

Management of head injury for anaesthetists

Traumatic brain injury is a common presentation in theatre.

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Many anaesthetists will regularly encounter patients with acute traumatic brain injury (TBI). Anaesthetists play a role during all phases of the management of these patients, including resuscitation, the early operative management of the head injury or associated injuries, and in their intensive care management. It is important to realise that in all patients with TBI the same pathophysiological processes are present, be it mild, moderate or severe TBI. Utilisation of the management principles for severe TBI may be even more critical in patients with mild or moderate TBI, because they have a far greater opportunity to return to their fully functional place in society.

Concept of primary versus secondary injury

The primary injury is caused by the destruction of neurons, the supporting glia and vascular structures due to deformation at the time of injury, which cannot be repaired or prevented after the event. There appear to be several processes initiated by trauma that result in further cell damage or death in the period following TBI. Ischaemia is probably the dominant process and is a multifactorial mechanism.¹

Factors during the post-injury period that impact on neurological outcome are termed secondary injuries. Manipulation of these factors and hence the preservation of injured neurons forms the basis of the current management of TBI.²

There is no 'magic bullet' to improve outcomes following TBI, but a fundamental knowledge of what improves outcome and what may indeed be detrimental will allow the provision of optimum care at all stages following TBI.³

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Important determinants of outcome

Non-modifiable

- **Age.** Very young children and older adults do worse with apparently equivalent injuries.

It is important to realise that in all patients with TBI the same pathophysiological processes are present, be it mild, moderate or severe TBI.

- **Sex.** Women appear to do worse than men.
- **Genetic factors.** The Apo E4 allele of apolipoprotein is associated with worse outcome.
- **Injury severity.** Penetrating injuries, a poor initial GCS and certain initial CT findings are associated with a poorer prognosis.
- **Raised intracranial pressure (ICP).** ICP resistant to medical therapy has a poorer outcome.

Modifiable

- **Blood pressure.** Even brief periods of hypotension (systolic BP <90 mmHg in adults) during the first 72 hours post-TBI correlate with a doubling of mortality.¹
- **Hypoxia.** Brief hypoxic periods are associated with worse outcomes and are compounded by associated hypotension.¹
- **Seizures.** Seizures result in a rapid and sustained rise in ICP. This can precipitate fatal brain herniation, and therefore all patients at risk of convulsions should receive anticonvulsants. Anticonvulsants should be continued until ICP normalises.
- **Hyperglycaemia.** Hyperglycaemia occurs in more than 70% of patients with severe TBI, but there is no definitive work demonstrating that controlling blood glucose improves outcome. Tight control is difficult to achieve and is associated with iatrogenic hypoglycaemia that may cause morbidity.⁴

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- **Hypocapnia.** Hyperventilation will result in an acute drop in ICP, but this effect lasts less than 2 hours. A return to normocapnia will then result in an increase in ICP. Routine hypocapnia is associated with a worse neurological outcome and is therefore not recommended.⁵
- **Temperature.** There is currently no evidence to support the use of hypothermia as therapy.⁶ Hyperthermia is associated with a worse outcome and pyrexia should be aggressively prevented and controlled.
- **Intracranial haematomas** are associated with an increased mortality if not rapidly evacuated.^{7,8}

Principles in the management of patients with TBI

Initial resuscitation

Consider the possibility of a cervical spine injury in all non-penetrating TBIs. Many patients with more severe head injuries arrive with cervical spine immobilisation devices in place. There is work demonstrating that rigid collars result in a rise in ICP, suggesting that we should aim to clear the cervical spine early and remove the collar.

Airway management

Several guidelines recommend that all patients with a GCS of 8 or less should be intubated and ventilated. This is a good generalisation, but exceptions are common and the decision needs to be individualised. While a formal rapid sequence induction is recommended, with attention to preventing and treating hypotension, there is no evidence that it is better than sedation. When strapping the endotracheal tube attention to simple issues such as checking the position of the tube and the strapping is important. Tight and incorrectly positioned ties across the neck can obstruct venous outflow and decrease arterial perfusion.

Ventilation

The use of PEEP remains controversial and

unless required for oxygenation, is probably best avoided. TBI patients are ventilated to prevent secondary injuries due to hypoventilation and to allow adequate and safe analgesia and sedation without the risks of hypoxia and hypercarbia. $P_{ET}CO_2$ should be maintained between 4.0 and 4.5 kPa.⁹

Avoid and treat hypotension

In adults a systolic blood pressure falling below 90 mmHg has repeatedly been demonstrated to impact negatively on outcome. Guidelines suggest aiming for systolic pressure of 120 mmHg and mean arterial pressure (MAP) of 90 mmHg to provide some cushion.⁹ Hypotension is almost never a consequence of the TBI, so spinal cord and haemorrhagic causes need to be excluded.

Mannitol and fluids

Mannitol has been extensively utilised for decreasing ICP in TBI. Recommended doses vary between 0.25 g/kg and 1 g/kg. The routine use of mannitol is not recommended, but in the setting of sudden increase in ICP or threatened brain herniation it can be life saving. Mannitol should be used as a bolus over 10 - 20 minutes and can be repeated safely up to 2 g/kg.

ICP and cerebral perfusion pressure (CPP)

Patients with severe TBI all warrant an ICP monitor (guidelines available at www.braintrauma.org). European and American guidelines agree that sustained ICP above 25 mmHg requires active control measures.⁹ CPP (MAP-ICP) should be kept around 60 - 70 mmHg. A CPP approaching 50 mmHg should be aggressively managed with ICP control and fluids/vasopressors to raise MAP.

Evacuation of intracranial haematomas

Early CT scanning is necessary to expedite the diagnosis and evacuation of intracranial haematomas.

Measures that are probably ineffective

- IV anaesthetic agents are used as a final component in the medical management of raised ICP. Ideally patients should be in a specialist neurocritical care centre by this stage. Barbiturates for this indication have many complications and no proven benefit.¹⁰ Propofol has largely replaced thiopentone in this role but there is no conclusive work demonstrating its efficacy.
- Steroids have no evidence to support their use in humans. Patients receiving steroids may have a worse outcome.¹¹

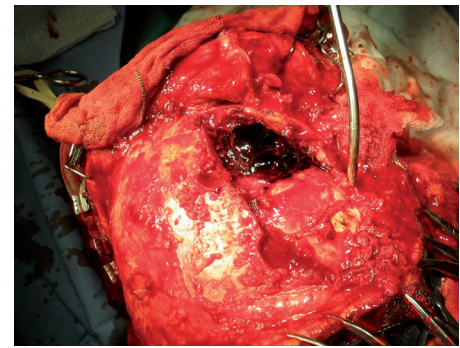


Fig. 1. At craniotomy the large extradural haemorrhage is clearly visible.

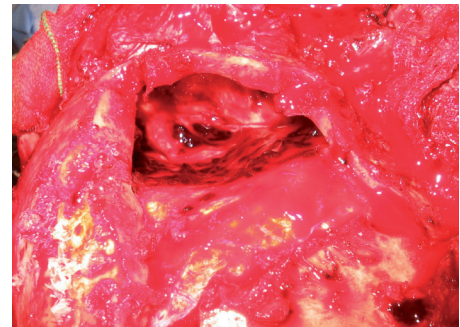


Fig. 2. After removal of this clot the brain is still severely retracted. This unfortunately indicates a high likelihood of permanent disability, despite early surgery.



Fig. 3. A young male patient with a large extradural haematoma.

- Hypothermia: current evidence indicates no benefit, even if patients are cooled to 33°C within 8 hours of injury.⁶
- Routine hyperventilation.⁵

Non-neurological extracranial surgery for the TBI patient

The majority of TBI patients require surgery for their non-neurological injury. Evidence required to guide decision making in this regard is not clear. Patients fall into 2 categories:

- those with a life-threatening injury, e.g. solid organ (liver, spleen, kidney) rupture
- those with a less serious, non-life-threatening condition, e.g. a long-bone fracture.

The timing of surgery depends on the severity of these injuries and their effect on the ability to maintain cerebral protection. When anaesthetising a patient with a TBI the primary goal is the prevention of further cerebral injury.

Head injury with a life-threatening extracranial injury

As many as 70% of all patients sustaining blunt trauma have some degree of head injury and may require resuscitative surgery before head scanning. Once these patients have been haemodynamically stabilised, management of the head injury should take priority. This may include transfer to a centre with a neurosurgical service.

If the patient is haemodynamically stable in the presence of ongoing resuscitation there may be sufficient time to perform required somatic and head CT scans. These will aid in the surgical management of both cranial and extracranial injuries.

Head injury with a non-life-threatening extracranial injury

A limb-saving vascular procedure may be considered as a matter of urgency in all trauma patients.¹² Long-bone fractures are more controversial. Early (<24 h) fixation of long-bone fractures appears to improve outcome and may reduce pulmonary complications, mechanical ventilation time and ICU and hospital stay. Several studies have shown that the timing of surgery is predictive of perioperative hypotension and thus the promotion of secondary brain injury.

The following factors are relevant when assessing the appropriateness of early long-bone stabilisation:

- severity of brain injury
- severity of pulmonary dysfunction, coagulopathy and presence of hyperthermia
- evidence of hypotension and response to initial physiological stabilisation
- risks of delaying surgery.

Long-bone fixation should probably best be undertaken when physiological parameters have stabilised, usually within 48 - 72 hours.¹² Compound fractures can be cleaned and debrided as part of the resuscitative effort. Skeletal traction can be instituted until fracture fixation can be performed.

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Anaesthetic considerations

Widely accepted anaesthetic goals in patients with cerebral injury are:

- the prevention of an increase in ICP during induction and maintenance of anaesthesia
- the maintenance of adequate brain oxygenation and cerebral perfusion pressure
- the maintenance of cerebral autoregulation
- rapid and smooth induction and emergence from anaesthesia.

To achieve this the following perioperative strategies are important:

Monitoring

In addition to all the minimum mandatory monitors the following should be used whenever possible:

- arterial line – provides beat-to-beat measurement of arterial pressure and can be used for blood gas and blood sugar analysis
- central line – particularly useful in polytrauma patients and for patients who require vasopressor support
- pupillary size and reactivity – the only monitor of tentorial herniation in the sedated and ventilated patient
- ICP monitor – a very valuable monitor in the TBI patient undergoing extensive surgery or surgery at multiple sites (e.g. abdominal surgery and long-bone fracture fixation); if such surgery is deemed necessary the patient may need to be referred to a unit where ICP monitoring can be instituted.

Patient position

A 10 - 15° head-up position should be maintained perioperatively whenever possible. Head-down position should be avoided.

Fluid administration

Isotonic crystalloids (normal saline) are the preferred fluids for TBI patients. There is no benefit to the brain in utilising colloids, as the blood-brain barrier reflectance characteristics dictate that the concentration of ions in the plasma determines the movement of free water. Colloids are limited to their role in resuscitation of the intravascular volume. Hypotonic crystalloids (Balsol and Ringer's lactate) should be used with caution. Rehydration solutions (sodium concentrations typically half that of plasma) and 5% or 10% dextrose should not be used.

Hyperventilation

Acute hyperventilation should only be used as a rescue measure to prevent tentorial herniation when all other modalities have failed to alleviate an acute rise in ICP. $P_{ET}CO_2$ should be maintained between 4.0 and 4.5 kPa intraoperatively.

Anaesthetic drugs

Inhalational v. intravenous anaesthesia

Clinical studies comparing inhalational and total intravenous anaesthesia have failed to show any difference in outcome. The best inhalational agents for maintenance of anaesthesia in TBI are isoflurane and sevoflurane. They decrease cerebral metabolic rate ($CMRO_2$) and may cause an increase or decrease in regional CBF depending on the concentration administered. Carbon dioxide reactivity is preserved, as is autoregulation at normocapnia.

Propofol decreases ICP, CBF and $CMRO_2$. It also causes the least interference with autoregulation. When used for maintenance of anaesthesia it allows for rapid recovery and assessment.

Etomidate has a stable cardiovascular profile, but appears to cause a proportionally greater decrease in CBF than $CMRO_2$. Its use in TBI is thus best restricted to the haemodynamically unstable patient.

Thiopentone has anticonvulsant properties, relative cardiovascular stability and allows a smooth induction.

Ketamine causes an increase in CBF, cerebral blood volume and cerebrospinal fluid volume, and is not recommended.

Nitrous oxide

Even though the deleterious effects of nitrous oxide, namely raised ICP, raised CBF, an increase in $CMRO_2$ and impairment of autoregulation are largely antagonised by other anaesthetic agents, its use in the TBI patient is best avoided.

Head injury

Opioids

Continuous infusions and small bolus doses of fentanyl, sufentanil and alfentanil have little effect on CBF and ICP. Large bolus doses, which cause a decrease in MAP, are associated with a transient rise in ICP, further decreasing cerebral perfusion pressure (CPP).

Remifentanyl causes no significant changes in ICP during anaesthesia, but a dose-dependent decrease in MAP and CPP may cause significant alterations in cerebral haemodynamics. Sudden withdrawal may cause agitation and hypertension.

Morphine does not have any direct cerebrovascular effects. Its use as perioperative analgesia for extracranial surgery may aid cerebral protection in the TBI patient.

Non-narcotic analgesia with non-steroidal anti-inflammatory agents and paracetamol should be considered for its excellent analgesic and opioid-sparing effects.

Muscle relaxants

Succinylcholine causes a brief increase in ICP. However, during rapid sequence intubation the potential risks of hypoxia and hypercarbia far outweigh this small and very transient increase in ICP. All of the modern non-depolarising muscle relaxants are suitable for use in TBI patients.

Conclusion

In patients with TBI we need to ensure delivery of oxygenated blood to the brain at all times. Adequate ventilation and

supplementary oxygen need to be ensured and the delivery of oxygen maintained by preventing and aggressively treating any hypotension. We should then exclude any intracranial haematoma. In the event of threatened brain herniation or deterioration in clinical signs, hypocapnia and mannitol can be used acutely to control ICP.

Life-threatening non-neurological injuries should be treated immediately. All other injuries should be considered once the head injury has been stabilised. Attention to the identified principles of care at all times will provide benefit for the patient's TBI.

Once the patient has been stabilised, attention to good nursing care and adequate analgesia/sedation form the backbone of medical care. The use of ICP monitors is not mandatory, but centres with specialist expertise and experience utilise them more frequently and have improved outcomes compared with low-use centres.

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In a nutshell

- The injured brain is susceptible to insults that, under physiological conditions, would not cause damage.
- Preventing and treating secondary insults seems to offer the most hope for optimising outcome following TBI.
- Prevention of hypoxia, hypotension, seizures and the rapid evacuation of intracranial haematomas offer the best advantages.
- Hypothermia, routine hyperventilation and steroids offer no benefit.
- Life-saving surgery may take priority over the TBI.
- Non-life-saving surgery should be delayed until the TBI has stabilised, ideally 48 - 72 hours or more after injury.
- If used carefully most anaesthetic techniques and drugs are suitable, with a few exceptions.
- Attention to the general principles of care, especially avoiding hypotension, is key to maximising the neurological outcome.