Moderate-to-Severe Traumatic Brain Injury (TBI) Management: ADHB Guideline

Authors: **Dr Chen Chen**
Anaesthetic Fellow

Dr Nadia Forbes
Specialist Anaesthetist

Dr Phoebe Matthews
Consultant Neurosurgeon

February 2022

Background

Trauma to the head is one of the most common presentations to ED. Approximately 75% of TBI patients have mild brain injury, 15% moderate, and 10% severe[1]. In New Zealand approximately 1750 people suffer moderate to severe traumatic brain injury (TBI) per year and 500 people will require specialised residential rehabilitation. TBI is the leading cause of disability in under 40 age group, and thus a significant health burden. In 2020 at ADHB, there were 93 admissions of moderate-severe TBI patients to Department of Critical Care Medicine (DCCM). These patients had a 30% mortality.

Guideline Principles

- To provide guidance on the management of patients with moderate/severe TBI at ADHB
- To provide physiological management principles to help prevent secondary brain injury
- To provide timely multidisciplinary assessment of moderate/severe TBI patients
- To ensure urgent neurosurgical intervention where appropriate
- To ensure appropriate disposition of the patient

Definition of Head Injury Severity

Head injury severity is graded using the Glasgow Coma Scale, assessed after the initial resuscitation.

- Mild: GCS 14 – 15
- Moderate: GCS 9 – 13
- Severe: GCS < 9

Eye opening

Rating	Score
Spontaneous	4
To sound	3
To pressure	2
None	1
Non testable	NT

Verbal response

Rating	Score
Orientated	5
Confused	4
Words	3
Sounds	2
None	1
Non testable	NT

Best motor response

Rating	Score
Obeys commands	6
Localising	5
Normal flexion	4
Abnormal flexion	3
Extension	2
None	1
Non testable	NT

Institute of Neurological Sciences NHS Greater Glasgow and Clyde

Primary Injury occurs at the moment of trauma and reflects the mechanical events in the brain at that time. It is not preventable.

Secondary injury occurs minutes, hours, days or even weeks after the initial injury and damage can be modified or lessened by appropriate management.

Timing of Surgery

- **TIME IS BRAIN** – a rapid expanding mass lesion will lead to increased ICP, brain ischaemia and cell death.
- Currently the Brain Trauma Foundation Guidelines state that both extradural and subdural haemorrhage should be operated on "as soon as possible".
    - Acute extradural haemorrhage – A neurosurgical emergency. Mortality rate of 17% and 67% good neurological outcome if operated on within 2 hours, but the mortality increases to 65% with only 13% good neurological outcome if surgery delayed > 2 hours.
    - Acute subdural haemorrhage – earlier evacuation is associated with a lower mortality and a higher chance of good neurological outcome. 73% of patients achieved good recovery when operated on within 2 hours of trauma, compared with only 5% if the operation is delayed > 6 hours.

Section 1:

Initial management of TBI in ED / Resus

TBI in the setting of multi-trauma

See: Multi-trauma with Suspected Traumatic Brain Injury (TBI) - Guideline No.1

All patients arriving at ADHB with moderate (GCS 9 – 13) or severe (GCS 3 – 8) TBI should be assessed by trauma team.

The on-call neurosurgical registrar should be contacted (021 782 472) for prompt discussion to see if neurosurgical intervention is required.

Initial management of trauma should be addressed after appropriate handover from the pre-hospital team e.g. AT MIST (Allergies, Timing, Mechanism, Injuries, Signs, and Treatment).

EMST principles should be followed with primary survey addressing life-threatening injuries.

In patients that are haemodynamically unstable requiring urgent intervention e.g. trauma laparotomy or angiography, investigation of a suspected brain injury (CT) is usually deferred until life-threatening issues have been addressed.

Hypotensive resuscitation should **NOT** be utilised for patients with isolated CNS injury because of associated adverse outcomes in this population (Target MAP 80 – 90mmHg).

Prevention of secondary brain injury is best achieved by avoiding hypotension and hypoxia. A single systolic under 90mmHg is associated with 150% increase in mortality.

BP targets:

- SBP > 100mmHg (50 – 69 yrs)
- SBP > 110mmHg (18 – 49 yrs and > 70 yrs)

For damage control patients with suspected/known TBI – Target SBP > 110mmHg until definitive surgical control of bleeding, as per Damage Control Resuscitation guidelines.

Mannitol is contraindicated in shocked hypotensive patients.

Although hypertonic saline does NOT improve mortality in haemorrhagic shock it should be considered patients with TBI and evidence/suspicion of raised Intracranial Pressure (ICP).

Consider tranexamic acid 1g STAT, if within 3 hours of injury.

Any patient taking anticoagulant (Warfarin or DOAC) is at high risk of developing significant intracranial haemorrhage even from a minor head injury. Antiplatelet therapy also increases the risk, but to a lesser degree. Consider urgent reversal.

Primary Survey

See: Management of Traumatic Brain Injury – Guideline No. 2

Airway and C-spine protection

- Attempt to gather a response from the patient noting:
  - GCS /AVPU
  - Pupils (> 1mm difference is abnormal)
  - Focal neurology
- Open the airway and apply oxygen.
- Assist ventilation as required.
- Patients with GCS ≤ 8, inability to protect airway, inability to maintain SpO_2 > 90% despite oxygen, persistent combativeness or signs of cerebral herniation will require early intubation.
- Manual in-line stabilisation for C-spine protection.
- Modified rapid sequence induction with tight control of haemodynamic stability and oxygenation.
  - Ensure adequate pre-oxygenation and consider bag-mask ventilation while waiting for onset of neuromuscular blocker to avoid hypoxia (SpO_2 > 94%, PaO_2 > 60mmHg).
  - Maintaining haemodynamic stability – ideally MAP 80 – 90mmHg to ensure adequate CPP but systolic under 160mmHg, to avoid exacerbating bleeding and cerebral swelling.
  - Care with induction doses of anaesthetic drugs (consider titrated rather than fixed bolus in classic RSI) to avoid hypotension (any systolic BP < 90mmHg associated with worse outcome) as cerebral auto-regulation is disrupted.
  - Aim to avoid significant sympathetic response during laryngoscopy e.g. fentanyl 2 – 3mcg/kg or alfentanil 1mg.
  - Consider having vasopressor infusion running e.g. metaraminol 3 – 5mg/hr, with judicious vasopressor bolus 0.25mg if required to maintain haemodynamic stability.
- After intubation, keep sedated and paralysed using propofol and neuromuscular blockade to avoid coughing and straining.
- Avoid using long-acting sedatives (e.g. benzodiazepines) to allow early assessment of neurology.
- Use tape to secure endotracheal tube, ensure loose neck ties to avoid obstruction to cerebral venous return.
- Consider 30° head up.

Breathing

- Utilise $ETCO_2$ monitoring and maintain ventilation targets:
  - Aim for $PaCO_2$ 4.5 – 5.0kPa (35 – 40mmHg). Hypoventilation will raise ICP while hyperventilation may cause cerebral vasoconstriction and ischaemia.
  - Maintain SpO_2 > 94%, target PaO_2 > 13 kPa (100mmHg). Consider using lower FiO_2 to maintain oxygenation targets as hyperoxia may possibly be detrimental in TBI settings due to free oxygen radicals.
- PEEP 5 – 10 mmHg, avoid excessive PEEP > 15cmH_2O as this may reduce cerebral venous return.

Primary Survey - *Continued*

Circulation

- Ensure availability of large bore IV access.
- Consider Q1min NIBP until IABP monitoring available.
- Ensure adequate fluid resuscitation and consider early use of blood products if significant haemorrhage.
- MAP target 80 – 90mmHg (i.e. CPP 60 – 70mmHg) unless damage control patient. Ensure arterial line transducer is zeroed and placed at the level of external auditory canal.
- Use isotonic crystalloids, blood products and vasopressors as appropriate.
- Avoid albumin (SAFE study showed small increase in mortality).
- In the presence of intracerebral bleed and absence of brain herniation, systolic BP target should ideally be < 160mmHg as higher may increase bleeding and oedema.
- Approximately one third of patients with severe TBI demonstrate coagulopathy (release of tissue factor and phospholipids). This is associated with increased risk of haemorrhage enlargement, poor outcome and death.
- TXA 1g over 10 mins and then infusion 1g over 8 hours should be considered in patients with mild to moderate TBI (if within 3 hours of injury: CRASH 3).
- Consider urgent reversal of warfarin and DOAC as per guidelines.

Disability

- Recheck GCS, pupils, document focal neurology.
- Drowsy, confused or agitated TBI patients should NOT be sedated initially as it makes assessment difficult unless they are a management risk.
- Do not assume that altered consciousness is due only to intoxication. CT imaging is recommended in all intoxicated patients with signs of head injury.
- Check BSL (Aim glucose 4 – 10mmol/L).
- Antiepileptics initiated with 1g levetiracetam (1g in 100ml 0.9% NaCl over 15 mins) – this is usually continued prophylactically for 7 days at 500mg BD dosing 12hrs from initial 1g load unless evidence on-going seizure. Seizures increase ICP and place a large metabolic load on damaged brain tissue which may aggravate secondary injury.
- Assume uncal herniation in any unresponsive (comatose) patient with any of the following:
    - Bilateral / unilateral unresponsive pupil/s
    - Those exhibiting Cushing triad (hypertension, bradycardia and respiratory depression)

- URGENT CT and discussion with neurosurgical team
- See Emergent Management of Increased ICP – Guideline No.4
- Immediate TEMPORISING management includes:
    - Increased MAP target 100 – 110mmHg
    - 45° head up
    - Ensure no impediment to venous drainage (ETT ties, cervical collar).
    - Hyperventilate to ETCO2 to 4kPa/30mmHg.
    - Minimise PEEP (e.g. 0 – 5cmH2O).
    - Give osmotherapy
        - Primary agent: Hypertonic Saline 23.4% to target [Na+] 150 – 155mmol/L, may require different dosage depending on starting [Na+] – up to 50 – 100mL may be required to achieve this. Give over 10 – 20 minutes as can cause hypotension if given as a bolus push.
        - If using 3% saline, 3 – 5mL/kg over 30 minutes
        - Secondary agent: Mannitol 20% 0.5 – 1g/kg over 20 minutes ONLY if isolated head injury. DO NOT GIVE IN THE HYPOVOLAEMIC/HAEMODYNAMICALLY UNSTABLE PATIENT.
    - Ensure adequate sedation with propofol infusion
    - Paralysis to avoid coughing.

Primary Survey - *Continued*

Exposure

- Ensure normothermia (target 36 – 37°C).
- Avoid hyperthermia (temp > 38°C increases ICP and CO_2 production).
- Hypothermia < 35°C worsens coagulopathy.

Urgent non-contrast CT head

- Evaluate volume of intracranial bleeding, signs of hydrocephalus, herniation or mass effects.
- Urgent review of neuro-imaging and discussion with neurosurgeons to ensure rapid operative management if appropriate.
- Note: arterial phase imaging with contrast will take longer but should be considered when there are concerns for vascular injury e.g. carotid or vertebral arteries. (Skull base, facial or vertebral fractures).
- **See Blunt Cerebral Injury guidelines (Indication for CTA Neck in Trauma)**

Anticoagulation reversal / Treatment of coagulopathy

- Patients who are on anticoagulation will need URGENT reversal – reference to perioperative anticoagulation reversal guidelines and prompt discussion with on-call haematologist for advice if needed.
- Patients who are not on anticoagulation with abnormal coagulation screen may have trauma induced coagulopathy and should be aggressively resuscitated.
- General coagulation physiological targets: (specific cases may need to be discussed with neurosurgical team)
    - **Platelets > 100 x 10^9/L**
    - APTT < 38s
    - PR < 1.4
    - Fibrinogen > 1.5g/L
    - pH > 7.2
    - Ionised calcium > 1.1mmol/L
- Consider 1g TXA if within 3 hours of injury (survival benefit shown in CRASH-3 trial for moderate TBI).

Section 2:

Intracranial pathology considered for operative neurosurgical interventions at ADHB

TIME IS BRAIN

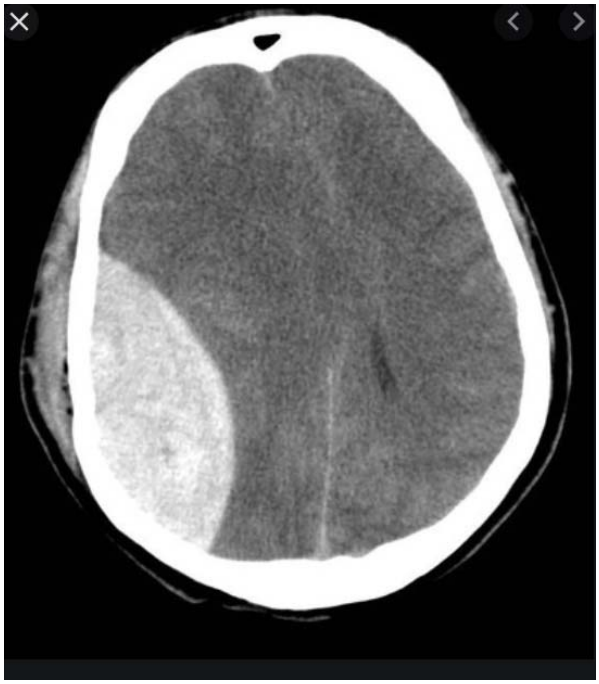
See: Trauma with a Positive Head CT – Guideline No. 3

CT confirmed intracranial pathology requires prompt discussion with neurosurgical teams, so that urgent theatre bookings can be made. On-call anaesthetist and theatre nurse coordinator should be notified ASAP, to allow appropriate theatre allocation and preparation.

Most patients with moderate (GCS 9 – 13)/severe (GCS 3 – 8) TBI will require intervention ASAP. The timeframe will be dictated by patient clinical state/GCS and imaging. (NB: Timing indicated is from deterioration to intervention.)

Extradural Haemorrhage:

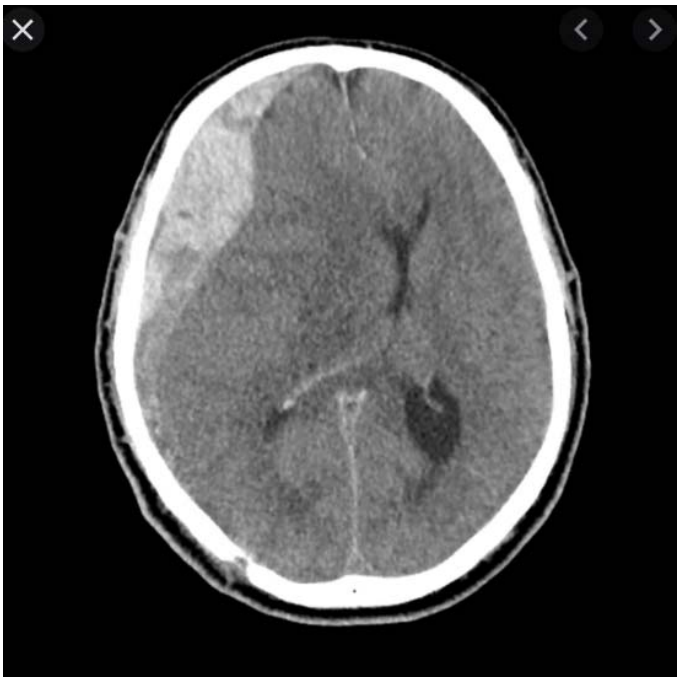
- A neurosurgical emergency
- TIMING: Operate ASAP/within 2 hours of deterioration
- Indication for surgery
    - Volume of blood > 30cm^3/30ml
    - GCS < 9 and asymmetric or fixed/dilated pupils
    - Focal deficit or midline shift



- Time from loss of consciousness to decompression is associated with outcome. (3% mortality rate if patients are conscious prior to surgery, but 28.6% mortality for patients with prior LOC)
- Skull fractures common (75 – 90%).
- Most commonly due to arterial injury (85% due to trauma to skull with tearing of the middle meningeal artery, 15% due to injury to dural sinuses/confluence of sinuses.)
- Significant bleeding may occur intraoperatively.
    - Monitor blood loss and correct coagulopathy as per coagulation targets/TEG.

Acute subdural haemorrhage

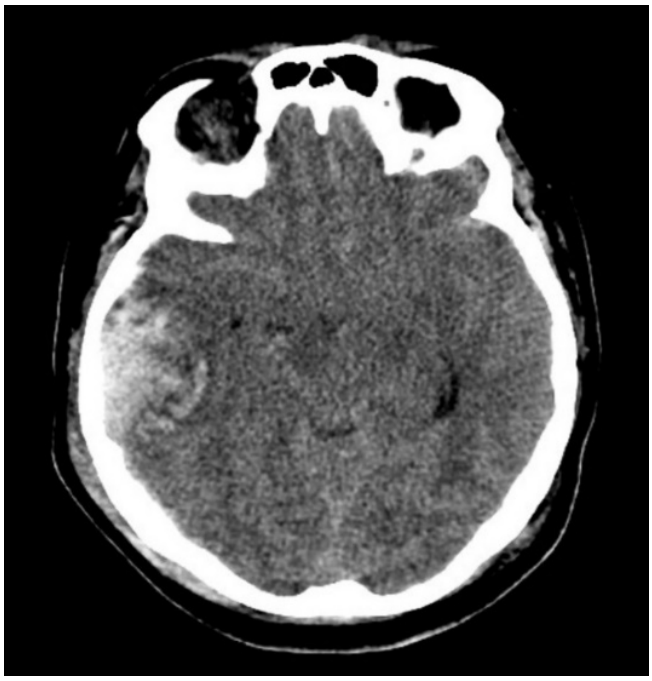
- Large craniotomy/craniectomy
- TIMING: Operate ASAP, aim within 2 hours of injury (NB: increased urgency if GCS drops 2 points).
- There is a correlation between time from neurological deterioration and evacuation, and outcome.
- Indication for surgery
    - Regardless of GCS if midline shift > 5mm or thickness > 10mm
    - Significant mass effect
    - GCS < 9
    - GCS decreased by more than 2 points from time of injury to admission
    - Asymmetric or fixed and dilated pupil
    - ICP measurements consistently > 22mmHg
- Decision to operate will depend on the above factors, along with salvageability and patient comorbidities
- Mortality rate correlates with clot thickness, with steep increase in mortality > 20mm
    - Mortality 10% < 10mm, increasing to 90% at 30mm thick.



Intracranial pathology considered for operative neurosurgical interventions at ADHB - *Continued*

Intraparenchymal/Intracerebral haemorrhage

- Craniotomy or ICP monitoring depending on imaging appearance and clinical state
- TIMING: Operate within 2 hours of presentation
- Indication for surgery
    - Lobar bleed with mass effect and poor GCS
    - Consider early intervention irrespective of GCS if significant lobar bleed i.e. > 50mm^3 or effacement of basal cisterns/herniation/> 5mm midline shift
    - Posterior fossa and > 30cm^3 volume and mass effect (brainstem compression, obliteration 4th ventricle, effacement of basal cisterns, obstructive hydrocephalus).



Depressed skull fracture

- TIMING: as soon as possible or first on emergency list if presented at night and GCS stable
- Dural penetration or depressed greater than thickness of cranium

Penetrating injuries

- TIMING: as soon as possible especially if foreign body present in skull

Section 3:

Anaesthesia Management of TBI

The aim of the anaesthesia is to allow emergent surgical decompression (TIME IS BRAIN), to reduce ICP, and prevent further secondary insult to the injured brain. This requires maintaining physiological targets to allow adequate cerebral perfusion pressure and decreasing $CMRO_2$.

The main physiological target is to maintain MAP 80 – 90mmHg in all phases of anaesthesia.

Pre-operative care

- Ensure primary survey/EMST principles are followed for management of trauma, associated injuries and initial management noted
- Re-assess/manage ABCDE
- If on anticoagulation – check indication, most likely require URGENT reversal, discuss with Haematology for advice if not done already
- Consider trauma induced coagulopathy if significant multi-trauma +/- activation of MTP
- Discuss with surgeon and DCCM re disposition of patient

Intra-operative care

- Resuscitate and correct hypovolaemia
- NB: The higher the ICP the more haemodynamically unstable the patient will be
- Suggested conduct and considerations
    - Awake arterial line and
    - Well running large bore IV access
    - Pump set + hot line (if considering blood product)
    - Consider propofol TIVA/remifentanil
    - Modified RSI with cricoid, noting C-spine precaution/requirements
        - Important to maintain cardiovascular stability during induction – ideally MAP 80 – 90mmHg, avoid significant hypotension (systolic < 90mmHg) or hypertension (systolic > 160mmHg) from laryngoscopy. Consider titrated bolus of propofol or use TCI pump to induce (smaller and slower bolus). Have metaraminol infusion running pre-induction 3 – 5mg/hr
        - Avoid hypoxia (SpO_2 > 94%) by ensuring patient is well preoxygenated, consider bag mask ventilation while waiting for onset of neuromuscular blockade
        - No neck ties for ETT
        - Head elevated 30^o
    - If using Mayfield pins – extremely stimulating, ensure adequate level of analgesia (consider increase remifentanil infusion rate or bolus fentanyl prior to pinning) to avoid hypertension response as this may worsen cerebral oedema and intracranial haemorrhage.
    - Consider if CVL required – NB: Do **NOT** delay starting evacuation of EDH for CVL insertion

- General physiologic targets
    - Traumatic brain injury disrupts blood brain barrier, perfusion to the brain is pressure dependent and autoregulation likely disrupted
    - Maintain normovolaemia, consider blood products early if significant blood loss. Avoid albumin if possible in TBI setting (SAFE study).
    - Monitor urine output as significant diuresis may occur with mannitol/hypertonic saline
    - Monitor and treat any coagulopathy, consider thromboelastogram (TEG) / coagulation profile
    - Maintain normothermia 36 – 37°C. Avoid hyperthermia < 38°C which increase $CMRO_2$,
    - Target $SpO_2 \geq 94\%$, normal PaO_2 > 13kPa, use lowest FiO_2 (hyperoxia may also be detrimental)
    - Maintain normoglycaemia BSL range 4 – 10mmol/L, avoid hypoglycaemia
    - Avoid and treat seizures / seizure prophylaxis discussed with surgeon if not initiated already from ED (levetiracetam 1g over 15 mins in 100mL 0.9% NaCl)

- **If evidence of brainstem herniation** (bradycardia, hypertension, fix dilated pupils) – the definitive treatment would be to get surgeons to decompress ASAP but temporising measures should include:

    - MAP 100 – 110mmHg to attempt to increase CPP
    - Hyperventilate to $PaCO_2$ 4kPa/30mmHg
    - Minimise PEEP 0 – 5cmH_2O
    - Hypertonic saline to achieve [Na^+] 150 – 155mmol/L – may require up to 50 – 100mL, depending on starting [Na^+].
    - Raise head of bed up 30 – 45°
    - Ensure deep anaesthesia and paralysis
    - Consider propofol TIVA
    - Consider mannitol 20% (100g in 500mL) – 0.5g/kg up to 1g/kg (ONLY IN ISOLATED TBI, NON-SHOCKED, HAEMODYNAMICALLY STABLE PATIENT) May be detrimental due to disrupted blood brain barrier and can increase swelling

- **If no evidence of brainstem herniation**

    - Continue to target cerebral perfusion pressure of 60 – 70mmHg
        - MAP 80 – 90mmHg, assuming raised ICP of ≥ 20mmHg for severe TBI (GCS ≤ 8 at presentation)
    - Avoid significant hypertension, target systolic < 160mmHg, especially if intracerebral bleed.
        - Increase remifentanil/propofol TIVA
        - Magnesium sulphate 2 – 5mmol (depending on starting systolic BP), up to 10mmol
        - Labetalol 10 – 20mg every 5 minutely
        - Hydralazine 10mg every 5 minutely
        - Nicardapine 0.5mg (care as BP may drop precipitously)
    - Ensure adequate depth of anaesthesia to decrease $CMRO_2$ i.e. BIS 40 – 60
    - Ventilation
        - Aim $PaCO_2$ 4.5 – 5.0kPa, PaO_2 > 10kPa
        - Do not hyperventilate ($PaCO_2$ < 4.5kPa) unless significant brain swelling / herniation as significant vasoconstriction of cerebral arterial vessels and will reduce cerebral blood flow and may worsen TBI outcome
    - Hypertonic saline 23.4% – bolus 10 – 20mL (40 – 80mmol) give over 10 mins. Faster rate can cause hypotension
        - Aim [Na^+] 145 – 150mmol/L, Osmolality < 320mmol/L
    - Ensure no obstruction/resistance to cerebral venous drainage
        - Head elevated 30 – 45°/no neck ties as discussed earlier
        - Ensure adequate neuromuscular block to avoid inadvertent coughing/straining which may increase CVP
        - PEEP < 10cmH_2O
    - CSF drainage – surgical management by inserting EVD to drain CSF

- **Other potential issues with TBI patients**

    - Monitor for electrolyte disturbance e.g. cerebral salt wasting syndrome (hyponatraemia [Na^+] < 135mmol/L, low plasma osmolality in context of fluid depletion and high urine [Na^+] > 40mmol/L and osmolality)
    - Paroxysmal sympathetic hyperactivity is a clinical syndrome which may occur in severe TBI due to loss of autonomic regulation. Features include periods/recurrent episodes of excessive sympathetic activity usually in response to internal or external stimuli (tachycardia, hypertension, tachypnoea, fever, sweating, and/or increased muscle tone with posturing).
        - Triggers are most common from external stimulation – endotracheal intubation, ETT suctioning, repositioning, urinary retention
        - Episodes are of rapid onset, with variable severity, with duration of 20 – 30min when untreated.
        - Treatment is mainly supportive by reducing stimulation, treating fever, and avoiding excessive ventilation
        - Pharmacologic treatment with beta-blockers and opioids may be necessary in the acute setting to maintain target MAP of 80 – 90mmHg, avoiding systolic BP > 160mmHg

Post-operative care

- Disposition to neurosurgical HDU or DCCM dependent on patient pathology and other associated trauma
- Early neurological assessment if possible
- **See: DCCM Management of Traumatic Brain Injury guideline**

Section 4:

Anaesthesia for non-neurosurgical procedures in patient with recent TBI

For multi-trauma patients with TBI needing return to theatre for other non-neurosurgical procedures – it is still important to use brain protective strategies to prevent further secondary brain injury.

The injured brain will remain vulnerable to further physiological insults in the weeks after initial injury. Recovery often can take months.

Discuss with neurosurgeon pre-operatively, to consider post-operative disposition and requirement for ICP monitoring, particularly if undertaking prolonged procedures (>4 hours).

The injured brain remains particularly sensitive to hypnotics/sedatives/analgesics. Consider Propofol TIVA and use short acting agents. Aim for early neurological assessment post-operatively.

The haemodynamic and physiologic targets for anaesthesia care remain largely the same:

- Avoid hypoxia – Sat. > 94%, PaO_2 > 10kPa
- Maintain appropriate cerebral perfusion pressure (CPP = MAP – ICP)
- Normocarbia – $PaCO_2$ 35 – 40 mmHg (4.6 – 5.3kPa)
- Consider head up to 30° (if appropriate for procedure)
- Avoid obstruction to venous return (ETT ties, C-collar etc)
- Limit intrathoracic pressure / avoid excessive PEEP
- Ensure normal Na^+
- Glucose 4 – 10mmol/L
- Maintain normothermia –temperature 36 – 37°C
- Continue seizure prophylaxis Levetiracetam 500mg BD (within 7 days)

Section 5:

Care for the Patient with ICP Monitor / External Ventricular Drain

ICP monitoring

https://adhb.hanz.health.nz/Policy/DCCM%20-%20Intracranial%20pressure%20monitoring.pdf

ICP is considered to require treatment if > 22mmHg
Intracranial hypertension is associated deleterious outcomes after traumatic brain injury (TBI)

Target CPP of 60 – 70mmHg reduces mortality.

Optimising CPP should first focus on treatment of ICP elevations (Severely disrupted autoregulation means that efforts should firstly be to minimise ICP, rather than elevating MAP, as hypertension worsens cerebral oedema).

At ADHB ICP monitoring is indicated in the following TBI patients:

- GCS 3 – 8 and abnormal CT scan
- GCS 3 – 8 and normal CT scan with any 2 of the following: age > 40 years, motor posturing, hypotension SBP < 90mmHg

May also be considered if:

- Evidence of brain swelling/cerebral oedema/hydrocephalus
- Patients with large bifrontal contusions
- Unreliable neurologic examination (e.g. maxillofacial trauma or spinal cord injury)
- Decompressive craniectomy performed as last resort for raised ICP refractory to medical management
- Following craniectomy but has risk factors for propagation of brain oedema e.g. hypoxia/hypotension/midline shift > 5mm/pupil abnormalities.

The monitor used at ADHB is the Codman® microsensor

- Inserted in theatre or DCCM in full aseptic manner
- Zero-ed by neurosurgeons prior to insertion and DO NOT RE-ZERO (see DCCM ICP monitor guideline for set up)

Normal range

- 0 – 10mmHg, upper 15mmHg
- Treatment required if ICP > 22mmHg
    - Follow DCCM protocol on tiers of treatment
    - Efforts to optimise CPP
    - Discuss with neurosurgeons re: potential for operative management i.e. decompressive craniotomy

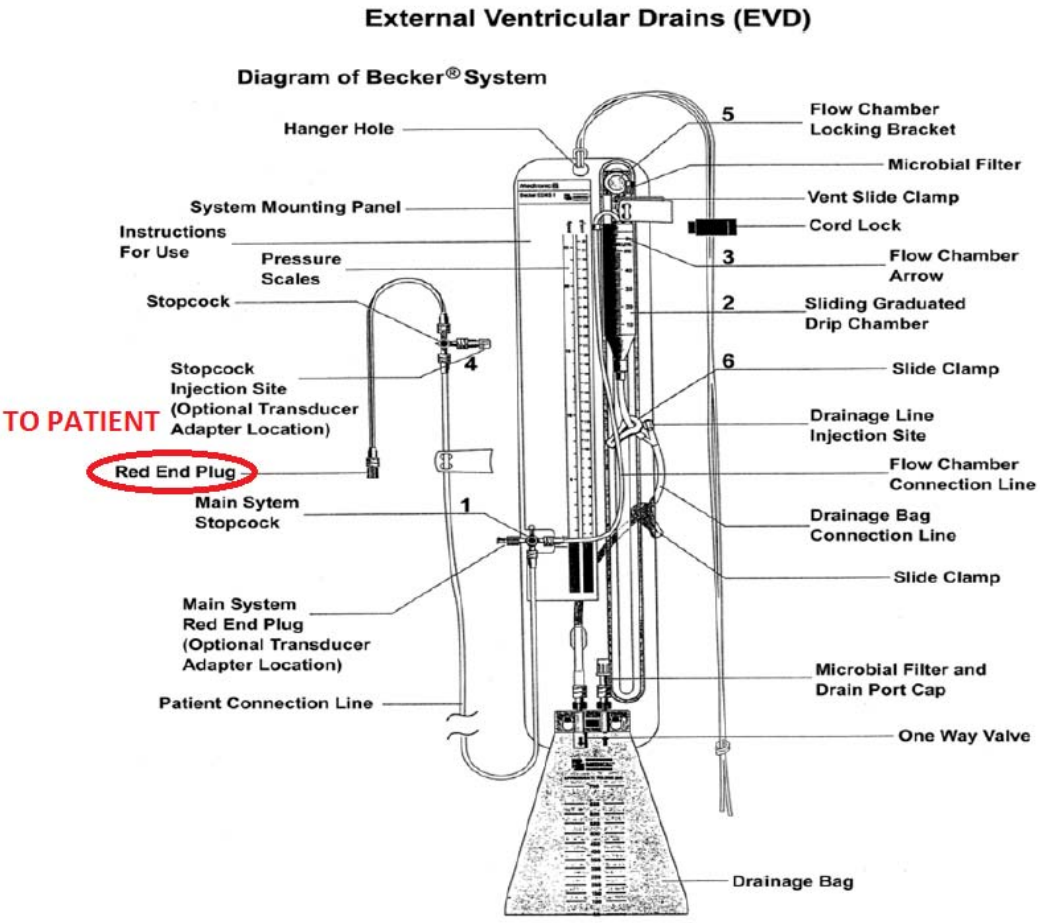
Complications associated with ICP monitors

- Malposition/dislocation/breakage – check with neurosurgeon if replacement required.
- Malfunction – intraventricular catheters can be blocked if too much CSF is drained, parenchymal catheters can drift and need re-zeroing (discuss with neurosurgeon).
- Infection – evidence of positive culture from CSF. Current infection rates increase significantly after 5 days. Can be reduced with subcutaneous tunnelling.
- Haemorrhage – rates vary, average incidence of 1.1% for intraventricular devices, careful balance of risk and benefit should be considered between DCCM and neurosurgeons regarding placement.

External Ventricular Drain (EVD)

https://adhb.hanz.health.nz/Policy/DCCM%20-%20External%20ventricular%20drain%20care.pdf

An EVD is a catheter that is inserted via a burr hole into the anterior horn of the lateral ventricle. It allows drainage of cerebrospinal fluid (CSF) as a way to reduce ICP, and also monitoring of ICP. At ADHB the Becker® system is used (see figure below).



The Becker® EVD system

The main aspects for looking after patients with EVD are:

- Measuring and checking the zero height of the drain – at the level of external auditory meatus (for patients lying flat), or the bridge of the nose (for patients lying on the side). Need to assess frequently if patient is moving in bed.
- Ensuring the drainage reference point on the Becker drain itself is set correctly – normally 10 – 15cmH_2O (NOT mmHg), this MUST BE PRESCRIBED by the medical (neurosurgical) team. This is the level at which CSF will drain into the collecting system.
- Monitor GCS closely – changes can be caused by too much CSF drainage or too little.
- Ensure patency of EVD, to detect malfunction/blockage or inadvertent closure of EVD system which can lead to an increase in ICP.
- Drainage should be recorded hourly and recorded in the ADHB CR5708 chart, reporting drainage, colour, and clarity, and contacting medical team promptly if concerns.
- Prevent infection – may provide direct route for microbes to the brain/central nervous system – strict asepsis must be maintained when handling insertion site/ports along the line. Report promptly if evidence of infection e.g. cloudy CSF or unexplained fever.

Current Evidence for Reference

Majority of evidence from the Brain Trauma Foundation 4th edition 2016 (Figure 1) and the 2020 update on indications for decompressive craniotomy (Figure 2).

TABLE 1. Updated Treatment Recommendations[a,b]

Topic	Recommendations
Decompressive craniectomy	Level IIA
	• [UPDATED 2020 (see above) Addition of results from RESCUEicp trial]
Prophylactic hypothermia	Level IIB
	• **Early (within 2.5 h), short-term (48 h post-injury), prophylactic hypothermia is not recommended to improve outcomes in patients with diffuse injury.**
Hyperosmolar therapy	Recommendations from the prior (Third) Edition not supported by evidence meeting current standards.
	Mannitol is effective for control of raised ICP at doses of 0.25 to 1 g/kg body weight. Arterial hypotension (systolic blood pressure <90 mm Hg) should be avoided.
	Restrict mannitol use prior to ICP monitoring to patients with signs of transtentorial herniation or progressive neurologic deterioration not attributable to extracranial causes.
Cerebrospinal fluid drainage	Level III
	• **An EVD system zeroed at the midbrain with continuous drainage of CSF may be considered to lower ICP burden more effectively than intermittent use.**
	• **Use of CSF drainage to lower ICP in patients with an initial GCS <6 during the first 12 h after injury may be considered.**
Ventilation therapies	Level IIB
	• Prolonged prophylactic hyperventilation with $PaCO_2$ of ≤25 mm Hg is not recommended.
	Recommendations from the prior (Third) Edition not supported by evidence meeting current standards.
	Hyperventilation is recommended as a temporizing measure for the reduction of elevated ICP.
	Hyperventilation should be avoided during the first 24 h after injury when CBF often is reduced critically.
	If hyperventilation is used, SjO_2 or $BtpO_2$ measurements are recommended to monitor oxygen delivery.
Anesthetics, analgesics, and sedatives	Level IIB
	• Administration of barbiturates to induce burst suppression measured by EEG as prophylaxis against the development of intracranial hypertension is not recommended.
	• High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.
	• Although propofol is recommended for the control of ICP, it is not recommended for improvement in mortality or 6-month outcomes. Caution is required as high-dose propofol can produce significant morbidity.[3]
Steroids	Level I
	• The use of steroids is not recommended for improving outcome or reducing ICP. In patients with severe TBI, high-dose methylprednisolone was associated with increased mortality and is contraindicated.
Nutrition	Level IIA
	• **Feeding patients to attain basal caloric replacement at least by the fifth day and at most by the seventh day post-injury is recommended to decrease mortality.**
	Level IIB
	• **Transgastric jejunal feeding is recommended to reduce the incidence of ventilator-associated pneumonia.**
Infection prophylaxis	Level IIA
	• Early tracheostomy is recommended to reduce mechanical ventilation days when the overall benefit is thought to outweigh the complications associated with such a procedure. However, there is no evidence that early tracheostomy reduces mortality or the rate of nosocomial pneumonia.
	• **The use of PI oral care is not recommended to reduce ventilator-associated pneumonia and may cause an increased risk of acute respiratory distress syndrome.**
	Level III
	• **Antimicrobial-impregnated catheters may be considered to prevent catheter-related infections during external ventricular drainage.**
Deep vein thrombosis Prophylaxis	Level III
	• LMWH or low-dose unfractioned heparin may be used in combination with mechanical prophylaxis. However, there is an increased risk for expansion of intracranial hemorrhage.
	• In addition to compression stockings, pharmacologic prophylaxis may be considered if the brain injury is stable and the benefit is considered to outweigh the risk of increased intracranial hemorrhage.
	• There is insufficient evidence to support recommendations regarding the preferred agent, dose, or timing of pharmacologic prophylaxis for deep vein thrombosis.
Seizure prophylaxis	Level IIA
	• Prophylactic use of phenytoin or valproate is not recommended for preventing late PTS.
	• Phenytoin is recommended to decrease the incidence of early PTS (within 7 d of injury), when the overall benefit is thought to outweigh the complications associated with such treatment. However, early PTS have not been associated with worse outcomes.
	• **At the present time there is insufficient evidence to recommend levetiracetam compared with phenytoin regarding efficacy in preventing early post-traumatic seizures and toxicity.**

[a] $BtpO_2$, brain tissue O_2 partial pressure; CBF, cerebral blood flow; CSF, cerebrospinal fluid drainage; DC, decompressive craniectomy; EEG, electroencephalogram; EVD, external ventricular drainage; GCS, Glasgow Coma Scale; GOS-E, Glasgow Outcome Scale—Extended; ICP, intracranial pressure; ICU, intensive care unit; LMWH, low molecular weight heparin; $PaCO_2$, partial pressure of arterial carbon dioxide; PI, povidone-iodine; PTS, posttraumatic seizures; RESCUEicp trial, Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of ICP trial; SjO_2, jugular venous oxygen saturation; TBI, traumatic brain injury.
[b] Bold: New or revised recommendations.

Figure 1 - Brain Trauma Foundation 2016 guidelines on treatment recommendations

Current Evidence for Reference - *Continued*

2020 Brain Trauma Foundation update for decompressive craniotomy

Level IIA–to improve mortality and overall outcomes

1. NEW–Secondary DC performed for *late* refractory ICP elevation is recommended to improve mortality and favorable outcomes.

2. NEW–Secondary DC performed for *early* refractory ICP elevation is not recommended to improve mortality and favorable outcomes†.

3. A large frontotemporoparietal DC (not less than 12 × 15 cm or 15 cm in diameter) is recommended over a small frontotemporoparietal DC for reduced mortality and improved neurological outcomes in patients with severe TBI.

Level IIA–for ICP control

4. NEW–Secondary DC, performed as a treatment for either early or late refractory ICP elevation, is suggested to reduce ICP and duration of intensive care, though the relationship between these effects and favorable outcome is uncertain.

†*Recommendation #2 should not be extrapolated to primary DC in which the bone flap is left off when an intracranial mass lesion is evacuated early after injury.*

Figure 2 - Brain Trauma Foundation 2020 update on decompressive craniectomy

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