

Current neuromonitoring techniques in critical care

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Abstract

Early detection of secondary events is a major target of neuromonitoring in critically ill patients. Intracranial pressure (ICP) and cerebral perfusion pressure are the most widely accepted neuromonitoring parameters. Many studies have shown both to be related to mortality after traumatic brain injury. However, the benefit of ICP monitoring has not been established by a randomized controlled trial, and the efficacy of ICP-guided management has indeed been challenged. This review considers current neuromonitoring techniques in critical care medicine.

Key words: Monitoring, neurocritical care, secondary brain injury

INTRODUCTION

Primary damage incurred in traumatic brain injury (TBI) is defined by the accident and impact itself and cannot be influenced by medical treatment. Neurocritical care must focus on treating the secondary injury resulting from swelling, metabolic derangements and ischaemia, which endanger formerly intact brain tissue. The degree of secondary injury has a critical impact on outcome,^[1] and early detection of these secondary events is a major target of neuromonitoring. The most widely accepted neuromonitoring parameters, intracranial pressure, cerebral perfusion and seizure activity, have been shown in many studies to be related to mortality after traumatic brain injury (TBI).^[2-5]

This review considers current neuromonitoring techniques in critical care medicine.

Clinical assessment

An altered state of consciousness is often the first sign of a clinical deterioration caused by acute brain dysfunction. A standardized scoring system can help to compare current neurological status to prior assessments, thus allowing for detection of aggravation as early as possible. The Glasgow Coma Scale (GCS) is a widely used scoring system for this purpose. It consists of three components: Assessing eye-opening, verbal and motor responses, the latter being the most important regarding outcome after TBI. The GCS is easy to use and has a high interobserver reliability. The more recently developed Full Outline of UnResponsiveness score (FOUR Score) evaluates brain stem reflexes and respiration.^[6] Thus, more subtle changes in neurological status can be detected; likewise more information can be collected in deeply comatose patients. The FOUR score also allows the explicit testing of eye movements or blinking, facilitating detection of a locked-in state. As a verbal response is not assessed, the FOUR score avoids the problem of underrating intubated but alert patients, a drawback of the GCS.

Intracranial pressure monitoring

Intracranial pressure monitoring is the most widely used technical neuromonitoring device. According to the Monro-Kellie doctrine,^[7] the incompressible skull, the blood, cerebrospinal fluid (CSF) and brain tissue exist in a state of volume equilibrium, such that any

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increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another. The initial compensatory mechanism for intracranial volume expansion following TBI or ischaemic oedema, for example, would be a reduction in CSF. Once all compensatory mechanisms have been exhausted, ICP rapidly rises resulting in decreased cerebral perfusion pressure (CPP) as demonstrated by the formula $CPP = MAP - ICP$. MAP denotes mean arterial pressure. In the absence of autoregulation, CPP determines cerebral blood flow (CBF) and, therefore, oxygen supply.

ICP must be measured directly as it cannot otherwise be estimated by any clinical feature or computed tomography (CT). Normal ICP depends on age and body positioning, and ranges from 7 to 15 mmHg in healthy adults. Above 20–25 mmHg, aggressive treatment is started in most intensive care units (ICU). Lower thresholds have been suggested for children.^[8]

Currently, a catheter placed into one of the lateral ventricles and connected to an external pressure gauge is the most reliable and low-cost method for ICP monitoring [Figure 1]. This is the most frequently used method as it measures global ICP, provided there is no obstruction to CSF flow. It also allows therapeutic drainage of CSF to reduce ICP, and can be recalibrated *in situ*. However, ventricular catheters are associated with a higher infection risk compared to intraparenchymal catheters. These catheters can be difficult to place into brains with severe swelling.

Intraparenchymal catheters are easier to place, and infections are rare. This type uses a fibre-optic transducer or a piezo element on the tip of the catheter. Accuracy is usually good and although recalibration is not possible, baseline drift is low.^[9] However, it is possible that the pressure measured by the probe does not represent global ICP. The theoretical concept of an intracranial space as a single chamber with an equal distribution of pressure, however, is not always correct, as ICP in TBI patients for example may be unequally distributed throughout the cranial vault. Whether or not this is clinically important remains to be determined. Several studies have shown contradictory results in this matter. The risk of developing an ICP gradient may depend on the cause of damage. Focal lesions have been shown to cause an interhemispheric pressure shift, while in diffuse lesions no interhemispheric difference could be shown. Intraparenchymal probes should preferably be placed at the side of a mass lesion to avoid overestimation of CPP.^[10] An important disadvantage of intraparenchymal probes is the higher cost as well as the missing possibility of CSF drainage.

Other methods, such as epidural or subdural pressure probes are less accurate and, therefore, are rarely used.

Guidelines of the Brain Trauma Foundation recommend ICP monitoring in salvageable patients with GCS scores of 3–8 and an abnormal CT, defined as a scan showing haematomas, contusions, swelling, herniation or compressed basal cisterns. ICP monitoring is also recommended in patients with a GCS of 3–8 and a normal CT scan provided that at least two of the following criteria are fulfilled at admission: Age >40 years, unilateral or bilateral motor posturing or systolic blood pressure <90 mmHg (www.braintrauma.org). Some clinicians may choose to adapt these recommendations, e.g., they may monitor patients with a GCS score >8, if they will undergo major non-cranial surgery soon after admission.

Despite widespread use of ICP monitoring and clear guidelines, evidence on this matter is not straightforward. A meta-analysis published in 2010 found that ICP monitoring and aggressive treatment of intracranial hypertension in severe TBI is associated with improved outcomes.^[11] However, the benefit of ICP monitoring has never been proven in a randomised controlled trial. A study has even suggested that a CPP/ICP-oriented therapy will increase treatment intensity and respirator days without any improvement in outcome.^[12] However, it should be noted that the unmonitored group underwent intensive cross-sectional imaging and motor score assessment (GCS) instead of ICP monitoring.

The Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure (BEST: TRIP) trial published 2012^[13] raised further doubts about the uncritical application of ICP monitoring. This was a multi-centre, controlled trial in Bolivia and Ecuador in which 324 patients with severe TBI were randomly assigned to one of two specific protocols: Guideline-based management based on monitored ICP versus treatment based on imaging and clinical examination. The primary outcome was a composite of survival time, impaired consciousness, functional status at 3 and 6 months and neuropsychological status at 6 months. The study found that care concentrated on maintaining monitored ICP <20 mmHg was not superior to care based on imaging and clinical examination. However, the authors of the trial stated that they do not feel that given the ease and safety diminished ICP monitoring should follow this report, as “the study did not test the value of ICP monitoring *per se*” but “specifically compared two aggressive TBI treatment approaches, one of which was ICP-guided therapy.” Given the current evidence, it seems that the value of ICP monitoring in severe TBI should rather be seen as a part of a multi-modality approach to targeted therapy rather than the only basis for decision-making. In a critique of the BEST: TRIP trial published 2013 in the BMJ, author Peter Hutchinson stated that he also believe(s) that a “normal” ICP should

not be considered only in light of a particular cut-off value, because waveform analysis of the ICP is also important.^[14] Ongoing research has shown that ICP waveform analysis can provide information on the state of cerebrovascular reactivity (PRx index) and can be used to estimate optimal CPP levels for individual patients.^[14] It is important not to rely on one monitor alone, but rather to gain information from multimodal monitoring including haemodynamic measurements like CPP and ICP, as well as global measures of oxygenation and metabolism. These additional monitors including brain tissue oxygenation, jugular bulb saturation, near-infrared spectroscopy (NIRS), cerebral microdialysis and continuous electroencephalography (EEG) will be briefly summarised later in this text.

Cerebral perfusion pressure

CPP is considered an important treatment target for TBI patients in neurointensive care units. In order to calculate CPP ($CPP = MAP - ICP$), it is clinically important to set the reference point of the arterial pressure as well as for the ICP at the level of the foramina of Monro, or the external auditory meatus, as patients with brain injury are nursed in a 20–30° “head-up position.” MAP measured at the level of the heart as is frequently done, will overestimate CPP.

The Brain Trauma Foundation Guidelines for the Management of Traumatic Brain Injury recommend that CPP be maintained at 50–70 mmHg in the brain-injured patient. Evidence demonstrates that a CPP <50 mmHg in TBI patients is associated with poor outcome.^[15] Target CPP >70 mmHg increases the risk of acute lung injury through the administration of large fluid volumes and vasopressors, often leading to poor outcome.^[16] Therefore, the Lund concept for the treatment of TBI targets a CPP >50 mmHg and includes a volume-targeted therapy to minimise increases in intracapillary hydrostatic pressure and intracerebral water content to avoid secondary increases in ICP.^[17] Auto-regulation is impaired by too low or too high CPP. In healthy individuals, autoregulation is effective in the CPP range 50 to 150 mmHg. However, after traumatic head injury this range is typically reduced in a highly variable manner.^[18] It is very likely that rather than targeting an absolute threshold for CPP, therapy would be better guided by an individualised CPP target. Optimising CPP by monitoring cerebral vascular pressure reactivity may prevent both injurious hypotension and hypertension. Bedside monitoring of pressure autoregulatory capacity is possible by various methods, with Czosnyka’s pressure reactivity index (PRx) being the most frequently used.^[19] Pressure reactivity is a key component of cerebrovascular auto-regulation. The PRx can be derived through analysis of slow waves in mean arterial blood pressure MAP and ICP. In normal autoregulation,

MAP is inversely correlated with ICP. A negative PRx value indicates intact autoregulation, and a positive value signifies a non-reactive cerebral circulation [Figure 2]. PRx correlates well with transcranial Doppler ultrasonography data with abnormal values being predictive of poor outcome after TBI.^[20,21]

Cerebral blood flow

CBF can be accurately measured by cerebral CT, direct angiography, or positron emission tomography (PET), but these procedures are expensive, time consuming and put patients at risk because of the need for transport, the use of contrast agents and radioactivity.

Alternatively, CBF can be measured indirectly by transcranial Doppler, which measures the velocity of blood flow in the cerebral arteries using an ultrasound probe. The flow velocity of the blood causes a phase shift in the specific sound wave frequency emitted and recorded by the probe, wherein the wave frequency is either increased or decreased in correlation with the speed of the blood. The blood flow volume can be determined if the diameter of the vessel is known. Transcranial Doppler is primarily a technique for measuring relative blood flow changes, limiting its usefulness in neuromonitoring.

Furthermore, manipulating CBF is difficult, and there is no convincing evidence that a strategy attempting to regulate CBF is superior to an approach using ICP or CPP to guide therapy. There is no absolute threshold for CBF with regard to ischaemia, and therapeutic interventions that influence the metabolic demands of the brain (e.g., sedation or hypothermia) have an unpredictable effect on CBF-related values.

Jugular bulb saturation

Measuring SJO₂ by a catheter placed in the jugular bulb, which samples of blood almost exclusively drained from the intracranial circulation, is one way of gaining information about global brain metabolism and ischaemia. Jugular oximetry can provide information about cerebral oxygen extraction and the adequacy of global CBF. This, however, is only correct if coupling between flow and metabolism is intact (i.e. changes in the metabolic rate for oxygen [CMRO₂] are linked to changes in CBF).

Low CBF and ischaemia increase oxygen extraction, consequently decreasing SJO₂. Normal SJO₂ values range from 50 to 75%. Oxygen extraction, or the arterio-jugular oxygen content difference (AJDO₂), is calculated as the difference between the arterial and jugular oxygen content in paired blood samples. Normal AJDO₂ values range from 4 to 9 ml/100 ml. Inherent shortcomings of this method that have led to decreased use include: (1) Only two-thirds of the sampled blood is

drained from the ipsilateral site; and (2) there is a large inter-individual variability in cerebral venous drainage. Therefore, SJO₂ and AJDO₂ can only provide information about global metabolism. Smaller lesions might not be detected. A study in head-injured patients using PET to quantify the ischaemic brain volume found that on average 170 cc of brain tissue were ischaemic at an SJO₂ of 50%.^[22] Therefore, adjusting CPP/ICP management based on SJO₂ results is difficult. Too low and too high SJO₂ values are associated with poor outcome.^[22,23] However, the question whether treatment directed at restoring normal SJO₂ improves outcome remains unanswered.

Cerebral tissue oxygenation monitoring

Cerebral tissue oxygenation (PbtO₂) is measured by a microcatheter inserted into the white matter of the frontal lobe [Figure 3]. Oxygen diffuses through a permeable membrane surrounding the probe, entering an electrolyte solution within the probe, which creates an electrical current within the solution proportional to the O₂ tension of the blood/tissue being measured. Normal baseline PbtO₂ values range from 25 to 35 mmHg (3.3 to 4.7 kPa). Mortality increases with time at or below a PbtO₂ of 20 mmHg or with the occurrence of any PbtO₂ values ≤ 6 mmHg.^[24] PbtO₂ is strongly influenced by CPP and can also be used to define an individually acceptable lower limit of CPP.^[25] However, the focal nature of PbtO₂ must be emphasised, as only approximately 15 mm² of tissue around the tip is sampled. Global assumptions can only be made in normal tissue or in a diffuse injury and not in tissues at risk. Therefore, adequate positioning of the sensor becomes crucial when using PbtO₂ as an endpoint of optimised CPP.

Studies suggest beneficial effects of clinical algorithms that target normalisation of PbtO₂ levels. The value of adding PbtO₂ measurement to the standard ICP/ CPP-guided therapy has been assessed in two prospective observational studies. Meixenberger *et al.*, compared a group where the ICP/ CPP algorithm was combined with a PbtO₂ target >1.33 kPa (10 mmHg) to a historical control group with an ICP/ CPP target algorithm.^[26] Although there was no significant difference in the 6-month outcome, there was a positive trend in the PbtO₂-guided group. Stiefel *et al.*, also compared an ICP/ CPP target group with a PbtO₂-guided group targeting a PbtO₂ of >3.3 kPa (25 mmHg). the PbtO₂-directed protocol produced better 6-month clinical outcomes than standard ICP/ CPP-directed therapy. A review on PbtO₂-based therapy combined with ICP/ CPP-based therapy published 2012 suggests that this combination is associated with better outcome after severe TBI than ICP/ CPP-based therapy alone.^[27,28] PbtO₂ measurements may contribute to the prevention of secondary injury after TBI and may allow treatment adaptation tailored to the individual needs. Brain Trauma Foundation guidelines

recommend monitoring PbtO₂ as a complement to ICP/ CPP-guided care in patients with severe TBI. Although no specific treatment protocol exists, the lower threshold for critical ischaemia has been defined as <2 kPa (15 mmHg). MAP and CPP are important determinants of PbtO₂. However, we have to keep in mind that other factors, such as pCO₂, pO₂ and haemoglobin significantly influence PbtO₂ as well.

Microdialysis

Cerebral microdialysis can be used as a bedside monitoring to detect cerebral hypoxaemia on a cellular level by measuring the energy substrate glucose and metabolites like lactate and pyruvate. A dialysis catheter (Ø 0.9 mm) is introduced into the brain parenchyma [Figure 4], and by calculating the lactate-pyruvate ratio, information on the brain's redox state, a marker of mitochondrial function, can be obtained. An increased lactate/pyruvate ratio and a decreased brain glucose level is associated with poor outcome after TBI and SAH.^[29] Whether or not microdialysis can be used to guide CCP managements remains to be determined. Although microdialysis can reveal important biochemical changes in injured brain tissue, some studies have found no relationship between elevated lactate/pyruvate levels and CPP changes.^[30]

Near infrared spectroscopy

NIRS is a non-invasive monitoring technique that measures the relative concentrations of oxygenated to deoxygenated haemoglobin through the transmission and absorption of near-infrared light as it passes through tissue. The normal range of regional oxygen saturation (rScO₂) is generally stated to be 60 to 75%. However, due to substantial inter- and intra-individual baseline variability it should rather be used to monitor trends.^[31] No absolute threshold exists for cerebral hypoxia and confounders, such as extracerebral or subdural haematoma can change the ratio of cerebral to extracerebral haemoglobin and, thus, offset tissue oxygen saturation values by a variable amount.^[32] Some clinical studies have shown the ability of cerebral oximetry monitoring to uncover otherwise clinically silent episodes of cerebral ischaemia in a variety of clinical settings despite the above-mentioned limitations.^[33]

Conservative and quantitative electroencephalography

EEG provides a continuous real time, non-invasive measure of brain function. When CBF is reduced, changes occur both in metabolic and electrical activity. The classical indications of EEG are for detecting seizures and for the prognosis of coma. A recently published review summarises the utility of the current

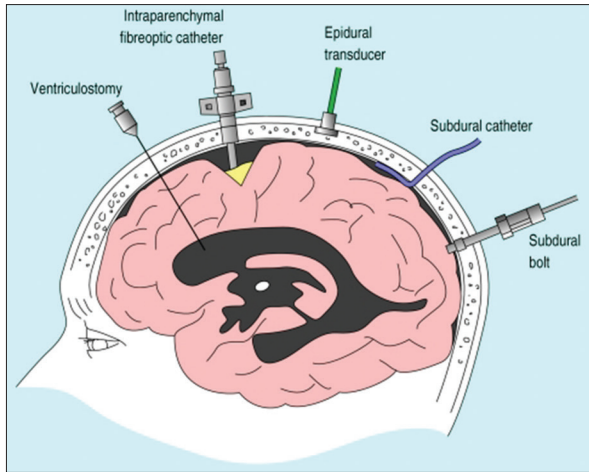


Figure 1: Intracranial pressure monitoring

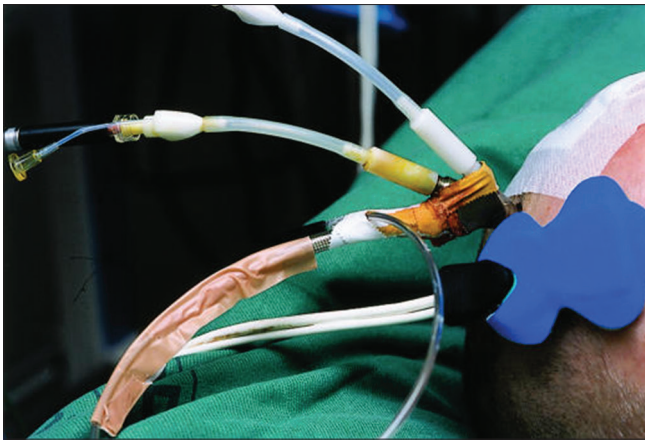


Figure 3: Brain tissue oxygenation monitoring

EEG recommendations to detect non-convulsive seizures in patients with severe brain injury in the ICU setting.^[34] However, EEG may also be useful for detection of deterioration in CBF in patients with TBI.^[35] When normal CBF declines, the EEG first loses the higher frequencies (alpha and beta bands), while the lower frequencies (delta and theta bands) gradually increase. When the CBF decreases further towards an infarction threshold, the EEG becomes isoelectric. This transition from ischaemia to infarction may provide a window of opportunity to treat. However, the EEG interpretation is difficult and highly subjective. Therefore, a variety of quantitative EEG have been developed based on the Fourier transformation principle. It is well beyond the scope of this review to explain the mathematical background, and the reader is kindly referred to specific literature on this matter. These quantitative EEG (qEEG) derived indices such as the alpha power or the alpha/delta ratio, have been used to detect delayed cerebral ischaemia in poor-grade SAH and severe stroke patients.^[36] qEEG is capable of detecting electrical

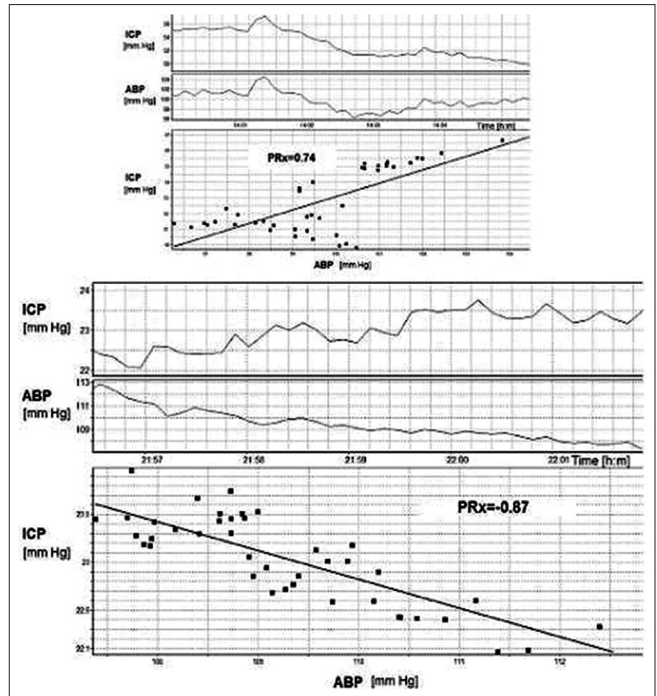


Figure 2: Intracranial and haemodynamic pressure monitoring

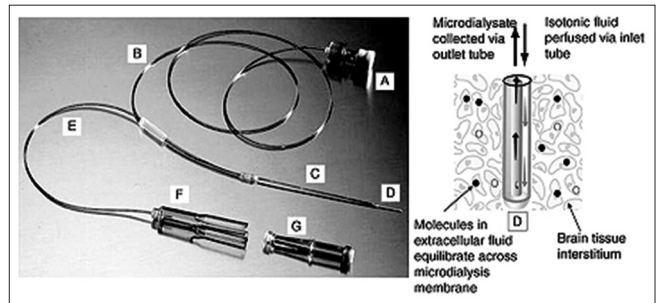


Figure 4: Brain microdialysis monitoring

changes correlating to blood flow and metabolism in as little as 28–100 seconds.^[37] It is important, however, to consider the effect of a large number of confounders like medication (sedatives), body movements or electrical artefacts from other monitoring intensive care unit devices. In addition, increasing use of continuous EEG reveals clinically undetected epileptiform activity in up to 70% of critically ill patients depending on the underlying neurological illness^[4,35,38] and results in higher detection rates than routine EEG because of the intermittent nature of occult seizures. Using EEG monitoring, 56% of seizures are detected during the first hour of patients in a general ICU, and 88% during the first 24 hours.^[39] Furthermore, a delay in diagnosis of non-convulsive status epilepticus and prolonged seizure duration has been independently associated with increased mortality.^[40] Continuous EEG seizure detection and treatment has been associated with improved outcome.^[40]

CONCLUSIONS

In severe brain injury, monitoring of ICP and CPP remains the cornerstone, particularly if a brain CT scan is abnormal despite the lack of first-class evidence due to difficulties in providing prospective randomised studies. It is important, however, not to rely on these two components alone. There is clinical evidence that demonstrates that so-called multimodal brain monitoring might help to optimise CBF and the delivery of oxygen/energy substrate at the bedside, thereby improving the management of secondary brain injury. Looking beyond ICP and CPP, and applying a multimodal therapeutic approach for the optimization of CBF, oxygen delivery and brain energy supply and early detection of clinically hidden epileptic seizures by EEG improve overall care of brain injured patients. Promising newer techniques for monitoring of oxygen and substrate delivery like PbtO₂ monitoring, NIRS or microdialysis, as well as quantitative EEG will further help to guide therapy in intensive care medicine in the future.

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