Group arrangements:
Salford Royal NHS Foundation Trust (SRFT)
Pennine Acute Hospitals NHS Trust (PAT)

Northern Care Alliance NHS Group

Traumatic Brain Injury Management Guideline

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Applies to	Salford Royal Care Organisation
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Protocol Summary Sheet

Stage ZERO

- **Head up or bed tilt 30 degrees**
- **Assess GCS hourly for 8 hours then 2-hourly then 4-hourly**
- **Avoid venous congestion**
- **Assess and optimise analgesia needs**
- **SpO_2 94-98%**
- **MAP >90mmHg and systolic Bp >110mmHg**
- **Early fixation of any long bone injury**
- Measure HbA1c and provide Glycaemic control
- Consider anticonvulsant therapy

Neuroworsening is defined as a drop in GCS of 1 point, or a change in pupillary signs, or an ICP >20mmHg for 5 minutes and should prompt a medical review and the consideration of the escalation in therapy.

Is any neuroworsening due to an intracranial cause?

Consider repeat CT head and insertion of an EVD or evacuation of any surgical lesion

Tier ONE

- **Head up or bed tilt to a maximum of 30 degrees**
- **Avoid venous congestion and remove any collar**
- **Sedation to a RASS -5 & avoid coughing**
- **$PaCO_2$ 4.5-5kPa & PaO_2 >10-12kPa Sats 94-98%**
- **Target CPP 60-70mmHg with arterial transducer zeroed at the ear**
- **Treat hyperthermia >38°C**
- Measure HbA1c and provide glycaemic control
- Ensure analgesia optimised
- Consider anticonvulsant therapy
- Consider neuromuscular blockade

If Hb <100g/L at any time randomise into the Hemotion trial if eligible.

Is any neuroworsening due to an intracranial cause?

Consider repeat CT head and insertion of an EVD or evacuation of any surgical lesion

Tier TWO

- $PaCO_2$ 4-4.5kPa
- Treat extracranial causes of increased ICP
- Ensure adequate cardiac output using continuous monitoring and maintain CPP 60-70mmHg
- **IBW based dosing of osmotherapy**
- Consider loop diuretics if >3l +ve balance
- Ensure normothermia (36-37°C) with active targeted temperature management
- Perform a MAP challenge to assess cerebral autoregulation and inform consultant MDT individualisation of the CPP target
- Consultant MDT individualisation of the ICP threshold (20-30mmHg)
- Consider a Ketamine infusion

Osmotherapy and the SOS trial

Is any neuroworsening due to an intracranial cause?

Consider repeat CT head and insertion of an EVD or evacuation of any surgical lesion

Tier THREE always requires consultant discussion

- Ensure normothermia (36-37°C) with active targeted temperature management
- Consider a large decompressive craniectomy
- Consider barbiturate infusion to 50% burst suppression using continuous EEG monitoring and ensure adequate cardiac ouput with invasive monitoring

The change in volume obtained by any operative management should be accompanied by an intervention targeting a reduction in brain water content before safe de-escalation of therapy

1. Overview (What is this guideline about?)

This document outlines a tiered management structure for patients with a traumatic brain injury.

It is available in a smartphone enabled version online at www.neuroicu.guru

If you have any concerns about the content of this document please contact the author or advise the Document Control Administrator.

2. Scope (Where will this document be used?)

The document defines the care of patients with severe traumatic brain injury admitted to Salford Care Organisation.
It should be referred to by all staff who manage adult patients with severe Traumatic Brain Injury:

- Medical – Neurosurgery, Emergency Department, Anaesthetics, Critical Care
- Nursing – Emergency Department, Critical Care, Theatres, neurosurgical wards, TAU
- AHPs – Emergency Department, Critical care, Theatres, neurosurgical wards, TAU

Associated Documents

- ICP/EVD
- RASS
- VTE hub
- NCA Critical Care bowel management guideline

3. Background (Why is this document important?)

- Traumatic brain injury (TBI) accounts on average for approximately 25% of the admissions to Salford Royal Critical Care.
- TBI is a heterogeneous disease.
- Our focus is to limit the degree of secondary brain injury occurring as a result of brain swelling or as a result of systemic deterioration. As the injured brain swells, the closed box that constitutes the skull will rapidly lead to a rise in intracranial pressure (ICP). This rise in pressure may then compromise brain perfusion, local oxygen delivery and further damage neuronal pathways resulting in death or worsening functional outcome.
- The treatment of TBI is aimed at keeping the ICP at low levels (<20mmHg) and maintaining cerebral perfusion pressure (CPP) at reasonable levels (generally 60-80mmHg).

- **There is no 'magic bullet' therapy for TBI, the key to maximising outcome lies in attention to detail and ensuring evidence based practice is reliably implemented in all of the patients all of the time.**
- A multidisciplinary review led by the consultant intensivist and consultant neurosurgeon happens at least twice a day. In this review we assess the response to treatment, the control of ICP and may individualise targets for therapy. The review may decide on further imaging and provide guidance on the escalation or de-escalation of treatment when indicated.
- Functional outcome deteriorates and mortality increases when there is sustained intracranial hypertension (>22mmHg for 37 minutes in total).
- All interventions to lower ICP or increase CPP are all associated with potential harm and therefore a balanced approach to decision making is needed before escalating therapy. When possible, endeavour to use the lowest tier of treatment.
- At any time if the ICP rises to greater than 20 mmHg for 5 minutes, there should be an urgent medical review.
- Further imaging may be required, in the form of a CT head, to ensure no surgically correctable lesion is present.
- The consultant MDT guidance on the appropriate escalation of therapy should then be implemented.

4. What is new in this version?

- Emphasis on our research portfolio. We should randomise into trials rather than provide random care on the clinical questions in which we have equipoise.
- The introduction of the term neuroworsening. Neuroworsening is defined as a drop in GCS of 1 point, or a change in pupillary signs, or an ICP >20mmHg for 5 minutes and should prompt a medical review and the consideration of the escalation in therapy.
- Ideal body weight based osmotherapy dosing as per the SOS trial protocol.
- MAP challenge in tier 2 to assess cerebral autoregulation and inform the consultant level MDT individualisation of CPP targets.
- Increased emphasis on the utilisation of invasive cardiac output monitoring in the higher tiers of therapeutic intervention.

5. Guideline

5.1 Stage ZERO

Stage ZERO therapy in un-intubated TBI patients considered to have a minor injury or in those recently de-escalated from level one therapy:

- Documented neurological examination and observations by a trained nurse including the Glasgow Coma Score APPENDIX and the pupillary response to light. This should be performed hourly for 8 hours then de-escalated to 2-hourly for a further 8 hours and then performed 4-hourly. A trained nurse is defined as one who has completed the mandatory training on neurological observations.

- 30-degree head of the bed elevation or whole bed tilt if the spine is suspected to be unstable or awaiting formal clearance.

    - Cervical, thoracic and lumbar spinal imaging must be complete before admission to Critical care
    - The spinal imaging must be reviewed and a plan documented, as a clinical note, by the neurosurgery or radiology team within 12 hours of admission.
    - All extraction or hard plastic collars should be removed.
      Hard extraction collars should never be in place >2hours after arrival at SRFT. Prolonged use is associated with pressure ulceration.
    - An Aspen or a Philadelphia collar can be applied if a suspected unstable spine is present or prior to formal documented clinical spinal clearance.

- Ensure no cerebral venous congestion from positioning.
  Most individuals have a dominant right sided cranial venous drainage system. **Maintain an aligned neutral head position to avoid venous congestion.**

- MAP should be maintained ≥90mmHg and systolic Bp ≥110mmHg with blood & blood products, intravenous Plasmalyte-148 and or vasopressor therapy as deemed clinically appropriate.
  Even in the absence of another injury, up to 70% of TBI patients can demonstrate significant haemodynamic instability.

- **Correct any coagulopathy and immediately reverse anticoagulant therapy if present as per Salford Thrombosis and Anticoagulation hub guidelines.** In addition to iatrogenic coagulopathy, Trauma induced coagulopathy (TIC) on admission is also associated with worse clinical outcomes. Recent evidence from the ITACTIC and PAMPER trials suggest a potential benefit to rapid identification and correction of TBI coagulopathy. At present, this evidence is hypothesis generating. Viscoelastic Haemostatic Assay (VHA) testing is available 24 hours a day and can be used to identify and guide correction of extreme TIC or other coagulopathy, as per the

separate trust guideline, at clinical discretion. VHA at Salford is accessed via the on call ODP using the machine in theatre recovery (TEG 6S), with results reported on a separate webserver.

- **Ensure that a valid Group & Save sample is in the lab.** A repeat sample is needed each 72 hours and an additional sample will be required when a temporary hospital number is superseded by the confirmed identification hospital number.

- DVT prophylaxis should be considered in all patients. Appropriate prescribing information can be found at the Thrombosis and Anticoagulation Hub and with accessible decision support using the EPR HAT risk assessment tool (selecting emergency surgical admission / cranial surgery)

- Pain should be assessed and an appropriate analgesia regime instituted. This should include regular paracetamol +/- small doses of opiates.
  Pain which requires analgesia in excess of this regimen mandates medical review and consideration should be given to repeat imaging.

- **ANTICONVULSANT** therapy:

  - A 7-day prophylactic limited course of sodium valproate should be prescribed in patients with temporal lobe injury or a depressed skull fracture:
  - Loading dose 800mg IV over one hour followed by 1.6g IV over 23 hours, then:
  - <60kg 600mg bd sodium valproate NG/IV
  - >60kg 600mg tds sodium valproate NG/IV
  - Intravenous sodium valproate should be commenced for witnessed seizure activity at any time or if non-convulsive status epilepticus is suspected.
  - **In women of childbearing age the preferred agent is levetiracetam.**
  - Levetiracetam 2g stat IV
    then 1g bd NG/IV in the presence of normal renal function.
  - When prescribing anticonvulsant therapy, the indication must be clearly recorded (eg. Witnessed seizure, prophylaxis due to depressed skull fracture, suspected non-convulsive status).
  - All anticonvulsant prescriptions should be reviewed at 3 months following injury.

- Maintain euvolaemia and plasma Na^+ ≥135mmol/L, Mg^{2+} 0.7-1.0mmol/L and check the serum Lactate <2.0mmol/L

- All patients unable to eat and drink should receive enteral feed as per the enteral feeding calculator (at least 25kcal/kg/day Adjusted Body Weight). There is good evidence that cumulative caloric deficits are associated with worsened outcomes in TBI.
  Dietician referral is indicated in all patients with BMI <18 or >35.

- All patients should be commenced on laxatives as per the NCA bowel management protocol.

- A full tertiary survey should be performed and documented within the first 24 hours. This warrants a thorough clinical examination and correlation with the trauma pan-scan findings. It may need to be repeated as patients recover from TBI reporting pain or they exhibit bruising not previously noted.

- **FLUID THERAPY** in Traumatic brain injury
  - We aim to maintain euvolaemia. This is particularly important in individuals who require vasoactive support to maintain optimal cerebral perfusion pressure and blood flow.
  - Positive fluid balance is associated with increased mortality in TBI.
  - Fluid responsiveness is normal in a euvolaemic state and early cardiac output monitoring can be required to ensure safe use of vasoactive therapies.
  - The total maintenance daily fluid requirement for an individual patient is 30ml/kg day.
  - In normothermic individuals after the first 24 hours of admission to Critical Care their daily fluid requirements are met by:

  Enteral feed + drug infusions + bolus drugs
  E.g. for a 70kg individual: 2100ml total fluid required by calculation

  1270ml Osmolite HP @1.0kcal/ml
  + 600ml (Propofol and Alfentanil)
  + 400ml Paracetamol ≈ 2270ml delivered fluid

  i.e. supplemental continuous parenteral infusion of crystalloids once full enteral feed is established and being absorbed should be avoided.

  High insensible or enteral losses are exceptions to this guidance and parenteral Plasmalyte-148 should be given to replace losses and maintain euvolaemia.
  Remain vigilant to the possible development of diabetes insipidus. APPENDIX.

  Hypotonic parenteral fluids (5% glucose and 0.18%NaCl + 4% Glucose) or enteral water should be avoided in the first 10 days following a severe traumatic brain injury, unless brainstem death is clinically suspected or

under specific MDT instruction. If a clinician is in doubt, discuss with the critical care consultant.

5.2 Escalation to Tier ONE therapy

If GCS falls by 1 or below an absolute value of 13 then there should be immediate senior medical review.

Early discussion with the duty Critical Care Consultant or senior trainee is advised.

Care should be escalated when appropriate.

TBI is often associated with alcohol or drug intoxication. However, alcohol or illicit drug use should never be assumed to be the cause of a drop in GCS in a trauma patient.

A CT scan demonstrating signs of herniation, effacement of the cisternal or ventricular architecture should prompt early intubation and ventilation regardless of the absolute GCS, to enable prevention of secondary injury.

If GCS equals 8
often we intubate too late

The actual drop in GCS may be a late indicator of a worsening brain injury and as such other triggers for escalation may include:

- *A worsening headache*
- *Increasing agitation or behavioural changes*
- *Unexplained hypertension or changes in heart rate or heart rate variability*
- *New changes in the pupillary response to light*
- *The development of a new motor deficit*
- *Nursing staff report that it is becoming more difficult to achieve the same GCS or the duration that this level of GCS is maintained is reducing.*

Remember in a young patient the motor score is the most sensitive component of the GCS. If the Motor score is less than 5 then intubation and ventilation for a further CT is indicated.

In the elderly or in a patient with a degree of cerebral atrophy the verbal component is more sensitive and Verbal score less than 3 may indicate a need to escalate therapy.

Is the worsening of the patient's condition due to an intracranial cause?
Consideration must be given for repeat imaging and operative intervention for CSF drainage or significant space occupying lesions, prior to the escalation of medical therapy for intracranial hypertension.

Patients who demonstrate signs of coning, transtentorial herniation or progressive neurological deterioration not attributable to extra cranial causes, prior to the establishment of ICP monitoring, should be treated with Mannitol at a dose of 0.25-0.5g/kg Ideal Body Weight.

This may be given as an IV bolus during the preparation for induction of anaesthesia.

100ml 10% Mannitol contains 10g

Mannitol dose for a 70kg individual: **175-350ml 10% Mannitol solution**

All patients who receive Mannitol are highly likely to subsequently need intravenous fluid resuscitation to maintain cerebral blood flow following their diuresis.

Anaesthetic conduct

Ketamine is the anaesthetic agent of choice in trauma:

A suggested induction regimen is:	Fentanyl	1mcg/kg
	Ketamine	1-1.5mg/kg
	Rocuronium	1mg/kg

Immediately following the induction of anaesthesia, the pupillary response to light should be reviewed and ventilation established to $ETCO_2$ 4-4.5kPa.

MAP should be maintained throughout ≥90mmHg and systolic Bp ≥110mmHg with the use of appropriate vasopressor therapy, blood and blood products and intravenous fluids.

5.3 TIER ONE THERAPY (in all mechanically ventilated patients)

Invasive arterial and intracranial pressure monitoring should ideally be sited within the first 2 hours of admission to SRFT ICU or after the decision to escalate therapy to stage one therapy.

ICP monitoring may not be appropriate in patients with an uncorrected coagulopathy, or in those with no CT imaging evidence of raised intracranial pressure.

If there is no ICP monitor in situ, thorough repeated clinical examination must be performed every hour. Any unexpected changes or neuroworsening mandates repeated CT imaging and early discussion with the ICU consultant.

- Ensure there is no cerebral venous congestion from poor positioning or tight endotracheal tube ties.
  - **The head position should be aligned and neutral**
  - It is standard practice within SRFT Critical Care to remove all collars and place blocks either side of the head whilst the patient is fully sedated and mechanically ventilated.
  - Spinal precautions must be taken when log rolling.
  - An Aspen or a Philadelphia collar can be applied when a suspected unstable spine is present and a sedation hold or reduction is undertaken.

- A green pillowcase should be used to identify the head pillow (and aid in the reduction of hospital acquired infection).

- 30-degree (maximum) head of the bed elevation or whole bed tilt if spine is suspected to be unstable or awaiting formal clearance.

- Invasive ventilation targeting a minute volume to maintain $PaCO_2$ 4.5-5kPa

- ARDS low tidal volume ventilation is not appropriate. High respiratory rates >22 should be avoided and lung recruitment maintained with tidal volumes ~8ml/kg IBW and appropriate use of PEEP. Any change in ventilation may increase intrathoracic pressure and contribute to increased intracranial pressure.

- Maintain PaO_2 10-12kPa and Oxygen saturations 94-98%

- All TBI patients admitted to critical care should have their HbA1c checked routinely on admission. A growing evidence base suggests that chasing normoglycaemia in TBI patients with poor glycaemic control may precipitate a cerebral metabolic crisis and worsen outcome. Glycaemic control should be maintained with insulin by infusion using the web based calculator.

- **Sedate to a Richmond Agitation Sedation Scale - 5**
  - Propofol 1% 0-25ml/hr (max dose 4mg/kg/hr IBW)
  - Alfentanil 0-2ml/hr (25mg/50ml)

- A third sedative agent may also be required Midazolam 0-20mg/hr.
  Midazolam has a long half-life. Achieving a steady state by continuous infusion can take many hours.
  Repeated bolus doses should be used initially prior to increasing the rate.
- Coughing in a patient with poor intracranial compliance must be avoided.
- Pre-emptive boluses of sedative agents (Midazolam 2-5ml, Alfentanil 1ml, or Propofol 1% 2ml) may be required to ensure ICP control during stimulating procedures or care e.g. endotracheal suctioning or changing bed linen. These boluses should be recorded on the observation chart.

- Sedated patients must be subject to good clinical examination including brainstem function at least every hour.
  - The pupillary size and response to light should be assessed. Any changes should prompt medical review.

It is unacceptable to just write sedated across the GCS section of the observation chart.

If any GCS component cannot be tested record it as NT (not tested) on the chart.

- Maintain optimal CPP 60-70mmHg:
  - Use up to 0-10ml/hr 4mg:50ml Noradrenaline and up to 4 x 250ml boluses of Plasmalyte-148 as appropriate.
  - **The arterial transducer used to estimate the MAP for the calculation of CPP should be zeroed and positioned at the level of the ear when an ICP probe is in-situ.**

NO patient should receive >10ml/hr 4mg: 50ml Noradrenaline without medical review.

Always consider the early use of continuous flow monitoring in addition to echocardiography to ensure euvolaemia and titrate cardiovascular support.

Always examine the patient to assess adequacy of perfusion and beware of excessive vasoconstriction. If the peripheral skin microcirculation is poor, so is the cerebral microcirculation.

High doses of noradrenaline may contribute to cerebral pyrexia by preventing peripheral heat loss.

Any patient receiving >8ml/hr 12:50 NA should be screened for hypopituitarism (check the serum cortisol). If the serum cortisol is inappropriately low, start hydrocortisone 50mg tds for 7 days.

- Ensure full volume-based enteral feed is prescribed per the web-based calculator. There is good evidence that cumulative caloric deficits are associated with worsened outcomes in TBI. Dietician referral is indicated in all patients.

- Ensure analgesic requirements are reviewed regularly.
  Appropriate analgesia: 6-hourly weight-based dosing of paracetamol NG/IV +/- opiates.

Additional tier one therapy in selected patients includes:

- External ventricular drainage of CSF. The siting of an EVD mandates the use of the yellow paper EVD pathway and clear marking of an exclusive green pillow for the head. If an EVD has been sited, patient handwashing with soap and water is mandated three times daily and nail-cleaning once a day- both as measures to reduce medical device related ventriculitis.

- Treatment of hyperthermia >37^{o}C by simple measures.
  Remember that intracranial temperature is routinely >1 ^{o}C higher than tympanic temperature. APPENDIX

- Treatment of infection as per trust infection control and antibiotic guidelines

- Consider establishing non-depolarising neuromuscular blockade in conjunction with neuromuscular junction monitoring if bolus therapy results in an improvement in ICP control.

- Consider a multichannel EEG/ employ cEEG monitoring or empirically start/escalate any anticonvulsant therapy.
  When prescribing anticonvulsant therapy, the indication must be clearly recorded (eg. Witnessed seizure, prophylaxis due to depressed skull fracture, suspected non-convulsive status). All anticonvulsant prescriptions should be reviewed at 3 months following injury and their indication and 3-month review date made clear in the patient handover of care/critical care discharge summary.

- A patient intubated for severe brain injury (GCS<12) within 24 hours of injury may be eligible for recruitment into the Hemotion trial if Hb<100g/L..

An **ICP ≥ 20mmHg sustained for 5 minutes** should prompt medical review and further intervention or escalation to a higher level of the protocol.

Each intervention to control ICP or maintain CPP should be recorded clearly on the observation chart, e.g. sedation bolus, change in minute volume or escalation in the level of care. There is a sticker to facilitate this.

The total number of interventions required in the last 12-24 hours can then be used to inform the MDT plan for the next 12-24 hours.

Is the loss of ICP control due to an intracranial cause?
Consideration must be given for repeat imaging and operative intervention for CSF drainage or significant space occupying lesions, prior to the escalation of medical therapy for intracranial hypertension.

A multidisciplinary neurocritical care and neurosurgical plan should be clearly documented, as to which level two therapies are to be offered if required.

5.4 Tier TWO THERAPY – triggers further senior medical review

Why has the ICP risen? Is the prescribed CPP maintained?
Check if all Tier ONE measures actually in place?
Is it due to an intracranial or extracranial cause?
Is there adequate venous drainage?
Is a repeat CT head indicated?

- **Prescribe an increase in minute ventilation to target PaCO2 4-4.5kPa**:

ARDSnet-style low tidal volume ventilation is not appropriate in isolated TBI patients.

High respiratory rates >22 should be avoided

Maintain lung recruitment with tidal volumes ~8-10ml/kg IBW

Titrate PEEP to achieve oxygenation goals SpO2 94-98%.

Any change in ventilation may increase intrathoracic pressure and contribute to a rise in intracranial pressure.

An increase in minute ventilation should be recorded on the observation chart as an ICP intervention.

An arterial blood gas should be checked after 30 minutes to assess the response.

A wide alveolar-end-tidal CO2 gradient >0.5kPa may indicate a low cardiac output state or significant ventilation-perfusion mismatch.

- **Treat extracranial causes of compromised venous outflow**:

Remove all cervical collars

Ensure the head is in the neutral aligned position

Loosen off any tight ETT ties

Avoid high mean airway pressures and be aware of the possibility of breath stacking with a high set respiratory rate.

Check for intra-abdominal hypertension and treat as appropriate.

- **A cardiac output monitor should be sited to confirm adequate intravascular volume and to ensure adequate blood flow. Fluids, inotropes or higher doses of noradrenaline may be required to optimise blood flow and maintain an optimal CPP 60-80mmHg.**

- **External ventricular drainage of CSF.**
  This may require a BRAINLAB© CT scan.
  The siting of an EVD mandates the use of the yellow paper EVD pathway and the use of an exclusive green pillow for the head.

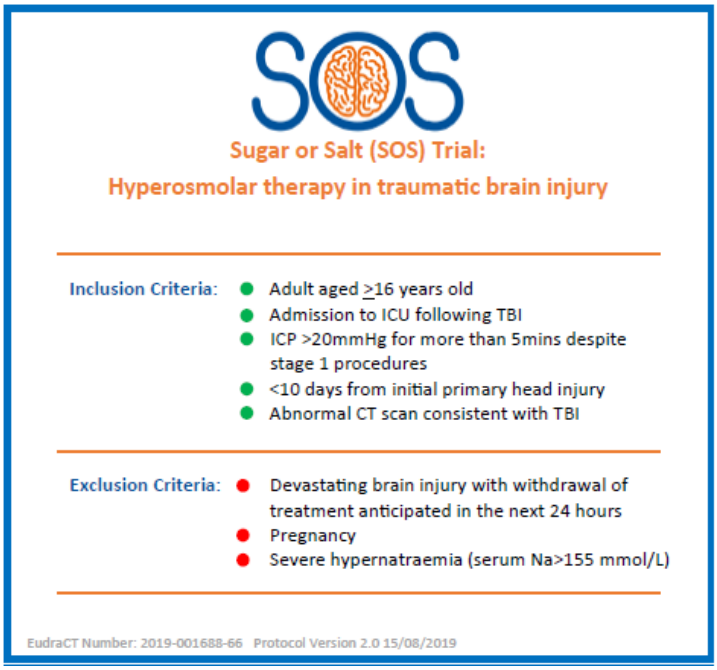
- **Target normothermia 36-37ºC**
  Utilising an external or invasive cooling device, or by infusing small volumes of fridge cold intravenous fluids (only when fluid therapy is indicated). Remember that intracranial temperature is routinely >1 ºC higher than tympanic temperature. APPENDIX

- **OSMOTHERAPY**

Osmotherapy is a standard worldwide intervention for neuroworsening, as a result of cerebral oedema, but it is unknown whether hypertonic NaCl or Mannitol is the agent of choice and the current evidence base is low grade.

The Salford neurocritical care and neurosurgical team have a position of equipoise and our unit is participating in the NIHR SOS randomised trial to answer the research question comparing these two agents.

Do NOT exclude patients that have received osmotherapy prior to ICU admission. Co-enrolment has been agreed between the SOS trial and ADAPT-sepsis, and SOS and Hemotion.



Following randomisation or outside the trial under senior medical discretion osmotherapy can be administered as follows:

Hypertonic 30% NaCl

30% NaCl can only be given via a CENTRAL LINE.

Consultants (*or senior trainees)* **may give an ideal body weight defined bolus of 30% NaCl (15ml contains 75mmol Na+) using an infusion pump OVER 15 minutes, and repeated to establish ICP control.**
Each bolus could increase plasma Na^+ by 2-3mmol/L.
This dose does not account for any ongoing naturesis, free water loss or sodium intake.

One hour after each bolus check the serum Na^+ level.
A rebound increase in ICP within 2 hours requires discussion with consultant.
If serum Na^+ ≥155mmol/L or calculated plasma osmolality ≥320mosmol/L then 30% NaCl is contraindicated.

The use of osmotherapy mandates close attention to the detail of fluid and sodium balance, a minimum of 4-hourly medical review is standard practice.

The falling serum Na^+ must continue to be managed closely to prevent rebound cerebral oedema.

If required a target serum Na^+ 150-155mmol/L can be maintained by infusing 3-5ml/hr 30% NaCl or by scheduled boluses of 30%NaCl every 6 hours and then weaned to a maximum fall of 5mmol/L/24 hours.

10% Mannitol

10% Mannitol can be given via CENTRAL LINE or a PERIPHERAL cannula (18G or larger).

Consultants (*or senior trainees)* **may give an ideal body weight defined bolus of 10% Mannitol using an infusion pump OVER 15 minutes, and repeated to establish ICP control.**
Each bolus will increase the plasma osmolality and will be followed by a marked diuresis.

Once a day the plasma osmolality must be measured if the plasma osmolality ≥320mosmol/L then further Mannitol is contraindicated.

A rebound increase in ICP within 2 hours requires discussion with consultant.
If serum Na^+ ≥155mmol/L or measured plasma osmolality ≥320mosmol/L then further osmotherapy is contraindicated.

The use of osmotherapy mandates close attention to the detail of fluid and sodium balance, a minimum of 4-hourly medical review is standard practice.

Remain vigilant for the development of rebound cerebral oedema over the subsequent 72 hours.

- **Consider the use of loop diuretics to reduce brain water content if fluid balance is cumulatively greater than 3 litres positive since admission**:
    - Furosemide 10-20mg qds for one day.

- **Perform a MAP challenge to assess cerebral autoregulation and inform the individualisation of the optimal CPP target and individualisation of ICP threshold during the consultant MDT ward round.**

    - These are **consultant level MDT decisions** dependent on the pattern of injury and the degree of cerebral auto regulation thought to be present.

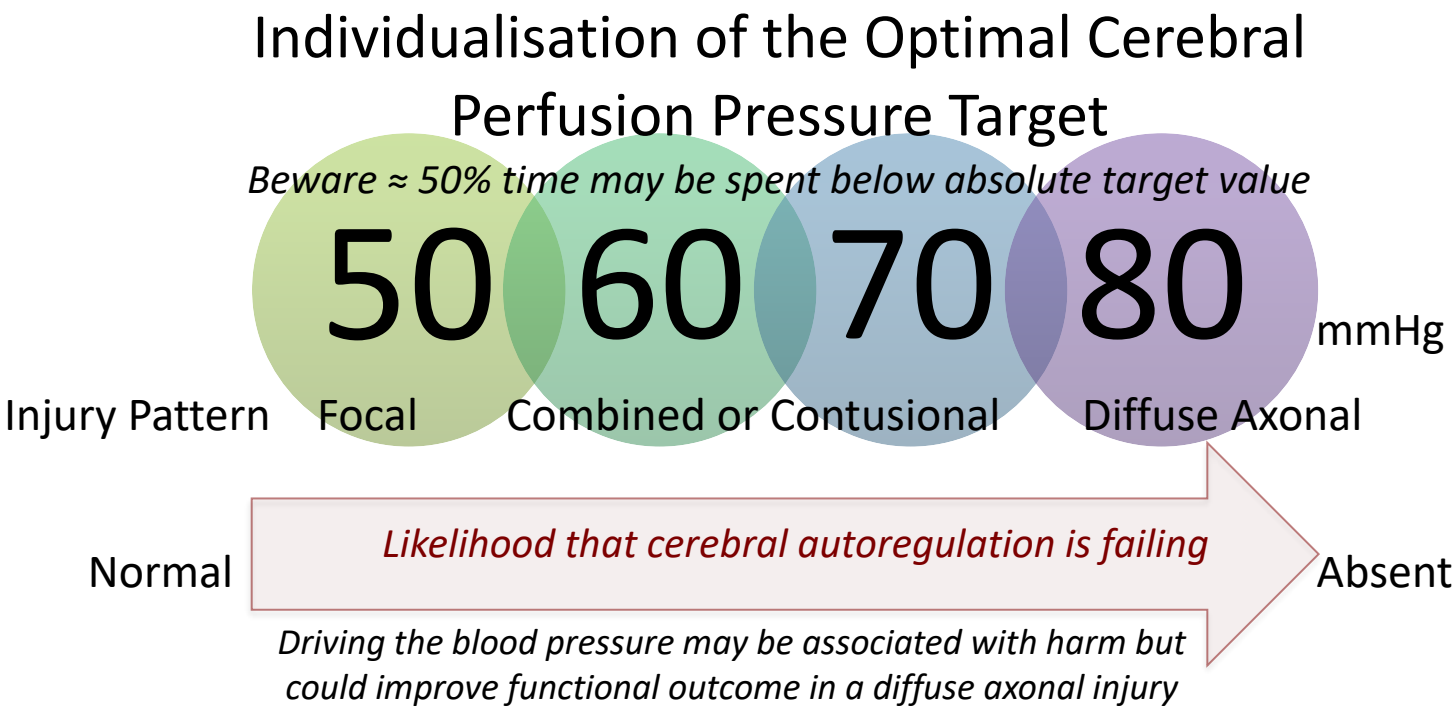
    - A discussion of ICP stability, during vasopressor 'piggybacking', with the bedside nursing staff together with a review of over the last 24 hours on the monitoring can be very informative.

Performing a MAP challenge to assess cerebral autoregulation

This should only be undertaken by a member of the consultant body or senior medical team.

- NO other therapeutic adjustments (i.e. sedation) should be performed during the challenge
- Initiate or titrate up a noradrenaline infusion to increase MAP by 10mmHg for 20 minutes.
- Monitor and record key parameters (MAP,CPP, ICP and the number of unprovoked spikes in ICP) before during and after the challenge.

    - Cerebral autoregulation is more likely to be impaired in patients with midline shift, traumatic subarachnoid haemorrhage or diffuse axonal injury.



- **Consider commencing antihypertensive therapy if the natural CPP ≥110mmHg and a mechanism involving vasogenic oedema postulated.**
  The target should be to reduce MAP by 25%.
  This rare event and is always a **consultant level MDT decision**.

  - 1st line Labetalol 300mg: 60ml 0-20ml/hr
  - 2nd line Clonidine 750mcg: 50ml 0-10ml/hr

- **Consider the induction of ketamine anaesthesia**
  This is a **consultant level decision**.

  - Trial a 2mg/kg IBW bolus of Ketamine to assess if there is any decrease in ICP
  - If a clinically relevant effect (decrease in ICP by 2mmHg or more) occurs consider starting an infusion of 1-2mg/kg/hr IBW (for 70kg patient 23-47ml/hr of 200mg/50ml solution or 10-20ml/hr of 500mg/50ml solution).
  - Any co-administered Alfentanil infusion can be then stopped for the duration of the ketamine infusion. This improves cerebral perfusion pressure independently of any decrease in ICP as the patient's haemodynamic state improves. Subsequently ICP lability will then decrease.

An **ICP ≥20mmHg sustained for 5 minutes** should prompt medical review and further intervention or escalation to a higher level of the protocol. Is a further CT head indicated?

Each intervention to control ICP or maintain CPP should be recorded clearly on the observation chart, e.g. sedation bolus, change in minute volume or escalation in the level of care.

The number of ICP spikes over 24 hours can be visualized via the monitor through the graphical trends menu after selecting ICP as a variable.

The total number of interventions required in the last 12-24 hours can then be used to inform the MDT plan for the next 12-24 hours.

A multidisciplinary consultant level neurocritical care and neurosurgical plan should be clearly documented as to which level three therapies are to be offered if required.

Before escalating to tier THREE therapy it is paramount that all tier ONE and appropriate tier TWO measures are actually in place.

5.5 Tier THREE THERAPY

The evidence base is limited for all tier 3 therapies. All can cause harm.
A considered MDT discussion should be documented to ascertain which therapy may be offered if full tier TWO therapy fails.

Is there adequate venous drainage?
Why has the ICP risen?
Is the CPP maintained above the target value?
Is a repeat CT head indicated?

- **Ensure normothermia has been achieved.**

  This may require the maintenance of central temperature 36-37^{o}C using an appropriate cooling device.
  Remember that intracranial temperature is routinely >1 ^{o}C higher than tympanic temperature and that external cooling technologies may have limited effect when used in conjunction with noradrenaline.

- **Consider a large decompressive craniectomy**.

  - The rapid change in volume obtained by a decompressive craniectomy does not treat the underlying pathological cerebral oedema and therefore should always be combined with non-surgical treatments aimed at a slow and lasting reduction in brain water content.

    - This may include loop diuretics or osmotherapy.
    - Aim for a negative fluid balance of -500-1000ml for the first 72 hours following decompression.
    - Avoid 20% human albumin solution in the first week after injury
    - Remove the head bandage, if present, on return from theatre to ICU. Ensure the no bone flap label is in clear view.

  - Continue sedation for 48-72 hours following decompression, unless brainstem death is strongly suspected i.e. MAP=CPP and pupils are fixed and dilated.
    - The ICP waveform will appear damped and of low amplitude. The absolute ICP value will no longer be a reliable measure of the degree of cerebral injury, look at the trend.
    - Obtain a further head CT or perform transcranial ultrasound at 24-48 hours following decompressive craniectomy to help determine the timing of de-escalation.

- **Consider a barbiturate infusion to control intracranial pressure.**

    - Thiopentone infusion to a target of 50% burst suppression using continuous EEG monitoring.
    - Load with 500mg-1.5g Thiopentone and then start an infusion at 0.5-6mg/kg/hr IBW (2-15ml/hr of 25mg/ml solution for 70kg individual)
    - A progressive reduction in the dose of Thiopentone required to attain 50% burst suppression is expected given the long context sensitive half-life of Thiopentone by infusion.
    - All patients will require advanced cardiovascular monitoring during barbiturate coma. Unpredicted cardiovascular collapse and death can occur in otherwise healthy individuals with raised intracranial pressure.
    - Ventilator-associated pneumonia is common and the threshold for treatment should be lower than in standard practice for the duration of the barbiturate induced coma.
      Clinical Pulmonary Infection Scores do not have an evidence base for use in this setting.

The use of any tier three therapies should be subject to root cause analysis to inform future practice and the development of this protocol.

The RCA should be carried out contemporaneously by the duty senior trainee or consultant present when the decision to undertake a tier three intervention was made. The results should then be forwarded to Dr Naisbitt or Dr Ferris for discussion at neurocritical care governance.

5.6 Osmotherapy and the SOS trial

Osmotherapy is a standard worldwide intervention for neuroworsening, to ameliorate the mass effect of cerebral oedema, but it is unknown whether hypertonic NaCl or Mannitol is the agent of choice and the current evidence base is low grade.

The Salford neurocritical care and neurosurgical team have a position of equipoise and our unit is participating in the NIHR SOS randomised trial to answer the research question comparing equi-osmolar doses of these two agents.

Randomisation packs, stickers for the bedside chart and prescription guidelines are available in the hub offices between PODs A/B and C/D.
In hours, contact the Acute research delivery team and out of hours contact Dr Jay Naisbitt.

Do NOT exclude patients that have received osmotherapy prior to ICU admission. Co-enrolment has been agreed between the SOS trial and ADAPT-sepsis and Hemotion.

Following randomisation the assigned intervention Hypertonic 30% NaCl or 10% Mannitol should be prescribed through EPMAR using “Free text” and by selecting “clinical trial” from the drop down options.

Hypertonic 30% NaCl

30% NaCl can only be given via a CENTRAL LINE.

Consultants (*or senior trainees)* **may give an ideal body weight defined bolus of 30% NaCl (15ml contains 75mmol Na+) using an infusion pump OVER 15 minutes, to establish ICP control.**
Each bolus could increase plasma Na^+ by 2-3mmol/L.
This dose does not account for any ongoing naturesis, free water loss or sodium intake.

One hour after each bolus check the serum Na^+ level.
A rebound increase in ICP within 2 hours requires discussion with consultant.
If serum Na^+ ≥155mmol/L or calculated plasma osmolality ≥320mosmol/L then 30% NaCl is contraindicated.

The use of osmotherapy mandates close attention to the detail of fluid and sodium balance, a minimum of 4-hourly medical review is standard practice.

The falling serum Na^+ must continue to be managed closely to prevent rebound cerebral oedema.

If required a target serum Na^+ 150-155mmol/L can be maintained by infusing 3-5ml/hr 30% NaCl or by scheduled boluses of 30%NaCl every 6 hours and then weaned to a maximum fall of 5mmol/L/24 hours.

10% Mannitol

10% Mannitol can be given via CENTRAL LINE or a PERIPHERAL cannula (18G or larger).

Consultants (*or senior trainees)* **may give an ideal body weight defined bolus of 10% Mannitol using an infusion pump OVER 15 minutes, to establish ICP control.**
Each bolus will increase the plasma osmolality and will be followed by a marked diuresis.

Once a day the plasma osmolality must be measured if plasma osmolality ≥320mosmol/L then further Mannitol is contraindicated.

A rebound increase in ICP within 2 hours requires discussion with consultant.
If serum Na^+ ≥155mmol/L or measured plasma osmolality ≥320mosmol/L then further osmotherapy is contraindicated.

The use of osmotherapy mandates close attention to the detail of fluid and sodium balance, a minimum of 4-hourly medical review is standard practice.

Remain vigilant for the development of rebound cerebral oedema over the subsequent 72 hours.

Osmotherapy should be avoided in patients with long-standing hyponatraemia Na^+ ≤130 mmol/L and used with caution in patients with cardiac or renal problems.
An acute plasma sodium rise of up to 10mmol/L over 24-hours is reported to be safe.
Pontine demyelination could occur if the serum Na^+ falls by >0.5mmol/L an hour.
Marked falls in serum Na^+ (>5mmol/L/24hours) in the context of traumatic brain injury are to be avoided and may be associated with rebound cerebral oedema.

Untreated or undertreated diabetes insipidus contraindicates the use of hypertonic NaCl.

Remain vigilant for new biochemical evidence of acute kidney injury.

A number of commonly infused solutions also contain a high Na+ content:

Solution Name	Na+ content mmol/L
0.9% NaCl	154
Plasmalyte-148	140
Phosphate polyfusor	162
4.5% human albumin solution	160
20% salt poor HAS	145
8.4% $NaHCO_3$	1 mmol/ml
30% NaCl	5 mmol/ml

5.7 Cranial Diabetes Insipidus

Cranial Diabetes Insipidus is characterised by a decreased secretion of ADH. This results in polyuria by diminishing the patient's ability to concentrate urine. It is a common, although usually transient, complication of traumatic brain injury or neurosurgical procedures performed in the sellar and suprasellar region.
Polyuria occurs, up to 18L a day, resulting in a rapid rise in plasma osmolality as body stores of free water are lost.
The diagnosis of diabetes insipidus (DI) is often made clinically, whilst the laboratory tests provide confirmation after a few hours.
If urine output >200ml/h for 2 consecutive hours then DI should be suspected and the pathway below followed:

1. Send simultaneous plasma and urine osmolalities and measure urine specific gravity
2. Start a Plasmalyte-148 infusion at an input rate to match the previous hour's urine output
3. Measure plasma Na^+ using ABG hourly
4. Rule out secondary causes of polyuria *(Diabetes mellitus, physiological excretion of excess resuscitation fluid or as a result of an intentional osmotic diuresis- post Mannitol)*
5. If plasma Na^+ is rising by ≥2mmol/L/h give a STAT bolus of DDAVP 0.5micrograms IV before the laboratory confirmation of DI
6. Only if plasma Na^+ ≥160mmol/L AND in the presence of a devastating brain injury or suspected brain death, then start additional hypotonic fluid (5% glucose at 100ml/h or enteral water) to aim to lower plasma Na+ by 0.5mmol/L/h and titrate this hypotonic infusion rate to effect.
7. If the diuresis worsens or recurs 4 hours after a DDAVP bolus then give a repeat bolus of 1microgram IV and then start a continuous infusion of DDAVP as per critical care order set (neuro). The DDAVP infusion may be required for up to 6 days.
8. If the laboratory confirms DI (**Urine osmolality<Plasma osmolality)** but plasma Na^+ remains unchanged or has risen by ≤1mmol/L/h, then continue to match fluid input with urine output and seek consultant advice.
9. Stop any input/output matching Plasmalyte-148 infusion once the urine output is reliably ≤100ml/h

A urine specific gravity of 1.005 or less and a urine osmolality less than 200 mosmol/kg is the hallmark of diabetes insipidus.
Random plasma osmolality is generally ≥287 mosmol/kg.

Be vigilant for the risk of rebound cerebral oedema following DDAVP administration and do not administer further hypotonic fluid.

5.8 Troubleshooting: The approach to an ICP >22mmHg for 5 minutes in a deteriorating patient

The approach to ICP >22mmHg for 5 minutes

Examine the patient

Why has the ICP risen? Think about venous drainage.

Remember >1.5l/min blood must be able to drain from the head

Ensure the ETT ties are loose

Can you fit two fingers comfortably under the tie?

Head up to 30 degrees

Is the head in neutral alignment? (see pictures opposite)

Remove all collars but maintain spinal precautions

Trial head in slightly flexed position if no neck injury is present

Make sure the ICP monitor is functioning. Check the ICP trend and trace

Is it an acute spike or this part of a trend?

If there is an EVD in situ, is it open? Is it oscillating?

Is the EVD zeroed to the tragus?

Are they adequately sedated? (RASS -5)

Check ventilation; aim $PaCO_2$ (4-5kPa); Ensure SpO_2 94-98%

Avoid high airway pressures and beware of breath stacking

Is CPP above the target set by MDT ward round?

Is the Arterial transducer zeroed to the level of the tragus?

Is there any evidence of seizure activity?

Check the temperature – if pyrexial consider cooling measures

Registrar review. Why has the ICP risen?

Bolus the sedation with 5-20mg Midazolam

CPP must be maintained above the target

Ensure adequate cardiac output

If ICP is refractory to sedation boluses consider neuromuscular blockade

Refer to the tiered protocol

Communicate the situation to the neurosurgical team. Is a further CT needed?

Consider osmotherapy prior to transfer.

Parkin, Wallace, Wright

1. If there are new pupillary changes or a new focal motor deficit has developed or if there has been any drop in motor component of the GCS, repeat the CT head urgently.

2. **Ensure the head is in an aligned neutral position.**

3. Make sure the head of the bed is tilted up 30 degrees, with the Arterial line transducer zeroed to the tragus of the ear? Any more than 45 degrees head up may cause the head to flex or extend at the craniocervical junction causing impaired venous drainage.

4. Is the ICP waveform indicative of raised ICP? If not is it working?

5. Is the patient adequated sedated?
   Bolus up to 20mg Midazolam but ensure the patient remains haemodynamically stable with adequate CPP.

6. Could they be seizing?
   Non-convulsive status is reported to be present in up to 50% of TBI patients.
   Consider loading with Valproate.
   Always clearly document the reason why antiepileptics were started.

7. Ensure ventilatory targets are met SpO2 94-98% and PaCO2 4-5kPa.
   Check the ETT is patent and has not migrated out or is endobronchial.
   Avoid high plateau airway pressures and beware of breath stacking.

8. Is there adequate cerebral blood flow?
   Ensure CPP is maintained greater than 60mmHg.
   Remember with normal autoregulation an increase CPP should decrease ICP.
   Do not use more than 10ml/hr 4:50 Noradrenaline without consultant approval and consideration of cardiac output monitoring.

9. Is there adequate venous drainage?
   Remember more than 1.5l blood/min needs to drain from the head:
   Systematically check ETT ties are not compressing venous outflow
   Remove all collars, but maintain spinal precautions if indicated
   Check the intra-abdominal compartment pressure.
   Constipation can cause raised ICP.

10. What is the patient's temperature?
    If pyrexial cool and target normothermia; place ice packs in the patients hands; administer paracetamol and investigate and treat infection if present; Place the external cooling cap on the patient; If all the above has not worked institute invasive cooling. Administer cold fluid only if fluid therapy is indicated for another cause.

If all of the above has been optimised the patient may require a further CT.
Bolus therapy may be required for ICP management in CT scan:

- Give up to 20mg Midazolam
- consider a dose of osmotherapy prior to transferring to CT
- incremental 125mg Thiopentone may be used to reduce ICP transiently but has no evidence base for routine practice.

11. **Communicate the situation to the Critical Care consultant and the neurosurgical team.**
    Consider evacuation of any mass lesion or drainage of CSF via an EVD.
    Refer to the tiered protocol.

5.9 Stepdown Protocol

The early wake up test

Traumatic brain injury patients who have been neurologically stable requiring no intervention for raised intracranial pressure should undergo a wake-up test at 24 hours to assess their neurology formally.

If the initial GCS is less than 5 or if there is evidence of basal cistern effacement, midline shift >5mm or cortical sulcal effacement on the most recent CT, then the wake-up test should be deferred until the MDT consensus opinion agrees to a sedation hold.

Stepdown Protocol

Beginning the de-escalation of therapy to control ICP or changing the ICP threshold:

The number of interventions to control the ICP or maintain adequate CPP over the last 12-24 hours should be presented on each MDT ward round. This figure then informs the decision whether to perform a sedation hold or alter the ICP threshold for intervention, in conjunction with other clinical observations and monitoring.

The bedside nurse should take part in this discussion and MUST be present throughout any subsequent sedation hold or reduction.

Always consider the natural history of each patients individual injury pattern. The peak swelling in a contusional injury may be greater than 72 hours post injury.

Do not undertake a sedation hold in a period of predicted peak swelling.

After 72 hours of ICP stability it is appropriate to remove the ICP probe.

Algorithm for the de-escalation of ICP therapy, changing the ICP threshold or deciding on the appropriateness of a sedation hold.
Consultant MDT input is mandatory.

DO NOT de-escalate ICP therapy or attempt a sedation hold if:
any ICP intervention has been required in last 12 hours
Escalation in level of therapy in last 24 hours
Abnormal ICP waveform P_2>P_1 Appendix 3
Worsening neurological examination or pupil abnormality in last 24 hours
Sustained rise in ICP on stimulation for ≥1 minute requiring a sedation bolus e.g. turns and ETT suctioning
Worsening CT appearances:
- *Midline shift ≥5mm*
- *Absent basal cisterns*
- *More cortical sulcal effacement*

De-escalate ICP therapy or reduce sedation if:
Less than 2 interventions in last 24 hours
Bilateral slowly reactive normal sized pupils
Stable motor score within GCS
P_1≥P_2>P_3 ICP waveform
On stimulation or coughing the ICP spontaneously falls back to less than threshold within 1 minute
ICP has spontaneously trended down over last 24 hours
Relaxing the $PaCO_2$ goal is well tolerated
Stable CT abnormalities

Sedation hold or change ICP threshold if:
Improving neurological examination
Normal pupils
$M_{5/6}$ on GCS when sedation reduced
Normal ICP waveform
On stimulation or coughing the ICP rapidly returns to less than threshold
No interventions to control ICP required over the last 24 hours
A CT scan is not compatible with raised ICP

5.10 Management of the agitated waking TBI patient

This can be one of the most challenging things to manage in neurocritical care.
It is a cause of considerable anxiety across the MDT but is a normal part of the job, all of us will care for many agitated patients each year.

Impaired cognitive function is expected following a Traumatic Brain Injury.

The combination of cognitive impairment, emotional instability and a lowered stress threshold to environmental change manifest themselves very commonly as agitated delirium.

Typical features include behaviours, which are repetitive and non-purposeful, are often inappropriate or excessive, and can involve restlessness, aggression, and disinhibition.

The anatomical site of traumatic brain injury can be predictive of different patterns of agitation. Frontal injuries may cause aggression; Temporal injury can lead to distressing memory loss; Deep brain injuries will cause emotional instability and unmask fear and anxiety.

New onset delirium should always be investigated appropriately:

- Drug withdrawal (pain medications, other meds from ICU)
- Sepsis
- Electrolyte disturbance
- Alcohol substance withdrawal
- Seizure disorders.
- Neuroendocrine dysfunction

Increasing agitation can be an early sign of deterioration.
Prolonged chemical restraint and sedation is harmful.

Hypoxia is a very potent cause of agitation.

Use the Richmond Agitation and Sedation Scale to aid in the approach to the agitated waking patient.

If a patient has a RASS +2, all of the guidance applies +1 to +2.
If a patient has a RASS +3, all of the guidance applies +1 to +3 e.t.c.

RASS +1 *Restless* *Anxious but movements are not aggressive or vigorous*

The first line always involves simple measures:
Speak slowly and calmly in a low volume, one person at a time.
Introduce yourself, shake hands with the patient.
Use non-confrontation body language
Never show aggression, argue or look for conflict with the patient
Limit the amount of direct eye contact
Frequently re-orientate the patient
Educate the family how to re-orientate the patient.
Give the patient tubing to handle or provide an appropriate toy.
Obtain any hearing devices or vision aids from home to improve orientation
Minimise noise or start their preferred music therapy.
Maintain nursing continuity if possible with a consistent schedule and staff
Try to create a familiar environment:
Allow family to bring in personal possessions and photographs
Reinstitute normal circadian variation in ambient light levels.
Reduce stimuli: Light, noise, distractions (especially at night), place patient in bed, draw curtains, turn off television, etc.
Monitor sleep cycle and sleep quality, consider use of Melatonin
Limit the number of visitors and staff in the bed area at one time
Help with inattention and psychological management by focusing on sequencing and staying on task.

Provide adequate analgesia.

Any distortion or irritation of the dura, subarachnoid haemorrhage or late post-traumatic hydrocephalus can cause pain.
Is there an extracranial missed injury or fracture?
Use simple analgesia with Paracetamol +/- opiate.

Ensure patient is not in gastrointestinal distress with constipation or an ileus and is on the appropriate laxative agents using the NCA critical care bowel management guideline.

Propanolol 40-60mg tds PO/NG is the only evidence based medicine to reduce aggression and agitation following TBI. In patients with frontal lobe injury and no contraindication for β-blockade, it should be started prophylactically for a 7 day course.

RASS +2 ***Agitated*** *frequently, no purposeful movements*

Perform a verbal risk assessment: enlist senior nursing support from POD lead nurse or shift leader and the duty medical senior registrar or consultant.

The safety of patients and staff are paramount at all times.
Plan a strategy and act before peak agitation is reached.

Remove invasive monitoring as soon as appropriate.
Retain peripheral IV access if at all possible but disconnect infusions.
Cover up invasive lines, out of sight, out of mind (even a PEG can be covered with an abdominal binder)
Avoid repeated NG removal and insertion overnight but replace in the morning.
Do not stand within an arms reach or linger in a position in which you could be struck.
Position yourself at the hip of the patient, not the foot or head of the bed.
Hold their hand in a non-threatening manner
Slowly and calmly in a low volume voice reassure the patient they are safe remember they are afraid.
Ensure breaks are adequately covered- safe staffing arrangements use the whole MDT.
Timed toileting

RASS +3 ***Very agitated*** *Pulls or removes invasive lines tubes etc, aggressive*

Special nursing.
Consider Padded hand mittens.
In an exception consider Nursing the patient on multiple mattresses on the floor.
Move into side room if one is available (not 12).
Always maintain patient dignity but ensure that another member of staff can visualize your position in the bed area.

Subclinical epilepsy can present as intermittent aggression.

Propanolol 40-60mg tds PO/NG is the only evidence based medicine to reduce aggression and agitation following TBI.

With good care often even very agitated patients can be managed in a safe way by riding out the period of peak agitation.

RASS +4 *Combative* *Overly combative, violent, of immediate danger to staff or themselves*

Continue to speak slowly in a calm respectful low volume voice.
Any verbal abuse, threat or violence is not personal.

Pharmacological management of excessive agitation

The first line chemical therapy is a Benzodiazepine:

Lorazepam 1-2mg IV or IM
Midazolam 5mg IV or IM
Give IV wherever possible, remember that IM absorption is variable and may take 10-15 minutes to have any effect. The peak effect may be much later.

Seek senior medical review if a single dose does not control the agitation.

Clonidine can be effective in those withdrawing from substances.
Bolus 150-300mcg IV over 10-15min then start an infusion 0.1-2mcg/kg/hr IBW titrated to effect (0-10ml/hr of 750mcg/50ml solution).
As clonidine has a long half-life, infusion may take some time to reach steady state and therefore repeat the bolus as necessary.
Once control is obtained, clonidine can be changed to enteral route and weaned.

Olanzapine can be of value in selected patients.
5-10mg IM/IV repeated 5-10mg after 2 hours maximum 20mg in 24 hours
If effective consider regular oral dosing.

Avoid polypharmacy, often a patient with a RASS +4 can be managed in a safe way by riding out the period of peak agitation.

NEVER EVER give haloperidol or droperidol to patients with TBI, it causes increased long-term cognitive dysfunction and delays cognitive recovery in neurorehabilitation.

Remember excessive sedation may result in significant respiratory depression.
This could in turn lead to further preventable secondary brain injury.
In addition to constant nursing observation, hourly medical observation is mandatory for 6 hours following any chemical sedation.

If GCS falls below an absolute value of 13 then there should be immediate senior medical review.

Always plan future strategy to manage agitation once control has been achieved.

Intubation and ventilation is may be required to ensure patient safety across the critical care unit.

Remember prolonged chemical sedation and restraint is harmful and will delay recovery.

5.11 Paraoxysmal Sympathetic Hypereactivity or PAID syndrome

As a consequence of severe brain injury and axonal disruption, a non-noxious stimuli may be perceived as noxious.

Following the stimulus, which could be minor e.g. positional changes, suctioning or washing the patient, often there is impaired descending inhibition resulting in increased excitatory interneuronal cord activity and increased sympathetic output often accompanied by increased abnormal motor tone or dystonia.

PSH can be diagnosed using an assessment method (PSH-AM):

A Clinical feature scale (CFS) score

	0	1	2	3
Heart rate (beats per min)	<100	100–119	120–139	≥140
Respiratory rate (breaths per min)	<18	18–23	24–29	≥30
Systolic blood pressure (mm Hg)	<140	140–159	160–179	≥180
Temperature (°C)	<37·0	37·0–37·9	38·0–38·9	≥39·0
Sweating	Absent	Mild	Moderate	Severe
Posturing during episodes	Absent	Mild	Moderate	Severe

B Diagnosis likelihood tool (DLT): one point per feature present

Antecedent acquired brain injury
Clinical features occur simultaneously
Episodes are paroxysmal in nature
Sympathetic over-reactivity to normally non-noxious stimuli
Absence of parasympathetic features during episodes
Features persist for >3 consecutive days
Features persist for >2 weeks post-brain injury
Two or more episodes daily
Absence of other presumed causes of features
Features persist despite treatment of alternative differential diagnoses
Medication administered to decrease sympathetic features

C Interpretation of scores

- **CFS subtotal=**
  sum of CFS scores for each of the six features (0–3 points for individual features; maximum subtotal=18);
  CFS subtotal severity scores:
  0=nil; 1–6=mild; 7–12=moderate; ≥13=severe
- **DLT subtotal=**
  sum of points for each feature present (one point per feature; maximum subtotal=11)
- **PSH-AM=**
  CFS subtotal + DLT subtotal;
  PSH-AM score:
  <8=PSH unlikely; 8–16=PSH possible; ≥17=PSH probable

If PSM is probable then treatment should be commenced to minimise distressing symptoms and contractures associated with dystonia.

Even with regular treatment after 2-4 weeks the dystonia can be severe and persistent. It is often resistant to neuromuscular blockade and the intubation of these patients can be difficult.

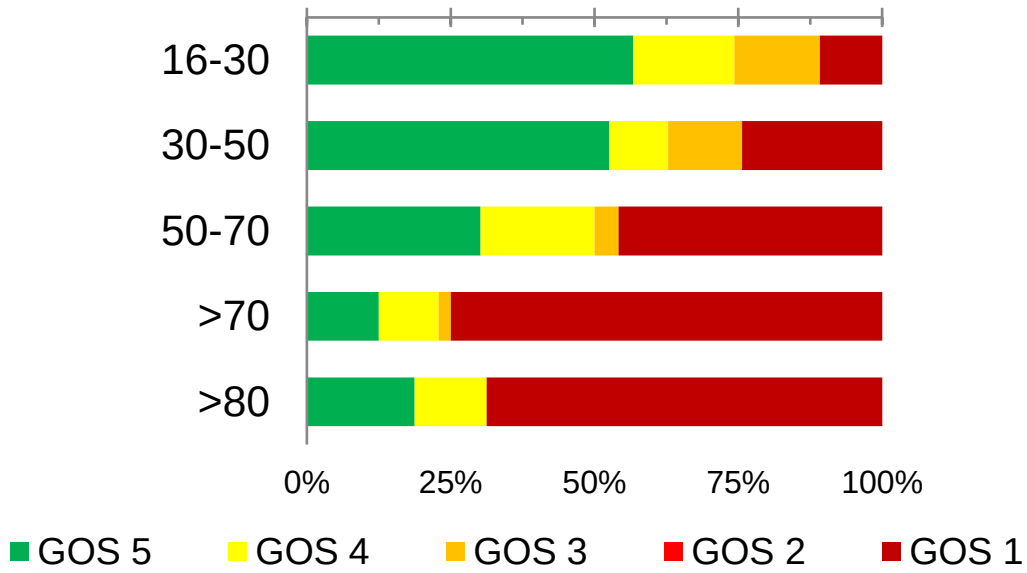
1st Line treatment:	Propranolol 20-60mg qds enterally Clonidine 100mcg tds enterally (gradually titrated to clinical effect up to maximum of 1200mcg over 24 hours)
2nd Line treatment:	Gabapentin 100mg tds (gradually titrated to clinical effect up to maximum of 4800mg over 24 hours)
3rd line treatment:	Baclofen 5mg tds enterally
4th Line treatment:	Morphine and midazolam titrated to effect

Advice should be sought from the neurorehabilitation team or spasticity MDT ward round regarding weaning from therapies.

5.12 Prognostication following TBI and care of the patient with a devastating brain injury (DBI)

The 6-month functional outcomes (Glasgow Outcome Scale) in TBI patients are audited biannually.

The impact of Age on functional outcome 2019-20



The above graph illustrates the outcomes from 2019-20 audit in all patients with severe TBI.

Glasgow Outcome Score 5	Good neurological recovery
Glasgow Outcome Score 4	Minor disability
Glasgow Outcome Score 3	Severe disability

Glasgow Outcome Score 2 Persistent vegetative state

Glasgow Outcome Score 1 Death

Fixed unreactive pupils on admission do not always equate to death (In 2015-16 10% of these patients survived to a Glasgow Outcome Score of 5- all had emergency evacuation of an extradural haematoma).

Traumatic brain injury is a process not an event.

Prognostication following isolated severe traumatic brain injury is difficult.

The premise of conversations early in the disease process should be uncertainty.

The natural history of the process of recovery from brain injury demonstrates that recovery is not fixed for at least 2 years, but that the majority of recovery is seen within the first 3 months following injury.

Concordance between the multidisciplinary opinion of the predicted outcome, multiple investigations (CT, EEG +/- MR) and the clinical condition should be sought. If in doubt about the outcome, give the patient more time for further clinical observation and prognostication as required.

The advocacy of the next-of-kin is important in ascertaining how acceptable an individual outcome may be to the patient.

In a large observational cohort series, controlling for age, sex and TBI type, the independent factors associated with recovery of consciousness by the end of rehabilitation were the absence of intraventricular haemorrhage and the absence of residual intracranial mass effect.

All decision making should be taken in the patient's best interests.

Treatment may be withheld or withdrawn in accordance with GMC guidance.

It is crucial that the terminology used by the MDT is consistent.

The term devastating brain injury should be used rather than the term 'un-survivable or not surgical treatable' which may mean different things to different members of the MDT.

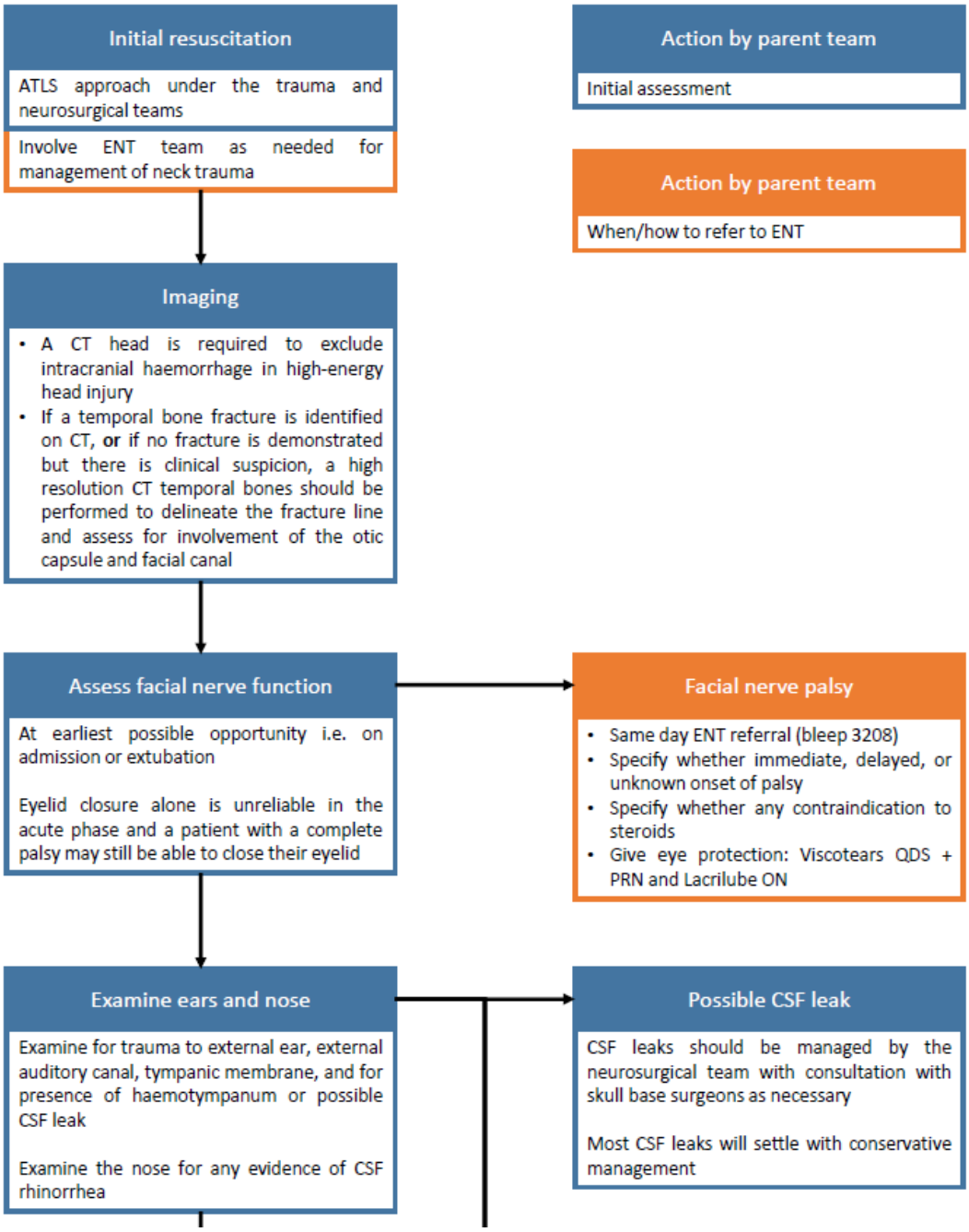
Clarify the meaning of any language used by other members of the MDT, in the notes or with the next-of-kin, on admission to critical care.

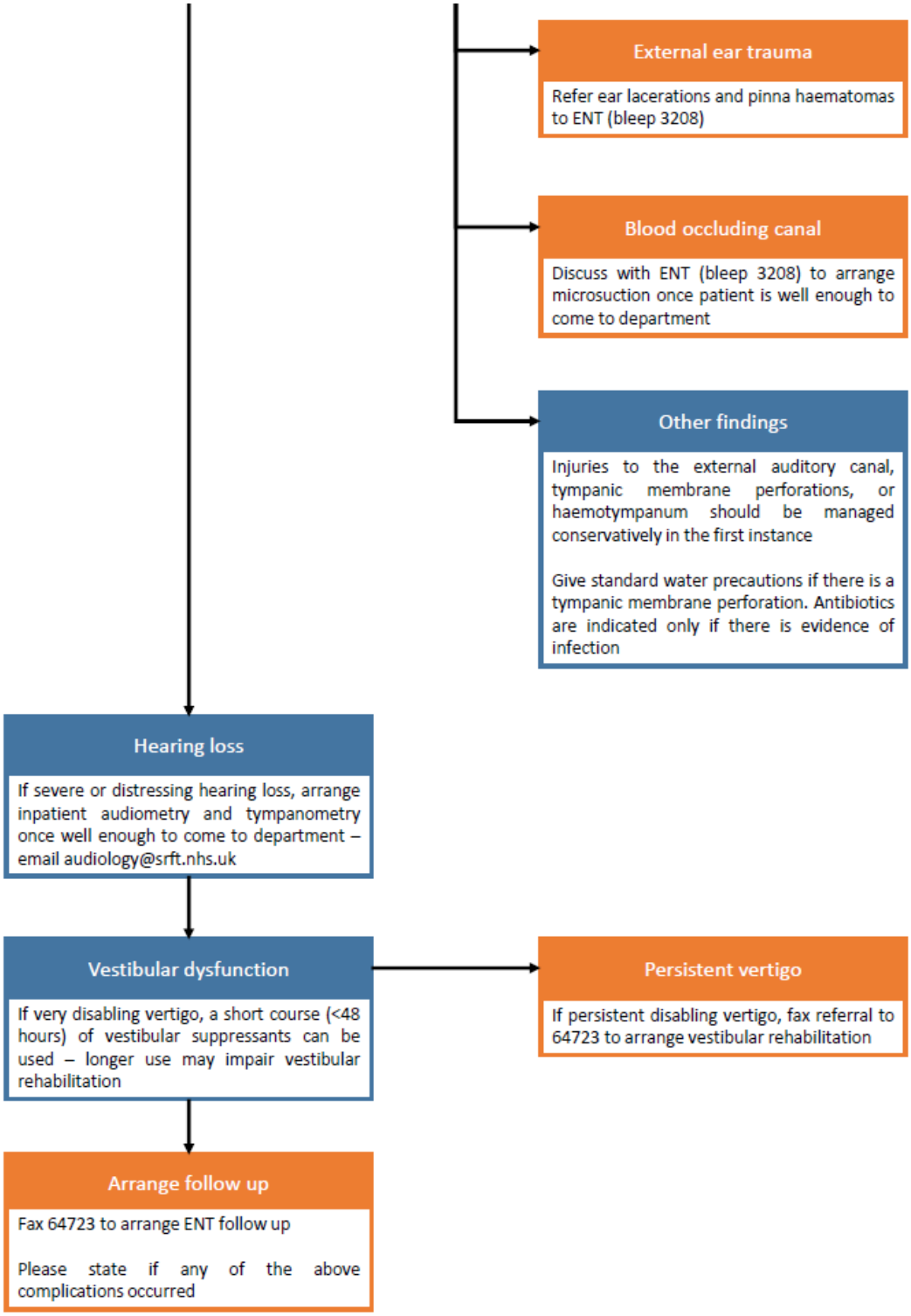
Current FICM guidance should be used in the care and prognostication of patients admitted with a presumed devastating brain injury.

Next of kin support can be accessed by early referral to the specialist nursing team.

5.13 ENT follow-up of temporal bone fractures

Management of suspected or proven temporal bone fractures




External ear trauma
Refer ear lacerations and pinna haematomas to ENT (bleep 3208)
Blood occluding canal
Discuss with ENT (bleep 3208) to arrange microsuction once patient is well enough to come to department
Other findings
Injuries to the external auditory canal, tympanic membrane perforations, or haemotympanum should be managed conservatively in the first instance
Give standard water precautions if there is a tympanic membrane perforation. Antibiotics are indicated only if there is evidence of infection
Hearing loss
If severe or distressing hearing loss, arrange inpatient audiometry and tympanometry once well enough to come to department – email audiology@srft.nhs.uk
Vestibular dysfunction
If very disabling vertigo, a short course (<48 hours) of vestibular suppressants can be used – longer use may impair vestibular rehabilitation
Persistent vertigo
If persistent disabling vertigo, fax referral to 64723 to arrange vestibular rehabilitation
Arrange follow up
Fax 64723 to arrange ENT follow up
Please state if any of the above complications occurred

6. Roles & responsibilities

Critical care neurogovernance group –

1. Ensure staff involved are educated about new clinical guideline and implications for practice

2. Ensure standards set out are audited and results fed back to critical care

7. Monitoring document effectiveness

1. Patients should be reviewed within 12 hours of admission to critical care by the critical care consultant and the neurosurgical consultant (ICS guidance)

2. Invasive arterial blood pressure monitoring and intracranial pressure monitoring should be instituted within 2 hours of commencing tier one therapy

3. A multidisciplinary neurocritical care and neurosurgical plan should be clearly documented. This should include the tier and choice of therapy to be offered if needed in the next 12-24 hours.

4. A multidisciplinary consultant level neurocritical care and neurosurgical plan should be clearly documented as to which tier three therapies are to be offered if required.

5. The guideline processes and individual patient functional outcomes are audited biannually and then presented through the neurocritical care governance group.

8. Abbreviations and definitions

List all abbreviations or acronyms in alphabetical order (even if they are explained within the document as well), for example:

NCA	Northern Care Alliance
NICE	National Institute for Health and Care Excellence

9. References

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https://www.ficm.ac.uk/sites/default/files/dbi-consensus-statement-2018.pdf

10. Appendices

Appendix 1

Richmond Agitation Sedation Scale (RASS) *

Score	Term	Description	
+4	Combative	Overtly combative, violent, immediate danger to staff	
+3	Very agitated	Pulls or removes tube(s) or catheter(s); aggressive	
+2	Agitated	Frequent non-purposeful movement, fights ventilator	
+1	Restless	Anxious but movements not aggressive vigorous	
0	Alert and calm		
-1	Drowsy	Not fully alert, but has sustained awakening (eye-opening/eye contact) to *voice* **(≥10 seconds)**	Verbal Stimulation
-2	Light sedation	Briefly awakens with eye contact to *voice* **(<10 seconds)**	Verbal Stimulation
-3	Moderate sedation	Movement or eye opening to *voice* **(but no eye contact)**	Verbal Stimulation
-4	Deep sedation	No response to voice, but movement or eye opening to *physical* stimulation	Physical Stimulation
-5	Unarousable	No response to *voice or physical* stimulation	Physical Stimulation

Procedure for RASS Assessment

1. Observe patient
   a. Patient is alert, restless, or agitated. **(score 0 to +4)**
2. If not alert, state patient's name and *say* to open eyes and look at speaker.
   b. Patient awakens with sustained eye opening and eye contact. **(score –1)**
   c. Patient awakens with eye opening and eye contact, but not sustained. **(score –2)**
   d. Patient has any movement in response to voice but no eye contact. **(score –3)**
3. When no response to verbal stimulation, physically stimulate patient by shaking shoulder and/or rubbing sternum.
   e. Patient has any movement to physical stimulation. **(score –4)**
   f. Patient has no response to any stimulation. **(score –5)**

* Sessler CN, Gosnell M, Grap MJ, Brophy GT, O'Neal PV, Keane KA et al. The Richmond Agitation-Sedation Scale: validity and reliability in adult intensive care patients. Am J Respir Crit Care Med 2002; 166:1338-1344.

* Ely EW, Truman B, Shintani A, Thomason JWW, Wheeler AP, Gordon S et al. Monitoring sedation status over time in ICU patients: the reliability and validity of the Richmond Agitation Sedation Scale (RASS). JAMA 2003; 289:2983-2991.

Appendix 2 Sticker for observation charts

TBI + ICP monitor: be cl**EAR**

30 head-up/bed-tilt

Art line zeroed at tragus of ear

Head neutral position

TARGET MAP _____ TARGET CPP_____

PLEASE COMPLETE THE TABLE BELOW WHEN THERE IS A SPIKE IN ICP ABOVE THE ACCEPTED LIMIT.(GREATER THAN 22mmHG)

TIME	ICP	TRIGGER IF APPLICABLE	INTERVENTION	WORKED Y/N?
9.20	*35*	*T*	*2P*	*Y - ICP 15*

Key

Trigger: C (cough) T (Turn) S (sedation reduction) U (unknown)

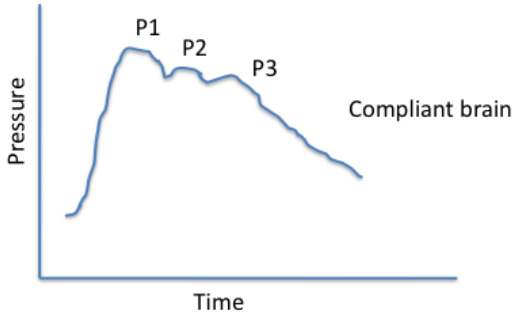
Intervention - Number of mls + P = propfol, M – Midazolam A- Alfentanil. e.g 2P.

Pos = position change, Vent = ventilation change, Sod = 30% sodium

Appendix 3

The ICP waveform has three components: pulse, respiratory and slow waves.

The pulse component of a normal ICP waveform generally consists of three peaks, decreasing in height to correlate with the arterial pressure waveform occurring with each cardiac cycle. These pulse waves represent arterial pulsations in large cerebral vessels as they produce a fluctuation in the volume within the ventricles.



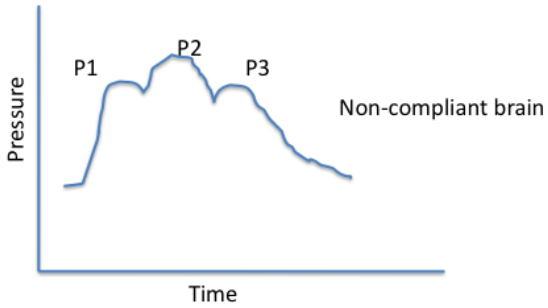
P1 the first and sharpest peak is called the percussive wave and results from the arterial pressure being transmitted from the choroid plexus. Arterial hypotension and hypertension will decrease or increase the amplitude of P1 respectively.

P2 the second peak referred to as the tidal wave varies in amplitude with brain compliance and ends on the dicrotic notch

P3 represents the dicrotic wave and is caused by closure of the aortic valve

The ICP waveform also has a slower pattern reflecting changes in intrathoracic pressure associated with respiration. This respiratory waveform generally cycles about 8-20 times per minute.

Analysis of the ICP waveform begins with an understanding of its shape and amplitude.
The shape of the ICP waveform resembles the shape of the arterial waveform.
The amplitude varies with changes in physiological state and is influenced by changes in intracranial compliance and cerebral blood flow.



As the ICP increases due to an excess of components within the cranial vault, the amplitude of all the components increase. If the ICP continues to rise, P2 becomes more elevated than P1

until eventually P1 may disappear within the waveform. This signifies a decrease in intracranial compliance and may warrant intervention. Amplitude increases as intracranial compliance falls, this may be evident prior to the actual elevation in ICP. Elevation of P2 can also indicate the patient will have a rise in ICP on stimulation.

Conditions resulting in a constriction of cerebral blood vessels, as seen with hypocapnia or vasospasm, will exhibit a decrease in the amplitude of the waveform whereas severe hypercapnia and hypoxia will exhibit an increase in amplitude with an inability to distinguish the individual waves due to a rounding appearance of the waveform.

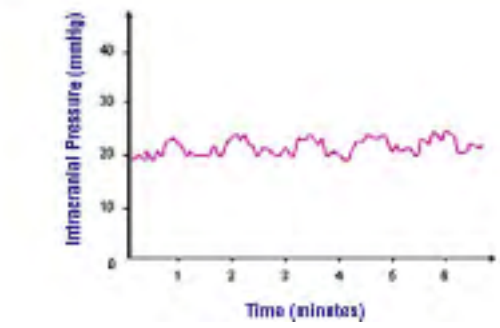
Patients who have undergone a craniectomy will have a dampened waveform.

When ICP is elevated and there is a decrease in intracranial compliance, pathological slow waves may appear. Lundberg described these as A, B and C waves. These waveforms are hard to distinguish because the sweep speed of the monitor is too fast to demonstrate them. Our monitors display the mean ICP value.

Lundberg A wave

A waves or plateau waves are characteristic of conditions that create low intracranial compliance and result from a pathological vasodilation of cerebral blood vessels as the brain stem responds to a decrease in cerebral perfusion pressure. As the ICP increases the A waves reflect steep increases in this pressure ≥50mmHg , lasting as long as 5-20 minutes before rapidly declining. They have been associated with poor outcomes related to cerebral hypoxia, ischaemia, infarction or impending herniation. The presence of these waves should prompt urgent treatment of ICP.

Lundberg B wave



B waves or pressure waves are of less clinical significance but are characterized as intermittent pathological waves whose amplitude sharply rises to between 20-50mmHg and fall every 1-2 minutes depending on changes in cerebral blood volume seen with decreased compliance. These waves can be seen with Cheyne-Stokes breathing pattern or during periods of apnoea

and may precede the development of A waves. They indicate a need to treat an elevated ICP or that the CPP is at the lower limit of autoregulation and needs to be increased .

Lundberg C wave

C waves are not thought to be of clinical significance and may be due to cyclical interactions between arterial blood pressure and respiration. They last ≤10 seconds and have pressures ≤20mmHg.

Appendix 4

Cooling can be achieved by a number of methods determined by consultant preference and the availability of equipment.

a. Surface cooling with damp towels/sheets, cool bathing and cool packs in axillae/groins
   This method is the simplest although also the least effective. Its use should be reserved for when other methods are unavailable
b. Cool water blankets
   We have an automated surface cooling machine; Blanketrol III, which is kept in the storage room (A3232) just past the middle corridor. This machine should be used in conjunction with the Maxitherm blankets found in the same storage room. This machine uses feedback from a patient temperature probe attached to the machine to maintain the desired temperature.
   Always utilise the cooling cap/hat first as body surface cooling alone can paradoxically cause brain temperature to rise as hot blood is shunted into central compartments. This effect can be compounded by noradrenaline use.
c. Intravenous cooling device
   This method is the most invasive, although also the most effective. We have two 'Coolgard 3000' machines available in the furniture store at the front of pods A&B (Room A3108).
   The instructions for how to set the machine up are attached to the front of the machine. (see below)
   In the first instance a dedicated cooling central venous catheter needs to be inserted, ideally into one of the femoral veins. These Alsius 'ICY' intravascular heat exchange catheters are found in the storage room near to the AB/CD dividing corridor.
   A dedicated core temperature monitor is required, which should be attached to the machine again to provide feedback to maintain the desired temperature.
d. If the patient is hypovolaemic then cold crystalloid can be infused.

Sedation/shivering: Shivering is not inherently dangerous. It may be tolerated if ventilation is not impaired. Shivering may, however, increase heat production and increase oxygen consumption by 40 – 100%. Shivering tends to be a particular problem on induction of cooling. It can be managed in the following stepwise manner:

a. Try gentle surface warming with a forced air mattress (obviously only if IV cooling is being utilised). This can sometimes prevent shivering while not interfering with core cooling.
b. If not already utilised, add an opioid agent for sedation
c. Ensure adequate sedation, if necessary add midazolam
d. Clonidine can be considered if haemodynamic status allows
e. If all these measures fail, neuromuscular blockade can be utilised. Once the target temperature is reached neuromuscular blockade can usually be discontinued.

Appendix 5

This is the suggested framework for the RCA.

Time to consultant review
MDT documentation
Could communication have been improved at any point?
Were the plans documented and followed through?
Where there any delays in care or in transfer to theatre?
Were the ventilation targets met?
Were the ICP or CPP targets met?
Why did ICP ultimately increase?
What was the number of interventions in each 24 hour period
Did we utilise flow monitoring?
Osmotherapy use. Did the serum Na+ fluctuate wildly?
Review the fluid balance from admission to use of the tier 3 therapy.
Was normothermia achieved?

An RCA should be carried out contemporaneously by the duty senior trainee or consultant present when the decision to undertake a tier three intervention was made. The results should then be forwarded to Dr Naisbitt or Dr Ferris for discussion at neurocritical care governance.

Appendix 6

Good neurological observations are a key part of quality neurocritical care.
They should be performed by a trained nurse, hourly for 8 hours then de-escalated to 2-hourly for a further 8 hours and then performed at least 4-hourly.
A trained nurse is defined as one who has completed their mandatory training on neurological observations.

1. Measure the GCS

The Glasgow Coma Scale is the most validated method of clinically assessing and tracking injury severity. The following video link demonstrates how to perform a GCS assessment:

http://www.glasgowcomascale.org/

It is never sufficient to only write sedated across the GCS section of the observation chart.
If a GCS component cannot be tested record NT on the chart.

2. Record the motor function and assess the tone of each limb

Remember the motor component of the GCS uses the best limb response.
Any new motor deficit is a cause for concern and may indicate a herniation syndrome is present.

Is the limb flaccid (floppy)? Is the limb spastic (stiff)?

3. Examine the brainstem functions (especially in the sedated patient):

The brainstem is the most important area of the brain.
Any change in function should always prompt medical review.
A patient with absent brainstem function may be dead.

EYES — Check for tracking and blinking to command

If open ask the patient to follow a finger or object horizontally and vertically.
If closed the examiner should open them and examine tracking. One eye will suffice in cases of eyelid or facial trauma.

Can the patient blink twice on command?
A patient with preserved tracking and blinking but GCS 3 may have a locked-in syndrome (the patient is fully aware).

BRAINSTEM REFLEXES — Any changes should trigger immediate medical review

Assess the pupillary reaction to light and record the size and shape of the pupils

Check the corneal reflex is intact by dropping 2-3 drops of sterile 0.9% NaCl onto the cornea from a distance of 8-10cm. The patient should blink.

The patient should cough on tracheal suctioning unless neuromuscular blockade is being used. Do not perform this if the ICP is problematic.

An awake patient should be able to stick out their tongue to command.

RESPIRATORY PATTERN Assess the pattern and rate of breathing

Is it regular or irregular? Is the patient apnoeic?

Does the patient exhibit Cheyne-Stokes breathing (gasping then periods of apnoea)?

Is the rate of breathing above ventilator rate?

Appendix 7

HEMOglobin transfusion threshold in Traumatic brain Injury OptimizatioN

What is the study about? Determine whether a restrictive or liberal blood transfusion strategy is best for patients with traumatic brain injury (TBI)

What do we do currently? We transfuse TBI patients when their Hb falls to 70 g/L

Who is eligible for this trial? Adults (18+ years) with a blunt (not penetrating) traumatic brain injury within the last 24 hrs whose GCS is 12 or less in the emergency department.

Who is excluded from this trial? (Any of the following) Patients who: are actively bleeding or have haemorrhagic shock, require urgent surgery, are not expected to survive (devastating brain injury), have received a blood transfusion prior to randomisation or whose identity is unknown

When can an eligible patient be randomised? As soon as their Hb falls below 100g/L on a full blood count or blood gas sample. This could be many days after admission to the ICU.

Who do I contact if I think a patient is eligible for the trial? A member of the Research Team or Jonathan Greenbaum. Note: Research Nurse attends medical handover every morning. The research team will assess eligibility, organise consent and randomise an eligible patient when their Hb falls below 100 g/L

What happens at the time of randomisation? If randomised to the liberal group then immediately organise transfusion of 1 unit red cells. The blood needs to be given within 3 hours of randomisation. If randomised to the restrictive group then do not give blood until Hb falls to 70 g/L.

Looking after patients who are in the trial. Apart from the blood transfusion trigger, all care is the same as any other patient with a TBI. When the patient reaches their transfusion threshold (100 or 70 g/L) then ONE unit of red cells is given within 3 hours and the Hb is checked again. Further blood is given only when the Hb falls below the transfusion threshold again.

When does the study intervention end? When the patient is discharged from the ICU (including step down to NHDU) or dies.

Unsure about anything? Please speak with either a member of the Research Team (tel: 62188 from 7am to 7pm) or Jonathan Greenbaum (mobile: 07595 167794 – any time)

PLEASE SEE REVERSE FOR RESPONSIBILITIES

Nurse responsibilities	Prescriber responsibilities
Be aware that the patient is in the HEMOTION trial and which Hb threshold (100 or 70 g/L) they have been randomised to (sticker on chart)	Be aware that the patient is in the HEMOTION trial and which Hb threshold (100 or 70 g/L) they have been randomised to (sticker on chart)
Ensure patient has a valid Group & Save sample (lasts 72 hrs). Contact Blood Bank if unsure.	
Monitor Hb with laboratory bloods samples and blood gas samples	Monitor Hb with laboratory bloods samples and blood gas samples
When Hb drops below the patient's transfusion threshold on any sample (FBC, ABG, VBG) alert ACCP/doctor that patient needs transfusion within 3 hours of that result	When Hb drops below the patient's transfusion threshold on any sample (FBC, ABG, VBG) act quickly to organise transfusion
When contacting Blood Bank to request a unit of red cells, inform the Blood Bank BMS that the patient is in the HEMOTION trial.	Call - or ask nurse to call - Blood Bank to request one unit of red cells (inform Blood Bank staff that patient is in the HEMOTION trial).
Organise prompt collection, bedside checking and administration of red cells	Complete the purple transfusion prescription chart
Document start and finish times of transfusion	Ensure patient has iv access for transfusion
Only give one unit and then recheck Hb. Only give further blood if Hb remains below the Hb threshold or directed otherwise by ACCP/doctor (e.g. patient actively bleeding)	Aim to transfuse red cells within 3 hours of the time that Hb fell below threshold. Check Hb after one unit red cells transfused. Only give more if Hb remains below threshold or actively bleeding.

THANK YOU!

CCCTG Canadian Critical Care Trials Group

NHS Salford Care Organisation Northern Care Alliance NHS Group

CTRC Canadian Traumatic Brain Injury Research Consortium

UKCCRG UK Critical Care Research Group

Appendix 8

NHS
Salford Care Organisation
Northern Care Alliance NHS Group

Hypertonic Sodium Chloride (HTS)

Dosing Table

SOS
Sugar or Salt trial:
Hyperosmolar therapy in traumatic brain injury

Dosing to be based on IDEAL body weight

mL/KG DOSING

Kg	<35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	90-94
Weight for calculation	35	40	45	50	55	60	65	70	75	80	85	90
30% HTS (ml)	7	8	9	10	11	12	13	14	15	16	17	18

Kg	95-99	100-104	105-109	110-114	115-119	120-124	125-129	130-134	135-139	140-144	145-149	150-154
Weight for calculation	95	100	105	110	115	120	125	130	135	140	145	150
30% HTS (ml)	19	20	21	22	23	24	25	26	27	28	29	30

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Appendix 9

NHS
Salford Care Organisation
Northern Care Alliance NHS Group

Mannitol Dosing Table

Dosing to be based on IDEAL body weight

SOS
Sugar or Salt trial:
Hyperosmolar therapy in traumatic brain injury

mL/KG DOSING

Kg	<35-39	40-44	45-49	50-54	55-59	60-64	65-69	70-74	75-79	80-84	85-89	90-94
Weight for calculation	35	40	45	50	55	60	65	70	75	80	85	90
Mannitol 10% (ml)	140	160	180	200	220	240	260	280	300	320	340	360

Kg	95-99	100-104	105-109	110-114	115-119	120-124	125-129	130-134	135-139	140-144	145-149	150-154
Weight for calculation	95	100	105	110	115	120	125	130	135	140	145	150
Mannitol 10% (ml)	380	400	420	440	460	480	500	520	540	560	580	600

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11. Document Control Information

Part 1 Must be fully completed by the Author prior to submission for approval			
Name of lead author:	M J Naisbitt		
Job Title:	Consultant in Intensive care medicine		
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Consultation: List persons/group included in consultation. N.B. Include Pharmacy/PADAT/D&T/MMG for documents containing drugs. Indicate whether feedback used (FU), not used (FNU) or not received(NR)			
Name of person or group	**Role/Department/Service/Committee/ Corporate Service**	**Date**	**Response: FU/ FNU / NR**
Critical care consultants	Governance meeting (SCO)	May 21	FU
Neurosurgical Consultants	Governance meeting (SCO)	May 21	FU
Dr P Ferris	Consultant ICM	May 21	FU
Dr D Horner	Consultant ICM	May 21	FU
Mr D Holsgrove	Consultant Neurosurgeon	May 21	FU
EqIA sign off: See Appendix 11			
Name: (Insert named lead from EDI team		**Date:**	
Yasmin Bukhari		13th October 2021	
Communication plan:			
Education and core framework for MDT treatment of TBI			

Part 2 Must be fully completed by the Author following committee approval. Failure to complete fully will potentially delay publication of the document. Submit to Document Control/Policy Support for publication		
Approval date:	**Method of document approval**	
27/07/2021	Formal Committee decision Yes	Chairperson's approval Yes
Name of Approving Committee	ICU Governance Committee	
Chairperson Name/Role	Dr R C Haslett	
Amendments approval: Name of approver, version number and date. Do not amend above details		

Part 3 Must be fully completed by the Author prior to publication	
Keywords & phrases:	Traumatic Brain Injury, TBI, Intracranial Pressure, ICP
Document review arrangements	Review will occur by the author, or a nominated person, within five years or earlier should a change in legislation, best practice or other change in circumstance dictate.

12. Equality Impact Assessment (EqIA) screening tool

- The below tool must be completed at the start of any new or existing policy, procedure, or guideline development or review. **N.B.** For ease, all documents will be referred to as 'Policy*'. The EqIA should be used to inform the design of the new policy and reviewed right up until the policy is approved and not completed simply as an audit of the final Policy itself.
- All sections of the tool will expand as required.
- EqIAs must be sent for review prior to the policy* being sent to committee for approval. Any changes made at committee after an EqIA has been sign off must result in the EqIA being updated to reflect these changes. Policies will not be published without a completed and quality reviewed EqIA.

Help and guidance available:

- Click here for the Policy*EqIA Tips for Completion QRG
- Email the Group EDI Team: eqia@pat.nhs.uk for advice or training information.
- Submission of policy* documents requiring EqIA sign off to: eqia@pat.nhs.uk. Allowing an initial four week turnaround.
- Where there is a statutory or significant risk, requests to expedite the review process can be made by exception to the Group Equality & Inclusion Programme Manager tara.hewitt@pat.nhs.uk

1. Possible Negative Impacts			
Protected Characteristic	**Possible Impact**	**Action/Mitigation**	
Age	Outcomes worse with advancing age	This data is part of the document and can be used to discuss with relatives/advocates and decide on best treatment options	
Disability	n/a		
Ethnicity	Communication can be challenging due to language barriers.	Utilisation of translation services when discussing with relatives/advocates	
Gender	n/a		
Marriage/Civil Partnership	n/a		
Pregnancy/Maternity	No in-patient maternity services at Salford	Patients in 3rd trimester of pregnancy are transferred to Preston so can be managed on neuro-intensive care unit with local in-patient maternity services. As per major trauma pathway	
Religion & Belief	n/a		
Sexual Orientation	n/a		
Trans	n/a		
Other Under Served Communities (Including Carers, Low Income, Veterans)	n/a		

2. Possible Opportunity for Positive Impacts		
Protected Characteristic	**Possible Impact**	**Action/Mitigation**
Age	Outcomes worse with advancing age	This data is part of the document and can be used to discuss with relatives/advocates and decide on best treatment options
Disability	n/a	
Ethnicity	Good early communication utilising translation services	
Gender	n/a	
Marriage/Civil Partnership	n/a	
Pregnancy/Maternity	n/a	
Religion & Belief	n/a	
Sexual Orientation	n/a	
Trans	n/a	
Other Under Served Communities (Including Carers, Low Income, Veterans)	n/a	

3. Combined Action Plan			
Action *(List all actions & mitigation below)*	**Due Date**	**Lead** *(Name & Job Role)*	**From Negative or Positive Impact?**
Understanding and communicating adverse outcomes with advancing age	**Complete**	**Paul Ferris (ICU consultant)**	**Both**
Patients transferred to Preston if in 3rd trimester of pregnancy as per major trauma pathway	**Complete**	**Sheila Tose (trauma lead for Salford)**	

4. Information Consulted and Evidence Base *(Including any consultation)*			
Protected Characteristic	Name of Source	Summary of Areas Covered	Web link/contact info
Age	Data in this document	Outcomes worse with advancing age	
Disability			
Ethnicity			
Gender			
Marriage/Civil Partnership			
Pregnancy/Maternity			
Religion & Belief			
Sexual Orientation			
Trans			
Other Under Served Communities (Including Carers, Low Income, Veterans)			

5. EqIA Update Log *(Detail any changes made to EqIA as policy has developed and any additional impacts included)*		
Date of Update	**Author of Update**	**Change Made**

6. Have all of the negative impacts you have considered been fully mitigated or resolved? *Yes*
7. Please explain how you have considered the duties under the accessible information standard if your document relates to patients? Use of translation service
8. Equality Impact ~~A~~ssessment completed and signed off? *(Insert named lead from EDI Team below). Please also add this information within Section 11.* **Name: Yasmin Bukhari** **Date: 13th October 2021**