NEURO-INTENSIVE CARE

Guidelines
TO BE USED IN CONJUNCTION WITH CURRENT ST GEORGES HOSPITAL GREY BOOK

http://stginet/greybook
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To

**Organisation**

1. The Neuro -ITU acts as a tertiary referral centre for neurosurgery, neurology and stroke services, as well as a high dependency post-operative ward.

2. We have consultant cover for the neuro ITU by dedicated consultants whose sole responsibility is NITU including on call cover. **There will be a ward round every day at 10:30am & 5pm.**

3. **A telephone ward round will take place around 10pm with the consultant on call**

4. The admissions policy at present is via one of the above named teams. Any requests made directly to the unit from areas outside these (that is not accepted by either neurosurgery, neurology or stroke) should be discussed with NITU consultant. We occasionally take non-neuroscience patients due to lack of capacity on GITU, however **this should be a discussion with the NITU consultant on call.**

5. All patients admitted to NITU must have a named consultant in charge.

6. Care of patients on NITU is a combined care with input from their admitting team. However ultimate responsibility falls with the NITU team.

7. **There is always a consultant available for consultation.**

8. There is a dedicated office space for trainees in the trainees’ room- shared office.

9. Please leave your room clean in the morning- between 8-20:00 is used as an office by SNODs, and network manager.

10. **We use Yummer & WhatsApp to communicate so please download both apps and make sure you are following/added to the neuro ITU group.**

11. M&M meeting: Weekly on Wednesday

12. Protected teaching on Neuro Curriculum : every Thursday Morning
Ward round & documentation

1. A formal ward round occurs with the NITU consultant in the morning (10:30 am).

2. Brief ward rounds with neurosurgical registrars usually occur at 9am, 4pm and 10pm.

3. All NITU admissions (except Spinal Injury patients) should have admission forms used. (Picture 1)

4. It is expected all NITU patients will have notes written daily. For all NITU patients we expect you to use the standardised proforma. (Picture 2)

5. All patients should have notes written in each shift (even if to say “no change”) and also when any significant event occurs (e.g. CT scan, starting inotropes or antibiotics, re-intubation, central line insertion, new arrhythmias etc) and a brief explanation of why or the result of.

6. All patients should have a formal ICU episode summary on iClip. This should not be named discharge summary. Please follow instructions on Picture 3.

7. In all patients with Spinal cord injury T8 & above, the relevant Integrated Care Pathway should be used as part of the medical and nursing notes. This includes admission documentation (Picture 4). Please note this document should be used for patient with Spinal cord INJURY T8 and above. All patients with a spinal injury should be referred to all the spinal injury units, within 48 hours of admission. A proforma is on one of the desktop computers.

8. DVT prophylaxis assessment and antibiotic prescription should follow Trust’s guidelines. All patients should have DVT prophylaxis assessment form filled on admission (or within 12hours). Antibiotics prescription should ALWAYS be discussed with microbiologist on call. Always write the length and reason for the antibiotic prescription. This information needs to be filled both in the notes and iClip.

9. Before any invasive procedure you should fill and file in the notes a WHO checklist (Picture 5)

The following is a brief guide to common conditions, as with all problems in medicine we may have to tailor individual treatment and variations from this may occur.
### Picture 4

**Integrated Care Pathway for patients with Spinal Cord Injury at T8 or above**

- Days 1 – 7 only
- Revised pilot version
  - (10th June 2015)
- Attach patient label here
- All clinical staff should use this ICP to record assessments and care.
- The ICP should be used from admission to day 7.
- Please keep to the daily format.

**Nurses**
- There is a care plan for the week on p.5. You do not need to write a daily MEAD.
- There is an assessment form to be completed at the start of each shift (blue borders).
- Evaluations should be written on the continuation sheets for each day.
- The bowel care record MUST be completed daily from day 2 onwards.
- All other Trust paperwork will need to be completed as usual.

### Picture 5

**ICU "WHO Safety Checklist" for invasive procedures (for bronchoscopy, tracheostomy and any other 'surgical procedure")**

<table>
<thead>
<tr>
<th>PRE PROCEDURE</th>
<th>Site (if applicable)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient identity confirmed</td>
<td>Y ☐ / N ☐</td>
</tr>
<tr>
<td>Procedure</td>
<td>Indication</td>
</tr>
<tr>
<td>Clinician responsible for decision to perform this procedure</td>
<td>Clinician performing procedure (if different to previous)</td>
</tr>
<tr>
<td>Assistance provided by: Bedside nurse ☐ AND / OR Other ☐ (state who)</td>
<td>Observed / supervised by</td>
</tr>
<tr>
<td>Patient informed</td>
<td>Y ☐ / N ☐</td>
</tr>
<tr>
<td>Patient able to indicate consent</td>
<td>Y ☐ / N ☐</td>
</tr>
<tr>
<td>Next of Kin informed</td>
<td>Y ☐ / N ☐</td>
</tr>
<tr>
<td>All equipment needed to complete the procedure collected together</td>
<td>Patient position</td>
</tr>
<tr>
<td>Analgesia + / or sedation: Required</td>
<td>Y ☐ / N ☐</td>
</tr>
<tr>
<td>Administered and monitored by</td>
<td></td>
</tr>
<tr>
<td>Physiological monitoring: ETCO2 ☐ SpO2 ☐ ECG ☐ IABP ☐ NIBP ☐ CVP ☐ Ventilator ☐ Other</td>
<td></td>
</tr>
<tr>
<td>Foreseeable problems and the plan for each: Known allergies ☐ Difficult airway ☐ Coagulopathic ☐ Others</td>
<td></td>
</tr>
</tbody>
</table>

| POST PROCEDURE | All sharps accounted for | |
|----------------|-------------------------|
| Standard procedure performed | Y ☐ / N ☐ |
| All specimens labelled and sent | Y ☐ / N ☐ |
| Post procedure instructions confirmed | Y ☐ / N ☐ |
| Cleaning and re-stocking of equipment performed by | |
| Any untoward events | Y ☐ / N ☐ |
| Events (e.g. equipment failure, cardiac arrest etc etc. Give details below and complete Datix) | |
Useful numbers

Neuro Reg on call: Bleep 7242

Senior Anaesthetic reg: Bleep 7647

Senior General Anaesthetic reg: Bleep 6111

Duty Floor anaesthetist: Bleep 8011
Head Injury Management in NITU
The aim of managing a patient with a severe head injury is to avoid further secondary injury and control intracranial pressure.

For details about pre-NITU care please refer to the following documents:
Head injury: triage, assessment, investigation and early management of head injury in infants, children and adults - NICE Guidance CG56

Recommendations for the Safe Transfer of Patients with Brain Injury – Association of anaesthetists

Patients with a mild or moderate head injury
Are sometimes intubated for transfer. If the neurosurgical opinion is to wake these patients up then sedation should be stopped and neurological assessment performed and if appropriate, extubated. If they are not suitable for extubation, they should be re-sedated pending a more senior opinion (this process should take into consideration other injuries, difficulty in possible re-intubation, staffing levels etc.).

Initial Management of Adults with Severe Head Injury

The initial management will be greatly influenced by neurosurgical opinion. Patient’s management will vary according to severity of injury, CT scan, initial GCS and presence or absence of an ICP monitor.

- Patients with a severe head injury +/- evidence of raised ICP or a poor neurological assessment.
Should follow the following guidelines.
Waking these patients up has the potential to cause significant harm. Consideration for insertion of an ICP monitor should be raised if not already insitu since a percentage of these patients will develop raised ICP.

- Patients with unclear neurology.
It is clear that an initial GCS performed at the referring hospital is sometimes inaccurate; this greatly influences management and prognosis. In this situation the neurosurgical team may request for “lightening” and a neurosurgical assessment. This should NOT be performed if there is evidence of raised intracranial pressure, or a significantly abnormal CT scan.
If there is no evidence of raised ICP and there is a neurosurgical decision to obtain an accurate neurological assessment, sedation may be stopped initially. Patients should not be weaned from the ventilator and all the physiological parameters outlined in the following guidelines should still apply.
Once a clear assessment of their GCS is obtained (the most significant factor is motor response, localising, flexion, extension or nil), they should either be re-sedated and treated with the following guidelines or treated as a patient with a mild or moderate head injury, depending upon their neurological assessment.
Patients as a general rule under this situation should not be weaned or extubated unless they (at a minimum) are localising appropriately. If the patient has an ICP monitor in-situ, this process may lead to a period of raised ICP. Provided this process is short in duration and CPP is maintained this can be tolerated for a short period while a satisfactory neurological assessment is made. These patients by definition
have no evidence of raised ICP, either clinically, on CT scan, or immediately after a surgical decompression and thus are at minimal risk of herniation. It should be clear from the neurosurgical team what the aim of stopping sedation is, and if the patient has a poor neurological assessment they should be re-sedated and treated with the following guidelines.

Any concerns about what to do with an individual patient should be directed to the consultant who covers the unit.

**Sedation**
Patients should initially be sedated with the aim of being unresponsive to suctioning and painful stimuli. This will often require the addition of inotropes to maintain a satisfactory CPP.

**1st Line:** Propofol - max dose 5mg/kh/hr
Add Midazolam if problems with sedation or Propofol > 5mg/kg/hr
Check plasma lipids every 2 or 3 days on patients on high dose lipids.
All patients should receive a narcotic infusion, usually Fentanyl (up to 200mcg/hr).

Weaning off propofol may lead to a rise in ICP and should be avoided if possible until the patient is stable or contra-indications arise.
The temptation to wake a patient up to perform a neurological assessment in a patient with a severe head injury should be avoided for the first 48 hours and until ICP is stable. If the ICP is normal, after a period of stability sedation may be withdrawn provided ICP continues to remain normal.

**Paralysis**
Patients should not be routinely paralysed. If required as part of the management of elevated ICP, Atracurium infusion is the preferred agent, titrated to 2 TOF twitches, and assessed 12 hourly. The aim should be to wean it as soon as possible.
Paralysis has been shown not to affect ICP (and is associated with other critical care complications) and thus cannot be recommended except in exceptional circumstances.

**Ventilation**
Use a volume controlled mode of ventilation (VC or PRVC)
Aim: PaCO₂ 4.5-5.0 kPa if ICP stable
        4.0-4.5 kPa if ICP elevated, whilst initiating 2nd line treatments
PaO₂ > 10 kPa (minimum Saturation 95-98%) for first 48 hours

Hyperventilation may be particularly harmful in the first 24 hours after injury when cerebral blood flow is often critically reduced.
Maintain minimum PEEP level of 5 cmH2O. PEEP may be increased to maintain adequate oxygenation as required and is unlikely to influence ICP.
After any significant change to ventilator settings a repeat ABG should be performed after 20-30 minutes.

**Haemodynamics**
Arterial transducer should be zeroed at the level of the external auditory meatus. This is to ensure correct calculation of CPP.
Aim is for an absolute minimum CPP of 60 mmHg.
In the absence of ICP monitoring, aim for a MAP of 90 mmHg.
The use of LIDCO is highly recommended to guide fluid resuscitation and inotrope requirements. Noradrenaline is the preferred initial inotrope. In certain circumstances, when supported by LIDCO measurements, Vasopressin or Dobutamine may also be considered.

**Head elevation**
Patients should initially be placed 15 degrees head up. Head elevation can be increased once the spine is cleared, but generally not more than 45 degrees.

**Temperature**
Pyrexia should be aggressively managed to maintain normothermia at < 37.5 degrees Celsius. Sources of infection should be considered and actively treated. Patients should receive regular Paracetamol, with the addition of NSAIDs for resistant cases and active cooling (ice packs etc.) If patient remains pyrexial despite these measures, insertion of a Coolguard line and intravenous cooling should be initiated.

Whilst there is no evidence to support induced prophylactic hypothermia to prevent secondary injury, patients who are hypothermic at presentation should not be actively warmed in the initial management phase

**Fluids**
Normal saline should be used as initial maintenance fluid. Aim is for normovolaemia. Fluid requirements should be reviewed on a daily basis. Remember to include drugs in this calculation. **Serum Na should be maintained at more than 140 mmol/L**
The assessment of volume status is an imprecise task. In the measurement of blood volume status the best approach is to integrate blood pressure, heart rate, urinary output and central filling pressures (where available). LIDCO monitoring and stroke volume variability may be useful if uncertain.

**Transfusion threshold**
Aim is for a minimum Hct of 0.30 (30%) for the first 48 hours post injury and while unstable. Some treatment algorithms suggest Hb should be maintained > 100g/L.

**Nutrition**
Commence early enteral feeding. Supplement with iv normal saline to achieve daily fluid requirements. Remember to prescribe laxatives (Initially Senna and Sodium docusate +/- Movicol as required).

Failure to reach full caloric replacement by Day 7 post injury may be associated higher mortality; therefore consideration should be given to parenteral nutrition in these circumstances.

If patient has reduced GI motility (aspirates > 300mls) start Metoclopramide 10mg iv tds, and/ or Erythromycin 250 mg ng qds

Request dietician review early.

**Blood Sugar**
Hypoglycaemia is extremely detrimental to the brain and should therefore be avoided. Whilst close glycaemic control is desirable the risk of hypoglycaemia associated with tight control regimes must be offset against any benefit. Insulin infusion should be used to maintain blood glucose < 10 mmol/L at all times, with an ideal range of 4-8 mmol/L.
**Stress ulcer prophylaxis**
All patients should receive some form of gastric protection. If no known GI co-morbidities prescribe Ranitidine (50mg iv tds until absorbing feed, then 150mg ng bd) in addition to enteral feeding while patient is receiving ventilatory support. If the patient is already taking a PPI this should be continued.

**Antibiotics**
Not routinely required, unless surgically indicated. Avoid prophylactic antibiotics for aspiration unless an obvious infection exists, septic, or already on antibiotics for >48 hours (in which case limit these to 5 days or modify according to cultures).

Antibiotic policy changes regularly and is reviewed by microbiology. Current hospital and intensive care guidelines should be available on NITU. Antibiotic policy changes regularly and is reviewed by microbiology. Current hospital and intensive care guidelines should be available on NITU.

**Seizure control**
If surgically indicated, prophylactic anticonvulsant should be limited to 7 days post-injury. First line agent is currently Levetiracetam. Patients with witnessed seizure should continue on anticonvulsants or commence on anti-convulsants (if not already on an anti-epileptic drug - AED). For further information please refer to section on epilepsy in these guidelines. **Preferred route of administration is the enteral route after IV loading dose.**

**Transfer patients to radiology/theatres**
Patients should be fully monitored, mechanically ventilated and if unstable paralysed. ICP monitoring should continue. During transfer, you should aim to maintain the same parameters as on the NITU.

**Management of raised intracranial pressure**

Initial target is ICP < 25 mmHg and CPP > 60 mmHg.

If ICP > 25 mmHg
Ensure optimal head position and no venous constriction
Ensure optimal sedation and analgesia (Consider BIS monitoring and target <20)
Ensure optimal blood gases
Treat pyrexia and seizures if present
CONSIDER REPEAT CT SCAN. Is there a surgical lesion or will an EVD help??

**Second tier therapies such as**

1. Osmotherapy / Induced hypernatraemia (Serum Na 150-160 mmol/L)
2. CSF drainage
3. Therapeutic hypothermia
4. Decompressive Craniectomy
5. Barbiturate coma

Are potentially hazardous and should only be authorised by someone experienced in managing head injuries – consultant level
The following is a flow chart outlining therapeutic options.

- Sedation so initially unresponsive
- Head Elevation 15-30 degrees
- Propofol (max 5mg/kg/hr), Fentanyl or Morphine infusion add Midazolam if required
- Blood Glucose 4-8 mmol/L, Temp < 37.5 °C, Normovolaemia, Hct > 0.3
- PaCO2 4.5-5.0 kPa, PaO2 >10 kPa, (SaO2 95-98%)
- CPP > 60 mmHg

**INTRACRANIAL HYPERTENSION**
- Initial threshold of 25mmHg, may be increased by senior medical staff

**NO**
- Carefully De-Escalate
- ICP Treatment after period of stability

**YES**
- Ensure Airway and ventilation are adequate
- Check ABGs - Check if Intrathoracic pressure is raised?
- Consider pneumothorax / Obstruction / sputum retention
- Check if circulation is adequate?
- Is CPP > 60 mHg? / Normovolaemia?
- Head position optimal? Evidence of venous obstruction?
- Ensure adequately sedated – Consider BIS monitoring
- No evidence of pyrexia or seizures

**CONSIDER REPEAT CT SCAN**

**INTRACRANIAL HYPERTENSION**
- 1. Administer osmotic agent, REPEAT as necessary
- 2. PaCO2 4.0-4.5 kPa
- NOTIFY NEUROSURGICAL TEAM

**OSMOTHERAPY**
- Mannitol 0.25-1g/kg
- Repeated as necessary PRN AND/ OR
- Hypertonic saline (5%) 1-2ml/kg (4-hrly max 8ml/kg/24hrs)
- PROVIDED serum osmolality < 320

**PROVIDED**
- normovolaemic

**SECOND TIER THERAPIES ONLY AFTER DISCUSSION WITH RESPONSIBLE CONSULTANT**
- “Recommend using EEG or BIS monitoring (Maximal effect occurs with a BIS level of 5-20)

- Barbiturate Coma
- Decompressive Cranectomy
- Hypothermia to 35 C
- CSF Drainage

**OR Reconsider treatment aims and/ or futility**
Guidelines for the use of high-dose thiopentone for raised ICP

The use of thiopentone to control ICP is controversial with no clear evidence of benefit in terms of morbidity or mortality. (Cochrane database)
The current brain trauma foundation guidelines suggest that high-dose barbiturate therapy may be used to control elevated ICP refractory to maximum standard medical and surgical treatment. Hameodynamic stability is essential before and during barbiturate therapy.
Thus the decision to institute this therapy must be:
- made by a senior experienced doctor (consultant only)
- only considered for salvageable patients (i.e. those with a potentially good outcome)
- only considered on haemodynamically stable patients
- only considered after maximal medical therapies have been exhausted and surgical therapies are not an option

Dosing regimen
There is a wide variability in response to achieve EEG burst suppression, and an unclear end point as to what we are aiming for. Previous recommended regimens have been described for pentobarbital and what follows is based on them.
- Use the BIS monitor (this is to avoid overdose, and titrate the level of sedation.
A BIS level of approx 10 correlates with EEG burst suppression). Ensure the BIS monitor is working correctly. If unavailable follow the guidelines based on ICP alone until EEG monitoring is available. Repeated loading doses may be required to achieve adequate levels of sedation.
- Loading dose: 10mg/kg over 30 minutes.
  If ICP is not controlled and BIS > 10 further loading doses of 5mg/kg every half hour until either ICP is controlled or BIS < 10.

Once BIS < 10 the effect of thiopentone is maximal and further doses will not have any additional benefit, but increase the severity of side effects.
If ICP is still uncontrolled and BIS < 10 seek senior medical advice.
Once ICP is controlled commence an infusion.

The aim is control of ICP. If this is achieved with a BIS of 30 then there is no need to give further bolus doses of thiopentone.

- Infusion range: 1-8mg/kg/hr (start on 3-4 mg/kg/hr)
  Infusion should be titrated to control ICP.
  If ICP is not controlled and BIS > 10 give a further loading dose of 5mg/kg and increase the rate of infusion.
  If the ICP is controlled titrate the infusion to maintain a stable ICP.

If BIS < 5 reduce the rate of infusion even if ICP is unstable.
ONCE THE PATIENT’S ICP IS STABLE WEAN OFF PROPOFOL SLOWLY.

Weaning infusion: Once ICP has been stable for 24-48 hours (preferably 48 hours), thiopentone should be slowly weaned.
If there is a sustained rise in ICP on weaning discuss with NITU consultant (reload with thiopentone, use propofol infusion, osmotic therapy).
NOTE: The use of thiopentone should be titrated to target ICP and not a BIS level.

- If the ICP is stable with a BIS level of > 20 then there is no need to increase the dose of thiopentone.
- If the ICP is not stable and BIS is < 5 then there is no benefit in increasing the dose of thiopentone and alternative therapies should be considered.

**Thiopentone blood levels:** There is no need to routinely do thiopentone blood assays. They are only indicated for cases where the patient has not woken up after receiving an infusion and you are concerned that accumulation of the drug may be a contributory factor. The typical example, is to exclude thiopentone coma as a cause of a patient being unresponsive prior to brain stem tests.

**Complications of thiopentone coma:**
- Hypotension: correct with fluids, vasopressors +/- inotropes as necessary
- Sepsis
- Rhabdomyolysis
- Hypokalaemia at high doses. On stopping thiopentone rebound hyperkalaemia can occur
- Polyuria
**LICOX & ICP treatment Guidelines**

Aim for **ICP < 20mmHg and PbtO2 > 20mmHg**
(PbtO2 > 50mmHg may indicate hyperaemia)
If a concern about reliability of probe – perform a 100% oxygen test – increase inspired oxygen to 100% for a short period and observe if PbtO2 improves, if not probably is a faulty probe.

**ICP<20 & PbtO2>20**
Continue current therapy

**ICP>20 & PbtO2>20**
Ensure CPP>60mmHg
Aim to reduce ICP (see guidelines)
Ventilation, CO$_2$, temp, sedation

**ICP<20 & PbtO2<20**
Consider the following - no specific order
Increase CPP>65mmHg
Increase FiO$_2$ to 100% (up to 2hrs) then reduce slowly to maintain PbtO$_2$>20
LiDCO - ensure good CO
Hb>10g/dl, Temp 35-37°C
(Paracetamol/NSAIDs)
Increase CO$_2$ by 0.5kPa, provided ICP remains <20

**ICP>20 & PbtO2<20**
In this order:
Ensure CPP>60mmHg
Increase FiO$_2$ to 100%
Treat ICP according to guidelines
(osmotherapy, sedation, decompression, hypothermia, barbiturates)
If PbtO$_2$ < 20 persisting:
Increase CPP 70-80mmHg
Increase CO$_2$ by 0.5kPa
Hb> 10, temp 35-37°C
Consider: increasing sedation, hypothermia, craniectomy, barbiturates.
Use of Therapeutic Hypothermia in NICU

The indications for the use of the Coolgard 3000 are:
- To control temperature in a febrile patient with a neurological injury
- To induce moderate hypothermia in a patient with a head injury or CVA with raised intracranial pressure

Use central temperature monitor: oesophageal/ nasopharyngeal/ bladder or rectal

Use should only be started under the direction of the NICU consultant.

Indication: To control temperature in a febrile patient with a neurological injury
- Set machine target temperature at 36 or 37 degrees
- Ensure DVT prevention
- Routine care as per normal for that patient

Indication: To induce moderate hypothermia to control intracranial pressure
- Set machine target temperature at 34 to 35 degrees
- Ensure DVT prevention
- Administer 20 mmol MgSO4 IV.

Once machine is not being used, do not leave catheter in situ for more than 2 - 4 hours. Remove or exchange it for a conventional CVC line

The following physiological changes can occur, and should be monitored and treated accordingly.
- Reduction in cardiac output, mild acidosis and mildly increased lactate.
- Increased or decreased heart rate.
- Reduction in WCC, platelets and increased APTT.
- Increased risk of infection especially chest, especially if > 48 hours.
- Daily WCC and CRP and consider regular sputum cultures (every 2-3 days)
- Hypothermia induced diuresis and electrolyte depletion (esp K, Mg, PO4, Ca )
- Insulin resistance, requiring increased insulin requirements.
- Reduced drug metabolism.
- Electrolyte and fluid balance disorders
  - Monitor fluid balance, avoid hypovolaemia.
  - Electrolytes Twice daily including K, Mg, Ca, PO4
  - Maintain electrolytes in a high normal range
    - K > 4.5 mmol/l, Mg > 0.9 mmol/l, Adjusted Ca > 2.3 mmol/l, PO4 > 1.1 mmol/l
- Reduce propofol infusion rate by 30-50%. Add midazolam if needed.
- Monitor drug levels daily (phenytoin etc)
- Daily lipid profile
- Shivering may occur.
  Ensure that patient is adequately sedated, try clonidine (unless contraindicated or significant bradycardia), alternatively use paralysis.
  - If significant bradycardia occurs:
    Try glycopyrrolate or atropine. If resistant or severe, raise target temperature by 1 degree celsius.
Weaning off the coolgard 3000

The rate of re-warming is critical to avoid secondary injury. It should be slow and controlled. Select a target temperature of 36.5 degrees and controlled rate to \textbf{0.25 degrees per hour}. Once target temperature of 36.5 degrees has been achieved, leave the system running for a further 24 hours to prevent re-bound hyperthermia. After 24 hours at a controlled temperature of 36.5 degrees, stop the machine and remove the catheter, changing it for a CVC line as necessary.

Catheter use
The standard cool line cooling catheter may remain in-situ for 7 days and can be used in the internal jugular, subclavian and femoral vein. (Our experience is that it tends to work better in the SVC rather than the IJVC) There is a significantly increased risk of DVT associated with Coolguard use. It should be removed as soon as no longer required, and there should be a low threshold for Doppler examination following use if any suspicion of thrombosis.
Spinal Cord Injury Management

The following guidelines have been produced and should be completed on all patients with a spinal cord injury.

The diagnosis of an unstable spinal injury and its subsequent management can be difficult, and a missed injury can have devastating long-term consequences. All patients with significant injury should be considered at risk of spinal injury. The following is a brief summary of spinal immobilisation and is to be used in conjunction with already existing guidelines on NITU and General ITU with regards to clearance of the spine in trauma patients.

Spinal Immobilisation
Manual spinal protection should be instituted immediately

If the neck is not in a neutral position, an attempt should be made to achieve alignment. If the patient is awake and co-operative they should actively move their neck into alignment. If unconscious or unable to co-operate this is done passively. If there is any pain, neurological deterioration or resistance to movement the procedure should be abandoned and the neck splinted in the current position.

The spine board should be removed as soon as possible once the patient is on a firm trolley. Full immobilisation should be maintained and manual protection should be reinstated if restraints have to be removed for examination or procedures (eg. Intubation)

The log roll is the standard manoeuvre to allow examination of the back and transfer on and off backboards.

Rigid transfer slides (eg. PatSlides) are useful for transferring patients from one surface to another (eg. CT scan, operating table)

Patients who are agitated or restless may be impossible to immobilise adequately. Forced restraints or manual fixation of the head may risk further injury. It may be necessary to remove immobilisation devices and allow the patient to move unhindered. This should be a medical decision.

Spinal immobilisation should continue until documentation of spinal clearance in accordance with existing guidelines present on NITU and general ITU.
Management of spinal cord injury

**Within 4 hours from Admission:**
- Hard collar fitted correctly
- Vital capacity is measured
- Arterial line inserted
- Central line inserted
- Naso/orogastric tube is inserted & position confirmed
- Urine catheter inserted
- VTE prophylaxis
- Patient log-rolled & skin inspected, anal tone sensation assessed.

**Within 24 hours from Admission:**
- Surgical plan documented
- Correct bowel management prescribed and initiated
- Spinal clearance form filled
- ASIA assessment completed
- Trauma secondary survey completed
- Physiotherapy assessment
- Occupational therapy referral.

**Within 72 hours from Admission:**
- Repeat ASIA assessment
- Trauma Psychologist referral
- SLT referral
- Dietician referral
- Spinal Unit referral.

**Airway**
Look for neck swelling - cervical spine fractures can cause soft tissue swelling that can slowly compromise the airway - vital signs may be normal. Ask the patient if they breathe comfortably.

When securing the airway use always Manual-In-Line-Stabilisation (MILS). Glidescope or Airtraq can be useful aids but the use of Macintosh blades is not contraindicated (use the airway tool you are more confident with). The use of fibreoptic scope is not helpful in the acute management of the airway of spinal cord injured patients.

**Breathing**
Measure vital capacity twice daily on non-intubated patients.

**Ideal FVC:**
- Male 70ml/kg
- Female 55ml/kg
- Lesions C3-C6 vital capacity is 20% of normal
- Lesions T1-T4 vital capacity is 30-50% of normal

- Look for signs of fatigue
Consider use of prophylactic “birding” &/or BiPAP for lesions above T11
Consider ventilation if RR > 30 or VC < 15ml/kg
Some patients breathe better when lying completely flat.

Neurogenic pulmonary oedema may be present in high cervical cord injuries

Suxamethonium can be used up to 48 hours post-injury. After this time it can cause life threatening hyperkalaemia

Haemodynamic management
Spinal injury causes hypotension (neurogenic shock), bradycardia (T6 and above) and poikilothermia.
Hypotension may be due to combination of blood loss and vasodilation – so a central venous catheter would be helpful.
Exclude other injuries that can cause hypotension. A combination of fluid resuscitation and vasonstrictors may be needed. The use of LiDCo is helpful and is recommended.

Aim for MAP ~ 80-90mmHg.

Increased vagal activity may cause bradycardia (often triggered by airway manipulation): Pre-oxygenation and atropine are useful preventative measures.
Urine catheter insertion will allow an accurate measurement of fluid balance.

Gastrointestinal management
Paralytic ileus is common.
The incidence of aspiration may be as high as 35%
Enter a Naso/orogastric tube within 4 hours from admission.
Establish feeding slowly with the addition of pro-kinetics as needed.
Gastric acid protection is mandatory.
Commence early bowel care with two laxatives and PRN suppositories – please follow bowel management protocol as early as possible.

DVT prophylaxis
Prescribe DVT prophylaxis as per guidelines. Apply mechanical protection early if not contraindicated.

Spinal Immobilisation
Manual spinal protection should be instituted immediately
Spinal immobilisation should continue until documentation of spinal clearance in accordance with existing guidelines

Pressure areas and contractures
Allow the nurses and physiotherapists to do treat patients appropriately
Autonomic dysreflexia

Life threatening syndrome in spinal injuries above T6 (can be down to T10)
Pathological response to pain or noxious stimuli
Hypertension, bradycardia and vasodilation above level of cord injury. Facial flushing, headache, nasal congestion, blurred vision, nausea and diaphoresis.
Vasoconstriction below level of cord injury

Causes
- Bladder distension
- bladder infection
- faecal impaction
- pressure sores
- surgery on area below level of cord injury

Treatment
- Remove cause
- Treat hypertension (vasodilators)
- Analgesia

Steroids
Currently use of high steroid dose is not recommended.

Spinal Assessment
For prognosis it is important a formal documentation of spinal injury is documented. We use the ASIA score (American spinal injury association). All patients should have an assessment on admission OR as soon this is possible. The forms can be found on the NITU or the integrated care pathway for spinal cord injuries T8 and above. Teaching videos on ASIA scoring are available on: http://asia-spinalinjury.org/wp-content/uploads/2016/02/Motor_Exam_Guide.pdf

Referral
All patients with a spinal injury should be referred to all the spinal injury units, within 48 hours of admission. A proforma is on one of the desktop computers. Please speak to our specialist nurse OR any of our physiotherapists.
Aneurysmal Subarachnoid Haemorrhage

Deterioration in patients with subarachnoid haemorrhage can be due to a number of factors:
1. Rebleeding: usually only a problem with unsecured aneurysms
2. Hydrocephalus: often treated by an intraventricular drain (EVD)
3. Vasospasm causing cerebral ischaemia. Usually a focal deficit, but may present with generally decreased GCS
4. Intra-arterial thrombosis or embolism. More commonly seen after coiling. May require Reopro, aspirin, heparin or clopidrogel either singly or in combination
5. Neurogenic pulmonary oedema and cardiac stunning. May cause a low cardiac output, hypoxia and decreased brain perfusion, requiring ionotropes (typically dobutamine) +/- vasopressors (Noradrenaline).
6. Seizures. Re-bleeding may masquerade as a seizure
7. Electrolyte disturbances. Especially cerebral salt wasting syndrome, or less commonly SIADH.

Management
On admission:
1. Routine bloods plus Troponin and NT-proBNP
2. ECG
3. If serum Na < 135 mmol/L, send a urinary Na level
4. Fluids use 0.9% NaCl - 30 to 40ml/kg per day once normovolaemic

Medications
1. Nimodipine 60mg 4 hourly PO/NG for 21 days (If drops BP too much change to 30mg 2 hourly). Do not use IV (as it is associated with hypotension)
2. Fludrocortisone 0.1mg po TDS for 7 days
3. Paracetamol 1gm QDS PO/NG/IV
4. Bowel softeners and laxatives

Blood pressure
The brain loses autoregulation thus becomes reliant on a good blood pressure. If aneurysm is unsecured, a BP too high can contribute to re-bleeding.
In patients with unsecured aneurysms aim for MAP 70-90 or SBP 160-180
Patients with secured aneurysms MAP can be allowed to rise higher
Patients, who are thought to have deteriorated due to vasospasm, refer to next chapter.

Use the LiDCo to manage these patients

- Aim for serum Na > 140 and Hb > 8.0
Therapy for symptomatic vasospasm
Symptomatic vasospasm presents as a clinical deterioration in neurology post acute SAH; usually 4-10 days post bleed. Vasospasm occurs in the majority of patients who have a SAH (as assessed by angiogram) but only ~30% will become symptomatic. (The common term is delayed ischaemic deficit, DID) There is no evidence prophylactic HHH therapy confers any benefit in asymptomatic patients.
In asymptomatic patients ensure ABC’s. The patient is normovolaemic, well oxygenated, and well perfused with a good blood pressure.

Hypertensive therapy should only be used in symptomatic patients who have a secured aneurysm (i.e. not able to re-bleed) - either post-surgical clipping or post-radiological coiling.

Exclude other causes of neurological deterioration, such as re-bleed, hydrocephalus, seizures and fluid or electrolyte abnormality.

In patients post surgical clipping or radiological coiling it is appropriate to omit anti-hypertensives provided blood pressure is not too high Remember the ABC’s; there is no point in increasing cerebral blood flow if the patient is hypoxic.

Ensure the patient is well oxygenated. (SaO2 > 97%, PaO2 > 10 KPa)

Hypervolaemia
There is no evidence that hypervolaemia confers any benefit over normovolaemia in terms of cerebral blood flow. Thus the aim is not for aggressive fluid administration but to avoid hypovolaemia (usually by slightly excessive fluid administration and resuscitation). Note that most patients with an acute SAH are hypovolaemic on presentation to theatre or ITU. Unfortunately no indicator we currently have can tell us accurately a patient’s fluid status. Static parameters (CVP, PCWP) do not correlate with intravascular volume and more importantly do not tell us if administering more fluid will increase cardiac output and thus cerebral blood flow. Dynamic parameters such as stroke volume variation require positive pressure ventilation. Urine output is unreliable in this situation since a high percentage of these patients will have a cerebral salt wasting syndrome and thus be polyuric.
The best we can do is an educated guess based on all the combined parameters (BP, HR, CVP, Urinary output etc) and aim to slightly over doing it. Selecting a single parameter such as CVP and aiming for an arbitrary number is illogical and potentially dangerous.

Excessive fluid administration results in an unacceptably high incidence of pulmonary oedema and should be discouraged.
Remember acute SAH is associated with myocardial dysfunction / infarction even in patients who do not have significant risk factors for myocardial disease.

Haemodilution
The optimum haemoglobin level balancing flow, viscosity and oxygen delivery is unknown, but theoretically it is Hct of 0.30 (30%). Thus we aim for a Hct of 0.30, and transfuse blood if it is significantly below this level.
Hypertension
The definition of hypertension is not universally agreed. Common aims are for SBP > 180mmHg or increase in SBP or MAP by 20% above baseline. You have to assess each case individually with emphasis on their pre symptomatic BP and their cardiac function. Commonly we aim to increase SBP above their symptomatic level (typically by about 20%). If this does not produce the desired response we will raise it further. It is not unrealistic in this situation to be aiming for a SBP 200-220 mmHg.
Fluid administration will not commonly achieve a rise in BP unless the patient is hypovolaemic (a common situation). Thus patients will require inotropes. Currently there is no evidence to choose one over another, we commonly use Noradrenaline as our initial inotrope, but in difficult circumstances, and especially patients with myocardial injury it would seem sensible to employ a monitor of cardiac function (LiDCO).
In the presence of myocardial injury/stunning, we often require the addition of dobutamine to maintain a good cardiac output. Sometimes there is a clinical balance between increasing after-load and further myocardial injury which require careful discussion with senior ITU staff.

The principles behind managing SAH patients are:
- Good hydration
- Keep well oxygenated
- Good perfusion and blood pressure.

Like many other neurological conditions SAH results in loss of cerebral autoregulation, thus cerebral blood flow is directly related to perfusion pressure, and a decrease in perfusion pressure can result in deterioration while increasing perfusion pressure may improve CBF.

When to wean Hypertensive therapy

Although it is not very clear, some points are present:
1. Vasospasm is a mechanical narrowing of vessels that does not resolve quickly. It would be unrealistic to assume that if the deterioration was secondary to vasospasm it would resolve in a short period of time.
2. If a patient is not benefiting from therapy then there is probably no point continuing, but a senior colleague should make this decision.
3. If a patient develops a cerebral infarct, then continuing HHH therapy may result in an increase in cerebral oedema or haemorrhage.
4. If not responding to HHH therapy, we have limited options all the ones mentioned (angioplasty, SNP, barbiturates) are not readily available or experimental and should only be considered after discussion with a senior doctor.

Like most acute neurological conditions we prefer a period of stability before removing therapy, if HHH therapy has improved a patient’s condition it should not be withdrawn as soon as possible but when it is no longer needed.
**We usually maintain therapy while the patient is stable for 48-72 hours and then slowly withdraw therapy.**
If we decide the patient no longer needs therapy, we wean slowly. When we are weaning therapy we have made a conscious decision that they should be tested with a lower BP to see if they re-develop a neurological deficit. Thus weaning should be on the basis of neurology and provided the neurology remains good then we should
not be aiming for a specific BP. If there is neurological deterioration on weaning, then we should reintroduce HHHH therapy and continue for another 24-48 hours.

Symptomatic Vasospasm Post Acute SAH (a guide)
Often vasospasm is a diagnosis of exclusion.

Explain other causes of neurological deterioration

In patients who have a secured aneurysm & ventilated connect:

EUVOLAEMIA
HYPERTENSION
OXYGENATION

Neurological deficit resolves: continue hypertensive therapy for at least

No improvement after 6 hours: Increase the parameters.

No improvement

Consider CT/CTA/perfusion scan
Discuss with neuroradiology

• Daily routine ITU bloods (proBNP & TropI).
• Consider twice daily serum Na.

Patient unable to tolerate therapy e.g. cardiac ischaemia, arrhythmias
Extraventricular Drains (EVD’S)

How to take a CSF sample
1. Turn off the drain at the distal three-way tap
2. Identify sample port along tubing, closest to the patient, not the distal port.
3. Place sterile towel (from dressing pack) under sample port
4. Clean sample port and tubing with chlorhexidine. Wait for it to evaporate.
5. Put gloves on / change gloves. (The above steps may be slightly altered if you have some assistance to achieve the same goals. E.g putting gloves on while an assistant holds the tubing for you)
6. Carefully draw of 2 mls of CSF (over 2 min) and discard. Do not use needles. If CSF cannot be aspirate with ease then abandon procedure and discuss with specialist registrar.
7. Carefully draw of another 1-2mls of CSF (1-2 minutes), send this in a sterile universal container to microbiology for MC&S.
8. Ensure drain zeroed and the open all clamps.

How to give intrathecal vancomycin
1. Follow the above steps 1 to 7, however you may not need to send the sample to for MC&S.
2. Give the vancomycin via the sample port, over 2 minutes.
3. Draw up at least 2 ml normal saline and flush the tube slowly (over 1-2 minutes) to clear the dead space.
4. Turn of the proximal three way tap (The drain should now be completely off)
5. Inform the nursing staff of the time that the drain was turned off and ask them to open the drain in one hour.
6. Sign the drug chart that he drug has been given.
Convulsive Status Epilepticus

The initial management and presentation is covered in the “St George’s Grey Book”.
The duration of continuous seizure activity used to define status epilepticus has varied over time. Historically, the International League Against Epilepsy (ILAE) and others defined status epilepticus as a single epileptic seizure of >30 minutes duration or a series of epileptic seizures during which function is not regained between ictal events in a 30 minute period. Because of the clinical urgency in treating generalized convulsive status epilepticus (GCSE), however, a 30 minute definition is neither practical nor appropriate in clinical practice. Once seizures have continued for more than a few minutes, treatment should begin without further delay.

Considering the need for rapid evaluation and intervention in GCSE to avoid cardiovascular morbidity and refractory status, an accepted operational definition of GCSE consists of the following:

- ≥5 minutes of continuous seizures, or
- ≥2 discrete seizures between which there is incomplete recovery of consciousness

In 2015, the ILAE published a revised conceptual definition of status epilepticus that incorporates two operational dimensions, t1 and t2.

- Status epilepticus is a condition resulting from either the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms that lead to abnormally prolonged seizures (after time point t1)
- Status epilepticus is a condition that can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures

Definitions:

Generalised convulsive status epilepticus: These are convulsions with generalised rhythmic jerking of the limbs and mental status impairment.

Status epilepticus (SE): This is a seizure lasting > five minutes, according to the revised definition. This will inevitably include some patients with prolonged seizures not fulfilled traditional criteria (>30 minutes duration).

Non-convulsive status epilepticus: This is seizure activity on EEG without clear convulsive clinical features; this includes severely ill patients with ‘subtle status’ – note that ‘wandering confused’ patients are not included here.

Refractory status epilepticus: Status epilepticus that does not respond to traditional or first-line treatment regimens that include an adequate trial of one benzodiazepine and one appropriate non-benzodiazepine anticonvulsant. Convention of diagnosis is status epilepticus exceeding two hours despite treatment.

New-onset RSE (NORSE): prolonged period of refractory seizures with no readily identifiable cause in otherwise healthy individuals.
Super-refractory SE: SE which recurs or continues despite adequate continuous infusion of sedation + an anticonvulsant for > 24 hours. Inadequate dosing is often a cause of perceived refractory SE.

AETIOLOGY

Most cases of status epilepticus in adults are symptomatic of an underlying structural brain lesion or a toxic or metabolic disturbance. Many episodes come from a combination of an earlier lesion and a superimposed new metabolic, infectious, or pharmacologic stressor such as uremia or a medication change.

Status epilepticus also commonly arises in patients with an established diagnosis of focal or generalized idiopathic epilepsy. Status epilepticus is occasionally the presenting manifestation of epilepsy.

Common causes of convulsive status epilepticus:

- Acute structural brain injury (eg, stroke, head trauma, subarachnoid hemorrhage, cerebral anoxia or hypoxia), infection (encephalitis, meningitis, abscess), or brain tumour. Stroke is the most common, especially in older patients.
- Remote or longstanding structural brain injury (eg, prior head injury or neurosurgery, perinatal cerebral ischemia, cortical malformations, arteriovenous malformations, and benign brain tumors).
- Antiseizure drug nonadherence or discontinuation in patients with prior epilepsy.
- Withdrawal syndromes associated with the discontinuation of alcohol, barbiturates, or benzodiazepines.
- Metabolic abnormalities (eg, hypoglycemia, hepatic encephalopathy, uremia, hyponatremia, hyperglycemia, hypocalcemia, hypomagnesemia) or sepsis.
- Use of, or overdose with, drugs that lower the seizure threshold (eg, theophylline, imipenem, high dose penicillin G, cefepime, quinolone antibiotics, metronidazole, isoniazid, tricyclic antidepressants, lithium, flumazenil, cyclosporine, lidocaine, bupivacaine).

It is important to terminate seizure activity promptly because the longer the fit lasts the more difficult it is to stop: this is time-dependent pharmacoresistance.

This algorithm is a continuation of the status epilepticus protocol found in the St Georges guidelines booklet and is only for use within the intensive care setting by doctors competent at airway management.

It should be implemented with the full involvement of the on-call neurology team.

Treatment of status epilepticus in adult ICUs

Stage 1:

- Position patient to avoid injury
- Administer oxygen via FM
- Establish IV access
- Administer **Lorazepam 4mg IV; rate 2mg/min, up to 8mg/kg** (alternatives when cannot obtain IV access: Diazepam 10-20mg PR or Midazolam 10mg IM).
- Administer **IV fluids**
- **Send bloods for:** U&Es (Na, K, Ca, Mg, Glucose), FBC, Clotting screen, Arterial Blood Gas, Toxicology (blood & urine), Serum drug levels (if already on anticonvulsants)

Consider Pabrinex (1 pair of ampoules in malnourished or alcohol-related)

**If seizures persist consider admission to Neuro Intensive Care unit**
**Call Neurology SpR on call**

**Stage 2: Neuro Intensive Care**

- **Repeat bloods (including PRIS monitoring)**
- **Give or optimise initial anticonvulsant therapy** (if inadequate dose has been given, administer the rest):

<table>
<thead>
<tr>
<th>Anticonvulsant</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenytoin</td>
<td>18-20mg/kg IV</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>30-40mg/kg IV</td>
</tr>
<tr>
<td>Sodium Valproate</td>
<td>20-40mg/kg IV</td>
</tr>
</tbody>
</table>

- **Continue regular anticonvulsants**

**Discuss with on-call neurology SpR. Intubate, ventilate and sedate**

### Induction:
- Propofol 1.5-2.5mg/kg
- Fentanyl 1-2mg/kg
- Suxamethonium 1-2mg/kg (unless contra-indicated)

### Sedation:
1. Propofol up to 5mg/kg/hr
2. Fentanyl up to 5mcg/kg/hr
   - Avoid paralysis, though this may stop visible fits, cerebral seizure activity may continue.
3. Ensure electrolytes are normal
4. Continue antiepileptic medication and check levels
5. Maintain blood pressure at normal or supra-normal levels (+/- inotropes)
6. Maintain normocapnia and adequate oxygenation
7. Seek cause

### Persistent seizures: up to 60 minutes from onset
- Midazolam bolus 0.2mg/kg bolus then infusion up to 0.5mg/kg/hr

If all other measures fail:
- Refer to Refractory Status epilepticus guidelines (New Onset RSE)
Continue anaesthetic for 12-24 hours post termination of last clinical or electrographic seizure and then wean sedation and extubate. EEG monitoring is important as non-convulsive status (NCS) may be or become present. (Non-convulsive status: no seizure activity is seen but activity continues at neuronal level. NCS is a cause of continuing coma after weaning of sedation)

References


Refractory Status Epilepticus in Adults

Refractory status epilepticus is defined as ongoing convulsive or nonconvulsive seizures following administration of an initial benzodiazepine and a nonbenzodiazepine antiseizure drug, given in appropriate doses. Whereas there is reasonable agreement upon the initial treatment of generalized convulsive status epilepticus (GCSE), the optimal treatment of refractory status epilepticus is more controversial; there are no randomized trials comparing various treatments. Regardless of the specifics of pharmacologic therapy, it is critical to provide adequate ventilatory and hemodynamic support. Patients with refractory status epilepticus should be intubated and monitored with continuous electroencephalogram (EEG).

Status Epilepticus Severity Score (STESS)

<table>
<thead>
<tr>
<th>Features</th>
<th>STESS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consciousness</strong></td>
<td></td>
</tr>
<tr>
<td>Alert or somnolent/confused</td>
<td>0</td>
</tr>
<tr>
<td>Stuporous or comatose</td>
<td>1</td>
</tr>
<tr>
<td><strong>Worst seizure type</strong></td>
<td></td>
</tr>
<tr>
<td>Simple-partial, complex-partial, absence, myoclonic</td>
<td>0</td>
</tr>
<tr>
<td>Generalized-convulsive</td>
<td>1</td>
</tr>
<tr>
<td>Non-convulsive status epileptic in coma</td>
<td>2</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
</tr>
<tr>
<td>&lt;65</td>
<td>0</td>
</tr>
<tr>
<td>≥65</td>
<td>2</td>
</tr>
<tr>
<td><strong>History of previous seizures</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0</td>
</tr>
<tr>
<td>No or unknown</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>0-6</td>
</tr>
</tbody>
</table>

**STESS 0-2:** Favourable outcome (P<0.001)
**STESS 3-6:** Unfavourable outcome

**STESS has an excellent negative predictive value – can tell you accurately who is going to do well!!!**
New onset Refractory Status Epilepticus

Make sure you have followed stage 1 and stage 2 steps of status epilepticus

Treatment

Involve Neurology SpR

- Begin a continuous infusion of general anaesthetic
  - Treatment goal: complete suppression of seizure activity - aim for burst suppression with high suppression to burst ratio
  - EEG monitoring is paramount (preferably continuous or intermittent).
  - Continue General Anaesthesia for at least 24 hours
  - Start an IV steroid
    - Methylprednisolone 1g IV/day for 3 days followed by 1mg/kg/day IV.

- LiDCo
- Antithrombotic therapy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Bolus</th>
<th>Continuous infusion</th>
<th>Things to consider</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midazolam</td>
<td>0.1-0.6mg/kg at a rate 2mg/min</td>
<td>0.1-0.4mg/kg/h can go up to 1.2mg/kg/h increase rate by 0.1mg/kg/h every 4 hours until flat EEG or burst suppression</td>
<td>Hepatic metabolism Tachyphylaxis occurs</td>
</tr>
<tr>
<td>Propofol</td>
<td>2-5mg/kg</td>
<td>5-10mg/kg/h Avoid using above 5mg/kg/h for more than 48 hours Increase infusion rate by 0.2mg/kg/h until flat EEG or burst suppression.</td>
<td>Involuntary movements Propofol infusion syndrome: check serum lactate (&lt;2.5), triglycerides (&lt;5), CK (&lt;2000). Use ideal body weight.</td>
</tr>
<tr>
<td>Thiopentone</td>
<td>1-3 mg/kg</td>
<td>1-5mg/kg/h</td>
<td>Zero order pharmacokinetics Needs loading with repetitive boluses and accumulates in adipose tissue.</td>
</tr>
</tbody>
</table>

Stage 3: Ongoing management in Neurointensive Care Unit

- Start maintenance anticonvulsant during continuous anaesthetic infusion
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose/Route</th>
<th>Caution/ Things to consider</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenytoin</td>
<td>100mg TDS, IV until stable levels</td>
<td></td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>1000mg-1500mg BD, IV/NG</td>
<td></td>
</tr>
<tr>
<td>Sodium Valproate</td>
<td>10mg/kg divided in 2-3 doses requires loading dose:20-30mg/kg/min</td>
<td>Avoid in liver disease, Mitochondrial disease, porphyria.</td>
</tr>
</tbody>
</table>

- **AFTER 24 hours of continuous anaesthetic in adequate dose (according to EEG):**
  - Gradually withdraw the anaesthetic infusion (over 6-12hours), optimally under continuous EEG monitoring while continuing the antiepileptic drugs.
- **If there is reoccurrence of SE**
  - Transfer patient in NITU (if not already there)
  - Repeat continuous infusion of GA over 48hours (think using a different agent).
  - Consider hypothermia, magnesium, IVIG, Plasma exchange, neurosurgery (see table below).

### Intervention/management | Things to consider
---|---
Hypothermia | 32°C - 35°C **for <48hours**
Do not use high doses of propofol & hypothermia at the same time.

Magnesium | 20mmol IV and continuous infusion 10mmol/h.
Target 3.5mmol/l.
Risk of neuromuscular blockade.

IVIG & Plasma exchange | If autoimmune encephalitis is possible (NORSE without a structural or infectious cause).
IVIG:0.4g/kg over 5 days

Neurosurgery | Lesional SE

The most common causes of pseudo-refractory SE are:
- **Inadequate drug dosage** (failure to suppress EEG activity)
- **Wrong diagnosis** (non-epileptic attacks or paroxysmal autonomic dysregulation)

- **If SE continues despite applying the above steps correctly:**
  - Re-imaging
  - Review EEG again
  - Add Ketamine: loading dose 3mg/kg. Maintenance 1-5mg/kg/hr.
    Potential neurotoxicity-use in combination with benzodiazepines.
    Electroconvulsive therapy
  - Consider Ketogenic diet (**Should NOT BE USED** in conjunction with propofol). 4:1 ratio, check for ketonouria.
Discuss more “extreme” approaches: Lidnocaine, Verapamil, Isoflurane, Vagal nerve stimulation, Isoflurane.

**Common treatment errors:**
SE was never controlled EEG-wise
SE was controlled but no maintenance or inadequate anticonvulsant therapy prior to stopping GA agents
SE was controlled but withdrawal of sedatives was too rapid.

**References**


Summary of Management of Status Epilepticus

Manage status according to St. George's Grey Book

consider admitting to NITU

On Admission to NITU

max Midazolam & Propofol
Maintenance dose of anticonvulsants (maximum 2 agents)
Diagnosis: repeat LP
EEG to check control of seizures

Day 2 on NITU

Wean slowly GA agents
Consider steroids
MRI
Use continuous EEG

Day 3 on NITU:

Consultant to consultant discussion & planning
consider plasma exchange or other management

Treatment Targets:

**Primary aim:** control seizures
**Secondary aim:** neuroprotection
**Third aim:** avoid or treat systemic complications of prolonged unconsciousness and prolonged anaesthesia.

Do not stop treatment unless there is evidence of irreversible brain damage
Use EEG (preferably continuous) to avoid inadequate suppression of seizure activity
Exclude other causes that can mimic SE.
DO NOT change antiepileptic drugs too often.
Continuous EEG in Critical Care

Critical illness
- Generalized convulsive status epilepticus
- Refractory status epilepticus

Neurological illness (other than status epilepticus)
- Cardiac arrest
- Traumatic brain injury
- Subarachnoid haemorrhage
- Encephalitis
- Intracerebral haemorrhage
- Ischaemic stroke
- All neurological illnesses

Non-neurological illness
- Comatose patients without primary brain injury

Continuous EEG
- No return to neurofunctional baseline after antiepileptic therapy and/or concern for ongoing seizures
- Unexplained altered level of consciousness
- Persistent coma
- Undetermined
- Rhythmic and periodic patterns along the ictal-interictal continuum

Indications of CEEG IN ICU
- Treatment of Status Epilepticus
- Management of burst suppression
- Detection of subclinical seizures
  - After convulsive status epilepticus-50% in 1 hr, 80% in 24hrs, 87% in 48 hrs
  - Non convulsive status Epilepticus -8% in TBI,70% in SAH,42% in HIE
- Detection of Evolving Ischemia
  - After subarachnoid haemorrhage-Total power and alpha variability in CEEG
  - Acute stroke- risk Maximum at 24-48hrs
- Other conditions
  - Intracranial Haemorrhage-pts with seizures/fluctuating GCS
  - Encephalitis-seizures in 88% in 24hrs & 93% in 48hrs
  - TBI
  - Cardiac arrest
  - During and after vascular neurosurgical procedures
  - During and after interventional neuro radiology procedures
  - In patients with hemodynamic lesions and borderline flow
  - In other patients at risk for in-hospital acute ischemia
- Assessment level of sedation
- Prognostication
Indications

1. Status Epilepticus:
   - Following generalized convulsive status epilepticus (GCSE) in patients with persistent encephalopathy to assess for ongoing non convulsive status epilepticus (NCSE).
   - For monitoring the response of SE to treatment, especially when using continuous intravenous anaesthetic medications.
   - To assess for NCSE in patients with unexplained coma or altered mental status.
   - For determination of whether repetitive, involuntary movements represent SE versus non epileptic events.

2. Ischemic Stroke:
   - Stroke patients in the ICU who are candidates for cEEG monitoring should include: Fluctuating neurologic deficits, unexplained coma or altered levels of consciousness.
   - Malignant increased intracranial pressure requiring induced coma or paralytics.
   - Treatment modalities that preclude reliable neurologic examination such as Paralytics for respiratory indications.
   - Unexplained “spells” such as variations in vital signs or twitching.
   - Seizures or status epilepticus that require active adjustment of therapy.

3. SAH:
   - Patients with persistent coma or unexplained neurological deterioration following SAH
   - Detect nonconvulsive seizures and NCSE.
   - The use of cEEG for the detection of DCI as a compliment to the clinical neurological exam, TCD, and radiographic evaluations.
   The duration of cEEG monitoring after aSAH is dependent upon the indication for monitoring.
   A minimum of 24 to 48 hours of monitoring is required in order to detect nonconvulsive seizures while more prolonged monitoring over several days or weeks is needed to monitor for DCI.

4. ICH:
   Recommendations for cEEG in Patients with ICH
   - Any patient with suspected seizure
   - Abnormal or fluctuating mental status
   Duration of up to 72 hours

5. Encephalitis:
   - Patients with fluctuating levels of consciousness
   - Patients with seizure presentation
   - Patients with lateralised periodic discharges need longer duration of monitoring
6. TBI:
CEEG useful if
- GCS score less than 10
- Cortical contusion
- Depressed skull fracture
- Subdural hematoma
- Epidural hematoma
- Intracerebral hematoma
- Penetrating head wound
- Seizure within 24 hours of injury

7. Prognostication:

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>POOR OUTCOME</th>
<th>GOOD OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracranial haemorrhage</td>
<td>PDs, LPDs, SIRPIs</td>
<td></td>
</tr>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>No reactivity, no state change first 24 hr, NCSE within 24 hr, GPDs, BIPDs</td>
<td>Presence of sleep, architecture, state changes, reactivity, absence of PDs</td>
</tr>
<tr>
<td>Nonconvulsive status epilepticus</td>
<td>Generalized SE, burst-suppression, LPDs, ictal discharges after control of SE</td>
<td>Focal SE, normalization of EEG background</td>
</tr>
<tr>
<td>Cardiac arrest without therapeutic hypothermia</td>
<td>Myoclonic SE within the first 24 hr, generalized suppression less than 20 mV/mm, burst-Suppression pattern, GPDs on flat background, alpha/theta/spindle coma without reactivity</td>
<td>Presence of reactivity</td>
</tr>
<tr>
<td>Cardiac arrest after therapeutic hypothermia</td>
<td>No reactivity, early myoclonus, burst-suppression, generalized suppression, SE</td>
<td>Continuous EEG background with generalized slow-wave activity, reactivity</td>
</tr>
</tbody>
</table>
Length of CEEG

Correlate clinically as may need further periods discuss with Consultant and Neurophysiology team.
1. If level of consciousness stable and awake – Max 24hrs
2. If fluctuating GCS or Depressed -72 hrs

Electrode placement

10-20 System of Electrode Placement

- F = Frontal
- P = Parietal
- T = Temporal
- O = Occipital
- C = Central
  A = Auxiliary

- Odd # = Left
- Even # = Right

References:
EEG Monitoring in ICU – S.LORCHE
Nature reviews - Neurology
Neuro critical Care society Guidelines
Critical care EEG monitoring research Consortium
**Intracerebral Haemorrhage**

Key points:
The second most common form of stroke (15–30% of strokes)
the volume of the hematoma correlates highly with morbidity and mortality
the clot enlarges in at least 33% of cases within the first 3 hours of onset
Common site: 50% (Basal ganglia); internal capsule, globus pallidus, 15% Thalamus, 10-15% Pons, 10% cerebellum.
Causes: hypertension, drugs, alcohol, leukemia, previous stroke, vascular abnormalities, tumors, history of recent trauma.
Complications: rebleeding, edema, hydrocephalus, intraventricular extension, seizures
Clot volume carries prognostic significance.
Investigation of choice – CT scan, cerebral angiogram as warranted. MRI/MRA in certain scenarios (discuss with Neuroradiologist if needed)

**Initial Management:**
Patients need ICU care. Close monitoring especially cerebellar Bleed

1. Hypertension: may contribute to further bleeding, especially within the first. Suggested target BP ≈ 140/90. Current trials to assess if acute BP reduction within 8-12 hrs of presentation will reduce Haematoma expansion are ongoing. Avoid overcorrection.

2. Monitor need for airway support, euglycemia, normothermia, anticonvulsants - also when lobar haemorrhage noted, Seizures are treated with appropriate AEDs like Levetiracetam, phenytoin.

3. Haemostatic issues: correct coagulopathies,

4. Steroids: No significant benefit from prophylactic dexamethasone in ICH, but noted to have significantly more complications. May need to be used if significant peri haemorrhage oedema on imaging.

5. For raised ICP secondary to intraventricular bleed or hydrocephalus may need mannitol/frusemide or external ventricular drain (EVD) and ICP monitoring.

6. Surgical Intervention:
**Factors that favour medical management:**
- Minimally symptomatic lesions. situations with little chance of good outcome-high ICH score, massive haemorrhage with significant neuronal destruction, large haemorrhage in dominant hemisphere, poor neurologic state.
- Basal ganglion (putamin) or thalamic haemorrhage: surgery is no better than medical management.

**Factors favouring surgical intervention:** with the exception of cerebellar Haemorrhage and superficial lobar haemorrhage (less than 1 cm from cortical surface) surgery generally not indicated but some situations may need as like:
- marked mass effect/ midline shift on imaging (removal is considered due to the potential for herniation)
Lesions where the symptoms (e.g. hemiparesis/plegia, aphasia, or sometimes just confusion appear to be due to increased ICP or to mass effect.

Symptoms attributable directly to brain injury from the haemorrhage are unlikely to be reversed by surgical evacuation.

Moderate volume: surgery for moderate volume hematomas (i.e. > 10-30 cc)

Persistent elevated ICP in spite of therapy (failure of medical management). Evacuating clot definitely lowers ICP, but the outcome is uncertain.

Rapid deterioration (especially with signs of brain stem compression) regardless of location.

**Cerebellar Haemorrhage: Recommendations:**

Patients with a Glasgow Coma Scale (GCS) score ≥ 14 and hematoma <4 cm diameter treat conservatively

Patients with GCS ≤ 13 or with a hematoma ≥4 cm: surgical evacuation

Patients with absent brain stem reflexes and flaccid quadriplegia: intensive therapy is not indicated. But debatable as loss of brain stem reflexes from direct compression may not be irreversible, and that cerebellar haemorrhage represents a surgical emergency (and that the above criteria would thus deny potentially helpful surgery).

Patients with hydrocephalus: ventricular catheter (if no coagulopathy). Caution: do not over drain to avoid upward cerebellar herniation. Most cases with hydrocephalus also require evacuation of the clot.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Finding</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCS score</td>
<td>3–4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>5–12</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>13–15</td>
<td>0</td>
</tr>
<tr>
<td>Age</td>
<td>≥ 80 years</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;80</td>
<td>0</td>
</tr>
<tr>
<td>Location</td>
<td>infratentorial</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>supratentorial</td>
<td>0</td>
</tr>
<tr>
<td>ICH volume</td>
<td>≥ 30 cc</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>&lt;30 cc</td>
<td>0</td>
</tr>
<tr>
<td>Intraventricular blood</td>
<td>yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>no</td>
<td>0</td>
</tr>
<tr>
<td>ICH Score</td>
<td>Total Points 0–6</td>
<td></td>
</tr>
<tr>
<td>ICH Score</td>
<td>30 day mortality</td>
<td></td>
</tr>
<tr>
<td>0</td>
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</tr>
<tr>
<td>1</td>
<td>13%</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>26%</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>72%</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>97%</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>100%</td>
<td></td>
</tr>
</tbody>
</table>

**References:**

2. World stroke Association –ESO guidelines 2014
Contrast nephropathy

Contrast nephropathy refers to an acute kidney injury occurring soon after (within 24–48 hours) the administration of intravascular radiocontrast material. Its pathogenesis is not completely understood, but is most likely due to acute tubular necrosis arising from the vasoconstricting and cytotoxic effects of radiocontrast material. Although generally reversible, it may be problematic in the context of critical illness and/or other risk factors for renal injury.

Identifying patients at risk of contrast nephropathy

Patients with normal or near-normal renal function, who are euvoalaemic and do not have other significant risk factors for renal injury, are at little risk of contrast-induced nephropathy. No special precautions are required in these patients.

Risk factors for contrast nephropathy include established renal disease (acute or chronic), other risk factors for renal injury, and factors related to the proposed radiological study or procedure.

Established renal disease

- **Chronic kidney disease**: the incidence of contrast nephropathy is directly related to the severity of existing renal impairment (incidence 22% in patients with serum creatinine concentration 177–260 μmol/L; 31% with serum creatinine >260 μmol/L)
- **Diabetic nephropathy** appears to confer a higher risk than non-diabetic nephropathy, for the same serum creatinine concentration
- **Acute kidney injury** is likely to increase the susceptibility to nephrotoxic effects of radiocontrast material, although the complexity of such patients makes risk stratification difficult

Other risk factors for renal injury

- Any state of reduced renal perfusion, whether acute (e.g. shock) or chronic (e.g. advanced heart failure)
- Concurrent treatment with nephrotoxic drugs (e.g. aminoglycosides, non-steroidal anti-inflammatory drugs)
- Myeloma

Factors related to the radiological procedure

The incidence of contrast nephropathy is dose-dependent, and varies with the type of contrast material used. The highest risk is associated with ionic hyperosmolal agents, and the lowest risk with nonionic iso-osmolal agents. At the time of writing, the agent used at St George’s is Omnipaque (iohexol), which is a nonionic low-osmolol (*not* iso-osmolal) agent, with osmolality 672 or 844 mOsm/kg for Omnipaque 300 or 350, respectively.
Studies involving intra-arterial injection (e.g. angiography) are associated with higher risk than those involving intravenous injection (e.g. CT scans). Percutaneous coronary interventions present a particularly high risk due to the high doses of contrast used (especially with left ventriculography), its intra-arterial injection, and the risk of atheroemboli associated with the procedure itself.

**Evaluating the risk–benefit profile of the proposed study or procedure**

Contrast-enhanced studies should not be avoided solely because of the risk of contrast nephropathy. Rather, case-by-case judgments must be made, weighing the risk of contrast administration (and the other risks of the procedure and radiation exposure, as applicable) against its anticipated benefits.

Where possible, studies that do not require contrast administration should be considered (e.g. ultrasonography, or CT or MR imaging without contrast). Magnetic resonance imaging with paramagnetic contrast administration should generally be avoided in patients with renal impairment, due to the risks of nephrotoxicity (similar to that seen with radiocontrast) and nephrogenic systemic fibrosis.

**Interventions to the reduce the risk of contrast nephropathy**

In patients at risk of contrast nephropathy, in whom it has been determined that the benefits of a contrast-enhanced study outweigh its risks, the following options may be considered to reduce the risk of adverse renal effects:

- Ensure radiology are aware of the risk, as they may be able to reduce the contrast dose or, possibly, select an agent with lower propensity to cause renal injury
- **Ensure euvoilaemia and avoid other nephrotoxins** if possible (especially non-steroidal anti-inflammatory drugs)
- In the absence of contra-indications, consider **plasma volume expansion** immediately prior to the study, by administering 500 mL of intravenous crystalloid solution, less than 30 minutes before contrast administration (see below for a discussion of fluid choice)
- Consider continuing intravenous crystalloid infusion for 6 hours post-procedure (e.g. at approximately 1 mL/kg/hr)

The choice of intravenous fluid for peri-procedural volume expansion will be influenced by the individual circumstances. Theoretical considerations suggest that sodium bicarbonate (near-isotonic, e.g. 1.4% or 1.26%) may be preferable, although clinical data are equivocal. Sodium chloride 0.9% and Hartmann’s solution (sodium lactate IV infusion, compound) are alternative and probably equally acceptable options, depending on other factors particular to the case (avoid Hartmann’s solution in patients with, or at high risk of, raised intracranial pressure).

The data on the use of acetylcysteine (oral or IV) are conflicting. Although there is evidence that it may reduce the incidence of contrast nephropathy (as defined by serum creatinine concentration), the quality of studies has been variable and their results inconsistent. IV acetylcysteine is associated with a risk of anaphylatoid reaction, and enteral administration the day before the procedure is often impractical.
in a critical care setting. On balance, therefore, our practice is not to administer acetylcysteine routinely to patients at risk of contrast nephropathy. If, on an individual risk–benefit assessment, and after discussion with the NICU consultant, it is decided that acetylcysteine should be administered, the dosage regimen is 1200 mg orally (or by enteral tube) twice daily for 2 days, starting the day before the study (i.e. four 1200-mg doses).

References


Thromboprophylaxis

Risk assessment

Patients requiring neurointensive care are at high risk of venous thromboembolism (VTE). They may also be at high risk of bleeding complications. These risks may change substantially over short timeframes. As such, regular assessment of the risk–benefit balance of thromboprophylaxis is essential.

At minimum, the VTE and bleeding risk should be assessed (and appropriate prophylaxis instituted) at admission and around 24 hours after admission, and reassessed after any significant change in the patient’s condition.

Initial risk assessments (within 10 hours of admission and within 24 hours of admission) are triggered automatically on the iCLIP system, where they must be completed (a guide may be found here). The relatively crudely-specified risk factors in the electronic document mean that all patients will (appropriately) be categorised as ‘at risk of thrombosis’ (by virtue of their critical care admission), and many will be ‘at risk of bleeding’ (e.g. due to neurosurgery, active bleeding, or another risk factor for bleeding). Thus while the electronic risk assessment is required, it does not replace the need for an individualised risk–benefit assessment for that patient, taking account of the subtleties of their case. This risk assessment, and the decision on the type of thromboembolism prophylaxis required, should be a part of the daily review, and documented accordingly in the physical notes.

Non-pharmacological thromboprophylaxis

Non-pharmacological measures to reduce the risk of VTE include:

- **Mobilisation**, which offers numerous advantages beyond those associated with VTE risk, and should be a priority in all patients, wherever feasible
- **Full length graduated anti-embolism stockings** should be fitted to all patients in the absence of contraindications (see box)
- **Intermittent pneumatic compression devices (in addition to anti-embolism stockings)** should be considered in all patients who are not receiving pharmacological thromboprophylaxis, in the absence of contraindications (see box)

Where it is not possible to fit full length antiembolism stockings or intermittent pneumatic compression devices, calf stockings should be considered.
Contraindications to mechanical thromboprophylaxis

- Severe leg oedema
- Pulmonary oedema due to cardiac failure
- Peripheral arterial bypass grafting
- Peripheral vascular disease
- Fabric allergy
- Major limb deformity preventing correct fit
- Unusual leg size or shape
- Recent skin graft
- Severe dermatitis

*Exercise caution* in venous ulceration/wounds.

Patients with [acute stroke should have intermittent pneumatic compression devices](#), but *not* anti-embolism stockings (risk of ulceration and blistering outweighs benefits).

Pharmacological thromboprophylaxis

All critical care patients should be considered for pharmacological thromboprophylaxis. As the VTE risk is high in almost all cases, it is the bleeding risk that principally determines whether pharmacological thromboprophylaxis is indicated.

The agent used for pharmacological thromboprophylaxis at St George’s is [dalteparin](#) (a low molecular weight heparin) or, in the context of significant renal impairment, [unfractionated heparin](#). See box for dosing instructions. Fondaparinux (2.5 mg SC daily) is an option for patients who are unable to take heparin due to allergy or a history of heparin induced thrombocytopenia — this should be discussed with haematology.

**Pharmacological thromboprophylaxis**

Choice of agent and dosage (in patients in whom pharmacological thromboprophylaxis is judged to be indicated)

<table>
<thead>
<tr>
<th>Most patients (who are not among the ‘special cases’ listed below)</th>
<th>Dalteparin 5000 units SC daily</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Special cases:</strong></td>
<td></td>
</tr>
<tr>
<td>Body weight 101–150 kg</td>
<td>Dalteparin 5000 units SC 12-hrly</td>
</tr>
<tr>
<td>Body weight &gt;150 kg</td>
<td>Dalteparin 7500 units SC 12-hrly</td>
</tr>
<tr>
<td>Renal impairment with creatinine clearance &lt;30 mL/min (calculated using the <a href="#">Cockcroft-Gault equation</a>)</td>
<td>Heparin 5000 units SC 12-hrly</td>
</tr>
<tr>
<td>Patients unable to take heparin due to allergy or history of heparin-induced thrombocytopenia</td>
<td><em>Discuss with haematology regarding the use of fondaparinux 2.5 mg SC daily</em></td>
</tr>
</tbody>
</table>
Neurosurgical patients

Always review the clinical notes and post-operative plan for specific instructions on when or whether to start pharmacological thromboprophylaxis.

In the absence of specific instructions, aim to start pharmacological thromboprophylaxis *with agreement of the neurosurgical team*, provided there are no other bleeding risk factors (including a significant risk of time-critical invasive procedures), at the following time points:

<table>
<thead>
<tr>
<th>Scenario</th>
<th>When to consider initiating pharmacological thromboprophylaxis*</th>
</tr>
</thead>
<tbody>
<tr>
<td>After elective neurosurgical procedures</td>
<td>24 hours post-op, unless specific instructions or circumstances suggest an increased risk of bleeding (if imaging is planned on post-operative day 1, defer administration until this has been performed and reviewed)</td>
</tr>
<tr>
<td>After securing a cerebral aneurysm</td>
<td>24 hours after the aneurysm is secured, provided the risk of bleeding is not unusually high, and time-critical invasive procedures are unlikely (e.g. withhold if there is hydrocephalus that might necessitate urgent placement of an external ventricular or lumbar drain)</td>
</tr>
<tr>
<td>Traumatic brain injury <em>not</em> associated with significant haemorrhagic component</td>
<td>48 hours post injury, provided time-critical invasive procedures are unlikely (e.g. withhold if intracranial hypertension that might require EVD placement)</td>
</tr>
<tr>
<td>Traumatic brain injury <em>with</em> significant haemorrhagic component</td>
<td></td>
</tr>
<tr>
<td>Spontaneous intracerebral haemorrhage</td>
<td>Should be considered on a case-by-case basis</td>
</tr>
<tr>
<td>Acute spinal cord injuries</td>
<td></td>
</tr>
<tr>
<td>Decompressive hemicraniectomy</td>
<td></td>
</tr>
</tbody>
</table>

*After discussion with the neurosurgical team*

Patients with acute ischaemic stroke

In patients with acute ischaemic stroke, evidence indicates net benefit from intermittent pneumatic compression devices, but *not* anti-embolism stockings. Clinical trial data also indicate that low-dose low molecular weight heparin (<6000 units/day) reduces the risk of DVT and PE, without a significantly increased risk of haemorrhage, although clinical practice varies in this respect.

Our preferred practice for patients with acute ischaemic stroke is:
- **Intermittent pneumatic compression devices** should be fitted in all cases, unless contraindicated.
- Anti-embolism stockings should **not** be used.
- **Pharmacological thromboprophylaxis** should be considered for initiation at 24–48 hours after an acute stroke, provided:
  - haemorrhagic stroke has been excluded
  - the risk of haemorrhagic transformation (or bleeding elsewhere) is not unusually high

**Patients with major trauma**

**Mechanical thromboprophylaxis** should be applied in all cases, unless contraindicated. **Pharmacological thromboprophylaxis** should be considered on a case-by-case basis. Insertion of an **inferior vena cava filter** may be appropriate in selected cases (see below).

**Patients taking other antithrombotic drugs**

Do **not** prescribe additional pharmacological thromboprophylaxis for patients who are receiving full anticoagulant doses of warfarin or other coumarins (with INR >2), fondaparinux, heparin (low molecular weight or unfractionated), rivaroxaban, dabigatran, apixaban, edoxaban, argatroban or danaparoid.

Pharmacological thromboprophylaxis **should** be considered, if indicated, in patients who are taking antiplatelet drugs.

**Other patient groups**

Risk assessments should be performed according to the trust protocol, individualised as appropriate to the circumstances of the case.

**Timing of pharmacological thromboprophylaxis in relation to elective and semi-elective invasive procedures**

Wherever possible, pharmacological thromboprophylaxis should be interrupted or adjusted around the time of invasive procedures, spinal anaesthesia, and insertion or removal of epidural catheters.
<table>
<thead>
<tr>
<th>Anticoagulant (at prophylactic dose)</th>
<th>Invasive procedures</th>
<th>Epidural catheter insertion/removal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><em>Hours post-dose before commencing invasive procedure</em></td>
<td><em>Hours post-op before giving next dose</em></td>
</tr>
<tr>
<td>Dalteparin</td>
<td>12 hr</td>
<td>≥6 hr after haemostasis</td>
</tr>
<tr>
<td>Heparin</td>
<td>12 hr</td>
<td>≥6 hr after haemostasis</td>
</tr>
</tbody>
</table>

Emergency procedures should **not** be delayed because of recent thromboprophylaxis administration if the risks of delay are likely to exceed the small excess risk of bleeding. Reversal (with protamine) may be considered if the patient has received unfractionated heparin, but is unlikely to be worthwhile for low molecular weight heparin (for which there is no reliable reversal agent) at thromboprophylaxis doses.

**Inferior vena cava filters**

Radiological placement of an inferior vena cava (IVC) filter is an alternative or additional option in selected cases, particularly patients who are at very high risk of VTE, but cannot receive pharmacological thromboprophylaxis. This may include patients who are expected to be immobilised for a prolonged period, with a significant ongoing bleeding risk or need for frequent surgical procedures (most commonly in the setting of major trauma).

**References**

Venous thromboembolism: reducing the risk for patients in hospital. NICE Clinical guideline CG92; last updated June 2015.

| **TABLE 1: Risk factors for venous thromboembolism.** |
|----------------------------------|-----------------------------------------------|
| **AGE**                          | Exponential increase in risk with age. In general population |
|                                  | <40 annual risk 1/10,000                       |
|                                  | 60-69 annual risk 1/1,000                     |
|                                  | >80 annual risk 1/100                         |
|                                  | May reflect immobility and coagulation activation |
| **OBESITY**                      | 3 x risk if obese (BMI > 30 kg/m2)            |
|                                  | May reflect immobility and coagulation activation |
| **VARICOSE VEINS**               | 1.5 x risk after major surgery / orthopaedic surgery |
|                                  | But low risk after varicose vein surgery |
| **PREVIOUS VTE**                 | Recurrence rate 5% / year, increased by surgery |
| **THROMBOPHILIAS**               | Low coagulation inhibitors (antithrombin, protein C or S) |
|                                  | Activated protein C resistance (e.g. factor V Leiden) |
|                                  | High coagulation factors (I, II, VII, IX, XI) |
|                                  | Antiphospholipid syndrome |
|                                  | High homocysteine |
| **OTHER THROMBOTIC STATES**      | Malignancy 7 x risk |
|                                  | Heart failure |
|                                  | Recent myocardial infarction / stroke |
|                                  | Severe infection |
|                                  | Inflammatory bowel disease, nephritic syndrome |
|                                  | Polycythaemia, paraproteininaemia |
|                                  | Bechet’s disease, paroxysmal nocturnal haemoglobinuria |
| **HORMONE THERAPY**              | Oral combined contraceptives, HRT, raloxifene, tamoxifen 3 x risk |
|                                  | High dose progestogens 6 x risk |
| **PREGNANCY, Puerperium**        | 10 x risk |
| **IMMObILITY**                   | Bed rest > 3 days, plaster cast, paralysis 10 x risk |
|                                  | Increases with duration |
| **PROLONGED TRAVEL**             | |
| **HOSPITALIZATION**              | Acute trauma, acute illness, surgery 10 x risk |
| **ANAESTHESIA**                  | 2 X general vs spinal/ epidural |
| **FEMORAL VENOUS CATHERISATION** | 6-10 x risk c/w subclavian |
Table 2: **Use of Graduated Compression Stockings**
[Use above knee stockings if possible unless contraindicated (e.g. thigh circumference > 81cm, incontinence)]

<table>
<thead>
<tr>
<th>CONTRAINDICATIONS</th>
<th>CAUTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Massive leg oedema</td>
<td></td>
</tr>
<tr>
<td>Pulmonary oedema (Heart failure)</td>
<td></td>
</tr>
<tr>
<td>Severe peripheral arterial disease</td>
<td></td>
</tr>
<tr>
<td>Major leg deformity</td>
<td></td>
</tr>
<tr>
<td>Dermatitis</td>
<td></td>
</tr>
<tr>
<td>Select correct size</td>
<td></td>
</tr>
<tr>
<td>Apply carefully, align toe hole under toe</td>
<td></td>
</tr>
<tr>
<td>Check fitting daily for changes in leg circumference</td>
<td></td>
</tr>
<tr>
<td>Do not fold down</td>
<td></td>
</tr>
<tr>
<td>Remove daily for no more than 30 minutes</td>
<td></td>
</tr>
</tbody>
</table>

Table 3: **Contra-indications and caution for heparins** (LDUH & LMWH) in prophylaxis of VTE

<table>
<thead>
<tr>
<th>CONTRAINDICATIONS</th>
<th>CAUTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncorrected bleeding disorder</td>
<td></td>
</tr>
<tr>
<td>Haemophilia</td>
<td></td>
</tr>
<tr>
<td>Oral anticoagulants</td>
<td></td>
</tr>
<tr>
<td>Platelet count &lt; 70 x 10⁹ / L</td>
<td></td>
</tr>
<tr>
<td>Recent Intracranial Neurosurgery (&lt; 3 days)</td>
<td></td>
</tr>
<tr>
<td>Severe head injury (&lt; 5 days)</td>
<td></td>
</tr>
<tr>
<td>Unstable ICP</td>
<td></td>
</tr>
<tr>
<td>Intra-cerebral bleed (ICH or SAH) &lt; 4 days</td>
<td></td>
</tr>
<tr>
<td>Ruptured Cerebral aneurysm (not coiled or clipped)</td>
<td></td>
</tr>
<tr>
<td>Bleeding or potentially bleeding lesion</td>
<td></td>
</tr>
<tr>
<td>Oesophageal varices</td>
<td></td>
</tr>
<tr>
<td>Active peptic ulcer</td>
<td></td>
</tr>
<tr>
<td>Recent (3 months) GI bleed</td>
<td></td>
</tr>
<tr>
<td>Allergy</td>
<td></td>
</tr>
<tr>
<td>Heparin associated thrombocytopenia or thrombosis (heparin)</td>
<td></td>
</tr>
<tr>
<td>Severe liver impairment, alcoholism</td>
<td></td>
</tr>
<tr>
<td>Severe kidney impairment</td>
<td></td>
</tr>
<tr>
<td>Major trauma or surgery to brain, eye or spinal cord</td>
<td></td>
</tr>
<tr>
<td>Spinal or epidural block, LP (&lt; 12 hours)</td>
<td></td>
</tr>
<tr>
<td>Recent Neurosurgery</td>
<td></td>
</tr>
<tr>
<td>Intra-cerebral bleed (ICH or SAH)</td>
<td></td>
</tr>
<tr>
<td>Intra-cerebral tumour</td>
<td></td>
</tr>
<tr>
<td>Major head injury (&gt; 5 days) with intracerebral contusions</td>
<td></td>
</tr>
<tr>
<td>Unstable ICP</td>
<td></td>
</tr>
<tr>
<td>Subdural or extradural haematoma</td>
<td></td>
</tr>
</tbody>
</table>

Table 4: **Contra-indications to Intermittent Pneumatic Calf Compressors**

<table>
<thead>
<tr>
<th>CONTRAINDICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Only able to use one leg</td>
</tr>
<tr>
<td>Trauma (fractured leg), tissue loss</td>
</tr>
<tr>
<td>Skin condition or infection</td>
</tr>
<tr>
<td>Severe peripheral vascular disease</td>
</tr>
<tr>
<td>Skin condition (e.g. dermatitis)</td>
</tr>
<tr>
<td>Pulmonary oedema / Heart Failure</td>
</tr>
</tbody>
</table>
Sepsis (severe sepsis) and septic shock

Terminology and diagnosis

The terminology, and its associated definitions, relating to sepsis has recently changed. **Sepsis** (which now encompasses the syndrome known previously as **severe sepsis**) is defined as *life-threatening organ dysfunction caused by a dysregulated host response to infection*. In this context, **organ dysfunction** is defined as an increase in the Sequential (Sepsis-related) Organ Failure Assessment (SOFA) score of ≥2 points.

This scoring system not particularly suitable for use at the bedside, where instead a **quickSOFA (qSOFA)** score of ≥2 (see box) may be used to identify patients at significant risk of mortality due to infection.

<table>
<thead>
<tr>
<th>QuickSOFA (qSOFA) score</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate ≥22 breaths/min</td>
<td>1</td>
</tr>
<tr>
<td>Altered mentation</td>
<td>1</td>
</tr>
<tr>
<td>Systolic blood pressure ≤100 mmHg</td>
<td>1</td>
</tr>
</tbody>
</table>

*A score of ≥2 points in patients with suspected infection identifies patients at high risk of mortality*

**Septic shock** is a subset of sepsis, defined by a vasopressor requirement to maintain a mean arterial pressure of ≥65 mmHg, with serum lactate concentration >2 mmol/L, in the absence of hypovolemia (i.e. after adequate fluid resuscitation).

Sepsis is associated with an in-hospital mortality rate >10%, rising to >40% in patients with septic shock.

Management

The management of sepsis and septic shock in neurointensive care is, in general, no different to other critical care settings (although see the relevant sections of this handbook for advice on the management of **central nervous system infection**). The **guidelines of the Surviving Sepsis Campaign** (see box) offer a reasonable practical framework for management of sepsis, although they must be individualised to the circumstances of the case. Noradrenaline is the preferred first-line vasopressor in most cases of sepsis managed in the neurointensive care unit. Hydrocortisone (see below) and vasopression may be added in selected cases.
### Surviving sepsis campaign bundles

**To be completed within 3 hours:**
1. Measure lactate level
2. Obtain blood cultures prior to administration of antibiotics
3. Administer broad spectrum antibiotics
4. Administer 30 mL/kg crystalloid for hypotension or lactate 4 mmol/L

**To be completed within 6 hours:**
5. Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥65 mmHg
6. In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate 4 mmol/L (36 mg/dL):
   - Measure central venous pressure (CVP)*
   - Measure central venous oxygen saturation ($S_{cv}O_2$)*
7. Remeasure lactate if initial lactate was elevated*

*Targets for quantitative resuscitation included in the guidelines are CVP of ≥8 mm Hg, $S_{cv}O_2$ of 70%, and normalization of lactate.

### References


Hydrocortisone infusion

Hydrocortisone has direct vasoconstrictor effects, and has theoretical rationale as an adjunctive treatment in patients with refractory shock, who may have relative adrenal insufficiency. There is some evidence that it may (at ‘low’ doses, <300 mg/day) improve outcomes in severe septic shock, although this is controversial. There is no evidence that measurement of the serum cortisol concentration (random or after stimulation by synthetic ACTH) has any value in identifying a population more likely to be benefit.

Pragmatically, we consider a trial of hydrocortisone in patients with a sustained, high vasopressor requirement (e.g. noradrenaline >0.2 micrograms/kg/min), despite adequate volume resuscitation.

<table>
<thead>
<tr>
<th>Use of IV hydrocortisone in the neurointensive care unit</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient identification</strong></td>
</tr>
<tr>
<td><strong>Initial bolus dose</strong></td>
</tr>
<tr>
<td><strong>‘Steroid responder’</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Withdrawal and weaning</strong></td>
</tr>
</tbody>
</table>

References

**Red blood cell transfusion**

Transfusion of red blood cells is a central component of the management of patients with haemorrhagic shock and severe anaemia associated with features of inadequate tissue oxygen delivery (e.g. lactic acidosis, altered consciousness, myocardial dysfunction).

In patients who are not bleeding, and do not have clear evidence of inadequate tissue oxygen delivery due to anaemia, the role of red cell transfusion is less well defined. While it seems biologically plausible that maximising tissue oxygen delivery by maintaining near-normal haemoglobin concentrations should be beneficial, this is not supported by clinical trials. Trials comparing ‘liberal’ and ‘restrictive’ transfusion triggers (<100 and <70 g/L, respectively) in a range of settings (critical illness, cardiac surgery, hip fracture surgery, acute gastrointestinal bleeding) suggest that there is no advantage to a liberal transfusion strategy, and indeed that it may be harmful. In this context, it must be noted that red cell transfusion is associated with risks (see box), and that transfused red cells may behave differently to native red cells. These factors may contribute to what has been termed the 'blood transfusion anaemia paradox': namely, that anaemia is associated with adverse physiological effects and worse outcomes, but that correcting anaemia and its associated physiological effects by transfusion does not improve outcomes (and may even worsen them).

<table>
<thead>
<tr>
<th>Risks of red blood cell transfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haemolytic reactions</strong></td>
</tr>
<tr>
<td>Acute haemolytic reactions</td>
</tr>
<tr>
<td>Delayed haemolytic transfusion reactions</td>
</tr>
<tr>
<td><strong>Immunologically mediated non-haemolytic reactions</strong></td>
</tr>
<tr>
<td>Febrile reactions</td>
</tr>
<tr>
<td>Urticarial and anaphylactic reactions</td>
</tr>
<tr>
<td>Post-transfusion purpura</td>
</tr>
<tr>
<td>Transfusion-related immunomodulation (TRIM)</td>
</tr>
<tr>
<td><strong>Volume effects</strong></td>
</tr>
<tr>
<td>Transfusion-associated circulatory overload (TACO)</td>
</tr>
<tr>
<td><strong>Transmission of infection</strong></td>
</tr>
<tr>
<td>Transfusion-transmitted bacterial infection</td>
</tr>
<tr>
<td>Transfusion-transmitted viruses, parasites and</td>
</tr>
</tbody>
</table>

59
<table>
<thead>
<tr>
<th>prions</th>
</tr>
</thead>
</table>

**Metabolic effects**

| Hypocalcaemia | Due to citrate in the preservative solution |
| Hypoglycaemia | Due to cellular leakage, most often after massive transfusion or transfusion of irradiated products |
| Hyperkalaemia | Due to citrate in the preservative solution |
| Iron overload | After multiple transfusions for chronic anaemia |

There has been concern that studies favouring a restrictive transfusion strategy in general and cardiac intensive care patients may not be applicable to the neurointensive care population. Anaemia is associated with adverse outcomes in traumatic brain injury, subarachnoid haemorrhage and ischaemic stroke. Brain tissue has high oxygen demand, and operates at a high baseline oxygen extraction ratio, such that the compensatory mechanisms for anaemia are more limited than in other tissues. On the other hand, increasing haemoglobin concentration increases blood viscosity, reducing cerebral blood flow. The improvement in brain tissue oxygen tension following transfusion is small, and in some patients it actually falls. There is little robust clinical evidence to guide practice, and recommendations are generally based on expert consensus.

In the absence of good evidence pertaining specifically to neurointensive care, it is probably best to be guided by the wider data that favours a restrictive transfusion strategy. **Maintaining a haemoglobin concentration of 70–90 g/L is therefore reasonable in most cases in neurointensive care**, although expert recommendations suggest that the higher end of this range may be preferred in patients with cerebral ischaemia. It is crucial to recognise that the measured haemoglobin concentration forms only one part of a global assessment of the appropriateness of transfusion (see box) — it should never be the only reason for transfusion.

**A global assessment of the appropriateness of red cell transfusion**

<table>
<thead>
<tr>
<th>Clinical context</th>
<th>Patient/illness characteristics</th>
<th>Markers of tissue oxygen delivery</th>
<th>Haemoglobin concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Favours transfusion</strong></td>
<td>Haemorrhagic shock&lt;br&gt;Acute coronary syndrome&lt;br&gt;Cerebral ischaemia&lt;br&gt;Symptomatic anaemia</td>
<td>Older patients&lt;br&gt;More severe illness</td>
<td>High lactate&lt;br&gt;Low $S_{\text{cv}}O_2$</td>
</tr>
<tr>
<td>Against transfusion</td>
<td>No evidence of acute organ ischaemia</td>
<td>Younger patients&lt;br&gt;Less severe illness</td>
<td>Normal lactate&lt;br&gt;Normal $S_{\text{cv}}O_2$</td>
</tr>
</tbody>
</table>

**References**

Patients with abnormal clotting

Therapeutic anticoagulation

The complexity of therapeutic anticoagulation has increased recently due to the approval of several new antithrombotic agents (particularly the direct oral anticoagulants, DOACs). Agents currently licensed for anticoagulation in the UK are listed in the table. Therapeutic anticoagulation accounts for a significant minority of intracranial (including intracerebral) haemorrhage, and mortality from anticoagulation-related intracerebral haemorrhage (ICH) is high. The direct oral anticoagulants may be associated with a lower risk of ICH, although such cases may be complicated due to the difficulty measuring and reversing the anticoagulant effect. Potential reversal strategies are outlined in the table, but specialist advice should always be sought.

<table>
<thead>
<tr>
<th>Currently available anticoagulant drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug</strong></td>
</tr>
<tr>
<td><strong>Oral vitamin K antagonists</strong></td>
</tr>
<tr>
<td>Warfarin, acenocoumarol and phenindione</td>
</tr>
<tr>
<td><strong>Direct oral anticoagulants</strong></td>
</tr>
<tr>
<td>Dabigatran</td>
</tr>
<tr>
<td>Rivaroxaban</td>
</tr>
<tr>
<td>Apixaban</td>
</tr>
<tr>
<td>Edoxaban</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td><strong>Heparins and derivatives</strong></td>
</tr>
<tr>
<td>Unfractionated heparin</td>
</tr>
<tr>
<td>Low molecular weight heparins (dalteparin, enoxaparin, tinzaparin)</td>
</tr>
<tr>
<td>Fondaparinux</td>
</tr>
<tr>
<td><strong>Intravenous anticoagulants</strong></td>
</tr>
<tr>
<td>Danaparoid</td>
</tr>
<tr>
<td>Argatroban</td>
</tr>
<tr>
<td>Bivalirudin</td>
</tr>
</tbody>
</table>

*Always seek specialist haematology advice, especially for bleeding associated the newer agents (direct oral anticoagulants and fondaparinux). Minor bleeding can usually be managed conservatively. Non-emergency surgery can often be deferred to allow the effects of anticoagulants to dissipate. Where reversal of warfarin is required for non-urgent surgery (e.g. to avoid cancellation), a small dose of phytomenadione (e.g. 1–2 mg IV, or orally using the IV preparation, 24 hours before surgery) may be considered.

ACS, acute coronary syndrome; AF (prevention of embolic complications thereof); VTE, venous thromboembolism (prevention and/or treatment thereof)
PCC, prothrombin complex concentrate; FFP, fresh frozen plasma.
Assessment and management of other causes of coagulopathy

Non-therapeutic causes of coagulopathy are diverse, including liver disease, malnutrition, massive haemorrhage/transfusion, disseminated intravascular coagulation and sepsis.

In any patient with an INR >1.2 and intracranial haemorrhage give phytomenadione (vitamin K₁) 10 mg IV/orally daily for 3 days. Prefer IV administration for the first dose.

In coagulopathy not associated with bleeding, discuss with senior colleagues before giving phytomenadione as, provided it does not present a significant bleeding risk, it may represent a useful marker with which to track illness trajectory. In major or life threatening bleeding, always seek specialist haematology advice.

Thromboelastography (TEG) may be very useful in the assessment of coagulopathy and to guide its treatment. TEG analysers can be found in the cardiac intensive care unit, general intensive care unit and theatres. If you are unfamiliar with TEG, seek training from senior colleagues in how to perform and interpret the assay. If you need staff from Cardiac or General ICU to run the assay for you, always telephone in advance of collecting the sample, to ensure someone will be immediately available to assist you. A useful guide to the use and interpretation of TEG can be found in the General ICU introductory handbook (the 'CHAOS book').

References


Sodium and fluid balance

General considerations

Disorders of sodium and fluid balance are common on the neurointensive care unit. Moreover, because of the effects of sodium on brain volume (and therefore intracranial pressure) and brain cellular function, they — and their treatment — arguably present a greater risk of harm than in other settings.

It is imperative to avoid causing or exacerbating a fall in serum sodium concentration in patients with, or at significant risk of, raised intracranial pressure. For this reason, intravenous infusion solutions that are effectively hypotonic (e.g. glucose 5% and compound preparations with a sodium concentration less than that of serum) must not be administered except under the direction of a neurointensive care consultant. Hartmann’s solution (sodium lactate intravenous infusion, compound) has a sodium concentration of 131 mmol/L, which may also be too low in some patients (particularly those with raised intracranial pressure and hypernatraemia).

Cardiac dysfunction is also common in patients with several neurological illness. Use of a calibrated cardiac output monitor (we use the LiDCO® device) should be a routine part of management wherever there is haemodynamic instability or any uncertainty about volume status and its management.

<table>
<thead>
<tr>
<th>Normal reference ranges for sodium and osmolality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum sodium concentration</td>
</tr>
<tr>
<td>Serum osmolality</td>
</tr>
<tr>
<td>Urinary sodium concentration (spot sample)</td>
</tr>
<tr>
<td>Urinary osmolality</td>
</tr>
</tbody>
</table>

Rate of correction of serum sodium abnormalities

The safe and effective rate of correction of abnormalities of serum sodium concentration (hypo- and hypernatraemia) depends on the rate at which the abnormality developed.

- **Acute (<24 hrs):** serum sodium abnormalities that have developed within the last 24 hours can (and, if symptomatic, should) be treated rapidly. In this context, it is generally appropriate to shift the sodium concentration towards normality by about 5 mmol/L within a few hours. This should be sufficient to relieve any acute symptoms, and further correction within the first 24 hours should be kept to a minimum.
- **Subacute (24–48 hrs) and chronic (>48 hrs):** cerebral adaptive changes to the osmotic effects of hypo- and hypernatremia develop over about 24–48
hours. Rapidly normalising the sodium concentration after this period can provoke fluid shifts that may be harmful, causing seizures, osmotic demyelination, cerebral oedema and brain herniation, depending on the context. The rate of correction should be no more than 0.5 mmol/L per hour, and limited to a maximum of 8 mmol/L over 24 hrs.

**Hypernataemia**

**Definition and causes**

Hypernataemia is defined as a serum sodium concentration >146 mmol/L. In broad terms, it may be caused by a deficit of water relative to sodium, or an excess of sodium relative to water. The most common causes in the neurointensive care unit are **administration of hypertonic sodium chloride** and **cranial diabetes insipidus**, which are discussed more fully below. Other causes are outlined in the table.

<table>
<thead>
<tr>
<th>Causes of hypernatraemia</th>
<th>Mechanism</th>
<th>Cause(s)</th>
<th>Diagnosis</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium excess</td>
<td>Administration of hypertonic sodium chloride or bicarbonate</td>
<td>Clinical context (hypertonic fluid administration)</td>
<td>Moderate hypernatraemia (Na⁺ 146–155 mmol/L) is generally tolerated in patients with intracranial hypertension. See notes below for further discussion.</td>
<td></td>
</tr>
<tr>
<td>Insufficient water intake (‘simple dehydration’)</td>
<td>Impaired thirst sensation</td>
<td>Thirst; clinical signs of dehydration; oliguria; negative fluid balance; disproportionately elevated serum urea concentration, relative to creatinine</td>
<td>Rehydrate, preferably enterally (i.e. encourage oral intake if drinking, otherwise add/increase NG water); IV rehydration if necessary (use isotonic fluid, e.g. 0.9% sodium chloride).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inability to access sufficient water</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Iatrogenic water under-replacement</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of hypotonic fluid</td>
<td>Cranial diabetes insipidus</td>
<td>Polyuria (&gt;200 mL/hr for ≥2 hrs), rapidly rising serum sodium concentration and inappropriately dilute urine (urine osmolality &lt;300 mmol/kg, for which specific gravity &lt;1.01 is a simple bedside surrogate)</td>
<td>Alert patients with intact thirst sensation should be given free access to water, and encouraged to drink to thirst. Selected cases require desmopressin (DDAVP) — see notes below.</td>
<td></td>
</tr>
<tr>
<td>Osmotic diuresis due to mannitol</td>
<td>Clinical context (mannitol administration); polyuria; urine osmolality ≥300 mmol/kg and specific gravity ≥1.01</td>
<td>Replace volume loss with 0.9% sodium chloride</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nephrogenic diabetes insipidus</td>
<td>Although common in its mild form (in elderly patients or as part of a wider renal impairment syndrome), it is rarely the primary cause of a significant sodium or water imbalance</td>
<td>Depends on the cause and clinical context; seek advice</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Hypernatraemia due to hypertonic fluid administration**

Invariably, hypertonic sodium chloride infusion causing hypernatraemia occurs in the setting of life-threatening raised intracranial pressure (ICP). In this case, moderate hypernatraemia (Na⁺ 146–155 mmol/L) is tolerated, since correction (especially if too rapid) may cause a rebound elevation of ICP. Hypernatraemia may also occur following mannitol administration due to osmotic diuresis (provided there is intact renal function; if not, hyponatraemia — due to expansion of extracellular volume — may result). The loss of circulating volume due to osmotic diuresis may be replaced with 0.9% sodium chloride.

**Cranial diabetes insipidus**

*Causes.* Cranial diabetes insipidus (CDI) affects around 3.7% of neurointensive care patients. It occurs in patients with tumours encroaching on the hypothalamus or posterior pituitary gland (particularly craniopharyngioma; also suprasellar meningioma and pituitary adenoma) and after their resection, and in other conditions affecting the hypothalamus and/or posterior pituitary (including traumatic brain injury, subarachnoid haemorrhage, hypoxic–ischaemic encephalopathy and intracerebral haemorrhage). It also occurs in any condition associated with raised intracranial pressure, and is frequently seen after brainstem death.

*Clinical manifestations.* In patients with intact thirst sensation and free access to water, CDI does not usually cause significant hypernatraemia because the patient increases their water intake to compensate for their increased losses. In critically ill patients, however, both thirst sensation and water intake may be impaired or obliterated, and severe hypernatraemia may develop rapidly. The resulting fluid shifts can lead to seizures, impaired consciousness and osmotic demyelination.

*Diagnosis.* In patients who are not alert and/or cannot drink water freely, the diagnosis of diabetes insipidus requires:

- Polyuria: urine output >200 mL/hr for ≥2 hrs, usually leading to marked negative fluid balance
- Rapidly rising serum sodium concentration
• Inappropriately dilute urine: ideally measured on urine osmolality (which will be <300 mmol/kg)

Urine specific gravity is a useful bedside surrogate for laboratory measurement of urine osmolality, and a value <1.01 is supportive of the diagnosis. Values ≥1.01 do not exclude CDI and should prompt laboratory measurement of osmolality.

Treatment. Patients with intact thirst sensation, and who can drink freely, should be encouraged to drink to thirst. Acute treatment is unnecessary provided they can maintain neural fluid balance and the serum sodium concentration is normal or only mildly elevated.

Patients who cannot reliably drink to thirst may require enteral or intravenous fluid replacement. Intravenous fluid replacement is most safely administered using a near-isotonic fluid preparation (e.g. 0.9% sodium chloride). Treatment with desmopressin (DDAVP; an analogue of arginine vasopressin/antidiuretic hormone which does not have vasopressor activity) should be reserved for patients with marked polyuria, negative fluid balance and rapidly rising sodium concentration that cannot be controlled by other means. It must be recognised that inappropriate administration of desmopressin in the context of neurocritical illness carries substantial risks in promoting cerebral oedema. Discussion with the NICU consultant and/or neurosurgical team is essential if there is any doubt about its appropriateness. In the acute phase, desmopressin is preferably given intravenously, usually at a dose of 0.5 micrograms (repeated maximum 1-hrly). Desmopressin must not be administered concurrently with hypotonic fluids, due to the risk of cerebral oedema.

**Hyponatraemia**

**Definition and causes**

Hyponatraemia is defined as a serum sodium concentration <133 mmol/L, after excluding causes of 'pseudohyponatraemia' (an increase in the non-water fraction of serum volume, e.g. due to hypertriglyceridaemia or hyperproteinaemia). It is common, and has a diverse range of causes. Broadly, the causes may be divided into those associated with low extracellular water volume (implying a loss of sodium in excess of water), normal extracellular water volume (implying a loss of sodium) or increased extracellular water volume (i.e. oedema; implying an excess of water relative to sodium).

In the neurointensive care population, the most important causes are:

• **Syndrome of inappropriate antidiuretic hormone secretion (SIADH)**, which can occur with any central nervous system disorder (including traumatic brain injury, intracranial haemorrhage, stroke and CNS infection), tumours (most commonly small cell lung cancer), drugs (many, including carbamazepine, valproate, antidepressants, antipsychotics) and other disorders.

• **Cerebral salt wasting (CSW)**, which is most commonly seen in subarachnoid haemorrhage, but can occur in other central nervous system disorders
Other causes of hyponatraemia are listed in the diagnostic flow chart.

**Clinical features**

The symptoms and signs of hyponatraemia are mainly neurological. Outside of the neurointensive care population, mild hyponatraemia (125–133 mmol/L) is usually asymptomatic. Headache and lethargy emerge as the sodium concentration drops below 125 mmol/L, and progress to obtundation, seizures and coma at concentrations below 120 mmol/L. Milder degrees of hyponatraemia can cause symptoms if the rate of fall was rapid.

Patients with neurocritical illness may be more susceptible to adverse effects of mild hyponatraemia. Any degree of hyponatraemia is a concern (and requires correction) in a patient at risk of raised intracranial pressure. As such, a more proactive approach to the diagnosis and management of any degree of hyponatraemia is often required in NICU than it would be elsewhere.
### Diagnostic approach in NICU

Identifying the cause of hyponatraemia depends on an assessment of extracellular volume status together with the urine sodium concentration (see flow diagram). This, together with the clinical context (e.g. overt gastrointestinal losses, renal impairment, diuretic therapy, heart failure), will often allow a reasonably confident diagnosis to be made.
Usually the most difficult distinction in neurointensive care is between SIADH and cerebral salt wasting, both of which are common in the brain injured population. These are difficult to separate because both involve to ADH secretion and the consequences thereof; the difference is in why ADH is secreted.

In CSW, the primary disturbance is naturesis (the cause of which is not fully understood, but may involve excess secretion of nauretic peptides). This causes volume contraction, stimulating appropriate ADH secretion and, therefore, water retention and hyponatraemia. In SIADH, by contrast, the primary disturbance is with ADH secretion, which occurs inappropriately. Both processes lead to hyponatraemia associated with a relatively high urine sodium concentration, and a urine osmolality greater than serum osmolality (i.e. urine that is not maximally dilute). Clinically, the distinction is made on the basis of volume status and the response to 0.9% sodium chloride infusion:

- In **CSW**, the extracellular fluid volume is contracted. As euvolaemia is restored by infusion of 0.9% sodium chloride, ADH secretion is suppressed and the serum sodium concentration should improve.
- In **SIADH**, the extracellular fluid volume is normal (although in practice, a mixed picture, e.g. with oedema from another cause, is common). Infusion of 0.9% sodium chloride will not suppress ADH secretion (because it is being secreted independently of osmotic sensing). The serum sodium concentration generally does not improve, and may indeed worsen. This is because renal sodium handling is largely unaffected (allowing the whole sodium load to be excreted), whereas renal water excretion is impaired (because inappropriate ADH secretion prevents excretion of maximally-dilute urine). Thus the sodium load may be completely eliminated, whereas the water load is only partially eliminated, exacerbating the hyponatraemia.

### A pragmatic approach to hyponatraemia management in NICU

Where the cause of hyponatremia is readily evident from the clinical context, volume status and urine sodium concentration, appropriately directed treatment may be given. In particular, where extracellular volume status is clearly contracted (implying a deficit of both water and sodium, but more so of sodium), this should be treated with appropriate enteral and/or intravenous salt and water replacement (e.g. 0.9% sodium chloride). If the clinical picture is suggestive of cerebral salt wasting or pituitary pathology, consider also fludrocortisone 100 micrograms 6-hrly orally or NG (monitor for hypokalaemia).

In the common situation in which no overt cause for hyponatremia is evident, the urine output and volume status are not overtly abnormal, and the urine biochemistry is supportive of either SIADH or CSW (urine Na⁺ >30 mmol/L, urine osmolality > serum osmolality), a reasonable approach is to:

1. Supplement enteral sodium intake with sodium chloride (e.g. as Slow Sodium®, (600-mg tabs), 2–4 tabs 6-hrly)
2. Consider infusion of 0.9% sodium chloride (e.g. 100 mL/hr) initially, as a diagnostic and (potentially) therapeutic intervention
3. If hyponatraemia improves with 0.9% sodium chloride infusion, the diagnosis is more likely to be salt wasting – which may be CSW if the clinical context supports this
   a. Continue isotonic fluid infusion until volume status is normalised and can be maintained enterally
   b. Add fludrocortisone 100 micrograms 6-hrly orally or NG (monitor for hypokalaemia)

4. If hyponatraemia does not improve (or worsens) with 0.9% sodium chloride infusion (and there is not overt volume depletion or extra-renal losses), the pathology is more likely SIADH
   a. If mild (125–133 mmol/L) and not associated with a significant brain injury, treat initially with fluid restriction (to 1000 mL/day)
   b. If mild in the context of a significant brain injury (e.g. TBI, SAH), or severe (<120 mmol/L) or symptomatic (seizures, altered consciousness, etc), infusion of hypertonic sodium chloride is likely to be necessary (e.g. sodium chloride 1.8% at 50 mL/hr initially). See ‘Rate of correction of serum sodium abnormalities’, above, for a discussion on the optimum rate of correction.

References


Algorithm for difficult intubation on NITU

In NITU there is a dedicated airway trolley

Unanticipated difficult tracheal intubation in an adult patient

Direct laryngoscopy

Any problems

Call for help (Bleep 7647/6111)

Aspirate feeding tube if present
Preoxygenate
Cricoid pressure
Direct laryngoscopy
If poor view:
Reduce cricoid pressure
Introducer (Bougie) ± Alternative laryngoscope / Glidescope

Failed intubation

ILMA or LMA
NOT MORE than 3 insertion attempts
Oxygenate and ventilate

Failed oxygenation via LMA/ILMA

Revert to face mask
Oxygenate and ventilate
1 or 2 person technique (oral or nasal airway)

Failed ventilation and oxygenation

Scalpel cricothyroidotomy

Tracheal intubation
Verify with capnograph

NOT MORE than 4 attempts maintaining oxygenation and anaesthesia

Confirm: Ventilation, Oxygenation, Anaesthesia, Muscle relaxation then fiberoptic intubation through ILMA or LMA

Confirm ventilation & oxygenation
Discuss with consultant on call
alternatives (Airtraq, Glidescope)
References

DAS Guidelines 2015 - amended
Tracheostomies

A full guidance on tracheostomies can be found at Guidelines and standards | Intensive Care Society AND http://www.tracheostomy.org.uk

Some of the recommendations are as follows:
A planned tracheostomy should be performed by medical practitioners who have been trained, and are competent in the procedure, or are under direct senior bedside Supervision.
The minimum staffing required is 2 trained medical practitioners plus a third member of staff to assist.
All Critical Care Areas should have their own Difficult Airway trolley.
Endoscopy should be readily available and its routine use is likely to improve the safety of insertion procedures and management of airway problems thereafter.
A recent NCEPOD study has confirmed a relatively low frequency of serious periprocedural complications in ICU patients receiving a tracheostomy.

Tracheostomy wherever performed is a surgical procedure. As highlighted in the recent NCEPOD report; practices would be improved by national data collection of tracheostomy and other airway procedures, introduction of WHO style preoperative checklists and more consistent consent procedures.

Tracheostomy tube choice
Tracheostomy tubes should be chosen to suit the individual patient. The requirements of a patient may change over time.
The need for excessive cuff volume or pressure suggests that the tube size may be too small or that it may be misplaced.
Tracheostomy tubes with an inner cannula are inherently safer and are normally preferred. In situations where ventilation is difficult (high FiO2 or high airway pressures) the risks of repeated circuit disconnection to facilitate inner cannula care must be balanced on a daily basis.
Most tracheostomy tubes are sized by internal diameter in millimetres, but this may not take account of the inner cannula in all cases. Tracheostomy tubes with the same internal diameter (‘manufacturer’s reference size’) may have quite different external diameters and length.
Fenestrated tracheostomy tubes should be used with caution in mechanically ventilated patients and only in patients who are weaning from ventilation.
It is essential that the staff caring for a patient with a tracheostomy know the type of tube that is in place. This should be clearly documented.
Obese patients, or those with abnormal anatomy, or a low tracheostomy stoma may require a longer adjustable flange tube or a tube with a fixed extended distal or proximal portion.

Routine Care of tracheostomies:
In order to avoid complications, the main priorities are humidification, ensuring the cleanliness and patency of the inner tube, secure fixation of the tube and attention to cuff pressure.
The same care should be taken when moving, handling and nursing a patient with a tracheostomy as with any other invasive airway device.
In non-ventilated patients the inner cannula should be regularly removed, cleaned or changed at a maximum interval of 4 hourly in a patient with a productive chest, and at least 8 hourly in all cases, being cognisant of the patient’s need for sleep and rest. In mechanically ventilated patients, an inner cannula is desirable as a safety measure but routine cleaning or changing needs consideration against the risks of derecruitment and infection. In this group replacement inner cannula, should be immediately available at the bedside and replaced if ventilator parameters suggest narrowing of the cannula with secretions. The entire tube should be changed at least every 30 days or as per manufacturer’s recommendations.

**Cuff pressure** should not exceed 25 cm H2O. If an air leak occurs with the cuff pressure at the maximum recommended, the tracheostomy may have become displaced or may require changing: medical or other professionals who are competent in tracheostomy management should review the patient. **Humidification** is essential for patients with temporary tracheostomies.

Any difficulty in passing the suction catheter should lead to consideration that the tube may be partially blocked, badly orientated or misplaced and requires immediate attention.

**Changing tracheostomy tubes**

Tracheostomy tubes should only be changed by staff, competent to do so. Patients should be monitored at levels appropriate to their condition, and the predicted ease or difficulty of replacement. Technologies to confirm correct placement should be immediately available (capnography and endoscopy). It is essential that if the new tube cannot be inserted or is misplaced, there is an agreed procedure for managing the situation. A ‘Plan B’ should be discussed and agreed, with appropriate equipment and drugs available as necessary.
Common Indications for tracheostomy

- **To maintain the airway:** e.g. reduced level of consciousness, upper-airway obstruction, intubation difficulties
- **To provide some protection to the airway:** e.g. bulbar palsy
- **For bronchial toilet:** e.g. excessive secretions/inadequate cough
- **For weaning from IPPV:** e.g. patient comfort, reduction of sedation

Cautions and contraindications

In the absence of airway obstruction, the only absolute contraindication to either an open or PDT is severe local sepsis or an uncontrollable coagulopathy. Surgical tracheostomies often require a transfer to theatre, which may present logistical challenges and potential delays. However, an open surgical procedure may be judged to be the best option in the following circumstances, depending on local expertise:

Cautions and relative contraindications to PDT

- **Difficult anatomy:** e.g. morbid obesity, lack of neck mobility, proven or potential cervical spine injury, known difficult intubation, tracheal pathology,
- **Thyroid pathology, and aberrant vessels.**
- **Significant coagulopathy**
- **Proximity to site of recent surgery or trauma:** e.g. carotid endarterectomy, anterior cervical fixation, sternotomy, oesophageal drainage, and burns.
- **Potential instability:** e.g. patients unable to tolerate cardiovascular or respiratory changes, such as those with unstable intra-cranial pressure (ICP)
- after brain injury
- **Severe gas exchange problems:** e.g. FiO2 >0.6 and PEEP >10 cm H2O
- **Age:** children under 12 years of age
Preparing for a tracheostomy

Comments on procedure & Tips (with use of Blue Rhino Set)
See also on YouTube: https://www.youtube.com/watch?v=2svtzRn0qHQ

1. Always check clotting and PLT count before deciding to proceed.
2. Before starting/sterilizing the area, check with the ultrasound probe (Sonosite) for any vessels overlying the tracheal area that could bleed during the procedure. Set the patient’s ventilator on FiO2 of 100% throughout rest of procedure.
3. Placement of the percutaneous tracheostomy should be between 1st-2nd or 2nd-3rd tracheal rings. Though not usually performed, one can mark anatomic landmarks, like thyroid cartilage, cricoid cartilage and sternal notch. The bronchoscope light illuminating through trachea can also be used as a guide for correctly identifying the site of trachea if there is any doubt, before proceeding.
4. The anaesthetist managing the airway should at this stage withdraw the ETT so that the cuff stays just below the vocal cords. This should protect from puncturing the cuff and hitting the ETT with needle or dilators. Depending on the neck height of the patient, the distance between puncturing site and cuff/distal ETT tip might be not enough. Bronchoscopy with tracheal illumination may give an approximation of this distance, though not a certain measurement. In exceptional circumstances an LMA can replace the ETT tube.
5. After sterilizing and injecting lignocaine-adrenaline locally, insert the appropriate (provided) needle with mounted catheter to puncture the anterior trachea wall perpendicularly. Before puncturing, some prefer to make a 2 cm horizontal skin incision and dilate with a curved mosquito to facilitate digital palpation of trachea and also insertion of the tracheostomy tube.
6. The attached syringe should have 3-4 ml of saline so that the aspirated air can be visualised easily when the needle is in correct position. Insert the bronchoscope to check needle placement, which should ideally emerge from 12 o’clock position (anterior, midline, intercartilaginous tracheal site). Beware not to damage the bronchoscope with the needle. The needle should also not be hitting the posterior tracheal wall. Under bronchoscopic visualization, push catheter forward, remove needle and thread the J-tip guidewire through the catheter.
7. Remove the catheter. Make a <0.8cm horizontal skin nick with a scalpel (if not previously done on stage 5). Introduce the (small) dilator over guidewire and after removing this, the (long) 8fr white guiding catheter. There is a safety ridge on the distal end of this catheter that should sit on the level of the skin. Moreover, the black mark of the guidewire should be aligned to the proximal tip of the guiding catheter. Insert the rhino dilator over the guidewire and guiding catheter and place it so that it is sitting on the ridge with its distal tip. Push dilator in the trachea up to the marked black line. Do not use lubricant to insert any of these components, but only sterile NS or water.
8. Remove dilator and introduce lubricated trache tube together with provided blue catheter over the guidewire and guiding catheter. Remove blue catheter,
guiding catheter and guidewire. Connect trache tube with ventilator tube. **Don’t let go placed trache tube before securing!**

9. Check tube placement and distance from carina with **bronchoscope**.

**Table 1: Preparation Check list**

<table>
<thead>
<tr>
<th>Consent / Consent form/ WHO check list</th>
<th>Airway trolley</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tracheostomy set (Rhino)</td>
<td>Laryngoscope</td>
</tr>
<tr>
<td>Tracheostomy tubes x2 (normal + one size smaller)</td>
<td>LMA</td>
</tr>
<tr>
<td>CVC insertion pack, sterile gloves, hat, mask</td>
<td>ETT</td>
</tr>
<tr>
<td>Chlorhexidine 2% with 70% alcohol</td>
<td>Glidescope</td>
</tr>
<tr>
<td>Suture</td>
<td>Pair of scissors</td>
</tr>
<tr>
<td>Scalpel</td>
<td>Syringe 10ml</td>
</tr>
<tr>
<td>Forceps for dissection</td>
<td>Suction + yanker</td>
</tr>
<tr>
<td>AquageL</td>
<td>Extra Suction + Suction Tubing</td>
</tr>
<tr>
<td>Sonosite (ultrasound)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Single Use Bronchoscope (from consultants’ office)</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchoscope screen</td>
<td>fentanyl infusion</td>
</tr>
<tr>
<td>Catheter mount with port</td>
<td>propofol bolus + infusion</td>
</tr>
<tr>
<td>ETCO2 monitoring</td>
<td>paralysis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extra Suction + Suction Tubing</th>
<th>metaraminol</th>
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<tbody>
<tr>
<td></td>
<td>adrenaline / atropine</td>
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</tbody>
</table>

**Table 2: Safety Checklist to be filled and filled in the notes**

ICU “WHO Safety Checklist” for invasive procedures (for bronchoscopy, tracheostomy and any other “surgical procedure”)

<table>
<thead>
<tr>
<th>PRE PROCEDURE</th>
<th>POST PROCEDURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site (if applicable)</td>
<td>All sharps accounted for Y □ / N □</td>
</tr>
<tr>
<td>Procedure</td>
<td>Standard procedure performed Y □ / N □</td>
</tr>
<tr>
<td>Clinician responsible for decision to perform this procedure</td>
<td>All specimens labeled and sent Y □ / N □</td>
</tr>
<tr>
<td>Assistance provided by: Bedside nurse □ AND/OR Other □ (state who)</td>
<td>Post procedure instructions confirmed Y □ / N □</td>
</tr>
<tr>
<td>Patient informed Y □ / N □</td>
<td>Cleaning and re-stocking of equipment performed by</td>
</tr>
<tr>
<td>Next of Kin informed Y □ / N □</td>
<td>Any untoward events Y □ / N □ (e.g. equipment failure, cardiac arrest etc etc. Give details below and complete Data)</td>
</tr>
<tr>
<td>Patient position</td>
<td></td>
</tr>
</tbody>
</table>
TRACHEOSTOMY EMERGENCIES, COMPLICATIONS AND THEIR MANAGEMENT

The main life threatening complications associated with tracheostomies are blockage, dislodgement and bleeding.

Blockage and displacement

![Emergency Tracheostomy Management - Patent Upper Airway](image_url)

- **Call for expert airway help – Anaesthetist bleep 6111 and ENT SpR – call switchboard to aircall SG818**
- **Is the patient breathing?**
  - CPR if no pulse / signs of life
- **Apply high flow oxygen to both the face and the tracheostomy**
- **Remove speaking valve or cap**
  - Remove inner tube / check patency
  - Some inner tubes need re-inserting to connect to breathing circuits
  - Look, listen and feel at the mouth and tracheostomy
  - Deflate the cuff (if present)
  - Can you pass a suction catheter?
    - NO
  - YES
- **Is the patient stable or improving?**
  - NO
  - YES
  - Consider Tracheostomy Change / FNE (call ENT)
  - Continue ABCDE assessment
- **The tracheostomy tube is patent**
  - Ventilate/Oxygenate (via tracheostomy)
  - Connect CO₂ Monitoring
  - Continue ABCDE assessment
- **Remove the Tracheostomy tube / cover stoma**
  - Look, listen and feel at the mouth and tracheostomy. Ensure oxygen re-applied to face
  - Call Resuscitation Team CPR if no pulse / signs of life
- **Is the patient breathing?**
  - NO
  - YES
  - Continue ABCDE assessment
- **Primary Emergency Oxygenation**
  - Standard Oral airway manoeuvres
    - Cover the stoma (swabs / hand)
    - Use: Bag-valve-mask
    - Oral or nasal airway adjuncts
    - Supraglottic airway device e.g. LMA
  - Tracheostomy Stoma ventilation
    - Paediatric face mask applied to stoma (in Resus Trolley)
    - OR LMA applied to stoma (in Difficult Airways Trolley)
- **Attempt Oral intubation**
  - Prepare for difficult intubation
  - Uncut tube, advanced beyond stoma
  - Small Tracheostomy tube / 6.0 cuffed ETT
  - Consider Airway catheter and fiberoptic scope / Bougie / Airway exchange catheter
A blocked or displaced tracheostomy tube generally presents with respiratory difficulty. The nature of the problem will often be obvious, but if not, it is important to adopt a systematic approach and be aware that acutely ill patients may have other cardio-respiratory reasons for difficulty in breathing. A partly dislodged tracheostomy tube is just as dangerous, if not more dangerous, as a completely removed tracheostomy tube.

**Airway: is the airway (at least partially) patent?**

If the tube is displaced, the patient may be breathing through their nose or mouth. The patient may be safe in the short term, requiring urgent but not emergency action. Only experienced staff should try to replace the tube under such circumstances. If in doubt it will usually be safer to remove a partly dislodged tube, although a suction catheter or airway exchange catheter may be first advanced through it to allow oxygen administration. The airway should be maintained by other methods until experienced help arrives.

If tube is partially occluded, the patient may still be able to breathe through it, but with difficulty. If the tube has an inner cannula, this should be removed and changed. If the tube does not have an inner cannula, but a suction catheter can be passed down the tracheostomy tube, then it must be at least partially patent. It may be possible to change the tube over a catheter or other airway exchange device.

*Chest X rays do not give useful information about the position of the tube in this situation. Fibreoptic bronchoscopy may be useful if it is immediately available, but this should not delay treatment.*

**Bleeding from a tracheostomy**

Bleeding is the most common complication of tracheostomy. Bleeding may occur early (within 48 hours of formation of the tracheostomy) or late (several days afterwards). It may be minor (settles with simple conservative management) or major (requiring transfusion of blood and/or blood products) and surgical exploration may be needed to identify and deal with the source of bleeding.

The management of bleeding from a tracheostomy therefore depends upon the context in which the bleeding occurs.

**Early minor bleeding**

Oozing from the stoma site is the most common type of bleeding seen following formation of a tracheostomy. Most commonly, this is the result of the effects of the vasoconstrictor used to infiltrate the incision site wearing off. Blood staining of the dressings may be noted, or there may be blood staining of tracheal secretions. Large volumes of fresh blood represent significant bleeding, which may require surgical exploration.

1. **DON’T PANIC!**
2. Call for help—senior medical and nursing staff, other AHPs with tracheostomy care skills (e.g. physiotherapist).
3. Reassure patient.
4. Whilst maintaining control of the tracheostomy tube, remove the tracheostomy tube holder and dressing.
5. Clean stoma site with sterile saline.
6. Inspect stoma site, looking for any obvious bleeding point.
7. Apply manual pressure to any obvious bleeding point – this may be sufficient to stop minor oozing. Suturing locally may also be effective.
8. If still bleeding, infiltrate any obvious bleeding point with dilute adrenaline (1:80,000 to 1:200,000). If no obvious bleeding point, infiltrate the stoma margins with dilute adrenaline.
9. If still oozing, apply Kaltostat packing to stoma to promote local clot formation. The Kaltostat may also be soaked in dilute adrenaline.
10. Check full blood count and a coagulation screen. Correct any abnormalities in the standard fashion.
11. If bleeding is not stopped by these measures, refer to ENT or other appropriate surgeon for surgical exploration. Then seek surgical haemostasis in theatres with appropriate senior surgeon. Junior surgeons should not explore such wounds alone as major vessel bleeding and a compromised airway will require senior ENT, cardiothoracic or vascular expertise.

**Major early bleeding**
Proceed as minor bleeding guideline. Note that large volumes of blood in the trachea may cause respiratory embarrassment – the patient may need further respiratory support in a critical care area.
Beware of the risk of the tracheostomy tube becoming occluded by blood clot. If this occurs, proceed as per blocked tracheostomy tube guideline. If airway obstruction occurs due to blood clot in the airway (look with a laryngoscope) then direct suction on the tracheal tube with suction tubing may be required to remove it. Large clots will not pass through a suction catheter or a fibreoptic bronchoscope suction channel.
1. In most situations of significant bleeding, secure the airway by translaryngeal intubation with the cuff below the stoma so the airway is protected from blood entering the trachea from the stoma. Then temporary haemostasis of the stoma can be achieved by digital pressure, packing the wound with gauze or deep tension sutures.
2. Early referral to an appropriate SENIOR surgeon (e.g. ENT, cardiothoracic, vascular expertise) for urgent surgical exploration is necessary.
3. Ensure that cross-matched blood is made available.

**Late bleeding**
Late bleeding may occur because of erosion of blood vessels in and around the stoma site. This is more likely if there has been infection of the stoma site. Such bleeding may settle with conservative management, as described in the early bleed guideline. More worryingly, however, is the prospect of such bleeding being the result of erosion of a major artery in the root of the neck where there has been pressure from the tracheostomy tube itself or the cuff tube (another reason why it is important to ensure that the tracheostomy tube cuff pressure is monitored to avoid over-inflation). Most commonly, this erosion occurs into the right brachio-cephalic artery (also known as the innominate artery), resulting in a tracheo-innominate artery fistula. This situation may be heralded in the preceding hours by a small, apparently insignificant, sentinel bleed. Bleeding from such a fistula will be massive. THIS IS A LIFE-THREATENING EMERGENCY and so decisions need to be rapidly made. It is well recognised that fatalities occur in this situation, and that this may be the mode of death for some patients with head and neck cancers. The appropriate management
may, therefore, be palliation. Such a decision should, ideally, have been made in advance and in discussion with the patient and family, clearly documented in the patient’s medical notes and communicated to the nursing staff (who will undoubtedly have to deal with this situation). If active treatment is still the plan, proceed as below:

1. **DON'T PANIC**
2. Call for help—senior medical and nursing staff, other AHPs with tracheostomy care skills (e.g. physiotherapist).
3. Reassure the patient.
4. Proceed as described in the major early bleeding guideline.
5. Bleeding may be temporarily reduced or stopped by applying finger pressure to the root of the neck in the sternal notch, or by inflating the tracheostomy tube cuff (if present) with a 50ml syringe of air. This inflation should be done slowly and steadily to inflate the balloon to a maximum volume without bursting it. Depending on the type and size of the tracheostomy tube this may be anywhere between 10 and 35 ml.
6. Urgent referral for surgical exploration must now be made, if not already done so. In addition to an ENT or maxillofacial surgeon, it may be necessary to get help from a vascular surgeon. Sometimes, the damage can only be repaired utilising cardio-pulmonary bypass, and so a cardiothoracic surgeon may also be needed to help. The operating theatres must be informed that this is an NCEPOD Category One emergency, as any delay will increase the risk of death significantly.
End of Life Care

We aim to provide high quality end of life care in Neuro Intensive Care Unit.

If the decision of withdrawal of life sustaining treatment has reached the following actions should be take place:

- Referral to the Specialist Nurse for Organ Donation (SNOD) BEFORE speaking to the family.
- When possible have the SNOD present when break bad news to the family
- Initiate comfort measures if the sedation has stopped (includes management of secretions)
- After talking to the family document in the notes the content of your conversation
- Please fill in the DNAR form
- If you think that the patient will maintain spontaneous circulation for more than 24 hours involve the palliative care team.
- Discharge the patient to an appropriate ward environment (side room)
- Clearly document decisions for NOT escalating treatment – DISCHARGE

SUMMARY

If the patient has suffered a catastrophic neurological event and seems that he/she is brain stem dead:

- Initiate catastrophic Brain Injury pathway
- Call the SNOD
- Prepare to perform Brain Stem Tests (regardless donation)
- With the SNOD present explain to the family the course of events, brain stem tests and offer to the family to witness brain stem tests.
- Follow NICE guidelines and national guidelines for confirming death

Documentation for deaths occurring in NITU:

The confirmation of death should ALWAYS follow the “Code of practice for diagnosis and confirmation of death”

- Confirmation of death according to Neurological criteria Form (after brain stem death tests
- Certification of Cardiac death Form
- Death Certificate if it is not a coroner’s case
- Coroner’s report form – UPLOAD ONTO ICLIP
- Cremation form
- **Discharge summary on iClip** (if you have completed a coroner’s report this can be used as discharge summary)
#### Certification of Cardiac Death

**Record of death**

<table>
<thead>
<tr>
<th>Patient's Name</th>
<th>Date of birth</th>
<th>Gender (circle)</th>
<th>M / F</th>
</tr>
</thead>
<tbody>
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<table>
<thead>
<tr>
<th>St George's Hospital No.</th>
<th>NHS number</th>
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</tbody>
</table>

PLEASE fill this form on the FIRST page in the patient's notes folder

1a. **Clinical confirmation of death (see notes overleaf):**

- Name of Practitioner (please print)
- Sleep no. AND Mobile phone no.
- I confirm the absence of:
  - a central pulse
  - any audible heart sounds
  - any respiratory effort
- AND if present OR in the context of a specific / complex clinical setting e.g. non-heart beating organ donation, I confirm the absence of:
  - any pulsatile flow in an arterial line
  - AND/OR any contractile activity on echo
  - AND/OR any rhythm other than asystoles on continuous ECG for at least 5 minutes.
- Following this, I confirm the absence of:
  - any pupillary response to light
  - any corneal reflexes
  - AND any motor response to supra-orbital pressure

- Date and time of death

1b. **Hospital classification of type of death (see definitions and actions overleaf):**

- Expected
- Unexpected but unavoidable
- Unexpected and potentially avoidable

**Brief explanation of reasoning behind classification:**

<table>
<thead>
<tr>
<th>Signature</th>
<th>GMC / NMC No.</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

2. **Information recorded on the Medical Certificate of Cause of Death (see notes overleaf):**

   a.

   b.

   c.

   d.

   e.

   f.

   g.

   h.

   i.

   j.

   k.

   l.

   m.

   n.

   o.

   p.

   q.

   r.

   s.

   t.

   u.

   v.

   w.

   x.

   y.

   z.

If not completed, why?

Case has been discussed with the coroner's officer (see overleaf for indications and contact no.)

Yes ☐ No ☐ If yes, what was the outcome.

Cremation 4 - Medical certificate completed ☐

Name(s) and contact details of other team members present AND / OR able to complete cremation form 4

<table>
<thead>
<tr>
<th>Consultant in charge of care</th>
<th>Name</th>
<th>GP</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>

3. **This death has been communicated to:** (more space provided overleaf)

<table>
<thead>
<tr>
<th>Next of kin</th>
<th>Name(s)</th>
<th>Consultant in charge of care</th>
<th>Name</th>
<th>GP</th>
</tr>
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</table>
Catastrophic Brain Injury Pathway

Catastrophic Brain Injury Care Pathway

Do you suspect brain stem death? Yes/No
Are pupils fixed and dilated and GCS 3/15 Yes/No
Is the patient apnoeic (not triggering ventilator)? Yes/No
Are cough and gag reflexes absent? Yes/No
Has a decision to stop neuroprotection been made? Yes/No

If ‘Yes’ to all of above questions please commence the following checklist.

Time starting the protocol

Page Specialist Nurse on Organ Donation: 07659100103 (time: )

Ventilation Nurse and NICU Dr

<table>
<thead>
<tr>
<th>Targets:</th>
<th>pO₂ 8-14 kPa</th>
<th>pCO₂ 5-6.5kPa</th>
</tr>
</thead>
</table>

Additional Actions
- Sit up the patient at an angle of approx 30° - 45° and turn 3hrly
- Recruitment manoeuvre by medical team to optimise lung ventilation (eg. CPAP mode 25-40 cmH₂O for 30-50 secs)
- Set PEEP 8-10 cm H₂O
- Lung Protective ventilation (TV 6-8mls/kg, Peak pressure ≤30cm H₂O)
- Repeat recruitment manoeuvre if pO₂ ≤ 10.0kPa
- Review ventilation 2 hourly – repeat recruitment manoeuvre if deteriorating

Circulation Nurse & NICU DR

- Insertion of Central Line
- Calibrated LiDCO (Please record LiDCO number: …….)
- Start cardiovascular algorithm (time: )

Renal and Electrolytes

<table>
<thead>
<tr>
<th>Targets:</th>
<th>Urine output 0.5-2.5ml/kg/hr</th>
<th>Na 135-150mmol/L</th>
<th>K+ 4.0-5.5mmol/L</th>
<th>Mg &gt; 0.8mmol/L</th>
<th>Ca ionised 1.0-1.3mmol/L</th>
</tr>
</thead>
</table>

Additional Actions
- If polyuria (>300mls/hr for 2 hours) ensure adequate volume replacement
- If DI, bolus DDAVP 0.5mcg - consider vasopressin infusion if not started
- If oliguria, despite optimisation of CVS, consider Dobutamine

Hormones and Haematology

<table>
<thead>
<tr>
<th>Targets:</th>
<th>BM 4.0 – 9.0 mmol/L</th>
<th>Hb ≥ 8g/dL, Plt &gt; 50 x 10⁵/L</th>
<th>INR &lt; 2.0, APTTR &lt; 1.5, Fib &gt; 2.0g/L</th>
<th>Temperature 35.5 – 37.5</th>
</tr>
</thead>
</table>

Additional Actions
- Methylprednisolone 15mg/kg IV bolus (repeat after 12 hours)
- Start Insulin at one unit per hour and titrate to achieve BM control of 4.9mmol/L. If hypoglycaemia, continue insulin and supplement with 20% dextrose – do not stop Insulin altogether
- Continue enteral feed at low volume (10-30mls/h)
The catastrophic brain injury pathway is initiated for patients that appear to be brain stem dead and the decision for neuroprotection to stop has been made. The sole purpose of the pathway is to manage patients in order to be able to perform brain stem tests as soon as possible. In order to introduce safely the pathway you should be able to answer “YES” to all 5 questions at the beginning of the checklist.

Referral to Specialist Nurses for Organ Donation is mandatory as per hospital policy, NICE guidelines and GMC recommendations. Early involvement will allow to access if the patient is potential donor and to support the family and their needs (regardless donation).

100% referral of all Deaths to specialist Nurses for organ donation is mandatory 100% Brain Stem Testing is aimed
Organ Donation

Identification and Referral of DBD donor

- Patient suspected of being Brain Stem Dead (comatose, Fixed pupils, apnoeic)
- Discuss with Consultant Intensivist and Nurse In Charge
- Medical Team support for Brain Stem Testing (treat Diabetes Insipidus, support with inotropes, fluid resuscitation)
- Contact Specialist Nurse for Organ Donation (SN-OD)
- Consultant to discuss Brain Stem Death Tests (BSDT) with family. SN-OD present for this discussion—known as collaborative approach
- SN-OD spends time with family assessing understanding of BSD & meeting family needs
- 1st set of BSD tests performed
  CONTACT CORONER - seek permission for donation
- 2nd set of BSD tests performed
1. If donor unsuitable for solid organ donation, consider Cornea/ Tissue donation

2. If patient is not brain stem dead but further support considered futile, please assess suitability for Donation after Cardiac Death.
Identification and referral of DCD donors

Criteria for Identification
Significant Neurological injury including hypoxia

**DECISION ABOUT WITHDRAWAL OF SUPPORT**
GCS ≤ 5/15 (WITH NO SIGNIFICANT AMOUNT OF SEDATION)
Plan to withdraw ventilation and remove Endotracheal/Tracheostomy Tube

Refer to Specialist Nurse for Organ Donation (SN-OD)
Page 07659100103

CONTACT CORONER
Seek permission for donation

Unsuitable

Consider Cornea /Tissue Donation

Yes

Page Tissue Coordinator 0207-253-1199

No

Coordinator role:
1. End of life care as ICU protocols
2. Support family
3. Meet spiritual and cultural needs
4. Inform coroner if required
5. Document in patient records for audit
6. Tissue retrieval teams will liaise with mortuary and consent will be provided

ICU senior staff discusses futility and withdrawal of support with the family.
SNOD present – collaborative approach

Organ Donation to be raised only when the family accepted futility/withdrawal of support by a trained requester

Suitable

**Anaesthetist role:**
Withdrawal all treatment including ETT
1. Death certified using cardiac criteria
2. Death declared following 5 min asystole
3. Palliative care measures can be administered

If Asystole does NOT occur 120min after extubation donation process stopped patient returns to ICU or Ward as previously planned.