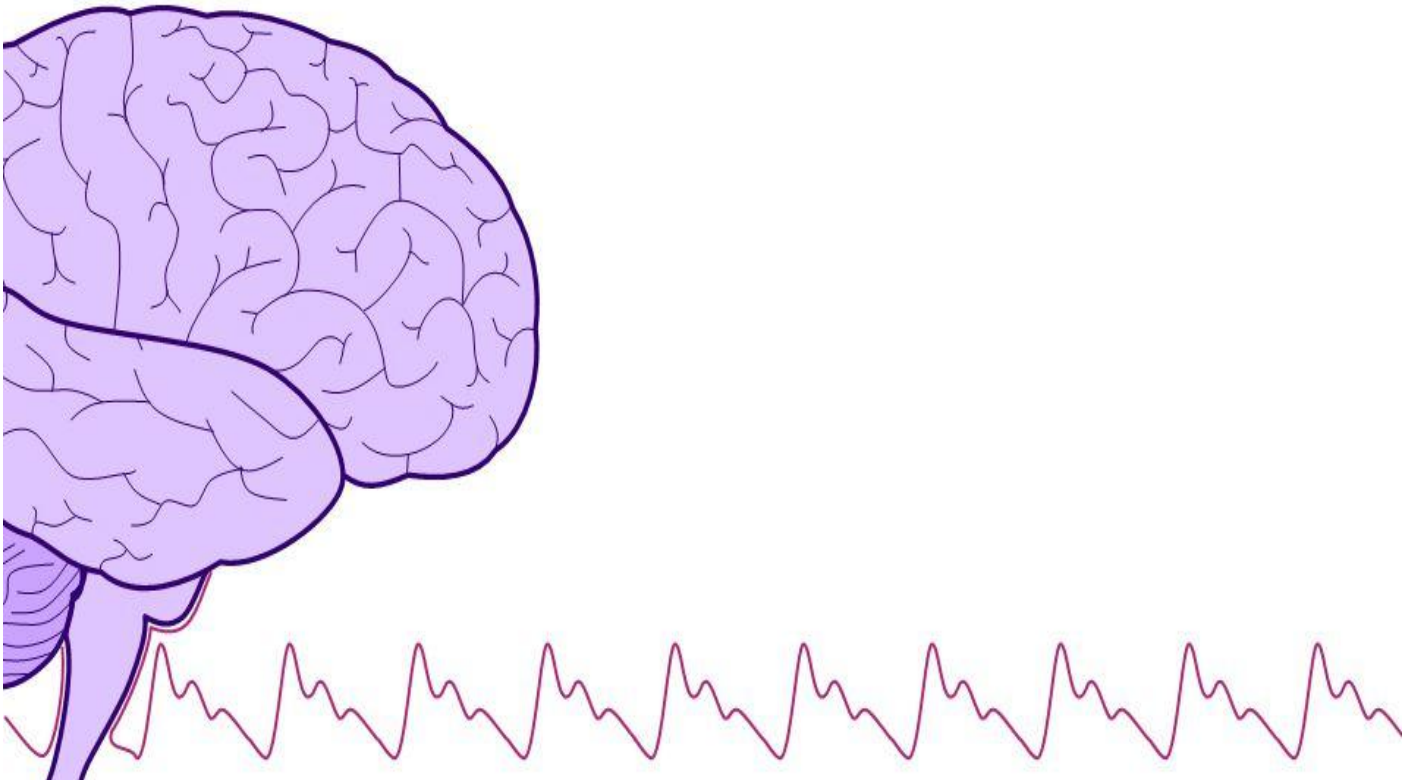


# NEURO-INTENSIVE CARE

## Guidelines

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

<http://stginet/greybook>



**2020**  
**COVID-19 edition**

Edited by AZ  
To be reviewed: 2021

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## Medical documentation

Currently all documentation is electronic using iCLIP. Please use instructions (Picture 1) to set up your profile correctly.

**It is expected all NICU patients will have notes written daily (using standardised proformas) (Picture 2 &3).**

There are admission forms, daily assessment forms and discharge/episode summaries. All patients should have notes written in each shift 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.

*Picture 4 has instructions on how to use iCLIP during ward rounds and how to solve the "corruption bug".*

**Please note that spinal cord injury patients have different forms: Spinal Cord Admission and daily review forms.**

**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 duration and reason for the antibiotic prescription. This information needs to be filled both in the daily proforma and antibiotic prescription.**

The "**Adult Critical Care Core Plan**" can be found in Orders and includes all the usual orders/prescriptions in NICU. Please use it for your prescriptions.

All patients should have a formal **ICU episode summary** on iClip. This should not be named discharge summary unless the patient dies. Please follow instructions on Picture 5.

**Before any invasive procedure you should fill an Invasive Safety Procedure Record & LocSSIP**

## Picture 1

# Clinical documentation on iCLIP

### Setting the *Default Note Type*

This is important to allow critical care clinical entries to be filtered from the mass of other documents that litter most patients' records. You only need to do it once:

1. Open any patient record
2. Click **+Add** next to 'Documentation' in the sidebar
3. Open the **'View'** menu at the top of the screen
4. Select **'Customise'** (last option in menu)
5. Open the **'Document Types'** tab
6. Wait for the system to catch up with the enormity of this request (there will be a few seconds in which it looks like it's died)
7. In the **'Default Note Type'** drop-down menu, scroll down to the 'Crit Care Medicine' section and select **'Crit Care Medicine – Med Progress Note'**
8. Click **'Okay'**

Now when you create a new document it will by default have this tag. This means that when faced with pages and pages of notes in the document list, you can select *Display > Only... > Note type > Crit Care Medicine – Med Progress Note*, so all others are filtered out.



## Picture 2

### Accessing neurointensive care (NICU) notes templates

1. Add a new document by clicking '+Add' next to 'Documentation' in the sidebar
2. Select the 'Pre-configured' tab
3. Type 'NICU' into the Search box
4. This will bring up the various template documents, which currently comprise:
  - NICU admission
  - NICU daily review
  - NICU elective post-op daily review and discharge summary
  - NICU discharge summary
  - NICU referral record
  - NICU discussion with family/friends/others
  - NICU capacity and best interests assessment
  - (And two non-medical templates)
5. For convenience, select all the above documents (hold 'Ctrl' to select them simultaneously) and then click 'Add to Favourites'
6. You can then access them by selecting the 'Favourites' tab (if none appear, click the 'All Pre-configured template' radio button)

## Picture 3

### Completing notes entries

1. Open the relevant template by double-clicking on it, either in your 'Favourites' tab or after searching in the 'Pre-configured' tab, as described above.
2. Enter the necessary information in the template.
3. When you have finished working on the document (whether or not it is fully completed), click on 'Sign/Submit'. You can still re-open and amend the document later, as can others.
4. Please *avoid* using the 'Save' option except as a means to save what you are doing *while working on the document*. If you click 'Save & Close', the document will not be part of the patient's record, and nobody else will be able to view or complete it.

## Picture 4

### Managing multiple users and the 'corruption bug'

Appropriately, only one user can modify a document at a time. This is generally unproblematic. There are, however, a few considerations:

**1. On ward rounds, it is probably better to allocate workstations to *tasks rather than people*.** For example, have one workstation for documentation, and the other one (or perhaps two) for pathology/radiology/prescribing. When moving to the next patient, the doctor who reviewed that patient should go to the 'documentation workstation' to open and update their daily review. Resist the temptation to open this on whatever computer happens to be in front of you, in case someone else simultaneously does the same. The system dislikes this, and will seek to exact revenge in cruel and unexpected ways (see 'occasional bug', below).

**2. Always close notes when you've finished working on them.** Either 'Sign/Submit' if you've made changes, or 'Cancel' (+/- 'Discard changes') if not.

**3. If you have an over-enthusiastic index finger, control it.** There is a habit among some users to double-click on notes entries (and therefore open them for editing) when they only need to view them. This is mildly problematic if someone else needs to edit the note at the same time. It is *intensely annoying* if that user then removes their smartcard and goes home, having still not closed the document...

There is an **occasional bug** in which, when attempting to Sign/Submit a note, an error message appears telling you that the note is corrupted. Whatever option you choose, the changes you have just made will be lost. We think that the problem occurs more often when users (especially different users) open/modify the same note in rapid succession. The simple but tedious workaround is to cut and paste the note before saving. Until we have identified and corrected the source of this problem, my suggestion is that if you don't want to risk losing your work, press 'Ctrl+A' then 'Ctrl+C' before clicking Sign/Submit. Then, if you encounter the bug, you can simply close the error message and press 'Ctrl+A' then 'Ctrl+V' to re-insert the whole (modified) note. Inexplicably, it should then be possible to Sign/Submit it without problems.

## Picture 5

The screenshot shows a 'New Note' form with the following details:

- \*Type: General Clinical Note
- \*Date: 25/01/2018 11:23 GMT
- Title: NICU discharge summary

The 'Pre-configured' tab is circled in green. Below the form is a search bar with 'Nicu' and a 'Contains' dropdown. A 'Restricted display by' section includes checkboxes for 'My notes only', 'Note Type', 'Associated Diagnosis', 'Recent', and 'Favourites'. A table of notes is displayed below:

Title	Document template	Shared	Last Changed By	Perform/Service Date/Time	Sentences
NICU Caseload Discussion	Dynamic Content Creation	Y	Tudor , Rosie	16/02/2017 17:30:15	
NICU daily review (DRAFT)	Dynamic Content Creation	Y	Hitchings , Andrew William	27/11/2017 10:38:33	
NICU discharge summary	Dynamic Content Creation	Y	Hitchings , Andrew William	10/01/2018 09:49:48	
NICU Physiotherapy Initial Assessment	Dynamic Content Creation	Y	Gawned , Sara Louise	27/04/2016 14:24:19	
NICU referral record (DRAFT)	Dynamic Content Creation	Y	Hitchings , Andrew William	24/01/2018 17:17:57	

## Useful numbers

Neurosurgical Registrar on call: Bleep 7242

Senior Anaesthetic Registrar: Bleep 7647

Senior General Anaesthetic Registrar: Bleep 6111

Duty Floor Anaesthetist: Bleep 8011

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.

## Head Injury Management in NICU

The aim of managing a patient with a severe brain injury is to minimise **secondary injury**.

You may be involved with care of these patients prior to ICU admission. For guidance, please refer to the following document:

[Head injury: assessment and early management – NICE Clinical Guideline 176](#) <sup>1</sup>

### Mild or moderate brain injury

These patients are sometimes intubated for transfer. If the neurosurgical advice is to wake such a patient then sedation should be **stopped**, neurological assessment performed and if appropriate, the patient should be extubated. If they are not suitable for extubation, sedation should be recommenced pending a more senior opinion. This process should take into consideration issues such as other significant injuries, difficult airway, staffing levels etc.

### Patients with unclear neurology

The initial GCS assessment performed is sometimes inaccurate. This can greatly influence 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 (ICP), 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 component is the motor response), the patient should either be re-sedated and treated as per the following guidelines **or** treated as a patient with a mild or moderate head injury, depending upon their neurological assessment.

As a rule, patients in this situation should not be weaned or extubated unless they are localising appropriately, or better. 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 cerebral perfusion pressure (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 because they have undergone surgical decompression. Therefore, they are at minimal risk of herniation.

Prior to stopping sedation, **it should be clear from the neurosurgical team what the goal is**. If the patient has a poor neurological assessment, they should be re-sedated and treated according to the following guidelines.

**Any concerns about what to do with patients in this situation should be directed to the Neuro ICU consultant on-call.**

## Severe brain injury +/- evidence of raised ICP or poor neurological assessment

The initial management of these patients will be greatly influenced by the neurosurgical opinion. It will also vary according to the severity of injury, CT scan, initial GCS and presence of advanced neurological monitoring. **Waking these patients up has the potential to cause significant harm.**

### Monitoring

- Intracranial pressure monitors should be inserted in all salvageable patients with a severe TBI and an abnormal CT scan, ideally in the least trauma-affected frontal lobe
- An abnormal CT scan of the head is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns
- ICP monitoring is indicated in patients with severe TBI with a normal CT scan if two or more of the following features are noted at admission: age over 40 years, unilateral or bilateral motor posturing, or systolic BP < 90 mmHg<sup>2</sup>
- If available, a brain tissue oxygen (PbtO<sub>2</sub>) monitor should also be inserted. The Raumedic Neurovent-PTO is currently on trial at SGH. It allows ICP, PbtO<sub>2</sub> and temperature monitoring via one catheter

### Sedation

Patients should be sedated with the initial target of unresponsive to suctioning and painful stimuli.

This will often require the addition of inotropes to maintain a satisfactory CPP

- All patients should receive an opioid infusion, usually fentanyl - up to 200 mcg/hr
- **1st line sedative:** Propofol - max dose 4mg/kg/hr
- **2<sup>nd</sup> line sedative:** Add midazolam if propofol requirements > 4mg/kg/hr
- Check plasma lipids every 2 - 3 days on patients on high dose propofol
- Weaning propofol may lead to a rise in ICP and should be avoided until the patient is stable, or if complications arise
- Avoid the temptation to wake a patient with a severe TBI **for the first 48 hours** and not until ICP is stable. Sedation may then be slowly withdrawn, provided ICP does not deteriorate

### Paralysis

- Patients should not be routinely paralysed
- If required as part of the management of elevated ICP, atracurium is the preferred infusion, titrated to two train-of-four twitches
- The aim should be to wean paralysis off as soon as possible
- Paralysis has not been shown to improve outcomes, is associated with complications and cannot therefore be recommended, other than in exceptional circumstances

### Ventilation

- Use a volume-controlled mode of ventilation (VC or PRVC)
- Target PaCO<sub>2</sub> 4.5-5.0 kPa if ICP stable
- PaO<sub>2</sub> > 10 kPa (SpO<sub>2</sub> 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
- Target 4.0-4.5 kPa if ICP elevated, whilst initiating 2nd line treatments
- Prolonged hyperventilation should be avoided
- Maintain minimum PEEP level of 5 cmH<sub>2</sub>O. PEEP may be increased to maintain adequate oxygenation as required and is unlikely to influence ICP
- After any changing ventilator settings, a repeat ABG should be performed after 30 minutes

### Haemodynamics

- The arterial transducer should be zeroed at the level of the external auditory meatus. This is to ensure correct calculation of cerebral perfusion pressure <sup>3</sup>
- The LiDCO haemodynamic monitor is highly recommended to guide fluid and inotrope requirements
- Target a minimum CPP of 60 mmHg
- Avoid aggressive attempts to maintain CPP above 70 mmHg <sup>2</sup>
- In the absence of ICP monitoring, aim for a MAP of 90 mmHg
- 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

- Target core temperature should be  $37 \pm 0.5$  °C
- Sources of infection should be considered and actively treated
- Patients should receive regular paracetamol, with the addition of NSAIDs and external cooling for resistant cases and active cooling
- If pyrexia persists despite these measures, endovascular cooling should be initiated
- Early prophylactic hypothermia does not improve neurologic outcomes <sup>4</sup>

### Fluids / transfusion

- Normal saline should be used as initial maintenance fluid
- Serum sodium should be maintained at  $\geq 140$  mmol/l
- Target euvolaemia. Fluid requirements should be reviewed daily. Remember to include drugs in the calculation
- Assessment of volume status is an imprecise task. The best approach is to integrate blood pressure, heart rate, urinary output and central filling pressures (where available). LiDCO monitoring, including stroke volume variability, may be useful
- Avoid albumin and non-protein colloids, including hydroxethyl starch <sup>5</sup>
- Haemoglobin should be maintained  $> 90$  g/l. A restrictive transfusion strategy may be associated with harm in TBI patients <sup>6</sup>

## Nutrition

- Severe TBI is associated with increased energy expenditure
- Commence early enteral feeding. Supplement with IV normal saline to achieve daily fluid requirements
- Prescribe laxatives - initially senna and sodium docusate +/- polyethylene glycol
- Failure to reach full caloric replacement by day 7 post-injury may be associated with higher mortality; consider parenteral nutrition in these circumstances
- If GI motility is reduced (aspirates > 500ml) commence metoclopramide 10mg IV TDS
- Transpyloric feeding may reduce incidence of ventilator-associated pneumonia
- Request dietician review early

## Glycaemic control

- Hypoglycaemia is extremely detrimental to the brain and should be avoided
- Intensive glycaemic control does not improve mortality but does increase the risk of hypoglycaemia <sup>7</sup>
- Intensive control may improve neurologic outcome but requires further investigation
- Glucose should be maintained below 10 mmol/l, with an insulin infusion if necessary

## Stress ulcer prophylaxis

- All patients should receive some form of gastric protection
- PPIs lower the risk of clinically important GI bleeding, as compared to H2RAs, without increasing the risk of pneumonia, mortality or length of ICU stay <sup>8</sup>

## Antibiotics

- Not routinely required, unless surgically indicated
- Although early-onset ventilator-associated pneumonia is common in TBI <sup>9</sup>, there is little current evidence for prophylactic antibiotics
- Prescribe antibiotics according to current hospital and ICU guidelines if evidence of infection or sepsis, or if already on antibiotics for > 48 hours

## Seizure control

- There is modest evidence that antiepileptic drugs reduce the incidence of early post-traumatic seizures (PTS) in severe TBI <sup>10</sup>
- Prophylactic anticonvulsant should be limited to 7 days post-injury
- First-line agent is currently levetiracetam
- Patients with witnessed seizures should continue or commence anticonvulsants
- Preferred route of administration is enteral, after IV loading dose
- For further information please refer to section on epilepsy in these guidelines

## Transfer patients to radiology/theatres

- Patients should be fully monitored throughout and mechanically ventilated
- If unstable, muscle relaxants should also be used
- ICP monitoring should continue
- The aim should be to maintain the same parameters as on the neuro ICU



## Management of raised intracranial pressure

- Initial targets are ICP < 22 mmHg and CPP > 60 mmHg<sup>2</sup>
- If ICP is elevated, follow guidance in flowchart “management of raised intracranial pressure”
- If brain tissue oxygen (PbtO<sub>2</sub>) monitor also available, follow guidance in flowchart “management of brain tissue oxygenation and intracranial pressure”
- 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.

# Management of raised Intra-Cranial Pressure

## Initial Targets

Sedation level so patient is unresponsive: Propofol (max 4mg/kg) + Fentanyl ± Midazolam  
Head Elevation 15-30°  
Temp < 37.5 °C / Blood Glucose 4-8 mmol/L / Euvolaemia  
PaCO<sub>2</sub> 4.5-5.0, PaO<sub>2</sub> >10kPa  
CPP = 60-70 mmHg

**ICP > 22 mmHg?**



## STEP 1

Check airway and breathing: ABGs, airway pressures  
Consider pneumothorax, airway obstruction, sputum retention and treat if necessary  
Check circulation: CPP > 60mmHg, Euvolaemia – Use LiDCO  
Check head position and venous drainage (remove collar if present)  
Ensure fully sedated, Normothermia - consider the use of BIS  
AED if seizures suspected



**ICP > 22 mmHg?**



## Step 2

Administer osmotic agent: Hypertonic saline (5% 1-2 ml/kg) or mannitol (0.25-1g/kg)  
Repeat osmotic agent PRN if serum osmolality < 320 mmol/kg and patient euvolaemic  
PaCO<sub>2</sub> 4.5 kPa  
**Notify neurosurgical team**



**ICP > 22 mmHg?**



## Step 3

### Consider repeat CT scan

BIS (maximal effect at 5-20) or EEG monitoring is recommended  
Discuss second tier therapies with responsible consultant

## Second tier therapies – consultant discussion

Barbiturate coma  
Hypothermia to 35 °C  
CSF drainage  
Decompressive craniectomy  
**Or reconsider goals of treatment**

# Management of brain tissue oxygenation (PbtO<sub>2</sub>)

**Aim ICP < 22mmHg and brain tissue oxygenation (PbtO<sub>2</sub>) > 20mmHg**  
 PbtO<sub>2</sub> > 50mmHg may indicate hyperaemia  
 Perform 100% O<sub>2</sub> test if concerns over reliability: FiO<sub>2</sub> 100% and observe for improved PbtO<sub>2</sub>: if none, consider probe fault

PbtO<sub>2</sub> > 20mmHg?

### Type A

ICP < 22 mmHg  
 Continue current therapy

### Type B

ICP > 22 mmHg  
 Ensure CPP > 60mmHg  
 Target ICP reduction as per guidelines  
 Check airway, breathing, CO<sub>2</sub>, sedation, temperature

PbtO<sub>2</sub> < 20mmHg?

### Type C ICP < 22 mmHg

**Tier 1 (no specific order)**  
 Increase CPP to maximum 70 mmHg  
 Increase FiO<sub>2</sub> to 60%  
 Increase PaO<sub>2</sub> by adjusting PEEP  
Optimise CO – use LiDCO  
 Ensure temp < 38 °C  
 Consider AEDs

**Tier 2 (no specific order)**  
 Increase FiO<sub>2</sub> to 100%  
 Increase CPP to 70-80 mmHg  
 Increase PaO<sub>2</sub> by 0.5 kPa, provided ICP remain < 20  
 Increase sedation  
 Hb > 100g/L

### Type D ICP > 22 mmHg

**Target ICP reduction as per guidelines, then,**

**Tier 1 (no specific order)**  
 Increase CPP to maximum 70 mmHg  
 Increase FiO<sub>2</sub> to 60%  
 Increase PaO<sub>2</sub> by adjusting PEEP  
Optimise CO – use LiDCO  
 Ensure temp < 38 °C  
 Consider AEDs

**Tier 2 (no specific order)**  
 Increase FiO<sub>2</sub> to 100%  
 Increase CPP to 70-80 mmHg  
 Increase PaO<sub>2</sub> by 0.5 kPa, provided ICP remain < 20  
 Increase sedation  
 Hb > 100g/L

**Tier 3 (consultant discussion as per ICP guidelines)**  
 Barbiturate coma  
 Temp 35 °C  
 EVD  
 Craniectomy

## Guidelines for the use of high dose thiopentone to control ICP

The use of thiopentone to control ICP is controversial with no clear evidence of benefit in terms of morbidity or mortality. Thiopentone can also lead to profound haemodynamic instability. The Brain Trauma Foundation guidelines suggest high-dose barbiturate therapy may be used to control elevated ICP refractory to maximum standard medical and surgical treatment, but that haemodynamic stability is essential before and during barbiturate therapy.

Thiopentone must therefore:

- be instituted only following discussion with the NICU consultant
- be considered only for salvageable patients (those with a potentially good outcome)
- be considered only if ICP is refractory maximal medical and surgical therapies
- be used only in the context of haemodynamic stability

### Dosing regimen

There is wide variability in dose required to achieve EEG burst suppression and an unclear end point. Previous recommended regimens have been described for pentobarbital and what follows is based on these:

- Use BIS monitor to avoid overdose and titrate the level of sedation
- Ensure the BIS monitor is working correctly
- A BIS level of approximately 10 correlates with EEG burst suppression
- Loading dose: 10mg/kg over 30 minutes
- Repeated loading doses may be required to achieve adequate levels of sedation

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. If ICP is not controlled and BIS > 10, further loading doses of 5 mg/kg may be given 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 will increase the risk of side effects. If ICP is still uncontrolled and BIS < 10 seek senior medical advice.

- Once ICP is controlled commence an infusion, range: 1-8mg/kg/hr (start at 3-4 mg/kg/hr)
- 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 ICP IS STABLE, SLOWLY WEAN OFF PROPOFOL

Thiopentone should only be weaned once ICP has been stable for 24-48 hours (preferably 48 hours).

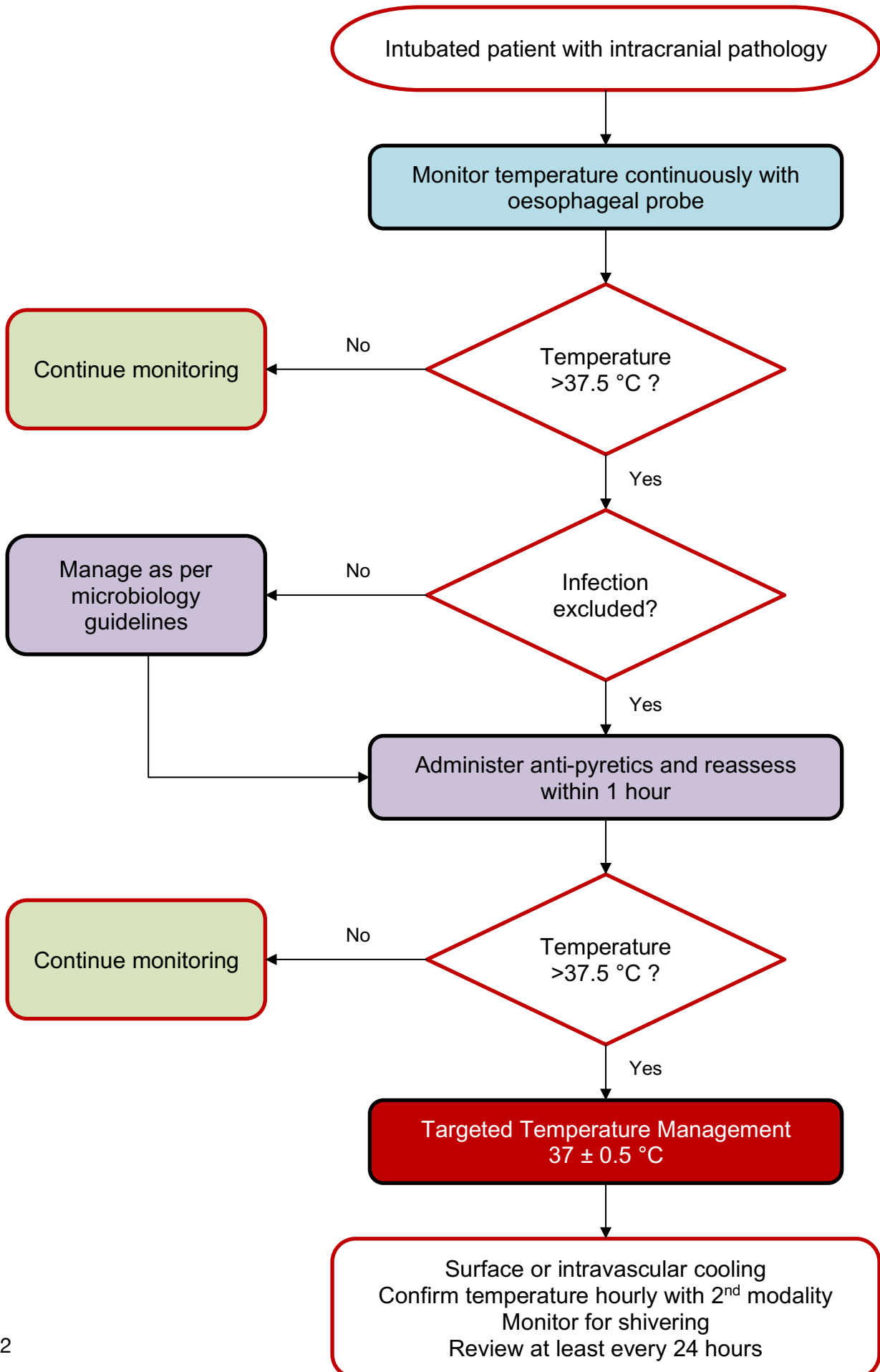
Plasma thiopentone levels are not routinely indicated. They should be measured if there is concern that accumulation may be preventing a patient from waking up. It is important example is to exclude thiopentone coma as a cause of a patient being unresponsive prior to performing brain stem tests.

Complications of thiopentone coma include hypotension, sepsis, rhabdomyolysis, hypokalaemia, rebound hyperkalaemia and polyuria. It is important to be vigilant for these side effects.

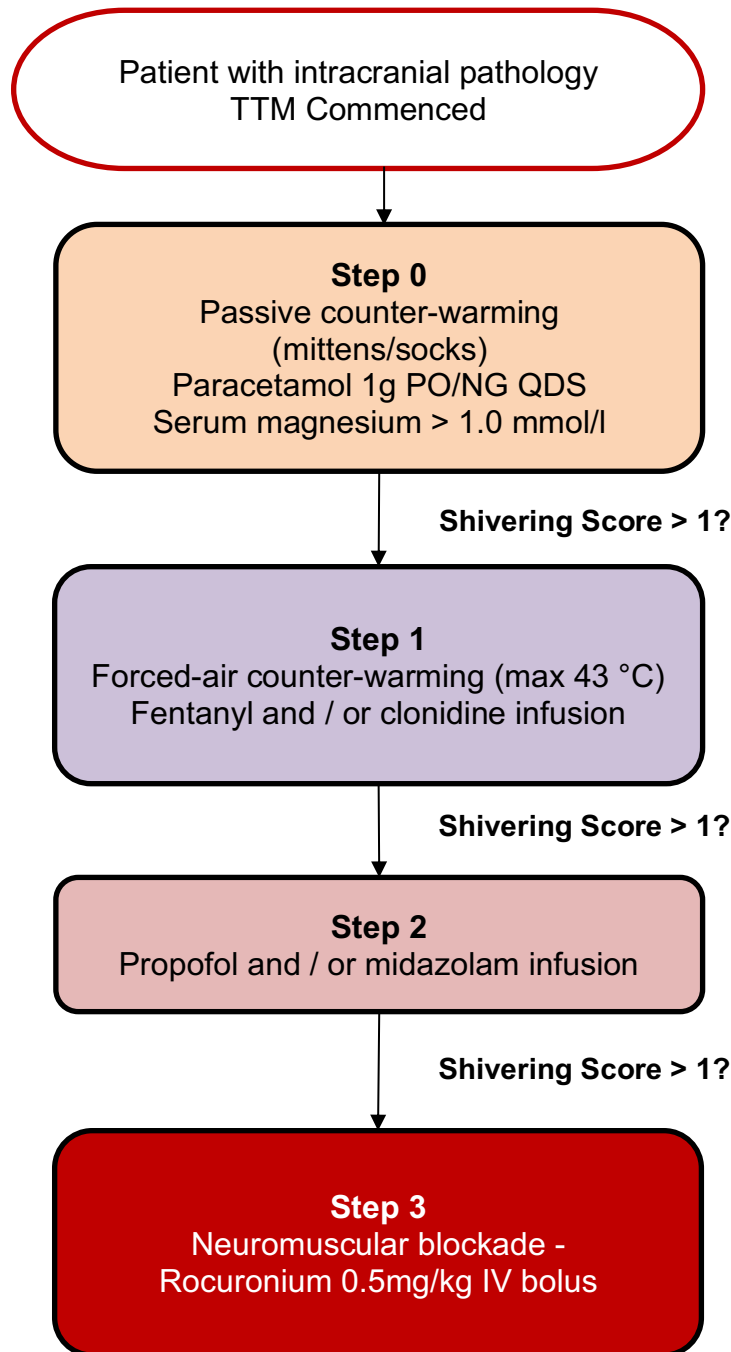
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## Temperature management in NeuroICU



## Management of shivering during TTM



Shivering score	Type	Features
0	None	No shivering detected on palpation of masseter, neck or chest
1	Mild	Shivering localised to neck or chest, or seen on ECG only
2	Moderate	Shivering involves intermittent movement of upper extremities
3	Severe	Gross movements of upper and lower extremities

## 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 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 MgSO<sub>4</sub> 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, PO<sub>4</sub>, 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, PO<sub>4</sub>
  - Maintain electrolytes in a high normal range  
K > 4.5 mmol/l, Mg > 0.9 mmol/l, Adjusted Ca > 2.3 mmol/l, PO<sub>4</sub> > 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 **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

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 traumatic injury should be considered at risk of spinal injury.**

### 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.**

## Integrated Care Pathway in NICU

All patients with Spinal injury T8 or above should be admitted to NICU

### **Within 4 hours** from admission

Hard collar fitted correctly  
Vital capacity is measured and recorded  
Arterial line inserted  
Central line inserted (if needed)  
Naso/orogastric tube is inserted & position confirmed (if needed)  
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  
Speech & Language Therapy referral  
Dietician referral  
Spinal Unit referral.

## Management of Spinal cord injury (by systems)

### Airway

Look for neck swelling- cervical spine fractures can cause soft tissue swelling that can slowly compromise the airway- vital signs & arterial blood gases may be normal.  
Ask the patient if they breathe comfortably. Prepare for difficult intubation.

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 fiberoptic scope is not helpful in the acute management of the airway of spinal cord injured patients.*

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

### Breathing

The most common respiratory complications are respiratory failure, neurogenic pulmonary oedema (high cervical cord injuries), pneumonia and pulmonary embolism.

Ineffective cough, reduced secretion clearance, atelectasis and hypoventilation are results of diaphragmatic and chest wall muscle weakness.

Other pulmonary physiologic changes after SCI include changes in lung and chest compliance, airflow limitation and bronchial hyperresponsiveness and changes in ventilatory control.

- Measure forced vital capacity twice daily on non-intubated patients. Normal physiological range: male 70 ml/kg, female 55 ml/kg. Lesions C3-C6 vital capacity is 20% of normal, lesions T1-T4 vital capacity is 30-50% of normal.
- Signs of impending respiratory failure (Increased RR, reducing FVC, rising PaCO<sub>2</sub>, falling PaO<sub>2</sub> are indications for intubation and mechanical ventilatory support
- Consider use of prophylactic “birding” &/or BiPAP/ High flow O<sub>2</sub> for lesions above T11
- Consider invasive ventilation if FVC < 15ml/kg
- Some patients breathe better when lying completely flat.

Physiotherapy should be initiated as soon as possible with a goal to prevent atelectasis and pneumonia.

### Haemodynamic management

**Neurogenic shock** refers to hypotension, usually with bradycardia (especially injuries T6 and above), due to interruption of autonomic pathways in the spinal cord causing decreased vascular resistance. Patients with traumatic SCI may be suffer also from haemodynamic shock due to blood loss. Exclude other injuries that can cause hypotension. A combination of careful fluid resuscitation, blood products and vasoconstrictors may be needed. The use of LiDCO is helpful and is recommended to guide management.

Adequate MAP is believed to be important in maintaining adequate perfusion to the spinal cord and minimizing secondary ischaemic injury. Current guidelines are based on empirical data and expert opinion

### **Aim for MAP ~ 80- 95 mmHg.**

Increased vagal activity may cause **bradycardia**, often triggered by airway manipulation. In these cases, pre-oxygenation and atropine are useful preventative measures. Severe bradycardia that does not respond to atropine may require external pacing and cardiology referral for temporary pacing.

Urine catheter insertion will allow an accurate measurement of fluid balance.

### **Gastrointestinal management**

High risk for stress ulcers, especially for patients with cervical injuries. Gastric acid protection is mandatory. Prophylaxis with proton pump inhibitors is recommended upon admission for four weeks.

Paralytic ileus is common. The incidence of aspiration may be as high as 35%. Insert a naso/orogastric tube within 4 hours from admission. Establish feeding slowly with the addition of pro-kinetics as needed. Monitor bowel motility (bowel sounds, bowel emptying, gastric emptying).

Commence early bowel care with two laxatives and PRN suppositories – please follow bowel management protocol as early as possible. Refer to relevant chapter.

### **DVT prophylaxis**

SCI patients are amongst the highest risk groups for developing VTE. The greatest risk is between 72 hours and 2 weeks. LMWH should be administered as soon as contraindications are excluded. Mechanical methods, intermittent pneumatic compression (IPC) or graduated compression stockings are used as adjuncts to LMWH.

In the post-fixation period, LMWH is usually safe to be given 24 hours postoperatively unless the neurosurgical team has advised differently.

The VTE prophylaxis is continued for at least 8-12 weeks.

### **Steroids**

Current guidelines **do not recommend** the use of high doses of steroids.

### **Pressure sores**

Can develop quickly, even within hours. Most common on the buttocks and heels. After spinal stabilisation patients should log rolled (side to side) every 2-3 hours to minimise the risk for pressure sores. Early spinal fixation helps with the overall nursing of these patients.

### **Temperature control**

Patients with high spinal cord injury become poikilothermic (temperature varies according to the environment). They lack vasomotor control (cannot vasoconstrict) and they are not able to sweat.

## Spinal Assessment

It is important to have a formal documentation of the spinal injury for prognosis. We use the ASIA score (American Spinal Injury Association). **All patients should have an assessment on admission** OR as soon this is possible. This should be repeated after spinal shock resolves (usually in 72 hours).

A chart to record your findings can be found either in the document drawers or online:

[https://asia-spinalinjury.org/wp-content/uploads/2016/02/International\\_Std Diagram Worksheet.pdf](https://asia-spinalinjury.org/wp-content/uploads/2016/02/International_Std Diagram Worksheet.pdf)

Advice on how to perform an ASIA assessment can be found on:

[https://asia-spinalinjury.org/wp-content/uploads/2016/02/Key\\_Sensory\\_Points.pdf](https://asia-spinalinjury.org/wp-content/uploads/2016/02/Key_Sensory_Points.pdf)

[http://asia-spinalinjury.org/wp-content/uploads/2016/02/Motor\\_Exam\\_Guide.pdf](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 **72 hours of admission**. A proforma is on the desktop computers. Please speak to our specialist nurse OR our physiotherapists.

## Advanced Spinal Cord Injury Course

The NICU educational team has designed and running an MDT course on management of the spinal cord injured patient. This has online and hands-on components. Please speak to the practice educators on NICU to book a place on it.

## ISCOPE study

Since 2010 we are running in NICU the Injured Spinal cord evaluation study. It is an observational study that involves monitoring of intraspinal pressure (ISP) after traumatic spinal cord injury (TSCI). Research fellows are heavily involved and available to answer any questions.

## Autonomic dysreflexia

Autonomic dysreflexia is unusual within the first month of SCI but usually appears within the first year. **It is a life-threatening syndrome** in spinal injuries above T6 (can be down to T10). Effectively is a manifestation of the loss of coordinated autonomic responses to demands on heart rate and vascular tone. Uninhibited or exaggerated sympathetic responses to noxious stimuli below the level of the injury lead to diffuse vasoconstriction and hypertension. A compensatory parasympathetic response produces bradycardia and vasodilation above the level of the lesion, but this is not sufficient to reduce elevated blood pressure. SCI lesions lower than T6 do not produce this complication, because intact splanchnic innervation allows for compensatory dilatation of the splanchnic vascular bed.

### Typical stimuli include:

- bladder distention or infection,
- bowel impaction,
- pressure sores,
- bone fracture
- occult visceral disturbances
- surgery on area below level of cord injury

### Recognition and avoidance of inciting stimuli are important in preventing attacks

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### Typical stimuli include:

- bladder distention or infection,
- bowel impaction,
- pressure sores,
- bone fracture
- occult visceral disturbances

### Recognition and avoidance of inciting stimuli are important in preventing attacks

Clinical manifestations are hypertension, bradycardia, headache, vasodilation above level of cord injury, facial flushing, nasal congestion, blurred vision, nausea and diaphoresis.

### Management of acute attacks includes:

- Measuring and monitoring blood pressure.
- Immediately sitting the patient upright to orthostatically lower blood pressure.
- Removal of tight-fitting garments.
- Searching for and correcting noxious inciting stimuli. Bladder distension and fecal impaction are the most common precipitants. Bladder catheterization and evaluation

for urinary tract infection (UTI) should be undertaken; indwelling catheters should be checked for obstruction, and a rectal examination should be performed.

- Prompt reduction of blood pressure with a rapid-onset/short-duration agent, depending on the severity of attack and response to above measures.

#### **Medications often used in this setting include**

- Nitrates (should be avoided in patients who may use Sildenafil)
- Nifedipine 10mg po
- Hydralazine 10mg IV
- Labetalol 10mg IV
- Analgesia

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# Aneurysmal Subarachnoid Haemorrhage Management

## Initial admission

1. ABCDEFG
2. Head up 30 degrees
3. Maintain oxygenation,  $\text{PaO}_2 > 10 \text{ kPa}$  & normocapnia.
4. Routine bloods on admission plus **troponin**
5. Neurogenic cardiomyopathy – treat dysrhythmias, acute pulmonary oedema and cardiogenic shock as required.
6. Fluid and inotrope therapy guided by the use of CO monitoring eg LiDCO
7. Baseline ECG
8. If hyponatraemia present ( $\text{Na} < 135 \text{ mmol/L}$ ), send urinary Na
9. Correct coagulopathy, eg vitamin K and prothrombin complex for warfarin reversal
10. Avoid over sedation which may impair assessment of neurological status
11. Treat seizures; occur in up to 20% of patients with aSHA, load with anti-epileptic usually levetiracetam in this Trust.

## Glucose control

Start insulin if hyperglycaemic (aim for blood glucose 6- 10mmol/L). Caution to avoid hypoglycaemia which can be detrimental to the injured brain.

## Anaemia

Anaemia is common in aneurysmal SAH. The optimum haemoglobin level which balances flow, viscosity and oxygen delivery is unknown, but theoretically it is Hct of 0.30 (30%).

Aim is for a minimum Hct of 0.30 (30%). Some treatment algorithms suggest Hb should be maintained  $> 90 \text{ g/dl}$  but be cautious.

## Blood pressure management

Autoregulation is lost after aSAH thus the brain is reliant on MAP for brain perfusion. Hypertension (SBP  $> 160 \text{ mmHg}$  or MAP  $> 110 \text{ mmHg}$ ) should be treated in patients with unsecured aneurysms. Use rapid acting onset/offset drugs eg labetalol, hydralazine. Hypertension can be tolerated once the aneurysm has been secured.

- 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 relevant chapter.

## Fever

Pyrexia ( $T > 38.3$ ) occurs in up to 70% of patients with SAH. Although cause is usually non-infectious, need to seek and treat infection: LRTI's, UTI's, lines and EVD-associated ventriculitis.

Pyrexia is associated with poor neurological outcome and needs aggressive control. Regular use of paracetamol +/- infusion of NSAID. Senior clinician involvement. Please refer to TTM chapter.

Effective methods: use of surface cooling devices eg Artic Sun although patients normally need to be sedated if actively cooled.

Treat shivering: magnesium, pethidine, sedation, paralysis if indicated. Please refer to relevant chapter/algorithm.

### Thromboprophylaxis

There is up to 20% chance of VTE, higher risk with higher grades of aSAH. Avoid LMWH until at least 24 hours after aneurysm is secured. Normally decision made after discussion with neurosurgeons/neuroradiologists.

### Fluids

Close monitoring of fluid balance and fluid status. Avoid hypotonic solutions – monitor Na.

**Use the LiDCo to manage these patients-** All fluid boluses should be NaCl 0.9% unless Na > 140 mmol/L.

### Electrolyte abnormalities

Electrolyte abnormalities are common after SAH. The most common abnormality is hyponatraemia, defined as a serum Na of < 133 mmol/L, which normally results from CSW and SIADH less often. Please refer to relevant chapter.

Diagnosis is based on both clinical and biochemical markers (see table below). Serum Na levels should be monitored carefully on a daily basis and if Na starts to drop quickly or Na < 135, a sodium screen should be sent (urinary Na, paired osmolalities and a urine SG). Sodium therapy in the form of slow Na tablets (600 mg 4-6 hourly), slow NaCl 0.9% 100ml/hour and +/- fludrocortisone tablets 100mcg 6 hourly can be started.

A senior doctor should be involved in making these decisions. Further information on electrolyte abnormalities can be found in the Sodium and Fluid balance chapter.

## *SIADH and CSW<sup>11</sup>*

<b>Characteristic</b>	<b>SIADH</b>	<b>CSW</b>
Extracellular Fluid Volume	Normal, Increased	Decreased
Urine Osmolality	High	High
Plasma Osmolality	Low	Low
Serum Sodium	Low	Low
Urine Sodium	High	Very high
Urine Output	Normal or Low	High
Treatment	Fluid Restriction	Fluids &/or mineralocorticoids

### Medications

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

### 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 clopidogrel either singly or in combination
5. Neurogenic pulmonary oedema and cardiac stunning. May cause a low cardiac output, hypoxia and decreased brain perfusion, requiring inotropes (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.

## 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 or Delayed Cerebral Ischaemia-DIC).

DCI is defined as a change in the level of consciousness (decrease in two points on the GCS scale) or the development of a new focal deficit lasting for at least one hour and not explained by other causes.

Exclude **other causes of neurological deterioration**, such as re-bleed, hydrocephalus, seizures and fluid or electrolyte abnormality. Often vasospasm is a diagnosis of exclusion.

Vasospasm (as assessed by angiogram) occurs in the majority of patients who have a SAH but only ~30% will have clinical signs of vasospasm. Symptoms can be reversible if treated promptly and aggressively, otherwise DCI progresses to cerebral infarction, associated with higher mortality.

**There is no evidence prophylactic HHH therapy confers any benefit in asymptomatic patients.** In asymptomatic patients ensure ABC's. The patient should be normovolaemic, well oxygenated, and well perfused with a good blood pressure.

Remember the ABC's; there is no point in increasing cerebral blood flow if the patient is hypoxic.

**Ensure the patient is well oxygenated (SaO<sub>2</sub> > 97%, PaO<sub>2</sub> > 10 KPa).**

### Prevention of DCI

- Ensure the patient is well oxygenated. (SaO<sub>2</sub> > 97%, PaO<sub>2</sub> > 10 KPa)
- Maintenance of euvolaemia, consider use of CO monitoring eg LiDCO
- Avoid hyponatraemia. High risk of CSW/SIADH, consider fludrocortisone if indicated.
- In patients who are either post-surgical clipping or radiological coiling, it is appropriate to omit anti-hypertensives provided blood pressure is not too high.
- Avoid hypo/hyperglycaemia
- Maintain normothermia

### DCI Management

- Exclude **other causes of neurological deterioration** prior to starting hypertensive therapy, such as re-bleed, hydrocephalus, seizures and fluid / electrolyte abnormality
- Institute haemodynamic augmentation (stepwise SBP/MAP increase titrated to neurological response) (see below)

- Hypertensive therapy should **only to 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.
- **Mild** hypervolaemia is acceptable in patients with DCI
- If no improvement, consider urgent endovascular therapy (eg intra-arterial nimodipine- neuroradiology suite)

## 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 ICU.

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 do 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 which balances flow, viscosity and oxygen delivery is unknown. Aim for Hb ~ 90

## 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 an 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 vasopressor therapy.

Currently there is no evidence to choose one vasopressor 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 afterload and further myocardial injury which require careful discussion with senior ICU staff.

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 hypertensive therapy may result in an increase in cerebral oedema or haemorrhage.
4. If not responding to hypertensive 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- usually it is a decision of the whole MDT.

Like most acute neurological conditions, we prefer a period of stability before removing therapy, if hypertensive therapy has improved a patient's condition it should not be withdrawn as soon as possible but when it is no longer needed.

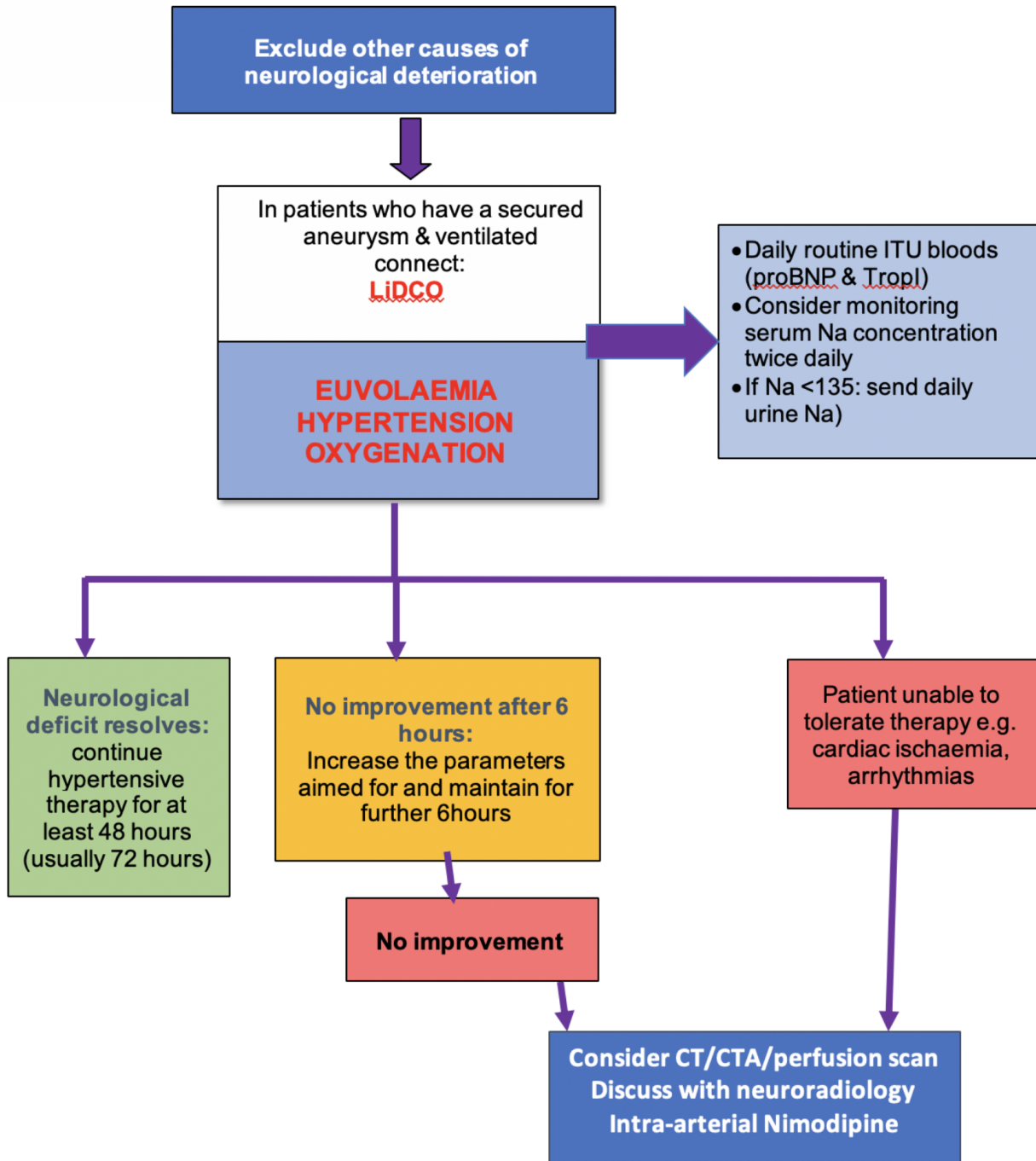
**We usually maintain therapy while the patient is stable for 48-72 hours and then slowly withdraw therapy.**

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 hypertensive therapy and continue for another 24-48 hours.**

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## Management of symptomatic DCI Algorithm



## 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 are 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:

- $\geq 5$  minutes of continuous seizures, or
- $\geq 2$  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 (>30minutes 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 pharmaco-resistance.

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: NeuroICU

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

<b>Phenytoin</b>	<b>20mg/kg IV</b>
<b>Levetiracetam</b>	<b>60mg/kg IV (max 4500mg)</b>
<b>Sodium Valproate</b>	<b>40mg/kg IV (max 3000mg)</b>

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

**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)

**Induction:****Propofol 1.5-2.5 mg/kg****Fentanyl 1-2 mg/kg**Suxamethonium 1-2mg/kg (unless contra-indicated) or **Rocuronium****Sedation:**

Propofol up to 4 mg/kg/hr

Fentanyl up to 5 mcg/kg/hr

Avoid paralysis, though this may stop visible fits, cerebral seizure activity may continue.

Ensure electrolytes are normal

Continue antiepileptic medication and check levels

Maintain blood pressure at normal or supra-normal levels (+/- inotropes)

Maintain normocapnia and adequate oxygenation

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)

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## 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)

	Features	STESS
<b>Consciousness</b>	Alert or somnolent/confused	0
	Stuporous or comatose	1
<b>Worst seizure type</b>	Simple-partial, complex-partial, absence, myoclonic	0
	Generalized-convulsive	1
	Non-convulsive status epilepticus in coma	2
<b>Age</b>	<65	0
	≥65	2
<b>History of previous seizures</b>	Yes	0
	No or unknown	1
<b>Total</b>		<b>0-6</b>

**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 24hours**
  - **Start an IV steroid**
    - **Methylprednisolone 1g IV/day for 3 days followed by 1mg/kg/day IV.**
- **LiDCO**
- **Antithrombotic therapy**

Drug	Initial Bolus	Continuous infusion	Things to consider
<b>Midazolam</b>	0.1-0.6mg/kg at a rate 2mg/min	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	Hepatic metabolism Tachyphylaxis occurs
<b>Propofol</b>	2-4mg/kg	4 -10mg/kg/h <b>Avoid using above 5mg/kg/h for more than 48hours</b> Increase infusion rate by 0.2mg/kg/h until flat EEG or burst suppression.	Involuntary movements <u>Propofol infusion syndrome</u> : check serum lactate (<2.5), triglycerides (<5), CK (<2000). <b>Use ideal body weight.</b>
<b>Thiopentone</b>	1-3 mg/kg	1-5mg/kg/h  1-2mg/kg boluses + increase infusion by 0.5mg/kg/h every 6hours max 800mg/h	Zero order pharmacokinetics Needs loading with repetitive boluses and accumulates in adipose tissue.

### Stage 3: Ongoing management in Neurointensive Care Unit

- **Start maintenance anticonvulsant during continuous anaesthetic infusion**

Drug	Dose/Route	Caution/ Things to consider
Phenytoin	100mg TDS, IV until stable levels	
Leveriracetam	1000mg-1500mg BD, IV/NG	
Sodium Valproate	10mg/kg divided in 2-3 doses requires loading dose: 20-30mg/kg/min	Avoid in liver disease, Mitochondrial disease, porphyria

- **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 NICU (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 <b>for &lt;48hours</b> 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: Lidocaine, 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.

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## Summary of Management of Status Epilepticus in NeuroICU

consider admitting to NICU

### On Admission to NICU

max Midazolam & Propofol

Maintenance dose of anticonvulsants (maximum 2 agents)

Diagnosis: repeat LP

EEG to check control of seizures

### Day 2 on NICU

Wean slowly GA agents

Consider steroids

MRI

Use continuous EEG

### Day 3 on NICU:

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.

## Intracerebral Haemorrhage

Intracerebral Haemorrhage (ICH) is the second most common cause of stroke after ischemic stroke in frequency (9-27% of all strokes). Incidence increases by Age, doubling every 10 years after 35 y.o. Rate is highest in Asians, intermediate in black race and lowest in white race. No clear gender distribution although it might be higher in men than women.

### Risk Factors - Aetiology

Hypertensive vasculopathy (HTN): Most common factor and aetiology. Causes deep ICH

Cerebral Amyloid Angiopathy (CAA): Associated with Old Age. Primary cause of Lobar ICH

Anticoagulant/Antithrombotic therapy:

Warfarin: Increase risk of ICH 2-5-fold. INR >3 =>Poor Outcome

New Oral Anticoagulants (NOA) (Dabigatran, Rivaroxaban), lower risk than warfarin

Antiplatelets: AAS + Clopidogrel have an increased risk of ICH.

Drugs (Cocaine, amphetamines, alcohol):

Alcohol is associated with a 3-fold increase in ICH

Vascular abnormalities: AVM, Dural fistulas, Haemorrhagic Infarctions, Moyamoya disease, Vasculitis, Cerebral Hypoperfusion Syndrome

Infections: Septic embolism, CNS Infections

Others: Low Cholesterol, Black Race, Brain Tumours

### Presentation

Headache, Vomiting (50% of patients), Decreased Level of Consciousness (LoC).

Stupor/Coma is an ominous sign.

Seizures occur in 15% of patients during the first few days (often in the setting of a lobar ICH)

Cardiac Abnormalities: Prolonged QT, ST-T waves changes with mild elevations of Troponin. ECHO: Global or regional wall motion abnormalities, and Reduced Ejection Fraction. Ventricular arrhythmias may occur if the brainstem is compressed.

### Signs and Location

- Putamen/Globus Pallidus (56%): Hemiplegia, Hemisensory loss, Gaze palsy, Stupor and coma
- Internal Capsule Haemorrhage: Mild dysarthria, Contralateral hemiparesis and sensory deficit
- Thalamic (31%): Hemiparesis, Sensory loss, transient hemianopsia. Can present Aphasia.
- Lobar (14%): Affects often parietal and occipital lobes. High incidence of Seizures. Contralateral hemianopsia.
- Cerebellar (7%): Unsteady balance, Vomiting, Headache (occipital, usually referred to neck/shoulder), neck stiffness, gaze palsy, facial weakness. Patients are usually stuporous due to hydrocephalous or brain stem compression. Surgery is essential.

- Pontine: Leads to deep coma over the first few minutes. Total paralysis, pinpoint but reactive pupils. There might be ocular bobbing, facial palsy, deafness and dysarthria once patient is awake

## Diagnosis

Lab tests: FBC, Electrolytes, Urea, Glucose, Creatinine, Clotting profile, Troponins, Liver Function Profile, Toxicology screen if suspected drug abuse

Brain imaging: CT head: define size and location.

Brain MRI: 100% sensitivity and accuracy

## Treatment

During the acute phase they may require intubation with mechanical ventilation, blood pressure control, reversal of anticoagulation, ICP treatment, treatment for seizures, and surgical treatment. Prognosis for individual patients is uncertain, therefore full aggressive care for the initial stage (48-72h) is recommended. DNAR orders should be postponed until the second day (ICS prognostication guidelines).

## BP management

If SBP is between 150-200mmHg: lower to SBP to 140mmHg. SBP below this is not beneficial for reducing death/disability and may increase the risk of renal complications. If SBP > 220mmHg: keep SPB 140 -160mmHg (reasonable target).

For BP management IV infusions are recommended: Labetalol, phentolamine, Calcium antagonist.

## ICP Management

- Head Up to 30 degrees, sedation, avoidance of neck compression devices
- Osmotic therapy: NO differences between Mannitol and Hypertonic.
  - Mannitol: 0.5-1g/kg with further boluses of 0.25-0.5g/Kg.
  - Hypertonic 3% titrated to a sodium goal of 155mEq/L maximum.
- Hyperventilation: PaCO<sub>2</sub> between 4-4.5kPa. Less than 4 may result in ischemia and adverse outcomes. The effect of this intervention only lasts for few hours.
- Neuromuscular blockade and pharmacological coma: Attention to adverse effects: Propofol infusion syndrome after more than 48-72h. Increased risk of Ventilator Assoc. Pneumonia secondary to NMB

## Medical Care

- DVD prophylaxis: Flow-Tron Boots
- Glucose control: Hyperglycaemia after ICH associates with worse outcomes. Hypoglycaemia should be avoided. Optimal goal of glucose is uncertain, but for guidance: 8-10mmol/L
- Avoid Hypervolemia. Use of hypotonic fluids is contraindicated (worsening cerebral edema)
- NG Feeding (Dysphagia is a common sign)
- Seizures: Risk is reported to be around 15%. More common in lobar ICH. AHA/ASA 2015 guidelines recommend against the use of prophylactic seizure drugs. In further

studies there is no evidence to recommend prophylaxis. If seizures do occur the suggestion is to treat them as per protocol.

- Reversal of Anticoagulation: Main goal is to rapidly reverse anticoagulation and to maintain the reversal for at least 72h, limiting the haemorrhage enlargement. This is important as hematoma growth (specially within the first 24h after ICH is an independent predictor of mortality and poor outcome).
  - Warfarin
    - Prothrombin Complex concentrate (PCC) (Beriplex/Octaplex) normalises INR faster than Plasma or Vit K alone (within 10 min of administration).
    - FFP requires large volumes and time (around 30h with a range between 14-50). 3.
    - Vit K iv should be administered with PCC due to the shorter life of the later.
  - NOA: Can be treated with PCC or specific antagonists: idarucizumab for Dabigatran, Andexanet alfa for rivaroxaban, apixaban. Activated charcoal can be used if the last dose was taken between 6-8h.
  - Heparin:
    - UNH can be treated with Protamine based on the aPTT. Use a slow infusion.
    - LMWH (enoxaparin, dalteparin) Protamine is a reasonable treatment although less effective than with UNH
  - Antiplatelets: Transfusion of platelets are not generally indicated during ICH, unless the Plat count is <100.000 or there is known platelet function defect. Evidence suggests that patient with ICH and taking antiplatelets should not receive platelet transfusion unless they meet the above criteria.

## **Surgery**

Cerebellar: Recommended when area of ICH>3cm, neurological deterioration, brainstem compression and/or hydrocephalus due to ventricular obstruction. EVD alone is not recommended. Surgery is associated with decreased mortality but not improved functional outcomes.

Supratentorial: Controversial. Reserved for life threatening mass effect (routine evacuation not recommended). Avoid if patient is either fully awake or comatose. Intermediate level of arousal (stupor-obtundation) is a better feature.

Intraventricular: ICH with intraventricular extension are at risk of hydrocephalous. If deterioration, consider them for EVD.

## **Resumption of Anticoagulation/Antiplatelets**

Antiplatelets: AAS is probably safe to resume after the acute phase of ICH (provided BP is controlled, and the indication and benefits>risks). Avoid antiplatelet in patients with prior lobar ICH in general. Antiplatelets are reserved as secondary prevention of IHD or Stroke/TIA. There is no agreement regarding timing after ICH:1-2 weeks in general. AAS could be safely started within days (48-72h?)

Anticoagulation: AHA/ASA guidelines suggest delaying oral anticoagulation for at least 4 weeks after ICH

## Prognosis

Mortality at 30 days ranges from 30-50% and half of those happen during the first 2 days.

Risk Factors:

- Increasing age
- Low GCS
- Increasing ICH volume: Hematoma growth (within the first 24h) is an independent predictor of mortality
- Presence of IHV
- Deep or Infratentorial ICH location
- Anticoagulation and probably Antiplatelet (oral anticoagulation relates to a mortality rate of 52-73%).

An ICH volume of 60cm<sup>3</sup> or greater on initial CT and GCS<8 predicts a 30-day mortality of 91%

An ICH volume of <30cm<sup>3</sup> and a GCS>9 or more predicts a 30-day mortality of 19%

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## Acute ischaemic stroke: (AIS)

AIS is a leading cause of death and disability in the UK with an incidence of around 85,000 people a year, of which an estimated 20% will require critical care admission during their hospital stay.

The therapeutic aim of available interventions is to restore perfusion to ischaemic penumbra (area of brain tissue surrounding core infarct) either by intravenous thrombolysis with tissue plasminogen activator (tPA) or mechanical intra-arterial thrombectomy or both.

Intensive care for stroke patients is focused on management of complications of reperfusion and minimizing secondary brain injury including brain oedema and progressive stroke by haemodynamic and neurological monitoring and maintaining physiological homeostasis. The decision to admit stroke patients to ICU should be made with careful consideration to likely prognosis, known patients' wishes and presence of co-morbidities.

### Airway management/ventilatory support:

- Oxygen therapy only indicated to maintain SpO<sub>2</sub> > 94%.
- Endotracheal intubation is indicated in reduced GCS <8, respiratory failure, or brain stem dysfunction.

### Blood pressure control:

- Correct hypotension and hypovolaemia to maintain systemic organ perfusion.
- Maintain BP < 185/110 in patients who have received tPA or thrombectomy for the first 24 hours.
- Patients ineligible for tPA/thrombectomy with BP >220/120 should have antihypertensives started aiming at a 15% reduction.

### Temperature:

- Fever > 38<sup>0</sup> C should be treated with antipyretics
- Infective sources should be investigated and treated.

### Glucose control:

- Target serum glucose between 7.5 – 10 mmol/L

### Antiplatelets and thromboprophylaxis:

- Aspirin should be started within 48 hours after onset of stroke
- Thromboprophylaxis with intermittent pneumatic compression devices only.
- Anticoagulation in the acute phase is not recommended but will need to be discussed with stroke teams on an individual basis.

### Management of brain swelling:

- Intermittent use of Hyperosmolar agents such as Mannitol 20% or hypertonic saline maybe used for cerebral oedema treatment. Mannitol is given in boluses of 0.25 – 1 g/kg and is generally discontinued once the osmolar gap is greater than 20mOsm/L; Hypertonic saline 5% can be given in 1-2mls/kg boluses and discontinued once serum sodium is higher than 160mEq/L.

- Decompressive craniectomy significantly reduces mortality and improves functional outcomes in malignant MCA infarctions when performed within 48 hours of stroke onset and in particular in patients < 60 years of age.
- Suboccipital decompressive craniectomy should be considered in patients with cerebellar infarctions causing brain stem compression. Also, ventriculostomy with EVD is recommended if causing obstructive hydrocephalus

### Management of haemorrhagic complications:

The most reliable predictor of haemorrhagic transformation is infarct size.

- Symptomatic intracranial haemorrhage occurring within 24 hours of tPA administration can be reversed with cryoprecipitate 10 units and tranexamic acid 1 gram , it would also be reasonable to maintain BP < 160 mmHg.

### Management of tPA associated orolingual angioedema:

- Awake fibroptic intubation if oedema involving larynx, oropharynx, palate, floor of mouth with rapid progression
- Methylprednisolone 125 mg (IM)
- Cyclizine 50 mg
- Consider 0.5 mg adrenaline (IM) if worsening condition.
- Icatibant (30 mg) is a bradykinin receptor antagonist maybe given subcutaneously and repeated every 6 hours (max 3 doses/24 hours), **will need discussion with pharmacist.**

# Management of Guillain-Barre Syndrome

## Investigations

- Lumbar puncture
- Nerve Conduction studies
- Spirometry
- HIV
- MRI spine
- Anti-ganglioside antibody
- Stool culture
- CXR

A multidisciplinary approach to the acute phase combining supportive and disease modifying therapy is required.

## Respiratory management

Risk factors for progression to mechanical ventilation include

- rapid disease progression
- bulbar dysfunction
- bilateral facial nerve weakness
- dysautonomia

Pulse oximetry and arterial blood gases (ABGs) should not be relied on, as hypoxia or hypercarbia is a late sign and patients will decompensate very quickly.

Bedside spirometry should be performed every 6 hours initially.

Early spirometry will also help triage the patient between the intensive care unit (ICU) and regular ward.

Patients with bulbar dysfunction, high risk of aspiration (i.e., infiltrates on chest x-ray [CXR]), and new atelectasis on CXR should be intubated early for airway protection and impending respiratory failure.

In patients with no bulbar dysfunction or with mild bulbar dysfunction without aspiration risk, the 20/30/40 rule should be used as detailed below.<sup>1</sup> The patient should be monitored in the ICU and elective intubation considered if:

- Vital capacity is <20 mL/kg
- Maximal inspiratory pressure is worse than -30 cmH<sub>2</sub>O (negative inspiratory force)
- Maximal expiratory pressure is < 40 cmH<sub>2</sub>O
- Vital capacity, maximal inspiratory pressure, or maximal expiratory pressure is reduced by 30% from baseline initial measurement.



The mean duration of ventilation is 15 to 43 days, and weaning should be guided by serial pulmonary function tests (PFTs) and assessment of strength.

The need for tracheostomy should be addressed from week 2 onwards, especially if PFTs do not show improvement. If there is improvement of PFTs above baseline, tracheostomy may be delayed by an additional week before reassessment.

## **Cardiovascular management**

Haemodynamic monitoring of pulse and blood pressure (BP) should be started on admission.

If dysautonomia is present, continuous cardiac monitoring and placement of a Foley catheter should be initiated on admission.

All patients with severe disease should have their pulse and BP monitored until they are off ventilator support and have begun to recover.

Fluid balance should be monitored carefully, especially because the autonomic dysfunction renders clinical determination of hydration status very difficult.

Hypotensive episodes can be managed with fluid boluses.

If BP is very labile, then intra-arterial BP monitoring should be initiated. Hypertensive episodes should be treated with short-acting agents (e.g., labetalol, esmolol, and nitroprusside) to prevent abrupt hypotension.

Other factors that may potentiate dysautonomia include manoeuvres such as suctioning, changing position (i.e., lying to sitting), and medicines (antihypertensive drugs, succinylcholine).

## **DVT prophylaxis**

Immobility and hypercoagulability from treatments such as intravenous immunoglobulin (IVIg) can increase the risk of DVT in these patients.

Subcutaneous heparin or enoxaparin and support stockings are recommended for non-ambulatory patients until they are able to walk independently.

## **Pain management**

Gabapentin or carbamazepine are generally recommended in the ICU in the acute phase. Adjuvant therapy with tricyclic antidepressants, tramadol, gabapentin, carbamazepine, or mexiletine may be helpful for long-term management of neuropathic pain.

Opioids may be effective; they may aggravate autonomic gut dysmotility and bladder distension.

## **Immunotherapy**

Immunotherapy comprises IVIg or plasma exchange.

Both have been shown to be equally efficacious.

The choice between them is often institution dependent.

If there is a contraindication to IVIg, such as IgA deficiency or ongoing renal failure, then plasma exchange would be a better option.

Plasma exchange requires close monitoring for electrolyte abnormalities and coagulopathies.

When started within 2 weeks from symptom onset, IVIG has equivalent efficacy to exchange in hastening recovery in patients who require help with walking.

Combination treatment (plasma exchange followed by IVIG) is not recommended.

Unlike in other immune-mediated disorders, corticosteroids in monotherapy do not significantly shorten time to recovery or prevent long-term disability, with oral corticosteroids delaying recovery when compared with placebo,<sup>9</sup> possibly due to harmful effects on denervated muscle.

### Plasmapheresis

The recommended dose is given by central venous catheter, 50 mL/kg bodyweight over 7 to 14 days started within 2 weeks of disease onset.

### Intravenous Immunoglobulin

Dose: 400mg/kg/day for 5 days

The goal of IVIG is to hasten recovery and reduce long-term morbidity. It is recommended for ambulatory patients within 2 weeks from the onset of neurological symptoms.

Possible mechanisms for beneficial effects include blockade of Fc receptors on macrophages preventing antibody targeted attachment on Schwann cell membrane and myelin or on axolemma in axonal variants of GBS; regulation of autoantibodies or cytokines by anti-idiotypic or anti-cytokine antibodies in the pooled immunoglobulin; and interference with the complement cascade or regulatory effects on T cells.<sup>10</sup>

### Rehabilitation

This is recommended in the acute phase.

It comprises gentle strengthening involving isometric, isotonic, isokinetic, and manual resistive and progressive resistive exercises.

It should be focused on proper limb positioning, posture, orthotics, and nutrition.

A multi-disciplinary approach has been shown to improve disability and quality of life as well as reduce fatigue.

### Complications

Complication	Time Frame	Likelihood
<b>Respiratory failure</b>	Short term	Medium
<b>Bladder areflexia</b>	Short term	Medium
<b>Adynamic ileus</b>	Short term	Medium
<b>Paralysis</b>	Short term	Medium
<b>Fatigue</b>	Long term	High
<b>Hypercalcemia from immobility</b>	Long term	Low
<b>DVT</b>	Variable	Medium

## Mortality

The overall prognosis of GBS is good, with approximately 85% of survivors making a good functional recovery. Mortality of 20% has been shown in ventilated patients.

Prognosis worsens with older age.

Factors associated with poorer outcome include

- more severe weakness
- rapid onset
- older age
- muscle wasting
- electrically in-excitabile nerves
- Preceding diarrhoeal illness

Miller-Fisher syndrome has a better prognosis than other GBS subtypes. Most severely disabled patients with acute motor axonal neuropathy have been found to walk independently within a few years.

Most patients with a poor outcome have been mechanically ventilated. Mortality of 20% has been demonstrated in these patients. Recovery from severe disease may be prolonged, but most patients are able to walk independently.

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# Myasthenia Gravis

Myasthenia Gravis is an autoimmune disorder caused by antibodies against acetylcholine (ACh) receptors in skeletal muscle. The clinical features are weakness and fatigability.

Myasthenic Crisis is considered to be MG complicated by respiratory weakness, oropharyngeal weakness or inability to clear secretions, requiring intubation.

Exacerbations may be spontaneous or triggered by factors such as: infection (particularly respiratory), electrolyte abnormalities (eg. hypokalaemia, hypocalcaemia, hypermagnesaemia), drugs (eg. aminoglycosides, b-blockers), surgery (eg. thoracic, abdominal), pregnancy, pyridostigmine dosing errors, or initiation of high-dose corticosteroids.

Predictive factors of requiring mechanical ventilation post-surgery include long pre-operative duration of myasthenia (> 6 years), chronic respiratory disease, high acetylcholinesterase requirements (eg. pyridostigmine > 750mg/day) and pre-operative VC < 2.9L.

## Principles of management

- 1) Chest physiotherapy including monitoring vital capacity and maximal inspiratory pressure.
- 2) Identify and treat the precipitant.
- 3) Optimise anticholinesterase medication.

Neostigmine (IV infusion) is indicated to avert intubation or facilitate liberation from mechanical ventilation, but where enteral pyridostigmine is insufficiently effective due to unreliable absorption or unavailability of the enteral route.

(See next chapter for details on converting between pyridostigmine daily dose enterally to neostigmine IV continuous infusion starting rate.)

If mechanically ventilated, consider temporary cessation or reduction of anticholinesterase drugs to reduce respiratory. An optimal dose of pyridostigmine should ideally be found prior to extubation to maximise success.

- 4) Avoid hypokalaemia, hypocalcaemia and hypermagnesaemia.
- 5) Escalate treatment if the above steps fail to improve the clinical condition.

High-dose corticosteroids (eg. methylprednisolone 1g IV loading if mechanically ventilated, then prednisolone 50-100mg/day enterally) AND either PLEX (5 exchanges of 3-4L over a 2-week period) or IVIG (total dose 1-2g/kg over 1-2 consecutive days) to be commenced simultaneously.

- 6) Muscle relaxation.

Suxamethonium can be used safely with dosages 1.5-2mg/kg. Patients are very sensitive to non-depolarising muscle relaxants and reduced dosages (eg. 10%) should be used.

## Drugs that may unmask or worsen myasthenia gravis

<b>Drugs that may unmask or worsen myasthenia gravis</b>
<b>Anesthetic agents</b>
Neuromuscular blocking agents ¶
<b>Antibiotics</b>
Aminoglycosides ¶ (eg, gentamicin, neomycin, tobramycin)
Fluoroquinolones (eg, ciprofloxacin, levofloxacin, norfloxacin)
Ketolides ◇ (eg, telithromycin)
Macrolides (eg, azithromycin, clarithromycin, erythromycin)
<b>Cardiovascular drugs</b>
Beta blockers (eg, atenolol, labetalol, metoprolol, propranolol)
Procainamide
Quinidine
<b>Other drugs</b>
Anti-PD-1 monoclonal antibodies (eg, nivolumab and pembrolizumab)
Botulinum toxin
Chloroquine
Hydroxychloroquine
Magnesium
Penicillamine
Quinine

<b>Drugs usually well tolerated in myasthenia gravis but occasionally associated with an exacerbation*</b>
<b>Anesthetic agents</b>
Inhalation anesthetics (eg, isoflurane, halothane)
Local anesthetics <sup>Δ</sup> (eg, lidocaine, procaine)
<b>Antibiotics and antiviral agents</b>
Antiretroviral agents (eg, ritonavir)
Clindamycin
Metronidazole
Nitrofurantoin
Tetracyclines (eg, doxycycline, tetracycline)
Vancomycin
<b>Anticonvulsants</b>
Carbamazepine
Ethosuximide
Gabapentin
Phenobarbital
Phenytoin
<b>Antipsychotics and other psychiatric drugs</b>
Butyrophenones (eg, haloperidol)
Lithium
Phenothiazines <sup>§</sup> (eg, chlorpromazine, prochlorperazine)
<b>Glucocorticoids<sup>¥</sup></b>
Dexamethasone
Methylprednisolone
Prednisone
<b>Ophthalmic drugs</b>
Betaxolol
Echothiophate
Proparacaine
Timolol
Tropicamide
<b>Other drugs</b>
Cisplatinum
Emetine (Ipecac syrup)
Fludarabine
Glatiramer acetate
HMG CoA reductase inhibitors (statins)
Interferon alpha
Interleukin-2
Iodinated contrast agents
Riluzole

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## Neostigmine IV Infusion for Myasthenia Gravis

The first-line cholinesterase inhibitor for myasthenic crisis is pyridostigmine, administered enterally (oral or by enteral tube). It has poor absorption and a short half-life. Despite this, an effective enteral dosage regimen can be found for most patients. In a minority, unreliable absorption may hinder effective symptom control. Neostigmine, administered by IV infusion, may be an option in these cases.

### Key Points

Indications	<p>Myasthenic crisis in which symptomatic treatment with a cholinesterase inhibitor is indicated to avert intubation or facilitate liberation from invasive ventilation, but where enteral pyridostigmine is insufficiently effective due to unreliable absorption or unavailability of the enteral route.</p> <p>This is to be prescribed under the direction of a consultant in neurointensive care with support from a consultant neurologist.</p>
Starting Dose	Based on current total daily enteral pyridostigmine dose – see below for details
Dose range	60–360 micrograms/hr
Preparation and administration	<p>Neostigmine 2.5 mg/1 mL solution for injection ampoules.</p> <p>Dilute two 2.5 mg/mL ampoules to 50 mL (100 micrograms/mL) with sterile water.</p> <p>Set as 'DRUG X' on infusion pump.</p>
Adverse effects	<p>Common or important adverse effects include flushing, dizziness, drowsiness, diarrhoea, excess secretions (bronchial, oropharyngeal, gastrointestinal), bronchospasm, cardiac arrhythmias (bradycardia, atrioventricular block) and hypotension. Patients with coronary artery disease may be at increased risk of adverse cardiovascular effects, including cardiac ischaemia.</p> <p><b>Atropine or glycopyrronium should be available at the bedside.</b></p>

### Starting Dosage

To convert the total daily enteral pyridostigmine dose (mg) to an hourly IV neostigmine infusion rate (micrograms/hr), follow these steps:

- Calculate the patient's current **total daily enteral pyridostigmine dose**. For example, if the patient is currently taking pyridostigmine 60 mg orally 4-hrly, his/her total daily dose is 60 mg/dose × 6 doses/day = 360 mg/day.
- Refer to the **Dosage conversion table**, below, to identify the starting IV infusion rate.

Total daily enteral pyridostigmine dose	Hourly IV neostigmine infusion rate	Pump rate
180 mg/day orally	60 micrograms/hr IV	0.6 mL/hr
210 mg/day orally	70 micrograms/hr IV	0.7 mL/hr
240 mg/day orally	80 micrograms/hr IV	0.8 mL/hr
270 mg/day orally	90 micrograms/hr IV	0.9 mL/hr
300 mg/day orally	100 micrograms/hr IV	1.0 mL/hr
360 mg/day orally	110 micrograms/hr IV	1.1 mL/hr

**Dosage conversion table:** Starting doses for IV neostigmine infusion based on current total daily enteral pyridostigmine dose. \*Pump rates assume dilution of 5 mg to 50 mL (100 micrograms/mL).

### Dosage titration

The infusion rate may be titrated against effects (therapeutic and adverse) under direction of the neurointensive care consultant, with neurology advice. An interval of at least 4 hours should be allowed between dosage adjustments to observe the effects at steady state. A longer interval may be necessary in patients with impaired renal function.

### Stopping treatment

Patients who require endotracheal intubation for myasthenic crisis do not necessarily need a cholinesterase inhibitor while they are receiving invasive ventilation, and this may contribute to problematic respiratory secretions. In patients who are not expected to be extubated imminently, consideration should be given to suspending it. Once disease activity has been reduced with immunomodulatory treatment and the patient is successfully extubated, it may be reintroduced if necessary, or earlier if required to facilitate extubation.

IV neostigmine should be stopped in favour of oral pyridostigmine administration as soon as viability of the enteral route is re-established. The **Dosage conversion table** may be used

to convert the patient's current stable IV neostigmine infusion rate back to an equivalent enteral pyridostigmine total daily dose. Divide this as appropriate for the intended dosage interval.

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## 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 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.

Use of IV hydrocortisone in the neurointensive care unit	
<b>Patient identification</b>	Sustained, high vasopressor requirement (e.g. noradrenaline >0.2 micrograms/kg/min) despite adequate volume resuscitation.
<b>Initial bolus dose</b>	Hydrocortisone 100 mg IV as bolus
<b>'Steroid responder'</b>	<p>Patients who have a <b>≥20% reduction in vasopressor requirement*</b> to achieve the same blood pressure target 4 hours after a 100-mg bolus dose, should be started on a hydrocortisone infusion at a dose of 10 mg/hr IV.</p> <p>Patients who <b>do not</b> respond to the bolus dose should not receive a continuous infusion.</p> <p>*Taking account of the trajectory of the underlying process, as applicable</p>
<b>Withdrawal and weaning</b>	The hydrocortisone infusion should be stopped or weaned 6 hours after withdrawal of vasopressors. Provided the duration of infusion was less than 14 days and the patient is not otherwise expected to have adrenal suppression (e.g. because of prior corticosteroid therapy), it can be stopped without a weaning period.

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## 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.

Currently available anticoagulant drugs			
Drug	Main inhibitory target(s)	Main licensed indications	Potential reversal strategies (in patients with major bleeding or requiring emergency surgery*)
<i>Oral vitamin K antagonists</i>			
Warfarin, acenocoumarol and phenindione	Vitamin K dependent clotting factors (II, VII, IX, X)	AF; prosthetic heart valves; VTE	Dried prothrombin complex 25–50 units/kg IV <b>and</b> phytomenadione (vitamin K <sub>1</sub> ) 5 mg IV, as the effect of PCC alone is short-lived
<i>Direct oral anticoagulants</i>			
Dabigatran	FIIa	VTE; non-valvular AF	Always seek specialist advice. Consider tranexamic acid (1 g IV 3 or 4 times/day) ± prothrombin complex concentrate. Consider activated charcoal if a dose was taken within the last 2 hours. Reversal agents (idarucizumab for dabigatran; andexanet alfa for FXa inhibitors) have been licensed but, at the time of writing, are not widely available. Dabigatran (but not the others, which are extensively protein-bound) may be removed by haemodialysis or haemofiltration.
Rivaroxaban	FXa	VTE; non-valvular AF; ACS	
Apixaban	FXa	VTE; non-valvular AF	
Edoxaban	FXa	VTE; non-valvular AF	
<i>Heparins and derivatives</i>			

Unfractionated heparin	FIIa and FXa	VTE; ACS; thromboprophylaxis; extra-corporeal circuits; bridging therapy	Protamine 1 mg per 100 units of heparin to max 50 mg, given IV at max 5 mg/min
Low molecular weight heparins (dalteparin, enoxaparin, tinzaparin)	FXa (and FIIa, less so)	VTE (especially in cancer); ACS; thromboprophylaxis	Protamine may be given but its effect is partial and unreliable (dose 1 mg per 100 units of LMWH to max 50 mg, given IV at max 5 mg/min). A reversal agent (andexanet alfa) has been licenced but is not yet available (discuss with haematology for latest guidance).
Fondaparinux	FXa	Thromboprophylaxis; VTE; ACS	Seek specialist advice. A reversal agent (andexanet alfa) has been licenced, but is not yet widely available.
<b>Intravenous anticoagulants</b>			
Danaparoid	FXa	Thromboprophylaxis in surgical patients; heparin-induced thrombocytopenia	No reversal agent. Protamine has minimal effect.
Argatroban	FIIa	Heparin-induced thrombocytopenia	Factor concentrates (PCC/FFP) may be effective — seek specialist advice
Bivalirudin	FIIa	ACS	No reversal agent, but its half-life is short
<p>*Always seek specialist haematology advice, especially for bleeding associated with 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.</p> <p>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.</p>			

## 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 K<sub>1</sub>) 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.

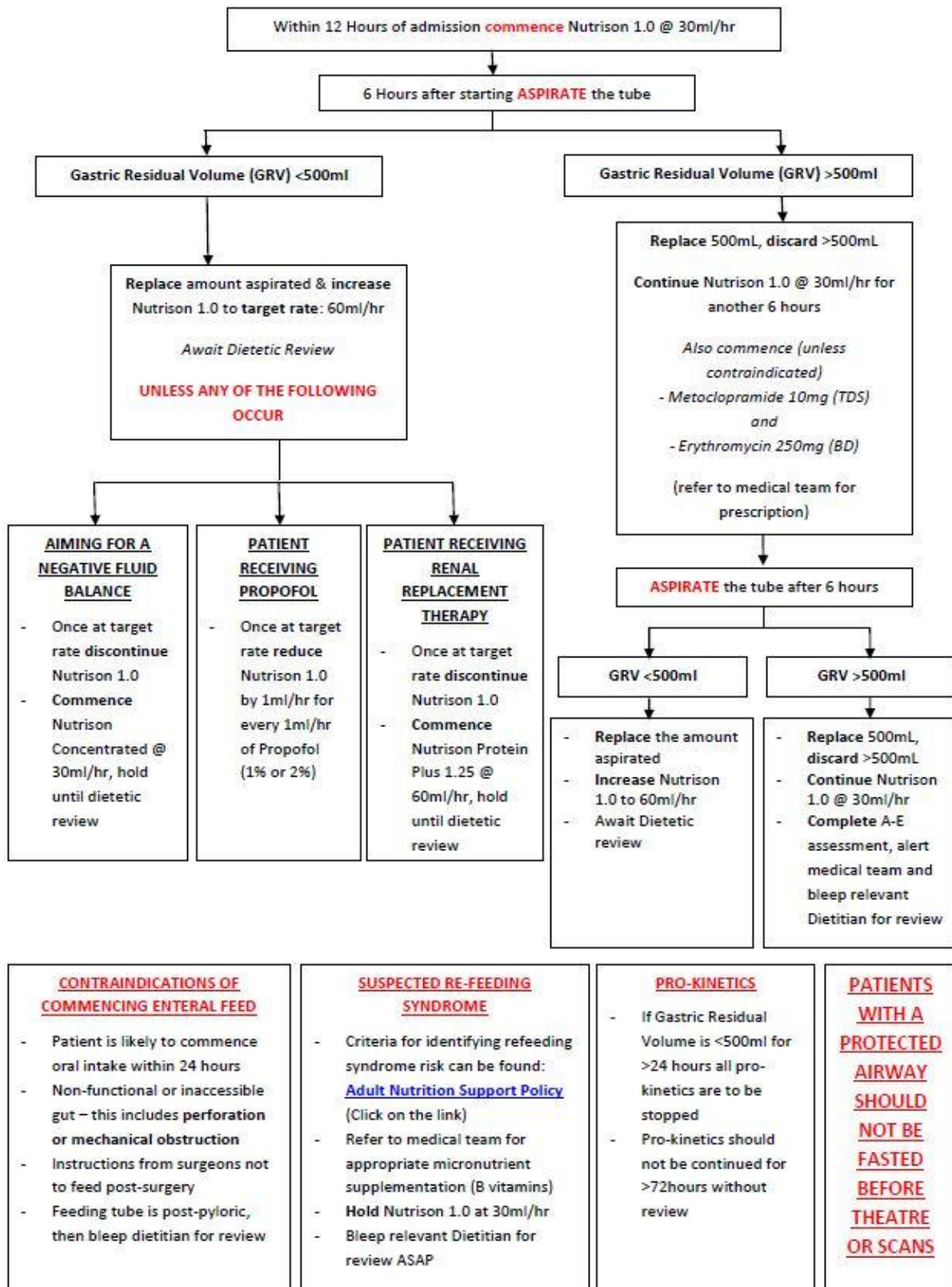
**Thromboelastography (TEG)** may be very useful in the assessment of coagulopathy and to guide its treatment. TEG analysers can be found in the cardiac intensive care unit, general intensive care unit and theatres. If you are unfamiliar with TEG, seek training from senior colleagues in how to perform 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

1. Keeling D et al. Peri-operative management of anticoagulation and antiplatelet therapy. A British Society for Haematology Guideline (01/08/2016). Available online: <http://www.b-s-h.org.uk/guidelines/guidelines/peri-operative-management-of-anticoagulation-and-antiplatelet-therapy/> (accessed 18/01/2017).
2. Makris M, et al. Guideline on the management of bleeding in patients on antithrombotic agents. Br J Haematol 2013;160:35-46.

# Enteral feed Pathway

## Intensive Care Gastric Enteral Feed Pathway



Written by Elizabeth Viner-Smith (Critical Care Lead Dietitian) & Faye Crathern (Senior Staff Nurse & Nutrition Link CTICU)



# 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<sup>®</sup> device) should be a routine part of management wherever there is haemodynamic instability or any uncertainty about volume status and its management.

Normal reference ranges for sodium and osmolality	
Serum sodium concentration	133–146 mmol/L
Serum osmolality	275–295 mmol/kg
Urinary sodium concentration (spot sample)	20–40 mmol/L (varies with Na <sup>+</sup> intake and fluid status). Can be checked on ABG machine using a normal non-heparinised syringe.
Urinary osmolality	100–1400 mmol/kg (should be interpreted in light of the serum osmolality and volume status, which indicate what the 'appropriate' urine osmolality should be)

## 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.

## Hypernatraemia

### Definition and causes:

Hypernatraemia 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.

Causes of hypernatraemia			
Mechanism	Cause(s)	Diagnosis	Management
Sodium excess	Administration of hypertonic sodium chloride or bicarbonate	Clinical context (hypertonic fluid administration)	Moderate hypernatraemia (Na <sup>+</sup> 146–155 mmol/L) is generally tolerated in patients with intracranial hypertension. See notes below for further discussion.
Insufficient water intake ('simple dehydration')	Impaired thirst sensation	Thirst; clinical signs of dehydration; oliguria; negative fluid balance; disproportionately elevated serum urea concentration, relative to creatinine	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).
	Inability to access sufficient water		
	Iatrogenic water under-replacement		
Loss of hypotonic fluid	Cranial diabetes insipidus	Polyuria (>200 mL/hr for ≥2 hrs), rapidly rising serum sodium concentration and inappropriately dilute urine (urine osmolality <300 mmol/kg, for which specific gravity <1.01 is a simple bedside surrogate)	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.

Osmotic diuresis due to mannitol	Clinical context (mannitol administration); polyuria; urine osmolality $\geq 300$ mmol/kg and specific gravity $\geq 1.01$	Replace volume loss with 0.9% sodium chloride
Nephrogenic diabetes insipidus	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	Depends on the cause and clinical context; seek advice

### Hyponatraemia due to hypertonic fluid administration

Invariably, hypertonic sodium chloride infusion causing hyponatraemia occurs in the setting of life-threatening raised intracranial pressure (ICP). In this case, moderate hyponatraemia ( $\text{Na}^+$  146–155 mmol/L) is tolerated, since correction (especially if too rapid) may cause a rebound elevation of ICP. Hyponatraemia 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 hyponatraemia 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 hyponatraemia 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 ADH secretion and the consequences thereof; the difference is in *why* ADH is secreted.

In CSW, the primary disturbance is natriuresis (the cause of which is not fully understood but may involve excess secretion of natriuretic 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  $\text{Na}^+$   $>30$  mmol/L, urine osmolality  $>$  serum osmolality), a reasonable approach is to:

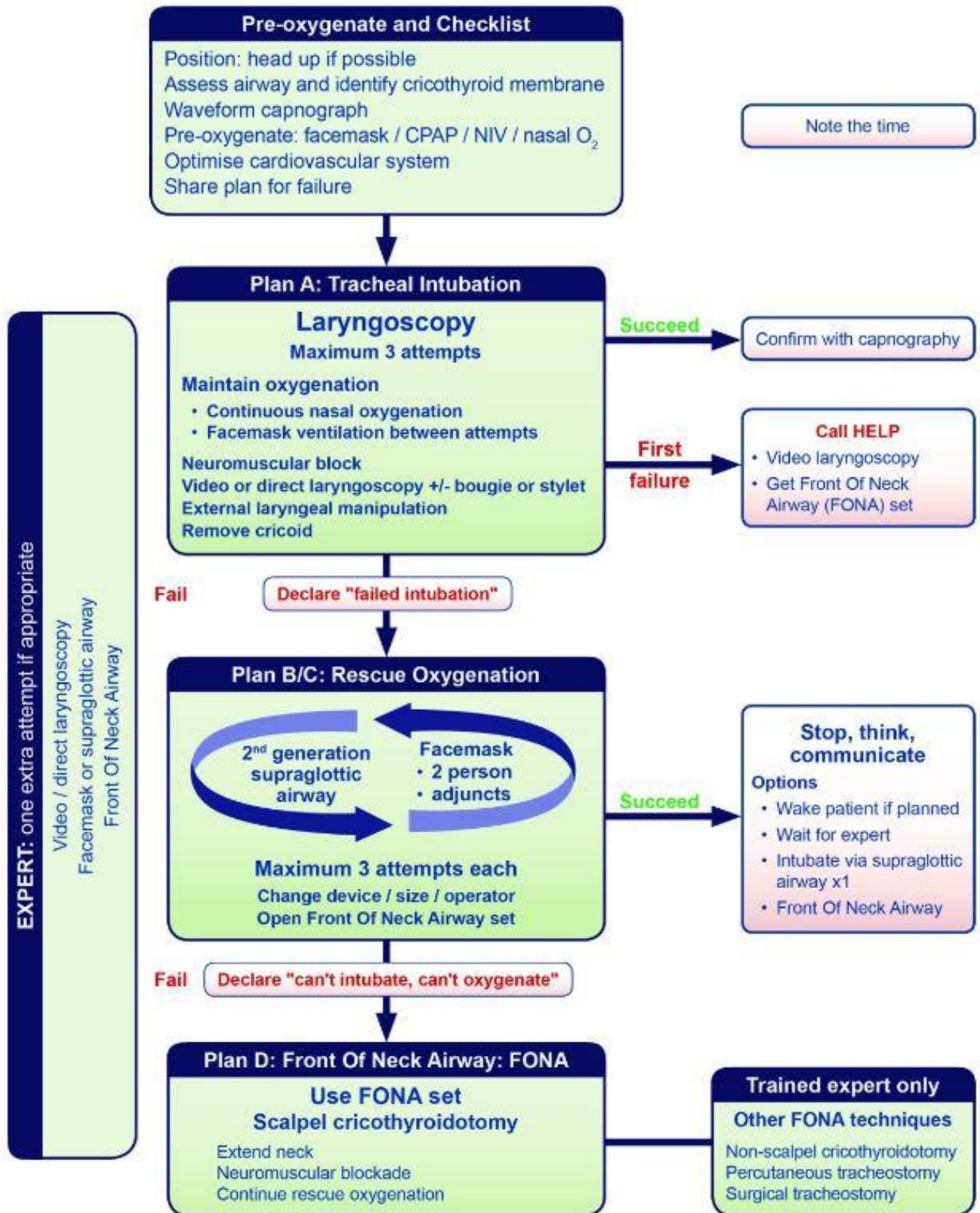
1. Supplement enteral sodium intake with sodium chloride (e.g. as Slow Sodium<sup>®</sup> (600-mg tabs), 2–4 tabs 6-hrly)
2. Consider infusion of 0.9% sodium chloride (e.g. 100 mL/hr) initially, as a diagnostic and (potentially) therapeutic intervention
3. If hyponatraemia improves with 0.9% sodium chloride 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 no 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

1. Yeates KE et al. Salt and water: a simple approach to hyponatremia. *CMAJ* 2004; 170:365-9.
2. Sherlock M et al. The incidence and pathophysiology of hyponatraemia after subarachnoid haemorrhage. *Clin Endocrinol* 2006; 64:250–254.
3. Silva ADH et al. Electrolyte and endocrine disturbances. In: Smith MM et al (Eds.). *Oxford Textbook of Neurocritical Care*. Oxford University Press (2016).

# Algorithm for difficult intubation on NICU

In NICU there is a dedicated airway trolley





# Can't Intubate, Can't Oxygenate (CICO) in critically ill adults



The Faculty of  
Intensive Care Medicine



## CALL FOR HELP

Declare "Can't Intubate, Can't Oxygenate"

## Plan D: Front Of Neck Airway: FONA

Extend neck

Ensure neuromuscular blockade

Continue rescue oxygenation

Exclude oxygen failure and blocked circuit

### Scalpel cricothyroidotomy

- Equipment:**
1. Scalpel (wide blade e.g. number 10 or 20)
  2. Bougie ( $\leq 14$  French gauge)
  3. Tube (cuffed 5.0-6.0mm ID)

#### Laryngeal handshake to identify cricothyroid membrane

#### Palpable cricothyroid membrane

- Transverse stab incision through cricothyroid membrane
- Turn blade through 90° (sharp edge towards the feet)
- Slide Coudé tip of bougie along blade into trachea
- Railroad lubricated cuffed tube into trachea
- Inflate cuff, ventilate and confirm position with capnography
- Secure tube

#### Impalpable cricothyroid membrane

- Make a large midline vertical incision
- Blunt dissection with fingers to separate tissues
- Identify and stabilise the larynx
- Proceed with technique for palpable cricothyroid membrane as above

#### Trained expert only

#### Other FONA techniques

- Non-scalpel cricothyroidotomy
- Percutaneous tracheostomy
- Surgical tracheostomy

#### Post-FONA care and follow up

- Tracheal suction
- Recruitment manoeuvre (if haemodynamically stable)
- Chest X-ray
- Monitor for complications
- Surgical review of FONA site
- Agree airway plan with senior clinicians
- Document and complete airway alert

## References

1. DAS Guidelines 2017

## 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 24hours 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 NICU:

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

# 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

- |          |                           |                          |
|----------|---------------------------|--------------------------|
| Targets: | pO <sub>2</sub> 8-14 kPa  | <input type="checkbox"/> |
|          | pCO <sub>2</sub> 5-6.5kPa | <input type="checkbox"/> |

#### 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 cmH<sub>2</sub>O for 30-50 secs)
- Set PEEP 8-10 cm H<sub>2</sub>O
- Lung Protective ventilation (TV 6-8mls/kg, Peak pressure ≤30cm H<sub>2</sub>O)
- Repeat recruitment manoeuvre if pO<sub>2</sub> ≤ 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

- |          |                              |                          |
|----------|------------------------------|--------------------------|
| Targets: | Urine output 0.5-2.5ml/kg/hr | <input type="checkbox"/> |
|          | Na 135-150mmol/L             | <input type="checkbox"/> |
|          | K+ 4.0-5.5mmol/L             | <input type="checkbox"/> |
|          | Mg > 0.8mmol/L               | <input type="checkbox"/> |
|          | Ca ionised 1.0-1.3mmol/L     | <input type="checkbox"/> |

#### 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**

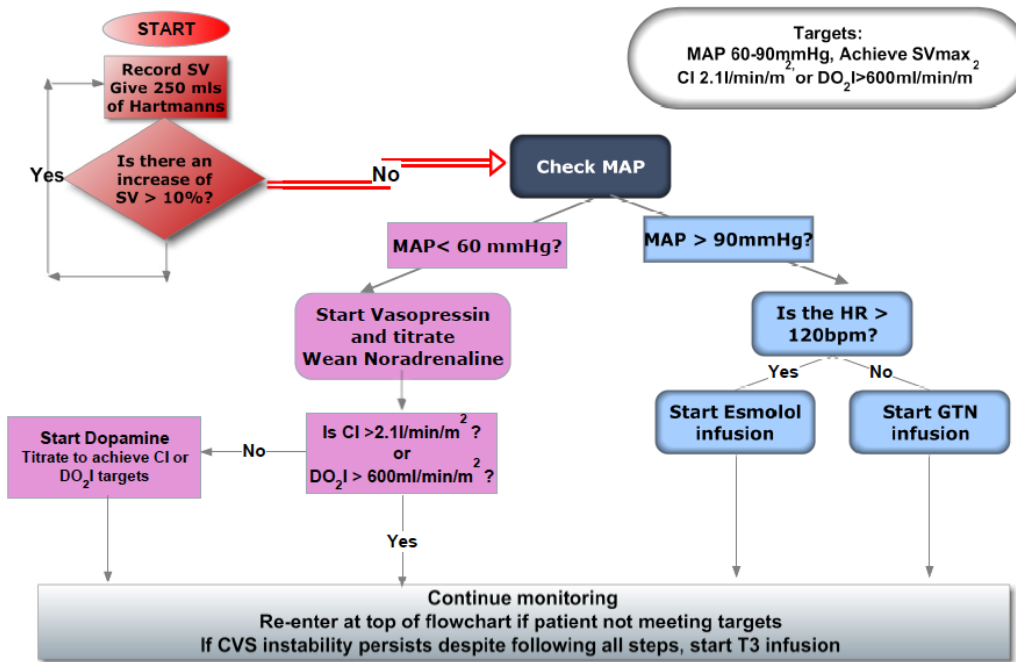
### Hormones and Haematology

- |          |   |                          |
|----------|---|--------------------------|
| Targets: | BM 4.0 – 9.0 mmol/L                       | <input type="checkbox"/> |
|          | Hb ≥ 8g/dL, Plt > 50 x 10 <sup>9</sup> /L | <input type="checkbox"/> |
|          | INR < 2.0, APTTR < 1.5, Fib > 2.0g/L      | <input type="checkbox"/> |
|          | Temperature 35.5 – 37.5                   | <input type="checkbox"/> |

#### 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)

## Cardiovascular algorithm



Ashleigh Sherrington, Argyro Zoumprouli May 2012

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 imminent Deaths to specialist Nurses for organ donation is mandatory**

**100% Brain Stem Testing is aimed**

# Organ Donation

## Organ donation during COVID-19

**There are three things we need if we are to continue to offer lifesaving transplants to those in desperate need. These are:**

**1. Exclusion of COVID-19 in donors.**

The completion of the COVID-19 SNOD Checklist is absolutely required and testing for COVID-19 must be completed before any potential donor can proceed. Latest requirements always available at <https://www.odt.nhs.uk/deceased-donation/covid-19-advice-for-clinicians>

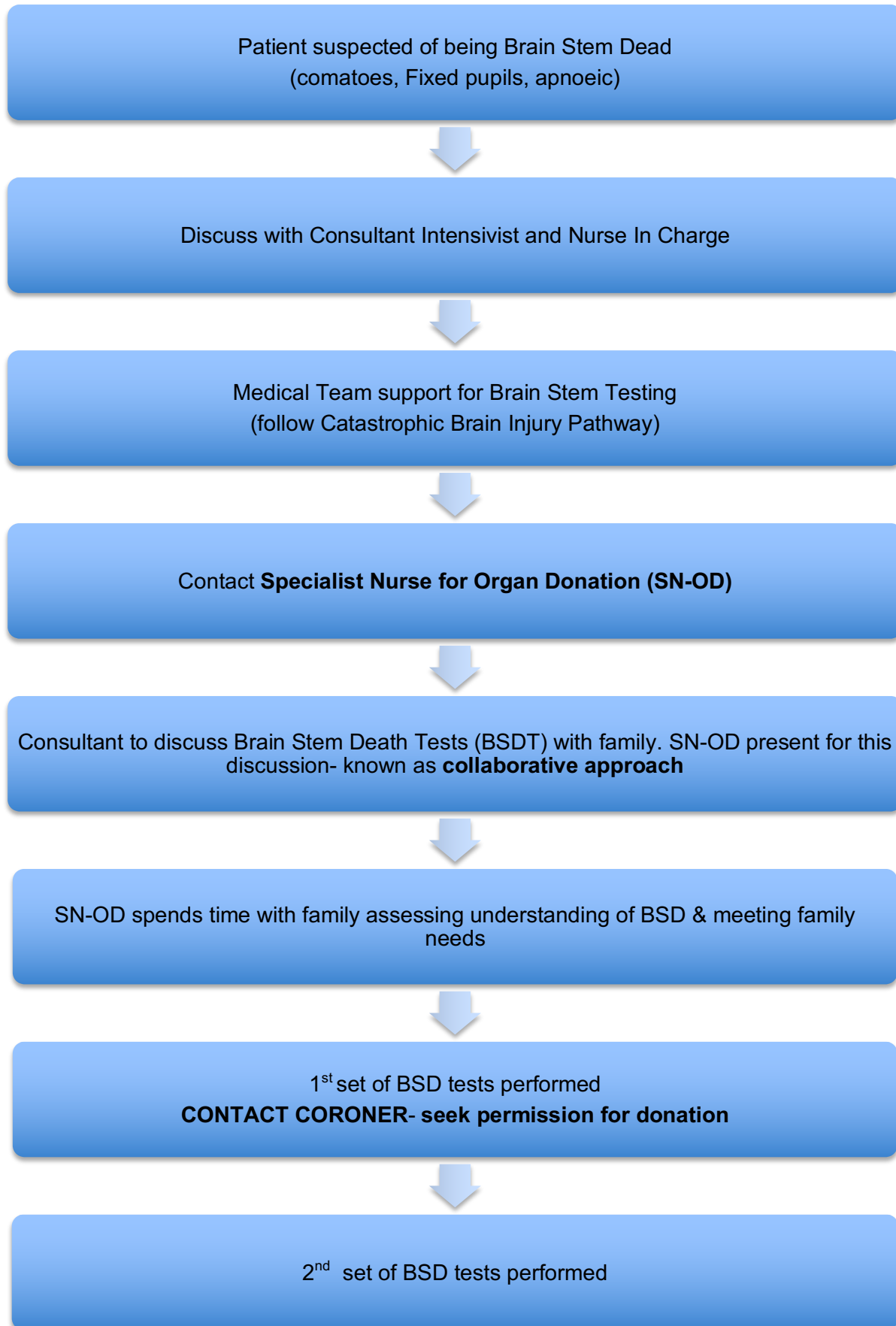
**2. Personal leadership and availability for communication by medical staff in the donor's ICU.** Every donation in the UK is now being progressed or stood down on a case by case basis. Donation is not occurring without ongoing local ICU leadership and discussion, often requiring direct input from Dr Ushiro-Lumb, NHSBT's virologist.

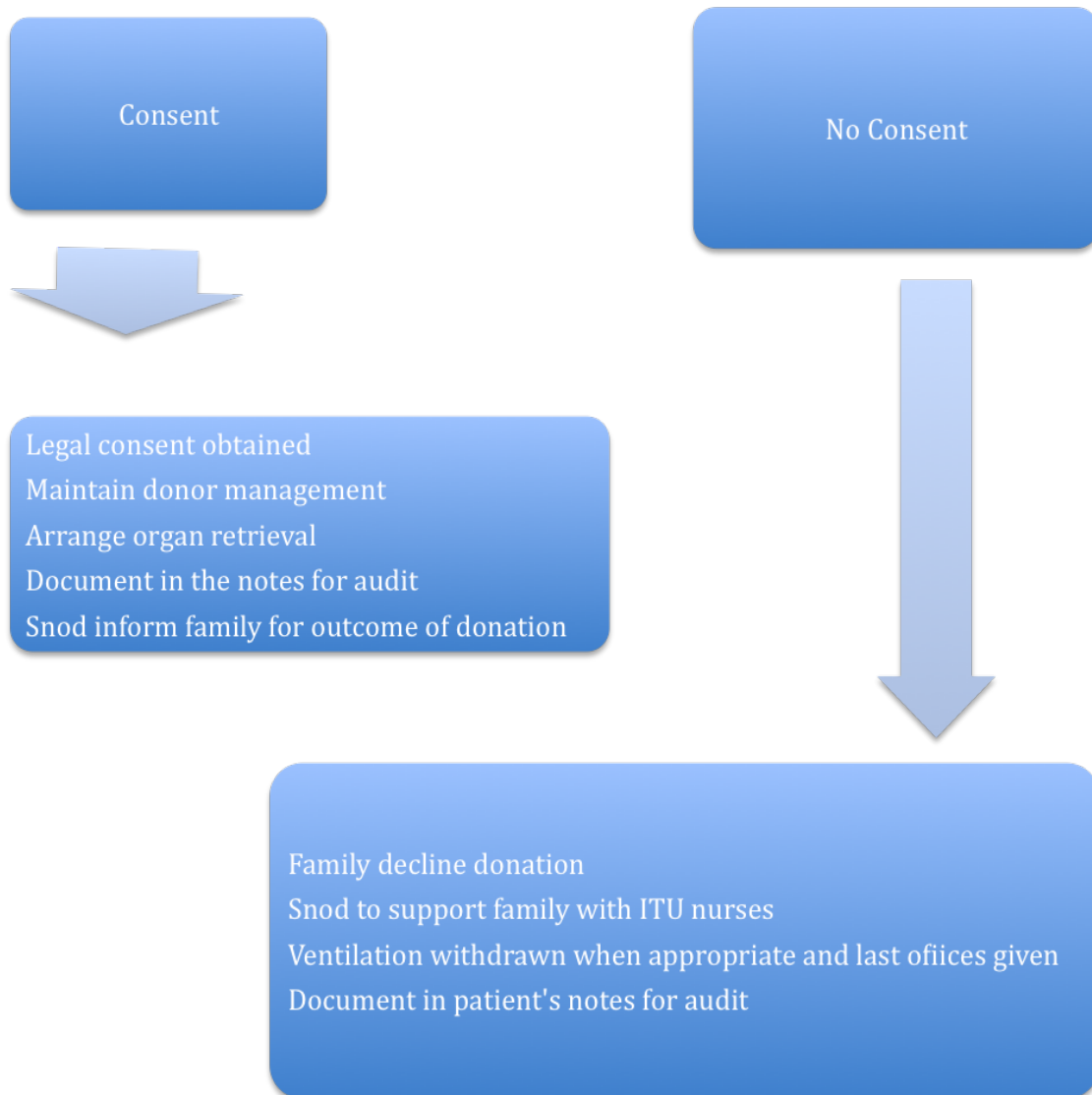
**3. Please refer as normal.**

- To free up ICU beds, we made the difficult decision to drop the upper age for both DBD (age 60) and DCD (age 50). These acceptance criteria may well change again, perhaps even altering on a region by region basis. The important point is that the SN-OD or Team Manager taking the referral will have the latest acceptance criteria.
- There is no longer any expectation that SNODs complete the PDA. This means that referral, and the information you give us by phone, is one of the only ways we have for decision-making and advising government and anxious transplant list patients.
- Capacity constraint is a reason for donation not to be explored further but please refer.

We know many SNOD/SRs are offering their services to their embedded or local ICU. As well as providing ICU nursing care, our specialist nurses have expert skills in communication and supporting bereaved families, even via the phone. We will be supporting them through this process, but we also ask you to reach out to them where possible.

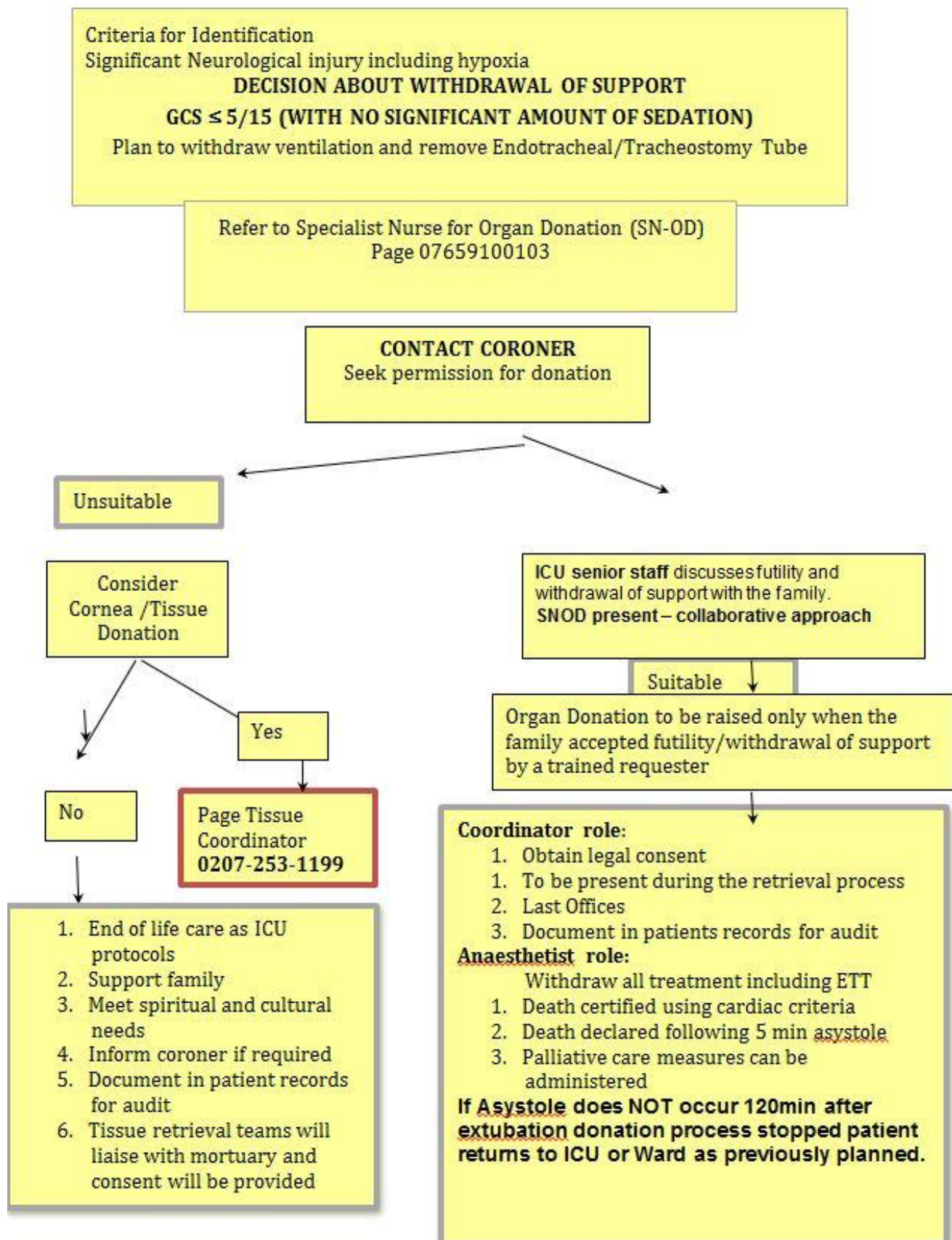
## Identification and Referral of DBD donor





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





## Advice on how to minimise risk aerosol generation during apnoea testing

During the COVID-19 pandemic it is essential to ensure safety from cross infection during an aerosol generating procedure (AGP).

While the apnoea test for diagnosing death using neurological criteria does not meet the specific [PHE definition of an AGP](#) there is enough in common with other airway interventions to suggest the apnoea test is best considered an AGP.

### Steps to minimise risk aerosol generation during apnoea testing

- **Follow local PPE infection control guidance in local ICU for an aerosol generating procedure.**
- **Turn off ventilator**
- **Clamp endotracheal tube <sup>a</sup>**
- **Attach Mapleson C with an attached HMEF with oxygen off**
- **Unclamp endotracheal tube**
- **Turn on oxygen**
- **Perform apnoea test**
- **Turn off oxygen**
- **Clamp endotracheal tube <sup>a</sup>**
- **Reattach ventilator**
- **Unclamp endotracheal tube**
- **Turn on ventilator and carry out a recruitment manoeuvre**

#### Note

a repeat clamping has been associated with delamination of endotracheal tubes and subsequent complete obstruction and need for reintubation in both the UK and France. If clamping considered necessary, proceed with care.

Approved by National Organ Donation Committee - April 2020

## Recommended practice for airway management in WLST for DCD during COVID-19 pandemic

Deceased donation is proceeding in the patient because, as far as can be known, COVID-19 infection is considered very low risk. This conclusion will have been based on a comprehensive SNOD screening history and negative respiratory tests. Where there is any doubt discussion with NHSBT's virologist will have occurred.

**Always ensure comfort and dignity of the patient are prioritised. Follow all your other usual end of life care practices.**

**Always follow local PPE infection control guidance.**

Two Recommended Options -Choose the most appropriate for your unit and patient.

Extubation

May be considered to be preferable if family present.

Less likely to artificially prolong the dying process.

Extubation is a familiar practice in many ICUs and allows the most natural dying process.

Leaving endotracheal tube in situ

Option if there is concern regarding aerosol and droplet generation.

### Steps

Stop ventilator

Disconnect the ventilator leaving the HMEF attached at the patient side

If HMEF obviously soiled or waterlogged – replace.



Disconnect here

Approved by National Organ Donation Committee - April 2020