Traumatic Brain Injury Guidelines

Salford Royal NHS Foundation Trust

University Teaching Trust

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Who should read this document?

- Staff who manage patients with 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

Key Messages

All patients should be reviewed within 12 hours of admission and at least twice a day by both critical care and neurosurgical consultants.

A clear ceiling of care decision in high risk patients should be documented and discussed with the patient or their advocate within 24 hours of admission to SRFT.

Variance from the guideline may be indicated and should be documented accordingly.

Management of traumatic brain injury (TBI) relies on attention to detail and ensuring all prescribed interventions are reliably instituted in every patient every time.

Care is delivered in a staged manner, and escalation or de-escalation between stages should be done following multi-disciplinary review and with a clear documented plan.

Background & Scope

Traumatic brain injury (TBI) accounts on average for approximately 25% of the admissions to Salford Royal Critical Care.

Our focus is to limit the degree of 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).

The treatment of TBI is aimed at controlling the ICP at low levels (<20mmHg) and maintaining cerebral perfusion pressure (CPP) at reasonable levels (generally 60-70mmHg).

There is no 'magic bullet' therapy for TBI, the key to optimising 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.

At anytime if the ICP rises to > 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 can then be implemented.

What is new in this version?

1. Addition of a 'Stage Zero' which will explain management of TBI patients in a level 2 enviroment or following stepdown from 'Stage One' TBI management.
2. Guidance around de-escalation of ICP control measures
3. Consideration of individualised CPP targets depending on the type of TBI
4. Bolus dosing of 30%NaCl for ICP control
5. Guidance on the investigation and treatment of cranial diabetes insipidus
6. A change in measurement of arterial blood pressure.
   When a patient has an ICP monitor in-situ, we will now be measuring arterial blood pressure from the level of the tragus. *This change is being implemented on a test of change basis from December 2014 for 3-6months.*

Protocol Details

Summary Flow Chart

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**
- Normoglycaemia
- Consider anticonvulsant therapy

A drop in GCS of 1 point or to <8 should trigger medical review and consideration of escalation of therapy

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

Stage ONE

- **Head up or bed tilt 30 degrees**
- **Avoid venous congestion and remove collar**
- **Sedation to a RASS -5 & avoid coughing**
- **$PaCO_2$ 4.5-5kPa & PaO_2 >10-12kPa Sats >97%**
- **CPP 60-70mmHg with fluids & up to 10ml/hr 4mg: 50ml Noradrenaline**
- Normoglycaemia
- Ensure analgesia optimised
- Consider anticonvulsant therapy
- Consider neuromuscular blockade

A rise in the ICP >20mmHg for 5 minutes triggers a medical review and consideration of escalation of therapy

Is the loss of ICP control due to an intracranial cause?

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

Stage TWO

- $PaCO_2$ 4-4.5kPa
- Ensure adequate cardiac output using continuous monitoring and maintain CPP 60-70mmHg
- Osmotherapy
- Consider loop diuretics if >3l +ve balance
- Maintain normothermia by cooling
- Consultant MDT individualisation of the CPP target (50-80mmHg)
- Consultant MDT individualisation of the ICP threshold (20-30mmHg)

Is the loss of ICP control due to an intracranial cause?

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

Stage THREE always requires consultant discussion

- Consider a large decompressive craniectomy
- Consider therapeutic hypothermia
- Consider barbiturate infusion to 50% burst suppression with CFAM or BIS monitoring

The change in volume obtained by any operative management should be accompanied by treatment targeting a reduction in brain water content

STAGE ZERO THERAPY

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 a minimum of Glasgow Coma Score and the pupillary response to light. This should be performed hourly for 8 hours then reduced to 2-hourly for a further 8 hours and then 4-hourly. A trained nurse is defined as one who has completed the competency assessment.

- 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 results must be documented, as a clinical note, by the neurosurgery or radiology team within 12 hours.
    - All hard plastic collars should be removed. Hard extraction collars should not be in place >2hours after arrival at SRFT. Prolonged use is associated with a high incidence of 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

- MAP should be maintained ≥90mmHg with intravenous fluids and the use of appropriate vasopressor therapy.

- Correct coagulopathy or reverse anticoagulant therapy if present as per SRFT MTC guidelines.

- DVT prophylaxis should be considered in all patients in accordance with the existing protocol.

- Pain should be assessed and an appropriate analgesia regime instituted. This should include paracetamol +/- opioids.

- **ANTICONVULSANT** therapy:
    - A 7-day prophylactic 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

      <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 anytime or if non-convulsive status epilepticus is suspected.
- The second line therapy is Levetiracetam 500mg bd NG/IV.

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

- All patients should receive enteral feed as per the enteral feeding calculator (at least 25kcal/kg/day), there is strong evidence that a cumulative caloric deficit is associated with worsened outcomes in TBI.

- A full secondary survey should be performed and documented within the first 24 hours.

- **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.
  - Fluid responsiveness does not equate with a euvolaemic state and cardiac output monitoring can be required early in the treatment course.
  - The total maintenance daily fluid requirement for an individual patient is approximately 30ml/kg day.
  - In most 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 1.0kcal/ml
  + 600ml (Propofol and Alfentanil)
  + 400ml Paracetamol *≈ 2270ml delivered fluid*

  I.e. there is little need for continuous supplemental parenteral infusions of crystalloids once full enteral feed is established and being absorbed. 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. If urine output >200ml/h for 2 consecutive hours then refer to guidance in APPENDIX.

Escalation to Stage One Therapy

IF total GCS falls by 1 or below an absolute value of 8 then there should be immediate medical review and escalation of care as appropriate. The actual drop in GCS may be a late indicator of a worsening brain injury. Earlier triggers for escalation may include:

- *A worsening headache*
- *Increasing agitation*
- *Unexplained hypertension or a drop in heart rate*
- *New changes in the pupillary response to light*
- *The development of a new focal motor deficit*
- *Nursing staff report that it is becoming more difficult to achieve the same GCS (e.g. greater patient stimulation necessary) or the duration that this level of GCS is maintained is reducing(after stimulation).*

Is the worsening of the patient's condition due to an intracranial cause?
Consideration must be given to 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 Actual Body Weight.

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

100ml 10% Mannitol contains 10g
100ml 20% Mannitol contains 20g

Mannitol dose for a 70kg individual: **175-350ml** **10% Mannitol solution**
Or **88-175ml** **20% Mannitol solution**

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

Immediately following the induction of anaesthesia, the pupillary response to light should be reviewed and consideration given to a period of hyperventilation to $PaCO_2$ 4-4.5kPa.

STAGE ONE THERAPY (in all mechanically ventilated patients)

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

- Ensure no cerebral venous congestion from positioning or endotracheal tube ties.
    - The head position should be 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, unless specifically directed otherwise by the spinal team.
    - 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.
- 30-degree 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
- Maintain PaO_2 10-12kPa and Oxygen saturations 94-98%
- Maintain normoglycaemia, plasma glucose 5-10mmol/L, with insulin by infusion utilising the intranet calculator.
- Sedate to a Richmond Agitation Sedation Scale -5
    - Propofol 1% 0-25ml/hr (max dose 4mg/kg/hr)
    - Alfentanil 0-3ml/hr (25mg/50ml)
    - A third sedative agent may also be required Midazolam 0-10mg/hr.
    - Coughing in a patient with poor intracranial compliance must be avoided.
    - Boluses of sedative agents 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.
- Maintain optimal CPP 60-70mmHg with intravenous fluid loading:
    - Use 250ml boluses of Plasmalyte-148 and up to 0-10ml/hr 4mg: 50ml Noradrenaline as required.
    - **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 consultant approval. Consider the use of continuous flow monitoring in addition to echocardiography to ensure euvolaemia and titrate cardiovascular support. AUDIT

- Ensure full enteral feed is prescribed per the web-based calculator.
- Ensure analgesic requirements are reviewed regularly. Analgesia should be delivered with paracetamol +/- opioids.

Additional level one therapy in selected patients includes:

- External ventricular drainage of CSF.
- Treatment of infection as per trust infection control and antibiotic guidelines
- Consideration of non-depolarising neuromuscular blockade in conjunction with neuromuscular junction monitoring.
- Consider starting or escalating anticonvulsant therapy
- Consideration given to individualisation of optimal CPP target.
    - This is a **consultant level MDT decision** dependent on the pattern of injury and the degree of cerebral auto regulation thought to be present.
    - A CPP >70mmHg has been historically associated with a higher risk of Acute Lung Injury but may be indicated in patients with diffuse axonal injury.
- Consideration given to commencing antihypertensive therapy if the natural CPP ≥110mmHg and a vasogenic oedema mechanism postulated. The target should be to reduce MAP by 25%. This is a **consultant level MDT decision**.
    - 1st line Labetalol 300mg: 60ml 0-20ml/hr
    - 2nd line Clonidine 750mcg: 50ml 0-10ml/hr

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. AUDIT

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. AUDIT

STAGE TWO THERAPY – triggers further senior medical review

- Why has the ICP risen? Is the CPP maintained?
- Are all Stage ONE measures are actually in place?
- Is it due to an intracranial or systemic cause?
- Is a repeat CT head indicated?

- Increase minute ventilation to target PaCO2 4-4.5kPa
  - 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 cardiac output monitor is required to confirm adequate intravascular volume and to ensure adequate blood flow. Fluids, dobutamine or higher doses of noradrenaline may be required to optimise blood flow and maintain an optimal CPP 60-70mmHg.

- External ventricular drainage of CSF. This may require a BRAINLAB© CT scan.

- Target normothermia by infusing cold intravenous fluids and/or utilising an external or internal cooling device.

- OSMOTHERAPY (APPENDIX)

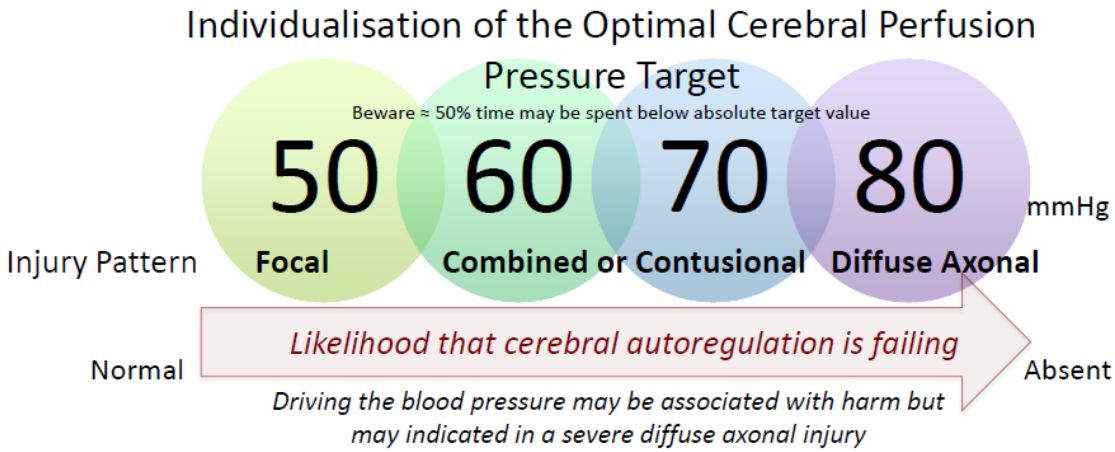
The use of osmotherapy mandates close attention to the detail of all fluid and sodium balance, a minimum of 4-hourly medical review is standard practice.

- **30% NaCl can only be given via a CENTRAL LINE.**
- **Consultants (***or senior trainees)* **may give a 15ml bolus of 30% NaCl over 10 min (75mmol) using an infusion pump, to establish ICP control .**
- **A maximum of 4 x 15ml boluses can be given within a 24 hour period.**
- Each bolus will increase plasma Na^+ by 2-3mmol/L.This dose does not account for any ongoing naturesis, changes in free water or sodium intake.
- **If serum Na^+ ≥155mmol/L or calculated plasma osmolality ≥320mosmol/L then 30% NaCl is contraindicated.**

Plasma Na^+ must be checked every 4 hours if 30%NaCl is used.

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

- Consideration may be given to 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.
    - Consider a trial of CPP >70mmHg. A CPP >70mmHg has been historically associated with a higher risk of Acute Lung Injury but may be indicated in patients with diffuse axonal injury.



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 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.

STAGE THREE THERAPY (always requires consultant discussion)

Are all stage ONE and appropriate stage TWO measures actually in place?
Why has the ICP risen?
Is the CPP maintained ≥ target value?
Is a repeat CT head indicated?

- Consider a large decompressive craniectomy.
  - The rapid change in volume obtained by a decompressive crainectomy 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, osmotherapy or on occasion by bolstering colloidal oncotic pressure.

- Consider a barbiturate infusion to control intracranial pressure.
  - Thiopentone infusion to a target of 50% burst suppression utilising CFAM/BIS monitoring.
  - Load with 500mg-1.5g Thiopentone and then start an infusion at 0.5-6mg/kg/hr (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.
  - Most patients will require advanced cardiovascular monitoring during barbiturate coma

- Consider instituting therapeutic hypothermia to control intracranial pressure. This may require the maintenance of central temperature 32-35^{o}C utilising an appropriate cooling device. *In view of pending results from the EUROTHERM 3235 clinical trial this treatment option may no longer be appropriate.*

As a bridge to theatre, under consultant supervision, a 15ml 30% NaCl bolus may be infused via the **central line**. This can only be administered when plasma Na^+ ≤155mmol/L and will increase plasma Na^+ further by approximately 2mmol/L.

Mannitol may be administered instead (providing calculated plasma osmolality ≤320mosmol/L). It's use MUST be documented and verbally handed over to the theatre anaesthetist.

Mannitol dose for a 70kg individual: **175-350ml 10% Mannitol solution**
Or **87.5-175ml 20% Mannitol solution**

All patients who receive Mannitol are highly likely to subsequently need intravenous fluid resuscitation to maintain cerebral blood flow following their diuresis.
Repeated Mannitol doses may paradoxically worsen cerebral oedema and have been historically associated with acute kidney injury.

Osmotherapy with hypertonic NaCl

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

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

Consultants (*or senior trainees)* **may give a 15ml bolus of 30% NaCl over 10 min (75mmol) using an infusion pump, to establish ICP control .**

A maximum of 4 x 15ml boluses can be given within a 24 hour period.

Each bolus will increase plasma Na^+ by 2-3mmol/L.This dose does not account for any ongoing naturesis, free water change or sodium intake.

If serum Na^+ ≥155mmol/L or calculated plasma osmolality ≥320mosmol/L then 30% NaCl is contraindicated.

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 or rebound cerebral oedema could occur if the plasma Na^+ falls by >0.5mmol/L an hour.

Marked changes in plasma Na^+ are to be avoided and may be associated with rebound cerebral oedema.

Diabetes insipidus contraindicates the use of hypertonic NaCl.

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
30% NaCl	5 **mmol/ml**

Cranial Diabetes Insipidus and management pathway

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

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 free water is 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. Measure urine specific gravity. Inform the ICU Consultant *(or senior trainee).*
2. Start a Plasmalyte-148 infusion at an input rate to match the previous hour's urine output
3. Measure plasma Na^+ using ABG machine 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^+ rising by ≥5mmol/L/h give a STAT bolus of DDAVP 0.5micrograms IV before the laboratory confirmation of DI
6. If plasma Na^+ ≥160mmol/L, inform the ICU consultant *(or senior trainee)*, then start additional hypotonic fluid (5% glucose at 100ml/h for a 70kg man) to aim to lower plasma Na+ by 0.5mmol/L/h and titrate this hypotonic infusion rate to response using the hourly measured ABG machine Na^+.
7. If the diuresis worsens, continues at the same rate or recurs ≥4 hours after a DDAVP bolus then give a repeat bolus of 1mcg IV. Then start a continuous infusion of DDAVP. The DDAVP infusion may be required for up to 6 days.
8. If the laboratory confirms DI **(Urine osmolality<Plasma osmolality and plasma osmolality is >285mosmol/kg)** but plasma Na^+ remains unchanged or has risen by ≤5mmol/L/h, then give a DDAVP bolus 0.5mcg IV
9. Stop any input/output matching Plasmalyte-148 infusion once the urine output is ≤100ml/h for 2 consecutive hours.

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.

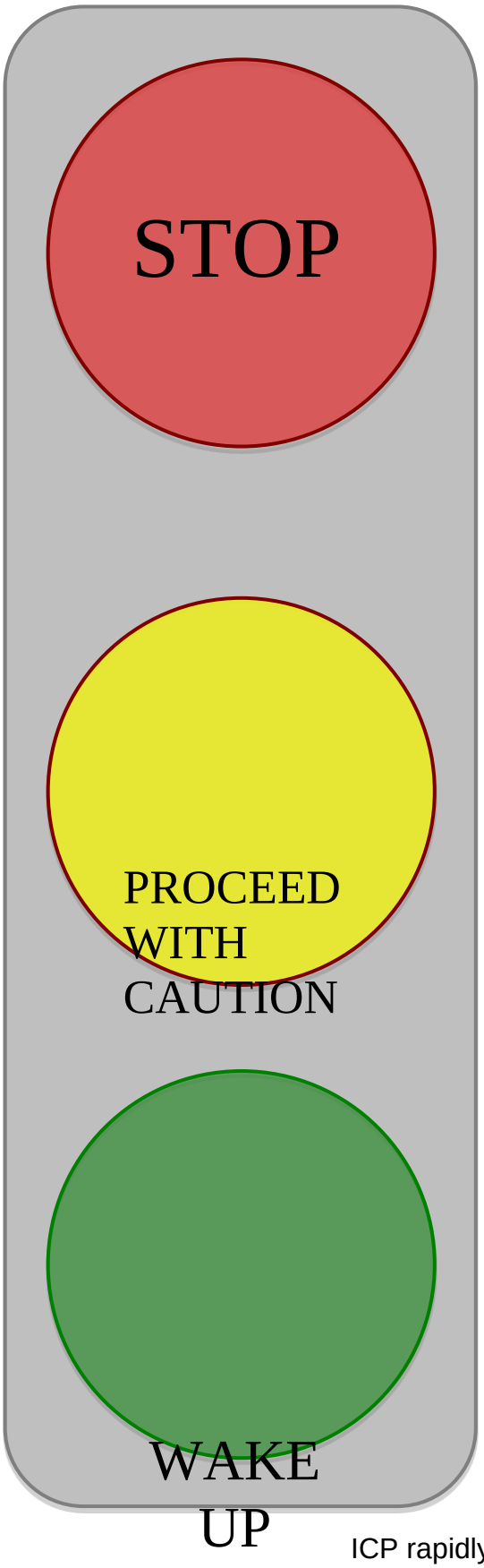
Stepdown Protocol

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

Algorithm for beginning the de-escalation of ICP therapy, changing the ICP threshold or deciding on sedation hold as appropriate. **Consultant MDT input is mandatory**.



DO NOT de-escalate ICP therapy or attempt a sedation hold if:

- ≥ 2 interventions 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

De-escalate ICP therapy or reduce sedation if:

- ≤ 2 interventions in last 24 hours
- Bilateral slowly reactive normal sized pupils
- Stable motor score within GCS
- $P_1 \geq P_2 > P_3$ 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 not compatible with raised ICP

Standards

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. Patients should be reviewed twice a day by a critical care consultant and neurosurgical consultant
3. Invasive arterial blood pressure monitoring and intracranial pressure monitoring should be instituted within 2 hours of commencing Stage 1 therapy
4. No patient should receive >10ml/hr 4mg: 50ml Noradrenaline without consultant approval. Continuous flow monitoring in addition to echocardiography should be used to ensure euvolaemia and titrate cardiovascular support
5. A multidisciplinary neurocritical care and neurosurgical plan should be clearly documented. This should include the stage and choice of therapy to be offered if needed in the next 12-24 hours.
6. 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.

Explanation of terms & Definitions

Terms explained in the document

References and Supporting Documents

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Roles and responsibilities

Critical care neurogovernance group –

1. Ensure staff involved are educated about new clinical guideline and implications for practice
2. ensure trial method of measuring MAP/CPP is monitored and reviewed pending this monitoring process
3. ensure standards set out are audited and results fed back to critical care

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)**	
-3	Moderate sedation	Movement or eye opening to *voice* **(but no eye contact)**	
-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	

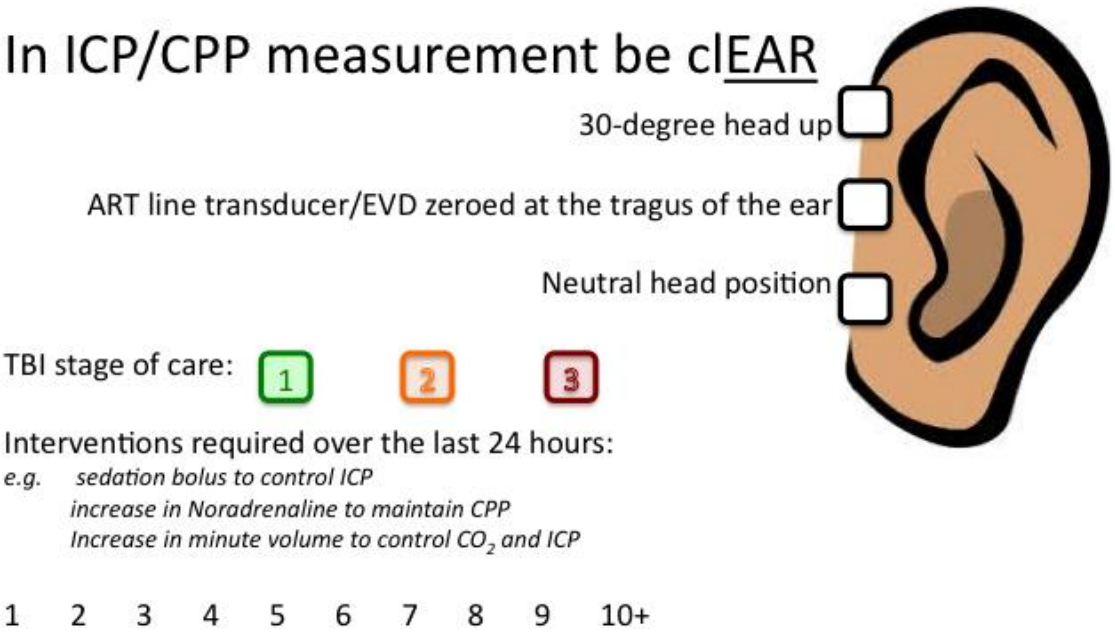
Procedure for RASS Assessment

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 attached to observation chart

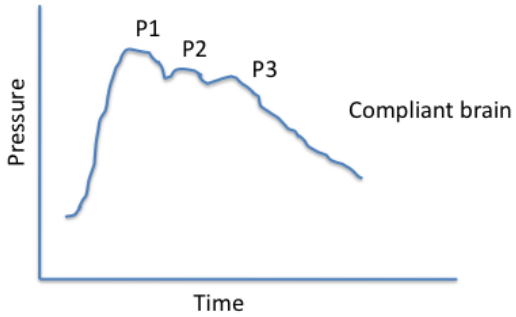


Appendix 3

ICP waveform analysis

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.



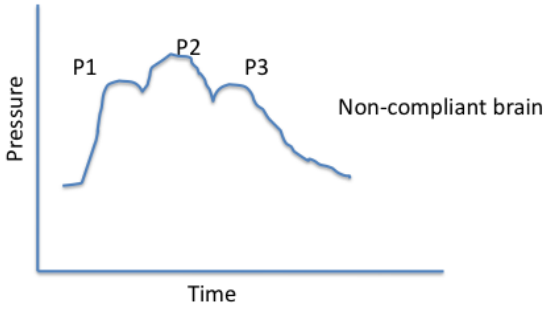
P1 the first and sharpest peak is called the percussive wave and results from the arterial pressure being transmitted from the choroid plexus. Arterial hypotension and hypertension will decrease or increase the amplitude of P1 respectively.

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.

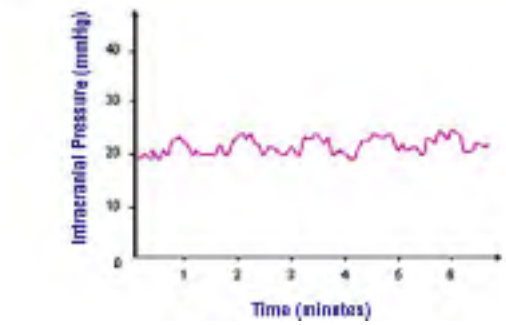
Patients who have undergone a craniectomy will have a dampened waveform.

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, indicating the need to treat an elevated ICP.

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 – Active cooling

Induction of cooling

The first step in the induction of hypothermia is to infuse 20mls/kg of refrigerated crystalloid over 20 minutes. A further 10ml/kg can be infused over another 10minutes if the desired temperature is not reached. Central or peripheral IV access can be used.
The induction can be continued by one of the methods available. The choice will be guided by consultant preference and availability of equipment and disposables.

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 secretaries office. 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.
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 secretaries' office.
   A dedicated core temperature monitor is required, which should be attached to the machine again to provide feedback to maintain the desired temperature.

THE CONSEQUENCES OF HYPOTHERMIA INDUCTION DEPEND UPON THE TARGET TEMPERATURE AND SPEED OF INDUCTION.
AT TEMPERATURES ≤34^{O}C THE PATIENT WILL BECOME MORE UNSTABLE.

Observations and Investigations

*ABG and blood glucose***:** Oxygen consumption and CO_2 production may decrease with the onset of hypothermia. Insulin requirements may decrease with the onset of hypothermia. Check at 1 hourly intervals during induction. Ensure ABG are corrected for patient temperature.
*FBC, Clotting Screen, U+E, Lab glucose***:** Check these lab parameters at least 6 hourly during induction phase. Risk of leucocyte dysfunction, coagulopathy and large renal electrolyte losses.
*Fluid balance***:** Monitor overall fluid balance hourly. Risk of hypothermia induced diuresis.

Arrhythmias: Monitor for occurrence of arrhythmias.

Interventions
*Electrolyte supplementation***:** Maintain K^+ > 4.0 mmol/l; PO_4 > 1.0 mmol/l; Mg^{2+} > 1.0 mmol/l
Magnesium: Commence $MgSO_4$ infusion 40mmol/24hours.
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.

Ventilation: Adjust ventilatory parameters according to ABG results.
Arrhythmias: These should be managed with electrolyte supplementation. Resistant arrhythmias or those with haemodynamic consequences may require amiodarone infusion or other anti-arrythmic drug medication.

Precautions during induction phase
Pressure damage: Increased risk of tissue damage – frequent observation and patient repositioning are required.

<u>Maintenance of Cooling</u>

The semi-automated methods (b and c above) can be continued as long as the reason for cooling is indicated.

MANY OF THE PRINCIPLES ESTABLISHED DURING INDUCTION ARE ALSO FOLLOWED DURING MAINTENANCE AS OUTLINED BELOW.

Observations and Investigations
ABG and blood glucose: Check at 4 hourly intervals during maintenance. Ensure ABG are corrected for patient temperature.
FBC, Clotting Screen, U+E, Lab glucose: Check these lab parameters at least 12 hourly during maintenance phase.
Fluid balance: Monitor overall fluid balance hourly.
Arrhythmias: Monitor for occurrence of arrhythmias.

Interventions
Electrolyte supplementation: Maintain K^+ > 4.0 mmol/l; PO_4 > 1.0 mmol/l; Mg^{2+} > 1.0 mmol/l
Sedation: Administer sedation boluses to minimize shivering. Neuromuscular blockade should **not** be required. See above.
Ventilation: Adjust ventilatory parameters according to ABG results.

Arrhythmias: These should be managed with electrolyte supplementation. Resistant arrhythmias or those with haemodynamic consequences may require amiodarone infusion or other anti-arrythmic drug medication.
Gastrointestinal: Ensure that prokinetics are prescribed.

Precautions during maintenance phase
Pressure damage: Increased risk of tissue damage – frequent observation and patient repositioning are required.
Infection: Increased risk – have lower threshold for surveillance cultures and use of antibiotics. Monitor line insertion sites and wounds closely.
Pharmacokinetics: Altered clearance of various medications. Review drug chart daily on an individual patient basis with the ICU pharmacist.
Gastrointestinal: Impaired GI tract motility, altered LFTs and mild pancreatitis are common. Consider the significance of these issues in your daily clinical assessment of the patient.
Bleeding: Increased risk due to impaired platelet function. Have lower threshold for use of blood products prior to invasive procedures.

Rewarming Phase

Rewarming depends on the method of cooling used and the indication. Ideally the rate should be controlled and not more than 0.5 ^{o}C per hour, usually less at 0.25 ^{o}C per hour.
When using the cooling blankets, the desired temperature should be reset up by 0.5 ^{o}C every two hours until normothermia is reached.
With the intravenous cooling system, the desired target temperature and the rate at which it is reached should be entered into the machine. (see below)
The patient may become unstable as they rewarm, similar to the instability observed during cooling.
In those patients who have been cooled for a raised intracranial pressure, any increase in ICP above 20mmHg requires re-initiation of the cooling phase.
It is not unusual to have a rebound hyperthermia following a period of therapeutic hypothermia, which can occasionally be profound. There is also a risk of infection with hypothermia so a high index of suspicion should be maintained.
Electrolyte levels should also be carefully monitored in the re-warming phase. Hyperkalaemia is a particular risk.
When removing the Alsius 'ICY' IV catheter, it is imperative that the cooling ports are not connected or bunged off so that any saline left in is allowed to freely drain as it is removed. **Failure to do this could lead to a serious injury to the blood vessel.**