The Newcastle upon Tyne Hospitals NHS Foundation Trust
Critical Care Management of Severe Traumatic Brain Injury

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1 Introduction

Traumatic Brain Injury (TBI) is one of the most important causes of death in the UK for adults under 45 and is a growing problem in the elderly population. Survivors of severe TBI live with significant disabilities, and need extensive health and social care support. This causes a considerable socio-economic burden for society.
Over the past thirty years, the care of patients with severe TBI has been standardised by the development of international guidelines.
Evidence shows that treatment in large trauma centres offering neurosurgical treatment and access to specialised neuro-critical care is associated with better patient outcomes, especially when such units use a protocolised based care approach.

2 Guideline scope

This guideline is intended for the treatment of adult patients with severe traumatic brain injury on ward 18 in the RVI. The management of 'clearing' the cervical spine, intracranial hypertension and disorders of sodium and water balance are described in associated guidelines.

3 Main body of the guideline

3.1 Initial Assessment and Neuroimaging

The initial assessment should be guided by ATLS standards.

A Airway (with cervical spine control)
B Breathing
C Circulation (with haemorrhage control)
D Disability (neurological assessment)
E Exposure (to identify all injuries)

The assessment of ABC should not be delayed by neurological assessment. Indications for intubation and ventilation include:

1. GCS < 8 or a rapidly falling GCS
2. Loss of protective laryngeal reflexes
3. Hypoventilation: hypoxaemia (PaO_2 < 13 kPa on oxygen), hypercarbia ($PaCO_2$ > 6 kPa)
4. Spontaneous hyperventilation causing $PaCO_2$ < 3.5 kPa

5. Bleeding into oropharynx
6. Inability to lie still for CT when indicated
7. Seizures

Patients admitted with TBI should be assessed for cervical spine injury as there is a clear association. Full cervical spine protection should be applied until it can be cleared either clinically or radiologically.
The patient should have two large bore peripheral venous cannulae (≥ 16 G) and fluid resuscitation should be undertaken with 0.9% Sodium Chloride to correct hypovolaemia and avoid hypotension. A patient with persistent hypotension should not be moved to the radiology department or the Critical Care Unit before further evaluation for associated injuries and possible ongoing haemorrhage. Blood samples should be immediately send for U&E, LFTs, CRP, glucose, full blood count, coagulation screen and group & save for urgent analysis.
Indications for an urgent CT scan are:

1. GCS less than 13 at any point since the injury.
2. GCS equal to 13 or 14 at 2 hours after the injury.
3. Suspected open or depressed skull fracture.
4. Any sign of basal skull fracture (haemotympanum, “panda” eyes, cerebrospinal fluid otorrhoea, Battle’s sign).
5. Post-traumatic seizure.
6. Focal neurological deficit.
7. More than one episode of vomiting
8. Amnesia for greater than 30 minutes of events before impact.

A CT should also be immediately requested in patients with any of the following risk factors, provided they have experienced some loss of consciousness or amnesia since the injury:

1. Age greater than or equal to 65 years.
2. Coagulopathy (history of bleeding, clotting disorder, current treatment with Warfarin).
3. Dangerous mechanism of injury (a pedestrian struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or five stairs).

3.2 Critical care management of severe TBI

The focus of critical care management is to limit secondary brain injury. Treatment efforts focus on

1. intracranial pressure management
2. maintenance of cerebral perfusion
3. optimisation of oxygenation and blood pressure
4. management of temperature, glucose, seizures, and other potential secondary brain insults.

A secondary brain injury chart is used for patients with severe TBI to facilitate regular monitoring and goal directed therapy.

3.2.1 Monitoring

1. ECG.
2. Pulse oximetry.
3. End tidal CO_2.
4. Invasive arterial pressure monitoring
5. Urinary catheter: hourly urine output and fluid balance recording.
6. Core temperature.
7. ICP monitoring.
8. Cardiac output monitor in haemodynamically unstable patients
9. Cerebral function monitor for suspected seizures or need to achieve burst suppression.

3.2.2 Oxygenation and Blood pressure

Hypoxia and hypotension are significant factors leading to secondary brain injury.
Oxygenation should be monitored and hypoxia (PaO_2 < 8kPa or Sat O_2 < 90%) should be avoided (Level III). The FiO_2 should be adjusted to achieve a PaO_2 at normal physiological levels, ideally above 10.0 kPa. Patients with persistent hypoxia should receive humidification and physiotherapy and a CXR.
Ventilation should be volume controlled (usually SIMV autoflow) to achieve a $PaCO_2$ of 4.0 - 4.5 kPa. Prophylactic hyperventilation ($PaCO_2$ < 3.5 kPa) is not recommended (Level II). Hyperventilation may be used as a temporary measure to control a critically raised ICP but should be avoided during the first 24 hours following injury. (Level III).
Blood pressure should be monitored and hypotension (systolic blood pressure < 90mmHg) should be avoided. (Level II). The aim of blood pressure management is the maintenance of the cerebral perfusion pressure (CPP). For patients without ICP monitors an ICP of up to 30 mmHg may be assumed. To achieve a CPP of above 60 mmHg the mean arterial blood pressure should therefore be maintained at 90 mmHg. A CPP of < 50 mmHg should be avoided (Level III).
Hypotension should initially be treated with correction of hypovolaemia using 0.9% Sodium Chloride. Albumin and colloids should not be used to resuscitate patients with severe TBI. Severe TBI can cause a systemic inflammatory response syndrome which should be aggressively addressed using adequate vasopressor or inotropic therapy. Aggressive attempts to maintain the CPP above 70 mmHg with fluids and vasopressors should be avoided because of the risk of adult respiratory distress syndrome (Level II).

3.2.3 Anaesthesia, analgesia and sedation

Ventilated patients with severe TBI should be adequately sedated with propofol and fentanyl. High doses of propofol carry the risk of high lipid loads

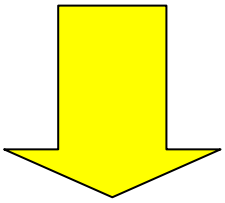
and rarely propofol infusion syndrome, therefore a maximum dose of 4mg/kg/hour is recommended.
Prophylactic high dose barbiturate administration to induce burst suppression is not recommended (Level II) but might be used to control raised ICP refractory to maximum standard surgical and medical treatment.

3.2.4 Hyperosmolar Therapy

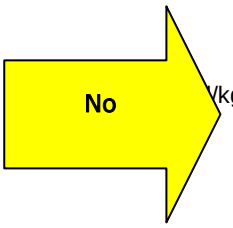
Mannitol and hypertonic saline are hyperosmolar agents both commonly used for the management of raised ICP in severe TBI. A target serum osmolarity should be >300mOsm/kg.
Mannitol is recommended by the European Brain Injury Consortium and the Brain Trauma Foundation (Level II). The dose is 2ml/kg 20% solution, infused over 20 minutes, repeated every 4-6 hours if required (Level II). ICP is reduced within a few minutes and distribution is rapid with a half-life of approximately 10 minutes. A urinary catheter is required due to a profound osmotic diuresis within an hour of administration.
Hypertonic saline is an attractive alternative to mannitol. It produces an increase in circulating blood volume and consequent rise in CPP and shows no diuretic properties. It also modulates the inflammatory response by reducing leucocyte adhesion to the endothelium. A variety of concentrations are available but a reasonable starting dose would be 300ml 2.7% NaCl or 150ml 5% NaCl centrally over an hour targeted to serum osmolarity. Higher doses can safely be used but should be discussed with senior staff Caution should be exercised with hypertonic saline in the context of chronic hyponatraemia due to the risk of central pontine myelinolysis

Hyperosmolar Therapy on Ward 18

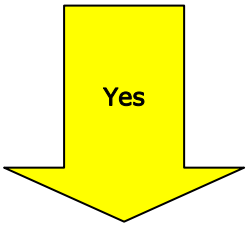
Patient requiring level 2 care for management of raised ICP.
Aim of serum osmolality >300osm/kg.



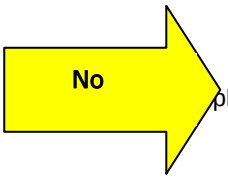
Is the patient hypovolaemic or hyponatraemic



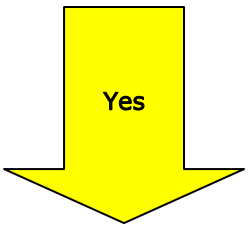
/kg 20% mannitol over 20 mins. Repeat every 4-6h.



Use hypertonic saline.
Does the patient have a central line in situ?



300ml 2.7% NaCl pherally*



150ml 5% NaCl centrally*

*Dosages are a suggested starting point and can be altered according to individual circumstances.
Speak to Duty Consultant if any doubt.

3.2.5 Avoidance of hyperthermia

Fever worsens outcomes after stroke or severe head injury presumably, by aggravating secondary brain injury. Temperature monitoring and interventions to avoid hyperthermia are currently recommended. To achieve this, regular treatment with paracetamol, surface cooling, cooling mattresses or intravascular cooling devices to keep the patients temperature below 37ºC should be used.
The benefit of mild to moderate hypothermia for patients with severe TBI is currently investigated in the Eurotherm study. Until the results are available this remains an intervention that is only used for patients which are enrolled in the study protocol.

3.2.6 Infection prophylaxis

Standard infection control procedures should be observed. The insertion of invasive devices should follow strictly aseptic techniques.
Prophylactic antibiotics are currently not recommended unless there is evidence of infection although prophylactic cefuroxime around the time of intubation has been shown to reduce nosocomial pneumonia in patients with TBI and is described in the latest BTF guidelines.
Intravascular devices or extraventricular drains should not be routinely changed unless there is evidence of a device related infection or malfunction.
The prophylactic use of antibiotics for ventricular catheter placement is not recommended (Level III).
Patients with open depressed cranial fractures and base of skull fractures with CSF leak may require prophylactic antibiotic treatment. Initiation should be discussed with the microbiology and neurosurgical teams.

3.2.7 Venous thromboembolic prophylaxis

Patients with severe TBI are at significant risk of thromboembolic complications. Graduated compression stockings or pneumatic sequential compression devices are recommended unless lower extremity injuries prevent their use (Level III).
The use of low molecular weight heparin needs special consideration in view of a possible concurrency of intracranial haemorrhage, other severe injuries or coagulopathy. A balanced decision on the use of LMWH should be taken in cooperation with the neurosurgical team on admission and again 48 – 72 hours following admission to critical care.
Prophylactic LMWH should be used in combination with mechanical prophylaxis (Level III).

3.2.8 Intracranial pressure monitoring

Intracranial pressure (ICP) cannot be reliably predicted by CT scan alone. ICP should be monitored in patients with

1. severe TBI (GCS ≤ 8 following resuscitation) and
2. abnormal CT
   - haematoma,
   - contusion,

- swelling,
- herniation,
- compressed basal cisterns (Level II)

ICP monitoring is indicated in patients with severe TBI and a normal CT scan if two or more of the following are present at admission:

1. age > 40 years,
2. unilateral or bilateral motor posturing,
3. systolic blood pressure < 90mmHg. (Level III)

Although recent publications suggest that ICP monitoring might not decrease mortality, there is a role of ICP monitoring in guiding targeted interventions to reduce increased ICP. External ventricular drains (EVD) connected to a strain gauge are the most accurate, low cost and reliable method to monitor ICP. Alternatively micro strain gauge or fibreoptic devices measure ICP similar to EVD.
Treatment should be initiated with ICP thresholds above 20 mmHg (Level II). Treatment thresholds, targets and methods should be discussed with the neurosurgical team. The treatment of a raised ICP is discussed in a separate guideline.

3.2.9 Diabetes insipidus

Patients with severe TBI are at risk of developing diabetes insipidus (DI). Regular monitoring of plasma and urine osmolarity is recommended and should be documented on the secondary brain injury chart. Patients with high Na, a urine output of > 200ml/hr for two consecutive hours and a plasma osmolarity > 320 mOsm and a low urine osmolarity may require DDAVP. Electrolyte disorders are discussed in a separate guideline.

3.2.10 Nutrition

Patients with severe TBI should receive enteral nutrition as early as possible unless contraindicated or if early extubation is planned. Enteral feeding should be initiated within the first 24 hours via a gastric feeding tube to meet full caloric requirements. Care must be taken during the insertion of feeding tubes in view of possible associated base of skull fractures. A gastric feeding tube might be placed orally in the case of a displaced base of skull fracture. In patients with suspected malnutrition or vitamin deficiency, Pabrinex® should be given twice daily for 48-72 hours followed by vitamin B complex and thiamine. These patients should be carefully monitored for the development of re-feeding syndrome.

3.2.11 Glycaemic control

Hyperglycemia is associated with worsened outcome in a variety of neurologic conditions including severe TBI. This has been presumed to be at least in part related to the aggravation of secondary brain injury. The blood glucose levels of patients with severe TBI should be monitored closely, at least four-hourly. Hypoglycaemia can also have deleterious effects on critically ill patients, especially on patients with severe TBI. An insulin infusion should aim to control the blood glucose level to 4.4 – 10 mmol/l.

3.2.12 Anti-seizure prophylaxis

Patients with severe TBI are at risk of posttraumatic seizures. Seizures can increase cerebral blood flow and thereby have the potential to increase ICP. Another concern is that seizures place an increased metabolic demand on damaged brain tissue and may aggravate secondary brain injury. However posttraumatic seizures are not associated with worse outcome. Patients with severe TBI and with the following risk factors may be considered for prophylactic anticonvulsants for 1 week after discussion with the neurosurgical team:

1. GCS < 10
2. Cortical contusion
3. Depressed cranial fracture
4. Subdural haematoma
5. Penetrating brain injury
6. Seizures within 24 hours of injury

Any seizures occurring in patients with severe TBI should be treated promptly with lorazepam. Phenytoin is the drug of choice for anticonvulsant therapy. It is given as a loading dose of 18mg/kg (at ≤ 50 mg/min) followed by a maintenance dose of 3–4 mg/kg per day. If Phenytoin is given enterally, the feed should be stopped for 2 hrs before and after the dose to facilitate absorption.
Prophylactic use of antiepileptic medication is not recommended for the prevention of late posttraumatic seizures (Level II).
Patients with pre-existing epilepsy should have their usual medications continued unless there is a specific reason to change.

3.2.13 Steroids

The use of steroids for patients with severe TBI is not recommended to improve outcome or reducing ICP. Steroids for patients with moderate or severe TBI are associated with increased mortality (Level I).

3.2.14 Haemostatic therapy

Patients with severe TBI are at risk of developing a coagulopathy. This is associated with an increased risk of further haemorrhage and poor neurological outcome. A coagulation screen should be sent for urgent analysis at the patient's arrival in the emergency department. Any coagulopathy should be corrected immediately.
Patients on Warfarin may be given prothrombin complex concentrate (Beriplex®) and vitamin K following an urgent consultation with the on call haematologist.
Patients with a thrombocytopenia should be given platelets to maintain a count > 80,000. The use of platelet transfusions in patients with severe TBI who are on antiplatelet medications is unknown. The use of tranexamic acid is established for patients with trauma. Recent publications could not confirm

harm or benefit of tranexamic acid in patients with severe TBI. The results of the ongoing CRASH-3 study are currently awaited.

3.2.15 Surgery

Patients with severe TBI may benefit from surgery and so the early involvement of the neurosurgical team is paramount. The critical care team can play an important role in the facilitation of the logistics for theatre including anaesthetic management and theatre availability.
Guidelines have been published by the Congress of Neurological Surgeons and the American Association of Neurological Surgeon. These are summarized in the appendix

4 Training, Implementation, Resource Implications

All medical and nursing staff should be aware of this guidance. The guideline should be included in the induction pack for rotating junior doctors.

5 Monitoring Section

The recommendations given in this guideline should be subject to a periodic audit process.

6 Evidence Review and Evaluation

Latest international recommendations and relevant review articles were reviewed. When available and appropriate the level of recommendation is stated. Levels of recommendation are level I, II and III, derived from class I, II and III evidence, respectively.
Level I recommendations are based on the strongest evidence of effectiveness, and represent principles of patient management that reflect a high degree of clinical certainty.
Level II recommendations reflect a moderate degree of clinical certainty.
For level III recommendations, the degree of clinical certainty is not established.

7 References

Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons. Guidelines for the management of severe traumatic brain injury. J Neurotrauma. 2007;24 Suppl 1:S1-106.
Rosenfeld JV, Early management of severe traumatic brain injury.2012 Lancet;380(9847):1088-98
Hemphill J C et al, Management of acute severe traumatic brain injury, 2013 UpToDate®
Bullock MR et al; Surgical Management of Traumatic Brain Injury.Neurosurgery. 2006 Mar;58,S2:1-60
Haddad SH, Arabi YM.Critical care management of severe traumatic brain injury in adults. Scand J Trauma Resusc Emerg Med. 2012 Feb 3;20:12.

Appendix

Surgical Management of acute extradural haematomas

Bullock MR et al; Surgical Management of Traumatic Brain Injury.Neurosurgery. 2006 Mar;58,S2:1-60

Indication for surgery

- An extradural haematoma > 30 cm^3 should be surgically evacuated regardless of the patient's GCS score.
- An extradural haematoma < 30 cm^3 and with < 15 mm thickness and with < 5 mm midline shift in patients with a GCS > 8 without focal deficit can be managed non-operatively with CT scanning and close neurological observation in a neurosurgical centre.

Timing

- It is strongly recommended that patients with an acute extradural haematoma in coma (GCS < 9) with anisocoria undergo surgical evacuation as soon as possible.

Methods

- There are insufficient data to support one surgical treatment method. However, craniotomy provides a more complete evacuation of the haematoma.

Surgical Management of acute subdural haematomas

Indication for surgery

- An acute subdural haematoma with a thickness > 10 mm or a midline shift > 5 mm on CT scan should be surgically evacuated, regardless of the patient's GSC score.
- All patients with acute subdural haematoma in coma (GCS < 9) should undergo ICP monitoring.
- A comatose patients (GCS < 9) with a subdural haematoma < 10 mm thick and a midline shift < 5 mm should undergo surgical evacuation of the lesion if the GCS decreases between the time of injury and hospital admission by 2 or more points on the GCS and/or the patient presents with asymmetric or fixed and dilated pupils and/or the ICP exceeds 20 mmHg.

Timing

- In patients with acute subdural haematoma and indication for surgery, surgical evacuation should be performed as soon as possible.

Methods

- If surgical evacuation of an acute subdural haematoma in a comatose patient (GCS < 9) is indicated, it should be performed using a craniotomy with or without bone flap removal and duraplasty.

Surgical Management of traumatic parenchymal lesions

Indication for surgery

- Patients with parenchymal mass lesions and signs of progressive neurological deterioration referable to the lesion, medically refractory

intracranial hypertension, or signs of mass effect on CT scan should be treated operatively.

- Patients with GCS 6 - 8 with frontal or temporal contusions > 20 cm^3 in volume with midline shift of at least 5 mm and/ or cisternal compression on CT scan, and patients with any lesion > 50 cm^3 in volume should be treated operatively.
- Patients with parenchymal mass lesions who do not show evidence for neurological compromise, have controlled ICP, and no significant signs of mass effect on CT may be managed non-operatively with intensive care monitoring.

Timing and Methods

- Craniotomy with evacuation of mass lesion is recommended for those patients with focal lesions and the surgical indications listed above, under Indications.
- Bifrontal decompressive craniectomy within 48 hours of injury is a treatment option for patients with diffuse, medically refractory posttraumatic cerebral oedema and resultant intracranial hypertension.
- Decompressive procedures, including subtemporal decompression, temporal lobectomy, hemispheric decompressive craniectomy, are treatment options for patients with refractory intracranial hypertension and diffuse parenchymal injury with clinical and radiographic evidence for impending transtentorial herniation.

Surgical Management of posterior fossa mass lesions

Indication for surgery

- Patients with mass effect on CT scan or with neurological dysfunction or deterioration referable to the lesion should undergo operative intervention. Mass effect on CT scan is defined as distortion, dislocation, or obliteration of the fourth ventricle, compression or loss of visualisation of the basal cisterns, or the presence of obstructive hydrocephalus.
- Patients with lesions and no significant mass effect on CT scan and without signs of neurological dysfunction may be managed by close observation and serial imaging.

Timing

- In patients with indications for surgical intervention, evacuation should be performed as soon as possible because these patients can deteriorate rapidly, thus, worsening their prognosis.

Methods

- Suboccipital craniectomy is the predominant method reported for evacuation of fossa mass lesions, and is therefore recommended.

Surgical Management of depressed cranial fracture

Indication for surgery

- Patients with open (compound) cranial fractures depressed greater than the thickness of the cranium should undergo operative intervention to prevent infection.

- Patients with open (compound) depressed cranial fractures may be treated non-operatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial haematoma, depression > 1 cm, frontal sinus involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination.
- Non-operative of management of closed (simple) depressed cranial fractures is a treatment option.

Timing

- Early operation is recommended to reduce the incidence of infections.

Methods

- Elevation and debridement is recommended as the surgical method of choice.
- Primary bone fragment replacement is a surgical option in the absence of wound infection at the time of surgery.
- All management strategies for open (compound) depressed fractures should include antibiotics.