TBI Management

1. Purpose of the Document

To guide the management of patients with acute traumatic brain injury (TBI) admitted to the Department of Critical Care Medicine (DCCM).

2. Responsibility

All medical and nursing staff providing care and treatment for patients admitted to the DCCM with TBI.

3. Document Principles and Goals

The primary role of intensive care for the patient with acute TBI is the prevention of second-ary neuronal injury.

A diverse range of pathophysiological processes contribute to secondary neuronal injury and these not only vary between individual and injury type but are also dynamic in time and heterogeneous across brain regions. Current monitoring modalities remain imperfect for characterising an individual's perturbations of cerebral physiology and few treatment options have established efficacy.

In practice, intensive care management aims to optimise cerebral physiology primarily by maintenance of cerebral perfusion pressure (CPP) and ICP within a strict target range. This is achieved through use of a TBI protocol in which initial baseline therapy and monitoring are applied to all patients (tier zero) and refractory problems are managed by therapy escalation, targeted to causative pathology where possible. Interventions that are more difficult to implement or present significant risk (e.g. rescue decompressive craniectomy) are used as last resorts.

The indications for ICP monitoring, treatment thresholds and therapies used in the DCCM align with internationally recognised guidelines and expert consensus. However, significant controversy regarding the use of ICP monitors in TBI patients remains; this extends to indications, duration and treatment thresholds. It is essential to consider patient characteristics, radiological imaging and findings from regular clinical examination in treatment decisions and therefore intensivist directed deviations from this document may be appropriate for some patients.

The goals of this document are to provide DCCM team members with information to manage patients admitted following TBI, it includes guidance on indications for insertion of ICP monitors, management of raised ICP and established treatment parameters.

4. Inclusion Criteria

All patients with severe TBI admitted to the DCCM, either for therapy or for prognostication reasons.

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5. Exclusion Criteria

Patients may not receive this treatment if:

- The duty intensivist (in consensus with the stroke and neurosurgical teams) determines a clinical or other indication for deviation from this document.
- The ICH is unsurvivable and the patient has been admitted as a potential organ donor or for palliative care.

6. Process of treatment

6.1 Initial Assessment of the patient with suspected TBI

This usually takes place in the emergency department and is performed by the DCCM registrar or intensivist alongside the ED team and in liaison with the neurosurgical registrar.

The nature of the TBI and other important clinical information will be ascertained and all patients requiring transfer to the DCCM will be discussed with the duty intensivist.

TBI is commonly associated with severe multi-trauma, the important differences in management of multi-trauma patients with associated TBI include strict avoidance of hypotension (MAP target of >90mmHg where appropriate-once stabilised this can be reduced to 80mmHg), strict avoidance of hypoxia, maintenance of brain orientated physiological targets where possible and a higher platelet target of > 100x109/L.

Patients with clinical evidence of trans-compartmental brain herniation (eg sudden dilatation of a pupil) should receive osmotherapy (e.g. 40ml of 4molar sodium chloride) and hyperventilation targeting hypocarbia (CO_2 <4.3Kpa) for a maximum of 30minutes while awaiting brain imaging and surgical planning.

a. All patients will have a CT brain, including arterial phase contrast if there is concern for arterial injury and CT venogram where skull fractures oppose a major venous sinus or there is suspicion of cerebral venous injury.

Important information to be recorded in the admission note are:

- Nature of the TBI, signs of hydrocephalus, trans-compartmental herniation or significant mass effect.

b. All patients will have a clinical exam performed by the DCCM Registrar or Intensivist

- Record will be made of the GCS, pupillary examination and the character of any focal neurological deficit at presentation as a minimum.

c. All patients will have the requirement for reversal of anticoagulants or coagulopathy considered

- Tranexamic acid is not routinely administered in the setting of isolated TBI but may be ad-ministered in the setting of severe multi-trauma.
- Reversal of warfarin or the DOACs should be undertaken as per ADHB policy or following advice from the on call haematologist.
- Patients receiving aspirin, ticagrelor or clopidogrel only do not usually require any specific treatment except for cessation of therapy.

- Maintain platelet count > 100x109/L, APTT/PT <1.5x normal, fibrinogen >1.5g/L and iCa >1.13mmol/L.

d. All patients will be discussed with the Neurosurgical registrar on call and this discussion including the name of the responsible Neurosurgical consultant will be documented

As a minimum this initial discussion will determine the need for evacuation of mass lesions or emergent EVD placement and when indicated initiate consensus discussion for patients with devastating brain injury. Surgical intervention is individualised and directed by the Neurosurgical team.

All patients who undergo surgical intervention should have the requirement for concomitant ICP monitor placement considered prior to transfer to the operating theatre (see guidance below).

6.2 Initial Management - All Patients

A. Intubate and ventilate if indicated

- A suggested induction regime is ketamine (1-1.5mg/kg) or propofol (dose modified to ac-count for hemodynamic status) and fentanyl (1mcg/kg) and rocuronium (1mg/kg) or sux-amethonium (1mg/kg). Have a vasopressor eg metaraminol available to treat any hypoten-sion.

B. Initiate standard monitoring including in all patients a CVL, NGT, IDC and arterial line

C. Sedation for ventilated patients

- Propofol (e.g. 1-2mg/kg/hr) and an opiate (e.g. fentanyl 20-50mcg/hr + PRN) titrated to RASS score documented by the intensivist on the treatment chart.
- Neuromuscular blockade should be used sparingly when indicated as neurological assess-ment forms an important part of management.

D. Maintain MAP >90mmHg until either clinical assessment or ICP monitor placement, after which the ICP protocol should be followed.

E. Maintain ICP protocol tier zero interventions for all patients.

F. Fluid Therapy should be administered to maintain normovolemia

- Hypovolemia from blood loss should be replaced with blood

and blood products in accordance with the principles of hemostatic resuscitation

- Albumin should not be administered to TBI patients
- No maintenance fluid is prescribed for TBI patients
- No hypotonic fluids or NG water is prescribed for at least the first 10 days following TBI

G. Haemoglobin should be maintained above 90g/L

- TRAIN, NEJM 2024

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H. GCS and pupillary size and response to light will be recorded hourly by nursing staff

- Change in pupillary symmetry, deterioration in GCS or evidence of a new focal neurological deficit will be notified to the ICU registrar urgently.

I. EVD Management

- The EVD, if present, is usually positioned at 15cm above the EAM unless documented oth-erwise.
- The bedside nurse will notify the registrar if there is no drainage from the EVD, new blood evident in the CSF or CSF volume exceeds 20mls/hr.
- Remedy of a blocked EVD and removal of the EVD is the responsibility of the Neurosurgical service.

J. Seizure Management and Prophylaxis

- Anti-epileptics should be administered to all patients who have a witnessed seizure.
- Seizure prophylaxis with leviteracetam may be considered with the intention of reducing the incidence of seizures if; GCS <9, cortical contusions, depressed skull fracture, penetrat-ing brain injury, evacuated intra-cerebral hematoma or extradural hematoma.
- Standard dose 1g IV/NG 12 hourly. (500mg for patients <50kg).

K. Enteral nutrition, glucose management and stress ulcer prophylaxis

- As per DCCM policy.
- 2kCal enteral nutrition (with psyllium 5ml NG Q8H) may be required to maintain sodium in the target range.

L. DVT prophylaxis

- All patients should receive pneumatic compression stockings.
- The initiation of chemo-prophylaxis should be by 48-72 hours and if there are concerns it can be discussed with the neurosurgical team but ideally not delayed.
- Heparin is the preferred agent usually dosed at 5000U 8-12 hourly SC.
- Once stable they can be transitioned to enoxaparin once daily, usually administered at night to allow interventions to occur safely the following day.

M. Volume status

- Volume status will be assessed clinically. Non-invasive, semi-invasive or invasive forms of volume status assessment or cardiac output monitoring are not routinely used for TBI pa-tients in the DCCM.

N. Fever

- Initial targets are 36-38 degrees Celsius.
- Fever may be associated with deterioration in neurological status or elevation in ICP, fol-lowing exclusion of other causes of neurological deterioration it is reasonable to initiate cooling measures to control fever.
- The aetiology of fever should be investigated in all cases including a sample of CSF in all patients with an EVD - the most useful markers of infection from EVD samples are the glucose concentration, a WCC:RBC >500:1, gram stain and culture.

O. Diabetes Insipidus.

Diabetes insipidus may occur due to the disruption of the pituitary function and reduced re-lease of anti-diuretic hormone (ADH).
The diagnosis should be considered when there is a sudden production of a large volume of dilute urine (>200ml/hr for 4 hours) with increasing serum sodium concentration and/or osmolality.
The diagnosis can be confirmed by sending paired serum and urine samples for osmolality and sodium concentrations. The urine should have an osmolality of <200mOsm/kg and be less than 50% that of the serum.
Treatment is with desmopressin (DDAVP) 2-4mcg IV and this should be discussed with the duty intensivist prior to initiating.

P. ICP monitoring

ICP monitors are placed by the neurosurgical team in theatre or sometimes in the DCCM, these are usually intra-parenchymal and generally placed in the frontal lobe of the non-dominant hemisphere. Alternatively the ICP motor may be positioned ipsilateral to the site of predominant pathology, particularly in the setting of bifrontal traumatic brain contusions at risk of abrupt deterioration, without warning, due to posterior displacement of the brainstem.
Placement of ICP monitors in TBI patients should be individualised to the clinical scenario but expert consensus and guidelines suggest consideration of ICP monitor insertion in the following circumstances:

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Guidance for ICP Monitor Placement	
GCS 3-8 following resuscitation and:	Action:
Normal CT *AND* 2 of; age >40years, unilateral or bilateral motor posturing or episode of systolic blood pressure <90mmHg	Recommended
Normal CT *AND NOT* meeting above criteria	Interval CT or expedited CT if clinical deterioration and ICP monitor only if radiographic progression
Minimal CT abnormality (i.e. traumatic SAH or small millimetric petechie)	Interval CT or expedited CT if clinical deterioration and ICP monitor only if radiographic progression (i.e. contusions develop, basal cistern or sulcal effacement develops)
CT with diffuse injury *AND* signs of raised ICP or brain swelling (i.e. compressed/absent basal cisterns, sulcal effacement, herniation or midline shift > 5mm)	Recommended
CT with cerebral contusions *AND NO* signs of brain swelling	Recommended if sedation cannot be withheld or clinical assessment is confounded and in patients with large bifrontal contusions or lesions close to the brainstem. Routine CT followup highly recommended.
Other abnormal CT scan with intra *OR* extra axial hematomas *OR* signs of brain swelling *OR* trans-compartmental herniation *OR* compressed basal cisterns	Recommended
Routine interval CT scan at 12-24hrs (or earlier if deterioration) should be considered in all patients to exclude progression of intracranial lesions that may require surgical intervention or modification of medical therapy.	

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Following Evacuation of Acute Supratentorial Lesion	
Motor score <5 Pupillary abnormalities Prolonged or severe hypoxia Prolonged or severe hypotension Compressed basal cisterns Midline shift >5mm or > than thickness of extra-axial clot Additional extra-axial hematoma, parenchymal injury or swelling Brain swelling intra-operatively	Recommended if any present
All patients who undergo secondary/rescue decompressive craniectomy should have post-operative ICP monitoring	
See also: DOI 10.1007/s00701-014-2127-4 for further guidance.	

Management of Raised Intracranial Pressure

Although significant variation exists, it is typical for the ICP to rise over the first few days, usually reaching a peak by day 5-7 before slowly subsiding. Even brief (>5mins) elevations in ICP are associated with higher rates of mortality and worse outcomes.

There is a lack of certainty about the threshold of ICP that justifies initiation of therapies that have intrinsic toxicity. The standard ICP target in the DCCM is <20mmHg. A guide to escalation in therapy to attain this target is described below but **all escalations in therapy beyond stage 0 therapies must be discussed with the duty intensivist.**

Each step must be preceded by confirmation of arterial and ICP monitor accuracy and consideration of need for repeat CT, EVD placement or surgical referral for evacuation of mass lesion. Ensure to cross-check the ICP if there is an EVD present

Tier 0 – Standard Brain oriented intensive care "BOIC"- All patients

- Adequate resuscitation to attain normovolaemia and Hb of at least 90g/L
- Head of bed 30^{o}
- Neck inline, c-spine collar removed if appropriate
- Target saturations >94%, C02 4.6-5.3kpa, CPP 60-70mmHg
- Maintain temperature 36-38 degrees with active cooling if required
- Propofol 2-4mg/kg/hr + opiate infusion
- Ensure serum sodium is high normal (140-145mmol/L)
- Ensure EVD if present is 15cm above EAM and patent

If ICP >20mmHg notify intensivist and move to tier 1

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Tier 1 - Consider repeat CT, need for evacuation of mass lesion or placement of EVD

1. Consider seizures – Empiric treatment and/or EEG monitoring (levetiracetam 1-1.5g IV Q12H)
2. If EVD present lower to 5-10cm above EAM and ensure patency
3. Elevate sodium target to 145-155mmol/L and administer 4molar Sodium in 20ml aliquots to attain new sodium target
4. Deepen sedation (target RASS -5) with benzodiazepine e.g. Diazepam 20mg IV x1 then 10mg IV 6 hourly
5. Administer muscle relaxant and initiate TOF monitoring – see NMB SOP
6. Control temperature to target 36-37^{o}C and ensure propofol <3mg/kg/hr
7. Ensure Hb target of 90g/L
8. Consider trial of frusemide if fluid balance is positive (e.g. 10mg IV Q6H x24hrs)
9. If no EVD ask surgeons whether one should be considered.

If ICP >20mmHg notify intensivist and move to tier 2

Tier 2 - **Consider repeat CT, need for evacuation of mass lesion or placement of EVD**

1. Administer anti-epileptic if not done already and consider EEG monitor
2. Trial CPP target 70-80mmHg and assess the effect on ICP or consider MAP challenge to assess cerebral auto-regulation. (The MAP challenge is to increase the MAP by 10mmHg for up to 20min and observe ICP- if it reduces then may consider raising MAP target).
3. Consider mild hypocapnia, CO2 4.3 to 4.6kPa (caution should be taken in lowering further in the absence of PbtO2 monitoring)
4. Consider mild hypothermia – 35-36^{o}C and ensure propofol <3mg/kg/hr, daily CK, lipid and ECG
5. Consider addition of ketamine as a continuous infusion (1-2mg/kg/hr)
6. Consider relaxing ICP target to 25mmHg

If ICP remains elevated notify intensivist and move to tier 3

Tier 3 –These are high level therapies and need to be discussed in conjunction with the duty intensivist and neurosurgeon. Consider repeat CT, need for evacuation of mass lesion or placement of EVD

1. Consider therapeutic hypothermia – 32-34^{o}C (Change propofol to alternative sedative) *or*
2. Consider barbiturate coma. Thiopentone 3mg/kg as a bolus repeated to a maximum of 40mg/kg or 5g (no faster then 1g per hour) followed by an infusion of 4-8mg/kg/hr o*r*
3. Consider secondary/rescue decompressive craniectomy *or*
4. Consider consensus discussion to limit further escalation in therapy or active treatment withdrawal and consider opportunity for organ donation

Important Caveats

1. **Critical Neuroworsening** defined as deterioration in GCS, decrease in pupillary reactivity, new pupillary asymmetry or mydriasis, new focal deficit or signs of herniation, should be evaluated emergently. If herniation is suspected, empiric treatment with osmotherapy and hyperventilation (to CO2 <4kpa) should be initiated and urgent repeat CT imaging obtained.
2. **Systemic hypertension and cerebral hyperaemia** with CPP persistently >90mmHg may drive progression of vasogenic cerebral edema and consideration may be given to administration of an antihypertensive agent. This is a consultant only decision.

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7. De-escalation of ICP Protocol and removal of ICP monitors

De-escalation of ICP directed therapies, progression to sedative free neurological assessment and timing of ICP monitor removal are consultant lead decisions. There are no established guidelines to guide de-escalation.

Factors that should be taken into account when deciding to wean ICP therapies include:

- Expected time course of injury and peak swelling
- Intensity of required therapy
- Results of clinical examination, imaging and monitoring

De-escalation of ICP Protocol/Sedation free Neurological Assessment Guidance	
Consider	**Do not de-escalate therapy or attempt sedation hold if any present:** - Any intervention required for raised ICP in last 12hrs - Escalation in tier of therapy in last 24hrs - Worsening neurological exam or pupillary reactivity - Worsening CT appearance: sulcal effacement, absence of basal cisterns or midline shift >5mm - Abnormal ICP waveform - P2>P1
Proceed with caution if any criteria met	**De-escalate ICP therapy or attempt sedation hold with caution if:** - <5 days post injury - < 2 interventions for ICP in last 24hrs - Stable but abnormal CT
Proceed if all criteria met	De-escalate ICP therapy or attempt sedation hold if: - > 5 days post injury - ICP waveform - P1>P2>P3 - No interventions for raised ICP in last 24hrs - Normal pupils - No CT evidence of raised ICP - Spontaneously declining ICP - Relaxing CO2 target is well tolerated

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Deescalation of ICP Protocol/Sedative free Neurological Assessment Guidance	
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Proceed with caution if any criteria met	**De-escalate ICP therapy or attempt sedation hold with caution if:** - <5 days post injury - < 2 interventions for ICP in last 24hrs - Stable but abnormal CT
Proceed if all criteria met	**De-escalate ICP therapy or attempt sedation hold if:** - > 5 days post injury - ICP waveform - P1>P2>P3 - No interventions for raised ICP in last 24hrs - Normal pupils - No CT evidence of raised ICP - Spontaneously declining ICP - Relaxing CO2 target is well tolerated

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Deescalation of ICP Protocol/Sedative free Neurological Assessment Guidance
There are no guidelines to direct timing of ICP monitor removal. Expert consensus suggests that 72 h of acceptable ICP is almost universally accepted whereas removal at 24 h is recommended only for patients with fairly benign CTs and favorable exams. The therapeutic intensity required prior to acceptable ICP and the Glasgow Coma Scale is an important guide to decision making and is a consultant led decision. (For further information refer to : https://doi.org/10.1007/s00134-019-05805-9) ICP monitoring can generally be removed if receiving tier 0 therapies only and: - The patient has been extubated *or* - GCS >8 (V score allocated 1 point in intubated patients)with a motor score of 4-6 on all exams for 24hrs *or* - No evidence of raised ICP *and* stable CT *and* no intervention for ICP for 3 days *or* - No evidence of raised ICP *and* stable CT *and* no intervention for ICP for 24hrs if > 5days post injury In general the ICP monitor should not be removed if there is any surgical intervention planned within the next 24hrs. Removal of the ICP monitor is the responsibility of the neurosurgical team although they are increasingly removed by the DCCM registrar- for guidance see document:

9. Neuroprognostication of the TBI patient

Accurate prognostication in TBI is important but difficult. The key clinical features at presentation that determine outcome are age, initial GCS and CT characteristics. However there is a complex interaction between injury and host variables and early prognostication, which may be inaccurate, risks invoking a self-fulfilling prophecy. All decisions involving prognostication of TBI patients should involve a multidisciplinary consensus taking into account patient and family wishes, the trajectory of recovery and the results of multi-modal testing.

Standard Initial Targets for all TBI patients – Escalation as per raised ICP management below	
MAP (mmHg)	80-120 (Transducer zeroed at phlebostatic axis- right atrium)
ICP (mmHg)	<20 (Transducer zeroed at right atrium)
CPP (mmHg)	60 -70 (Transducer zeroed at right atrium)
Oxygen	Saturations 94-96%
CO2 (KPa)	4.6 – 5.3 Kpa
Temp (degrees Celsius)	36 - 38 degrees

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Standard Initial Targets for all TBI patients – Escalation as per raised ICP management below	
Patient Position	2hrs left, 2hrs right, Head up - Max
Sodium (mmol/L)	140-145
Glucose (mmol/L)	4.0-10.0 - as per DCCM protocol
Enteral nutrition	As per DCCM Protocol - 2.0 kcal/ml (with 5ml Q8H Psyllium) may be required to avoid hyponatremia
EVD	15cm above external auditory meatus or as per intensivist
Hemostatics	Platelets >100x10^9/L, PR<1.5, APTT<40secs, Fib>1.5g/L and iCa^{2+} >1.13, Hb >90g/L
Sedation	Propofol + opiate
DVT Prophylaxis	In discussion with neuro-surgical team
CS and pupil exam	Hourly
Neurological deterioration, elevated ICP or low CPP should be treated as an emergency in accordance with the ICP protocol and the duty intensivist notified	