Critical closing pressure

Denotes the lower limit of arterial blood pressure (ABP), below which small cerebral arterial vessels collapse and blood flow ceases.

Measurements in animals and in humans, have shown that the CrCP is significantly greater than zero. A simple mathematical model, incorporating the effects of arterial elasticity and active wall tension, shows that CrCP can be influenced by several structural and physiological parameters, notably intracranial pressure (ICP) and active wall tension. Due to the non-linear shape of the complete ABP-CBF curve, most methods proposed for estimation of CrCP can only represent the linear range of the pressure-flow (or velocity) relationship. As a consequence, only estimates of apparent CrCP can be obtained, and these tend to be significantly higher than the true CrCP. Estimates of apparent CrCP have been shown to be influenced by arterial PCO2, ICP, cerebral autoregulation, intra-thoracic pressure, and mean ABP. There is a lack of investigation, under well-controlled conditions, to assess whether CrCP is altered in disease states. Studies of the cerebral circulation need to take CrCP into account, to obtain more accurate estimates of cerebrovascular resistance changes, and to reflect the correct dynamic relationship between instantaneous ABP and CBF [1)].

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retrospectively analysed recordings of intracranial pressure (ICP), arterial blood pressure (ABP) and blood flow velocity from 27 severe TBI patients (mean 39.5 ± 3.4 years, 6 women) in whom a ventilation increase (20% increase in respiratory minute volume) was performed over 50 min as part of a standard clinical CO2 reactivity test. CrCP was calculated using the Windkessel model of cerebral arterial flow. Arteriolar wall tension (WT) was calculated as a difference between CrCP and ICP. The compartmental compliances arterial (C a) and cerebrospinal fluid space (C i) were also evaluated. RESULTS:

During hypocapnia, ICP decreased from 17±6.8 to 13.2±6.6 mmHg ($p < 0.000001$). Wall tension increased from 14.5 ± 9.9 to 21.7±9.1 mmHg ($p < 0.0002$). CrCP, being a sum of WT + ICP, changed significantly from 31.5 ± 11.9 mmHg to 34.9±11.1 mmHg ($p < 0.002$), and the closing margin (ABP-CrCP) remained constant at an average value of 60 mmHg. C a decreased significantly during hypocapnia by 30% ($p < 0.00001$) and C i increased by 26% ($p < 0.003$). CONCLUSION:

During hypocapnia in TBI patients, ICP decreases and WT increases. CrCP increases slightly as the rise in wall tension outweighs the decrease in ICP. The closing margin remained unchanged, suggesting that the risk of hypocapnia-induced ischemia might not be increased [2)].

1)
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2)
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