

Management of head injury in the intensive-care unit

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Key points

Head injuries are common and have a major impact predominantly on young individuals.

Key principles of head-injury management can be started outside the intensive-care unit.

Management is based on maintenance of normotension, normoxia, normocapnia, normothermia and normoglycaemia.

The monitoring of intracranial pressure may allow early identification of patients requiring surgical intervention.

Few good data exist for evidence-based practice, and collaborative studies are required.

Approximately one million patients present to hospital in the UK each year having suffered a head injury. The vast majority of these patients have minor (GCS 13–15) or moderate injuries (GCS 9–12) and approximately half are less than 16 yrs old. In adults the age distribution is bimodal, comprising young people (15–29 yrs) involved in road traffic accidents (responsible for approximately 50% of head injuries) and elderly people involved in domestic accidents. Overall, males are 2–3 times more likely to have a head injury than females.

Head injury is associated with tremendous mortality and morbidity. One percent of all deaths in the UK are attributed to head injury; up to 85% of all severely head-injured patients remain disabled after 1 yr and only 15% have returned to work at 5 yrs. Even after apparently mild head injury, nearly 50% of patients have moderate or severe disability 1 yr later and only 45% return to full functional activity. Therefore, for both individual and economic reasons, small improvements in the management of head-injured patients may have a great effect on outcome. This article will address the main principles of head-injury management in the intensive-care unit (ICU) after severe isolated traumatic brain injury, the use of additional monitoring devices and alternative management protocols. Some of the pitfalls and failures of head-injury research and some of the potential areas of future development are discussed. The management of patients with multiple trauma will not be addressed.

Classification

Head injuries may be classified in different ways – for example, according to the nature of the insult (penetrating or blunt); concomitant injuries (isolated head injury or multiple trauma); and the timing of the injury (primary or secondary). Primary injury is that occurring at the scene and is usually outside the control of the intensivist. Secondary injury is anything that occurs to augment the primary injury; the prevention of this is predominantly where intensive therapy is aimed.

The Glasgow Coma Score (GCS) remains the most commonly used method of assessing the severity of the head injury; and although the overall score is predictive of outcome, the motor part of the score has the greatest predictive ability. Other scoring systems such as the Virginia prediction tree aim to take features other than the level of consciousness into account and to enhance the outcome prediction made.

Pathophysiology

A range of pathological processes may be involved in a head-injured patient.

Cerebral contusions

Cerebral contusions are essentially areas of ‘bruising’ within the brain tissue with relatively localized cellular damage, haemorrhage and oedema. These may be large haemorrhagic regions or small ‘point’ contusions. The effects of these on outcome will depend on location and size along with pressure effects that they may generate locally. As with contusions elsewhere in the body, the associated maximal swelling and bleeding is often not seen until up to 72 h after the initial insult.

Diffuse axonal injury

Diffuse axonal injury, depicted by loss of grey/white differentiation on the computed tomography (CT) scan, is caused by widespread shearing forces that occur as the brain undergoes stresses such as rapid deceleration.

Traumatic subarachnoid haemorrhage

Traumatic subarachnoid haemorrhage (SAH) is bleeding associated with tearing of an intracranial vessel by the shaking of brain tissue in a traumatic situation. As for non-traumatic SAH, traumatic SAH may be associated with vasospasm. Evidence for the beneficial effects of nimodipine in this situation has been limited by poor-quality studies, and it cannot be

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recommended unless vasospasm has been demonstrated by angiography or alternative imaging techniques.

Epidural or subdural haematoma

Epidural or subdural haematomata occur frequently after trauma; and if bilateral, the associated localizing signs may be absent. Epidural haematomata may have relatively little underlying associated 'brain damage'; although if of sufficient size, brain compression and ischaemia may occur. Early evacuation is generally associated with a good outcome. Subdural haematomata, because of the involvement of brain tissue, have a much worse prognosis. Surgical evacuation will usually be performed if there is evidence of any mass effect or increased intracranial pressure (ICP) to which the haematoma may be contributing.

Transfer of the head-injured patient

Patients admitted to a hospital in the UK should be considered for transfer to a neurosurgical centre if they meet the following criteria:

- severe head injury or focal signs (whether or not they need neurosurgical intervention); and
- needing ventilation, ICP monitoring, or both.

Transfer should take place in a manner consistent with the AAGBI (Association of Anaesthetists of Great Britain and Ireland) and ICS (Intensive Care Society) guidelines and should occur after full discussion with, and ideally after review of CT scans by, the regional neurosurgeons. The essential principles of the initial management of the patient with an isolated head injury before transfer are given in Table 1.

Factors influencing outcome

It should be clear from the outset that the evidence base for the treatment of head-injured patients with severe trauma is extremely limited. After fully reviewing the literature, the Brain Trauma Foundation (BTF), in collaboration with the American Association of Neurological Surgeons, concluded that there are insufficient data to support a treatment standard or a treatment guideline for the initial management of the head-injured patient. The report has proposed a number of options, with the underlying principle being complete and rapid physiological resuscitation.

Table 1 Checklist for safe transfers if the Glasgow Coma Score is less than 8

Airway secured definitively
Staff trained in airway and head injury
Large-bore intravenous access
End-tidal CO ₂ maintained at 4–4.5 kPa on transport ventilator
SpO ₂ and arterial gases checked for adequate oxygenation
Blood pressure adequate and fluids and pressors available
Brain CT imaging completed and hard copies available
Transfer complete within 4 h – no inappropriate delay (e.g. for central venous access)

There are five key principles that should guide the ongoing management of the head-injured patient on the ICU – normotension, normoxia, normocapnia, normothermia and normoglycaemia.

Normotension

It is well documented that even a single episode of systolic pressure below 90 mm Hg has a direct negative effect on outcome after traumatic brain injury. Strenuous attempts need to be made to maintain the blood pressure in the normal range. This should be initially with fluid resuscitation and then by the use of vasopressor agents. There is insufficient evidence to make a strong recommendation for one pressor agent over another, and in our unit nor-epinephrine is the agent most used. It is probably important to maintain a mean arterial pressure (MAP) of at least 70 mm Hg; although not tested in a blinded randomised study, this is consistent with cerebral perfusion pressure targets described below.

Normoxia

A significant body of evidence shows that hypoxaemia (defined as SpO₂ < 90%) is associated with worsened outcome. In one study, there was approximately a four-fold increase in mortality in patients with documented hypoxaemia (SpO₂ < 60%) compared with non-hypoxaemic patients. Maintenance of oxygenation needs to be balanced against the cardiovascular effects of additional PEEP (positive end-expiratory pressure); in patients with combined head and chest trauma, a compromise may have to be reached to provide the best possible conditions for the brain, potentially at the expense of a 'protective lung strategy' for ventilation.

Normocapnia

Hyperventilation (PaCO₂ < 25 mm Hg) should be specifically avoided in the first 24 h after traumatic brain injury and should not be a target for prolonged ventilation beyond this time period. Hyperventilation results in cerebral vasoconstriction and a subsequent decrease in cerebral blood flow. In the first 24 h after a traumatic brain injury, cerebral blood flow is reduced to approximately half that of normal, and aggressive hyperventilation may result in further cerebral ischaemia.

Normothermia

An increase in body and brain temperature is associated with an increase in cerebral blood flow, cerebral metabolic oxygen requirement and oxygen utilization, resulting in an increase in ICP and further potential brain ischaemia. Therefore, avoidance of hyperthermia should be one of the mainstays of head-injury management; it may require the use of pharmacological antipyretics and surface cooling measures.

Normoglycaemia

The brain is an obligate glucose user. Hyperglycaemia is associated with an increase in cerebral metabolism; because of decreased cerebral blood flow subsequent to trauma, this results in additional anaerobic metabolism. Blood sugar concentrations need to be controlled tightly with insulin infusions; administration of dextrose infusions should be avoided. We would normally aim to maintain blood glucose between 4–8 mmol litre⁻¹ in these patients.

Additional monitoring

All five key principles of care can be offered by any ICU. However, in patients with a severe head injury, additional monitoring may be helpful in management, particularly to guide the timing of repeat scans and neurosurgical intervention. The benefit of the additional monitoring modalities in terms of mortality or morbidity is unclear at the present time.

ICP

The BTF guidelines suggest that there are inadequate data to make ICP monitoring a treatment standard. However, they suggest the following guideline:

Intracranial pressure monitoring is appropriate in patients with severe head injury with an abnormal admission CT scan. An abnormal CT scan of the head is one that reveals haematomas, contusions, oedema or compressed basal cisterns.

ICP monitoring may be used to guide therapies to limit the increase in ICP or to allow calculation and maintenance of a cerebral perfusion pressure (CPP) (see below). Patients with severe head injury and a high ICP have a poorer prognosis than those with a normal ICP. The critical ICP at which action is taken to limit further increase is not clear and varies between 15 and 30 mm Hg. However, 20 mm Hg seems to be accepted as the treatment threshold by a large number of authorities. A management protocol for patients with an ICP above 20 mm Hg is discussed below.

However, it is possible that the combination of ICP and MAP is more important than the ICP alone. Thus, CPP may be a more appropriate measure (and target) than ICP, where CPP is taken as MAP – ICP.

ICP measurement has never been subjected to a randomized double-blind study, and to do so would be extremely difficult. However, there is a substantial body of evidence that suggests that it helps in early detection of mass lesions (*e.g.* epidural or subdural haematomata), may limit the indiscriminate use of therapies to control ICP (which in themselves may be harmful) and may be helpful in determining prognosis.

ICP may be monitored from various sites using a variety of devices. A solid-state intraparenchymal monitor is associated with a reduced risk of intracranial infections. This is not true of an intraventricular catheter; however, this will allow withdrawal of

cerebrospinal fluid (CSF) and thereby provide an alternative method of ICP control. Subdural or epidural catheters have also been used but carry the risk of infection without the potential benefits of CSF aspiration. The preferred site for the ICP monitoring device is the right frontal lobe (non-dominant hemisphere, minimal essential brain tissue). However, this may or may not be the tissue involved in the head injury, and interpretation of pressure readings may be difficult if the monitor is sited in the middle of an expanding contusion.

Alternative head-injury management protocols

CPP management (Rosner)

In 1995, Rosner and colleagues published a study in which 158 patients with severe traumatic brain injury were managed with vasopressors (norepinephrine or phenylephrine) to maintain CPP above 70 mm Hg. The outcome of these patients was compared with ICP-based management protocol patients collected in the Traumatic Coma Data Bank (TCDB). They found that in all GCS categories morbidity and mortality improved with CPP management when compared with the TCDB data. The overall mortality in this group was 29%; and 2% remained vegetative.

A number of studies have been performed to further validate this management protocol. However, there have been no randomized controlled studies that allow this to be confirmed as the optimal standard of care.

Lund protocol

In 1998, neurocritical care physicians in Lund, Sweden, questioned the use of CPP targeted treatment protocols. They suggested that high CPP management may have the adverse effects of triggering the development of vasogenic brain oedema through forces striving toward the classical Starling equilibrium causing an increase in ICP. The increase in ICP would counteract the desired increase in CPP and brain would become more likely to herniate. They also hypothesized that the use of vasopressor agents may enhance the vasoconstrictor response that is likely to be present after trauma. They proposed a treatment protocol that included the following:

- preservation of normal colloidal-absorbing force;
- a reduction in intracapillary pressure by antihypertensive therapy using clonidine and metoprolol;
- a simultaneous moderate constriction of the precapillary resistance vessels with low-dose thiopental and dihydroergotamine; and
- optimal general intensive care (*i.e.* fluids to maintain normovolaemia, monitoring of lung function, nutrition and electrolyte supplementation).

They compared 53 patients managed according to this protocol with historic controls and found mortality to be significantly lower

in the protocol group (8%); the ratio of patients with vegetative or severe disability was about the same (13%), resulting in a higher proportion of patients having a favourable outcome. Further studies have been performed using this protocol with similar results.

Jugular bulb oxyhaemoglobin saturation protocol

In 1998, Cruz published a review of 10 yrs of jugular bulb monitoring comparing the outcome of 178 patients with severe acute closed brain trauma managed by a combination of CPP management and jugular bulb oxyhaemoglobin saturation with 175 patients managed by CPP monitoring only. Mortality was 9% in the jugular bulb group compared with 30% in the CPP group. The jugular bulb protocol was aimed not only at maintaining normal ICP and CPP but also at maintaining normal coupling between cerebral blood flow and oxygen consumption (*i.e.* to normalize cerebral oxygen extraction). This was achieved by the use of hyperventilation, sodium thiopental and mannitol. There have been a number of subsequent reports, both of the benefits of using this approach in addition to CPP management and of potential problems with jugular bulb oximetry.

Summarizing management protocols

There are several alternative protocols for the management of the severely head-injured patients, all of which claim excellent results. Rosner's CPP management protocol remains the most widely used and accepted protocol, although 'multi-modality monitoring' is increasingly reported in the literature. 'Additional' modalities include jugular bulb oxygen saturation, transcranial Doppler ultrasound, SPECT (single-photon emission computed tomography) scanning and brain tissue oxygen monitoring. These additional modalities are often expensive, time-consuming, may overcomplicate management and are frequently only fully practical for research purposes. The evidence for the additional benefits of these modalities is also poor to date.

Management algorithms for increased ICP

Whatever the protocol being used to manage the severely head-injured patient, it is generally agreed that the higher the ICP, the worse the outcome. The BTF guidelines suggest that the ICP should be maintained below 20 mm Hg. A number of algorithms may be used to achieve this, commonly including the following medical and surgical practices.

Medical

Positioning

The patient should be nursed in a head-up ($\sim 30^\circ$) position to improve venous drainage and reduce ICP. In order to do this, it is essential to be certain about the integrity of the spine; good working protocols for early clearance should be in place.

Sedation and neuromuscular block

Deep sedation (to Ramsay score of 6 or equivalent) is used to reduce cerebral metabolism. In some units, neuromuscular blockers are used as standard; in others, they are used when ICP remains difficult to control, all other medical measures are in place and there is a concern that muscle activity may be contributing to the pressure. If the ICP remains difficult to control, a thiopental infusion may be used in conjunction with electroencephalographic (EEG) monitoring to bring about burst suppression. Once this is achieved, other sedation agents can be withdrawn.

Ventilation and carbon dioxide control

The objective of mechanical ventilation is to maintain P_{aCO_2} at 4–4.5 kPa. If ICP becomes dramatically increased, short-term hyperventilation may be used to gain control while other measures (*e.g.* mannitol) take effect.

Mannitol and osmolality management

An increase in serum osmolality will result in a tendency to decrease brain tissue water and hence decrease ICP. A serum osmolality of 300–310 mosm is targeted in our unit, achieved by incremental 100 ml doses of mannitol 20%.

Seizure control

Both clinical and subclinical seizures may have dramatic effects on cerebral metabolism and ICP; they should be prevented. In patients receiving neuromuscular blocking drugs or in whom subclinical seizures are suspected, EEG monitoring may aid detection of the fits.

Temperature control and induced hypothermia

For reasons given above, an increase in body temperature to more than 37°C should be actively avoided. Induced hypothermia remains contentious and there is conflicting evidence as to whether it affects outcome. There is some evidence that below 35°C brain tissue oxygenation may be impaired, but generally there is agreement that cooling will result in a decrease in ICP.

Surgical

CSF drainage

If hydrocephalus is demonstrated on CT scan in a patient with increased ICP, CSF drainage will usually decrease this pressure. In situations in which hydrocephalus is not demonstrated, great care must be exercised. In many patients, the ventricles will be flattened and further supratentorial CSF drainage is not possible. Lumbar drainage of CSF may be dangerous and should only be performed following neurosurgical advice.

Craniectomy

A bifrontal decompressive craniectomy may be performed to allow the brain tissue to expand and decrease the ICP. This technique has not been studied in a randomized trial, although scattered reports in the literature suggest that it may be beneficial.

Lobectomy/removal of contusion

Either lobectomy or removal of contusion may be possible surgically, depending on the nature and location of the brain injury and whether there is midline shift that may be exacerbated by removing non-dominant tissue. Again, there is little evidence in terms of improved outcome to support this.

Difficulties with head-injury research

Before concluding this review of head-injury management, with its many references to the lack of available data, it is worth considering why head-injury research is so difficult. Contributory factors include the ubiquitous use of the GCS, the dynamic nature of head injury over time and the lack of collaborative research.

The GCS is used virtually universally to determine the severity of a traumatic brain injury, and it shows a very good relationship to outcome. However, many different intracranial pathologies can result in a GCS of less than 8 (*e.g.* epidural, subdural or intracerebral haematomata, multiple supratentorial or single infratentorial contusions, diffuse axonal injury, or any combination of these). Each of these conditions is associated with a different outcome (see Virginia prediction tree for some further explanation). However, most interventional studies have grouped patients together as severe head injury (GCS < 8) whatever the aetiology, even though the intervention may be less appropriate for some patients than others. It is extremely likely that penetrating and blunt head trauma will need different management approaches, and yet these have rarely been explored.

A head injury is not a static event that occurs at time zero and recovers to normal at a defined later time point. It is a dynamic process that changes over days, weeks and months after the event as various physiological processes are involved, and final outcome cannot be assessed until at least 6 months after the head injury.

One method of management may be appropriate in the early phase of the injury and another method later on. Again, to date, most research has been directed at finding one treatment protocol that can be applied to all patients throughout their critical care stay; this is inappropriate. The only possible way forward is through widespread collaborative research. However, this is very expensive and time consuming, and appropriate networks do not currently exist.

Several avenues of research are being investigated for the prevention of secondary brain injury, including NMDA (*N*-methyl-D-aspartate) antagonists, steroids and magnesium. Results of studies in these areas are awaited.

Key references

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See multiple choice questions 39–43.