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COMMENTARY

Cerebral blood flow autoregulation during intracranial hypertension: a simple, purely hydraulic mechanism?

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Since Fog demonstrated that the diameter of the pial arteries changed in response to blood pressure changes, numerous studies, both in experimental animals and in man, have shown the role of the cerebral arterial tree in the cerebral blood flow (CBF) autoregulatory process, although the relative contributions of the various segments of the tree remain a matter of debate [1]. Several physiological theories (tissue pressure, neurogenic, myogenic and metabolic theories) have been proposed for the explanation of the underlying mechanism of this process. Other studies have shown that the same mechanism is operative, when the cerebral perfusion pressure (CPP) is reduced, as in this study, by increasing the intracranial pressure (ICP) [2, 3]. If CBF autoregulation is disturbed, the response to both blood pressure and ICP changes is affected [4].

The authors hypothesise that CBF is not regulated by the upstream arterial vascular compartment but solely by the downstream venous compartment and that CBF autoregulation is not a physiological but a purely physical, hydraulic phenomenon. Although not new, this is a daring concept in conflict with the above classical concept. The question is whether this hypothesis is validated by the experimental design and results of this study.

In this rabbit model, CBF remained constant up to an ICP close to the blood pressure level and thereafter (experimental point B) fell abruptly. In other animal preparations and in man, autoregulation fails at much higher CPP values. The amplitude of the ICP pulsations increased during the first phase, which can be explained by the exponential shape of the craniospinal volume–pressure curve (reduced compliance), and decreased when CBF started to fall. The latter finding is in contrast with other studies showing an increase in ICP pulsations when autoregulation is beginning to fail [5, 6]. I do not understand why from these results the inference of a hydraulic autoregulatory mechanism at the venous side of the vascular bed is made. The authors argue that the appearance of a reverberating wave at the arterial inflow in the situation of circulatory arrest (point D) excludes the possibility of the cerebrovascular resistance being located proximally, in the arterial segment, as this phenomenon requires that a large amount of non-circulating blood oscillates in a maximally expanded vascular compartment. However, this ‘requirement’ does not contradict the existence of arterial resistance vessels, as after the exhaustion of CBF autoregulation the resistance vessels cannot further dilate and are maximally dilated when circulatory arrest occurs, a state called vasoparalysis by Langfitt et al. [7].

I agree with the authors that the Starling resistor mechanism is responsible for the maintenance of venous outflow when the veins, during rising ICP, are progressively compressed at the ‘cuff-constriction’ site near the dural sinuses [8]. In this way, the resistor is part of the CBF autoregulatory process. The increase in venous outflow resistance is compensated by an increase in the upstream venous vascular pressure which further increases as a result of an active dilatation of the arterial resistance vessels. In my opinion, therefore, the cerebrovascular resistance is made up of two resistances in series: one at the arterial inflow section and one at the venous outflow section. During progressive elevation of the ICP, the flow resistance is shifting from the arterial to the venous side as demonstrated by Shulman and Verdier [9] and Lowell and

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Bloor [10]. If CBF autoregulation would be exclusively dependent on a purely hydraulic mechanism, it would be difficult to understand why in many clinical pathological conditions (head injury, subarachnoid haemorrhage, tumour) CBF autoregulation can be impaired at relatively low levels of ICP when venous compression plays a minor role.

A prerequisite for the Starling resistor model is a closed system as postulated by the Monro–Kellie doctrine. However, numerous studies on the craniospinal volume–pressure relationships have shown that this doctrine does not completely hold true, as the cranial compartment is in connection with the spinal compartment and open to the atmosphere through the vascular compartment. In this system, there are no pressure changes without volume changes, however small the latter may be. Therefore, the ICP pulsations 'require' an underlying volume change and I disagree with the authors that the cerebral blood volume during a cardiac cycle should remain constant [11].

The authors should be recommended for drawing attention to an often neglected side of the cerebral vascular bed: the venous outflow section.

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