

Monitoring intracranial pressure, perfusion and metabolism

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Key points

The intraventricular drain is considered to be the most accurate way of measuring intracranial pressure.

Intracranial pressure monitoring is not without risks.

Jugular bulb oximetry is a useful second-line monitor for refractory intracranial hypertension.

Transcranial Doppler can provide much useful information about cerebral perfusion.

Understanding the limitations of any monitoring device is crucial to its effective use.

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Cerebral monitoring is important for management of severe head injury. It is also used in subarachnoid haemorrhage, stroke, intracerebral haematoma, meningitis, encephalopathies, hepatic failure, after neurosurgery and in patients undergoing carotid artery surgery. This article provides an overview of cerebral monitoring techniques available in clinical practice.

Intracranial pressure measurement

The normal level of mean intracranial pressure (ICP) in a resting healthy adult in the horizontal position is 7–10 mm Hg. The normal ICP is pulsatile and reflects the cardiac and respiratory cycles, Fourier analyses give three different ‘slow’ waveforms (Table 1). An ICP >15 mm Hg is considered pathological, although this varies with the condition. Treatment would be instituted at a lower ICP in a patient with benign intracranial hypertension than one with an acute severe head injury. A number of studies have shown that prolonged elevation of ICP carries a poor prognosis¹ and treatment of elevated ICP decreases mortality. In head injured patients, levels >20 mm Hg are usually treated. An ICP monitoring device is often the earliest method to detect a surgically treatable cause of raised ICP, such as an expanding haematoma. The Brain Trauma Foundation publishes clinical guidelines regarding ICP monitoring.²

Derived values from ICP and its waveform give useful information:³

- (i) estimation of the *pressure–volume compensatory reserve* of the brain can be calculated by correlating the amplitude of the ICP pulse waveform with the mean ICP; and
- (ii) the *cerebrovascular pressure-reactivity index* is calculated by correlation of the ICP response to slow spontaneous changes in arterial blood pressure. This can be used to assess disturbances of cerebral autoregulation.

ICP is usually measured with devices placed in a ventricle, subdural space, subarachnoid space or directly into the brain parenchyma.⁴ The risks associated with these methods of ICP measurement include infection, haemorrhage, incorrect position, malfunction and obstruction. Clinically significant infection and haemorrhage is rare.

Intraventricular catheter

The gold standard for ICP measurement is the intraventricular catheter, which is traditionally inserted through a right frontal burr hole into the lateral ventricle. Placement can be difficult if the ventricle is either displaced or compressed. The intraventricular catheter can be used to remove cerebrospinal fluid (CSF) and administer drugs (e.g. antibiotics). It may be connected to either a saline manometer or a transducer. The reference point for the transducer is the foramen of Munro as this is close to the centre of the head. For ease the external auditory meatus is often used. If the head position is changed the transducer must be repositioned. Use of the intraventricular catheter is complicated by infection with quoted rates of 1–5%. Blockage may be overcome by flushing the system; repeated flushing should be avoided, as this will significantly increase the risk of infection. The use of antibiotic impregnated catheters may lower the infection rate.

Intraparenchymal monitors

The Camino transducer uses a fiberoptic cable to direct light to a miniature displaceable mirror at the catheter tip that is placed in brain tissue. ICP distorts the mirror and the reflected light intensity is transduced into pressure. This system is not dependant upon a saline-filled catheter. Codman have produced a microchip sensor placed at the end of a flexible nylon tube. ICP changes the resistance within the sensor and this is reflected as a voltage change. These sensors are inserted into the brain

Table 1 The waveforms seen on the ICP trace

	Associated with	Amplitude	Duration
A (plateau) waves	Cerebral vasodilatation Reduced cerebral compliance	50–200 mm Hg	5–20 min
B waves	Changes in respiratory pattern	<50 mm Hg	1 minute
C waves	Blood pressure and systemic vasomotor tone	<20 mm Hg	7–15 s

through a small burr hole via a 4 mm screw. They are considered to be almost as accurate as the ventricular drain, and have relatively low rates of infection and haemorrhage. They are particularly useful when the ventricles are inaccessible because of compression from raised ICP. The main disadvantages of this type of system are: (i) they cannot be recalibrated *in vivo*; (ii) they measure localized pressure, which may not be reflective of global ICP; (iii) therapeutic CSF drainage is not possible; and (iv) they may be subject to drift when used for long periods.

Subdural pressure transducers

Subdural pressure transducers are the least invasive and most easily placed of the ICP monitors described in this review. The dura is pierced and as the hollow device fills with CSF, the pressures equalize, and this closed fluid filled tubing transmits the pressure to a transducer. The rates of infection and haemorrhage are low, but the device is considered less accurate and there may be problems caused by occlusion with debris and misplacement. It is not possible to remove CSF with this device.

Jugular bulb oximetry

The jugular bulb is a dilatation of the internal jugular vein just below the base of the skull. It receives blood directly from the brain; measurement of oxygenation of this blood gives an estimation of cerebral oxygen consumption. The internal jugular vein is cannulated in a retrograde direction with a catheter containing a spectrophotometric fibreoptic probe and a lumen for aspiration of blood. Infrared light at three wavelengths measures haemoglobin concentration and oxygen saturations.⁵ The position of the catheter tip should be confirmed by a lateral x-ray, the ideal position is above the disc between the first and second cervical vertebrae and close to skull base. This approximates with the level of the mastoid air cells.

In the uninjured brain, reduced cerebral oxygen delivery (e.g. arterial desaturation) causes an increase in cerebral blood flow resulting in improved oxygen delivery (autoregulation). In patients with brain injury, autoregulation may be deranged and the cerebral vasculature may be unable to compensate for changing oxygen requirements. The normal jugular saturation (S_{jvO_2}) ranges from 55 to 71%, a figure that is lower than mixed venous saturations, reflecting the greater cerebral oxygen extraction compared with the rest of the body.

Table 2 Effects of altered S_{jvO_2}

Low S_{jvO_2}	1. Increased cerebral oxygen extraction Systemic hypoxia Reduced cerebral blood flow Increased ICP 2. Increased cerebral oxygen demand Seizures Pyrexia
High S_{jvO_2}	1. Abnormally high cerebral blood flow (loss of autoregulation) 2. High ICP causing shunting of blood past capillary beds

S_{jvO_2} is dependant upon arterial oxygen saturation, cerebral blood flow and cerebral metabolic rate. As long as the first two factors remain constant, the S_{jvO_2} varies with cerebral oxygen uptake. Significant increases and decreases of S_{jvO_2} are associated with poorer outcome (Table 2).

S_{jvO_2} monitoring is mainly used in the management of severe head injury. It confirms the deleterious effects a low cerebral perfusion pressure, and reflects the effects of interventional therapies. For example, hyperventilation is used to acutely reduce ICP, but can lead to critical cerebral vasoconstriction and ischaemia. S_{jvO_2} monitoring can be used to define how much hyperventilation can be safely used. S_{jvO_2} monitoring can also be used to optimize cerebral perfusion pressure: 70 mm Hg is considered optimal but it may be possible to lower this aim, minimizing the administration of vasopressors, if adequate cerebral oxygenation is confirmed.

Unfortunately the merits of S_{jvO_2} monitoring are limited by difficulties in obtaining accurate readings and in their interpretation. Frequent recalibration is required and protein build-up at catheter tip causes further error. If the catheter tip is not correctly positioned, accuracy will be affected by significant contamination with scalp and facial blood. Too rapid aspiration of a blood sample may also lead to inaccuracy as this may lead to contamination with extracerebral blood. The S_{jvO_2} reflects global rather than regional oxygenation, and may be affected by a number of variables other than cerebral oxygen uptake. Its relevance to focal injury should be questioned. As the monitor is invasive its insertion may lead to local complications. Because of these limitations the Brain Trauma Foundation only recommends S_{jvO_2} monitoring as a second-line device to help guide the treatment of raised ICP refractory to standard treatment.

Transcranial Doppler ultrasonography

Transcranial Doppler (TCD) is a non-invasive ultrasound-based technique used to measure blood velocity in the cerebral arterial system.⁶ Measurements are usually taken from the middle cerebral artery, although any major branch of the Circle of Willis or the basilar artery can be assessed if an appropriate 'window' can be found. A pulsed 2 MHz signal is transmitted through the temporal bone to a depth of 5–6.5 cm. Initially the beam is focused at a depth of 5 cm; the depth of focus is then varied to optimize the signal.

The signal is reflected by the solid components of blood (mostly red blood cells) and is distorted according to the Doppler shift principle; the change in wavelength is recorded in the same probe that delivers the signal. A waveform is displayed, which gives information on systolic, diastolic and the mean blood flow velocity (FV_{mean}).

The FV_{mean} is a weighted mean that takes into account the different velocities of formed elements in the blood vessel and has a normal value of $\sim 55 \text{ cm s}^{-1}$ in the middle cerebral artery. The shape of envelope from peak systolic flow to the end diastolic flow with each cardiac cycle is known as the waveform pulsatility. This pulsatility reflects the distal cerebral vascular resistance, providing there is not any stenosis or vasospasm and that blood rheology and pressure remain constant.

The main uses of TCD in anaesthesia and critical care are:

1. *To differentiate between vasospasm and hyperaemia in patients with subarachnoid haemorrhage and brain injury.* TCD is used for diagnosis of and differentiation between high velocity states such as cerebral vasospasm or hyperaemia (hyperperfusion syndrome). The Lindegaard ratio is the flow velocity of the middle cerebral artery divided by the velocity measured in the extracranial internal carotid artery. A high flow velocity ($>120 \text{ cm s}^{-1}$) in association with a Lindegaard ratio of <3 implies hyperaemia. A Lindegaard ratio >3 is likely to imply vasospasm.

2. *Determination of adequacy of collateral circulation during carotid surgery.* In order to perform a carotid endarterectomy, it is necessary to clamp the common, internal and external carotid arteries. At this point the adequacy of cerebral perfusion to the ipsilateral middle cerebral territory needs to be assessed. If there is an adequate collateral circulation through the Circle of Willis then no further action needs to be taken. However, if there is an inadequate collateral supply then a shunt needs to be inserted before a hypoperfusion stroke occurs. TCD is one of a number of modalities that can be used to assess the adequacy of the cerebral collateral circulation. A fall to one-third to half of baseline is generally felt to require shunt insertion. Having inserted the shunt it is possible to use TCD to confirm the restoration of flow, and that the shunt is serving its intended purpose.

3. *TCD can be also be used to monitor patients who have suffered strokes or transient ischaemic attacks arising from the carotid artery.*⁶ Immediately after such an event, microemboli can be detected in the middle cerebral artery, and the number of microemboli (the embolic load) is related to the risk of further embolic events. Microemboli are also seen after carotid endarterectomy, and again a high microembolic load is associated with an increased incidence of stroke. TCD can be used to detect these emboli, and can monitor the efficacy of antiplatelet agents which have been shown to both reduce the embolic load and reduce the incidence of stroke following carotid surgery.

4. *Estimation of perfusion pressure:* the pulsatility index (PI) gives an estimation of cerebral vascular resistance, $PI = (FV_{\text{systolic}} - FV_{\text{diastolic}}) / FV_{\text{mean}}$. The normal value for the

PI ranges from 0.6 to 1.1 and has been shown to correlate with cerebral perfusion pressure.

Near-infrared cerebral spectroscopy

Near-infrared cerebral spectroscopy (NIRS) is used as a non-invasive monitor of brain oxygenation.⁷ A forehead sensor shines infrared light through the surface layers of the brain and the light that re-emerges is sensed by a dual detector system. One detector is placed approximately 3 cm from the light source, and the other ~ 4 cm from the light source. The detector closer to the light source is assumed to detect reflected light that has passed through more scalp and subcutaneous tissues whereas the light detected by the distal detector would have a greater component of scalp, subcutaneous tissues and brain. The difference of the signals is assumed to represent a reading from the brain tissue ~ 2.5 cm below the surface. A computer algorithm based upon the Beer–Lambert law is used to display concentrations of oxygenated and deoxygenated haemoglobin. The monitor aims to give a real-time, non-invasive display of cerebral oxygen levels, and has been shown to correlate well with jugular bulb saturations in healthy volunteers under conditions of isocapnic hypoxia. Unfortunately the monitor is subject to significant error.⁸ Currently, cerebral NIRS remains an interesting research tool, with its place in clinical practice still being evaluated.

Invasive brain tissue oximetry

Two commercially available sensors are available which measure brain oxygen levels directly. One device (Licox) is a polarographic Clark electrode, the other (Neurotrend) is a multi-parameter sensor that measures temperature, PO_2 , PCO_2 and pH using a fiberoptic probe; tissue oxygen levels are measured by a phenomenon known as ‘fluorescent quenching’. These devices can measure local changes in regional oxygenation that would not be noticed using jugular oximetry, and some centres are now using these devices as part of a multimodal monitoring technique described below.

Microdialysis

Microdialysis is achieved via a fine coaxial catheter that is inserted into the brain. The catheter has a dialysis membrane on its outer surface and low flow rates of dialysis fluid are passed through the catheter using a pump mechanism. Vials of fluid are removed every 10–60 min allowing the measurement of concentrations of substances in the cerebral extracellular fluid. Fluid is taken to a remote machine for analysis although continuous on-line analysis is now possible. Although the concentration of any substance that will pass across the dialysis membrane can be measured, the following substances are currently of interest in the injured brain:

- (i) energy related metabolites (glucose, lactate, pyruvate, adenosine, xanthine);
- (ii) neurotransmitters (glutamate, aspartate, GABA);

- (iii) markers of tissue damage (glycerol, potassium, cytokines); and
- (iv) exogenous substances (drugs).

Elevation of the lactate/pyruvate ratio is associated with derangements in metabolism after severe head injury, and is considered to be a useful marker of tissue ischaemia. Currently, microdialysis is used as a research tool.

Multimodality monitoring

Continuous monitoring of more than one parameter can help overcome some of the limitations of each method described. An example would be using Sjv_{O_2} monitoring to help tailor therapy based upon ICP, another example would be using Sjv_{O_2} with TCD monitoring to help differentiate between the ischaemic and hyperaemic phases of head injury. The assumption is that the measurement errors from different monitors will occur at different times with the different monitors. Unfortunately multimodality monitoring incurs greater costs in equipment, manpower and time, and further increases the complexity of treatment.

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See multiple choice questions 99–101.