Acute Management of Traumatic Brain Injury

Management of traumatic brain injury focuses on stabilisation of the patient and **prevention of secondary neuronal injury** to avoid further loss of neurons. Full neuromonitoring including intracranial pressure measurement are rarely available prior to the patients arrival in the intensive care unit. Significant neurological damage can occur between the time of injury and CT scanning, accurate measurement of ICP and other parameters. The acute management of these patients is therefore directed towards assuming there is significant intracranial pathology and instituting measures to protect living brain tissue.

Assessment

Assessment of brain injury hinges on evaluation of the Glasgow Coma Score (GCS) and examination of the pupils. Traditionally a GCS of below 9 is considered to reflect severe brain injury. However with improvements in prehospital care and greater knowledge of brain injury, patients are arriving in emergency departments earlier and their brain injury may still be evolving. Therefore the following measures should be considered and porbably instituted in all patients with coma scores of 12 or below.

Hypotension will adversely affect cerebral perfusion and therefore lower the Glasgow Coma Score. However brain injury management should be instituted on the basis of the initial examination and there should be no delay to assess whether the GCS improves with volume resuscitation. It is important to identify signs of impending transtentorial herniation as this will affect the course of the immediate management of these patients. This is identified by unilateral abnormal posturing and/or the presence of a unilateral dilated pupil.

Serial assessment is vital. Patients may arrive with a mildly impaired GCS and rapidly deteriorate due to expanding haematomas or increasing cerebral swelling. Pupils may be initially normal and then dilate as intracranial pressure rises and the brain starts to herniate.

Management

The specific goals in the acute management of severe traumatic brain injury are:

1. Protect the airway & oxygenate
2. Ventilate to normocapnia
3. Correct hypovolaemia and hypotension
4. CT Scan when appropriate
5. Neurosurgery if indicated
6. Intensive Care for further monitoring and management

Hypoxia and hypotension are the greatest threat to functional outcome in brain injury. Early acute control of the above three parameters may have more impact than all other measures subsequently employed. Progressive neuronal loss occurs from the time of injury, not the time of arrival in hospital. Rapid sequence intubation should be used where available to secure the airway and maximally oxygenate the patient. Hypovolaemia and hypotension must be corrected early and take priority over other interventions for the brain injury. Other injuries causing haemorrhage must be addressed first (or simultaneously) so that an adequate cerebral perfusion pressure is maintained. Patients should be kept sedated to prevent coughing or valsalva maneuvers from fighting the ventilator, as these increase intracranial pressure.

Many of the interventions used in the management of intracranial pressure may have a detrimental effect on cardiopulmonary resuscitation (eg. mannitol) which in turn will have a detrimental effect on cerebral perfusion. In addition some measures may be counterproductive when used without adequate monitoring (eg. hyperventilation). Thus these further interventions are used without guidance from CT scans or ICP monitoring only when there is evidence of impending brain herniation (unilateral posturing and/or unilateral dilated pupil).

A CT scan of the brain should be obtained when appropriate, as dictated by the presence of other injuries and physiological disturbances. This will delineate the brain injury and determine whether surgery is indicated to remove an intracranial mass lesion (epidural / subdural haematoma), and the degree of diffuse injury and cerebral swelling present.

During this period there is potentially continuing cerebral ischaemia and neuronal death and timeliness is of the essence. There should be no unnecessary investigations or procedures and damage control techniques should be employed as necessary. No spinal or long bone imaging should be undertaken prior to CT scanning as these investigations will not affect the immediate patient management. The haemodynamically unstable patient should have minimum investigations, control of haemorrhage by the simplest means appropriate (and abbreviated surgery if necessary) and then CT scan and treatment of the brain injury.

If there are signs of impending transtentorial herniation (unilateral posturing and/or unilateral dilated pupil) or if there is rapid progressive neurological deterioration (without extracranial cause), then there is significant intracranial hypertension and measures should be instituted to control ICP immediately. Hyperventilation should be instituted to reduce the PaCO2 to no lower than 3.5kPa (25mmHg) and mannitol should be administered as a bolus. Oxygenation and cerebral perfusion must be maintained. CT scanning is emergent, as is surgery if indicated.

If there are other injuries leading to haemorrhage and hypotension these still take priority. However it may be necessary to consider treatment of the brain injury simultaneously with management of these injuries (laparotomy or thoracotomy), even without a CT scan to guide therapy. Blind burr holes to detect extra-axial collections may be appropriate as a last resort in these cases.

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Glasgow Coma Score

The GCS is scored between 3 and 15, 3 being the worst, and 15 the best. It is composed of three parameters : Best Eye Response, Best Verbal Response, Best Motor Response, as given below :

Best Eye Response. (4)

1. No eye opening.
2. Eye opening to pain.
3. Eye opening to verbal command.
4. Eyes open spontaneously.

Best Verbal Response. (5)

1. No verbal response
2. Incomprehensible sounds.
3. Inappropriate words.
4. Confused
5. Orientated

Best Motor Response. (6)

1. No motor response.
2. Extension to pain.
3. Flexion to pain.
4. Withdrawal from pain.
5. Localising pain.
6. Obeys Commands.

Note that the phrase 'GCS of 11' is essentially meaningless, and it is important to break the figure down into its components, such as E3V3M5 = GCS 11.

A Coma Score of 13 or higher correlates with a mild brain injury, 9 to 12 is a moderate injury and 8 or less a severe brain injury.

Teasdale G., Jennett B., LANCET (ii) 81-83, 1974.

Cerebral Perfusion Pressure

Cerebral Perfusion Pressure (CPP) is defined as the difference between the Mean Arterial Pressure (MAP) and the Intracranial Pressure (ICP).

CPP = MAP - ICP

This represents the pressure gradient driving cerebral blood flow (CBF) and hence oxygen and metabolite delivery. The normal brain autoregulates its blood flow to provide a constant flow regardless of blood pressure by altering the resistance of cerebral blood vessels.

These homeostatic mechanisms are often lost after head trauma (cerebral vascular resistance is usually increased), and the brain becomes susceptible to changes in blood pressure. Those areas of the brain that are ischaemic, or at risk of ischaemia are critically dependent on and adequate cerebral blood flow, and therefore cerebral perfusion pressure.

Key Recommendations

Maintenance of CPP reduces mortality in severe head injury.

CPP should be maintained above 70-80mmHg

Systemic hypotension is associated with poor prognosis

Maintenance of an adequate Cerebral Perfusion Pressure is a cornerstone of modern brain injury therapy.

After brain injury, and especially in the multiply injured patient, cerebral blood flow may be lowered to the ischaemic threshold. To prevent further neuronal death (the secondary brain injury), this flow of well oxygenated blood must be restored. There is no class I evidence for the optimum level of CPP, but 70-80mmHg is probably the critical threshold. Mortality increases approximately 20% for each 10mmHg loss of CPP. In those studies where CPP is maintained above 70mmHg, the reduction in mortality is as much as 35% for those with severe head injury.

Cerebral Perfusion Pressure may be maintained by raising the Mean Arterial Pressure or by lowering the Intracranial Pressure. In practice ICP is usually controlled to within normal limits (<20mmHg) and MAP is raised therapeutically. It is unknown whether ICP control is necessary providing CPP is maintained above the critical threshold.

Control of intracranial hypertension is discussed on the pages on intracranial pressure.

There is substantial evidence now that early hypotension (BP < 90mmHg) is associated with increased morbidity and mortality following severe brain injury. Even patients with one episode of hypotension during their ICU stay have a significantly reduced prognosis. Maintenance of an adequate MAP requires primarily a normovolaemic patient. Control of other sites of haemorrhage has the highest priority (with oxygenation). These patients should NOT be kept 'dry' with fluid restriction, but maintained in zero balance. Further elevation of MAP, once normovolaemia is achieved, is usually accomplished with norepinephrine, though dopamine may be used. There is little evidence to recommend any one agent over another.

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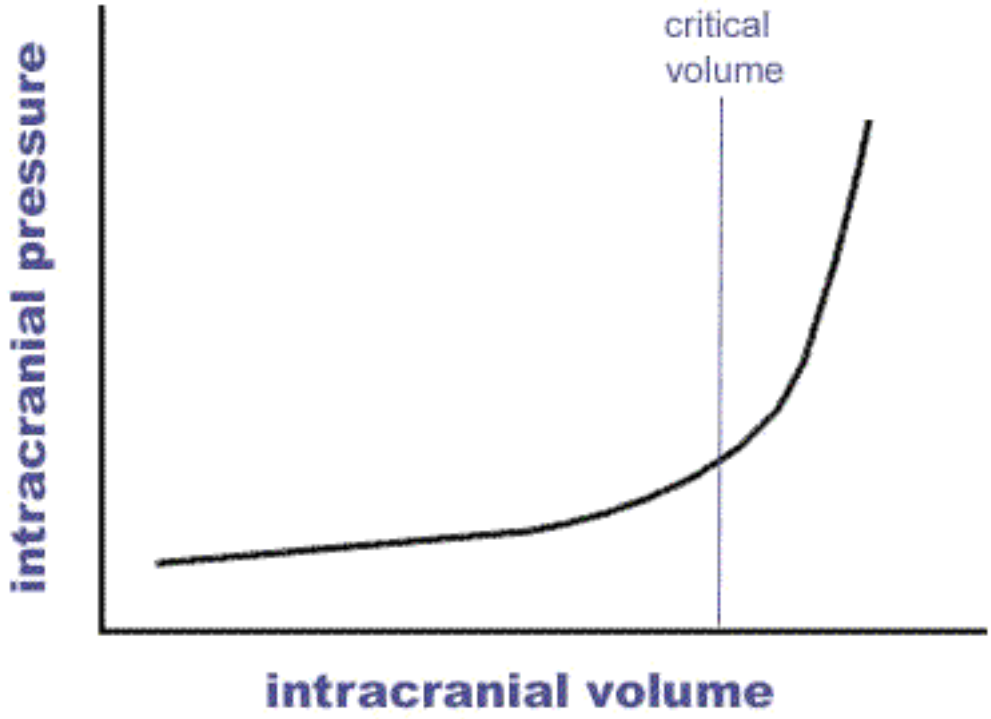
Intracranial Pressure

As the cranial vault is essentially a closed, fixed bony box, its volume is constant. This volume is described by the Monro-Kellie doctrine, proposed in the early part of the 19th century:

v.intracranial (constant) = v.brain + v.CSF + v.blood + v.mass lesion

As all these components are fluids, and non-compressible, once the cranial vault is filled, it's pressure rises dramatically. This intracranial pressure (ICP) rise can the lead to interruption of cerebral blood flow by reducing the cerebral perfusion pressure. As an intracranial mass lesion or oedematous brain expands, some compensation is possible as cerebrospinal fluid (CSF) and blood move into the spinal canal and extracranial vasculature respectively. Beyond this point, further compensation is impossible and ICP rises dramatically (figure 1).

Patients who are on the cusp of the graph have exaggerated ICP responses to events such as tracheal intubation, coughing or fighting a ventilator.



Key Recommendations
Maintenance of CPP reduces mortality in severe head injury.

ICP monitoring is recommended in most comatose patients with severe head injury.

ICP should be treated when above 20mmHg, but maintenance of CPP is probably more important.

The normal ICP is 0 - 10 mmHg. There is no defined set point at which treatment for intracranial hypertension should be initiated, but levels above 20mmHg are usually treated. However it is probably more important to maintain an adequate cerebral perfusion pressure. In a hypotensive patient, even a small increase in ICP could be harmful. Alternatively, an elevated mean arterial pressure may protect against a raised ICP.

ICP measurement is necessary to accurately determine CPP. However ICP measurement per se has not been conclusively shown to alter outcome in head injury patients. This is due to a combination of factors, primarily that ICP monitoring is now so accepted for severe head injury, and forms the basis for modern brain injury management, so that it would be difficult to conduct a study with a control arm. Secondly, as mentioned previously, previous studies have concentrated on controlling ICP, whereas modern brain injury protocols focus on maintenance of CPP. Thirdly, failure of a therapy should not necessarily implicate the measurement tool that therapy is directed against.

There is a substantial body of evidence to support the use of ICP monitoring. Several studies have reported substantial lowering in mortality after ICP monitoring and control was introduced. Similarly studies have shown a lower mortality in those patients whose ICP could be controlled compared to those in which it could not. ICP monitoring is now a central part of critical care management for the severely brain injured patient.

Indications for ICP monitoring.

All patients with severe head injury (GCS<9) and those patients with moderate head injury (GCS 9-12) at increased risk (see below) or who cannot be followed with serial neurological examination (eg. anaesthetised for other procedure).

Indications for ICP monitoring Risk of raised ICP

Severe Head Injury (GCS 3-8)

*** Abnormal CT scan**

50-60%

*** Normal CT Scan**

Age > 40 or BP < 90mmHg or abnormal motor posturing

50-60%

* **Normal CT scan**

No risk factors

13%

Moderate Head Injury (GCS 9-12)

* **If anaesthetised/sedated**
* **Abnormal CT scan**

approx. 10-20% will deteriorate to severe head injury

Mild Head Injury (GCS 13-15)

* **few indications for ICP measurement**

Only around 3% will deteriorate

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Control of Intracranial Hypertension

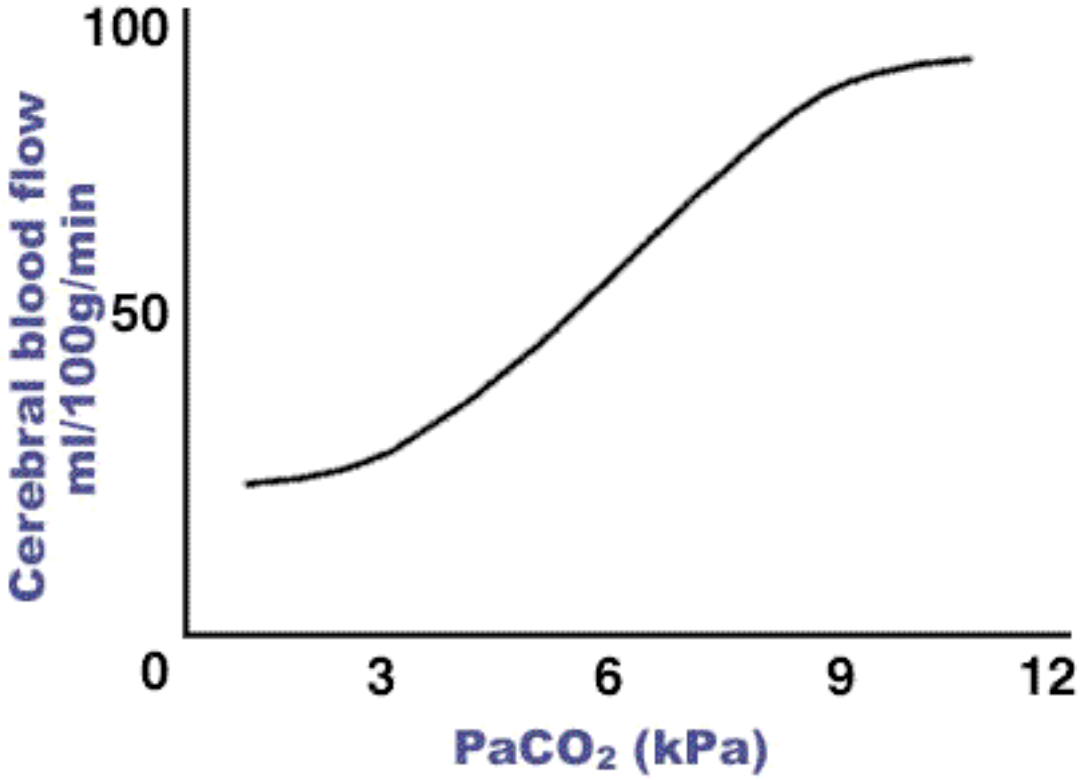
As part of intensive treatment of traumatic brain injury, intracranial pressure (ICP) should be controlled when the cerebral perfusion pressure (CPP) falls below 70mmHg and/or the ICP is greater than 20mmHg. Intracranial

hypertension occurs in approximately 40% of all patients with severe traumatic brain injury. Maintenance of an adequate cerebral perfusion pressure is more important than control of ICP per se. Measures to increase mean arterial pressure should be instituted prior to starting more complex methods of ICP control.

There are several methods for controlling ICP. These are usually applied in a stepwise fashion to achieve control, where possible. The absolute requirement for the potentially severely brain injured patient is tracheal intubation with a cuffed tube. This protects and maintains the airway and allows for maximal oxygenation and control of ventilation.

Ventilation

Carbon dioxide dilates the cerebral blood vessels, increasing the volume of blood in the intracranial vault and therefore increasing ICP. Patients should be ventilated to normocapnia (PaCO2 4.0 kPa / 30mmHg).



Key Recommendations

The baseline status for the severely brain injured patient is intubated, normovolaemic and normocapnic.

Hyperventilation should not be used routinely.

Mannitol should be reserved for acute control of ICP and administered in bolus form.

Previously, hyperventilation was used routinely to maximally reduce PaCO2. No studies have shown this to improve outcome in these patients. Additionally, transcranial doppler (TCD) assessment and positron emission tomography (PET) shows this can induce significant constriction of cerebral vessels and this increase in cerebral vascular resistance may reduce cerebral blood flow to below the ischaemic threshold. One study has shown an improvement in long-term outcome when hyperventilation is not used routinely.

Consequently hyperventilation should be used only for short periods when immediate control of ICP is necessary. For example in the patient who has an acute neurological deterioration prior to CT scanning and surgical intervention. Hyperventilation should not take the PaCO2 level to below 3.5-4 kPa as there is minimal beneficial effect on ICP below this level.

Occasionally hyperventilation may be necessary for longer periods in patients with persistently high ICPs who have not responded to other treatment modalities. These patients may benefit from more intensive neuromonitoring such as jugular venous oxygen saturation and transcranial doppler assessments to ensure cerebral perfusion is not being compromised at the expense of ICP. Persistent hyperventilation should not be used in the first 24 hours and preferably not within the first 5 days following brain injury.

Intravenous fluid therapy

Patients with severe brain injury should be kept normovolaemic. Previous regimens recommending that patients be kept 'dry' have essentially been discarded as there is significant risk of both hypotensive episodes (leading to a fall in cerebral perfusion) and systemic inflammatory respinse syndrome

(SIRS) or multiple organ failure (MOF) leading to failure of oxygenation and ventilation. Dehydration has little effect on cerebral oedema.

Free water (as dextrose solutions) should NOT be administered. This will decrease plasma osmolality and so increase the water content of brain tissue (the blood brain barrier acting as a semipermeable membrane). Elevated blood sugar levels are associated with a worsening of neurologic injury after episodes of global cerebral ischaemia. Ischaemic brain metabolises glucose to lactic acid, lowering tissue pH and potentially exacerbating ischaemic injury.

Hypertonic solutions and osmotic diuretics such as mannitol will have the opposite effect. This mechanism requires an intact blood brain barrier. If this is damaged, as may be the case following injury, low molecular weight, osmotically active particles may leak into the cerebral interstitium. In this case mannitol may have no effect in reducing brain water content, and maintenance of the colloid oncotic pressure in the vessels by administration of colloids, plasma proteins or other high molecular weight compounds may, theoretically, be of benefit. However in practice, colloids offer little benefit over crystalloid solutions.

There has been considerable interest in the use of hypertonic crystalloid solutions for the treatment of hypovolaemia in the presence of intracranial hypertension. Animal studies have proven the efficacy of hypertonic solutions in reversing shock, and sometimes in controlling ICP. Clinical trials suggest that survival after severe brain injury (GCS<9) may be improved with hypertonic solutions. However those injuries leading to a breakdown in the blood brain barrier show little or worsened response to hypertonic fluid administration.

There is no single best fluid for patients with traumatic brain injury, but isotonic crystalloids are widely used and have good scientific basis. Normal saline or lactated RInger's solution should be the standard resuscitation fluid until further studies show a clear benefit from other therapies. Regardless of the fluid type chosen, normovolemia must be maintained and episodes of hypotension avoided.

Mannitol

Mannitol, a 6-carbon sugar, is widely used in head injury management, though it has never been subjected to a randomised control trial against placebo and the methods and timing of administration vary widely. It is an osmotic diuretic and can have significant beneficial effects on ICP, cerebral blood flow and brain metabolism. Mannitol has two main mechanisms of action. Immediately after bolus administration it expands circulating volume, decreases blood

viscosity and therefore increases cerebral blood flow and cerebral oxygen delivery.

Its osmotic properties take effect in 15-30 minutes when it sets up an osmotic gradient and draws water out of neurons. However after prolonged administration (continuous infusion) mannitol molecules move across into the cerebral interstitial space and may exacerbate cerebral oedema and raise ICP. Mannitol itself directly contributes to this breakdown of the blood brain barrier.

Mannitol is therefore best used by bolus administration where an acute reduction in ICP is necessary. For example the patient with signs of impending herniation (unilateral dilated pupil / extensor posturing) or with an expanding mass lesion may benfit from mannitol to acutely reduce ICP during the time necessary for CT scanning and/or operation.

Mannitol is wholly excreted in the urine and causes a rise in serum urine and osmolality. Patients with poor renal perfusion (shock), sepsis, receiving nephrotoxic drugs or with a serum osmolality over 320mOsm are at risk of acute tubular necrosis. Hypolaemia should be avoided with the infusion of isotonic fluids as necessary.

Sedation and anaesthesia

All but the most severely brain injured patients (GCS 3) will require anaesthesia for intubation. The cardiovascular responses to intubation induce a rise in ICP which is exaggerated in those patients on the cusp of the pressure-volume curve. Rapid sequence intubation is probably the safest method of establishing an airway in these patients.

Continuing sedation will be necessary in most patients to allow adequate ventilation and to prevent coughing or fighting the ventilator. Ensuing valsalva-type maneuvers cause sharp rises in intracranial pressure. Which agents are used to achieve sedation is probably less important. However short acting preparations will allow finer control of the depth of anaesthesia and faster recovery from sedation. Agents with a longer duration of action such as diazepam may be best administered by intravenous bolus as required rather than by constant infusion to avoid build-up of active metabolites.

Sedation is not analgesia, and pain requirements must be addressed to provide a quiet, comfortable patient. Adequate analgesia will also reduce the requirements for sedation and neuromuscular blockade.

The use of neuromuscular blocking agents is not routinely required for continued ventilation. However some patients whose high sedative

requirements lead to adverse cardiovascular effects may benefit from pharmacologic paralysis.

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Neuromonitoring for Traumatic Brain Injury

The primary goal of management for traumatic brain injury is the prevention of secondary damage due to neuronal hypoxia and hypoperfusion. Monitoring modalities are aimed at identifying potential episodes of hypoxia and guiding therapy related to cerebral perfusion.

Patients with severe brain injury are intubated to protect the airway and allow maximal oxygenation. Standard monitoring for all such patients is required including oxygen saturation (SaO2), ECG, mean arterial blood pressure (MAP) and urine output. These patients will require frequent determination of arterial blood gases and an intra-arterial catheter is helpful. Patients are maintained euvolaemic and central venous pressure measurements are used to guide therapy.

Normocapnia is vital for maintenance of intracranial pressure (ICP), and patients should have continuous measurement of end-tidal CO2 (ETCO2) levels using a capnometer. These represent the baseline requirements for monitoring of these patients. Patients receiving inotropic agents to increase MAP and maintain cerebral perfusion pressure (CPP) may benefit from pulmonary artery occlusion catheters to guide therapy.

Key Recommendations

The baseline requirements are SaO2, ECG, MAP, ETCO2, CVP and urine output.

ICP monitoring should be used in most patients with severe brain injury.

Multimodality monitoring including SjO2 and TCD should be employed where ICP and CPP cannot be maintained by standard methods.

Intracranial Pressure Monitoring

Cerebral perfusion pressure is maintained by supporting mean arterial pressure and/or reducing intracranial pressure. The principles of, and indications for ICP monitoring are discussed with the physiology of intracranial pressure. ICP transducers should measure the pressure range of 0 - 100mHg with an accurracy of 2mmHg in the range 0-20mmHg, and at least 10% accurracy for the rest of the measurable range.

The most accurate and reliable method of monitoring intracranial pressure is with an intraventricular catheter connected to a pressure transducer. This system also allows intermittent drainage of cerebrospinal fluid from the ventricles to aid in control of ICP. Manometer type systems allow re-calibration whereas fibreoptic devices may suffer from baseline drift if used for several days. Catheters may also be placed in the cerebral parenchyma, or the subdural and subarachnoid spaces. While easier to insert in some cases these may not accurately measure the ICP when compared to an intraventricular catheter. Epidural devices are significantly less accurate.

In general complications related to ICP monitoring are rare. The greatest concern, ventriculitis, has never been demonstrated in prospective studies of clinically significant intracranial infections following ICP measurement. Bacterial colonisation does occur however (5% ventricular/subarachnoid, 15% parenchymal), and its incidence increases markedly after 5 days in situ. Irrigating ICP devices significantly increases the risk of colonisation. Treatment is removal of the ICP bolt.

It is difficult to assess the risk of haematoma formation associated with ICP monitors but the rate is around 1.4%, with 0.5% requiring surgical evacuation. Parenchymal catheters have a higher incidence of haematoma than other methods. Malfunction of the devices does occur, and readings over 50mmHg may be inaccurate with higher rates of obsturction and loss of signal.

Multimodality monitoring

While maintenance of cerebral perfusion pressure is important, it only measures one parameter affecting the delivery of oxygen to the neurons. Ultimately, the Cerebral Blood Flow (CBF) and oxygen content of the blood are the prime parameters. CPP provides a pressure gradient governing CBF, but flow is then affected by the resistance of the cerebral vessels. Neuronal demand for oxygen is governed by their metabolic rate. Neurons with high activity levels require greater amounts of oxygen than those which are quiescent. Globally this is described as the Cerebral Metabolic Rate for Oxygen (CMRO2).

CMRO2 = CBF x OEF x SaO2

OEF is the oxygen extraction fraction. How much oxygen is extracted can be measured by the Fick principle based on measurements of the arterial and venous oxygen content.

Thus monitoring only the ICP and CPP really gives very little idea of the overall state of the injured brain and no idea at all about oxygen delivery and usage. Multimodality monitoring allows using a combination of jugular venous bulb oximetry and transcranial doppler ultrasound allows a greater understanding of the state of the cerebral circulation and oxygen consumption. At present, methods to measure cerebral blood flow such as Positron Emission Tomography (PET), Xenon clearance or Single Positron Emission Computed Tomography (SPECT) remain too cumbersome for use in the ICU, but will no doubt play a greater role in the future.

Jugular Venous Bulb Oximetry

Jugular venous bulb oximetry involves placing a sampling catheter in the internal jugular vein, directed upwards, so that its tip rests in the jugular venous bulb at the base of the brain. Blood samples drawn from here measure the mixed venous oxygen saturation (SjO2) of blood leaving the brain. This is normally in the range 50-75%. Solving the Fick equation for SjO2 gives:

SjO2 = SaO2 - (Oxygen Consumption / Cardiac Output x Hb x 1.39)

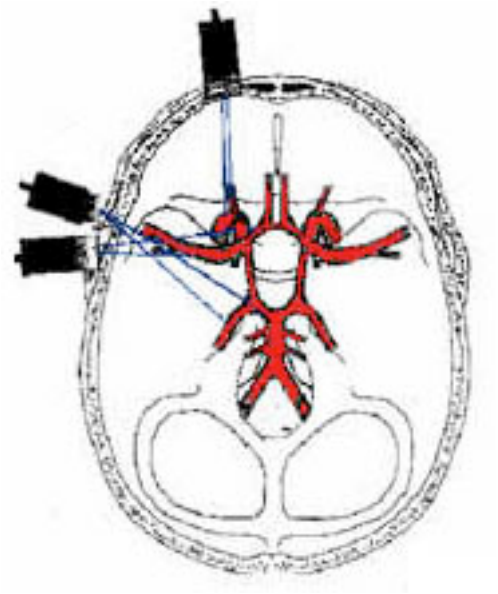
The SjO2 will fall when there is an imbalance between oxygen consumption and delivery. If the SjO2 falls below 50% (without a fall in arterial oxygen saturation - SaO2), this implies either a fall in CBF or a rise in oxygen utilisation (higher CMRO2). If cerebral perfusion pressure is maintained, a fall in CBF is due to an increase in cerebrovascular resistance (CVR). Vascular spasm and a rise in CVR are very common after brain injury and are significantly worsened by hyperventilation. Jugular venous bulb oximetry should be employed whenever there is prolonged hyperventilation

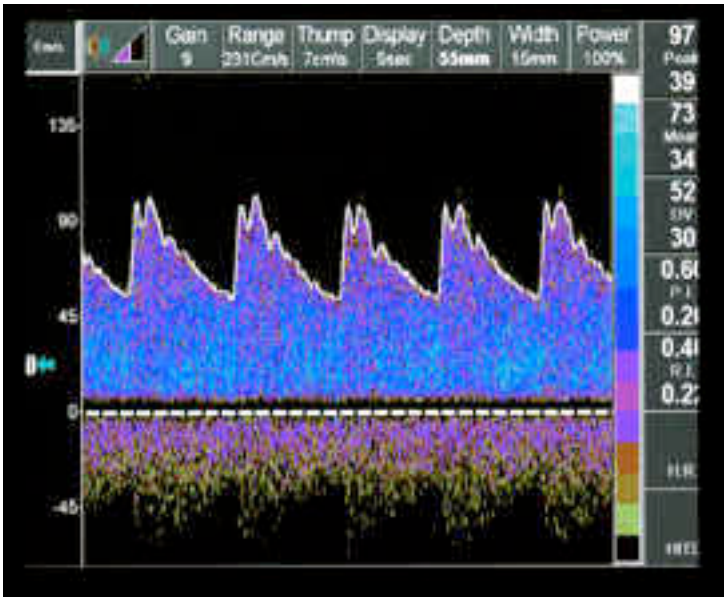
Burst suppression to reduce CMRO2 may be used in patients who have presistent intracranial hypertension not responsive to standard therapy. Barbiturates are the most commonly used agents. Multimodality monitoring should probably be employed when such therapy is instituted.

An increase in SjO2 to 85%+ implies either a hyperaemia with a rise in cerebral blood flow, shunting of blood away from neurons or a decrease in CMRO2 (impending cell death / brain death).

Transcranial Doppler Ultrasound

Transcranial doppler is a non-invasive method of assessing the state of the intracranial circulation. The velocity of flow can be measured in the middle, anterior and posterior cerebral arteries, the opthalmic artery and internal carotid. Flow cannot be measured from velocity because the cross-sectional area of the arteries cannot be measured directly. . However the doppler shift measured is inversely proportional to the diameter of the vessel, so that, all other factors remaining constant, vascular spasm leads to an increase in flow velocity. Doppler waveform analysis can give further information about the state of blood flow, such as flow acceleration and pulsatility index (systolic velocity-diastolic velocity/mean velocity). However the value and utility of these measurements is as yet unknown.





Position of TCD probes and a sample tracing of normal Middle Cerebral Artery (MCA) waveform

There is an an inverse correlation between the severity of head injury and the middle cerebral artery velocity. Low velocities in the intracranial circulation after head injury is due to low cerebral blood flow and high ICP levels. Low velocities on admission are indicative of a poor prognosis. A reduction in CPP and rise in ICP are also reflected in a rise in the pulsatility index.

Vasospasm is common after head injury and can be an important cause of neurologic deterioration. Vasospasm usually occurs where there is traumatic subarachnoid haemorrhage. TCD is useful for monitoring at-risk patients for signs of vasospasm. Detection is made more difficult by the presence of cerebral hyperaemia in many patients, and the ratio of intracranial to extracranial velocities should be used to detect and correct for this. Concomittant jugular venous bulb oximetry may also provide valuable information in this setting.

Interestingly, during hyperventilation to PaCO2 of below 3.5 kPa (25 mmHg) the TCD signal can fall off almost to zero showing how profoundly detrimental hyperventilation can be. Again, TCD is indicated for patients whose intracranial hypertension and cerberal perfusion pressure cannot be maintained by standard therapy. 2 dimensional TCD is now becoming available.

Cerebral Function Monitoring

All the above monitoring technologies assess oxygen delivery to and extraction by the brain. None measure cerebral activity directly. A full

electroencephalograph (EEG) is too complex for continuous use in the ICU, but the Cerebral Function Monitor (CFM) or Cerebral Function Analysis Monitor (CFAM) provide summed, averaged and in CFAM's case, analysed outputs of the general state of brain activity. Evoked potentials, the response to external stimulus (visual, auditory or somatic) may also be of value. Although their place has yet to be fully evaluated, they should probably be employed when efforts are made to control the CMRO2 - ie burst suppression using barbiturate therapy or brain cooling therapy.

Emerging Monitoring Technologies

The above measures are all global in nature, and it is likely that there are significant regional differences in cerebral blood flow and oxygen utilisation in the injured brain. Regional monitoring technologies include non-invasive PET or SPECT imaging. Also being investigated are brain tissue oxygen electrodes measuring tissue oxygen tension (PtiO2) in different regions of the brain and cerebral microdialysis catheters which can provide information on the nature of the cerebral interstitial fluid. Such technologies will no doubt become more prominent in the very near future.

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Intracranial pressure monitoring

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Radiology for Traumatic Brain Injury

The advent of CT scanning has had a huge impact for the treatment for traumatic brain injury. It is rapid, non-invasive and allows identification of surgically treatable lesions as well as diffuse injury. Plain skull -rays have no place in the management of severe blunt head injury. The indications for CT scanning in severe head trauma are not in question - all patients will require a CT scan (unless other injuries prevent this).

In general, indications for CT Scan in mild/moderate head injury are:

1. Neurological signs
2. Decreased level of consciousness
3. Mental state difficult to evaluate

(anaesthesia, drugs & alcohol, young children)

The patient with minimal external signs of injury who is fully alert & orientated (GCS 15) with a normal neurological examination and no symptoms other than headache may not need a CT scan. However they do need close observation for the next 24 hours by reliable observers. Should they require anaesthesia for treatment of other injuries they should have a CT scan prior to surgery.

Non-contrast CT scans are performed using contiguous 5mm slices for the skull base and 10mm slices for the rest of the brain. Bone and brain tissue windows should be examined. For severe brain injury where the patient is intubated, the upper cervical spine should be included in the scan (occiput - C2) as plain AP/odontoid films are difficult and may miss significant spine injury.

Epidural (extradural) haematoma

An epidural haematoma occurs when there is a tear in a vascular structure, usually arterial, in the potential space between the dura and the skull. The haematoma strips the dura off the skull vault and appears on CT as a biconvex lesion. Around 75% are associated with skull fractures. If there are no other brain injuries the patient may remain conscious until the haematoma expands to such a point that brain structures become compressed (the classic lucid interval). There is then rapid deterioration after this point. Surgical evacuation via a craniotomy is necessary and if performed early, before damage to brain structures occurs, can have an excellent outcome.

Subdural haematoma

Subdural haemorrhage usually occurs due to disruption of bridging veins between the brain and the dura. Blood can track around the brain and between the leaves of the falx. The presence of a subdural haematoma is an indication of underlying brain injury, and acute subdurals are associated with a worse outcome than epidural haematomas. On CT the subdural appears as a crescentic extra-axial collection. While surgical evacuation is usually indicated, the underlying brain injury will dictate the subsequent clinical course and functional outcome.

Cerebral contusion

Parenchymal contusions are a manifestation of direct injury to brain tissue. Contusions appear as bright signals within brain tissue, usually in areas abutting the skull or in areas near the zone of impact. Again contusions are more a reflection of underlying brain injury than clinically significant themselves and unless they are large, easily accessible and exert a significant mass effect surgical evacuation will not be of benefit. Multiple pin-point contusions are a sign of diffuse brain injury.

Diffuse Injury

Diffuse injury (or diffuse axonal injury) is due to acceleration and decelaration occurring at different rates across the brain as shear forces are applied during the moment of impact. There is a diffuse, non-focal pattern of injury. CT appearances vary, from a mild appearance with loss of grey-white differentiation, effaced ventricles and a small amounts of intra-ventricular blood, to a more severe picture with multiple contusions, diffuse swelling with loss of the basilar cisterns and brain stem involvement.

The initial CT scan appearance often underestimates the actual brain injury and the patients clinical condition may be much worse than the CT scan would suggest. Diffuse injury evloves and becomes more prominent on CT during the next 48-72 hours after injury. A diffuse injury grading system has been developed based on compression of the basal cisterns and the degree of midline shift apparent.

Diffuse Injury Grade	**CT appearance**	**Mortality**
I	Normal CT scan	9.6%
II	Cisterns present. Shift < 5mm	13.5%
III	Cisterns compressed/absent. Shift < 5mm.	34%
IV	Shift > 5mm	56.2%

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Barbiturate Coma

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Brain damage resulting from head injury is the leading cause of death among individuals younger than 24 years of age. The most hazardous is increased ICP. High-dose barbiturates are used to control intracranial hypertension in selected patients. ICP is decreased due to decrease in CBV due to vasoconstriction caused by increase in cerebrovascular resistance.

Indications:

1.
Potentially survivable head injury

2.
No surgically treatable lesion accounting for intracranial hypertension (except when used for preparation for surgery)

3.
Other conventional therapies of controlling ICP have failed (posture, hyperventilation, osmotic and tubular diuretics, corticosteroids)

4.
ICP > 20 to 25 mmHg for more than 20 min, or >40 mmHg at any time

5.
Unilateral cerebral hemispheric edema with significant (>.7 mm) shift of midline structures shown on CT

6.
A low Glasgow Coma score

Benefits:

1.
Decrease in cerebral metabolic rate (CMRO2), caused by decrease in synaptic transmission, presumably by affecting GABA transmission

2.
Decrease in cerebral blood volume and ICP, due to increase in cerebrovascular resistance, due vasoconstriction -Both CMRO2 and CBF are decreased in a dose dependent fashion: About 50% decrease at a dose sufficient to produce isoelectric EEG

3.
Promote or induce hypothermia

4.
Increase in IC glucose, glucagon, and phosphocreatine energy store

5.
Decrease in nitrogen excretion following acute head injury

6.
Shunt blood from regions of normal perfusion to those of reduced CBF due to vasoconstriction

7.
Anticonvulsant prophylaxis

8.
Stabilization of lysosomal membranes

9.
Decrease in excitatory neurotransmitters and IC calcium

10.
Free radical scavenging (thiopental only)

Risks:

1.
Direct myocardial depressant

2.
Increase in venous capacitance, due to central and peripheral sympatholytic action

3.
Impaired gastrointestinal motility Increased hepatic microsomal activity

4.
Direct CNS depressant, resulting in unreliable neurological examination

5.
Possible allergic reaction Impaired lymphocyte immune response and function

Goals

1.
Maintenance ICP < 20 mmHG

2.
Therapeutic EEG response: burst suppression or cortical electrical silence (with preservation of SSEP and BAEF)

Dosing Regimens

* **Pentobarbital:**
* High dose:

Loading:30-40 mg/Kg over 4 hours (~2500mg/70Kg)
Maintenance: 1.8-3.3mg/Kg/hr (~175mg/70Kg)

* Mid-level dose:

Loading: 10mg/Kg over 30min, 20-25mg/Kg over 4hr

maintenance: 5mg/Kg/hr for 3hrs,then 2-2.5mg/Kg/ with 5mg/Kg bolus prn if serum level < 3mg/dl

* Low dose:

Loading: 3-6mg/Kg over 30min
Maintenance: .3- 3mg/Kg/hr

* Therapeutic serum level: 2.5-4 mg/dl (6 mg/dl may be needed)

* **Thiopental:**

* Loading: 3mg/Kg bolus, followed by 10-20mg/Kg over 1 hr

Maintenance: 3-5 mg/Kg/hr
Therapeutic serum level: 6-8.5 mg/dl
Weaning: dosage is halved q 12 hr.

Monitoring

Cardiovascular

1. A-line: arterial BP, blood gases
2. PA catheter: CO, CI, SV, SVR, PVR, right heart filling pres., PCWP
3. Bladder catheter: urine output

Cerebrovascular and neurophysiological

1. ICP: maintain < 25 mmHg, preferably less
2. CPP: maintain > 70 mmHg
3. EEG: burst suppression, or cortical electrical silence optional
4. Brain temperature
5. Jugular bulb O2 monitor/ oxymeter catheter
6. Somatosensory or brainstem auditory evoked potentials (SSEP, BAEF)

Other monitoring

1. Core body temperature: NP, TM, E: 32 to 35 degrees C is acceptable
2. Serum barbiturate levels
3. nasogastric catheter: pH and output
4. intake and output

Therapy may be required for 7-14 days or longer, may be weaned after 3-6 days

Therapeutic end points

1. **Success**:
a. ICP < 20 mmHg for at least 48 hours, at a minimum
b. Resolution of intracranial mass effects or midline shift, preferably
c. ICP must remain controlled with conventional therapies
2. **Failure:**
a. Diagnosed brain death
b. Uncontrollable ICP despite adequate serum levels, EEG burst-
c. Suppression, or electrical silence
d. Intolerable side effects;
a. Hypotention not responsive to cardiac inotropes, peripheral vaso-pressors, or intravenous fluid therapy (cardiac isotopes: dopamine, dobutamine, epinephrine) (peripheral vasopressors: ephedrine, phenylephrine)
b. (IV fluids: packed RBCs, albumine, hetastarch, LR)
c. Progressive pulmonary dysfunction
d. Sepsis

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