

# Neuromonitoring

Martin Smith

## Abstract

The monitoring of critically ill brain injured patients has become increasingly complex. Several techniques are now available for global and regional brain monitoring that provide assessment of cerebral perfusion, oxygenation and metabolic status, and early warning of impending brain hypoxia/ischaemia. Developments in multimodality monitoring have enabled a move away from rigid physiological target setting to an individually tailored, patient-specific approach to the management of acute brain injury. Multimodal monitoring generates large and complex datasets, and systems that analyse and present information in a user-friendly format at the bedside are essential to maximize its clinical relevance. This review describes current neuromonitoring techniques used during the intensive care management of acute brain injury.

**Keywords** Cerebral microdialysis; cerebral oxygenation; intracranial pressure; multimodal monitoring; near infrared spectroscopy; neuro-intensive care

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In addition to the continuous monitoring and assessment of cardiorespiratory functions common to all critically ill patients, several techniques are now available for global and regional brain monitoring. These provide assessment of cerebral perfusion, oxygenation and metabolic status and early warning of impending brain hypoxia/ischaemia, and guide targeted treatment after acute brain injury (ABI).<sup>1</sup> Some monitoring modalities are well established whereas others are relatively new to the clinical arena, and their indications are still being evaluated (Table 1).

## Intracranial pressure

There are two main methods of monitoring intracranial pressure (ICP).<sup>2</sup> The gold standard is a ventricular catheter connected to a pressure transducer 'zeroed' at the level of the external auditory meatus. Ventricular catheters measure global ICP and have the additional advantages of allowing periodic external calibration and therapeutic drainage of cerebrospinal fluid (CSF). However, they are associated with significant complications, including haemorrhage, seizures and CSF infection (ventriculitis). Alternatively, fibreoptic and microtransducer (strain gauge) ICP monitoring devices are easy to insert and have minimal complication rates. They are placed in the brain parenchyma via a cranial access device at the bedside or during neurosurgery and, although they cannot be recalibrated *in vivo*, their zero and sensitivity drift over time is relatively small. Microtransducer

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**Martin Smith MBBS FRCA FFICM** is a Consultant and Honorary Professor in Neurocritical Care at the National Hospital for Neurology and Neurosurgery, University College London Hospitals, UK. Conflicts of interest: MS is part funded by the National Institute for Health Research via the UCLH/UCL Comprehensive Biomedical Research Centre.

## Learning objectives

After reading this article you should be able to:

- identify the key intracranial variables that can be monitored at the bedside
- understand the advantages and limitations of different monitoring techniques
- understand the role of multimodality monitoring to guide individualised patient management.

devices measure localized pressure and this may not represent true CSF pressure because of the presence of intraparenchymal pressure gradients in the injured brain.

ICP monitoring is supported by international consensus guidance for many ABI types, and particularly for traumatic brain injury (TBI). A 2010 meta-analysis suggested that ICP monitoring and management is associated with improved outcome after severe TBI,<sup>3</sup> but a recent randomized controlled trial found no difference in 3- or 6-month outcomes when treatment after severe TBI was guided by ICP monitoring compared to care based on imaging and clinical examination in the absence of ICP monitoring. Whether the findings of this study, conducted in Bolivia and Ecuador, are applicable to wealthier nations with superior pre-hospital care and rehabilitation services remains to be seen. Furthermore, the composite primary endpoint in this study was weighted towards neuropsychological outcomes, and a more conventional measure, the extended Glasgow Outcome Scale, showed a (non-significant) 5% lower mortality and improved outcome in the ICP monitoring/management group.

ICP monitoring does not provide a comprehensive picture of cerebral physiology and pathophysiology, and is best viewed as a key component of a multimodal monitoring technique rather than as a monitoring modality in isolation.

## Cerebral oxygenation

Cerebral oxygenation monitoring assesses the balance between cerebral oxygen delivery and utilization, and the adequacy of cerebral perfusion. Several bedside methods of monitoring global and regional cerebral oxygenation are available.

## Jugular venous oxygen saturation

Measurement of jugular venous oxygen saturation (SjvO<sub>2</sub>) was the first bedside measure of cerebral oxygenation. As well as having considerable historical relevance, SvjO<sub>2</sub> monitoring formed the basis of our understanding of cerebral oxygenation changes after ABI. SjvO<sub>2</sub> is a flow-weighted measure that reflects global cerebral oxygenation only if the dominant jugular bulb is cannulated, although in practice the right side is usually chosen. The jugular catheter must be correctly sited to avoid contamination from the extracranial circulation, which is minimal when the catheter tip lies level above the lower border of the first cervical vertebra on a lateral cervical spine radiograph. Because it is a global measure, SjvO<sub>2</sub> monitoring is unable to detect regional ischaemia.

Normal SjvO<sub>2</sub> is 55–75% and interpretation of changes is relatively straightforward (Table 2). Prolonged or multiple

### Advantages and disadvantages of bedside neuromonitoring techniques

| Technique                                    | Advantages  | Disadvantages   |
|--|---|---|
| Intracranial pressure (ventricular catheter) | Gold standard<br>Measures global pressure<br>Therapeutic drainage of CSF<br><i>In vivo</i> calibration                    | Placement technically difficult<br>Risk of haemorrhage<br>Risk of infection   |
| Intracranial pressure (microsensor)          | Intraparenchymal/subdural placement<br>Easy to place with low procedural complication rate<br>Low infection risk          | <i>In vivo</i> calibration not possible<br>Measures localized pressure  |
| Transcranial Doppler                         | Non-invasive<br>Assesses regional blood flow velocity<br>Real time with good temporal resolution                          | Measures relative cerebral blood flow<br>Operator dependent<br>Failure rate of 5–10% (absent acoustic window)                                 |
| Jugular venous oximetry                      | Assesses balance between flow and metabolism<br>Easy to perform   | Global and insensitive to regional changes<br>Risk of vein thrombosis, haematoma, carotid puncture  |
| Brain tissue pO <sub>2</sub>                 | Bedside gold standard for brain oxygenation monitoring<br>Represents balance between flow and metabolism<br>Continuous    | Invasive<br>Measures regional oxygen tension<br>Utility dependent on probe location   |
| Near infrared spectroscopy                   | Non-invasive<br>Real time<br>Assessment of regional cerebral oxygenation over several regions of interest                 | Dependent on manufacturers algorithms<br>Lack of standardization between commercial oximeters<br>Signals affected by extracerebral structures |
| Microdialysis                                | Measurement of local brain tissue biochemistry<br>Early detection of hypoxic/ischaemic injury                             | Focal measure<br>Thresholds for abnormality uncertain   |
| Electroencephalography                       | Monitor of cellular bioenergetic distress<br>Non-invasive<br>Real time<br>Correlates with ischaemic and metabolic changes | Skilled interpretation required<br>Affected by anaesthetic/sedative agents  |

Table 1

### Interpretation of changes in jugular venous oxygen saturation

| SjvO <sub>2</sub> | Relative blood flow & metabolic changes | Causes  |
|-------------------|---|---|
| Normal (55–75%)   | CBF and CMRO <sub>2</sub> balanced      |   |
| Low (<50%)        | ↓ CBF or ↑ CMRO <sub>2</sub>            | ↓ Blood pressure<br>↓ PaCO <sub>2</sub><br>↓ PaO <sub>2</sub><br>↑ ICP or ↓ CPP<br>Seizures                               |
| High (>80%)       | ↑ CBF or ↓ CMRO <sub>2</sub>            | Cerebral hyperaemia<br>Failure of oxygen utilization (mitochondrial failure)<br>Arteriovenous shunting<br>Brainstem death |

CBF, cerebral blood flow; CMRO<sub>2</sub>, cerebral metabolic rate for oxygen; CPP, cerebral perfusion pressure; ICP, intracranial pressure; PaCO<sub>2</sub>, arterial carbon dioxide tension; PaO<sub>2</sub>, arterial oxygen tension; SjvO<sub>2</sub>, jugular venous oxygen saturation.

Table 2

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