Cerebral perfusion pressure

- Characteristics and Changes of Cingulate Gyrus Function and Perfusion in Patients With Anti-N-Methyl-D-Aspartate Receptor Encephalitis
- Translational Insights Into Pericyte-Mediated Regulation of Cerebral Blood Flow: Implications for Ischemic Stroke
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Cerebral perfusion pressure, or CPP, is the net pressure gradient causing cerebral blood flow to the brain (brain perfusion). It must be maintained within narrow limits because too little pressure could cause brain tissue to become ischemic (having inadequate blood flow), and too much could raise intracranial pressure (ICP).

An alternative definition of CPP is:

The difference between the mean arterial blood pressure (MAP) and the ICP (CPP = MAP-ICP), and therefore it is a calculated variable.

where:

MAP is mean arterial pressure

ICP is intracranial pressure.

Increased cerebral blood volume: may result from loss of cerebral vascular autoregulation. This hyperemia may sometimes occur with extreme rapidity, in which case it has sometimes been referred to as diffuse or “malignant cerebral edema,” which carries close to 100% mortality and may be more common in children. Management consists of aggressive measures to maintain ICP < 22 mm Hg and CPP > 60-70 mm Hg [1]. (whether 60 or 70 is the optimal minimum for CPP is unclear).

Insufficient cerebral perfusion pressure (CPP) after aneurysmal subarachnoid hemorrhage can impair cerebral blood flow.

Guidelines recommend cerebral perfusion pressure (CPP) values of 50-70 mmHg and intracranial pressure lower than 20 mmHg for the management of acute traumatic brain injury (TBI). However, adequate individual targets are still poorly addressed, since patients have different perfusion thresholds [2].

A CPP > 60 mm Hg emerges as the crucial factor guaranteeing sufficient brain oxygenation. Any intervention used to further elevate CPP does not improve cerebral oxygenation, to the contrary, forced hyperventilation even bears the risk of inducing brain ischemia, but no class I evidence can currently advise the ideal CPP for any form of traumatic brain injury. 'Optimal' CPP is likely patient-, time-, and pathology-specific. Further, CPP estimation requires correct referencing (at the level of the foramen of Monro as opposed to the level of the heart) for MAP measurement to avoid CPP over-estimation and adverse patient outcomes [3] [4].

Aggressive CPP therapy should be performed and maintained even though apparently lethal ICP levels may be present. Further study is needed to support these encouraging results [5].

Cerebral perfusion pressure and cerebral autoregulation

Secondary brain injury (i.e., following the initial trauma) is attributable in part to cerebral ischemia. The critical parameter for brain function and survival is not actually ICP, rather is adequate cerebral blood flow (CBF) to meet CMRO2 demands.

CBF is difficult to quantitate, and can only be measured continuously at the bedside with specialized equipment and difficulty. However, CBF depends on cerebral perfusion pressure (CPP), which is related to ICP (which is more easily measured)

The actual pressure of interest is the mean carotid pressure (MCP) which may be approximated as the MAP with the transducer zeroed ≈ at the level of the foramen of Monro.

As ICP becomes elevated, CPP is reduced at any given MAP. Normal adult CPP is > 50 mm Hg. Cerebral autoregulation is a mechanism whereby over a wide range, large changes in systemic BP produce only small changes in CBF. Due to autoregulation, CPP would have to drop below 40 in a normal brain before CBF would be impaired.

In the head-injured patient, older recommendations were to maintain CPP ≥ 70 mm Hg (due to increased cerebral vascular resistance) & ICP < 20 mm Hg.3 However, recent evidence suggests that elevated ICP (≥ 22 mm Hg) may be more detrimental than changes in CPP (as long as CPP is > 60-70 mm Hg) (higher levels of CPP were not protective against significant ICP elevations).

Management protocol

see Rosner Concept

1)
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2)

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3)

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5)

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