Calculation of cerebral perfusion pressure in the management of traumatic brain injury: joint position statement by the councils of the Neuroanaesthesia and Critical Care Society of Great Britain and Ireland (NACCS) and the Society of British Neurological Surgeons (SBNS)

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The calculation of CPP is an integral part of this strategy, as described by Rosner and colleagues4 in their seminal paper. In their article, the mean arterial pressure (MAP) used was measured in supine patients at the level of the middle cranial fossa to estimate transcranial perfusion, described by the following equation:

\[
\text{CPP} = \frac{\text{MAP}}{C_0} - \text{ICP}
\]

In 2013, Subhas, Wilson and Jain conducted a national survey of CPP measurement practices in Great Britain and Ireland. Their results were presented at the NACCS meeting in Cardiff and the abstract published.5 They revealed that, in calculating CPP, 58% of neurosurgical intensive care units place the arterial transducer at the level of the heart and 42% place it at the level of the tragus. No-one routinely nursed their patients in the supine position, and 84% nursed patients 30 degrees head-up. They also demonstrated that 94% of respondents wished NACCS to endorse a consensus statement on standardization of CPP measurement practices in Great Britain and Ireland.
This has been considered by the Councils of NACCS and SBNS, who wish to make the following joint statements.

**Research involving cerebral perfusion pressure calculation or cerebral perfusion pressure-derived variables**

Councils of NACCS and SBNS recommend that all research articles relating to CPP measurement or CPP-derived variables in the management of TBI should explicitly state in their methodology where the arterial transducer was positioned (levelled) for relevant measurements.

Councils endorse positioning (levelling) the arterial transducer at the level of the middle cranial fossa, which can be approximated to the tragus of the ear.

**Clinical practice involving cerebral perfusion pressure-based targets and management based on recommendations by the Brain Trauma Foundation**

Whilst not wishing to dictate local clinical practice, based on the available evidence, the Councils of NACCS and SBNS would recommend that when calculating CPP in TBI the MAP used in the equation CPP=MAP–ICP should be the mean cerebral arterial pressure estimated to exist at the level of the middle cranial fossa, which can be approximated by positioning (levelling) the arterial transducer at the tragus of the ear.

They also recommend that the arterial transducer is repositioned to remain levelled with the tragus following changes in body elevation or position.

Councils do not endorse positioning (levelling) the arterial transducer at heart level (phlebostatic axis) for CPP-based treatment decisions because there is a requirement for subsequent cerebral MAP to be calculated, which is dependent on the relationship:

\[
\text{MAP}_{\text{brain}} = \frac{\text{MAP}_{\text{heart}}}{C} - (\text{water column between heart and brain} \times C)
\]

where \(C\) is a coefficient, always lower than 1, dependent on conditions of both the arterial and the venous elements of the cerebral circulation, which is not reliably predictable and is variable between individuals.

Centres that wish to continue to position (level) their arterial transducers at the level of the heart for CPP-based TBI management should have explicit guidance within their TBI protocols on how they take account of this difference and its subsequent effect on individual CPP calculation for patient management.

**Declaration of interest**

None declared.

**References**


**Editorials**


**Cerebral perfusion pressure**

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Monitoring and managing cerebral perfusion pressure (CPP) is a key component of the management of traumatic brain injury (TBI). It is easily measured, can be monitored continuously, and maintenance of CPP sufficient to sustain adequate cerebral blood flow (CBF) forms part of the management guidelines of the Brain Trauma Foundation (BTF).1

Although CPP has been the subject of significant research as a factor influencing outcome after TBI, there is little evidence from randomized controlled trials to support a specific CPP target.2

Traditional approaches have targeted higher CPP values after evidence that CPP >70 mm Hg is associated with improved outcome.3 The argument for this approach is based on the principle that autoregulation can be preserved but shifted rightwards after TBI, and therefore a higher CPP is required to maintain adequate CBF. Increasing CPP also reduces ICP by reversing or avoiding the vasodilator cascade, that accompanies a CPP at the lower limit of autoregulation.3 Despite these theoretical advantages, many studies have demonstrated that higher CPP is