NETS
Part of the Sydney Children's Hospitals Network

TRAUMATIC BRAIN INJURY

PRACTICE GUIDELINE ©

DOCUMENT SUMMARY/KEY POINTS

- Focus on preserving viable brain function by avoiding secondary brain injury after severe traumatic brain injury (TBI) including preventing hypotension, hypo/hypercapnia, hypoxia, hypo/hyperglycaemia, hyperthermia and adequate control of raised intracranial pressure.
- Anticipating and appropriately responding to acute deterioration by controlling intracranial hypertension, optimising ventilation and ensuring hemodynamic stability.
- In particular for neurosurgical emergencies, consider early activation of the mobile sub-specialist (neurosurgical) pathway.

CHANGE SUMMARY

- References updated

READ ACKNOWLEDGEMENT

- All NETS clinical staff are to read and acknowledge they understand the contents of this guideline.

Approved by:	SCHN Policy Procedure and Guideline Committee	NETS Executive
Date Effective:	1st August 2017	**Review Period**: 3 years
Team Leader:	Staff Specialist	**Area/Dept**: NETS

Rationale

- To avoid secondary brain injury after severe traumatic brain injury (TBI) by avoiding hypotension, hypoxia, hypo/hypercapnia, hypo/hyperglycaemia, hyperthermia, exacerbation of cerebral oedema.
- Respond to acute deterioration by managing intracranial hypertension, ventilation and haemodynamic instability.
- In neurosurgical emergencies, use mobile neurosurgical capacities to surgically relieve intracranial pressure at the referring hospital prior to patient transfer.

Equipment for Specific Requirements

- Paediatric bridge with paediatric packs
- Vacuum mattress
- Neurosurgical kit (if neurosurgeon tasked)

Evaluation procedure

Specific points on history:

- Mechanism of injury may point to severity and raise suspicion of other injuries including non-accidental injury (NAI).
- Timing of injury: severe TBI will generally present within 6 hours of incident. Delay in seeking medical help may raise suspicion of NAI.
- Symptoms suggestive of head injury: loss of consciousness, vomiting, headache, seizures, combative/agitated behaviour etc. Additional history of concurrent acute drug intoxication should be elicited and may be the cause of altered mental status or hemodynamic instability.
- Also look for symptoms suggestive of other injuries e.g. abdominal pain, chest pain, haematuria etc.
- Past medical history: focus on previous head injuries, history of coagulopathy, seizures or developmental delay, drug allergies, immunisation history.

First look/primary assessment: ABCDE

Suspect C-spine injury in all patients with head injury, especially when unconscious, uncooperative and/or suspected injury on imaging studies. If in any doubt, ensure in-line mobilisation or hard collar for all procedures, manoeuvres and movement of patient.

- **A:** Assess airway for patency and patient's ability to maintain airway (avoid chin lift, use jaw thrust if required). Look for blood, vomitus, secretions, foreign body (teeth, fractured bone) obstructing the airway.

- **B:** Assess breathing: air entry, chest movement, respiratory rate, oxygen saturation. Chest wall tenderness, bruising, lacerations, deformity may herald underlying thoracic injury. If already intubated, determine position and patency of ETT; adjust as required. Ensure end-tidal capnography ($ETCO_2$) is routinely used for all intubated patients.
- **C:** Assess circulation: skin colour and temperature, heart rate, peripheral and central pulses, capillary refill, blood pressure.
- **D:** assess level of consciousness: determine GCS, pupil size, form and reaction to light, observe for signs of seizures/abnormal movements. Look for focal neurological signs.
- **E:** measure temperature, assess for marks/signs of injury (bruises, wounds, swelling etc). Measure BSL if it has not been done in the previous 2 hours. Log roll to be performed to rule out spinal injuries.

Airway/ventilation

Primary Aim of treatment:

Prevent hypoxia and hypo/hypercarbia to prevent secondary brain injury

Monitoring parameters (set targets) :

- Maintain saturations > 95%, keep PaO_2 > 80 mmHg.
- Target end-tidal capnography ($ETCO_2$) between 35-40mmHg. Avoid prophylactic hyperventilation below $PaCO_2$ 35mmHg. Correlate $ETCO_2$ with $PaCO_2$ at initiation of ventilation and when changes are made.

Intubation:

- Intubate if patient unable to maintain airway, if saturation < 95% with maximum oxygen therapy, abnormal breathing pattern and/or GCS < 9. A child with fluctuating GCS who is in need of brain imaging may need intubation for transfer.
- Intubation needs to be done cautiously to prevent large swings in blood pressure, avoiding both hypotension and hypertension. The patient may need volume loading and vasopressors peri-intubation.
- RSI intubation should be performed by the most experienced person on site, with a second person immobilising the patient's cervical spine. Avoid nasal intubation and insertion of nasogastric tubes in patients with (suspected or proven) basal skull injury and/or coagulopathy. Ketamine should be considered as an induction agent if haemodynamically unstable.
- Previous literature suggested that Ketamine was associated with elevations in intracranial pressure (ICP), however recent evidence has refuted this theory. Fentanyl may be used as a pre-induction agent to prevent sudden spikes in ICP related to the intubation process. Use of endotracheal (ET) / intravenous (IV) Lignocaine to prevent procedural spikes in ICP remains controversial.

Ventilation strategies:

- Use PEEP 5 cmH_2O unless increase is required for concomitant pulmonary pathologies.

- Adjust FiO_2 to maintain saturations above 95%.
- Perform endotracheal suction only if clinically indicated. Ensure adequate pre-oxygenation while avoiding hyperventilation (drop of $ETCO_2$ not more than 5 mmHg from baseline level). Give additional boluses of analgesia/sedation prior to suctioning to prevent large spikes in ICP.
- Short term hyperventilation may be considered to treat persistent elevations in ICP. Longer term hyperventilation (to keep $PaCO_2$ 25-30 mmHg) may be considered in refractory persistent intracranial hypertension but should only be undertaken in consultation with the NETS consultant and receiving intensivist.

Circulation

Primary aim of therapy:

Avoid hypotension to maintain adequate cerebral perfusion.

Fluid resuscitation:

- Maintain mean arterial pressures (MAP) within age-appropriate limits and attempt to achieve euvolemia.
- If signs of shock are present (tachycardia, poor perfusion, hypotension) give boluses of crystalloid in aliquots of 20 mL/kg.
- Consider use of hyperosmolar solutions (Mannitol or 3% saline) for volume expansion in a child with raised intracranial pressure. However there are no clear recommendations for its use as resuscitative fluid in children.
- Massive transfusion protocol may need to be activated and packed red cell transfusion may need to be administered along with crystalloid resuscitative fluids in suspected haemorrhagic shock in trauma.

Management of hypotension:

Hypotension is the single most important factor causing secondary brain injury and should be aggressively treated to maintain cerebral perfusion

- Maintain age–appropriate systolic BP greater than or equal to the 75^{th} percentile to achieve better outcomes.
- If hypotension persists despite adequate volume resuscitation, suspect other injuries (especially trauma of abdomen, chest, pelvis, long bone fractures, spinal shock).
- Consider early inotropic/vasopressor support to maintain MAP and cerebral perfusion pressure (CPP) (CPP= MAP- ICP) in consultation with NETS consultant and receiving intensivist.

Monitoring parameters (set targets):

- Continuous cardiac monitoring is mandatory.
- Invasive arterial monitoring may be necessary to adequately titrate fluid and inotropes and to calculate CPP where an external ventricular drain (EVD) is insitu.

- Strict fluid balance: consider insertion of IDC to monitor fluid balance in all intubated children, patients needing large amounts of fluid resuscitation and where hyperosmolar therapy is used.

Guide for age-related mean arterial pressure[4]

Age	Mean BP mmHg
Term	40-60
3 months	45-75
6 months	50-90
1 year	50-90
3 years	50-90
7 years	60-90
10 years	60-90
12 years	65-95
14 years	65-95

Ongoing fluid management:

- Following fluid resuscitation, administer maintenance fluids (0.9% sodium chloride with 5% glucose) at 2/3 or 1/2 maintenance, if patient is well perfused.
- Ensure euglycemia at all times
- Maintain serum sodium over 140mmol/L.

Disability (Neurology)

Management of elevated intracranial pressure

Factors precipitating spikes of intracranial pressure are: hypoxia, hypercapnia, hypotension, valsalva manoeuvre, cough/pain/agitation, hyperthermia, position (flexed neck impairing venous return), inappropriately applied c-spine collar, seizures, progression of neurological process (cerebral haemorrhage or oedema).

If monitoring ICP and MAP, aim for ICP < 20 while maintaining appropriate age-related systemic blood pressure and therefore CPP.

An **ICP of 20 mmHg** for more than 5 minutes is generally accepted as a treatment threshold for infants and children of all age groups

A **CPP of 40 mmHg** is thought to be the lower limit of functioning cerebral flow autoregulation and below this, increases the risks of adverse outcome

Target CPP for age (CPP=MAP-ICP)[7]

Age	Desirable minimum CPP (mmHg)
< 1 year	> 45
1-10 years	> 55
> 10 years	> 65

- Manage ventilation and circulation as described above
- Ensure appropriate analgesia and sedation with narcotic +/- benzodiazepine infusions, and boluses prior to interventions/procedures
- Neuromuscular relaxation can be given to avoid coughing/straining and to facilitate control of ventilation. The caveat is the potential masking of epileptic seizures – watch for tachycardia, hypertension and pupillary changes. Prophylactic anti-convulsant medication is indicated in TBI
- Avoid and treat hyperthermia (exposure, paracetamol, cooling blankets). Keep in mind that vacuum mattress can overheat patients quickly
- Nurse with head in midline and head elevation (30 degrees). Exert caution in suspected spinal injury
- Although difficult to achieve on retrieval, be mindful of clustering nursing activities and medical procedures
- Reassess neurological signs including pupillary response frequently

Trouble shooting ICP spikes/CPP dips:

- **Check patient:**
  - SpO_2 and $ETCO_2$ (resolve ventilation issues), heart rate and blood pressure (treat hypovolaemia/hypotension)
  - New neurological signs (unequal or sluggish pupils, fall in level of consciousness, posturing, Cushing reflex [hypertension, bradycardia, irregular respirations]) (treat seizures, consider progression of neurological process)
  - Patient's position: neck flexed or rotated? (reposition as indicated)
  - Is the collar ill-fitting or too tight? (adjust as required)
  - If EVD dressing present, is it too tight? (adjust as required)
  - Adequate analgesia and sedation? If paralysed, is patient waking up or muscle relaxant wearing off? (bolus and adjust infusion rate)
- **Check equipment:**
  - Check that the ventilator is working correctly
  - Check that all pressure monitoring is properly aligned and zeroed
  - Check EVD (if present) is functioning properly
  - If invasive monitoring in situ, check arterial line site for leaks

Steps for intracranial hypertension refractory to above measures:

- Give boluses of analgesia and sedation, increase infusion rates
- If not already muscle relaxed, give intermittent muscle relaxant and, if still insufficient, consider commencing infusion. Note that muscle relaxation will obscure signs of seizures
- CSF drainage: if EVD in situ, open (usually at a height of ~15-20cmH_2O) for 5 min until ICP below target level
- Hyperosmolar therapy: 1st choice hypertonic 3% sodium chloride (bolus 5mL/kg, can be increased to 5-10 mL/kg in acute deterioration or infusion of 0.1-1 mL/kg/hour titrated to keep ICP <20 mmHg and serum Na 140-160 mmol/L). 2nd choice mannitol (initial bolus 0.5 g/kg, then 0.25 g/kg boluses).
- Contact NETS consultant and receiving intensivist urgently if ICP resistant to above measures. You could discuss short term hyperventilation with bagging to produce hypocapnia in the case of a critical fall in CPP. Barbiturate coma with thiopentone might be an ultimate measure however there is a very high likelihood of cardiovascular instability
- Consideration should be given to mild passive hypothermia for refractory intracranial hypertension
- Expedite retrieval for urgent neurosurgical intervention

Sedation and neuromuscular blockade

- Adequate sedation is needed to prevent spikes in ICP that occur with movement
- Commonly used infusions are morphine and midazolam titrated to achieve the desirable effect (refer to the NETS drug calculator for the dose and formulation)
- Propofol infusion may be considered and should be used with caution, after consultation with the receiving intensivist. Propofol potentiates haemodynamic instability and there are reports of propofol infusion syndrome after prolonged use
- Suxamethonium and rocuronium remain the commonly used neuromuscular blocking agents for intubating children with TBI and maintaining neuromuscular paralysis as needed

Seizure control

- Midazolam infusion used for sedation in ventilated children may also suffice as an anti-convulsant medication
- Seizures should be controlled acutely with IV benzodiazepines
- In post-traumatic status epilepticus consider using Phenytoin or Phenobarbitone or Levetiracetam
- Discuss prophylactic anticonvulsant (usually Phenytoin/Levetiracetam) and antibiotics with receiving neurosurgeon/Intensivist.

Environmental control:

Targeted temperature management

- Aggressively avoid hyperthermia as it increases cerebral metabolic requirements and thus increases CPP and ICP. Keep core body temperature below 37 degrees.
- For refractory cases of intracranial hypertension consider inducing hypothermia after consultation with the NETS consultant and receiving intensivist.

Mobile neurosurgical capability

In time-critical neurosurgical emergencies a paediatric neurosurgeon may be tasked to travel to the referring hospital. Please refer to the mobile sub-speciality pathway [8].

Care of patient during and after neurosurgical intervention in the referring hospital

- During neurosurgical intervention in the operating theatre at the referring hospital, the clinical care of the patient remains the responsibility of the local anaesthetist supported by the NETS team and the treating neurosurgeon.
- **The NETS team shall assist upon request, but are not responsible for anaesthesia in theatres.** The team may use the waiting time to set up and prepare equipment, drugs and fluids in order to facilitate smooth transfer to the final destination.

Important points to consider for patients post-neurosurgical intervention

- Follow procedures for airway, breathing, circulation and control of intracranial hypertension as outlined above.
- Set-up and operation of EVD and ICP monitoring is outlined in the EVD policy. Secure drain safely at exit sites and remember to turn drain off during movement/transfer of patient (e.g. from bed to stretcher, during log roll)
- Clarify target ICP and CPP with neurosurgeon and titrate management accordingly.
- Ensure that a contingency plan for ventilation, sedation, and circulation (after setting target parameters) is formulated in conjunction with the receiving intensivist prior to transfer.
- After craniectomy: clearly mark the bandage covering surgical area with “NO BONE – do not apply pressure” and avoid any pressure/compression of that particular area.
- Make sure the excised skull bone is stored appropriately (discuss with neurosurgeon for details of storage procedure) and is travelling with the patient to destination hospital.
- Consider transfusion of packed red cells for post-traumatic haemodynamic instability due to hypovolemia and blood loss or large intra-operative blood loss, and FFP/platelets for coagulopathy, especially if a long distance retrieval.

Documentation

- In addition to routine observations (including temperature), regular neurological observations should be recorded with special focus on GCS, pupillary size and reaction to light, any focal neurology appreciated and seizure activity if present.
- If ICP monitoring device insitu: record ICP & CPP (CPP = MAP-ICP). If CSF drained: record colour and amount.
- Record fluid input and output especially when hyperosmolar agents are used to manage intracranial hypertension and if there is a high risk of diabetes insipidus.
- Neurosurgeon to briefly document procedural details in NETS notes.
- Regularly record the need for sedation/neuromuscular blocking agents including boluses and background infusion adjustments.

Educational Notes

- **Intracranial hypertension, systemic arterial hypotension and hyperthermia** are the key pathophysiological processes resulting in **secondary brain injury**. It results in decreased perfusion of surviving neuronal tissue by decreased oxygen and metabolite delivery and/or focal or global cerebral ischemia. While the primary injury cannot be undone, management during retrieval aims to minimise secondary injuries and to respond to acute deterioration.
- **Ventilation:** Hyperventilation reduces ICP by producing hypocapnia-related cerebral vasoconstriction and reduction in cerebral blood flow in presumed hyperaemia in the initial phase after brain tissue injury. However, newer evidence points towards decreased blood flow in the damaged area in the first day after head injury, raising concerns that hypocapnoeic vasoconstriction significantly further decreases cerebral oxygenation with potential for ischaemia and, in the long term, worse neurological outcome. Therefore, the target should be a **normal $PaCO_2$ of 35-40 mmHg**, with hyperventilation (to more than 5mmHg below baseline) used only as a temporary emergency measure for uncontrolled intracranial hypertension to prevent acute herniation.
- **Hyperosmolar therapy: Mannitol** reduces ICP by reducing blood viscosity (resulting in an immediate reflex vasoconstriction) and via osmotic effect. Adverse effects with extensive use include the potential for accumulation in injured brain tissue (reversing the initial osmotic effect) and a risk for acute kidney injury. Despite widespread clinical use and experience, there are no controlled clinical trials for mannitol in children, and a Cochrane review did not reach conclusion regarding efficacy in adults. **Hypertonic (3%) sodium chloride** acts by increasing blood osmolality and hence draining fluid from interstitial and intracellular spaces. Apart from rebound in ICP, a rapid rise in serum sodium can lead to central pontine myelinolysis, diuresis and natriuresis as well as hyperchloraemic acidosis. It may mask the onset of diabetes insipidus. However, as

opposed to mannitol, there is better evidence for the efficacy of hypertonic saline in paediatric patients. Additionally, administration can be controlled by measuring serum sodium and osmolality. On these grounds, preference should be given to hypertonic saline over mannitol. However, many non-tertiary hospitals only stock mannitol.

- **Seizure management:** Seizures can increase metabolic demands thus increasing ICP and CPP and hence should be managed appropriately. The risk of post-traumatic seizures increases with the severity of the brain injury. Prophylactic phenytoin may be indicated in patients with abnormal neurology and abnormal imaging results especially if they are concurrently paralysed with neuromuscular blocking agents. Despite limited paediatric evidence, phenytoin appears to be superior to Phenobarbitone or valproate in preventing early post-traumatic seizures. However it does not seem to prevent late post-traumatic epilepsy nor does it have any beneficial effect on long term neurological outcomes. Levetiracetam may be as efficient as phenytoin at preventing early post-traumatic seizures, however more studies are required[6].
- To make up 3% sodium chloride: Mix 20% sodium chloride with 0.9% sodium chloride in the ratio 1:8 (e.g. 10mL 20% NaCl and 80mL 0.9% NaCl = 90mL 3% NaCl)
- The use of corticosteroids is no longer recommended, as it has not proven effective in improving outcome in paediatric or adult studies.

References

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