BJA

Editorial

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 not necessarily associated with a more favourable outcome,[2 4] and that the interventions to increase MAP and CPP, such as administration of large fluid volumes and inotropes/vasopressors, are not without risk.[5 6] Current consensus guidelines from the BFT recommend that CPP should be maintained between 50 and 70 mm Hg, with evidence of adverse outcomes if it is lower or higher.[1] It is increasingly accepted that CPP values after TBI are best adjusted individually rather than managed to a generic single threshold,[7] with target values identified by multimodal brain monitoring including measurement of autoregulatory status, brain tissue oxygen tension and cerebral metabolism.[8] Indices of cerebral autoregulatory reserve, including cerebrovascular pressure reactivity, can be used to identify 'optimal' CPP, when autoregulatory capacity is maximal.[9]

Whichever approach to CPP management is favoured, accurate measurement of CPP is a prerequisite. It goes without saying that physiological monitoring in the critically ill must be carried out in an accurate and consistent manner, but the measurement of MAP, in the context of the calculation of CPP, has received little attention. Although international guidelines recommend target values for CPP, the measurement of blood pressure, which directly influences calculated CPP values, is not described.[1 10]

The driving pressure for blood flow in most organs is the difference between arterial and venous pressures. CPP is the pressure driving blood through the cerebrovascular bed, and therefore the difference between inflow (cerebral arterial) and outflow pressures. As the brain is contained within a rigid enclosure, and the cerebral venous system is compressible and when collapsed acts as a Starling resistor, its outflow pressure is whichever of intracranial or cerebral venous pressures is higher.[11] The outflow pressure in the cerebral venous bed (i.e. in cortical or bridging veins) is difficult to measure, but approximates to ICP. For these reasons, CPP is determined in clinical practice as the difference between MAP and mean ICP.[2]

In general intensive care, MAP is most commonly measured at the level of the right atrium (RA) using the mid-axillary line at the level of the 4th intercostal space, as the zero reference point for the arterial transducer. This provides the most valid determination of arterial blood pressure and is equivalent to the pressure measured by standard sphygmomanometer techniques.[12] However, the definition of CPP, first described by Niels Lassen in 1959, is based on 'arterial blood pressure measured at the level of the head,' (i.e. the level of the midbrain using the tragus of the ear as external landmark).[13] This is of critical importance as most TBI patients are managed with head elevation, and the level of the arterial blood pressure transducer will affect the measured MAP, and therefore CPP.[12] In the supine position with the head resting in a neutral position, the tragus has roughly the same elevation as the RA and, when calculating CPP in a supine patient, it is reasonable to assume that the MAP at the level of the heart and brain is identical. However, when the head is elevated above the heart hydrostatic effects mean that cerebral arterial blood pressure, will be reduced by a magnitude dependent on the angle of elevation and distance between RA and brain reference points. To calculate CPP accurately in such circumstances the

measurement points for both MAP and ICP should be the same (i. e. at the level of the brain).[7]

The implications of using the RA rather than brain for MAP calibration level during measurement of CPP are substantial. In a patient with 30 degrees head elevation and 30 cm distance between heart and the head, the difference in measured MAP and CPP levels will be up to 11 mm Hg depending on where the blood pressure transducer is calibrated.[14] Discrepancies between CPP measurements derived using different blood pressure measurement levels are exacerbated with varying angles of head elevation, and in tall patients. For example, in patients in whom the head of the bed is elevated to 50 degrees, measuring ABP at the level of the heart results in a calculated CPP that is up to 18 mm Hg higher compared with when blood pressure is measured at the tragus of the ear.[15 16] As a result, a CPP reading of 60 mm Hg obtained with ABP measured at the level of the heart may actually represent a 'true' CPP of <45 mm Hg. This is lower than the minimum recommended by the BTF, and could potentially result in significant risk of hypoperfusion and cerebral ischaemia despite a displayed value of CPP that is 'normal'.[7]

Since the earliest days of neuroanaesthesia, blood pressure has been routinely measured at the level of the brain during procedures performed in the sitting position, and 're-zeroed' during changes in position.[17] It is then somewhat surprising that this practice has not translated into the neurointensive care unit, where clinical practice with regard to blood pressure measurement during calculation of CPP varies so widely.[14 18 19] Almost 20 years ago, Nates and colleagues[19] highlighted that, although TBI patients were routinely managed in the 30° head-up position, in more than 95% of Australian and New Zealand intensive care units surveyed, the arterial pressure transducer was calibrated at the level of the tragus in only 10%. A European clinical practice survey found that 62% of responding centres calibrated the blood pressure transducer at the level of the heart in TBI patients, and 36% at level of the head.[14] One unit had a different routine depending on measured ICP; initial calibration was performed at the level of the heart, but changed to calibration at head level if ICP rose above 20 mm Hg. A recent clinical practice survey of members of the Neurocritical Care Society (241 responses, 14.3% response rate) found that, among all respondents, 59% (142 of 241) measured CPP with reference to the RA and 41% (99 of 241) with reference to the tragus.[18] However, MAP was measured at the level of the RA in 74% and at the level of tragus in 16% of 31 of the 34 United Council for Neurologic Subspecialties accredited neurointensive care units in the USA.[18] Some respondents from the same institution gave conflicting responses, and the authors speculated that this raises concern as to whether physicians who make CPP-based decisions understand how CPP is being measured in their patients, and also appreciate the implications of doing this incorrectly.

Reflecting the variation in clinical practice, current guidelines for the management of CPP after TBI also rely on evidence from studies that have used different reference points for blood pressure measurement. A recent narrative review was unable to determine how MAP was measured in the calculation of CPP in 50% of 32 widely cited studies of CPP-guided management.[18] In the 16 studies in which the method of blood pressure measurement could be ascertained, MAP was referenced to the RA in 62% raising the possibility of underestimation of true CPP in these studies. Of note, ABP was measured at the level of the RA in two studies that describe worse outcomes when CPP is below 60 mm Hg.[20 21] As head elevation of 30–50° is common after TBI, unmeasured but possibly clinically significant differences in CPP (up to 18 mm Hg) related to the method of MAP measurement

may in part explain the failure of randomized controlled trials to demonstrate benefit from CPP-guided therapy.[22] There is therefore an urgent need to standardize CPP measurement practices.

The Neuroanaesthesia Society of Great Britain and Ireland (NASGBI) and Society of British Neurological Surgeons (SBNS) have recently issued a joint position statement regarding the calculation of CPP in the management of TBI. They recommend that the MAP used to calculate CPP should be the mean cerebral arterial pressure estimated to exist at the level of the middle cranial fossa, which can be approximated by 'positioning (zeroing) the arterial transducer at the level of the tragus of the ear'.[23] It is also recommended that the arterial transducer is re-positioned, to remain level with the tragus, after changes in head elevation. Positioning (zeroing) arterial transducers at the level of the heart during CPP based TBI management is discouraged, and centres wishing to continue this practice are urged to include explicit guidance in their management protocols about how this approach can affect measured CPP and the consequent risks of its underestimation.

The NASGBI and SBNS position statement is to be welcomed as the first attempt by professional bodies to standardize CPP measurement. UK neuroscience units should incorporate its recommendations without delay. It is also hoped that its publication will lead to the development and adoption of international standardization of CPP measurement methods, not just in clinical practice but also in clinical trials.

Declaration of interest

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