

Management of head injuries

Bilal Ali and Stephen Drage

*Correspondence Email: bilalali21@hotmail.com

INTRODUCTION

Trauma is now the leading cause of death in most developed countries in the 18-40 age group and head injury is a major contributing factor. The World Health Organisation estimates that 300 people per day are killed due to trauma on Africa's roads. The most common causes of head injury are falls, road traffic accidents and assaults, with young men and children the most affected. In the UK, around one million people per year attend Emergency Departments due to head injury.

Head injury is defined by the National Institute for Clinical Excellence in the UK (NICE, www.nice.org.uk) as any trauma to the head other than superficial injuries to the face. Mild head injury makes up around 90% of all cases (GCS 13-15), moderate 5% (GCS 9-12) and severe head injury 5% (GCS ≤ 8).

Head injury is a major cause of long term disability and economic loss to society. Much of the neurological damage resulting from a head injury does not occur immediately, but in the minutes, hours and days that follow. It is for this reason that so much emphasis is placed on immediate management of head-injured patients. The primary injury is due to irreversible mechanical injury, but secondary injury which leads to cerebral ischaemia, results from raised intracranial pressure (ICP), hypotension, hypoxia, anaemia, seizures, hypoglycaemia and hyperthermia. Prevention and correct management of these complications improves outcome from head injury.

PRINCIPLES OF MANAGEMENT

The main aim of assessment and management of head-injured patients is to maintain adequate cerebral blood flow (CBF) and to avoid cerebral ischaemia and hypoxia. In patients with a head injury, the normal auto-regulation of CBF is lost and CBF is proportional to cerebral perfusion pressure (CPP), which in turn is directly determined by both the mean arterial pressure (MAP) and the intracranial pressure (ICP):

$$CPP = MAP - ICP$$

The cranium is a rigid structure with a fixed capacity, which contains 80% brain, 10% blood and 10% CSF.

These structures are all non-compressible, therefore an increase in the volume of any of these contents, unless coupled by a decrease in volume of another, results in an increase in ICP.

The main mechanisms of maintaining CPP are to ensure adequate MAP (by the use of fluids and vasopressors) and to prevent excessive rises in ICP. In normal individuals the ICP is 0-10mmHg and this is largely determined by auto-regulation of CBF (i.e. the amount of blood in the cranium). Vasoconstriction or vasodilatation of cerebral vessels occurs in response to changes in MAP, PaO₂, PaCO₂ and blood viscosity. Although these responses may be obtunded in head injury, prevention of secondary brain injury involves manipulation of these variables. An increase in PaCO₂ causes vasodilatation and an increase in CBF, which may increase ICP; a decrease in PaCO₂ causes vasoconstriction leading to decreased CBF and ICP. Thus inappropriate hyperventilation may cause ischaemia. A fall in PaO₂ causes vasodilatation with a consequent rise in ICP.

INITIAL ASSESSMENT

Patients presenting with significant head injury may have multiple injuries. The history of the mechanism of injury is useful in determining the potential extent of the head injury and is also an indication of the likelihood of other injuries. For example, the driver of a vehicle travelling at 60mph and not wearing a seatbelt raises the suspicion of both major head injury and significant extra-cranial injury.

Initial management should be guided by protocols suggested by Advanced Trauma Life Support (ATLS) or Primary Trauma Care (PTC, www.primarytraumacare.org). Injury to the cervical spine should be assumed from the start of assessment. Brain injury may be worsened by airway or circulatory compromise; use the ABC approach to identify and treat life-threatening injuries early (see article on page 95).

Once the patient has a secure airway, is adequately oxygenated and has a stable cardiovascular system, consideration should be given to transfer to a neurosurgical unit (where available). When discussing the case with the neurosurgeon, it is important to

Summary

This article describes the basic principles of management of a patient with a head injury and includes CT images of the common intracranial injuries. It should be read in conjunction with the articles on general trauma management and management of cervical spine injury.

Bilal Ali
Registrar

Stephen Drage
Consultant
Intensive Care Unit
Royal Sussex County
Hospital
Brighton
UK

convey the mechanism of injury, any other injuries and the results of a brief neurological assessment. The surgeon will want to know the history, the Glasgow Coma Score (GCS) at the scene, on arrival at your hospital and the current GCS (especially the motor score), the pupillary size and reaction, and whether there are any signs suggesting a collection of blood on one side of the cranial cavity ('lateralising' signs).

THE GLASGOW COMA SCALE

The GCS is the globally accepted method of quantifying and recording the neurological status of the head-injured patient. It is also useful in determining any improvement or deterioration in neurological function and facilitates accurate communication between health professionals. The scale is made up of three sections, with a minimum score of 3 and a maximum of 15. The best score in each section should be recorded e.g. if the patient localises with the right arm but extends on the left, then the best motor score is 5/6).

The components of GCS are:

Eye opening

- Spontaneously 4
- To speech 3
- To pain 2
- None 1

Verbal response

- Orientated 5
- Confused 4
- Inappropriate 3
- Incomprehensible sounds 2
- None 1

Motor response

- Obeys commands (for movement) 6
- Purposeful movement to painful stimuli ('localises') 5
- Withdrawal from painful stimuli 4
- Abnormal (spastic) flexion, decorticate posture 3
- Extensor (rigid) response, decerebrate posture 2
- None 1

The standard painful stimulus applied to the patient should allow the differentiation of purposeful movement ('localising'), from withdrawal and abnormal flexion. Strictly speaking true localisation or purposeful movement should follow a stimulus from one site to another. Squeezing/pinching the trapezius muscle and supra-orbital pressure are preferred stimuli. Nail bed pressure and sternal rub are less reliable and not of use in patients with spinal injury. Care must also be taken when assessing motor response in those with a suspected cervical spine injury, as any response may cause the patient to attempt to move their head.

The **Blantyre Coma Score** was originally designed for treatment of children with malaria, but is useful for assessment of children with head injury:

Eye movements

- Watches or follows (e.g. the mother's face) 1
- Fails to watch or follow 0

Motor response

- Purposeful movement to painful stimuli ('localises') 2
- Withdraws from pain 1
- No response or inappropriate response 0

Verbal response

- Cries appropriately with painful stimulus, or if verbal speaks 2
- Moan or abnormal cry with painful stimulus 1
- No vocal response to painful stimulus 0

MANAGEMENT

The main aims of management of any moderate or severe head injury are initial assessment and resuscitation, deciding whether ventilatory support is necessary and establishing a diagnosis, with a CT head scan if this is available. Early contact with specialist neurosurgical units is key; they will often advise on specific therapies. Early transfer, when indicated, is also important. The Association of Anaesthetists of Great Britain and Ireland suggest a maximum time of 4 hours between injury and surgery. Throughout this process management should be equal to that in an ICU, directed at maintaining the MAP and CPP and preventing rises in ICP.

Airway

The main concern is whether the patient is able to protect their airway and therefore whether intubation is necessary. Indications for intubation include:

- GCS \leq 8
- Risk of raised ICP due to agitation (i.e. sedation required)
- Inability to control/protect the airway or loss of protective laryngeal reflexes
- A fall of 2 or more points in the motor component of the GCS
- In order to optimise oxygenation and ventilation
- Seizures
- Bleeding into mouth/airway
- Bilateral fractured mandible.

This is not an exhaustive list and clinical judgement is important. If there is doubt, it is safest to intubate and consider early extubation

rather than delay intubation and risk secondary brain injury from hypoxia.

Rapid sequence intubation is almost always required. Maintain cervical spine immobilisation during intubation, unless the cervical spine has been clinically and radiologically cleared. Avoid the temptation to use no drugs in profoundly unconscious patients; some hypnosis and analgesia is required to obtund the rise in ICP that is inevitably caused by laryngoscopy. Propofol, etomidate, benzodiazepines and barbiturates all reduce ICP and are preferentially used. Ketamine produces a rise in ICP, but may be the only induction agent available in certain countries. Opioids and depolarising neuromuscular drugs do not increase ICP. The fasciculations caused by suxamethonium may cause a transient rise in ICP. Nitrous oxide may also cause a rise in ICP via increased blood flow.

Detection of cervical spine injuries is described in the article on page 112. All patients with head injury should have plain Xrays of the cervical spine and some may require a CT scan.

Breathing

Hypoxaemia is associated with a significant increase in mortality. A drop in PaO₂ below 8kPa (about 60mmHg) causes an increase in CBF and ICP. Targets for gas exchange should be a PaO₂ greater than 13kPa (100mmHg) and a PaCO₂ in the low normal range - usually 4.5-5.0kPa (35-39mmHg). Prolonged hyperventilation is not recommended since cerebral vasoconstriction and ischaemia may result, but short bursts of hyperventilation (a few minutes) may help to control episodes of high ICP.

Circulation

The loss of the autoregulation of CBF can result in a reduction in oxygen delivery. Maintenance of the MAP and CPP is essential; resuscitation and treatment of life-threatening circulatory instability should take precedence over neurosurgical interventions. This may

include surgery for haemorrhage control.

Use fluids, and where necessary vasopressors to achieve a MAP greater than 80-90mmHg. This figure is recommended as a guide until ICP monitoring is established, and assumes that the ICP is 20mmHg and therefore ensures a CPP of at least 60-70mmHg (since CPP = MAP – ICP). Once ICP monitoring is established then treatment is targeted at maintaining CPP 60-70mmHg. Aiming for higher CPP targets has been associated with adverse cardio-respiratory outcomes.

Ideally the MAP is measured using an arterial line. A central venous catheter may be useful for monitoring and the administration of vasopressors. A urinary catheter allows monitoring of urine output and fluid balance, especially if mannitol or other diuretics are used.

MONITORING INTRACRANIAL PRESSURE

Some clinical signs are suggestive of raised ICP. These include:

- Headache
- Dizziness
- Loss of consciousness
- Confusion
- Hypertension and bradycardia (Cushing's reflex)
- Nausea
- Vomiting
- Focal weakness or paresis
- Other focal neurological signs
- Change or asymmetry pupils.

Measurement of ICP

ICP can be measured using the techniques described in Table 1.

Table 1. Measurement of ICP.

Method	Benefits	Disadvantages
Intraventricular catheter (EVD or external ventricular drain)	<ul style="list-style-type: none"> • Gold Standard method • Allows CSF drainage to lower ICP • Re-zeroing possible 	<ul style="list-style-type: none"> • Most invasive method • High infection rate • May be difficult to insert • Simultaneous CSF drainage and ICP monitoring not possible
Extradural probe	<ul style="list-style-type: none"> • Low infection rate (no penetration of dura) • Easy to insert 	<ul style="list-style-type: none"> • Limited accuracy • Relatively delicate
Subarachnoid probe	<ul style="list-style-type: none"> • Low infection rate • No brain penetration 	<ul style="list-style-type: none"> • Limited accuracy • High failure rate
Intraparenchymal probe	<ul style="list-style-type: none"> • Low infection rate 	<ul style="list-style-type: none"> • Measures local pressure
Transcranial Doppler	<ul style="list-style-type: none"> • Non invasive 	<ul style="list-style-type: none"> • Limited precision
Lumbar CSF pressure	<ul style="list-style-type: none"> • Extracranial procedure 	<ul style="list-style-type: none"> • Inaccurate reflection of ICP • May be dangerous when brain oedema present
Tympanic membrane displacement	<ul style="list-style-type: none"> • Non-invasive 	<ul style="list-style-type: none"> • Insufficient accuracy

MANAGEMENT OF RAISED ICP

Improving venous drainage from the brain

- Elevation of the head of the bed to 30°.
- Good neck alignment – head in the neutral position.
- Ensuring ties holding the endotracheal tube in place do not compress the neck veins. Alternatively tape the tube using 'trouser-legs'.
- Where possible immobilise the patient's cervical spine with sandbags and tape rather than restrictive neck collars.

Reducing cerebral oedema

- Use mannitol (an osmotic diuretic) 0.5-1g.kg⁻¹ (= 5-10ml.kg⁻¹ of 10% or 2.5-5ml.kg⁻¹ of 20% mannitol). Some units use small aliquots of hypertonic saline as an alternative.
- Use furosemide (a loop diuretic) 0.5-1mg.kg⁻¹.
- Maintain serum Na⁺ in the range 140-145mmol.L⁻¹.

Reduction of the cerebral metabolic rate for oxygen

- Close temperature regulation. Avoid hyperthermia, but do not actively induce hypothermia.
- Use of sedation and anaesthetic drugs. Ensure that the patient is appropriately sedated and has received adequate analgesia.
- If the patient has a witnessed seizure loading with an anticonvulsant, usually phenytoin 18mg.kg⁻¹, should be considered.
- In cases of intractable raised ICP, a thiopentone infusion can be used to reduce the cerebral metabolic rate to a basal level. This is identified on EEG monitoring as 'burst suppression'.

Reducing intracranial blood volume

- Consider whether the patient has suffered a new or worsening intracranial haemorrhage. Are there any new or lateralising signs? Is a repeat CT scan required?
- Hyperventilation can be used to reduce the PaCO₂ as a temporary measure, but cerebral ischaemia may result if this is prolonged (more than a few minutes).
- The final resort if ICP remains raised is to perform a decompressive craniectomy (part of the cranial bone is removed).

Reducing CSF volume

- In a neurosurgical centre, use of an external ventricular drain (EVD) allows drainage of CSF to relieve raised ICP.

TRANSFER TO NEUROSURGICAL UNIT

Where there is a regional neurosurgical service, you may need to refer a patient or obtain advice. Electronic transfer of CT images allows the neurosurgeon to see the scans straight away and reduces delay. If the patient's condition changes significantly you should seek further advice. Some patients will benefit from being transferred to a neurosurgery centre.

Full resuscitation and stabilisation of the patient and all injuries must be completed prior to transfer. A doctor with appropriate training and

experience should oversee the transfer of the patient, the goal being continuous management to the standard available in the ICU. Ideally, monitoring for transfer should include ECG, invasive blood pressure, pulse oximetry, urinary catheter/output and capnography. Pupillary size and reaction to light should also be monitored. It is useful to check an arterial blood gas prior to departure and to correlate the PaCO₂ to the end-tidal value as the end tidal value is usually 0.5 - 1kPa higher. As with all transfers, think what may go wrong and check you have the facilities to deal with it en route.

TYPES OF INJURY

Traumatic subarachnoid haemorrhage

This is the most common type of intracranial haemorrhage. Blood is seen in the CSF and subarachnoid space. It is often caused by tearing of small subarachnoid blood vessels. Vasospasm may complicate traumatic subarachnoid haemorrhage and the amount of blood is related to the patient's GCS and outcome.

Acute subdural haemorrhage (Figure 1)

This type of injury is often caused following forceful acceleration-deceleration events. Blood is seen on CT between the dura and the brain. Rapid neurosurgical intervention is often required, necessitating rapid transfer. On CT scan the border of the haematoma next to brain tissue is typically concave (i.e. curved inward) towards the midline.



Figure 1. CT scan showing large left fronto-parietal subdural haematoma (A), with midline shift and compression of the left lateral ventricle.

Epidural (extradural) haemorrhage (Figure 2)

This is seen in up to 1% of cases. Blood is seen on CT between the skull and the dura. The classical presentation is of a patient who initially has loss of consciousness and is then lucid, before deteriorating again. Extradural haemorrhages often occur in conjunction with skull fractures, particularly over the course of the middle meningeal artery.



Figure 2. CT scan showing bilat extradural haematomas (B).



Figure 3. CT scan showing a fronto-parietal intracerebral haematoma (C) with surrounding oedema (D) and midline shift.

Prognosis is good if surgery is performed promptly. On CT scan the border of the haematoma next to brain tissue is typically convex towards the midline.

Intracerebral haemorrhage (Figure 3)

This is an injury deep within the brain itself, and is caused by shearing forces between the cranium and brain. It is most common around the frontal and temporal regions, with 50% of cases suffering loss of consciousness on impact.

Diffuse Axonal Injury

This is the primary lesion in around 40-50% of severe head injuries, and is secondary to shearing and tensile forces. The prognosis is linked to the clinical cause, with prolonged coma suggesting severe, irretrievable injury.

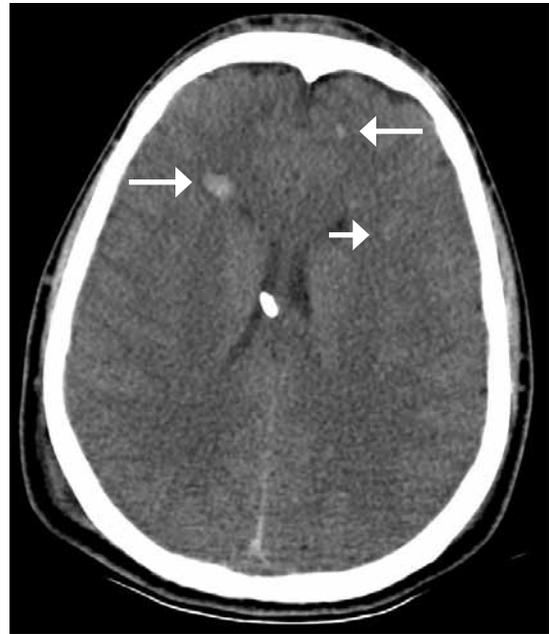


Figure 4. CT scan showing diffuse axonal injury with frontal petechial haemorrhages (arrows).

FURTHER READING

1. Recommendations for the safe transfer of patients with brain injury. The Association of Anaesthetists of Great Britain and Ireland (2006). Available at <http://www.aagbi.org/sites/default/files/braininjury.pdf>
2. NICE Guidelines. Head injury: triage, assessment, investigation and early management of head injury in infants, children and adults (2003). Available at: <http://www.nice.org.uk>
3. Primary Trauma Care. Trauma resuscitation guidelines for resource limited countries. Available at: www.primarytraumacare.org
4. Brain Trauma Foundation. Evidence based guidelines for head injury management. Available at: www.braintrauma.org