

A comprehensive Cerebrovascular Simulation Model for Teaching and Research

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The mathematical relationships between cerebral perfusion pressure (CPP), intracranial pressure (ICP) and cerebral blood flow (CBF) as well as the autoregulatory control of these quantities by the normal brain, have been well described [1]. However, the alterations occurring in these quantities in pathological conditions and their complex non-linear relations are difficult to be understood in simple qualitative terms; for this reason, their analysis can benefit from the use of mathematical models.

In past years, we developed a comprehensive mathematical model of the cerebral circulation, which included many different aspects of patho-physiological relevance: cerebral autoregulation, cerebrospinal fluid (CSF) circulation, CO₂ reactivity, intracranial compliance, ICP dynamics and venous collapsibility. With that model, we were able to simulate several physiological phenomena and clinical results on head injured patients [2].

Results show that the model is able to simulate autoregulation to arterial pressure changes and CO₂ reactivity very well, and to predict intracranial instability (plateau waves) in patients with reduced intracranial elasticity and reduced CSF outflow. Moreover, the model is able to reproduce the measured time pattern of middle cerebral artery velocity and ICP in different head injured patients through a best fitting procedure, and using values of parameters which lie within the range reported in the patho-physiological literature.

The previous model, however, included just a single blood pathway inside the brain, i.e., it assumed no difference in cerebral hemodynamic among the different brain regions. For this reason, that model was not able to simulate conditions characterized by regional differences in brain circulation, such as those occurring during occlusion or stenosis of an intracranial vessel, during ischemia in a brain territory, or as a consequence of regional differences in metabolism. In these conditions, the situation is made particularly complex by two phenomena: CBF regulation mechanisms work in a different way on the individual districts, depending on the local value of blood flow; the different districts communicate upstream via the Circle of Willis, and, although to a lesser extent, via downstream anatomical collaterals.

So we developed a mathematical model of cerebral hemodynamics, with separate regulation of multiple brain regions. The main sectors included in the model are: the Circle of Willis, the hemodynamic circulation in the six districts perfused by the cerebral arteries, venous return with collapsible veins, CSF circulation, and intracranial elasticity. Furthermore, each cerebral district is independently regulated following changes in CPP and CO₂ tension. This model can be used to analyze cerebral hemodynamics during unilateral stenosis or occlusion of an internal carotid artery (ICA), as well as to assess the compensatory role of the Circle of Willis and of cerebrovascular regulatory mechanisms. Results show that, in normal subjects, the action of local blood flow regulation mechanisms and compensation by the Circle of Willis ensure adequate ipsilateral blood flow even in the presence of total unilateral ICA occlusion. However, a steal phenomenon is observed (i.e., a decrease of blood flow in the ipsilateral region) following hypercapnia. Sensitivity analysis on the calibre of the communicating arteries reveals that the anterior communicating artery plays a pivotal role in ensuring blood flow in the ipsilateral side during ICA stenosis.

The model may have important clinical and educational applications, allowing assessment of blood perfusion to different brain regions in various patho-physiological cases.

References

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