBRAL CIRCULATION

A. PIECHNA* and K. CIESLICKI
Institute of Automatic Control and Robotics
Warsaw University of Technology
ul. św. A. Boboli 8 02-525 Warszawa, POLAND
E-mails: adam.piechna@gmail.com; k.cieslicki@mchtr.pw.edu.pl

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 the combination of those two mechanisms causes 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 the case of Internal Carotid Artery (ICA) stenosis combined with hypercapnia. We showed and analyzed the 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.

**Key words:** 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 on 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 fact that the brain is only of *2*% of the total human body weight, it is supplied by about *13-15*% of the 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, 3, 4, 5, 6 ,7, 8, 9, 10] of cerebral circulation were used for simulations of different pathological situations. Numerical models were based on different approaches: mechano-electric analogy, method of characterictics [8, 9, 10, 11, 12] or full Navier-Stokes solution using Finite Volume Method [12, 13, 14, 15]. 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 whose 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 the 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,

* To whom correspondence should be addressed

discriminate their circulation. The steal syndrome can occur both on the organ level (out-cranial) and tissue level (intracranial) dependent on the level of blocked vessel in relation to vascular tree and its anastomoses.

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 a situation can occur for example, when a patient with one-sided stenosis of ICA that induced a 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 using the commercial software ANSYS Fluent. The geometry of the CoW was created in CAD program based on a wax model used in previous experimental studies [16] (Fig.1).

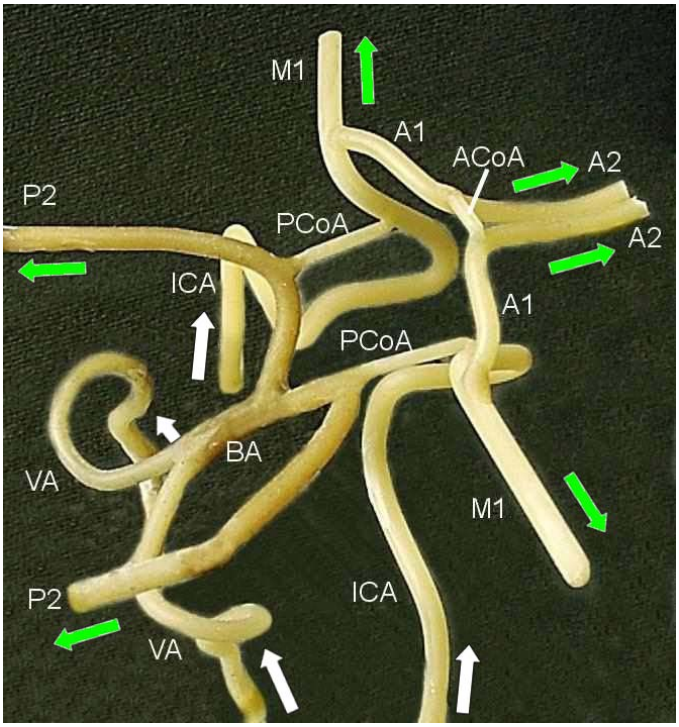


Fig.1. Modelled geometry. The abbreviations denote cerebral arteries: VA – Vertebral Artery, BA – Basilar Artery, P1 – Posterior Artery, M1 – Media Artery, A1 – Anterior Artery, ICA – Internal Carotid Artery, ACoA – Anterior Communicating Artery, PCoA – Posterior Communicating Artery. Arrows denote direction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

Diameters of the major arteries are presented in Tab.1.

Table 1. Diameters of the vessels.

| Vessel | *d[mm]* |
|---|---|
| ICA | *4.0* |
| VA | *3.0* |
| BA | *3.5* |
| A2 | *2.0* |
| M2 | *3.0* |
| P2 | *2.1* |
| PCoA | *1.5* |
| ACoA | *2.0* |

The created geometry was discretised using the ANSYS Meshing software. A 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 the finite volume method with SIMPLE algorithm to solve Navier-Stokes equations of flow. The pressure inlet boundary conditions were applied. The autoregulation model were based on the flow resistance characteristic presented in Fig.2.

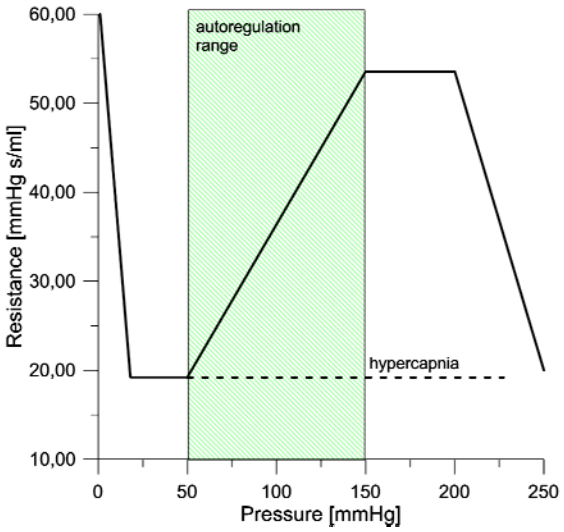


Fig.2. Model of autoregulation characteristic. The green area denotes the autoregulation zone. In the condition of hypercapnia the vessel resistance reaches minimum and is constant (arteries are expanded).

The bottom and upper limit of autoregulation was set on *50 mmHg*, and *150 mmHg* respectively. Termination resistances are different for each part of the CoW, their values were summarized in Tab.2. As there is no such boundary condition model provided in the used software, it was implemented using c code as the user defined function.

Table 2. Autoregulation model parameters.

| COW PART | MEAN BLOOD FLOW [*ml/s*] | MINIMUM RESISTANCE [*mmHg*min/ml*] | MAXIMUM RESISTANCE [*mmHg*min/ml*] |
|---|---|---|---|
| Anterior | *1.85 * 2* | *0.46* | *1.31* |
| Midle | *2.7 * 2* | *0.32* | *0.90* |
| Posterior | *2.0 * 2* | *0.44* | *1.20* |

## 3. Results

The following sequences of simulations 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

The obtained results are presented in Tab.3.

Table 3. Summary of the results. Breath Holding Index (BHI) is calculated beetween case IV and case III as $BHI=(Flow_{IV}-Flow_{III})/Flow_{III}$. The percent value in the last row denotes differences of CBF in regard to case I.

| | Reference CoW case I | | CoW with hypercapnia case II | | CoW ICAL ocluded case III | | CoW ICAL bl. with hypercapnia case IV | | BHI |
|---|---|---|---|---|---|---|---|---|---|
| | Flow [*ml/s*] | Pressure [*mmHg*] | Flow [*ml/s*] | Pressure [*mmHg*] | Flow [*ml/s*] | Pressure [*mmHg*] | Flow [*ml/s*] | Pressure [*mmHg*] | [-] |
| A2 L | *108* | *62* | *119* | *54* | *84* | *39* | *82* | *37* | *-3%* |
| A2 R | *108* | *62* | *119* | *54* | *97* | *45* | *93* | *42* | *-4%* |
| M1 R | *158* | *84* | *230* | *74* | *162* | *75* | *215* | *69* | *32%* |
| P2 R | *118* | *78* | *154* | *68* | *116* | *71* | *146* | *64* | *26%* |
| P2 L | *118* | *78* | *154* | *68* | *116* | *66* | *138* | *61* | *19%* |
| M1 L | *158* | *84* | *230* | *74* | *144* | *46* | *137* | *44* | *-5%* |
| sum | *769* | | *1007* | + 31 % | *720* | *- 6 %* | *811* | *+ 6 %* | |

## 4. Discussion

In the reference CoW the 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 the condition of hypercapnia the total blood flow is increased by about *31* % and this agrees with clinical findings [17]. The actual value of flow increment is strictly connected with the minimum resistance applied in the autoregulation model.

In the next step, we simulated 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.3 on the pressure-flow autoregulation characteristic of middle celebral artery.

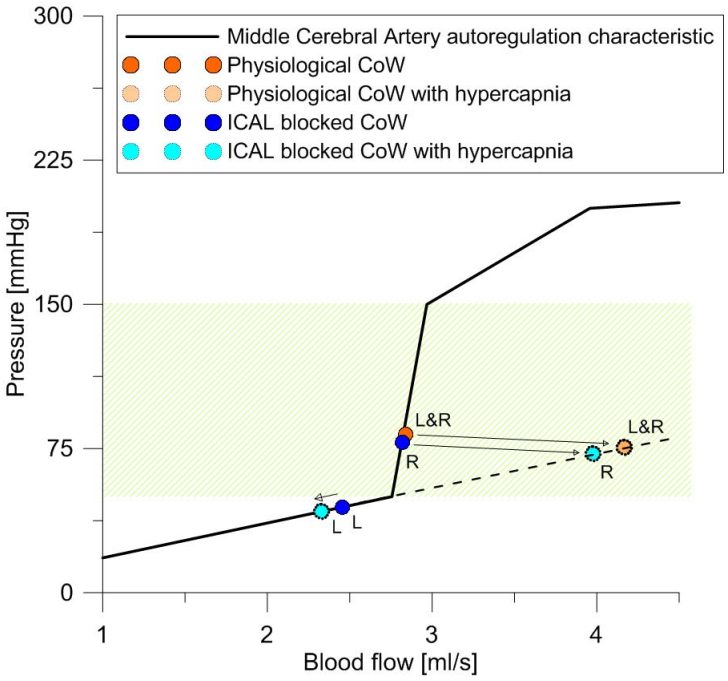


Fig.3. Reversed Robin Hood Syndrom mechanism presented on the autoregulation curve of middle cerebral artery. The green area denotes autoregulation range.

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 [17] report on six patients with ICA/MCA oclusions, the steal factor can vary beetween patients, from none existing up to *43.2* %. It depends on a specific CoW anatomy and vasomotor reactivity. In our computational model we can also observe stealing from the anterior part of the brain.

## 5. Conclusions

A numerical model of the CoW with implemented autoregulation mechanism was proposed. Four cases of cerebral blood flow were simulated and contrasted. It was shown that the 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.

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 the autoregulation mechanism is essential to capture clinically observed CBF phenomena.

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